On Typhoid Fever.

Part 1.

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I hereby certify that the thesis on Typhoid Fever, sent by me for the examination of the Medical Faculty of Edinburgh University, is my own composition.

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Cardiff. April 25. 1890

I hereby certify that
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my assistant for a
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William Taylor M.D. Edin
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On Typhoid Fever.

There is probably no disease of greater interest and importance to the General Practitioner of Medicine, than the endemic disease, known by the name of typhoid fever.

It confronts him often on the threshold of his career; and not uncommonly he is then, although well educated in the science of medicine, not in a position to diagnose the disease, as it presents itself to him in practice.

The young practitioner may mistake the case for a bilious attack, or other gastric derangement, and administer a purgative; a mode of treatment which may greatly increase the severity of the abdominal symptoms, by inducing an exhausting diarrhoea that cannot easily be stopped.

Sir Wm. Jenner has said that "More lives may be saved by the judicious treatment, and more lives lost by the improper treatment of

"of typhoid fever, than of any other acute disease".[1]

It is evidently therefore a disease with which the general practitioner should be well acquainted.

But enteric fever is so insidious in its onset, and so varied in its many manifestations, that even the most careful and experienced physicians, are sometimes at considerable difficulty in arriving at a correct diagnosis. Thus for example, a case occurred in which an experienced physician who was called into consultation, expressed the opinion that the patient, who had been ill for a fortnight, was suffering from typhoid fever. The case was altogether obscure, and nothing else could be found to account for the peculiar course of the temperature, until a deep seated abscess pointed in the lumbar region. The patient made

1. Sir Wm. Jenner. Midland Medical Society. Nov. 4th 1849. (Quoted by Dr. J. O. Affleck. Brit. Med. Jour. May 17th 1884. p 947.)

a perfectly good recovery, after the abscess was opened. There was no diarrhoea, and the characteristic rose coloured lenticular spots did not appear. Nevertheless the question is open, as to whether this was not a case of typhoid fever complicated by circumscribed peritoneal abscess similar to a case described by Dr. Charles Murchison.[1]

The name typhoid, as applied to this fever, has been subjected to a good deal of criticism by authorities. The Royal College of Physicians, in their nomenclature, call the disease, enteric fever; and in this country the disease is usually designated by that name. But Murchison criticises both of these names, and particularly the word typhoid.[2] He considers the name unscientific, as it literally means like typhus.

1. Murchison on Continued Fevers. 3rd Edition p. 381.
2. Murchison. page 419. Op. Cit.

Case LXXV.

Such a method of naming a disease he holds to be "at variance with all precedent in the accepted nomenclature of species in science." He further points out that the name is confusing. Because the word typhoid is used, adjectively, to designate the nature of a group of clinical phenomena, which may arise in the course of other diseases — for example, "the typhoid stage" of typhus — as well as during an attack of enteric fever. Again typhoid symptoms do not occur in all cases of enteric fever. With regard to the name enteric, it is objected, that in some cases of enteric fever, the symptoms referable to the bowels, are so little in evidence, that a physician might hesitate to apply the term enteric or bowel fever, to a case in which abdominal symptoms were conspicuous by their absence. Dr. Murchison therefore suggested the name Pythogenic Fever, from —

(πυθογενὴς, from πύθων (πύθομαι, putresco) and γεννάω).[1] But it is questionable whether at the present day, all authorities would be satisfied with the name Pythogenic; as many hold the view, that the specific cause of typhoid fever is a minute organism, demonstrated as occurring in the diseased intestines.

The bacillus in question was exhibited at the International Health Exhibition in 1884, in the Biological Laboratory, under the superintendence of Mr. Watson Cheyne[2]. The organism is described as "a small oval bacillus, which occurs constantly in great numbers, in the ulcers of the intestinal walls, at the acute stage of the disease, also forming plugs in the liver and spleen". The difficulty seems to be that of inducing typhoid fever in other animals, by the introduction of

1. Murchison on Continued Fevers p. 419.
2. British Medical Journal. Sept. 20th 1884. p. 579.

of the virus, without which, it cannot be definitely stated, that the bacillus is the cause of the disease. Thus for example: Dr. Murchison fed a pig for six weeks on the fresh stools of patients suffering from enteric fever.

The stools were mixed with barley meal, and given two or three times a day. The animals suffered no inconvenience, but got very fat, and when killed had perfectly healthy intestines! This difficulty is said to have been overcome, by neutralising the acidity of the gastric juice by means of Carbonate of Soda, and giving Opium to arrest the peristaltic action of the bowels, then introducing a pure cultivation of the typhoid bacillus into the stomachs of animals through a catheter. This operation usually results in the death of the animals operated upon, in which after death the intestinal walls are

1. Murchison on Continued Fevers. p. 498.

are found to be inflamed and altered; the intestines also containing large numbers of these bacilli.[1.]
It is unfortunate that in the case of the animals mentioned by Professor Charteris, in his lecture,[2.] no definite lesions were demonstrated, similar to those described by Dr. J. R. Roberts,[3.] of the Indian Medical Service, in the Indian Medical Gazette for June 1889. In the cases described by that gentleman, there were "enlarged spleen, swollen mesenteric glands, and bowel lesions closely resembling those which occur in the human subject during typhoid fever". Mr. J. Bland Sutton, also identified a disease, which occurred in Lemurs, Monkeys and Beavers, with enteric fever.[4.] He observed lesions after death in

1. British Medical Journal Dec.r 8.th 1888. p. 1274.
2. British Medical Journal Dec.r 8.th 1888. (Ibid.)
3. British Medical Journal Augt. 3.rd 1889. p. 824.
4. British Medical Journal May 9.th 1885. p. 944.

Canadian Beavers, similar to those which occur in the human subject after death from enteric fever. And many other instances are recorded of the occurrence of the disease amongst the lower animals. Thus for example at Natal in 1880, typhoid fever prevailed amongst dogs and oxen. The fact was corroborated by post-mortem examinations made by W. G. Thistle and Dr. Saunders, A. M. D. The observation is made that "In 1879-"-1880, in fresh encampments, the cattle were first attacked, and then after a few weeks, the men were down with genuine typhoid." 1. Therefore although Dr. Murchison states that there is no clear proof that any of the lower animals are liable to enteric fever; and although the statement, that the acute infective fever of pigs, known as swine fever, as well as the "cattle plague"

1. British Medical Journal. Augt. 10th 1889. p. 339.

"cattle plague", were pathologically analogous to human enteric fever, has been proved to be erroneous[1]; still it seems tolerably well ascertained, that enteric fever may occur among the lower animals.

In the outbreak of Typhoid Fever which occurred at Cluny in France,[2] the drinking water was contaminated, and Dr. Rodet cultivated typhoid bacilli from samples of it. Similar observations have been reported by others, with regard to the drinking water in certain epidemics. Dr. Alfred Carpenter, has detected the microbes of typhoid fever in sewer-gas.[3]

The presence of the typhoid bacillus in drinking water, and the emanations from sewers, gives support to the view that the materies morbi of typhoid fever, is of a specific bacillary nature.

The term pythogenic, suggested by Dr. Murchison, has not come into general use.

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1. Murchison on Continued Fevers, p. 498.
2. Lancet Oct: 22nd 1887. p. 842.
3. Brit: Med: Jour: June 22nd 1889. p. 1404.

But the different names by which typhoid fever has been known at different times and in different countries are in themselves sufficient to form a catalogue. They are given in great detail by Dr. Murchison.[1]

It is important to remember that this disease is called Abdominal Typhus, sometimes Ileo-typhus, by German writers. Also to know such synonyms, as, Gastric Fever, Bilious continued fever, Gastro-entérite, Dothiénentérite, Endemic fever, Autumnal or Fall fever, Infantile remittent fever, slow-fever, low-fever, common continued fever, Nervous or Hysteric Fever - Fever on the Spirits, Febris verminosa, and worm fever. Unfortunately not a few cases of typhoid fever are returned in this country and elsewhere, as cases of simple continued fever, leading to great confusion, and errors in statistics.

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1. Murchison on Continued Fevers p.p. 416–418.

The definition of typhoid fever is necessarily difficult, on account of the protean nature of its many manifestations.

Definition. Typhoid fever may be defined as an acute specific disease, usually endemic, but sometimes becoming epidemic, and it is said epizootic.

It is generated in, and propagated by certain forms of decomposing animal matter, containing a specific morbific material, which is usually classed amongst the miasmata. The nature of this materies morbi, however, must still be regarded as uncertain. Although it probably consists either of the "Bacillus Typhosa", or of the products of its vital activities. The morbific material is usually derived from the stools of persons suffering from the disease.

Typhoid fever is characterised by symptoms, which are insidious in their onset. As a rule there is slight general malaise, lasting for a few days, before the febrile

Professor Sir D. Maclagan

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Barnes on Typhoid.

I do not find anything original in the large Thesis but there is a full collection of all that has been written on the matter. Two stars may be given but it does not deserve a gold medal

A.H.

febrile condition becomes definitely established. The onset may be accompanied by feelings of chilliness or slight rigors. Sometimes there is diarrhoea, which may be profuse. The tongue is furred. Often it is white in the centre, and red at the tip and edges. Later in the disease the tongue tends to become dry and brown; sometimes raw-looking, glazed and deeply fissured.

There is a rise of temperature from the first, and the fever is remittent in type. The pulse as a rule is accelerated; but varies much in frequency and strength, in different cases, and at different times of the day in the same case.

Headache and aching of the limbs are complained of from an early stage, and the onset may be accompanied by violent retching and vomiting. Sometimes there is epistaxis. Pain and tenderness of the abdomen with tympanites are frequent symptoms. The tenderness is often more

more marked in the right-iliac region than in other parts of the abdomen; and gurgling can often be felt when pressure is made in that region. The skin is warm with occasional perspirations; and there is an eruption of rose coloured lenticular spots which appears between the 7th and the 14th day. The spots disappear completely on pressure, and come out in successive crops, each lasting for two or three days. The pupils are dilated. The patient is restless and sleepless, and there is often a tendency to delirium, or there may be stupor; but sometimes the mind is clear throughout the attack. There is marked prostration when the disease is advanced. In the earlier stages the prostration is not great, if there is any. The spleen is almost always enlarged. The disease may be protracted to thirty days or longer, and it may be followed by relapses.

Post Mortem. There is enlargement and ulceration of Peyer's Patches, and of the Solitary Glands of the Ileum, with enlargement of the Mesenteric Glands, and of the Spleen.

The following case was attended by the writer, in private practice, and illustrates a case of Typhoid Fever, of average severity.

N. R. Age 9 Years, was sent home from school, ill on the 16th Decr. 1888.

She complained of headache and general malaise. The tongue was covered with a pale yellow fur. She felt thirsty, and the mouth was dry and slimy. There were pain and tenderness in the Right Iliac Region, and diarrhoea. Temperature at 4 pm. 102·4°F. Pulse 96. Respirations 20. There were a few rose coloured, lenticular spots on the abdomen. There have been four motions in the course of the day.

Decr. 17th. The patient spent a restless night. Morning Temperature 102°; Evening Temperature 103·4°. Pulse 100. Respirations 22.

Decr. 18th. There is more marked prostration.

Four liquid motions have been passed during the day. Morning Temperature 102·8°, Evening Temperature 103·8°. Pulse 112. Respirations 24.
Dec^{r}. 19th. The bowels are still relaxed, and the motions characteristic. Morning Temperature 103·4, Evening Temperature 104° Pulse 98. Respirations 18.
Dec^{r}. 20th. Visit at 12 A.M. Appearance peaceful. Sleeps a good deal, and is drowsy. Pulse 106. Temperature 103°. Visit at 3·30 p.m. Very restless and excited. Feels thirsty and complains of abdominal pain. Takes milk well, about two quarts in 24 hours. Visit at 9 P.M. appears flushed, and has a troublesome cough. Vomited at 8·30 P.M. Pulse 112. Temperature 103·6°. Tongue dry and brownish. There are sordes on the teeth, and the lips are parched. The abdominal pain is not so severe.
Dec^{r}. 21st. The patient has slept fairly well; but was a little restless, and "talked in her sleep." During the night she perspired freely after a feeling of great heat. Cough still troublesome. Slight expectoration of rust coloured sputum. Tongue thickly furred. There is a feeling of

great nausea. The pain in the stomach is not so severe. She complains of occipital headache. Visit at 9 P.M. The head is burrowed back in the pillow. The expression of the face is anxious and the features are sunken. The pupils are dilated. The whole countenance indicates suffering. She cries out wildly and has been very restless since the morning visit. She does not take milk so well. The tongue is plastered. She is drowsy, and conscious only when roused, relapsing, almost immediately into unconsciousness.

Dec^{r} 22nd. Morning visit. Patient spent a restless night, with severe headache. She perspired freely during the night. Evening Visit. The headache is better. Has vomited twice since the morning, a very sour smelling fluid mixed up with curdled milk. She has been very excited, endeavouring to get out of bed. The skin is dry. The palms of the hands feel hot and dry. Fresh spots are still appearing on the abdomen. There is marked prostration.

Dec^{r} 23rd Morning visit. Has been very restless during the night, and tried to get out of bed. Face flushed. She is somnolent, and when aroused

complains of a dull headache. Eyebrows contracted. Tongue dry, and brown in the centre. Milk not well taken. Evening visit. No improvement. Has vomited once.

Dec^{r} 24th. Morning visit. Patient very delirious. She moans, and contracts her eyebrows.
Evening Visit. The condition of the patient has not improved.

Dec^{r} 25th. A marked remission of Temperature has taken place, and the patient appears much better.
Evening visit. Not so well.

Dec^{r} 26th. Morning visit. During the night, the patient was wildly delirious. She got out of bed twice. She shouted, and sang snatches of songs, after which she became drowsy. At 6 A. M. she was moaning; but at 9 A. M. she became quieter, and not so delirious. Motions slate coloured, numerous and copious. She is taking Dover's Powder with Bismuth for the diarrhoea. No perspiration. Prostration great. Temperature 103.6°. Evening visit, no improvement.

Dec^{r} 27th. Spent a better night. Conscious. Appetite improved. Skin dry. Complains of pains in all the limbs: particularly the legs.
Evening visit. Improvement still continues.

Dec^{r} 28th. Morning visit. Patient has again

spent a restless night. Complains of pain in the lower part of the abdomen. There is a short hacking cough. Towards evening the cough became very troublesome, and the breathing embarrassed. Temperature 103.8°, Pulse 120, Respirations 38. She moans and complains of pain in the right side. The sputum is scanty and rust coloured: and there are physical signs of a limited pneumonia at the base of the right lung.

Dec^{r} 29th morning visit. Patient much better. Poultices have been applied to the side. Cough still troublesome. Tongue red, dry and glazed There is much emaciation. Eyes sunken.

Evening visit. No change to note in condition of patient. On Dec^{r} 30th and 31st there was little alteration in the patients symptoms, but on the 1st January 1889, a marked improvement began. A favourable remission of the morning temperature occurred, and the tongue became moist. Temperature 99.8°. Evening temperature 101.2°. A further remission took place on the 2nd of January. morning temperature 98.8°. For several mornings after this the temperature was subnormal. On January 3rd the

DISEASE.

Enteric Fever

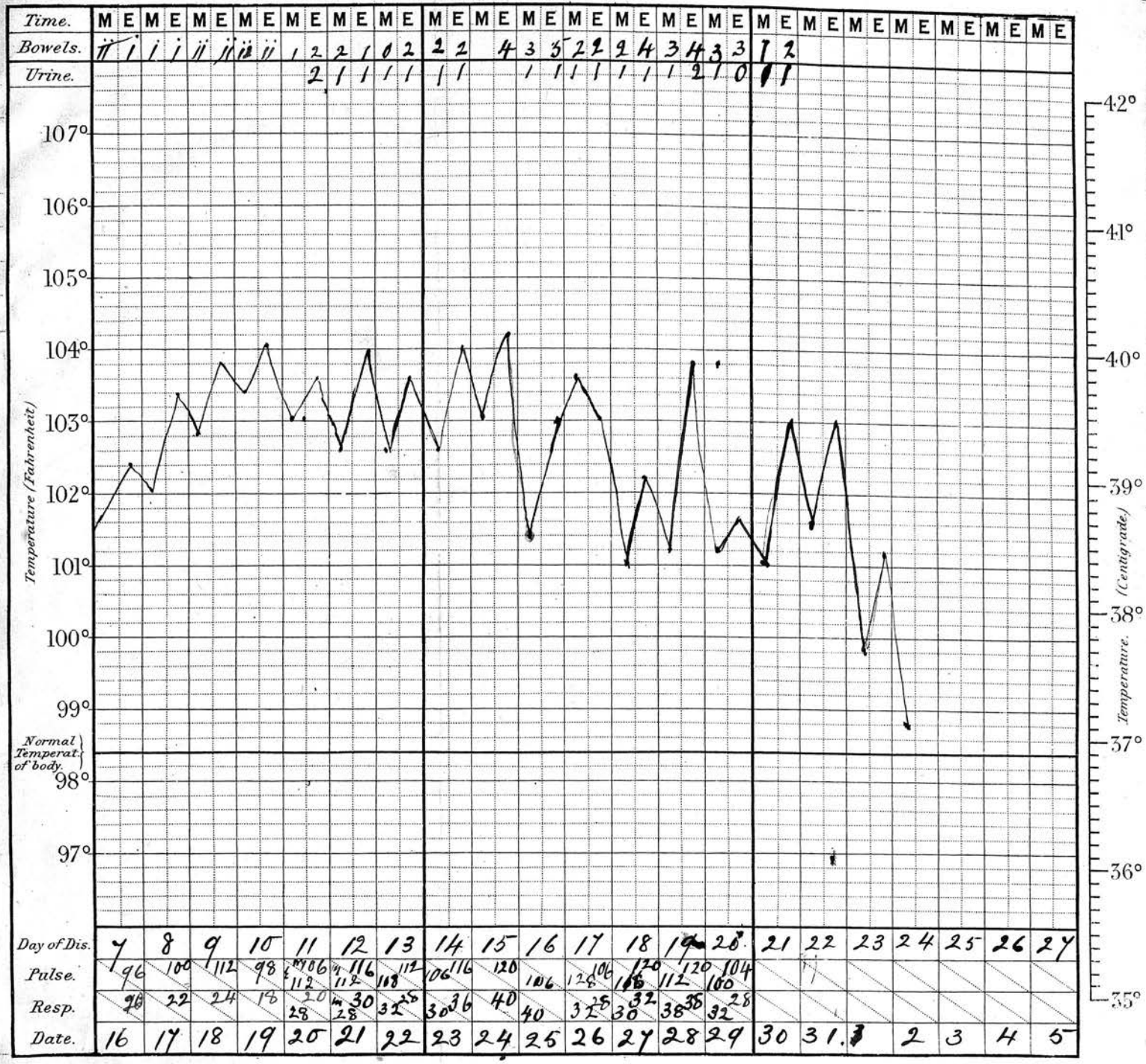
Notes of Case.

Name { Nellie Reece

Age 9 Years

Diet Milk -

Case Book No.



First seen

~~Date of admission.~~

Dec^r 16^th 1888.

Result Recovery

Entered at Stationer's Hall. Printed and Published by Wodderspoon & Co, 7 Serle Street, Lincolns Inn Gould's Clinical Chart.

morning temperature was 98·2°.
January 4th. Still further improvement in all the symptoms.
January 12th. The evening temperature has been normal now for some days. The tongue is clean. Patient apparently convalescent. Takes custards, milk and farinaceous puddings.
The chart which was kept at the patient's house, to mark the progress of the case, is opposite to this page. It is unfortunately not complete.
The chief features of the case were, the development of pulmonary complications, the marked abdominal symptoms, and the severe symptoms referable to the nervous system.
The patient made a perfectly good recovery. The diet during the fever consisted entirely of milk; but demulcent drinks acidulated with lemon etc. were allowed.
The diarrhoea was combated with Dover's Powders principally; but also with enemata of Starch and Opium.

A little Bismuth was administered with some of the Dover's Powders.
A mixture was given to relieve the cough, and poultices were applied to the affected side, for the relief of the pain and other symptoms.
Sponging with tepid vinegar and water, seemed to greatly comfort the patient, when the skin was hot and dry.
No other form of Antipyretic Treatment was attempted. A little wine was administered when the prostration became distressing, and the action of the heart feeble.
It seemed to greatly benefit the patient.

History. Typhoid Fever has probably been known from remote antiquity.
Thus for example Hippocrates described a disease which occurred in Autumn, and presented symptoms closely resembling those of enteric fever.
Dr. Murchison identifies it with the "Hemitritaeus" of Galen; and

"Spigelius speaks of it as common in various parts of Italy, early in the 17th Century".[1]

But it was only at the commencement of the present century, that the pathological anatomy of fever began to be carefully studied. Rost of Paris announced in 1804. that the lesions of Mucous, Gastric, Ataxic and Adynamic fevers, were found in the intestines. In 200 dissections, the intestines were invariably inflamed.[2] Broussais extended Prost's views. He even maintained that in Measles, Variola and Scarlet Fever, death was due to the same "Gastro-Entérite" — a view now generally held to be erroneous.

He advocated copious bloodletting as the treatment. Messrs. Petit and Serres, gave an accurate description of typhoid fever under the name of "Fièvre entéro-mésentérique in 1813.[3]

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1. Murchison on Continued Fevers: 3rd Edition p. 420.
2. Murchison Ib. p. 425.
3. Petit & Serres, 1813. p.p. 159, 165.

But to Bretonneau of Tours the honour is due, of demonstrating that the disease was always located in the solitary and agminated glands of the ileum. He named the disease dothiénentérie. Trousseau in his clinical lectures describes the disease under the name of "dothienenteritis" δοθιὴν ἔντερον.[1]

In 1829, Louis, who added an important contribution to the litterature of the subject, gave the disease the name of Fièvre Typhoïde. Chomel in his Clinical lectures published in 1834, adopted the name given to the disease by Louis, and it has been very generally used ever since.

These works showed that the fever of Paris, was invariably associated with disease of the solitary and agminated glands of the ileum. It was also noted that there was not necessarily any relationship between the severity

1. Trousseau. 1861. Eng. Trans. 1869. Vol. ii.

of the lesions and the intensity of the febrile phenomena. At this time, British observers were constantly remarking, that in most cases of infectious fever the intestines were healthy. This discrepancy arose from the fact that while the French physicians considered that the infectious fever of Camps and armies, was identical with the fever, they had to deal with at Paris, it was not really so; but was a different disease altogether. The fever of camps and armies being typhus fever, whereas the Paris fever was enteric or typhoid fever. Now it seems that the fevers in which the British observers so constantly found the intestines healthy, were cases of true typhus - a disease which is now almost universally considered to be perfectly distinct from typhoid fever - Quite a host of able observers in every part of the world have contributed towards the establishment of the

fact, that the two diseases are distinct. And the successive steps by which the conclusion has been arrived at, is an interesting chapter in the History of Medicine.

Sir Wm Jenner, during from 1849 to 1851, did a great deal by his careful observations, to set aside any doubt that might remain. He confirmed the observations of Gerhard, Stewart and others; and showed that the two fevers did not prevail together, and that they depended on different causes.

He adduced cases to prove that an attack of one fever did not protect from an attack of the other; but that it afforded immunity from a subsequent attack of the same fever. "Jenner maintained, that Typhus, and the so called "Typhoid Fever", were as distinct as any two of the Exanthemata."[1] He supported all his statements by carefully made observations on cases admitted into the London Fever Hospital.

1. Murchison on Continued Fevers. 3rd Edition. p. 433.

Since the time of Jenner's observations, many able physicians have abundantly verified his statements. Such workers as Dr. Peacock, Dr. Wilks, Sir Thomas Watson, Professor W. T. Gairdner etc., have assisted in elucidating the points of difference between these two important diseases. In America the non-identity of the two diseases is generally recognised. Bartlett, Austin Flint and West have contributed material in support of this view in America. In France and Germany also, much important work has been done in this direction by many eminent physicians, such as: Forget, Gondolier and Barrallier, in France; and Hirsch and Luelzer in Germany. Barrallier says: "They are distinct from one another in their causes, symptoms, progress, duration and anatomical lesions"[1]

Geographical Distribution.

1. Murchison on Continued Fevers. p. 434: and Barrallier Du Typhus épidémique à Toulon. Paris, 1861. p. 129.

Typhoid Fever occurs in every part of the world known to civilized man. But it is much more prevalent in some countries than in others. With regard to our own Islands, this fever appears to be more frequently met with in England and Ireland, than in Scotland. Although, since the introduction of water-closets into houses, typhoid fever has become, by no means so rare a disease in Edinburgh, as was formerly the case. In Ireland, Dublin and Cork, have acquired an unenviable notoriety on account of the prevalence of typhoid fever in their vicinity. There have been frequent outbreaks at the Royal Barracks, Dublin.[1] In January 1887, a serious outbreak of the disease at Cork, is reported in the medical journals. With regard to the Royal Barracks, Sir Charles Cameron, medical officer of health for Dublin, and Dr. J. W. Grimshaw, Registrar General, point out that the

1. British Medical Journal. Feb.y 11.th 1888.

unsanitary condition probably arises from geological defects in the site occupied by the barracks, and from defective drainage. The disease is so frequently met with in Paris, that it has been called the Paris Fever.
Typhoid fever occurs also in Germany, Norway and Sweden; in Russia, Turkey, Spain, Italy and Iceland. It occurs in Egypt, India and the Tropics. It has been observed by Heyman in Sumatra and Java; and it has also been shown to prevail in Syria[1]. It is very prevalent in the Colonies, particularly in Queenstown, Victoria and Brisbane[2]. In Melbourne also severe outbreaks have occurred[3].
Defective drainage and unsanitary conditions seem to play an important part in the etiology of the disease in the Colonies – as elsewhere. Typhoid fever is known to occur in New Zealand, and Van Diemen's Land,

1. Murchison on Continued Fevers. 3rd Edition p. 436.
2. Lancet. May 7th 1887.
3. Lancet. March 26th 1887. p. 640.

in China and Japan; in North and South America; in Greenland, Central America, California and the West Indies. Many outbreaks have been described in the various divisions of Africa, particularly in Algeria, and on the West Coast. But it also occurs in the Transvaal, Zululand and in other parts of Africa: seeming at times to follow the line of march of the troops, in their campaigns, as described by Surgeon R. Vachy Ash, A.M.D.[1]

Enteric fever is particularly virulent in Egypt. The young soldiers, sent out during the Egyptian Wars, died in great numbers.[2] The special correspondent of the British Medical Journal, in his "Medical Notes from the Nile Expeditionary Force", states that "The death rate from enteric fever is very high. This disease continues to be prevalent, and of a decidedly severe type"[3] - - - -

1. British Medical Journal. July 7th 1883. p.3.
2. British Medical Journal. Feb. 14th 1885. p.346.
3. British Medical Journal. Jan. 31st 1885. p.244.

---"enteric fever will claim far more victims than the Mahdi's bullets or spears"[1]. A large number of contributors belonging to the Army Medical Department, have furnished us from time to time with instructive reports of cases which have occurred at the various station hospitals under their charge: for example, a case is reported by Dr. G. S. Davies, Brigade-Surgeon M. S., as having occurred at the Station Hospital, Allahabad.[2] Nevertheless Sir Joseph Fayrer insists on the necessity of a closer investigation and elucidation of the climatic fevers of India. And as if in response to this appeal, Brigade Surgeon J. B. Hamilton M. D. has quite recently thrown much light on the subject. He states that more people die in India from Typhoid fever than from Cholera[3]. And the same fact

1. British Medical Journal. Jan.y 31st 1885. p. 248.
2. British Medical Journal Augt. 29th 1885. p. 394.
3. British Medical Journal. Oct. 4th 1890. p. 787.

has been previously noted by Dr. Joseph Ewart and others.[1] Dr. Hamilton proves the fact statistically in his valuable paper. But the unsatisfactory condition of statistics generally, relative to the death rate from enteric fever, not only in India, but at the other foreign military stations, is a subject which has been repeatedly and severely criticised by Epidemiological authorities. In the official returns, many more deaths are attributed to Simple Continued Fever, than one can conceive to be possible. There can be no doubt, but that many of the deaths from so-called Continued Fever, are really deaths from typhoid fever. That the disease met with in India is really typhoid fever, has been abundantly proved by many post-mortems,

1. British Medical Journal. Sept. 20th. 1884. p. 566.

made by competent observers.
Many cases of death which have
been returned as resulting from
Malarial Fever, likewise, were
probably deaths from enteric fever,
not diagnosed. Typhoid fever
occurring in an individual the
subject of malaria, might
present modifications, difficult
to recognise. Some authorities
have even asserted, that there
is a distinct climatic fever,
which they call "Typho-malarial",
inasmuch as it is a kind of
hybrid between typhoid fever and
malaria. But probably the
correct view is, that typho-
-malarial fever, so-called, is
simply enteric fever, as it
manifests itself in a malarious
subject: in which case the
symptoms of enteric fever are
masked by those of malaria.
Enteric fever occurs at Madras,
and in various parts of the Bengal

and Bombay presidencies, and in Burmah. It is prevalent in Bermuda.[1] Typhoid fever also sometimes occurs in ships: thus for example a persistence of the occurrence of enteric fever was noticed in certain ships of the Channel Squadron: viz., the Agincourt, Northumberland, and Sultan, in the Year 1883.[2]

Etiology. The etiology of typhoid fever, is a subject of the greatest interest and importance, insomuch as it is from a knowledge of its causes alone, that we can hope to make any progress in the prevention of the disease. The causes naturally divide themselves into two groups — predisposing and exciting. Concerning the predisposing causes, there is tolerable unanimity of opinion; but concerning the exciting causes, there is much uncertainty, and

1. British Medical Journal. Oct. 25th. 1890. p. 940.
2. British Medical Journal. Oct. 13th. 1883. p. 736.

discrepancy of opinion. For example, most authorities would admit, that the disease occurs with almost equal frequency in the two sexes; amongst rich and poor; and during the Autumn. But is the disease contagious? Is it communicable from the sick to the healthy? What is the nature of the materies morbi? Through what channels is it conveyed? What are the exciting causes of typhoid fever? These questions must still be regarded as requiring further elucidation; and they have been, and still are the subjects of keen, and as yet undecided controversy.

The predisposing causes may be briefly ranged in sequence, and dismissed, before passing on to the exciting causes.

1. Sex. "The disease occurs with almost equal frequency in the two sexes. Of this there is abundant evidence."[1]

2. Age. All practitioners who have

1. Murchison on Continued Fevers. 3rd Edition. p. 437.

any experience of the disease know, that it is amongst their younger patients that they most frequently meet with enteric fever. And the great majority of cases occur under the age of 25 Years. Often enough they may have cases occurring in children[1]; but still more frequently cases are met with between the ages of 15 and 25 Years. Sometimes however enteric fever does occur in people advanced in age. One old man suffered from typhoid fever, with characteristic symptoms, at the age of 67 Years, in the experience of the writer; and there are records of cases having occurred in infancy, and at very advanced ages. Thus for example, a case occurred in an infant aged 6 Months[2]; and Dr. Murchison quotes cases which occurred in newly-born infants[3]; and in one remarkable

1. Case of L.R. page 44 of this thesis: her sisters C.R. Aet. 8., and G.R. Aet. 4., also contracted the disease.
2. Murchison 3rd. Ed. pp. 439 and 440. 3. Ibid. 440.

case, the disease seems to have occurred in a seven months foetus.[1] Dr. Murchison further noted 27 cases above 60, and two above 75 Years of age. He quotes cases recorded by other observers, at the advanced ages of 86, and 90 Years respectively.[2]

Notwithstanding these exceptions, typhoid fever is essentially a disease of youth: and is rarely met with after the 50th year; and it is much more frequently met with under 30, than after that age. In this respect it presents a contrast to Typhus Fever.

The Mode of Prevalence. Enteric fever is essentially an endemic disease. It is the endemic fever of Britain, as it is of France and many other countries. It no doubt sometimes becomes epidemic, but with this peculiarity that the epidemic is

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1. Murchison on Continued Fevers. 3rd Edition. p. 440.
2. Murchison on Continued Fevers. 3rd Edition. p. 441.

localised to a certain area, and can generally be attributed to some sanitary defect in the locality. Apart from such localised outbreaks, the average number of typhoid cases does not vary much from year to year.

The Season of the Year, has a great influence on the prevalence of typhoid fever. Cases occur much more frequently during the Autumn, than at any other time. In October and November, the seasonal influence is at its maximum, and it gradually declines until April, when it is at its minimum. Cases are frequent likewise in August, September, December and January, as can be seen from the medical journals.

"It would seem as if the cause of the disease were only exaggerated or called into action by the protracted heat of summer and autumn, and that it required the protracted cold of winter and spring to impair its activity or to destroy it."[1]

[1] Murchison on Continued Fevers. 3rd Edition. p. 445.

It is easy to verify this fact by recalling to remembrance the cases of typhoid fever which have been attended in practice, or by reference to the Medical Journals. There was an outbreak at St. Helen's in November 1889[1]; at York in Dec^{r}. 1887[2]; at Sutton nr. Frodsham Cheshire in September 1884[3]; at Hebden Bridge in Nov^{r}. 1884[4]; at Kidderminster in Sept. 1884[5]; at Derby in Oct. 1884[6]; at Dareuth Asylum in Oct. 1885[7]; at Torpoint in August 1883[8]; at the Village of Muirkirk, Scotland, in Sept. 1883[9]; at Carrickfergus in Sept. 1883[10]; At Glasgow Royal Infirmary, in Sept. 1883[11]; At St. Pancras in Sept. 1883[12] and many other outbreaks have been recorded as occurring each Autumn.

1. British Medical. Nov^{r}. 2nd. 1889. p. 1013.
2. Lancet. Dec^{r}. 17th. 1887. p. 1239.
3. Brit. Med. Jour. Sept. 6th. 1884. p. 480.
4. Brit. Med. Jour. Nov^{r}. 8th. 1884. p. 919.
5. Brit. Med. Jour. Sept. 27th. 1884. p. 625.
6. Brit. Med. Jour. Oct. 18th. 1884. p 787.
7. Brit. Med. Jour. Oct. 24th. 85. p. 803. 8. Brit. Med. Jour. Aug 18th/83. p. 343.
9. Brit. Med. Jour. Sep. 29/83. p. 643. 10. Brit. Med. Jour. Sep. 29/83 p 644.
11. Brit. Med. Jour. Sept. 29/83. p. 643. 12. Brit. Med. Jour Sep. 29/83 p. 505.

Temperature moisture and soil have a distinct influence, as predisposing causes of enteric fever. After prolonged drought, cases are especially apt to arise, as occurs during hot, dry summers and autumns. There is a scarcity of water in consequence of the drought: drainage is not properly effected, and bad smells arise. The heat favours decomposition of the stagnating contents of sewers, soil-pipes, etc., giving rise to the generation of poisonous gases, which may diffuse themselves through the water contained in the water-closet traps; if the sewer-gas does not even force the traps altogether, thereby rendering them inefficient. In this way foul emanations get into houses which have drains communicating with their interiors. And moreover accumulations of putrefying material, in privies cesspools and other filth receptacles, are always more malodorous in warm weather. Thus we find that a warm dry summer and autumn favours the development of typhoid fever, whereas a cold and wet summer is unfavourable to its development. But mere moisture alone does not keep away the disease

because if it be associated with warmth, the conditions favourable to faecal fermentation are present. On the other hand cases are on record, where under certain unfavourable conditions and sanitary defects in the drainage system, an excessive rainfall has given rise to an outbreak of enteric fever. A remarkable outbreak occurred at Torquay from heavy rainfall (1½ inches in two hours) and defective sewers.[1] "The influence of a mild temperature and heavy rainfall are well exemplified by the severe epidemic which occurred at Kendall."[2] "The sewer gases were subjected to great pressure, and backed up in various directions." Von Pettenkofer's views as to the influence of the groundwater in the production of typhoid fever &c, are well known. The conditions necessary in his opinion are, a porous soil, in which water has accumulated at a certain depth, and a rise of this

1. Brit. Med. Jour. April 12th 1890. p. 735.
2. Brit. Med. Jour. May 17th 1884 p. 963.

this ground water to an unusual height.

In hot weather after a rise of the ground water, there is a sudden subsidence, leaving the soil moist, and under favourable conditions of temperature, for the development of disease germs. He believed that enteric fever was usually propagated from a sudden subsidence in the ground water, after it had risen to an unusual height; and never from the filtration of organic impurities into wells &c; as has been argued by some, to be the cause of enteric fever spreading under such conditions of the ground water. He has recently drawn attention to the remarkable diminution which at once ensued in Munich upon the completion of the drainage of that city.[1] And there is no doubt but that in Munich at all events, the influence of the ground water in the production and propagation of various infectious diseases has been

1. British Medical Journal. p. 369.

been considerable. And although in Egypt unsanitary conditions of every kind abound, still "the relation of the rise and fall of the Nile, with its influence on the ground water, strongly support Pettenkofer's views on the periodicity of enteric fever" But his views are probably too exclusive; because we know, that in this country, irrespective of the state of the ground water; there is a close relationship between unsanitary conditions, such as contaminated or defective water supply, and derangements in the drainage system, and the prevalence of enteric fever.

Previous Diseases. There is no evidence that previous diseases have any influence in predisposing to typhoid fever or otherwise. M. Carnot and his followers, have maintained, that there is a relationship between typhoid fever, and variola: that variola has not been influenced by vaccination in any other way, than that of driving the

the specific manifestations of variola from the skin into the intestines. And that the enlarged and ulcerated Peyer's patches, and solitary glands, are nothing more than an internal form of variola. That indeed to protect from typhoid fever, the intestinal mucous membrane ought to be vaccinated. M. Carnot's views have been refuted; but the doctrine has been brought forward by antivaccinators as an argument, to the effect, that the operation of vaccination has not produced any reduction of the mortality from acute infectious diseases; but that all that vaccination has done, has been to bring about "a displacement of mortality," — cases of small-pox, presenting the specific eruption on the mucous membrane of the bowels, instead of on the skin, and in this particular form, being accounted a distinct disease, called by medical practitioners typhoid fever. They argue that in proportion to the diminution of the number of cases of small pox, there is a corresponding

increase in the number of cases of enteric fever. Dr. Russel Wallace a troublesome antivaccinator, first stated the above doctrine to the writer.
It is said that people suffering from phthisis are rarely attacked by enteric fever; and Dr. Collie upholds this view.
Dr. Murchison does not adduce any evidence on the subject; but remarks that "an attack of enteric fever is often followed by tubercular deposit in the lungs."[1] This fact most practitioners are acquainted with from their own observations in practice. By some authorities it is supposed, that Malaria is antagonistic to enteric fever; and that in districts where malaria is prevalent, enteric fever is in a corresponding degree not prevalent. But it seems much more likely that there is no such antagonism but rather a similarity than an antagonism, between the two diseases.

1. Murchison. 3rd Edition. p. 452.

Malaria resembles typhoid fever, in the same way that typhoid, resembles Cholera and Dysentery. They prevail in a similar manner, and they often prevail together. As is now well known, Enteric fever, is quite prevalent in India, along with malaria. The notion of typho-malarial fever has been almost universally abandoned. There is no such hybrid disease. The same remarks apply to the so called antagonism between Scarlet fever and typhoid fever. It is not found that in proportion to the increase of cases of enteric fever, cases of scarlatina occur less frequently — or vice versa. The two diseases prevail together; they are both most prevalent in Autumn. The poisons of the two diseases may enter the body at the same time or at different times; and a patient suffering from scarlet fever, may develop symptoms of enteric fever. It is difficult to trace correctly the sequence of the two diseases occurring in the same patient.

But Scarlatina seems to predispose to enteric fever. A patient suffering from enteric fever, may contract Typhus.
Indeed enteric seems to predispose a patient to an attack of typhus fever.
Cases which at the outset present all the symptoms of Acute Rheumatism merge in rare instances into attacks of typical enteric fever.[1]
Occupation. The influence of occupation as a predisposing cause, is not very marked. Housemaids and others, who spend much time in basements are frequently attacked.
Also men who work in sewers.
Idiosyncrasy. There can be no doubt, but that certain peculiar states of the constitution, are more favourable to the development of enteric fever than others.
Overcrowding and deficient ventilation, do not seem to have much influence as predisposing causes. The disease

1. British Medical Journal. April 5th 1884. p. 666.

prevails in the densly populated and badly ventilated parts of large cities, not more markedly than it does in the best houses of the most sparsely populated suburbs.

It occurs in small towns and villages, or even in isolated houses, as frequently in proportion, as in large towns. The density of the population in fact, does not seem to have any influence on the origin or propagation of the disease. It is possible however, that defective ventilation may prevent dilution of the poison, and lead to its becoming more intense, if it should accumulate in the neighbourhood of the patient. Station of life. This does not seem to influence in any great degree the occurrence of enteric fever. It seems to be if anything more prevalent amongst the upper and middle classes, than amongst the very poor. In this respect it presents

a marked contrast to typhus fever.
Recent residence in an infected locality, undoubtedly predisposes to enteric fever. It occurs frequently in domestic servants, soon after they have changed from one house into another.
Often enteric fever occurs in healthy persons who have newly arrived in a district where the disease is prevalent. Long exposure to the causes of enteric fever apparently confers a power of resisting the poison; resembling the immunity from the poison of ague, which is acquired by people who have long resided in malarious districts.
Intemperance, mental emotion and fatigue do not seem to predispose to enteric fever.[1]

Exciting Causes.

The exciting causes of enteric fever are in many instances obscure; and

[1] Murchison on Continued Fevers. 3rd. Edition. p. 450.

much difference of opinion still prevails amongst the highest authorities with regard to the origin and nature of the poison, as well as to the manner in which it is propagated.

Dr. Murchison groups cases of enteric fever under two divisions, according to what he considers to be the exciting cause.

1. Cases in which enteric fever is communicated from the sick to the healthy, in some way or other.
2. Cases which have an independent origin.[1]

He contends that contagion in the strict sense of the word does not occur. Other authorities, such as Dr. Collie, and the followers of Dr. Budd, hold that contagion is the "masterfact" in the etiology of enteric fever. Dr. Collie's conclusions are the following:— "That the exhalations from the lungs, skin, urine or fresh stools must be infectious." "There is danger of infection, for some time after the stoppage

1. Murchison on Continued Fevers. 3rd Edition p. 458.

"of the diarrhoea". The 1st conclusion is directly opposed to that of Dr. Murchison; and the experience which medical men have of the disease, is that it is not at all usual for those who attend on the sick, to contract the disease. So that if the exhalations from the patient be contagious, they must cease to be active at a very short distance from the patient. Dr. Collie brings forward the fact that 19 nurses, at Homerton hospital, contracted enteric fever, whilst attending upon patients, labouring under the disease, as an argument in favour of contagion. But that such a large number of persons should be attacked by enteric fever, from direct contagion, is quite beyond the experience of the most careful observers. And the probability is that there was some local cause for the development of typhoid fever amongst those nurses — some defect of the drainage system. The arguments in favour of direct contagion are; a. That when enteric fever breaks out

Home Wednesday or Thursday
at 2. from

Miss M. C. Lemmonth;

111 Montgomery Street

in a household, it often spreads through the house, attacking several members of the family in succession. The following experience occurred to the writer, in private practice. A little girl was sent home from school ill, from typhoid fever. At short intervals her three sisters, were successively attacked; and a month later, the baby had an attack of infantile remittent fever.(?) All the patients recovered. All but the baby had enteric fever with rose spots and other typical symptoms. Such cases seem to favour the theory of direct contagion: but the difficulty is in such cases; that it is impossible to say that there may not be a local cause which has occasioned the outbreak. There was an accumulation of drain filth, under the floor of the front parlour, in the house where the above mentioned cases occurred. Another argument against the Contagion theory, is that several in a household may be seized simultaneously, giving rise to a suspicion of irritant Criminal poisoning. Or the interval between successive cases

may be too great, to render communication of the disease by direct contagion possible. Sometimes where a local cause of ~~infection~~ infection exists in a house, no case may have occurred for years, until a visitor or person newly arrived in the house, contracts the disease soon after arrival. Or in the event of a new tenant occupying the house, enteric fever, spreads through the family. Hence the necessity of having the drains thoroughly examined before taking possession of a new house.

It does occasionally occur that nurses contract enteric fever, while attending on cases; but apart from the possibility of a local cause existing, in the house in which the patient is being nursed, there is always the possibility that the disease may have been communicated through the agency of the stools of the patient. Dr. Murchison insists on the point that the fresh stools of enteric fever are harmless; and certainly his ponderous statistics, would tend

to prove that such is the case. Or at the least that the fresh stools of enteric fever are not nearly so virulent in their poisonous properties, as when they have undergone decomposition!

Dr. Cayley has reasons for thinking however, that the stools may develop contagious properties in the course of twelve hours, or less under favourable circumstances. Therefore if the specific stools be not at once disinfected and removed, it is easy to understand, how the disease might be communicated in this indirect manner. Granting that the fresh stools are harmless, and that they may become contagious in 12 hours or less; it would surely be unwise to adopt any other practice than that of at once disinfecting and removing the typhoid stools from the sick chamber. And if we cannot dogmatically assert that typhoid fever is never communicated by direct contagion, we know that

1. Murchison on Continued Fevers 3rd Edition pp. 462. 466 + 467.

by far the greater number of cases and epidemics arise from other causes. As a further argument in favour of the contagious nature of enteric fever, it is stated that when a person suffering from the disease, is introduced into a healthy locality or house, in which previous cases of fever have been unknown, he often communicates the disease to those around him; and it spreads from him as from a centre. That enteric fever may be sometimes spread in this way is an undoubted fact. But the number of cases in which after the introduction of a typhoid fever patient into a healthy house, the disease does not spread, far outnumbers those cases in which enteric fever spreads from the person affected, as from a centre. It is evident, then that in a certain number of cases, enteric fever is communicable from the sick to the healthy; and the method of communication is therefore important. The consensus

of opinion is that enteric fever is not contagious in the strict sense of the word; but communicable only through the agency of the motions of the typhoid patient. These motions seem to be particularly virulent after having undergone that fermentation, to which they are peculiarly liable. Instead of being acid, as the motions of health normally are, the motions of enteric fever are alkaline, and contain a large amount of ammoniaco-magnesian phosphate, ammonia, and other products of organic decomposition. Certain circumstances also, such as confinement in closed spaces, stagnation, and a certain temperature, corresponding rather to the warmth of summer, than to the cold of winter, greatly favour the development of the virulent properties of typhoid stools.

It may be confidently asserted that the nature of the poison is still unknown. But whatever be its nature, faecal fermentation seems to be particularly

favourable to its development.
Whether the 'Bacillus Typhosa' be the essential cause or not; it is well known that the process of putrefaction is characterised, by the presence of bacteria; and the question arises whether the poison may not be developed by these bacteria, certain conditions being necessary for its development. Can the passage of the ordinary causes and products of faecal fermentation or putrefaction, through the body of man, so aggravate the virulence of these causes and products, — viz. the virulence of the bacteria of ordinary putrefaction and any poisonous materials which they may generate by their activities — that they become not only the exciting causes of ordinary gastro-enteritis, but of the specific disease called enteric fever?
In other words, can the ordinary bacteria, with which putrefaction is always associated, become converted into the so called Bacilli of Typhoid?

Bacilli of Typhoid, in consequence of modifications which they may undergo, in passing through the human body? Or is the poison of the nature of a specific contagium vivum, like the bacillus which gives rise to Splenic fever? If there be a specific bacillus — the Bacillus Typhosa — from which alone enteric fever can originate; then there is the greatest difficulty in explaining the origin of a large number of cases of enteric fever, which arise in thinly populated villages or isolated houses. Dr. Murchison quotes a number of cases where it was impossible to trace any source of contagion; and in which the disease seemed to have an independent origin. And many cases are quoted by other observers, in which no possible source of contagion could be discovered. Sir Wm. Jenner also states, that it

it is in many instances impossible to trace the origin of enteric fever — neither contagion nor putrefaction sufficiently explaining the reason why an outbreak has occurred. He states that enteric fever must arise from the influence of man on his surroundings, and of his surroundings on man.

The contagionists on the other hand say, that although the source of contagion may not have been discovered, still the existence of contagion was not disproved in consequence. If faecal fermentation, and other putrefactions, per se, cannot be regarded as originating the poison of enteric fever; and if it be a necessary condition for the generation of the poison of enteric fever, that infection of the fermenting faeces or other putrefying material, with typhoid stools, or with the specific bacillus, must take place; then in

in those cases which are supposed to have originated de novo, the source of the specific infection must have been overlooked in all such cases: and if this were so, it would put an end to all discussion on the matter.
But cases are so numerous in which, after careful investigation, no such infection can be traced, although they are associated with defects in the drainage system, polluted water supply &c, that in the present state of our knowledge we are bound to admit, that whereas a certain number of cases of enteric fever, are communicated from the sick to the healthy, through the agency of the stools of patients suffering from the disease, yet there are a number of cases in which the disease appears to arise, independently of communication from the sick to the healthy.
The precise etiology of such cases is uncertain, but no doubt it is in some way intimately

connected with unsanitary conditions.
That enteric fever can be propagated by means of a polluted milk supply is now well known. Thus for example, an epidemic occurred at Aberdeen from this cause in December 1884, and January 1885.[1] at Larkhall, Scotland, in March 1885;[2] and at Derby in October 1884.[3] The milk epidemic transmitted from St. Albans to Camden Town is also historical. When enteric fever arises from the milk supply, it seems that in most instances the milk has been adulterated with water polluted with sewage, or that polluted water has been used for washing the milk cans. Often enough the dairy keepers have had cases of enteric fever in their families, at the time of the outbreak.
In one case related by Dr. Murchison

1. British Medical Journal. Jan.y 24th 1885. p. 193.
2. British Medical Journal. March 21st 1885. p. 615.
3. British Medical Journal. October 18th 1884. p. 787.

the milk seller and his family who were suffering from enteric fever, occupied a dirty room, close to which the milk was kept. The outbreak occurred in Dublin, and was reported by Dr. Cameron. In this case there probably was direct faecal contamination of the milk. 1.

It is said that diseased meat may give rise to the disease. Huguenin relates an outbreak, due to eating the flesh of calves which had undergone decomposition; but he stated that the calves were suffering from enteric fever, which they had contracted from their keepers. The flesh required to be putrid, as well as specifically infected, in order to give rise to the disease in man. Eating putrid bacon, and poisoning by the emanations from putrid hides, have also been cited as causes of the disease; and it is suggested that the animals may have been suffering from enteric fever in these cases. But much doubt is expressed as to the nature

1. Murchison on Continued Fevers. 3rd Edition p. 467.

of the disease, in many cases of animals stated to have had enteric fever. It is alleged that cases of enteric fever have arisen from eating oysters, in consequence of the oyster-beds having been polluted with sewage.

In the case of troops suddenly called into active service, it is a question whether the change to a hot climate and entirely different surroundings — camp life, and all the worries and fatigues, incidental to campaigning — may not bring the state of health into an abnormal condition, favourable to the development of enteric fever, in those subjected to such conditions. At all events, as already remarked, young soldiers suddenly placed under such circumstances, are particularly prone to contract the disease; even when such causes as defective or polluted water-supply, bad drainage, the state of the soil &c are not at all obvious.

The poison of enteric fever ceases to take effect at a short distance from the patient; particularly when the ventilation is good. But it is evident that by the motions, it may be carried to great distances, through the medium of the drainage or water supply; and in this way the poison may take effect at a great distance from the affected patient.

There is evidence that Clothing & other fomites, may be the means of conveying the disease to a distance. Clothing stained with typhoid stools may carry the poison, wherever the clothing is taken to be washed. Because there is no reason why, the changes necessary for the development of the most virulent properties of typhoid stools, should not take place in those stools discharged into the Clothing. Dr. Murchison quotes a remarkable instance of this kind.[1]

1. Murchison on Continued Fevers, 3rd Edition. p 468.

The period of incubation of the disease is very variable in different cases; and the investigation of the subject is difficult, owing to the insidious nature of the onset of the disease. The most usual period seems to be from 10 – 15 days, so far as can be ascertained. Dr. Budd states, from 10 to 14 days. Dr. Cayley has not found a well authenticated case in which the period of incubation was certainly less than 5 days, or more than 22 days. In Professor Quincke's cases, the shortest period was 8 days, the longest certain period between 16 and 18 days. Sometimes the period of incubation is very short, and a number of persons are attacked at one time so as to give rise to a suspicion of irritant poisoning.[1] There are no data apparently which would indicate at what

1. Murchison on Continued Fevers. 3rd Edition. p. 469.

what period the disease is most communicable; although it would be important to determine at what stage the stools are most virulent. There is no certain proof that enteric fever can be communicated by the dead body.

One attack of enteric fever seems to confer an immunity from a second attack; but there are well authenticated cases to show, that an individual may be attacked a second time after a longer or shorter interval. In this respect enteric fever resembles most other acute specific diseases.

Pregnancy has been supposed to confer immunity from enteric fever; but Dr. Murchison has observed several cases as occurring in pregnant women. In such cases abortion usually takes place[1];

1. Murchison on Continued Fevers. 3rd Edition. p. 582.

The Anatomical Lesions of enteric fever have been carefully and exhaustively studied. They are divided into two groups: 1st those specific lesions of the solitary and agminated glands of the ileum, and of the mesenteric glands, which are essential to, and invariably associated with enteric fever; and 2nd those accidental and non-essential morbid anatomical changes, which occur more or less frequently in cases of enteric fever, as they do in other diseases. It will be well to state the salient points relating to these two groups, arranging them as they occur in the different systems of the body.

The Cadaveric Rigidity is generally well marked. The body is much emaciated, if the disease has been prolonged previous to death. Putrefaction is not rapid in its onset, as in the case of typhus.

Where the typhoid state has existed

before death, putrefaction may set in rapidly. The lividity on dependent parts is not well marked. Greenish discolouration of the abdomen within 48 hours is rare.
The face as a rule is not livid.
Where pulmonary complications have existed during life, lividity may occur.
The rose-coloured lenticular spots do not persist after death.
Sudamina are sometimes seen.
The changes which occur in the muscular system have been described by Hoffman and Zenker, as parenchymatous degeneration.[1] Zenker describes the changes in muscles as being of two kinds: 1°. Granular degeneration. In this condition the striation in the muscular fibres becomes obscured, and a collection of granules takes place in the substance of the fibres. The muscles appear dark almost like smoked meat, and dry. 2°. Waxy degeneration. In this condition the contractile substance of the primitive bundles becomes converted into "an homogeneous, colourless, waxy, shining mass;" and the striation entirely disappears. "It reminds one of the lardaceous or amyloid

1. Ziemssen. Cyclopaedia of the Practice of Medicine. Vol. i. p. 108. (Liebermeister. – Typhoid Fever.)

amyloid degeneration"; but does not give the characteristic reaction with Iodine and Sulphuric acid. The striation entirely disappears. In both forms, the muscles are thicker and more brittle than normal. In the waxy form, they are often broken across. Ultimately a mass of fine detritus may take the place of the muscular bundles. In the granular form the detritus is absorbed, and the remaining more or less altered cellular elements proliferate, and the organ is as far as possible restored. In waxy degeneration a return to the natural condition is impossible. Often both varieties of muscular degeneration exist together, one or the other preponderating. During the 1st. week of typhoid fever the muscles appear very dark red or reddish brown — like smoked meat — in places they are paler where the degeneration is most advanced. In the granular degeneration, the muscles look yellowish; but in the waxy form they have a greyish appearance. In the highest degrees of muscular degeneration, the voluntary muscles appear yellowish grey or whitish grey. So that there is scarcely a trace of red colour

left in the muscular substance. On section the affected muscles are dull smooth dry and very brittle. At first the fibres are swollen, but later on decreased in volume. During convalescence the muscles become much moister, and their red colour returns. During the 2nd, 3rd and 4th weeks, Hoffman found the degeneration most marked. After the 4th week, the degeneration disappears or only its results are seen. Exceptionally degeneration of the voluntary muscles is found later than the 4th week. These degenerations occur most frequently in the adductors of the thigh and the Rectus and Transversalis abdominis muscles: Also in the Pectoral muscles. Amongst the results of these degenerations, are haemorrhages into the substance of muscles, and the formation of pseudo-abscesses, arising from the rupture of muscular fibres.

The organs of digestion. 1. Pharynx. As a rule the pharynx is healthy. It may be inflamed, covered with diphtheritic false membrane, or ulcerated. Sometimes the submucous tissue of the pharynx is infiltrated with abscess or pus. The pharyngeal ulcers as a rule are superficial, but may extend as deep as the muscular coat-

They are round oval or irregular in shape, varying in diameter from 2 lines, to ¾ inch; their edges are not thickened, and the surrounding mucous membrane may be normal or injected. Their usual seat is the lower part of the pharynx. They do not occur before the 3rd week of the disease.

2. Oesophagus. Like the pharynx, the oesophagus is usually healthy in typhoid fever. But it may be the seat of ulceration. These ulcers do not occur before the 3rd week of the disease. As a rule they are quite superficial, but sometimes they extend as deep as the muscular coat. They have never been known to lead to perforation; nor are they due to any deposit, similar to that which occurs in the essential lesions in the bowels. They are secondary to the specific intestinal lesions. Their most frequent situation is in the lower part of the oesophagus, where they may be larger than those higher up.

3. The stomach is often healthy; but it may be hyperaemic or pale. Softening of the stomach is often observed, especially in the great cul-de-sac. This softening may be considerable and extensive. It seems to be due to post mortem

digestion. Ulceration rarely occurs, and when observed, the ulcers are small and superficial. They are not accompanied by any deposit in the glandular structures of the stomach; and they occur in the course of other diseases. They have been attributed to the excessive exhibition of Alcohol during the treatment, in the last hours of life. Mamillation of the mucous membrane occasionally occurs. The duodenum is usually healthy; but may be injected, or present enlargement of its mucous follicles, conditions not peculiar to enteric fever. Minute ulcers similar to those which occur in the stomach, are said to have been found in very rare instances.

The Jejunum and Ileum. As a rule the jejunum and ileum are not distended, but are collapsed and almost empty — the tympanites observed in enteric fever being due to the presence of gas in the colon and large intestines. The contents of the bowels are of a thin consistency, of an orange or ochrey yellow colour, and often contain brownish portions of detached slough, or blood in small clots. They also contain

a large quantity of ammoniaco-magnesian phosphate. Sometimes worms (ascaris lumbricoides) are found in the intestines: and both round-worms and tape-worms have been passed by persons suffering from enteric fever.

It is said that when perforation occurs, these worms may be found in the peritoneal cavity, amongst the escaped contents of the intestines. From the presence of these entozoa in some cases, the term "Worm Fever", derived its origin. As in other diseases where there is profound affection of the cerebro-spinal system, invagination of the small intestine without inflammation, into a portion below, to the extent of one or two feet may take place.

The mucous membrane of the diseased intestine may be natural in colour or present increased vascularity.

The vascularity may be continuous or in patches. It is not restricted to dependent portions of the gut; but occurs equally all round the intestines. This increased vascularity of the mucous membrane, which sometimes occurs, is not an essential lesion of enteric fever.

After the 3rd " week, the mucous membrane

may present a greyish or slate colour.
Sometimes the mucous membrane is swollen
and extensively infiltrated with blood.
The blood can be squeezed out on pressure;
and the gut resumes its natural thickness.
In such cases the gut presents a dark,
reddish purple, shining, gelatinous
appearance. Softening of the gut has been
observed. It seems to be like that of the
stomach, a post mortem change.
The essential lesions of enteric fever consist
of changes which are brought about in the
solitary and agminated glands of the ileum.
These lesions are peculiar to enteric fever, and
are invariably present. The solitary glands of
the colon sometimes present a similar change.
The nature of the morbid process is no doubt,
as Dr. Murchison teaches, inflammatory.
A certain poison the nature of which is still
unknown, seems to be the exciting cause of
this inflammation. The agminated and solitary
glands of the ileum, seem to have an elective
affinity for the poison, which is no doubt
most frequently swallowed with the ingesta.
The structure of these agminated and

solitary glands is precisely similar to that of a lymphatic gland, or of the spleen – a fibrous stroma containing lymph corpuscles. They have been injected from the lacteals, and are compared by Virchow to lymphatic glands, spread out in the walls of the intestines. They are absorbent not eliminatory glands. The specific poison after being absorbed by these glands, no doubt excites inflammation in them; and swelling and proliferation of their tissue elements occurs. The lymphatic elements of the mesenteric glands, and of the spleen also proliferate. It has been thought that these glands become enlarged, in consequence of overstimulation in eliminating the poison. But the function of these glands is not eliminatory. They are absorbent glands, and probably carry poisons from without into the blood-stream – as in the case of the absorption of the poisonous material which occurs in pyaemia. The glandular enlargement of the intestines, cannot be regarded as an exanthem. There does not seem to be any specific "typhous matter", similar to the virus of a vaccine vesicle. The diseased agminated and solitary glands do not secrete an infectious virus: the

lesion consists essentially of an inflammatory condition of the normal tissues of the part. Microscopically there is proliferation of the lymph corpuscles proper to the gland. The corpuscles are larger than normal, and filled with granules. Some are much larger than others, and contain 1, 2, 3 or several nuclei. Later in the disease than the above early changes, the cellular elements disintegrate, a large quantity of oil globules and granular detritus becoming mixed up with the tissue elements. Recent observations have demonstrated the frequent presence of Eberth's Bacillus in the lymph spaces. The exact nature of the poison is a desideratum; and therein lies the solution of many of the difficulties surrounding the etiology of the disease. Eberth argues that a specific bacillus constitutes the poison. 1°. Because of its locality. 2°. Because of its reaction with certain reagents. 3°. Because in structural details, (delicacy of outline, spindle shape or flattened ovoid) it differs from the ordinary bacilli of putrefaction. But other observers have not found these typhoid bacilli constant in cases of enteric fever, even in the earlier stages of the disease. They have not always found them distinguishable

in outline, from the ordinary bacilli of putrefaction. And they have not been able to verify with certainty Eberth's observations, with regard to the behaviour of typhoid bacilli, in relation to staining reagents, distinguishing them from putrefactive bacilli. The subject requires further study.

Whatever be its exact nature, the poison excites lesions which are definite and characteristic. The lesions pass through various phases or stages which are described under four headings.

1. The stage of deposition or enlargement.
2. The stage of ulceration.
3. The stage of genuine typhoid ulcer.
4. The stage of cicatrisation.

1°. The stage of enlargement probably commences with the disease. It is doubtful whether the enlargement is preceded by congestion. The lesion has been observed without increased vascularity preceding the process. As taught by Dr. Murchison, any form whatever of congestion of the intestinal mucous membrane, without glandular enlargement, does not constitute enteric fever. Trousseau gives the 5th day, as the date of commencement of the enlargement; but Dr. Murchison has observed

the change in a case fatal on the 2nd " day.
Some authorities believe that the enlargement is due to a deposit from the blood. But probably it is inflammatory as already stated.
Proliferation of the cellular elements of the glandules constituting Peyer's Patches occurs, and the process extending to the fibrous stroma, the glandules coalesce; and the entire patch becomes involved. In this way the variety of the lesion termed a "plaque dure" is formed. The intestinal lesions have been divided by Louis, into "Plaques Molles" and "Plaques Dures"; by Chomel, into "Plaques Réticulées" and "Plaques Gauffrées."
They are essentially similar in nature, and various intermediate forms exist. The plaque molle is slightly raised above the level of the surrounding mucous membrane, soft in consistence, and has a pugose or granular aspect. Colour, pinkish grey, or more or less red. It is formed by the proliferation of the cellular elements of the glandules which make up the patch; but the process does not, in the plaques molles extend to the surrounding fibrous stroma, and consequently the glandules of the patch do not entirely

coalesce as in the case of the plaques dures. Some authorities attribute the formation of plaques dures to the rupture of the glandules into the surrounding submucous tissue. Professor Goodsir stated that the deposit took place 1st in the interior of the gland, which subsequently ruptured, discharging its contents into the submucous tissue. Dr. J. I. Maclagan believes that they are distinct varieties: the plaques dures being primary; and the plaques molles secondary lesions, due to infection from the plaques dures. "There does not seem to be adequate evidence in support of this view. "The plaque dure, is firmer in consistence, more raised and uniform in appearance than the plaque molle. "The mucous membrane covering it is pale-grey and smooth. Both varieties have steep edges, and are surrounded by a ring of vascular injection: and the peritoneal surface corresponding to the plaque is injected bright-red, purplish or of any colour intermediate.

"The solitary glands in the lower

portion of the ileum may be enlarged in the same way as the Peyer's Patches: in about $\frac{2}{3}$ of the cases (Murchison). Rarely the solitary glands above are affected: then their rounded forms and flattened surfaces, present an appearance not unlike the vesicles of small-pox.

The stage of enlargement does not necessarily pass on to the stage of ulceration. Sometimes the morbid products are absorbed; the process of absorption commencing about the 10th or 12th day of the disease, and being completed about the 21st day.

2. The stage of ulceration. This process begins in two different ways, according to whether it takes place in a plaque molle or a plaque dure. In the former, one or more superficial erosions appear in the softened mucous membrane covering the patch; and these extending unite to form one large ulcer, which may extend to various depths, through the mucous or muscular coats — sometimes even to complete perforation. In the plaques dures the whole diseased portion sloughs out, leaving an ulcer, whose base is formed

by the mucous or muscular coat, or by peritoneum. Sometimes the sloughing seems to take place through the whole thickness of the bowel, leading to perforation. When death takes place between the 12th and 21st days, the sloughs can be seen adhering to the surfaces of the ulcers. The plaques dures may slough out en masse, or in successive portions. Dr. Murchison believes that the sloughing of the plaques dures constitutes the most usual form of ulceration. The sloughs are yellowish or brownish from saturation with bile, or they may present a dark fungating aspect from infiltration with blood.

Dr. Aitken believed the glandules ruptured, and discharged their contents leaving a net-like appearance; and that this was the most usual way in which the morbid products were eliminated. But a better knowledge of the structure of the agminated glands, teaches that they are not eliminatory; and that the process in the plaque molles is ulcerative, just

as in the case of the plaques dures, except that in the latter, the ulceration commences at different points on the surface of the gland. It is important to know the time at which ulceration takes place. Usually it begins about the 9th or 10th day of the disease. But it seems that in rare cases, it may begin much earlier, and in some cases much later than this. The process of ulceration begins near the ileo-caecal valve, and extends upwards; and ulcers may be seen near the ileo-caecal valve before they appear in the glands higher up. The solitary glands undergo a destructive process essentially similar to that which occurs in the Peyer's Patches.

3. The stage of typhoid ulcer. This stage is variable in its duration. Sometimes close to the ileo-caecal valve, the sloughs may be detached, on or about the 14th day; whereas those higher up may not separate until the 3rd week, or later. This stage is therefore the period which intervenes between the

beginning of ulceration, and the beginning of cicatrisation. The characters of typhoid ulcers distinguish them from all others. Their situation is characteristic. They occur in the lower part of the ileum, increasing in size and number as the ileo-caecal valve is approached. Near the ileo-caecal valve, a large area of ulceration is sometimes formed, by the coalescence of several ulcers. The individual ulcers may vary in size, from about a line to an inch and a half in diameter. In shape the ulcers are round, elliptical or irregular, according to the shape of the affected glands. The round ulcers correspond to the solitary glands, the elliptical to the Peyer's Patches, and the irregular ulcers to portions of Peyer's Patches, or to the coalescence of several ulcers. The long diameter of the ulcer is in the direction of the longitudinal axis of the intestine, except when formed by the coalescence of several ulcerated solitary glands, when the ulcer may be transverse. Transverse ulcers are chiefly met with in the large intestine. In the elliptical variety

the ulcer always occurs in that portion of the gut which is opposite to the attachment of the mesentery. They never form a zone encircling the gut, as in the case of tubercular ulcers.
There is no thickening or induration of the edges or floor, when the sloughs separate, as in the case of the tubercular ulcer – although portions of slough may be adherent to the floor of the typhoid ulcer. The floor of the typhoid ulcer is formed by a layer of submucous tissue, by the muscular coat or by the peritoneum. The edges of the typhoid ulcer are detached from the subjacent submucous tissue: they are of a slate grey or purplish colour, and form a fringe round the ulcer, which can be best seen by floating the intestine in water. The edges are a line or more in width.
4. The stage of cicatrisation, usually commences some time during the fourth week; and the time occupied by the cicatrisation of each ulcer is

usually about a fortnight, but may be delayed much later than this — even for weeks after the subsidence of the primary fever. The ulcers are then said to be chronic or atonic. These chronic ulcers may give rise to severe diarrhoea, or even advance to perforation. The process of cicatrisation begins near the ileo-caecal valve, and extends upwards; so that, in cases which die about the 4th " or 5th " week of the disease, the specific lesions may appear most abundant, about two feet above the ileo-caecal valve. Cicatrisation is accomplished in the following manner. There is a deposit of delicate, shining granulation tissue, which covers the surface of the ulcer, and occupies the intervening space between the fringe of mucous membrane and the muscular coat. Through the medium of this granulation tissue, the fringe of mucous membrane becomes attached to the subjacent tissue, and adhesion takes place, from the periphery towards the centre.

The granulation tissue covering the ulcer becomes covered with epithelium, derived from the surrounding mucous membrane. At first this epithelium is not movable over the subjacent tissues, as in the normal mucous membrane; but ultimately, it becomes so: and it is said that even the intestinal villi are regenerated over the surface of the cicatrised ulcers. But the glandular structure which has sloughed is not regenerated.

The charlacters of the cicatrix resulting from a typhoid ulcer are thus stated:— It is depressed and smooth, firmer in consistence than the surrounding mucous membrane, and pale in colour. Held up to the light, the cicatrix is translucent; and the intestine at the seat of the cicatrix seems thinner than normal. In diameter, the cicatrix varies from a few lines to half an inch. It never gives rise to any puckering or diminution in the calibre of the gut.

Some authorities state that cicatrices

soon disappear; but others state that they have observed them to be distinct after 4, 5 or even 30 years.

The ulcers sometimes give rise to intestinal perforation, which may occur in the following ways:—

1°. Most frequently the process of ulceration or molecular disintegration extends, until a small portion of the peritoneal covering of the bowel is destroyed. In this way one or more small round pin-hole apertures are produced, about large enough to admit a stocking wire. These minute apertures usually occupy a position near the centre of the ulcer.

2°. A considerable portion of the peritoneum may slough, and perforation take place, in consequence of the complete or partial separation of the slough.

Often a small crescentic opening occurs, at the margin of the sloughing peritoneum: or on opening the bowel, it may be observed that an elliptical portion corresponding

to an entire Peyer's patch falls out:
3. Owing to some undue exertion, or without evident cause, a portion of denuded peritoneum is ruptured, and an elongated linear cicatrix results.
Although doubts have been expressed as to the possibility of this form of perforation, the elongated linear cicatrices, which are sometimes found post-mortem, admit of no other explanation. According to Dr. Bristowe, this indeed is the most common manner in which perforation is produced.
It may occur even after cicatrisation has commenced. Perforation occurs most frequently, in the lower portion of the ileum, but may occur higher up. It is said to have been, in rare cases observed in the jejunum. More rarely perforation occurs in the vermiform appendix, or in the Colon — in the caecum, ascending or transverse portion, or even in the sigmoid flexure.
Usually there is only one perforation but there may be two or more —

In a case observed in private practice by the writer, there was one perforation situated in the lower portion of the ileum. It was of the pinhole variety. The peritoneal cavity in the vicinity of the right iliac fossa, contained about half a pint of typical typhoid intestinal contents. When the perforation is large, the peritoneum may contain several pints of intestinal fluid, and even intestinal worms. But usually the escape is not copious, as it becomes limited by adhesions; and may give rise to a circumscribed peritoneal abscess, which may ultimately open externally, or into another portion of the bowel, into the gall-bladder, or urinary bladder.

It is said that in cases of enteric fever of very prolonged duration, there may be atrophy of the coats of the intestines.

The abdominal lesions vary greatly in extent in different cases: two or three glands only, may be implicated, or the affected glands may number

1. Murchison on Continued Fevers 3rd. Edition. p. 581. Case LXXV. (also Page 3. of this thesis.)

as many as thirty or forty. There is no relation between the extent of the intestinal lesion, and the severity of the cerebral and abdominal symptoms. But where the diarrhoea has been excessive, there is usually much congestion of the intestinal mucous membrane, and ulceration of the glands. There is rather an abrupt transition at the upper limit of the disease, between the healthy and diseased portions of the bowel, but the extent of the disease increases as the ileo-caecal valve is approached. Here the disease is most extensive, but is abruptly limited by the valve. The lesions are the same in children as in adults; but extensive deposit in, and ulceration of the intestinal glands are said to be more rarely met with; and it is stated that perforation is comparatively rare. The solitary glands are said to be more frequently attacked in children.

The characters above described are quite sufficient to distinctly differentiate the appearance of the intestines in cases of enteric fever, from the alterations

sometimes met with after death from other diseases, such as, Cholera, Variola, Scarlatina, Erysipelas and Pyaemia; also from the appearance likened to a shaven beard by French pathologists, and due to pigmentary deposit; and likewise from tubercular ulceration. The solitary glands of the large intestine, are affected in a manner similar to those of the small intestine. Usually the disease is most extensive in the caecum and ascending colon; but may extend as far as the sigmoid flexure. Occasionally the large intestine is more extensively affected than the small. Two such cases are mentioned by Dr. Roberts in his handbook of medicine 4th. edition page 128, & Dr. Murchison mentions a case in which, the solitary glands of the large intestine alone were affected. The ulcers in the large intestine are usually small and rounded, but may measure an inch and a half in their long diameter, and run transversely, corresponding to the folds of the gut. The large intestine is usually much distended with gas

sometimes to such an extent, as to displace the other abdominal viscera. Thus for example the liver has been displaced so as to simulate the dullness due to pneumonia. The changes in vascularity described in connection with the small intestine, may show themselves with varying intensity in the large intestines. They are not essential lesions of enteric fever. The enlargement of the mesenteric glands on the contrary are primary anatomical lesions peculiar to enteric fever. They begin to enlarge from the onset of the attack just as in the case of the intestinal glands. The enlargement of the mesenteric glands, therefore cannot be regarded, as simply due to irritation from the intestinal ulcers. The meso-colic glands, have been observed, enlarged, in a case where the mucous membrane of the colon was healthy; and diseased mesenteric glands may correspond to healthy intestinal mucous surface, in enteric fever. The nature of the change in the enlarged mesenteric

glands, is essentially the same as that which occurs in the intestinal glands. When the mucous membrane of the colon is affected, the mesocolic glands are likewise usually enlarged. As a rule, those mesenteric glands are most enlarged, which correspond to the portion of intestinal mucous membrane most diseased. Thus the mesenteric glands corresponding to the lower portion of the ileum are most affected; and they become less and less enlarged, as we proceed higher up. The enlarged mesenteric glands may attain the size of a hazel nut, or larger about the fourteenth day of the disease; and although they usually diminish in size from this period onwards, they may still be distinctly enlarged in cases which die about the thirtieth day. On section the glands present about the 14th day, numerous yellow points of friable disintegrated material, which ultimately soften into a puriform material, around the edges, so that on section the enlarged glands present numerous small

small collections of pus, each containing a central slough. Sometimes these run together forming a pseudo-abscess, only separated from the peritoneal sac, by the layer of peritoneal covering, over the affected gland. These so called pseudo abscesses may rupture into the peritoneum, exciting general peritonitis; but more frequently they undergo caseation, or ultimate conversion into a calcareous mass. In cases proving fatal after the 6th week, the glands are universally small, tough and shrivelled, and of a pale grey or bluish colour - The glands in the fissure of the liver, oesophageal, gastric, lumbar and inguinal glands, are sometimes also enlarged; but these are not essential lesions of enteric fever, such lesions being due to irritation.

The Spleen is almost invariably enlarged, in cases which die before the 30th day. In the earlier stages the enlargement is often considerable. The spleen may weigh from three to five times more

than normal; being increased from 4½ oz, to 12 or 20 oz. At first the consistence of the organ is increased, but later it frequently undergoes softening; the contents becoming diffluent. It is said that in some cases it may rupture. The colour of the organ is at first dark purplish, but later it becomes paler. The enlargement is most marked in persons under the age of thirty. This may account for the greater enlargement of the spleen, in enteric than in typhus fever. Haemorrhagic infarctions, are sometimes met with, which undergo softening and disintegration, and may excite peritonitis. It has been thought that those haemorrhagic infarctions consist of the same material as that deposited in the intestinal glands. But this is an error, the infarctions exactly resembling those met with in typhus and other diseases.

The liver and gall-bladder -

The liver may present a variety of morbid

appearances in enteric fever. As a rule the liver is normal in colour, but it may be either hyperaemic or paler than usual Its consistency is not unfrequently diminished; or there may be no apparent alteration in consistency. Where softening is present, (and often also where there is no evident softening,) the outlines of the liver cells may be obscured, their contents granular and cloudy, containing oil globules and pigment. Sometimes the cells are enlarged, and contain several nuclei. According to Hoffman and Zenker, who describe this condition as parenchymatous degeneration, the organ becomes friable and greyish or yellowish in colour. "In the liver the cells become more granular; in many of them are numerous fat granules; the nuclei can no longer be seen. In the higher degrees of degeneration the cells lose their sharp contour, they appear like a conglomeration of granules, and finally they lose all cohesion, and break down into a formless mass of granular detritus"[1].

1. Ziemssen Cyclopaedia of the Practice of Medicine Vol i. p.p. 105; 106.

The small blood-vessels in such livers contain very little blood. On section the organ presents an uniform greyish-red or yellowish-orange appearance. The right lobe is generally more affected than the left. If the patients recover, the granular detritus becomes absorbed; and in cells which have only reached the granular stage, a division of the nuclei takes place, and later a division of the whole cell. This fact was also noted by Hoffman in patients who died of accidental complications, during convalescence after typhoid fever.

Frerichs has related some curious cases, (one, a case of "Abdominal Typhus,")[1] in which, the liver underwent changes precisely similar to those which occur in acute yellow atrophy; with all the characteristic symptoms and lesions of that strange condition. Leucine, tyrosine, and other products of tissue disintegration were present in the diseased organs in some of these cases.[2]

1. A clinical Treatise on Diseases of the Liver, by Dr. Fried. Theod. Frerichs. New Sydenham Society. Eng. Trans. p.p. 215. 216.
2. Compare also: Ibid: p.p. 171; 174; 175. also 172; 173.

Hepatic abscess, Emphysema of the liver, circumscribed pyaemic deposits, and embolism, are other conditions found to occur in the livers of fatal cases of enteric fever. Dr. Murchison describes a case where the liver contained "an opaque yellow mass the size of a pigeon's egg, apparently due to embolism"[1].

Frerichs' case of "Abdominal Typhus" accompanied by circumscribed pyaemic deposits, has been already mentioned; in relation to the presence of Leucine and Tyrosine in the diseased organ, when the structure of the liver has been profoundly altered in the course of the disease[2].

The gall bladder is liable to inflammation in enteric fever. The inflammation may be catarrhal, diphtheritic or ulcerative. When catarrhal, the gall-bladder may be filled with pus. When diphtheritic, the gall bladder and ducts may be lined with diphtheritic exudation, causing hepatic obstruction. The ulceration to

1. Murchison on Continued Fevers. 3rd Edition p. 634.
2. Frerichs. (Op. Cit.) p. p. 172; 173; 174.

which the gall-bladder is liable may lead to perforation and fatal peritonitis; as in a case related by Dr. Murchison (Op. Cit. p. 566.): also in a case under the care of Dr. Russell of the General Hospital Birmingham.[1]
This case died on the 28th day of the disease. Two ulcers were seen near the neck of the gall-bladder. These ulcers had advanced to perforation, allowing the bile to escape into the peritoneal cavity, where a circumscribed peritonitis was set up in consequence. This is in other respects a most interesting case.
The ulcers in the gall-bladder, are not due to the sloughing of a morbid deposit similar to that in the intestines. Ulceration of the gall-bladder is not a primary anatomical lesion of enteric fever.
As might be expected from the material condition of the liver, already described, the condition of the bile is thin and watery, almost colourless and of a low specific gravity: (1010 to 1016, instead of 1026 to 1030.)[2]

1. British Medical Journal. July 12th 1884. p. 67.
2. Murchison (Op. Cit.) p. 635.

The reaction of such bile is acid according to Martin Solon. According to Hoffman, the salivary glands and pancreas undergo parenchymatous change[1]. The pancreas may be diminished in consistency.

Sometimes it is livid or reddish from hyperaemia.

Peritonitis is a common complication of enteric fever. It may result from extension of the morbid process, from the bowel to the peritoneum without perforation. From perforation of the bowel. From the rupture of mesenteric and other abdominal glands, or from rupture of the spleen. From the softening of haemorrhagic infarctions of the spleen. From perforation of ulcers of the gall-bladder. From an abscess in the wall of the urinary bladder or of the ovary; or the rupture inwards of a pseudo abscess, in the substance of the rectus muscle.

The peritonitis may be general or

1. Ziemssen Cyclopaedia of the Practice of Medicine Vol. I. p. 113. (Liebermeister: Typhoid Fever.)

circumscribed.
The condition of the blood varies in different cases. As a rule it presents white fibrinous clots; but sometimes it is dark and fluid, as in typhus. The blood is dark and fluid, when "typhoid symptoms" have existed for some time previous to death. Otherwise the blood is fibrinous and clotted: rarely it has presented a red currant jelly like appearance.
According to Virchow, the white corpuscles are always increased, and the fibrine diminished. He attributes the increase of the white corpuscles, to the enlargement of Peyer's Patches and of the mesenteric glands. Large numbers of dark granular bodies, smaller than red blood corpuscles, were noted in the blood by Virchow. He regarded them as disintegrating red corpuscles. Hoffman makes similar observations as to the increased number of white corpuscles, and the presence of dark coloured granules. He found them particularly in the Portal Vein. Lehmann states that the character of the blood varies, according to the stage of the disease. In the earlier stages it is like that of plethora, the solids being increased particularly the albumen; but also the corpuscles.

This obtains until about the 9th day, after which the blood becomes more fluid: the solids diminishing in proportion to the intensity of the intestinal lesions. The Pericardial Sac may contain a little serum. Rarely the serum is blood stained: and very rarely fragments of lymph indicative of pericarditis, are found in the serum.

The Heart: The heart and bloodvessels in typhoid fever participate in the granular degeneration already described. As a rule the heart is flabby and easily torn. In colour it is pale grey or yellowish brown. These changes may occur throughout the heart, or be more marked in the left ventricle. In the lesser degrees of the change, dark granules which are often highly refractive, are seen above and below the nuclei of the muscle cells. In more advanced cases the granules are seen in large numbers, partially arranged in rows in the lines of the muscular striae. Finally the muscle cells become quite filled with granules, and the striae disappear. Brown pigment-granules are sometimes seen as well as the usual fatty ones. The waxy form of degeneration already described as occurring in the voluntary

muscles, is rarely found in the heart. The functional disturbance of the heart during life, corresponds to the degree of the degeneration.

According to Dr. Hayem, the above process is associated with inflammatory changes in the heart and bloodvessels. Proliferation of the muscle nuclei, and small celled infiltration of the intermuscular connective tissue, due either to exudation, or to connective tissue proliferation Sometimes there is sanguineous infiltration of the intermuscular connective tissue. The above changes may be scattered in patches throughout the heart, and are frequently met with in the musculi papillares of the mitral valve. There is proliferation of the intima of the small arteries due to endoarteritis. Dr. Barié [1] states that there are two forms of endoarteritis, the obliterating and the parietal. Both are characterised anatomically by infiltration of all the coats of the artery with embryonic cells. The internal coat loses its polish, and in the obliterating form, the blood coagulates at the diseased spot, and plugs the vessel.

1. British Medical Journal. March 29th. 1884 p. 626.

In the parietal variety, small clots form on the internal coat, but the circulation is not arrested. These lesions occur most frequently, in the arteries of the lower limbs; but may be found in the Carotids, in those of the upper limbs, heart liver, spleen etc. The obliterating form generally leads to gangrene; whereas in the parietal form, recovery usually takes place. According to Hoffman, the changes in the smaller vessels of the brain kidneys etc. show fatty degeneration; and in the larger ones thickening and opacity of the inner coat: well seen in the pulmonary artery.

The Endocardium resembles the lining of the pulmonary artery, in being opaque and thickened — due to fatty degeneration of its endothelium. Recent endocarditis, with vegitations on the aortic or mitral valves, is also met with in some cases. The dusky-red staining of the endocardium, so frequently met with in typhus is not often found in enteric fever.

Respiratory organs. The epiglottis may be inflamed, oedematous, ulcerated or covered by false membrane. In such cases the disease is in an advanced

stage at the time of death.
Similar changes take place in the larynx.
Sometimes enteric fever is complicated with diphtheria[1]. In some epidemics throat-symptoms prevail.[2] An interesting case is recorded by Dr. G. E. Paget, in which diphtheria was well marked in the course of an attack of enteric fever.[3]
There may be only follicular tonsillitis, or there may be a membranous exudation resembling that of diphtheria.
Membranous deposits seem to be characteristic of certain epidemics.
Ulceration of the mucous membrane occasionally occurs in enteric fever. In some places it is common, in this country comparatively rare.
Trousseau believes that these ulcers are due to inanition in cases which have been kept on too low a regimen. They are met with late in the disease, and are usually associated with great debility.

1. Murchison on Continued Fevers. 3rd Edition. p. 538.
2. British Medical Journal. Sept. 29th 1883. p. p. 645; 646.
3. British Medical Journal. July 14th 1883. p. 64.

Their situation is near the posterior junction of the vocal cords. They may be superficial, or extend so as to destroy the laryngeal cartilages. Sometimes the larynx is perforated; and subcutaneous emphysema may be produced in consequence. They are not due to sloughing out of any morbid deposit similar to that in the intestines.
Bronchitis is often met with. The tubes are then filled with frothy mucous, and the bronchial mucous membrane is congested. This condition is not so frequent in enteric fever, however, as in typhus.
The lungs. Hypostatic congestion occasionally occurs, particularly where typhoid symptoms have existed during life: but it is not so frequent as in typhus.
Sometimes the lungs are healthy.
This is well seen in cases which die suddenly from peritonitis. Hypostatic congestion should be distinguished from true pneumonia, both by its symptoms and morbid anatomy. They are quite

distinct.

Oedema of the lungs is occasionally met with: in the upper lobes most frequently. True pneumonia, also, is frequently met with in enteric fever. It thus contrasts with typhus, in which true pneumonia is rare. The pneumonia may be lobular or lobar: most frequently the former. Patches of lobular pneumonia may degenerate into abscess of the lung, or even gangrene. Haemorrhagic infarctions, due to embolism of branches of the pulmonary artery may give rise to similar results. It is ordinary pneumonia and presents no special "typhus exudation". Recent tubercle may be found in the lungs in protracted cases.

The pleurae are inflamed more frequently than in typhus. This inflammation may give rise to effusion, recent adhesions or empyaema. Sometimes the pleural sac contains a reddish serum: usually in cases where hypostatic congestion of the lungs has occurred. As in ordinary pneumonia, enlargement of the bronchial

glands, sometimes occurs in consequence.
Nervous System. Sometimes there is congestion of the vessels of the cerebral membranes: but not so frequently as in typhus. "There is no relation between the amount of congestion of the membranes found after death, and the cerebral symptoms observed during life. Most frequently there is no congestion of the cerebral meninges: even where the cerebral symptoms have been severe. "The congestion does not give rise to meningitis; although in some rare cases, complicated by pyaemia, tubercle or disease of the temporal bone, meningitis may occur as a complication: as in the case, already referred to, under the care of Dr. Russell".
Haemorrhage into the cavity of the arachnoid is scarcely ever met with in enteric fever.
"The membranes are not easily torn from the brain tissue as a rule: as in typhus.
"The intracranial fluid is increased; but not so markedly as in typhus. It is

1. British Medical Journal. July 12th. 1884. p. 67.

colourless and transparent, and is not a sign of inflammation. There is no relation between the amount of cranial effusion observed after death, and the cerebral symptoms. The fluid seems to fill up the spaces left by the shrinkage of the brain tissues which takes place during the course of the fever. The shrinkage of the brain, and the oedema of its ventricles suggest a degeneration of its elements. According to Hoffman, the grey substance of the cortex presents a diffuse yellowish brown colouration. The corpus striatum and Optic Thalamus are of a diffuse yellow colour, and there is a deposit of small brown pigment granules in these parts of the brain. Sometimes the granules are closely crowded together so as to conceal the outlines of many cells. These appearances were seen in only a small number of cases and further observations are necessary. Softening and induration of the whole brain have been mentioned as occurring in rare cases. There is not any relation between the above-named conditions and the cerebral symptoms during life. Similar post mortem appearances are seen in the brains of cases which die of other diseases. So far as is recorded the Spinal Cord presents a normal appearance. Hoffman found pigmentary degeneration of the ganglion cells of the Sympathetic System.[2]

1 & 2. Ziemssen Cyclopaedia of the Practice of Medicine. Vol i. p. 112.
Liebermeister. — Typhoid Fever.

Virchow describes a similar condition.
Granular pigment is also found in some
nerve fibres, according to Hoffman.
The Urinary organs. The kidneys are often
congested, sometimes markedly so. They
may be dark red and intensely hyperaemic.
This appearance is usually observed in cases
which die during the first fifteen days of the
disease. Sometimes the kidneys are enlarged
and pale; the urinary tubules being blocked
with granular epithelium.
Hoffman and Dr. Murchison have observed
haemorrhagic infarctions as occurring in
the kidneys, in advanced stages of the
disease.
Symptoms. The clinical history varies
greatly in different cases of enteric fever.
The onset is in most cases gradual, and
the patient cannot say when he was
first taken ill, although the intellect
is clear and the memory good. Often at the
outset, there is a chilly feeling, or slight
shivering may take place; and this is
accompanied by lassitude, loss of
appetite, and pains in the limbs.

Headache may be complained of with giddiness and ringing noises in the ears. The tongue is furred, the pulse accelerated. There is a rise of temperature. Nausea and vomiting, with or without diarrhoea are often observed.
Occasionally diarrhoea, with or without sickness and abdominal tenderness are the first symptoms which attract attention. In such cases the injudicious administration of a purgative may induce dangerous diarrhoea.
The skin is hot, and dry with occasional perspirations; and bleeding from the nose may occur. The patient passes restless nights, and feels weak and ill; but still the prostration is not so great as to cause him to take to bed, during the first four or five days. And it is not uncommon for patients to walk about for the first week or ten days of the illness, or even for a longer period.
The fever is remittent in type, the remission taking place in the morning, and the exacerbation in the afternoon

or evening. The above symptoms may exist in various combinations, and with various degrees of severity, during the first week of the illness; and yet there is nothing absolutely distinctive of enteric fever in the case. But an evening temperature of 103° or 104°, with gastric disturbance or diarrhoea, and prostration, occurring in a young person, should make the practitioner suspicious of enteric fever. The patient does not often apply for advice before the 7th day of the disease, when the following symptoms may occur. The pulse is accelerated, may be, to 100 or 120; but it varies greatly in different cases, and in the same patient at different times of the day. It is generally most frequent in the afternoons and evenings, and least frequent in the mornings. It may be soft and compressible, but usually exhibits some resistance. The temperature is two or three degrees lower in the morning than in the evening. The skin is hot and usually

dry, but may be moist and clammy particularly in the morning.
On the 7th day of the disease, or between the 7th and the 12th day, an eruption of well defined, isolated, round or lenticular rose-coloured spots, appears on the abdomen chest and back. The spots are slightly raised above the surface of the skin, and disappear completely on pressure, returning when the pressure is removed. They come out in successive crops, and only a few or large numbers may appear. A circumscribed bright red or pinkish flush sometimes appears on one or both cheeks. It looks not unlike the hectic flush seen in phthisis, and is particularly marked after food, and in the evening. As a rule there is no congestion of the conjunctivae. The headache, pains in the limbs, and disturbed sleep continue, but there is no delirium at this time, and the mind is clear. There is complete loss of appetite,

the lips are dry and parched, and usually there is much thirst. Sometimes bilious vomiting is a troublesome symptom, at this stage of the disease. The tongue is coated by a thin white fur, and is red at the tip and edges. The abdomen is usually distended and gurgling may be felt on pressure in the right-iliac region. Diarrhoea is usually, although not always present. Two to six or more watery ochrey coloured motions may take place in the course of the day. There is enlargement of the Spleen. The urine is scanty, high coloured and concentrated. In mild cases the headache, distress and general pains begin to abate, about the middle of the second week, and at the end of the week, they may not be complained of. The remissions of temperature also become more marked, and a gradual improvement takes place in the patients condition. But in severe cases the temperature

still keeps high, and the headache and other nervous symptoms pass on to somnolence alternating with delirium. At first the delirium may be slight, and may occur only during the night; but at length it becomes more or less continuous. In the intervals the patient's mind may be clear or confused. His countenance is languid or depressed, often expressive of weariness or sadness. Sometimes he lies motionless, with his eyelids closed, but seeming to understand all that is going on around him. He protrudes his tongue, and does what he is told at once. But his answers although prompt are inarticulate and sometimes unintelligible. The character and degree of the delirium varies, but as a rule it is more acute, noisy and active than in typhus. The pulse is 120 or upwards and weak, the pupils are dilated; and the remissions of the fever are less distinct. The lips are dry and cracked; the tongue dry and brown at the base and along the centre; or glazed, red

and marked by deep fissures. Sordes collect on the lips and teeth, and the interior of the mouth is parched. There may be epistaxis. In severe cases the patient sometimes picks the nose and lips until they bleed, and tries to remove membranous looking deposits from the roof of the mouth. The tongue is tremulous when protruded; and general muscular tremors occur as a rule in these severe cases. The diarrhoea continues, and the abdomen becomes more distended and tympanitic. Membranous flakes or blood may occur in the stools. The spots continue to appear, and the old ones fade; but never persist and become converted into petechiæ. Bedsores may form over the sacrum, on the buttocks and other parts subjected to pressure. The patient gradually loses flesh and strength. The urine becomes pale and less dense. There is great prostration of the vital powers, and all the phenomena of the typhoid state become developed. The patient

becomes unconscious, has low muttering delirium and stupor, involuntary evacuations, tremors, subsultus tendinum, and picking at the bed clothes. The tongue becomes dry and brown and the pulse feeble. During this stage the patient may die comatose, or gradual recovery may take place, towards the end of the third, or some time during the fourth week of the disease. But even in the large number of cases which do not go on to the development of the typhoid state, many dangers may arise in the course of the illness. Apart from pulmonary and other complications, which may arise at any time and prove fatal; there are certain special dangers, which may arise from the intestinal lesions with which enteric fever is invariably associated. The dangers are peritonitis arising from perforation of the bowel, which may occur in the 3rd or 4th week of the disease, or earlier. Profuse and exhausting diarrhoea may also occur. Intestinal haemorrhage. In cases of

intestinal haemorrhage, the mental functions are usually unimpaired and the mind may remain clear to the last. It is difficult to decide precisely when the improvement begins, in those cases which recover. There is not a definite crisis as in the case of typhus, but a more gradual lysis; the first favourable indication consisting in a more marked remission of the morning temperature. Convalescence may be interrupted by complications of various kinds, one or other of which may prove fatal. Or after an interval of from ten to fourteen days during which the temperature was normal, and the case perhaps progressing favourably, a relapse of the fever may occur, accompanied by a fresh deposition in the intestinal glands, enlargement of the mesenteric glands, and eruption of rose coloured, lenticular, spots. There is a return of the fever lasting from 10 to 12 days; and as a second relapse may occur it is

evident, that the duration of an attack of enteric fever may be prolonged to two or three months.

The above Clinical description enumerates the symptoms which may be met with in ordinary cases of enteric fever; but the clinical aspects of different individual cases may vary almost indefinitely.

The principal symptoms which may occur in typhoid fever will now be considered in detail.

1. The facial expression may be little altered; and the patient may pass through an attack of enteric fever, without any further alteration than an expression of weariness and sadness, and dilation of the pupils. The heavy stupid expression characteristic of typhus is rarely met with; although in cases where the typhoid state is developed, the expression may be similar to that of typhus. Sometimes the face is unusually pale; but the circumscribed

pink flush already mentioned is characteristic of enteric fever.

2. The spots. The eruption of enteric fever consists of rose coloured lenticular spots, the "taches roses lenticulaires" of Louis. They are isolated, rounded and regular in outline, presenting distinct margins. They measure from half a line to two lines in diameter. Their colour varies slightly according to the complexion, in different cases. They are elevated, and feel slightly convex, when the finger is passed over them; but are never acuminated. In rare instances the spots are vesicular. They appear in successive crops, and are never converted into petechiae. They disappear completely on pressure, and return when the pressure is removed. Each spot lasts, 3, 4 or 5 days and then disappears, while fresh spots continue to show themselves. They are never seen on the dead body. When the rash is unusually copious, the spots may

may be darkish red, and indurated; and may not disappear completely on pressure. They then leave a brown stain after fading away. As a rule the spots are few in number, not more than 20 or 30, at one time. There may be only 3 or 4. But in rare cases the rash may be very copious, several hundreds of spots appearing. In one remarkable instance Dr. Murchison counted 1000. They are said to be less numerous in children than in adults. When numerous the edges of two spots may meet together; but there is never the formation of extensive patches, similar to what occurs in the eruption of typhus. The duration of the eruption varies from 7 to 21 days, according to the time it first appears, and the duration of the fever. Usually fresh spots cease to appear at the commencement of convalescence. But it is important to remember, that sometimes fresh spots continue to appear after the

the general symptoms have subsided; and that as long as fresh spots show themselves, any indiscretion, may bring about a return of the febrile symptoms. The duration of the eruption, is said to be shorter in children than in adults. About 4 or 5 days is the usual duration in children. The date of appearance of the eruption also, is said to be earlier in children than in adults, — as early as the 4th or 5th day occasionally. In adults the rash in rare cases appears before the 4th day — on the 5th or 6th day; or its appearance may be delayed until the 14th or even the 20th day. The most usual distribution of the eruption is over the front of the abdomen and chest, and on the back. Sometimes it appears on the back, before the abdomen and chest; or it may occur on the back, although absent from the front of the body. It is suggested that the early eruption on the back may be due to copious perspiration.

It has been noticed to appear early

also over regions, where a sinapism has been applied. A warm bath is said to favour the appearance of the eruption. The spots may occur on the arms and legs; and Dr. Murchison mentions an unique case, in which he noted the eruption on the face. Unfortunately, the eruption is not invariably present; but the spots can usually be found if a proper examination be made.

It has often occurred that a practitioner in doubt about an obscure case, on calling into consultation one of more extended experience, has had the difficulty at once removed, and the diagnosis cleared up, by a conscientious inspection of the skin covering the chest, abdomen and back.

The case which suggested the above remark, was that of a tall rapidly growing lad of between 14 and 15 years of age. He had catarrhal pneumonia, a fluctuating temperature, and a pulse which varied in frequency. There had been gradual, but marked loss of

of health and strength, with emaciation. There was no diarrhoea. His complexion was fair, eyes light blue, hair flaxen. The brightness of the eyes, and the circumscribed pink flush on the cheeks associated with the pneumonia and other symptoms, strongly recalled the aspect of a person, in the hectic condition, peculiar to phthisis. On consultation, 2 or 3 characteristic spots could be detected on the skin of the abdomen and chest, on careful examination. The lad later, passed through an ordinary attack of enteric fever; and made a good recovery.

The spots are most frequently absent before the age of 10, and after the age of 30 years. There is no relation between the copiousness of the eruption and the severity of the symptoms, in enteric fever; similar to that which exists in the case of typhus. Indeed those observers who have attended to the subject, believe that a copious eruption is a favourable sign, in enteric fever. The eruption above

above described, is peculiar to enteric fever. It is important therefore, to be acquainted with all the minutiae, concerning the characters presented by these lenticular spots.

Dr. Murchison has contrasted the eruption of enteric fever, with that of typhus in such a masterly manner, that his table on the subject, has become classical. It is as follows:—

Enteric Fever.	Typhus
1. Pink or rose-coloured throughout.	1. May be dirty-pink or red at first, but soon become reddish-brown.
2. Undergo no change until they fade or disappear. Never converted into petechiae.	2. Become gradually darker, and are often converted into petechiae.
3. Circular.	3. Of irregular form.
4. Isolated and few in number.	4. Numerous and adhere in patches.
5. No subcutaneous mottling.	5. Mottling common in addition to spots.
6. Elevated above the skin.	6. Not elevated, except at first appearance.
7. Disappear on pressure as long as they last.	7. Do not disappear on pressure, except at first.

Enteric Fever.	Typhus.
8. Rarely appear before 7th day.	8. Appear on 4th or 5th day.
9. Appear in successive crops.	9. Never in successive crops.
10. Each spot lasts only 3 or 4 days.	10. Many of the spots may last to the end of the fever.
11. Never present on dead body.	11. Often persist after death.
12. A large number does not indicate danger.	12. Direct ratio between the number and darkness of the spots, and the severity of the case.[1]

Certain observers have stated that they have met with rose coloured lenticular spots in other acute diseases besides enteric fever; for example in Acute Phthisis. These observations are probably erroneous. Ordinary pimples have been mistaken for the characteristic eruption - Dr. Murchison

1. Dr. Murchison on continued fevers. p.p. 513 & 514.

is of opinion, that an eruption presenting all the characters above numerated is to be found in enteric fever alone amongst acute diseases.

The following case exemplifies the quite rare condition, of some of the lenticular spots presenting a vesicular appearance. It was met with in private practice by the writer. The case was mild in all its symptoms, the only peculiarity being the vesicular eruption.

C. R. aet 7 yrs.

Decr. 20th 1888. On the 18th Decr. patient was unable to continue work at school, on account of lassitude, and a tendency to sleep at her desk. This is probably the 4th or 5th day of the disease - (perhaps a little later 6th or 7th day.) Tongue red at tip. White strawberry appearance on dorsum. Quite conscious. Eyes bright. Spots appearing. Several spots are vesicular, and others present an appearance of scabbing, like the eruption of chicken pox when it is fading.

Decr. 21. Quite conscious. drowsy. Pulse 120. good. Tongue slightly furred. Stomach not tender. Headache slight. Very thirsty. Cheeks flushed: takes milk well.

Decr. 22. Prostration not marked: pulse 120.

DISEASE.

Enteric Fever -

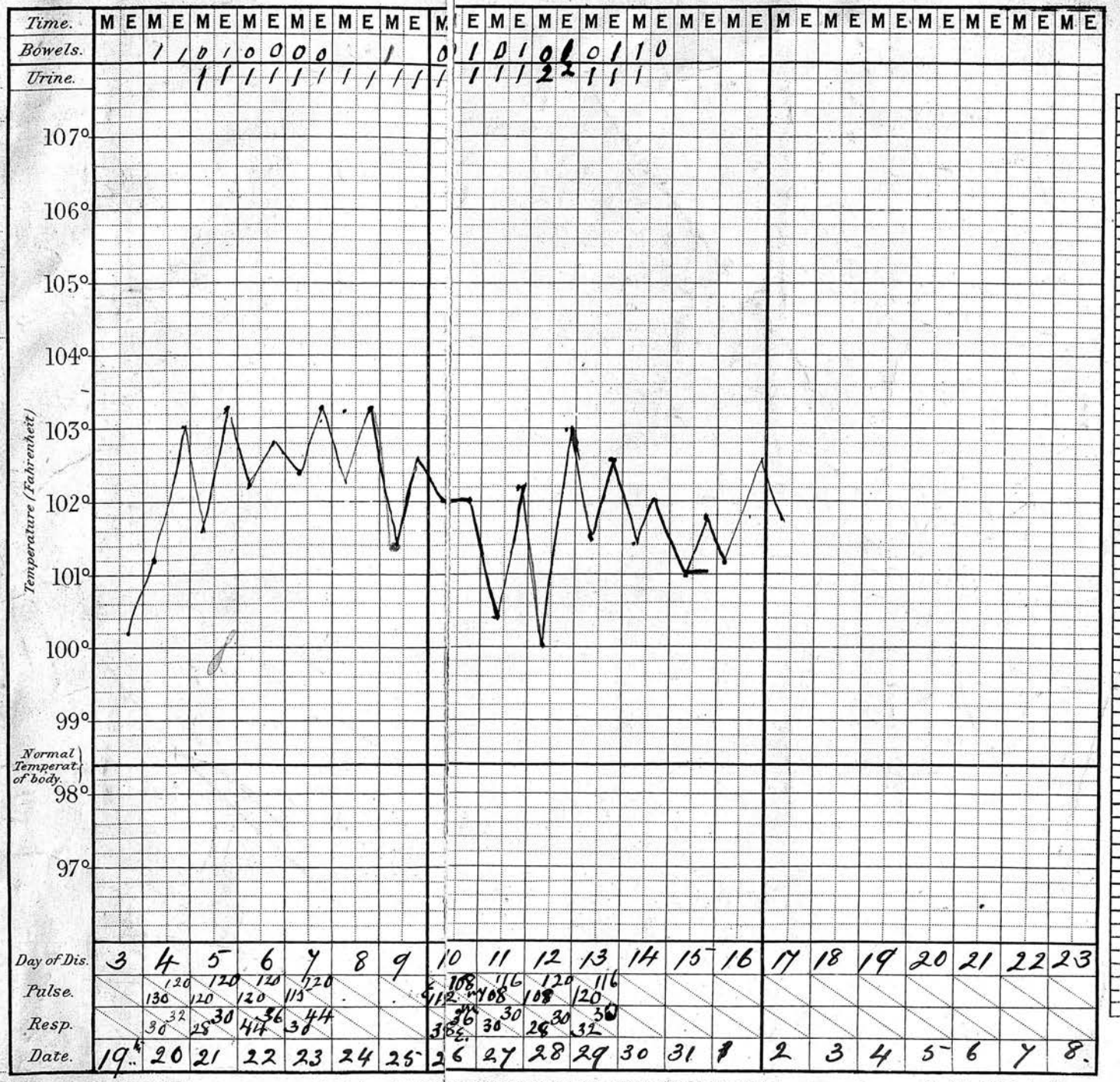
Notes of Case.

Name { Connie Reece -

Age 7 Years

Diet Milk.

Case Book N°



First seen

~~Date of admission.~~

Dec 19th 1888.

Result Recovered -

Entered at Stationers Hall. Printed and Published by Wodderspoon & Co, 7 Serle Street, Lincoln's Inn Gould's Clinical Chart.

Decr. 22. Nausea and pains in stomach - Signs of catarrhal pneumonia. Face flushed.
Decr. 23. Slept well. Face flushed. Eyes bright - Tongue white; mouth dry. Slight cough. There is slight cyanosis due to embarrassed breathing - Evening visit: Troublesome frontal headache + cough -
Decr. 24. Cough and obstruction of respiration considerable.
" 26. Flush on left-cheek - tongue dry. Slept well. but sleep was interrupted by slight delirium - Breathing embarrassed. Pulse. M. 108. E 112. Respirations M. 36. E. 38. Takes 1 quart of milk per diem.
Decr. 27. Cough better. Tongue fawn coloured strawberry with red tip. Complains of thirst and frontal headache.
" 28. Seems better. Tongue moist. Takes milk well. Cries on account of pain in stomach - sudden rise of temperature from 100° in the morning to 103° in the evening.
" 29. Rather better. slept well.
After this a more sudden defervescence than usual occurred: and the temperature became normal on the morning of the 21st. day of the disease, and soon the evening temperature became normal. For some reason, which is not obvious, the temperature chart has not been completed.

Sometimes the lenticular spots are preceded for two or three days by a delicate scarlet rash, which may persist throughout the fever — a reddish or purplish blush being observed on dependent parts in advanced stages of the disease. This condition of skin is not peculiar to enteric fever. The prodromal rash of Variola sometimes presents a diffuse scarlatiniform variety, and the appearance of a similar rash may be met with in other forms of pyrexia. It may be mistaken for Scarlatina, when associated with sore throat
Purpura spots and vibices are met with in rare cases. When petechiae occur, they are independent of the lenticular spots. Spots of a delicate blue tint "taches bleuâtres" are sometimes met with. They are said to occur in mild cases. They are neither elevated, nor affected by pressure. They do not pass through successive stages like typhus eruption, but preserve an uniform tint throughout. They are irregular or rounded: from

from 3 to 8 lines in diameter; and two or three of the vesicles may be confluent. They are met with in other diseases besides enteric fever. They are most common on the abdomen and thighs, and may often be seen following the course of the small subcutaneous veins. Sudamina are met with usually on the chest and abdomen, in cases where perspiration has been profuse. They are met with in other diseases.

Desquamation of the cuticle, is met with chiefly in cases in which sudamina have been present. But in other cases the skin may desquamate in minute branny scales during convalescence. During convalescence also the hair sometimes falls out; and the nails present a furrow which commences at the lunula: above the furrow there is an anaemic band. These changes in the nails commencing at the lunula, advance towards the free extremities. Similar changes in the nails are met with in typhus and other diseases.

The Temperature in enteric fever is

characteristic. The duration of the pyrexia varies, but is seldom of shorter duration than 21 days. It is remittent in type presenting morning remissions and evening exacerbations. In all cases remissions occur at the outset of an attack of enteric fever: and they become more marked towards the termination of cases which recover. sometimes they occur throughout the attack. The mode of ascent is peculiar. The temperature rises in a zig-zag manner so as to be one degree higher each morning and evening, than the morning and evening preceding. In this way a remission of two or three degrees or more, may take place in the morning, so that the evening temperature is about two degrees higher, than the temperature of the same morning. The temperature begins to rise about 12 noon, and attains its maximum, between 4 or 8 in the evening, and 12 midnight. From this point it falls, the most marked remission taking place between 6 and 8 o'clock in the morning.

As a rule the temperature attains its maximum about the 4th. or 6th. day of the disease; but may not reach its highest point until the 8th. day or later. After reaching this point, there is little change in the temperature from day to day, except that the highest point in each day may be slightly less. So that the high temperature is practically 'continued' as long as deposition is taking place in the diseased intestinal glands; and does not alter until about the 12th. day, when ulceration begins. In mild cases, a gradual improvement takes place from this time, and the case may terminate about the 21st. day. But in other cases the temperature may keep high, and indeed, may vary almost indefinitely, in different cases, according to severity and other circumstances connected with the case. The first indication of improvement in mild cases, is that the morning remission becomes more decided; and the evening

evening exacerbation also begins to manifest itself later in the day. At first the evening temperature may not be much altered, but ultimately this also becomes lower until in about 6 or 12 days from the beginning of lysis, the evening temperature is found to be normal — this being the only certain proof of the cessation of the fever. During this lengthened defervescence, the fever may be truly intermittent in type, the morning temperature being normal, but the evening temperature being raised two or three degrees, for a certain length of time. The temperature in severe and protracted cases, about the 12th day, shows a decrease of the morning remissions, and the high temperature may remain 'continued' from day to day, with only slight variations until some time during the fourth week; after which it gradually subsides as in mild cases. Sometimes however from

from the 12th. day, the temperature may rise even higher than before; there may be irregular remissions extending into the evening. Or after a marked remission, the temperature may rise up again, owing to complications, or a recrudescence of the fever. A very sudden fall of temperature, is also an unfavourable symptom - indicating as it usually does, intestinal haemorrhage, epistaxis or profuse diarrhoea. When death takes place by collapse, the temperature may sink below the normal. A sudden fall of the temperature accompanied by a rise of the pulse, occurring after the 14th. day of the disease is indicative of intestinal haemorrhage, even although no blood may have been passed by the bowel at the time that these indications occur. A sudden and irregular rise, especially one which has a marked influence on the morning temperature, more particularly, indicates some local complication. Sometimes

before death the temperature rises to 108° or even 110.3°. (Wunderlich).
The temperature during convalescence is often subnormal; but easily influenced by slight causes, such as emotional excitement, partaking of solid food for the first time &c.
Any persistent rise, however, indicates either complications or a relapse.
The temperature is important as an element in the diagnosis. Thus a temperature of 104°, occurring on the first or second morning of the disease; or a temperature approaching to normal, any evening during the first week, would exclude enteric fever from the diagnosis. But it would not be excluded, as stated by Wunderlich, if the temperature never rose to 103°. It is said that enteric fever may occur in an apyrexial form; all the symptoms and post-mortem appearances being present without any rise of temperature during the course of the disease. Such cases

are related frequently by military surgeons. They seem to occur in patients, who have been subjected to privations and great fatigue - Perspirations. The skin is hot and dry with occasional perspirations, which occur chiefly during the night. The effect of these perspirations on the temperature is only temporary. Sometimes the skin is merely clammy without actual perspiration.

There is often a peculiar febrile odour in cases of typhoid fever; but it said not to be distinctive as in the case of typhus.

The Pulse.

It is important to know that the fever may run its entire course, without any marked change in the frequency of the pulse, although the temperature at the same time reaches 104°. But as a rule the pulse is accelerated. It may be to over 90, 100, 120, or 140. The range varies on different days, and at different hours of the same day; and a variation of as much as 20 or 30 degrees may take place in the same case during the day. The average

frequency of the pulse in different cases also varies greatly. When the average frequency of the pulse, in an adult is over 130, the prognosis is unfavourable. But cases in which the pulse has never been very frequent during the illness may also prove fatal. During the first ten or twelve days of the illness, the pulse may exhibit some resistance; but during the later stages of the disease, it may become, soft, compressible, irregular, intermittent, feeble or even imperceptible Sometimes the pulse is unusually slow during the whole attack. It may fall to 60 or less. Dr. Murchison relates one case where it fell to 32. As might be expected from the parenchymatous degeneration of the heart, its action is impaired, sometimes to a marked degree. Hence the importance of examining the heart in all cases as well as the pulse; and of insisting upon the patient being kept recumbent during the attack. In severe cases, there is diminution or loss of the cardiac impulse, and impairment or loss of the 1st. sound. In mild cases, the impulses and sounds

may be unaltered, or only slightly diminished and impaired

Respiratory System. The frequency of the respirations is increased in enteric fever, independently of pulmonary complications. They may be increased to 20 or 40 in the minute. When they exceed 40, pulmonary complications are usually present. The frequency of the respirations varies with the pulse, except when the latter is unusually slow.

The breathing may be irregular and noisy — sometimes blowing or hissing, the 'Nervous respiration' of Sir D. Corrigan - "the mouth is kept closed, the cheeks puff out, and the nostrils dilate with each expiration." "There may be a long pause followed by a deep inspiration; and this by a number of often short and rapid inspirations" The nervous breathing usually indicates profound cerebral disturbance, and is an unfavourable symptom. The breath is very offensive when the typhoid stage is developed. In such cases the respired air has been found to contain ammonia.

The digestive apparatus. The appetite is usually lost, but may continue throughout the disease in mild cases.

The tongue is often tremulous when protruded. It has been suggested that this is due in part, to parenchymatous degeneration of its muscular fibres[1]; although the nervous system must exercise some influence on the phenomenon. Inability to protrude the tongue is much rarer than in typhus. In the earlier stages of the disease the tongue is moist. Usually it is covered with a thin white fur, and red at the tip and edges. It may remain like this, or with the white fur merely changing to a fawn colour, throughout the attack; or about the middle or end of the second week, the appearance of the tongue changes. It becomes dry and brown over a triangular area in front, and along the centre; and afterwards it may be covered with a thick brownish crust; or it may be red clean, dry, glazed, fissured, sometimes deeply, and looking like a piece of raw meat. The unusual redness of the tongue in typhoid fever is peculiar. Sometimes the tongue is unusually red, with enlarged papillae

1. Ziemssen's Cyclopaedia Vol i. (Liebermeister - Typhoid Fever.) p. 110.

like a red strawberry. Sometimes enlarged papillae present themselves, through a thin fawn coloured fur. Sometimes deep transverse fissures occur, which are very painful; and may proceed to the formation of ulcers, which lead to great cicatrisation of the tongue. Those cases, with a dry brown tongue are not necessarily fatal; and death often occurs in cases, where the tongue has remained moist during the attack. The lips are usually dry and parched; in severe cases, they may crack and bleed, a condition often aggravated by the patient picking his lips. Sordes collect on the teeth when the typhoid stage is developed. Haemorrhage from the gums is rare. Thirst is usually complained of in the early stages; sometimes it is excessive. Nausea and retching are common symptoms at the commencement of the attack; and may be among the earliest symptoms; sometimes retching and vomiting are slight, at other times they are persistent and distressing. Such symptoms are said to precede a mild attack, and often enough

in such cases, the subsequent attack is mild. After the second week of the disease, vomiting is a more grave symptom, and may indicate the onset of peritonitis. The vomited matters as a rule are greenish and bilious looking. But sometimes they contain blood; and when perforation occurs, stercoraceous vomiting may occur before death.

Tympanites is usually present, sometimes it is a prominent symptom. It is most marked in severe cases; and unlike that of typhus, is almost always associated with diarrhoea, abdominal pain, and tenderness. Its convexity has a direction from side to side, and not from above downwards; this direction of the convexity, being due to meteorism arising for the most part from distension of the colon. The meteorism in mild cases is often absent or not well marked. As a rule typanites does not supervene until after the sixth day of the disease, but has been observed as early as the third day. Sometimes a sensation of gurgling is felt, when sudden pressure is made in the right iliac region

This symptom is often absent. It is said to be more frequently present, than in ordinary diarrhoea. Abdominal pain and tenderness are frequent, but not invariable symptoms. Patients often complain of pain in the abdomen; and still more frequently, there is tenderness on pressure in the Right Iliac Region. When asked what part of the abdomen is most painful, patients will often place their hands over the right-iliac region. Cases in which abdominal pain is an early and severe symptom, often prove fatal. The pain is often absent or slight, in mild cases. The motions are rarely accompanied by pain, and never by tenesmus.

The spleen is enlarged, particularly in patients under thirty years of age; and towards the end of the second week of the disease. Often it can be felt through the abdominal walls

Diarrhoea. Although diarrhoea is usually a prominent symptom of enteric fever, it may be absent, or

or there may even be constipation. The period of commencement, and duration of the diarrhoea, vary in different cases. It may be an early symptom, and the first thing that attracts the patient's attention; sometimes lasting throughout the attack in such cases, or soon passing off. Or it may have existed for a few days, previous to the time at which the patient came under observation, and then passed off, not reappearing again during the course of the illness. Sometimes diarrhoea does not occur as an early symptom; but urgent diarrhoea supervenes after the administration of a purgative; and then lasts throughout the remainder of the illness. It seems to be the experience of most observers, that the severity and danger of an attack of enteric fever, is in direct proportion to the urgency of the diarrhoea. At the same time, some cases prove fatal from intestinal haemorrhage or perforation, where there has been no diarrhoea; or even where there has

On Typhoid Fever.

Part 2.

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has been constipation. The bowels may be constipated during the primary attack, and relaxed during a relapse of enteric fever. The stools of typhoid fever, present a characteristic appearance. They are of fluid consistence and usually the colour of yellow ochre. They separate into two portions on standing, a supernatant fluid, and a flocculent sediment. The fluid is pale yellowish or brownish in colour, and has a specific gravity of 1015. This fluid contains about 40 parts in 1000, of solid matter, which consists for the most part of albumen and Chloride of sodium. Traces of other soluble salts are found. The deposit consists of particles of disintegrating epithelium, shreds of sloughs from the intestinal ulcers, blood corpuscles, particles of undigested food, and large quantities of crystals of triple phosphates. The reaction of the motions in typhoid fever is alkaline, instead of acid — the normal reaction. This alkalinity is due to Carbonate of ammonia, and also to a fixed alkali. Schönlein believed that the

the presence of triple phosphate in the motions, was peculiar to enteric fever. But it is now known that this substance may occur in the motions in other diseases, in which the motions are prone to decomposition. Sometimes the motions present an appearance like that of bird-lime; or they may present a pultaceous appearance; being frothy as if fermenting. Under such circumstances (when frothy and pultaceous,) they are light enough to float in water. Sometimes the motions contain blood; their odour is very offensive, and often ammoniacal.

The above characteristics of typhoid stools, are best seen after the tenth day of the disease. Shreds of sloughs from the intestinal ulcers are not seen before the 14th day.

Intestinal haemmorrhage. This is an important symptom: it seems to occur less frequently in children than in adults. The quantity lost varies from a few drops to several pints. The blood is usually fluid, but may be clotted. The colour of the blood may be dark,

but more frequently it is bright red, on account of the alkaline condition of the intestinal contents. As a rule the cases in which intestinal haemorrhage occur, are severe, and preceded by diarrhoea; but sometimes the cases are mild, and there is no diarrhoea, or even there may be constipation preceding them. Most commonly the haemorrhage occurs, some time during the third or fourth weeks of the disease; but it may occur, any time between the second week, more particularly towards its close, and the 8th week of the disease. Sometimes it occurs as early as the fifth day, before ulceration has commenced. In three cases recorded by Dr. Murchison, haemorrhage took place on the 16th, 18th and 19th days, and recurred on the 49th, 33rd and 44th days. Sometimes extensive haemorrhage into the bowels occurs, causing death, before any blood is voided externally. A sudden fall of temperature, the pulse increasing in frequency while the temperature falls,

accompanied by sudden collapse and pallor of the surface, are indicative of copious intestinal haemorrhage, even when blood is not voided externally. In such cases the temperature speedily rises again, and may even exceed its previous height. Sometimes haemorrhage from the bowel, is associated with epistaxis, haemoptysis, haematemesis, haematuria, purpura spots, bleeding from the gums, and other haemorrhages, when the name of haemorrhagic putrid fever is given to the disease. The source of the haemorrhage varies, according to the time at which it takes place. When copious, and occurring at the end of the second week or later, the haemorrhage, probably takes place from ulceration into a capillary vessel in the intestinal mucous membrane; or it may result from a sungating condition of the semidetached slough. Sir Wm. Jenner has observed water flow freely, from one of the intestinal ulcers, when it was injected into the

the superior mesenteric artery. When the haemorrhage occurs before the end of the second week, and is associated with haemorrhages elsewhere; it probably results, from hyperaemia of the capillaries of the intestinal mucous membrane, or from a liquefied condition of the blood. This explanation is necessary to explain those cases of intestinal haemorrhage, which occur on the fifth or sixth day, before ulceration has commenced. There is some difference of opinion with regard to the prognostic significance of intestinal haemorrhage.

Some authorities such as Trousseau and Dr. Graves, maintain that it is not so dangerous, as has been supposed, and that it may even do good; and that most cases recover. But the consensus of opinion supports the belief, that intestinal haemorrhage, especially when it occurs after the 12th day, is a formidable symptom. Even a slight haemorrhage, at this time, may be the precursor of one which

which is copious. Fatal syncope sometimes succeeds copious haemorrhage from the bowels, in cases which were previously doing well. Cases in which haemorrhage occurs, are also particularly prone to peritonitis. In order to cause extensive haemorrhage, the ulceration must have proceeded deep enough, to penetrate the transverse muscular fibres of the gut; and it is easy to understand, how an ulcer of such depth may proceed to perforation, or cause peritonitis by extension of the inflammatory process to the peritoneum. In the early stages of the disease and when slight, it is possible that haemorrhage may relieve congestion of the intestinal capillaries. But although recovery may take place from the more formidable variety of intestinal haemorrhage, it is difficult to understand how it can under any circumstances prove beneficial.

Symptoms referable to the Urinary System.

The quantity, during the first week or ten days is diminished — may be to

to one third, one fourth, or even one sixth of its usual quantity. After this the quantity per diem increases, and the urine may be copious, and of low specific gravity, before the cessation of the fever. The quantity however, is not markedly increased until convalescence; when as much as 80—90 ounces of pale watery urine may be passed in the twenty four hours. The colour is at first darker than usual; in the advanced stages and during convalescence the urine is pale. The acidity of the urine is increased during the first week or ten days, owing to concentration. During the third and fourth weeks, it is very feebly acid, or even alkaline, due to the decomposition of urea, or the presence of fixed alkali. The specific gravity. At an early period of the disease, when the urine is scanty, the specific gravity may range from 1025—1030 or more. But when the disease is advanced, and particularly when the typhoid state becomes

developed, the specific gravity is low. It is low also during convalescence, when it may be only 1003 — 1005.

The amount of Urea excreted per diem is increased, on an average by one fifth; but sometimes, the amount excreted may greatly exceed the normal — 1200 grains have been found by A. Vogel. The increased elimination of urea during the first week, is most marked; but there is also elimination of urea, above the normal, during the whole time that the fever lasts. During convalescence, the elimination of urea is diminished, on account of the increased metabolism of nitrogenous tissues, which occurs during the fever, having come to an end, and being succeeded by diminished tissue metabolism and a renewal of the formative processes.

There seems to be a close relationship between the amount of urea excreted and the temperature. Consequently both are greatest during the first week of the disease. The quantity of urea excreted is greatest when the temperature is highest,

even although the temperature may be influenced by perspirations, and other causes which favour the abstraction of heat from the body; and although the amount of urea excreted, is not always an index of the amount of tissue metabolism which is taking place. Certain circumstances such as acute inflammatory affections, for example Pleurisy &c, may actually cause the amount of urea excreted to fall below the standard. Instead of being eliminated, the products of tissue metabolism may accumulate in the blood, on account of some defect in the excretory apparatus of the kidneys, as proved by the presence of tube casts and albumen in the urine in such cases. And under such circumstances, stupor somnolence, delirium, coma, convulsions and other phenomena of the typhoid state may become developed. Some pathologists have attributed the phenomena of the typhoid state to Septicaemia, due to absorption of gas, from the intestinal ulcers.

But although such absorption may be possible, and no doubt does occur in some cases; the typhoid state becomes developed in the course of other diseases in which there is no intestinal ulceration, and there is no relation between the severity of the cerebral symptoms, and the extent of ulceration in those cases in which ulceration does occur. It has moreover been proved, that the cerebral symptoms of typhoid fever, are independent of inflammatory conditions of the brain or its membranes. Therefore the phenomena of the typhoid state must be due to the accumulation in the blood of the products of destructive metabolism derived from the disintegrating tissues; which products of metabolism ought to have been eliminated by the kidneys. It has been observed that when the elimination of urea is diminished, on account of failure in the excretory function of the kidneys, cerebral symptoms become developed in

in certain cases, and that these symptoms soon disappear, after the elimination of urea is again increased.

Uric acid. The amount of uric acid excreted is always increased; the amount of increase may be up to three times the normal standard. The amount increases up to the fourteenth day, and then decreases. During convalescence, the amount excreted is below the normal. Deposits of urates may take place which are not necessarily critical. They may occur at any stage of the disease. The chlorides, are diminished to a trace; they seem to be retained within the system, and are discharged copiously during convalescence. It has been suggested that the chlorides are diminished in consequence of the small quantity of Chloride of Sodium, taken with the food, and to the large quantity discharged with the motions. But they have been found diminished in cases where there was no change in diet, and where there was neither

neither diarrhoea nor pneumonia
Albuminuria occurs in about one third
of the cases of enteric fever. It is most
frequently met with in the third week
of the disease; and rarely appears
before the sixteenth day. When
albuminuria appears, cerebral symptoms
often become developed, owing
apparently, to the non-elimination
of the products of tissue metabolism,
on account of the damaged state
of the kidneys. Cases of enteric fever
with much or persistent albuminuria
are usually severe, and have typhoid
symptoms well developed. In such
cases after death, the kidneys are often
found congested, the cortices hypertrophied,
and the urinary tubules blocked with
granular epithelium. Renal epithelium,
blood, and tube casts, may or may not
coexist with albumen in the urine.
Sometimes there is copious haemorrhage
from the kidneys, which is often
associated with other haemorrhages.
It is said that tube casts may be found

where there is no albuminuria.
Leucine and tyrosine have been found, under special circumstances.
Earthy and triple phosphates, are often copiously deposited in the advanced stages, when the urine is feebly acid.
Symptoms connected with the Nervous System. Headache is an almost-invariable symptom. As a rule it comes on with the invasion of the disease and exists from the first; in other cases it comes on, not later than the sixth day of the disease. It is most-severe during the first week, and as a rule passes away at the end of a fortnight. The headache is dull heavy and aching in character; its situation as a rule is frontal, but may extend over the head. It does not shoot, nor is the feeling described as bursting.
Vertigo is a frequent symptom, which may persist throughout the attack.
Pains in the limbs of an aching or indefinite character, are complained of from the outset; sometimes there is

true rachalgia — cervical or dorsal.
The pains are usually worse in the lower extremities, and sometimes they take on a neuralgic form: sometimes they are articular so as to simulate Rheumatism.

Mental Symptoms. Many patients pass through an attack of enteric fever, without any delirium or impairment of the mental faculties. Some moan and talk a little during sleep; but when awake, there is no mental confusion or stupor — they are perfectly rational. It is remarkable, that in cases where the mind has been unusually clear throughout the attack, intestinal perforation often takes place. But in the majority of cases of enteric fever, there is some degree of delirium and mental confusion, or somnolence.
The delirium may be slight and mild only occurring during the night, and the patient being quite rational for the greater part of the time; or the delirium may be more severe, and in character, it is often active and noisy at first.

The patient may attempt to get out of bed, sing snatches of songs, and shout laugh and vociferate. Sometimes they have jumped from windows, plunged themselves into deep waters, or done themselves other forms of injury. But later in the disease the delirium may become low and muttering, especially in cases where the typhoid state becomes developed — this form is the "typhomania" of Galen. Sometimes the patient is restless sleepless and fidgety, trying to get out of bed, apparently without any definite object. He is tremulous, and the character of the delirium is busy, and closely simulates the delirium tremens of the drunkard. The active, noisy form of delirium, is more common in enteric fever than in typhus, probably on account of the patients being as a rule younger and more robust in the former disease, than in typhus. In enteric fever, delirium rarely comes on before the end of the second week; but it may come on much earlier, or

or not until the third week. Sometimes delirium is one of the earliest symptoms noticed, and under such circumstances it may be mistaken for mania. Sometimes it comes on at the outset of the disease, and ceases on the appearance of the eruption. When it comes on late — in the third week — it only lasts a few days before death or recovery. Delusions are often present during the delirium, and sometimes they take very curious forms. But as a rule the acute noisy variety of delirium is accompanied by wandering of the mind, whereas in the quieter forms, the mind is centred on some fixed object. It is said that when patients in a delirious condition, suffering under the most dangerous symptoms, insist that there is nothing the matter with them, they almost invariably die. Delirium as a rule occurs earlier in children than in adults. During the early stages of enteric fever, the patient commonly is wakeful, and passes disturbed nights.

About the end of the second week, more or less somnolence becomes developed: the patient is then restless and delirious during the night; and more or less drowsy and stupid during the day. Somnolence may come on much earlier however, and show itself as early as the first week or even the first day of the disease. Sometimes the patient lies quite still, appearing to know what is taking place around him; but he is in a state of somnolence. His eyelids are half closed, the pupils dilated, the countenance apathetic, and calm rather than stupid; and he looks like a patient in a state of hysteric coma. In typhoid fever, as a rule somnolence is developed as a cerebral symptom, prior to delirium; in this respect presenting a contrast to typhus, in which, somnolence and coma, supervene upon delirium.
In enteric fever the somnolence becomes interrupted by delirium, the latter usually occurring during the night, and the former during the day. In children somnolence occurs in the same manner as in adults; sometimes it is markedly an early

symptom at the outset of the disease in children. In the case of C. R. attended by the writer;[1] the first symptom noticed was that the little girl fell asleep over her books at school, each time she tried to attend to her lessons. The degree of muscular prostration varies greatly in different cases of enteric fever. There is a certain amount of prostration in all cases from the commencement of the attack. But prostration as a rule is not marked at first in ordinary cases; and a patient may pass through an attack of enteric fever, without the occurrence of any marked prostration. In cases in which the prostration becomes complete, it rarely supervenes before the third week of the disease. Sometimes patients walk about throughout the whole of the attack, and even close up to the time of death in fatal cases. It is well known that patients have walked to hospital, and attended as out patients, throughout an attack of enteric fever, in a mild form; often such cases die suddenly from perforation of the bowel.

1. Case of C. R. p. 125 of this thesis.

Enteric fever contrasts with typhus, in the late development of muscular prostration in the former. It is the rule in enteric fever, that muscular prostration is not marked before the 4th day, and often it is not marked before the 14th day. The average time at which patients come under treatment is probably about the 10th day: so that practitioners rarely see cases before the disease has lasted for at least a few days. In enteric fever, the decubitus is usually dorsal. Involuntary evacuations may occur where there is complete prostration. Retention of urine is extremely rare in enteric fever. In grave cases there may be inability to protrude the tongue, and difficulty in swallowing. Sometimes there is marked dysphagia before the fatal event. Muscular tremors are a more or less prominent symptom; sometimes they are severe, and may occur in young people, who have not been intemperate in their habits prior to the attack. These tremors may exist

where there is no delirium, and where the mind is perfectly clear. The tongue is often markedly tremulous when protruded, and the muscles of the lips can be observed in a tremulous condition. The hands are tremulous, and there is ataxia associated with the tremor. Severe tremors occurring in cases in which the mind is clear, are said to indicate deep ulceration of the bowel, and sloughing of Peyer's patches.

Spasmodic movements such as subsultus tendinum, carphology, twitching of the mouth, choreic movements and protracted hiccough, may occur in the advanced stages of severe cases, and often usher in the fatal event. In some severe cases, muscular rigidity of various muscles may occur. The most common situations are the trunk, neck and extremities. The head may be rigidly retracted impeding deglutition and respiration.

There may be spasmodic constriction of the pharynx, strabismus, trismus or spasm of the glottis simulating laryngitis. Cataleptic rigidity may occur, particularly in females.

In one case attended in private practice by the writer, there was spasmodic rigidity of the muscles of the limbs, and of the neck. The rigidity was most marked in the lower extremities. The thighs were flexed on the abdomen, and the legs on the thighs. There was considerable contracture also in the muscles of the upper extremities. The muscles of the neck also were in a state of contracture, and the head was retracted. In addition to this bedsores formed on the buttocks, and other parts subjected to pressure. Altogether, the case presented a picture not soon to be forgotten. It was a very severe case in all its symptoms. Somnolence supervened early, the delirium was wild and maniacal, the temperature ran high, and the pulse

was much accelerated and feeble.
Hypostatic congestion of the lungs was a formidable complication. The condition of the mouth and tongue became foul; the tongue about the 3rd or 4th week was covered with a brown crust. Diphtheritic false membranes formed in the mouth and over the fauces. The patient picked the membrane off the palate in shreds, when he got the opportunity, causing the denuded surfaces to bleed. He also picked his lips and nose badly. His life was despaired of; but after the case had been prolonged to a duration of about two months from complications, convalescence set in. The delirium passed into a maniacal condition which lasted up till the commencement of convalescence: and for a considerable time after convalescence had set in, there was mental fatuity, and impairment of the mental faculties generally. The maniacal condition lasted so long, and so much interfered

interfered with the patient's sleep, that it was thought he would sink from exhaustion. For this condition, Paraldehyde seemed to do more good than any other drug. Chloral, Sulphonal, morphia and others were tried; but under the influence of full doses of Paraldehyde made up in Almond mixture, the mental symptoms subsided; and the patient had refreshing sleep, on the night following upon the first day that the Paraldehyde was administered.

The only disadvantage that could be noticed from its use, was that the breath and exhalations from the patient acquired a peculiarly disagreeable odour.

The muscular contracture in this case gradually yielded to massage, and became much relieved. But many months elapsed before the contracture entirely disappeared. When the patient had recovered strength sufficiently to go out of doors, he

he used to ride about on a tricycle with the object of moving the muscles of the lower extremities, and overcoming the contracture. Ultimately the boy whose age was between fourteen and fifteen years, made a good recovery.

General convulsions rarely occur in enteric fever; and they are rarely uraemic when they do occur. They seem to be more common in children, the fatal event in such cases taking place by coma preceded by convulsions.

Congestion of the conjunctivae so common in typhus is rare in enteric fever; and when it does occur, it is later in making its appearance than in typhus. There may be pain in the eyes, without congestion of the conjunctivae in enteric fever.

In typhoid fever, the pupil is dilated. This fact was first pointed out by Sir Wm. Jenner, who contrasted the condition with the small pupil of typhus. The dilation may occur in cases in which the mind is clear, or it may be associated with delirium and impairment

impairment of the mental faculties.
Dilation of the pupils is particularly marked in those cases, where the appearance of the patient, approaches to that of hysteric coma; and it usually comes on after the tenth day of the disease. Sometimes the pupils are contracted in enteric fever, particularly where there is complete insensibility and stupor. When the eye is congested contraction occurs; dilation of the pupil corresponding to anaemia of the eye. Sometimes inequality of the pupils is observed, without any tubercle or other lesion to account for the condition. Haziness of vision on sitting up, may be complained of; and strabismus is met with as a very rare condition.
The sense of hearing is sometimes affected. Ringing and buzzing noises in the ears, are often complained of in the early stages. These symptoms are said to be most marked in severe cases.
Deafness of one or both ears is a

frequent symptom. It is said to be of unfavourable prognostic significance when it affects only one ear, as it may then be due to suppuration of the ear, which may excite meningitis.
Deafness is rarely observed before the end of the second week.
Localised cutaneous hyperaesthesias are often met with, particularly in women and children. They may occur in the first week of the disease or not until convalescence. The chief seats of hyperaesthesia, are the abdomen and lower extremities. The hyperaesthesia always follows an ascending course, the upper limit being well defined, and the condition is present in every part of the limb below the upper margin.
Anaesthesia is said to be a grave symptom in children.
Epistaxis is a common symptom.
It may occur at any period of the disease; and repeated attacks may take place. It is said to be more common in Paris than in this country.

The quantity lost may be only a few drops or several pounds. Epistaxis has no favourable effect on the course of the disease; and may be the direct cause of death from its copiousness. Emaciation is marked, and often extreme, in cases which are prolonged to three or four weeks. In the case referred to, in which muscular contracture existed, the emaciation was remarkably pronounced. Although the patient lay on a water bed, it seemed as if the more prominent bony points would penetrate the skin.

The principal symptoms having now been enumerated; the clinical history may be reviewed as is usual, by considering the disease as it occurs in its different stages. These stages are purely artificial, but are useful for purposes of description.

The stages usually described, are those of incubation; invasion; deposition or enlargement; ulceration or sloughing; lysis, and convalescence.

It is evident that there is a parallelism between the division of the clinical phenomena into stages, and the description of the anatomical lesions as they occur in successive stages.

1. The period of incubation has already been considered under the heading of etiology.

2. The stage of invasion. This is the stage which extends from the first feeling of illness, to the development of decidedly febrile symptoms. It lasts for one or more days. The invasion is gradual, and it is often impossible for the patient or his friends to state precisely the day on which the illness began. Sometimes there may be irregular chills or slight rigors at the onset of the disease. But as a rule, distinct rigors are absent — not always however; and in rare cases the rigors may be prolonged and ague like. Headache, slight irregular chills, and general aching of the limbs, sometimes of a neuralgic character, lassitude and

and giddiness, are the earliest symptoms, which are usually met with. Nausea, vomiting, diarrhoea, boils and abscesses have also been noted among the early symptoms. But one symptom is never absent; there is invariably elevation of temperature from the first.

3. The stage of glandular enlargement. Strictly speaking, this stage includes the period of invasion. It extends from the first to the twelfth or fourteenth day of the disease; and is characterised by headache; giddiness; general aching; pains in the limbs; increasing muscular prostration; disturbed sleep; a remittent temperature rising in a regular manner, and attaining its maximum on the fourth or sixth day; a furred tongue, red at the tip and margins; abdominal tenderness and diarrhoea; increase in the amount of urea excreted; occasional epistaxis, and vomiting; and the appearance of the eruption. Occasionally acute delirium, bronchitis and pulmonary engorgement, leading to a fatal result. But as a rule

death does not occur during this period; and often the patient may not yet have taken to bed. Some of the intestinal glands probably continue to enlarge, after ulceration has begun in others. Sometimes there are no abdominal symptoms.

4. The stage of ulceration or sloughing, lasts from the twelfth or fourteenth day, to some time between the twenty-first and twenty-eighth day. It is characterised by persistence of the fever, with less decided remissions; successive crops of eruption; dry tongue — sometimes red, glazed and fissured; increase of the diarrhoea and abdominal distension. The motions often contain membranous flakes or blood. Retention of urea in the system, and the development of the phenomena of the typhoid state. The case may be prolonged by pulmonary, abdominal or other complications; or independently of such complications, may be protracted into the fourth or fifth week.

Wunderlich describes an amphibolic stage, or stage of changing fortunes,

as occurring during this period; in which after decided remissions, or even intermissions of the temperature, a recrudescence of the fever takes place, and the duration of the disease is prolonged in consequence. Such recrudescences are distinct from true relapses. A series of recrudescences may occur in succession.

5. The stage of lysis, in mild cases which do not go on to ulceration, may begin as early as the end of the second week; and then the morning remissions become more marked. At first there is not much difference in the evening temperature; but at length, the evening remissions also become more decided, until towards the end of the fever, the temperature may be truly intermittent — the pulse and temperature being normal in the morning, but the evening temperature raised two or three degrees.

In most cases however, the lysis does not begin until some time during the fourth week; and it may be interrupted by complications of

of various kinds. In addition to the marked remission in the morning temperature, which indicates the advent of lysis; the tongue becomes cleaner and moister; the cerebral symptoms subside, and fresh spots cease to appear. It will thus be seen that the defervescence is gradual. It takes place by lysis — not by crisis — and is not accompanied by critical evacuations.

6. Convalescence, cannot be said to have begun, before the bodily temperature has been normal on two successive evenings. It may be interrupted by the deposit of tubercle, peritonitis, and other sequelae. And even where it is not so interrupted, convalescence is always slow in protracted cases. Often, although the temperature does not rise, the pulse is more frequent than during the fever; and it is a long time before the patient regains appetite and strength. The duration of the illness in enteric fever, may be stated as three or four weeks. In many cases

however, are much more protracted, or of shorter duration. It is rare for fresh spots to be found appearing after the 35th day; but Dr. Murchison relates one remarkable case in which fresh spots continued to appear, up to the 60th day; so that even uncomplicated cases may be prolonged beyond the fourth week. No doubt many cases of so called febricula, are simply abortive attacks of enteric fever which have subsided during the second week of the disease, or before the twentieth day. Unless complicated, or associated with non cicatrisation of the intestinal ulcers, enteric fever is rarely protracted beyond the middle of the fourth week. Although cases are rarely fatal before the 14th day, the fatal event has been recorded as occurring as early as the 12th, 7th, 6th, 5th, or even in less than four days.[1] It is said that cases of enteric fever may terminate fatally even on the

[1] Murchison on Continued Fevers. 3rd Edition. p. 549.

the first or second day.
The symptoms in these rapid cases are said to be severe headache, acute delirium, profuse diarrhoea or great engorgement of the lungs.

Relapses. In a certain proportion of cases which varies in different epidemics, relapses occur. By a relapse is meant, a fresh evolution of the primary fever, with fresh deposition in the intestinal and mesenteric glands, and a fresh eruption of the rose coloured lenticular spots. The spots appear earlier in the secondary than in the primary fever — from the 3rd to the 4th day. In rare cases the eruption may be delayed beyond the seventh day in a relapse.
Usually there is an interval of ten or twelve days, during which the temperature is normal, intervening between the primary and the secondary attacks; but it seems that cases may occur, in which there is no apyrexial interval, but in which the secondary.

attack overlaps the primary. Some unusually prolonged cases of enteric fever are explained in this way.

True relapses have to be distinguished from recrudescences, which occur during the period of ulceration.

The points on which the diagnosis of relapses must be grounded are, the accession of fever usually after an apyrexial interval, the appearance of fresh spots, and fresh enlargement of the spleen; and the absence of all inflammatory and other complications, to account for the accession of fever.

The duration of the second attack is usually shorter than that of the primary attack, although this is not invariably the case. Sometimes the primary attack is mild and abortive, and the secondary attack, is of the ordinary duration. Such a case, occured to the writer, in private practice. After a mild attack which apparently subsided within three weeks, the patient, a young man, became careless, as he did not

not feel ill. And three or four days after the evening temperature had first become normal, he partook of a mutton chop. An ordinary attack of enteric fever, of between three and four weeks duration ensued. But in this connection it must be stated, that Dr. Murchison has never been able to trace relapses to errors of diet; and the above case may have merely illustrated the coincidence of an error of diet, with a relapse. The patient was ordered to resume a milk diet; and he did not again eat solid food until he was permitted to do so. The average duration of a relapse, seems to be about 16 days. But the duration may be as short as ten days, or as long as thirty-nine days. The apyrexial period may last only two days, or as long as thirty days. As a rule the relapse is milder than the primary case, and rarely proves fatal; but there are many exceptions to this rule, and fatal cases of relapse do occur.

1. Murchison on Continued fevers. p. 3-3-4.

In rare cases a second relapse or third attack takes place.
Post-mortem examination in fatal cases shows the recent affection of the intestinal glands, coexisting with the cicatrising ulcers of the first attack. But as those glands only are affected which escaped the first attack, the lesions are frequently not so extensive in the relapse, as in the primary attack; and for the same reason, they are often further removed from the ileo-caecal valve — or in the Colon. Trousseau alone among authorities denied that these were true relapses. He however, regarded the intestinal lesions, as the specific eruption of the fever.
Sex and age do not seem to have any influence in the causation of relapses; and their etiology has not been satisfactorily explained.
The explanations offered are:—
1st. The relapse may be caused by a fresh infection, from other cases of enteric fever in the same ward.

This seems improbable, as relapses occur in cases treated in isolated houses; and they also occur in cases removed from the original source of poisoning.

2nd It has been suggested, that the secondary infection is due to poisoning, by discharges and sloughs from the ulcers of the primary lesions, of those, or some of those intestinal glands, which had escaped poisoning and enlargement during the primary attack. It has been stated also that this poisoning takes place most frequently, where there has been constipation during the primary attack. But a priori it seems unlikely, that an acute specific disease like enteric fever, should afford no protection from a second attack, so soon after the patient had passed through the primary disease. And it is well known, that relapses are not confined to those cases in which there is constipation during the primary attack. Relapses have frequently followed cases, in which there was diarrhoea.

According to Liebermeister and others, relapses occur more frequently in cases subjected to the cold water treatment.

Complications and Sequelae.

Respiratory apparatus. Bronchitis may be an early or late complication. Bronchitis and hypostatic congestion of the lungs are frequent causes of death, in those cases which die in the first or second week of the disease. More commonly Bronchitis and Hypostatic Congestion are late complications, coming on in the fourth week, and prolonging the duration of the attack, if not even proving fatal. Bronchitis is not so common in enteric fever as in typhus. Sometimes spasmodic attacks of cough and dyspnoea are distressing symptoms in such cases.

Pneumonia is a common complication which does not usually occur before the third or fourth week; but it may occur early in the attack, and be mistaken for the primary disease[1]. The pneumonia may be catarrhal or lobar — usually catarrhal, when it

1. Pneumonia was developed early in the attack, in the case mentioned on page 121 of this Thesis: and was mistaken at first, for the primary disease.

it may end in minute abscesses, and rarely gangrene of the lung. It is more common than in typhus.

Pleurisy, is not uncommon, as a complication; and it occasionally terminates in empyaema or interlobular pleural abscess. It is more common in enteric fever than in typhus.

Those cases which terminate in empyaema, sometimes die from pulmonary phthisis; and, as is well known, tubercular deposits in the lungs and elsewhere, are not uncommon after enteric fever.

Laryngitis. Occasionally laryngitis is a serious complication. It presents four forms.

1. Ulceration of the mucous membrane of the larynx, "perichondritis typhosa" — Rokitansky. It may induce oedema of the glottis, necrosis and exfoliation of cartilages, permanent disease of the larynx, and abscesses in the neck. Such complications usually arise in cases marked by unusual prostration and long duration, which have been kept on too low a diet.

2. Acute oedema of the glottis may supervene apart from ulceration of the larynx.

in advanced stages of the disease.
3. Oedema of the glottis may occur in conjunction with erysipelas of the head and face. Oedema of the glottis may necessitate tracheotomy in enteric fever, or cause death by asphyxia.
In all the forms of laryngitis, abscesses may form in the submucous tissue.
Diptheria as a complication, has already been referred to.
General emphysema may occur, through the sloughing of an ulcer in the larynx.
And pneumothorax has been noted in rare cases to occur, from ulceration of a small bronchial abscess or gangrenous cavity in the lung.
Diseases of the circulatory system.
Haemorrhages. Epistaxis, and intestinal haemorrhage, have already been noted.
Haemorrhages also take place from the gums, kidneys, bladder, and other mucous surfaces.
Pyaemia; The formation of abscesses under the skin takes place not uncommonly during convalescence. Pus is deposited in the joints and internal organs in rare

instances.

Venous thrombosis, rarely occurs. Its most frequent situation is the left leg; but it may occur in the right or in both legs. In one case observed by the writer, where venous thrombosis occurred as a **sequela** after enteric fever; the thrombosis was in the left lower extremity. The patient made a good recovery, and resumed work as a telegraph messenger.

Arterial thrombosis. Spontaneous gangrene is comparatively rare. It may occur in the feet, ears, penis, labia, vagina, cornea, anterior wall of the abdomen. In one remarkable case recorded by Patry[1]; the gangrene spread from the left ear, to the forehead, eyelids and cheek. The cause of the gangrene in this case was arterial thrombosis of the external Carotid artery.

Diseases of the heart.

Valvular disease rarely results from enteric fever. The changes in the muscular fibres, have been already described.

1. Patry 1863. (Murchison 3rd Edn p. 560.)

Pericarditis, usually latent, is occasionally met with, and can only be detected by physical signs.

Diseases of the nervous system.

Meningitis is rare, but may result from tubercle, disease of the temporal bone, from suppuration in the internal ear, or pyaemia.[1]

In very rare instances it occurs independently of the above causes.

Mental imbecility and mania.

These mental conditions may result during convalescence, in protracted cases, from profound anaemia, and atrophy of the brain. They are not due to inflammation. Sometimes maniacal attacks occur under such circumstances, which readily yield to treatment. The mental fatuity and childishness of manner, (with sometimes delusions) take a longer time to disappear.

In the case already referred to, where muscular contracture occurred, mania and mental fatuity occurred during convalescence. And although

1. British Medical Journal. July 12th 1884. p. 67.

the mental faculties, continued to be impaired for a considerable time, after convalescence had become fairly established, they ultimately regained their normal condition.[1]

General Convulsions, have been mentioned. Paralysis, is noted as an occasional sequela. As a rule the various forms do not come on, until some weeks after the commencement of convalescence. They are therefore as a rule after paralyses; but during the attack, sudden hemiplegia may occur, probably from embolism. These paralyses are usually only temporary in duration, and ultimately pass away: their cause is obscure; but their analogy to post-diphtheritic paralyses is obvious. Nothnagel believes that they arise from the same cause, viz:- cell proliferation in the sheaths of the nerves, and between the fasciculi. Paraplegia is the most common form, but hemiplegia, strabismus, paralysis of the portio dura, motor paralyses of individual spinal nerves, such as the tibial or peroneal, and local anaesthesia, may all be consequent upon enteric fever.

1. Case mentioned on p. 162, of this thesis.

Such attacks, particularly in the legs, may lead to atrophy of muscles, and permanent distortion

Convulsions followed by temporary hemiplegia, temporary aphasia, and ptosis, are rare forms of post-febrile paralysis, associated with enteric fever. Neuralgia and hyperaesthesia are rare sequelae. The boy whose case is already mentioned, in connection with muscular contracture, suffered from Neuralgia of the back of the head and neck, during the earlier weeks of convalescence[1]. It was a distressing symptom, for which Belladonna Plasters, and hypodermic injections of morphia were tried; it ultimately quite disappeared.

Muscular tremors and Chorea, are also noted by Nothnagel as occasional sequelae.

Diseases of the organs of special sense.

Otorrhoea is not uncommon as a complication or sequela, especially in children. Suppuration of the internal ear may lead to meningitis, as already noted.

1. Case mentioned on p. 102, of this thesis.

Deafness occasionally occurs as a sequela, apart from otorrhoea.
Amaurosis, occurs occasionally during convalescence. It is usually incomplete. In Gillespie's case, there was total blindness for six weeks![1] There may be amblyopia.
Diseases of the digestive system.
Pharyngitis. Inflammatory affections of the pharynx may occur; often they are diphteritic. Or ulceration of the pharynx may take place. The dysphagia however, may be due to mere dryness of the throat, spasmodic closure due to nervous irritability, or in severe cases, from muscular paralysis. In some cases, where there is dysphagia, the pharynx is much injected.
Vomiting. Constant vomiting during convalescence is a nervous affection, according to Trousseau;[2] and can be stopped in most instances by giving solid food.
Diarrhoea. In cases in which the intestinal ulcers have become atonic

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1. Gillespie 1870. (Murchison p. 564.)
2. Trousseau 1861. Clin: Lect: "Dothienenteritis". Murchison p. 565.

they may give rise to exhausting diarrhoea after the cessation of the fever.

Dysentery, sometimes occurs as a complication of enteric fever: the symptoms during life, and the lesions after death, being present at the same time as those of enteric fever. Such a complication forms an element of difficulty in diagnosis.

Jaundice, is a complication not often met with. Remarkable cases, already referred to, are recorded by "Frerichs in his classical work on the Liver"[1]. Appearances indistinguishable, in their morbid anatomy, from those of Acute Yellow Atrophy of the Liver were observed by him in one interesting case of enteric fever associated with jaundice. In this case the liver was atrophied, with thin, sharp margins, and great diminution in thickness — the whole organ being soft and shrivelled. On section the cut surface was "pale brown, completely homogeneous, with no appearance of lobules." The bile ducts nowhere yielded coloured fluid, on pressure. The gall bladder contained only a little thin whitish "whey-like" fluid; and its walls were not bile-stained. "The glandular cells were in part disintegrated, some of them very pale, and several were filled with fat." Frequently in such cases, Leucine and Tyrosine, are found in the urine and other

1. Frerichs (Op. Cit.) p.p. 172; 173; 174; 215; 216.

excretions. But the cause of jaundice in enteric fever, varies, as it does in the case of typhus. Usually it occurs late in the disease; but in one of Frerich's cases, it occurred as early as the 5th day. Sometimes although rarely cases recover.

Peritonitis and perforation of the bowel, with escape of the intestinal contents, are the most important complications of enteric fever. Peritonitis may result from different causes; most commonly however, it results from perforation of the bowel. Sometimes it results from extension of the inflammatory process, by contiguity, from the mucous to the peritoneal covering of the bowel — without perforation. It may arise in this way before ulceration has commenced, even as early as the first week of the disease. Peritonitis may be excited by softened infarctions in the spleen; the bursting into the peritoneum of a softened mesenteric gland; an abscess in the wall of the urinary bladder, or in the ovary; the

the bursting inwards of a pseudo abscess in the sheath of the rectus muscle; or ulceration of the gall-bladder proceeding to perforation.[1]

In the great majority of cases peritonitis is due to perforation of the bowel; but it is impossible during life to distinguish, which of the above named causes has given rise to peritonitis.

Perforation of the bowel, with escape of the intestinal contents, into the peritoneal cavity, is by far the most important and fatal complication of enteric fever. According to Dr. Murchison, about one fifth of all the fatal cases which occur in this country, arise from perforation of the bowel. It seems to be met with about twice as frequently in fatal cases in this country as on the continent. Enteric fever moreover, is the only acute disease, with the exception of a few cases of tuberculosis and dysentery, in which perforation of the bowel takes place. It occurs

1. Brit. Med. Jour. July 12th. 1884. p. 67.

much more frequently in males than in females. This fact has been noted by all observers, who have attended to the subject.
Age does not seem to influence the occurrence of intestinal perforation in any marked degree. It is said that perforation is rare in children and old people; but Dr. Murchison has observed cases as early as 5 years of age; and as late as between 40 and 50 years of age. The case mentioned by the writer under "Anatomical Lesions", occurred at the age of 5 years[1]. The patient, a little boy, was said to have eaten some berries – nature unknown – a few hours before the fatal event; He had been running about, the same day that perforation occurred; and had only been medically attended, just prior to death: he was in a state of collapse when first seen. The post mortem examination, which was ordered by the coroner, showed typical lesions in the intestines, mostly in a state of deposition or enlargement; although ulceration had begun in a few patches. The seat and nature of the perforation has been already described. Another case occurred in

1. Case mentioned on page 87, of this thesis.

in a boy aged 10 years, F. R. 1st seen on 19th April 1892; T. 103.° Pains in stomach, and ochrey diarrhoea, Spleen enlarged, Pupils dilated, face pale. Tongue slightly furred, red at tip and edges; pulse 100: not particularly weak. Prostration not marked. No rose coloured spots, on abdomen, chest, back, limbs or face. The patient suffers from ichthyosis. Time of visit 5 pm. Ordered to bed at once; and to have a milk diet.
20th Visit morning. very thirsty, T. 102.8. p. 98. feels better. Pain in abdomen still complained of.
Evening visit not so well. T. 104. 2 ochrey motions since morning. very thirsty. circumscribed pink flush on cheek. Abdomen distended. Ordered Powders containing Dover's Powders and Bismuth: and linseed meal poultrices.
21st Prostration more marked. Abdominal pain severe Urine scanty: sleep disturbed during night; but mind clear; Evening; vomiting distresses the patient, & he has had several ochrey motions. The abdominal pain still unrelieved. Distension has increased so as to interfere with breathing. The urine is very scanty and passed with difficulty. Turpentine stupes, ordered in addition to the linseed poultices.
22nd Patients mind clear, perspires at intervals, no diarrhoea. Prostration more marked. Evening no change.
23rd The abdomen is much distended: there is occasional

vomiting of bilious looking fluid. T. 103°. P. 108.
Tongue moist, not dry and brown – slightly furred
Had a restless night but no delirium. Mind quite clear: answers promptly and in an articulate manner:
Visit 7 p. m. Great abdominal pain. Abdomen immovable: cannot pass water: the vomiting has much increased.
Visit at 9 pm. The patient presents all the symptoms of intestinal perforation. A large dose of solid Opium given, and linseed poultices frequently applied to the abdomen. Stercoraceous vomiting continues almost incessantly; features pinched; cheeks, and particularly nose quite cold to the touch; hands and feet cold; clammy cold perspiration on forehead. Great abdominal pain: legs drawn up; and abdomen hard and immovable.
Breathing thoracic. Mind perfectly clear; and he enquires anxiously about himself. T. 104.8°
Pulse feeble, 140 (?). Visit at 12 p.m., and remained till 2 p.m., at which time the face became a little warmer. Pulse very weak, and symptoms of perforation unmistakable: died at 12.30 a.m. on the 24th April, during time of visit.
The mind remained clear to the last.
This patient had been ill, for about fourteen days, as far as could be ascertained, before medical advice was sought. An apercient

had been given, early in the disease under the impression that the illness resulted from gastric derangement, or an ordinary cold; and the patient had been able to get about, until medical advice was called for, 14 days or more after the invasion of the disease -

The circumstances which favour the occurrence of perforation, are indigestable food; distension of the ulcerated bowel with faeces or flatulence. Sudden movements on the part of the patient, and the strain of vomiting. The administration of an enema, has sometimes been followed by intestinal perforation; as well as the injudicious administration of a purgative. Perforation is most likely to take place during the third, fourth or fifth week of the disease; but it may take place as early as the eighth, or as late as the 66th, 72nd, 76th or even 110th day (Morin.) The liability to perforation, long after convalescence has commenced is the important point to remember.

Those cases in which perforation occurs, have often been marked by abdominal symptoms. Diarrhoea is usually severe, and there is much abdominal pain. And often in cases which terminate in perforation intestinal haemorrhage has occurred during the attack. But cases which terminate in perforation have not invariably been severe. Sometimes intestinal perforation occurs suddenly in the mildest of cases, without any warning. Often it occurs even in cases where there has been no diarrhoea or abdominal pain, and in which the prostration has been so little marked, that the patient has been pursuing his ordinary avocations, at the time he was seized with symptoms of perforation of the bowel. It has indeed been said that these latent cases of enteric fever are more liable to be complicated by perforation, than other cases. But this is not so; perforation is an event to be feared, in any case of enteric fever. The symptoms

which indicate that perforation has occurred are the following: —

The sudden supervention of collapse — with or without rigors; great pain and tenderness of the abdomen, which is usually much distended and tympanitic.

The face is pale and pinched, and expressive of anxiety or suffering. The decubitus is dorsal, with the legs drawn up. The pulse is rapid, thready or imperceptible; and the temperature rises. The breathing is thoracic. There is often vomiting preceding the other symptoms by a few days; and there may be increase of diarrhoea. Sometimes the diarrhoea is preceded or accompanied by intestinal haemorrhage. Great thirst is complained of, and there may be suppression of urine. Soon the prostration becomes extreme, the extremities cold, and the face covered with large beads of perspiration. The patient gradually sinks; and the mind remains clear up to the last. Such unmasked cases are unmistakable But the symptoms may be obscure; and

and probably many cases of perforation are not diagnosed. There may be neither abdominal pain nor rigors; and the chief indications of the complication may then consist of, sudden increase of prostration; rise of pulse and temperature, and the distended motionless state of the abdomen. Sometimes the advent of perforation is latent on account of delirium, and the increase of prostration may then be attributed to the severity of the disease; the ordinary symptoms being absent. The period at which death takes place after perforation, varies from a few hours, to several days. In very rare cases as long as 15, 21, or even 28 days, have been recorded as elapsing between the first symptoms of perforation and the fatal event. But death from intestinal perforation most frequently occurs within 48 hours, after its occurrence; and life is rarely prolonged beyond two days, after the first appearance of the symptoms of intestinal perforation.

By many observers, intestinal

intestinal perforation is regarded as an invariably fatal complication of enteric fever. But it seems, that in certain rare instances, recovery may take place. The edges of the aperture may become glued by inflammatory exudation to the walls of the abdomen, to the mesentery, or to an adjacent coil of intestine, in this way preventing the extravasation of any considerable portion of the intestinal contents. Or even in cases, where escape of intestinal contents into the peritoneal cavity has taken place, the escaped contents, may become limited by adhesions, and lead to the formation of a circumscribed peritoneal abscess, which may discharge itself into the bowel, or open externally as in case LXXV of Dr. Murchison's already referred to. In such cases, a long interval may elapse, between the time of perforation, and the fatal event, which may be due to septicaemia; but recovery in rare cases may occur.

That the edges of the aperture, may become glued by inflammatory exudation, to the abdominal wall, is proved by the following instance, which was related to the writer, by Dr. Marsh, late of Clapham. During an attack of enteric fever, a boy aged 12 years, developed symptoms of an abscess in the vicinity of the umbilicus. The abscess discharged after having been poulticed; but it continued to discharge until the patient became much prostrated. A consultation was held, at which, it was proposed to administer a preparation of Iron, and observe whether the discharges from the umbilical abscess became blackened; in order to prove whether or not, there was a communication between the bowel and the external aperture. After a certain period, a black inky-looking fluid oozed from the umbilical opening. The patient died; but unfortunately there was no post-mortem.

Diseases of the urinary organs

Disease of the kidneys is a serious

complication. Haematuria has been already mentioned. Catarrh of the bladder, it is said may be troublesome, where retention of urine during the fever, has been neglected.

Generative System.

Menstruation may occur during enteric fever. It may be excessive. Pregnancy does not confer immunity from enteric fever. Abortion or miscarriage usually but not invariably occurs in consequence. And many pregnant women, attacked by enteric fever recover.

Diseases of the integumentary and osseous systems and of Connective tissues.

Erysipelas is a rare and unfavourable complication. It is sometimes associated with otorrhoea; and is often fatal.

Anasarca. Localised oedema, from venous thrombosis has been already mentioned. But swelling of both legs may occur, from weakness of the circulation in protracted cases. And in certain cases characterised by marked debility, or an adynamic state of the constitution the anasarca may be general, although

unaccompanied by albuminuria. In such cases, it comes on during the 3rd or 4th weeks of the fever, or during convalescence. It may be preceded by severe bronchitis or copious perspiration, lasts for two or three weeks, and then disappears, without any further inconvenience, than that of retarding convalescence. General or partial anasarca is not uncommon in children as a sequela of enteric fever. As a complication it may occur as early as the fifth day.

Bedsores are more frequently met with in enteric fever than in typhus, on account of the more prolonged duration of the disease, and the greater emaciation. They occur over the sacrum, trochanters, on the elbows heels, occiput &c, — situations similar to those in which they occur in other protracted diseases.

Noma, or Cancrum oris, is mentioned as a very rare complication of enteric fever, usually fatal, and occurring only in children.

Blisters applied during enteric fever are slow in healing, and may degenerate into

unhealthy sores.

Necrosis may occur as a sequela. It has been met with in the tibia, lower jaw, temporal bone and upper third of the femur. It is more common after enteric fever than after typhus. An interesting case was recorded, - in which "Necrosis of the upper jaw on the left side, supervened, five weeks after the apparent onset of enteric fever", - by Dr. Angel Money, at the Pathological Society of London on Nov.r 20.th 1883.[1] This case had the further peculiarity, of illustrating a case of enteric fever, supervening upon rheumatic fever.

Periostitis, may occur as a sequela of enteric fever. Sir James Paget[2] has called attention to the subject, and many other observers have since recorded cases. It is most frequently seen on the tibia; but may affect the femur, ulna, ribs, temporal and other bones. According to Sir James Paget, "it occurs at an advanced stage of the convalescence when the temperature has become normal, and the patient is regarded as free from fever".

1. British Medical Journal Nov. 24th 1883. p.p. 1019 & 1020.
2. St. Bartholomew's Hospital Reports. Vol. xii. 1876. Brit. Med. Jour. May 9th 1885. p. 939.

Dr. I. O. Affleck, relates cases to show that periostitis, may be no less a complication, than a sequela of enteric fever.[1] He attributes its causation, to lowered vitality of the osseous tissues, as the result of a severe or prolonged attack of enteric fever. Dr. Haywood of Liverpool, and Dr. E. Jackson of Manchester have among many others, given recent contributions to this interesting subject.[2]

Herpes of the lips, is sometimes met with in enteric fever; and large bullae have been observed to occur on various parts of the body.

Parotid and other buboes are rarely met with. They seem to be of unfavourable prognostic significance.

Marasmus. Most practitioners have met with cases, fortunately not frequently, in which, after an attack of enteric fever, usually severe, the patient does not get on during convalescence. Flesh and strength are not regained

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1. Brit. Med. Jour. May 9th 1885. p 939.

2. Brit. Med. Jour. Jan.y 3rd and Feb.y 28th 1885.

and the patient is anaemic, although the appetite may be good and the temperature normal, or even subnormal.
On examining the patient, the lungs seem to be healthy, and no local disease can be discovered. Still the wasting slowly makes progress, and the food does not seem to be assimilated. All kinds of artificially digested foods, and dietetic preparations, may have been administered. Cod liver oil does no good whatever; and malt extract is equally unsuccessful. The emaciation becomes extreme, and ultimately perhaps six months or a year after convalescence has set in, the patient dies of inanition. It seems that after death in these cases, the mesenteric glands are as a rule found to be shrivelled and atrophied; but not in all cases. Sometimes in such cases dyspeptic symptoms exist during life — the appetite is poor, and the patient may be much troubled by flatulent distension and diarrhoea.

It seems that occasionally the patient may survive for years, in this state of marasmus. The liability to sudden death in enteric fever, must be kept in remembrance.

It may occur during the height of the fever; but more frequently occurs during early convalescence. The cause of death is frequently softening of the heart, thrombosis or embolism of the pulmonary artery; but frequently death occurs suddenly where no such cause can be found. It has then been accounted for, by such probable causes, as reflex spasm, ischaemia of the brain, and pneumatosis of the blood. The fatal event is sometimes preceded by convulsions; and mental emotion or muscular exertion, seems in some cases, to have caused the accident. It may occur even after mild cases, therefore it is imperative, that patients should be prohibited from undue bodily and mental exertion during early convalescence after enteric fever.

The coexistence of enteric fever, with other acute specific diseases, has already been mentioned under etiology.

Patients suffering from enteric fever, may contract scarlatina, and cases of scarlatina may merge into enteric fever, when probably the two poisons enter the body at the same time. Enteric fever and measles may coexist; also a patient suffering from enteric fever, has been known to contract whooping-cough. The relationship of diptheria to enteric fever has been stated. A patient may suffer simultaneously from both typhus and enteric fever, or may contract typhus, while suffering from enteric fever[1]. Enteric fever may also be associated with rheumatism. Dr. Finlay read notes of five cases, in which enteric fever, followed upon an attack of acute rheumatism. In one of his cases it seems that the two diseases coexisted; but that, at the outset the symptoms of typhoid fever were so slight, as to be completely overshadowed by the rheumatic manifestations.

The discussion which followed, at the Clinical Society of London, brought out many other interesting points relative to the relationships of these two diseases.[2]

1. Murchison on Continued Fevers. 3rd Edition. p. 5-89.
2. British Medical Journal. April 5th 1884 p. 666.

The varieties of enteric must be briefly considered. Different writers have described enteric fever under a number of subdivisions such as; adynamic or low nervous fever, in which protracted pyrexia, and great prostration are prominent features; the abdominal form in which abdominal symptoms are prominent; the thoracic form, in which chest symptoms preponderate; and the haemorrhagic form already referred to. Also the ague like form prevalent in malarious districts, and ushered in by prolonged rigors.

The ataxic form — the so called 'brain-fever' is a further subdivision of adynamic or low nervous fever, in which delirium and the typhoid state are the distinguishing features.

However interesting it may be to regard the varied groupings of the protean manifestations of enteric fever, in this systematic manner; there are certain groups of cases considered as a whole, which are sufficiently distinct to form varieties, and it is practically useful to be well acquainted with each variety.

1. The abortive form. This variety probably includes many cases returned as febricula

The attack begins, like an ordinary attack of enteric fever. The temperature may reach 104° or even 105° on the fourth or fifth day of the disease. The pulse is not much accelerated, ranging as a rule from 70 to 80 beats per minute. There may be a good deal of headache, restlessness and general malaise. The tongue is furred, with red tip and edges. Sometimes there are vomiting and diarrhoea; but more frequently the bowels are constipated. The fever runs an irregular course, the stage of enlargement of the mesenteric glands does not proceed to the stage of ulceration; but absorption of the morbid deposit takes place, without ulceration. About the 8th or 10th day, a morning remission takes place, and lysis having thus been initiated defervescence follows, the process being complete by the middle or end of the second week. But the evening temperature, may remain slightly febrile, for a few days or even a week, during which time the fever is truly intermittent. The above is a clinical description of an average abortive case. Some erratic individual cases, have subsided on the 4th or

or 8th or even on the 5th day. According to Dr. Murchison even these irregular forms, as a rule subside by lysis. The proofs that abortive cases are really enteric fever are, that occasionally characteristic spots appear on or about the 7th day, and that abortive cases often occur in the same house as typical cases. This variety corresponds to the 'forme muqueuse' of French writers.

2. The insidious or latent form. In this variety, there may be little acceleration of the pulse, and all the symptoms are mild; still the temperature rises, and the disease has a duration of three or four weeks, and runs the ordinary course. Sometimes there are irregular chills, alternating with heat and flushing, slight headache, lassitude, loss of appetite, and disturbed sleep constituting the early symptoms. Sometimes there is diarrhoea, but usually the bowels are constipated. Bronchial catarrh may be the first and most prominent symptom; and the patient is thought to be suffering merely from a 'feverish cold'. Or again, it may begin

with nausea and vomiting, and a red tongue; and the patient may be then thought to be suffering from a bilious attack, or other gastric derangement. But in whichever of these ways, the early symptoms may manifest themselves, the patient sometimes passes through the attack, without much prostration or constitutional disturbance, and the illness may then be diagnosed as simple continued fever. But in certain of these cases, alarming symptoms of intestinal perforation sometimes suddenly manifest themselves, and the patient dies in a few hours. This variety has been called 'Typhus ambulatorius' on account of the patient walking about throughout the entire illness, or up to a few hours of the fatal event. The fact of the patient moving about in this way, would favour rupture of the denuded peritoneum, forming the base of the intestinal ulcers. In other cases acute maniacal delirium sets in suddenly, and a profuse haemorrhage from the

bowel takes place, which may prove fatal.
3. Gastric or bilious fever, has no separate existence as a species of fever.
Except in rare cases, where the temperature is elevated on account of gastric or bilious derangement apart from any specific cause, the cases of so called gastric fever all correspond to one or other of the varieties of enteric fever — often the abortive or latent variety.
The proofs of this fact are, 1st. That gastric fevers often occur in the same house as enteric fever; 2nd That in many cases of gastric fever, rose coloured spots appear; 3rd That many cases correspond with, what some Physicians would call gastric fever, for the first fortnight, but ultimately present the ordinary symptoms of enteric fever; 4th That in the course of gastric fever, the typhoid state may become developed, and the case prove fatal, the specific lesions of enteric fever being found after death in such cases; 5th That a case may run the course of gastric fever, with retching

and other gastric symptoms, and be followed by a relapse of typical enteric fever.

4. The acute form is that in which the disease sets in suddenly with great violence. From the commencement, or within a day or two, acute delirium exists, with or without diarrhoea. Pulmonary congestion appears early, and often spreads with great rapidity. The disease may prove fatal in the first, or early in the second week, before ulceration of the bowels has commenced.

5. Infantile remittent fever. According to Dr. Murchison, idiopathic remittent fever in children is almost invariably enteric; although it has been believed to be a distinct disease called, Infantile Remittent Fever, Worm Fever, Infantile Hectic, and Infantile gastric fever. Apart from those febrile conditions, often taking on a remittent type, common in children; and due to gastro intestinal disturbance;

and subsiding under proper treatment in the course of a week; all remittent fevers in children, in this country are probably enteric. Children are particularly prone to the disease; and the symptoms are modified, as already described, by the age of the patient. Besides, in cases of true enteric fever in children, with lenticular rose coloured spots, the temperature tends to be markedly remittent, and is benefited by Quinine. But the fact that they are benefited by Quinine, does not prove that the nature of the disease, is similar to that of malarial remittent fever — a disease to which children are no doubt subject, as well as adults, in malarious districts.

6. Enteric fever in aged people.

In aged people, the disease is usually very insidious in its onset, and the disease is protracted. The temperature does not run so high as in young people; and tends to be subnormal during convalescence.

The type of fever is essentially adynamic, and rose spots, acute delirium, and diarrhoea are not prominent symptoms. Collapse is not uncommon, and debility and tremors are frequently met with.

The diagnosis of enteric fever, is more or less difficult, according to the appearance, or non-appearance of the eruption, and the prominence or non-prominence of abdominal symptoms. During the first week of enteric fever, before the eruption has appeared, it may be impossible to positively diagnose the disease. All that can be arrived at, being a suspicion of its existence. The following symptoms, would make one strongly suspicious:— a febrile condition, increasing each evening, especially should this be associated with diarrhoea, vomiting, enlargement of the spleen or epistaxis. But when the disease has lasted a week or longer; and the characteristic

rose coloured lenticular spots appear, in successive crops, in the manner described, a positive diagnosis of enteric fever can be made, whatever be the nature of the other symptoms, and however few the spots, that are present at one time. But in those cases in which the spots do not appear, a positive diagnosis can still be made, when pyrexia has lasted over a week, is remittent in type, and associated with diarrhoea, ochrey stools, tympanites, abdominal pain, enlargement of the spleen; or epistaxis should it occur, further clinching the diagnosis.

If both the eruption and abdominal symptoms be absent, the diagnosis of enteric fever, can only be arrived at, by a process of exclusion, after carefully comparing the symptoms, with those of the principal diseases, with which it may be confounded. Dr. Murchison gives the following practical rule:-

"A fever which in this country (agnish districts excepted) persists beyond 7 days, and is

unattended by cutaneous eruption, or by signs of local disease, in the head, chest or elsewhere, is in all probability enteric fever, even although there be no symptoms of intestinal lesion. Almost the only source of fallacy is latent tuberculosis."

The following are the diseases from which typhoid fever must be distinguished:—

1. From Typhus, the diagnosis is easy; but the 'typhoid state', may be as distinctly developed in enteric, as in typhus fever. The eruptions are the great distinguishing features: and although that of enteric fever may be absent, the eruption of typhus is very rarely so. The onset is more sudden in Typhus than in enteric fever. The type of fever is more remittent in enteric fever than in Typhus; also the duration of enteric fever is longer than that of typhus. In typhus, defervescence is by crisis; in enteric fever lysis is the rule. Diarrhoea alone, does not distinguish the two diseases, for typhus may be complicated by diarrhoea, and in enteric fever, the bowels may be

constipated. But if a case in which pyrexia has lasted over a week, present symptoms of diarrhoea, with ochrey stools, abdominal pain, and tympanites; especially if epistaxis or intestinal haemorrhage be superadded, the disease is probably enteric fever.

The diagnosis will also be assisted by the tongue, the state of the pupil, the pink circumscribed flush on the cheek, contrasting with the dull suffusion of the face in typhus. And the circumstances under which the disease was contracted.

The symptoms differ so distinctly from those of relapsing fever, that the two diseases should not be confounded. But enteric fever with relapse, is sometimes spoken of as relapsing fever.

The diagnosis from remittent fever is often extremely difficult, particularly in malarious districts. In both, the spleen is enlarged; in both, the temperature is remittent in type; and the temperature in enteric fever, may even be intermittent at first, in malarious districts. Vomiting and diarrhoea are common to both, and

both may develop cerebral symptoms and the typhoid state. So that the presence of the eruption, is probably the only basis, on which a positive diagnosis of enteric fever can be grounded.

Enteric should not be mistaken for scarlet fever, even in those cases in which the lenticular spots are preceded by a scarlet rash, accompanied by a little sore throat. The throat is not really scarlatinal, in such cases, but only dry. Besides, the tongue is different; and the gradual rise of temperature in enteric fever, is very different from the abrupt invasion of scarlet fever. The prodromid erythematous rash of enteric fever further, does not appear, until the fourth or fifth day. The diagnosis of enteric fever, with copious eruption, from variola, is based upon the different characters and distribution and time of appearance of the two eruptions, among other distinctive characters. In enteric fever, the spots are never hard, gritty or acuminated. They rarely appear before

the 4th day, and are practically absent from the face. Besides the rachalgia, accompanying the onset of variola, is very characteristic.

Pyaemia. The diagnosis of enteric fever from pyaemia is difficult; and in some cases, a positive differential diagnosis may be impossible. When the eruption of enteric fever appears, the diagnosis is clear. But even when the spots do not appear, the following symptoms would point to pyaemia. The occurrence of rigors and profuse perspirations, an icteric tint of the skin. The circumstances under which the disease occurs, would also guide the diagnosis. The presence of abdominal symptoms, would not establish a diagnosis of enteric fever; because in puerperal pyaemia, there may be diarrhoea, and great distension of the abdomen, with variations of the temperature, closely simulating those of typhoid fever. Cerebral symptoms, may be marked in puerperal fever, and the typhoid state may be developed. Within the knowledge

of the writer, an undoubted case of puerperal pyaemia, (marked by the wildest delirium and delusions, ultimately merging into the typhoid state, and proving fatal,) was held to be puerperal mania, by an elderly physician, called into consultation. All the symptoms, including temperature, abdominal, cerebral and mental symptoms, and the development of the typhoid state, were of a very typhoid-fever like nature; but the circumstances under which the disease was contracted, the smell of the discharges, the arrested secretion of milk, the severe and repeated rigors marking the invasion of the disease, and the much more marked variations of temperature than occur in enteric fever, left no doubt but that the case died of puerperal fever. There were also in the above case, profuse perspirations; and an icteric tint developed itself, in a complexion which in health, was remarkably fair. It must be remembered however, that enteric fever, occurring during the puerperal state, may be

followed by pyaemia.

Influenza, especially when epidemic may closely simulate enteric fever. In both, there may be great prostration, occasional perspirations, sleeplessness, delirium, and even the development of the typhoid state. Bronchial catarrh and pulmonary complications, particularly pneumonia and pleuro-pneumonia, are common to both. In both, there may be deafness, discharges from the ears and giddiness. And in some cases of influenza there may be diarrhoea, dry tongue which may be even red and glazed; or there may be epistaxis. But in influenza, the onset is more sudden than in enteric fever, and the course of the temperature is different. In influenza the prostration is marked from the first; and there are often localised muscular pains, in the back, and at the nape of the neck, accompanying the onset of the disease in influenza: sometimes these muscular pains are located in the muscles of the chest wall, (simulating pneumonia or pleurisy,) or in the muscles of the abdomen, or in those of one or other of the extremities. Influenza occurs under different circumstances, and does not

run the protracted course of enteric fever.
The presence of the eruption, in enteric fever, would decide the case.

Tuberculosis. The various manifestations of tubercle, are conditions most difficult to diagnose from enteric fever. Tubercular meningitis, may sometimes present symptoms, so closely resembling those of enteric fever, that it may be impossible to distinguish the two diseases. Fever of remittent type, headache, delirium, vomiting, inequality of the pupils, cerebral maculae, rolling the head from side to side, the hydrocephalic cry and even partial paralysis, may be met with in both diseases.
The rose coloured spots, unfortunately, are most frequently absent, at the age when the difficulty of diagnosis is most liable to present itself.
The following are the points to be kept in mind, in coming to a conclusion.
The occurrence of previous cases of enteric fever in the same house would favour fever; whereas, the occurrence of tubercular disease, in other members of the same family, would favour acute tuberculosis. The vomiting

at the outset, is usually more urgent in meningitis than in enteric fever; and the tongue is rarely dry and brown, as it usually is in cases of enteric fever characterised by severe cerebral symptoms. The headache is more intense in meningitis than in enteric fever, persists after delirium has commenced; and is often associated with intolerance of light and sound. The temperature in meningitis does not run the regular course characteristic of enteric fever. In meningitis the temperature is liable to sudden falls, and for several days, in meningitis, the temperature may be normal, while other symptoms are getting worse, and while the pulse is rising. Towards the end in meningitis, the temperature may fall, while the pulse is rising.

The abdomen is distended and tympanitic, and there is often abdominal pain, in enteric fever; whereas in meningitis, the abdomen is usually retracted and

1. In enteric fever headache as a rule ceases after delirium has commenced.

painless.
Enlargement of the spleen, diarrhoea and epistaxis, as well as intestinal haemorrhage, are common in enteric fever; whereas they are rare in meningitis, in which constipation is the rule — or if there be diarrhoea, the stools are not ochrey, as in enteric fever. Inequality of the pupils; irregularity of respiration, partial paralysis, and rolling of the head from side to side, common in meningitis, are the exception as symptoms in enteric fever.
In meningitis Children may utter from time to time the hydrocephalic cry.
Also in meningitis, the patient is more irritable, and offers more resistance to examination.
Cöhnheim has shown, that tubercle may often be discovered in the choroid, on opthalmoscopic examination, in cases of tubercular meningitis.
Tubercular Peritonitis, sometimes closely simulates enteric fever.
The points of difference are, that in

Tubercular peritonitis, the abdomen is usually retracted;[1] whereas in enteric fever, the abdomen is distended, tympanitic and painful, as a rule.
Also that in tubercular peritonitis, the temperature usually becomes normal after a time; whereas the usual course of the temperature in enteric fever, would indicate the nature of the disease.
But the resemblance between the two diseases may be very close at first, both presenting pyrexia, occasional perspirations, vomiting, abdominal pain, hectic flush, diarrhoea, great prostration and emaciation, signs of bronchial catarrh and delirium.
Acute tuberculosis of the lungs, may present symptoms, extremely like those of enteric fever. Fever of remittent type, great emaciation and increasing prostration, perspirations, circumscribed hectic flush on the cheeks, dry tongue, delirium, stupor, dyspnoea and bronchitic rales, may be present in both cases; and while diarrhoea may

1. The writer recently saw a case of tubercular peritonitis, in which the abdomen was much distended.

be absent in enteric fever, in acute tuberculosis of the lungs, there may be diarrhoea, even with ochrey stools, when tubercular ulceration exists in the bowels; although, in pulmonary tuberculosis, the abdomen is usually retracted, and not distended and tympanitic as in enteric fever. Of course the presence of rose spots and splenic enlargement would be evidence of fever, although their absence does not prove the contrary.

The family history, and the circumstances under which the disease was contracted, are here, again valuable aids to diagnosis. But it must be remembered that acute tuberculosis of the lungs, sometimes follows upon enteric fever; and that cases occur, in which it would be impossible to differentiate the two diseases, from the physical signs in the chest, alone. An ophthalmoscopic examination also should be made in all doubtful cases, with a view to ascertaining the presence or absence of tubercles of the choroid.

Latent tubercle is another condition sometimes difficult to differentiate from enteric fever. Sometimes a patient has pyrexia with nocturnal exacerbations, increasing prostration, emaciation, perspirations and loss of strength; and no cause can be found, on the most careful physical examination, to explain the patient's condition. These symptoms may appear, many weeks before a sufficient development of tubercle, in the various organs has taken place, to give rise to physical signs; and it then may be thought that the patient is suffering from a low fever. Such cases can only be cleared up by exclusion. Sometimes the duration of the fever, being prolonged beyond a month, makes one suspect tubercle, the symptoms and signs of which, at a more advanced period become unmistakable.

When delirium sets in suddenly in enteric fever; or in cases which were up to the time of the appearance of the delirium comparatively mild, the case may be mistaken for insanity. The pyrexia and

other characteristic symptoms of enteric fever, will clear up the case.

Pneumonia, with typhoid symptoms, may be mistaken for enteric fever. Also pneumonia complicated by marked gastro-intestinal disturbance, as it often is, in children, may be mistaken for enteric fever.

In adults, the mistake may be made when pneumonia is complicated by dysentery. When enteric fever is complicated by pneumonia, the diagnosis is easy, when it occurs — as it usually does — late in the disease. But in those rare cases in which pneumonia occurs within the first week, an error in diagnosis may be committed, with respect to the question of the primary or secondary nature of the pneumonia.[1]

Gastro-enteritis. The diagnosis of enteric fever, has to be made, from all those derangements of the stomach and bowels, accompanied by fever, but where the fever is secondary, instead of primary. The abdominal symptoms

1. In the case mentioned on p. 121 of this thesis, pneumonia was an early symptom, and obscured the diagnosis.

of enteric fever may be mistaken for, irritant-poisoning, gastro-enteritis, colitis, typhlitis, or gastric irritation; and any of the above mentioned conditions may be mistaken for enteric fever.

In adults, the diagnosis is easy, from the course of the temperature, the greater loss of strength, and emaciation, headache delirium, the character of the stools, epistaxis, enlargement of the spleen, the presence of the eruption, and other symptoms of enteric fever.

But in children under the age of five years, slight causes of disturbance produce more profound constitutional changes, elevation of temperature, delirium &c; so that the diagnosis may be more difficult. But a careful observation of the symptoms, will often clear up the case, the diagnosis of which would be positive, should rose coloured spots appear, accompanied by enlargement of the spleen.

The mistake of confounding enteric fever, with an ordinary bilious attack

has already been mentioned. The value of the thermometer, as a means of diagnosis, is here evident.

Trichiniasis, excites pyrexia, vomiting, and diarrhoea, followed by typhoid symptoms. But the presence in trichiniasis of severe muscular pains, oedema of the eyelids, or of the whole body; and the absence of rose coloured spots, enlarged spleen and epistaxis, should make a mistaken diagnosis avoidable.

The prognosis of enteric fever is important. In all cases, even the mildest, it is evident from what has already been written, that the prognosis must be guarded. The patient cannot be said, to be out of danger, until convalescence has quite commenced; and the danger of perforation of the bowels occurring even a considerable time after convalescence has set in, is well known. It is well to bear in mind the conditions which influence the prognosis, such as rate of mortality, and other circumstances. In this country, the rate of mortality

in enteric fever, may be roughly stated as 1 in 5 or 6 — about 15 to 17%. This normal mortality becomes an important factor, when statistical methods are used in endeavouring to establish the efficacy of particular special methods of treating enteric fever; and more especially that particular form of antipyretic treatment called the cold bath treatment of enteric fever. In some foreign hospitals, the mortality has been enormously higher than 15%. Paris presents a mortality of 32% in a small number of cases!

Also, in the London Fever Hospital, the mortality varies widely in different years.[2]

Age does not seem to influence the prognosis in any marked degree, except in the case of aged persons: the age being over 50 years has an unfavourable influence on the prognosis.

Sex. The prognosis is rather more unfavourable in females; the mortality being about 1% higher in females than in males, and this difference occurs apart from the influence of child bearing.

1. Murchison on Continued Fevers. 3rd Edition p. 605. Table IV.
2. Murchison. (Ibid.) p. 604.

During the child bearing period indeed, the male mortality is greater than the female: also after the age of 40 Years, the male mortality is greater than the female. The mortality is greatest among female children, up to the age of 15 years – when the excess of female over male mortality is most marked. Months and seasons do not seem to have much influence on the prognosis. The mortality is sometimes less in Autumn, when the disease is most prevalent; and higher in cold weather, than during the Summer months; but these results are liable to variation in different years. Station of life. Destitution and poverty are not unfavourable, as in the case of typhus. The very poor and destitute do not die in greater numbers from enteric fever, than the rich. And in private practice, it seems that the better classes may suffer a higher mortality than the very poor.

Recent residence in an infected locality,

increases the mortality of enteric fever. It seems that after prolonged residence in a locality, where typhoid fever is endemic, the body acquires a power of resistance, whereby it suffers immunity from the poison. Place of birth, and race, have little influence on prognosis; but it is remarkable that in the London Fever Hospital statistics, the Irish presented a very low rate of mortality.[1] Family constitution, and the intensity of the poison are two factors, which seem to influence the mortality. Many members of one family die, and of another recover, even when they are taken with enteric fever, in different places, and at long intervals of time; whereas under similar circumstances many members of other families recover. But there must be another factor influencing the mortality in many epidemics, and isolated cases. In certain houses, the mortality is high amongst those attacked, although they are of different families. Further, it is well known, that different epidemics differ in

1. Murchison on Continued fevers. 3rd Edition p. 610.

their rate of mortality, according to the constitution of the disease, or epidemic type, as it is sometimes called. In some epidemics, the virulence of the poison seems to be greater than in others.

Debility from previous diseases or other causes, does not influence the prognosis unfavourably, as in typhus. On the contrary it is a common observation, that the strong and robust, more frequently die of enteric fever than the feeble. The following bodily conditions and habits influence the mortality. The prognosis is unfavourable, in very fat people, and amongst those of large muscular development; amongst those who have been intemperate in their habits; or who have suffered from gout and kidney disease.

The following symptoms, and other points, must be kept in mind as influencing the prognosis: —

1. Severe nervous symptoms, and great prostration are of unfavourable omen, in enteric fever, as in typhus.
2. The mode of invasion must not influence the prognosis: the mildest cases may

terminate suddenly in death. And cases mild at the outset, may frequently develop into severe cases; and those which have been characterised by severe symptoms, at the outset, may run a mild course.

3. The influence of the thermometer in guiding the prognosis is important. Those cases are severe, in which the morning remissions are not well marked. A temperature of 105° at any time during the course of the fever is unfavourable. A sudden rise or an irregular course of the temperature is a bad sign. And a sudden fall may indicate intestinal haemorrhage, or other unfavourable complication.

4. The pulse does not influence the prognosis so much as in typhus, where a sudden fall is favourable; because the frequency of the pulse, in enteric fever may vary greatly before the cessation of the fever.

5. Perspirations are not necessarily favourable or critical, in enteric fever.

6. The copiousness of the eruption,

has no relation to the danger of the case. Patients often die of enteric fever, in whom the eruption is absent.

7. Many cases of enteric fever, die, in which the tongue has not been dry and brown at any time during the disease.

8. Vomiting, early in the attack is not unfavourable: after the 14th day, it may be indicative of the onset of peritonitis.

9. Diarrhoea is unfavourable, in proportion to its frequency and urgency, also in proportion to its duration.

10. Abdominal pain, and marked tympanites are unfavourable.

11. Copious haemorrhage may induce fatal collapse, or precede perforation: slight haemorrhage is in itself, not of much importance, but may be followed by severe haemorrhage.

12. When peritonitis occurs, the case is almost hopeless, as in the great majority of cases, it is due to perforation of the bowels. But rare cases are on record, in which, patients presenting

all the symptoms of perforation have recovered.

13. Muscular tremors when severe and protracted, indicate deep and rapid ulceration of the bowels: especially when at the time of their occurrence the mind is clear.

14. Sudden collapse is almost invariably fatal, most frequently it results from perforation of the bowels, or copious intestinal haemorrhage — events, which may occur, although abdominal pain may be absent.

15. When coma, and congestion of the lungs, come on during the first week of the disease, death in such cases often takes place, before the 14th day.

16. Epistaxis, although usually unimportant in its relationship to prognosis, when profuse may lead to a fatal result.

17. Pregnancy does not add greatly to the danger; but in such cases miscarridge or abortion usually occurs.

18. A temporary remission of temperature during the second or third week, followed by a return of the pyrexia, and aggravation of the other symptoms, is often followed by a fatal result.

19. Even after convalescence seems to be fairly established, the patient is not out of danger. A relapse may occur; or the process of cicatrisation may not proceed satisfactorily. The ulcers may become atonic, giving rise to exhausting diarrhoea or intestinal haemorrhage; and, sometimes even advancing to perforation.

The above-named facts must be kept prominently before the mind, in giving a prognosis in cases of enteric fever.

The mode of fatal termination in typhoid fever, may be by asthenia, or coma; or by a combination of both. When death takes place by coma, caused by non elimination of urinary products, or deficient aeration of

the blood, the fatal event usually occurs at the end of the second, or the beginning of the third week. When death takes place by asthenia or anaemia, which it frequently does, the fatal event may not occur until the third or fourth week, or even later; and it is in such cases, usually preceded by severe abdominal symptoms. Death by sudden collapse, without any warning, is not uncommon in enteric fever; and, although usually due to perforation or haemorrhage, sudden collapse in the third or fourth week may occur, independently of these causes.

Treatment.

Enteric fever cannot be cured; but it must be treated. It may be confidently asserted, that no method of treatment as yet discovered, has any influence on the duration of the disease. Nor can the progress of the disease be arrested by any known means, after the poison has once initiated the morbid phenomena which characterise

the fever. The treatment of enteric fever is usually divided into prophylactic or preventive treatment, and therapeutic treatment. The general management is essentially similar, to that of other acute specific fevers.

The preventive treatment of enteric fever is closely associated with the etiology of the disease. It comprises the prevention of pollution of drinking water, milk &c, with feculent matter, and the prevention of the products of fecal fermentation from entering houses. Water butts and cisterns are to be scrupulously cleansed from time to time; and care must be taken that the cistern waste pipe does not communicate directly with a drain, so as to allow the drinking water to be contaminated with sewer gas. When the water supply is derived from surface wells or running streams, they must be at a distance from cess pools, sewers, privies and other filth accumulations; so that contamination of the drinking water may not occur from percolation of sewage. It ought to be held

a criminal responsibility on the part of water companies, to supply drinking water impure at its source, or contaminated during transit. Water should be filtered before drinking; and if suspicious, it ought to be tested by a drop or two of Condy's Fluid. If the colour is discharged and the suspected water turned brownish within half an hour, it is unfit to drink. The drainage must be kept in good order, free from leakage or obstruction. All openings into drains from water closets sinks etc., must be properly trapped. It must be remembered however, that if the water supply be deficient, the sewer gas may gain an entrance, although the water-closets etc, may be properly trapped; and if the drains be not properly ventilated beyond the traps, the sewer gas may force the traps, and thus be admitted into dwellings. The waste pipes of baths basins and sinks, ought therefore to be dis-connected from the main drains, as well as trapped. On the other hand, all water closet wastepipes, should be ventilated and deodorised, before connecting with the

main drain. When bad smells arise from sinks &c, disinfectants ought to be freely used, and free ventilation of the house maintained, until the cause of the escape can be found out. But the poison of enteric fever, although often accompanied by bad smells, may be itself inodorous. All house drains should be flushed, and all sinks scrubbed and cleansed, once or twice a week with plenty of water containing a disinfectant. A cess pool ought never to be within the walls of a dwelling house. When drains or cess pools are opened for repair or cleansing, disinfectants must be plentifully used, to render their contents less noxious. The residents of the house, should absent themselves, while the drainage, is being attended to. The following Chemical agents and disinfectants, are among the most useful.

1. Condy's fluid, although not an antiseptic is a good disinfecting agent. It acts as an oxidizing agent, on the products of decomposition.

Chloralum, and charcoal, act by absorbing the volatile products of putrefaction.
Carbolic acid 1 to 40, or mixed with sand or sawdust; Chloride of lime; Copperas, and Burnett's disinfecting fluid, are probably the best disinfectants to use.
These measures are particularly called for in autumn and during hot seasons; also when the house contains inmates under the age of 30 years.
When a case of enteric fever has arisen, it is important to prevent the propagation of the poison. The stools being the chief vehicles by which the disease may be communicated, they ought to be disinfected as soon as possible after being passed, with 1 to 40, Carbolic acid, before they are thrown into sewers or privies.
And they ought never to be thrown, where by any chance, they might percolate, so as to contaminate the drinking water. All soiled body linen, ought to be put into a solution of Carbolic Acid, (fl. 3iv ad Cj.) or boiled or baked before the fire, or in the sun, before being washed.

The sick room should be well ventilated, and vessels placed round it, containing Condy's fluid or Chloralum.
The original cause of the first case must be sought for and remedied. As mentioned, under Etiology, this is probably a more frequent cause of the occurrence of fresh cases, than the affected patient. While the various nuisances which may be the local cause of enteric fever are being remedied, all persons under the age of 30 years, ought to absent themselves from the house.

Therapeutic treatment.

Therapeutic treatment is directed towards the fulfilment of certain indications. The tendencies to death must be studied, and obviated if possible; it is therefore important to be acquainted with the precise duration of the disease.
The first indications, are to neutralize the poison, and improve the state of the blood.
The mineral acids, have long been favourite remedies, in the treatment of continued fevers. Their mode of action is not known.

Aromatic or dilute sulphuric acids, are certainly useful as astringents, where there is diarrhoea.

Antiseptics have been given, with a view to neutralising the poison in the bowels. Their beneficial results in this way are uncertain. Most practitioners have found them useful in reducing tympanites and diarrhoea. The agents used have been among others: Carbolic acid, Creasote, Iodine, Sulphurous acid, the Sulphites. Carbolic acid is said to act also as an antipyretic. M. A. Robin[1] protests against the use of Carbolic Acid in enteric fever: on account of its being eliminated as sulphocarbolate of potassium, and thereby depriving the tissues of their mineral constituents.

Sulphite of Soda ʒi, or ʒi – ʒii of Sulphurous acid, have been given every four hours. Probably they do not shorten the duration, or mitigate the severity of the fever. They may induce diarrhoea.

Iodine and Iodide of Potassium, have been given, by mouth & enema, to counteract

1. British Medical Journal. April 12th 1890. p. 743.

putrefaction of the intestinal contents.
The iodine treatment has been revived by Liebermeister and others. Probably it does not shorten the fever. Free Chlorine, is believed by Dr. Murchison, to be the best remedy of this class; and to have a beneficial action upon the abdominal symptoms. He recommends 20 drops of liquor Chlori, in Hydrochloric acid mixture.
Certainly many practitioners do not use mineral acids and antiseptics as a usual method of treating their enteric fever cases and they recover perfectly, under careful treatment, and good nursing, and general management. Often however sulphuric acid is used to dissolve the quinine which they may give, to reduce the temperature, when it runs unpleasantly high.
An indication which it has been thought imperative to fulfil, particularly on the continent, and in some hospitals in this country, within the last few years.
The second indication, is to relieve distressing symptoms; and this probably constitutes the chief therapeutic indication

to the ordinary private practitioner, who attempts to treat enteric fever, at the patient's home. Headache, sleeplessness, delirium, convulsions, hiccough, drowsiness, stupor and albuminuria, must be treated on the general principles which are indicated in their treatment in other diseases.

But the chief symptoms, peculiar to enteric fever, which call for medicinal treatment are:— Diarrhoea, Haemorrhage from the bowels, Vomiting, Tympanites, Abdominal pain and Epistaxis.

Diarrhoea.

May be controlled by starch enemata, containing from ten to twenty minims of Tincture of Opium. Or suitable doses of Dover's powder may be given either alone, or combined with Bismuth. If an acid mixture is being given, a little tincture of opium, or solution of morphia may be added. Lime water, or vegitable Charcoal, may be used, when acids are not well tolerated by the stomach. The acetate of lead also, either alone or combined

with Opium, acts powerfully as an astringent. Two or three grains of acetate of lead, may be given every four or six hours. Sometimes tincture of Catechu in drachm doses, every three or four hours may be required. These are the measures usually adopted to check diarrhoea in enteric fever. The activity of the treatment must be adopted to the severity of the symptom. In the majority of cases, five grains of Dover's powder, given every three or four hours will be quite efficacious; but should this not suffice, enemata of starch and opium may be given in addition; or some of the more astringent remedies named such as Acetate of lead, or Tincture of Catechu, may be administered. Many other special methods of dealing with the diarrhoea, have been suggested by different physicians. The following are a few examples: —

Small doses of Ipecac. combined with Phosphoric or Sulphuric acid; Alumn grs 24 — 3i; dissolved in gum; Alumn whey; Nitrate of Silver grs 1 — 3; Sulphate of Copper gr ¼ with Pulv. Opii gr ¼, in pill — or in

solution; with Sulphuric Acid, Quinine, and Tinct. Opii, every 4 or 6 hours.
The Copper sulphate, and Nitrate of Silver are probably, most useful, in the treatment of atonic ulcers, which cause diarrhoea.
Professor Trousseau gave laxative doses of Sulphate of Potash or Seidlitz Powders, at first. He believed them useful, where there was meteorism, as well as diarrhoea; and that this treatment checked diarrhoea, by altering the secretions. When this treatment failed, he used Mist. Cretae; Chalk and Bismuth; or Nitrate of silver.
Dr. G. Johnstone gives a tablespoonful of Castor oil, or a laxative enema when there is much meteorism, otherwise he leaves the diarrhoea alone.
As external applications, Poultices. Turpentine stupes, or wet flannel covered with oil silk or gutta percha tissue, are useful, in cases of diarrhoea, particularly when there is much abdominal pain and tympanites.

Haemorrhage.

During the first ten days, haemorrhage

may be easily checked by, acetate of lead, opium enemata, and the treatment recommended for diarrhoea. When haemorrhages occur elsewhere, large doses of perchloride or pernitrate of iron are useful. Tannic acid, turpentine, rhatany, opium and ergot, are the remedies most useful in the copious haemorrhage, which usually comes on at an advanced stage of the disease, and which is so dangerous. Ergot in large doses. ℥i of the tincture every hour, or the subcutaneous injection of full doses of Ergotine or Ergotinin, are very useful in formidable intestinal haemorrhage.

Perfect rest is to be enjoined and ice applied to the abdomen, and also given to suck.

Vomiting.

Vomiting at an early stage of the disease may be checked by emetics: if they fail, a sinapism, or turpentine stupe may be applied to the epigastrium; ice given to suck; and if acids are being taken; a change of medicine to Bismuth and Hydrocyanic acid may be prescribed. Lime water and milk often act beneficially

in arresting the vomiting. After the tenth day enemies are contraindicated. The vomiting then is often the first symptom of peritonitis.

Tympanites.

Sometimes this symptom is so excessive, as to embarrass the breathing. It increases the danger of perforation, and is a distressing symptom. Turpentine stupes and constant poulticing or fomentation of the abdomen may prevent tympanites. Sometimes enemata, containing, Carbolic Acid, Creasote, Vegitable Charcoal, Turpentine or Assafoetida and Rue, are efficacious remedies. Ice poultices, or ice applied between layers of flannel, often much relieve this symptom, and the treatment further lowers the febrile temperature, and is indicated in intestinal haemorrhage. If, with tympanites there be protracted constipation ℥i–℥ii of Castor oil may be given

Abdominal pain, is relieved by poultices or fomentations, to which Laudanum may be added; or turpentine stupes may be applied at intervals. When pain is severe

Opium may be given by mouth or rectum; or Morphia by hypodermic injection.
In young robust patients, it is said, that in the first week of the disease, a few leeches applied to the margin of the anus, or over the right iliac region, often give great and immediate relief
Epistaxis. Slight epistaxis requires no treatment. If profuse, it should be checked at once. Gallic Acid, Turpentine and Liquid extract of Ergot, may be given every hour. Probably hypodermic injection of ergotine or ergotinin, is a better method; it acts more rapidly than by the mouth. An ice-bag should be applied over the forehead and nose. Solution of alumn, Matico, Rhatany or Tannin, may be injected into the nostrils. Even plugging of the nares, may be required.

The third indication, is to treat complications, and aim at their prevention if possible. The complications common to enteric and other fevers, must be treated on general principals; but in enteric fever, all

purgatives must be avoided. The complications peculiar to enteric fever, which have to be combated are peritonitis and laryngitis. Peritonitis is by far the most frequent and important complication of enteric fever which calls for treatment. And although during life, the exact cause of the peritonitis cannot be diagnosed, by far the most frequent cause is perforation of the bowel.

Though almost invariably fatal, peritonitis is not an altogether hopeless complication of enteric fever.

The remedy is Opium, and it must be given in large doses, to do any good. Two grains should be given at once, and a grain every two or three hours until stupor is produced. In form of pill is probably the best method of administering opium by itself. Fomentations, Poultices of bran or linseed, and turpentine stupes must be assiduously applied; and the local application of ice to the abdomen is very useful in the treatment of peritonitis. The ice bladder or poultice are much more certain in their action

than the other external applications.
The application of leeches has been advocated; but few practitioners would use them in a patient so much prostrated, as one who was suffering from peritonitis in enteric fever. Besides the tendency to death in such cases is by asthenia. The patient must be kept at absolute rest in bed. The nourishment must be fluid and given in small quantities at a time, and often. Large quantities of food and stimulants are probably injurious, although they are often given. Cases have been conducted to a favourable termination on Opium and absolute starvation, — only a little water or toast water at frequent intervals being allowed. Purgatives must be carefully avoided, if the case progresses favourably: otherwise the adhesions will probably be broken up, and a fresh attack of peritonitis lighted up, which proves fatal. It has been recommended, to relieve great abdominal distension, by drawing off the intestinal gas by paracentesis. To prevent such a dangerous complication as peritonitis, all movements which might favour rupture of the denuded

peritoneum must be prohibited. This care is particularly required in mild cases, in which, the patient is able to get up without assistance. Throughout the disease avoid all strong purgatives and solid food; and in the later stages, after the 14th day, manipulations of the abdomen must be conducted with the greatest gentleness. Even after convalescence, if the ulcers are suspected of having become adonic, great care is also required not to perform any sudden or violent movement; such as getting up out of bed, to the night stool. The rule indeed ought to be, not to allow any patient to get up to the night stool, after the 14th day, until after convalescence is well established.
For laryngitis a blister may be applied below the angles of the jaw, on each side; and the whole neck enveloped in a poultice. Should suffocation appear imminent tracheotomy should at once be performed. The operation has often been successful. Should oedema of the glottis arise, the patient must be kept in a warm, moist, atmosphere

and carefully watched — sinapisms being applied to the neck. And glycerole of tannin, perchloride of iron, or finely powdered alumn, by insufflation are to be applied to the rima glottidis. If suffocation threaten laryngotomy must be performed, without delay.

The fourth indication is to promote elimination of the products of tissue waste, and of the fever poison. The importance of good ventilation and pure air, in fulfilling this indication is obvious. The patient should if possible be removed from the place in which he contracted the fever, and placed in a large airy appartment, from which all bedding, carpets, curtains, pictures and other fomites have been removed.
Diluents are useful to favour elimination. Toast water, barley water, milk and water, lemon-water, orangeade, lemonade, rice water &c, may be given according to the liking of the patient; but as a rule he prefers pure water after a time.
Diuretics are useful, but all those which

might tend to irritate the bowels must be avoided. Gin, Spirit. Aether. Nitros. and Digitalis are suitable diuretics to use in enteric fever. Common salt seems to favour elimination; it may be mixed plentifully with the patients beef tea.

Diaphoretics.

With regard to diaphoretics which might be useful to moderate the fever in the early stages; the natural periodical perspirations of the disease must be remembered. They do not apparently give much relief.

Emetics.

Emetics are undoubtedly useful in the 1st. week of the disease. They relieve the headache and vomiting, and some even claim that they diminish the duration, and severity — also the rate of mortality of the disease. They must not be given after the 10th. day, as under the effort of vomiting, the denuded Peritoneum may rupture.

The practice in this Country has been, not

to encourage elimination from the bowels in enteric fever; but to counteract diarrhoea, with astringents; and the bowels having once been locked up in this way, they are kept so, throughout the remainder of the attack. No inconvenience as a rule arises, even when the bowels do not act for four or six days in enteric fever. On the continent such physicians as Andral, Bretonneau, Louis and Trousseau, gave laxatives with the idea of eliminating a poison from the intestines, which poison they believed gave rise to the 'typhoid symptoms'. During the first week, they gave an antimonial emetic, then frequent doses of Calomel, Castor oil, Seidlitz water, laxative enemata, and cataplasms to the abdomen. Diarrhoea did not stop them from doing these things; and they did not regard meteorism and abdominal pain as contraindications. If the diarrhoea were excessive, they stopped the treatment for a day. Laxatives have recently been recommended in this

country and Calomel has long been used in Germany, to promote elimination in enteric fever. Sometimes inunctions of mercury are used; and a specific action in enteric fever, has been claimed for mercury, in cutting short the disease, or causing it to abort.

Most practitioners, in this country hold Dr. Murchison's views, that diarrhoea, is not a process of elimination, to be encouraged in enteric fever. And that the danger of enteric fever is in exact proportion to the severity of the diarrhoea; and that in cases in which diarrhoea is an urgent symptom, cerebral symptoms are often more marked; than in slight cases, where diarrhoea is either not urgent, or absent. Every one familiar with enteric fever, in practice, must have observed, the most alarming symptoms follow upon the administration of a purgative — possibly given under the impression that the patient was suffering from a bilious attack. All drastic purgatives, such as Jalap,

Colocynth &c should be avoided.

Very rarely, have the cases, seen by the writer, required even a teaspoonful of castor oil, when constipation occurred, at the outset. Such cases, have done perfectly well, without any aperient. And the only, ~~trouble~~ interference, which has been called for, in the writer's experience, with regard to the bowels, has been to check diarrhoea in enteric fever.

But on theoretical grounds, it is argued, that the poison which has been swallowed, might be swept away before absorption, by a mild aperient, of rhubarb or castor oil; or two or three doses of calomel given during the first week. Moreover, if the disease be caused by a bacillus, the calomel might act by destroying the poison in the intestine.

The fifth indication, is to reduce the temperature, and the frequency of the action of the heart.

1. Bloodletting. Bloodletting, once upon a time, was practised, with this object in view. No one would think of doing it, nowadays. But when the fever sets in with severe abdominal symptoms, a few leeches, applied around the margin of the anus, and over the right iliac region, might serve to relieve the pain of the abdomen and diarrhoea.

after the temperature is reduced by copious intestinal haemorrhage, it speedily rises again.
During late years, the cold bath treatment of enteric fever, has come into great prominence; and it has formed the subject of prolonged discussions. One of the most important during recent years, took place at the Medical Society of London, on Feb.y 18th. 1884.[1]
Dr. Sidney Coupland read a paper on the cold bath treatment of enteric fever, and Drs. Cayley, Bristowe, Austin Flint & others, took part in the discussion.
The principles on which it rests, are that the continued action of a high temperature on the body, is per se capable of producing many of the symptoms met with in enteric fever. Such as febrile oppression, headache delirium, stupor, and other cerebral symptoms. Liebermeister regards even the muscular degenerations, and other tissue changes, as in a great measure, if not entirely, due to the deleterious effects of the continued high temperature. And the objects of the antipyretic treatment, are to moderate the

1. Brit. Med. Jour. Feb. 23rd. 1884. p. p. 360 &c.

temperature, so as to keep the fever, from the outset, and during the attack, within due limits. The means adopted for this purpose, are baths of various temperatures, and certain drugs. But the baths are much more efficacious, for extracting calorie, from the febrile human body, than any of the numerous antipyretic drugs, which have been recently brought forward and advocated.

Dr. Currie, so long ago, as 1787, proposed Cold affusion, as a method of arresting and mitigating Continued fever.

Dr. Stallard introduced the Cold pack in 1846; and the name of Dr. Brand of Stettin is prominent in the advocacy of the Cold bath treatment.

In Germany, immersion in Cold water, is the usual method of treating enteric fever; but in this country, although the Cold bath has been used with apparent benefit, in certain hospitals, still this method of treatment, has met with much opposition from eminent authorities. The plans adopted vary

somewhat in detail, but the following, is the usual method of administering the cold bath.

The patient should be lightly clothed, and a bath at a temperature of from 70° to 75° F. given, every time the temperature exceeds 102.° F. The bath should be continued until distinct shivering is produced: from 5 to 20 minutes will usually be required to produce this effect, and the temperature should be reduced 2 or 3 degrees as the result of the immersion. If the bath does not produce the desired effect, it must be given at a lower temperature, or continued longer. In children a temperature of 80° will usually be low enough; or the child may be placed in a bath at a temperature of 90°, and cold water gradually added, until the temperature of the patient is sufficiently reduced. This method also, is an excellent plan to pursue, at the outset, in order to get the patient accustomed to the use of the bath, and thus overcome his

repugnance to the treatment.

It is found, as a result of the bath, that the febrile oppression, headache, delirium and stupor are diminished. The dry brown tongue, becomes moister, and refreshing sleep is obtained. The relaxation and paralysis of the vaso-motor system peculiar to the disease are counteracted, probably on account of the effect of the bath, in stimulating the vaso-motor centres. The excretion of carbonic acid and urea are diminished, the assimilation of food increased, and the febrile consumption of the body is diminished. There seems to be a great difference in the results, according to whether the baths are given regularly from the outset, or only irregularly and when the disease is advanced. If properly and regularly administered, it is said, by those enthusiastically in favour of the treatment, that the development of the typhoid state, is a clinical picture, no longer to be seen in enteric fever. And judging

by the numerous statistics adduced, there really does seem to be a diminution in the rate of mortality, in cases of enteric fever subjected to the cold bath treatment. In this connection it should be mentioned, that the advocates of the cold bath, hold, that the continued action of a temperature moderately high, is much more injurious to the system, than one temporarily much higher. At the same time, in cases of hyperpyrexia, where the chief, and indeed the imminent danger, for the moment, is the temperature per se, there can be no doubt, but that lives have been saved, by the use of the cold bath. Dr. Fox and others, have recorded cases where recovery has taken place after a temperature of 110°. F. Under every other method of treatment, such an elevation of temperature has been speedily followed by death.[1]

And even those who are opposed to the use of the bath, as a routine practice,

1. Murchison on Continued fevers. 3rd Edition p. 282.

and who deny that the mortality is diminished by this special mode of treatment, admit that in many cases, characterised by a high range of temperature, the bath, properly administered, greatly comforts the patient, and relieves the cerebral and other distressing symptoms.
Moreover, in ordinary private practice, it is found, that in the absence of conveniences for carrying out the cold bath treatment, much relief is afforded to the patient, by sponging with tepid water, to which a little Condy's fluid has been added.
The objections to the use of the bath, put forward by its opponents, are the dangers of fatal collapse, intestinal haemorrhage, congestion and inflammation of the lungs. These dangers, are not purely imaginary. But the cases must of course be properly selected, and systematically bathed, from the first. The difficulty in carrying it out, is the formidable objection to the treatment, in ordinary

practice. In hospitals where there is a good staff of attendants, this difficulty does not arise. And Mr. E. A. Fardon, of the Middlesex Hospital has invented an ingenious lift, which could be managed easily enough, were there means of possessing one!

Another formidable difficulty, is the distress and pain, and consequent repugnance of many patients to the treatment altogether.

The danger of causing fatal collapse, must be combated by the use of stimulants. It is well, indeed to administer some stimulant before or during the bath, as a general rule.

Death from collapse during a cold-bath however, does not often occur — only 1 in 2,068 of Dr. Goldammer's cases died.[2] It seems, however, that in order to obviate the danger of collapse, cases must be properly selected, and bathed systematically from the outset; and not only irregularly, at an

1. Murchison. 3rd Edition. p. 659.
2. Murchison. 3rd Edition. p. 658.

advanced stage of the disease.

In the selection of cases, it is important in this connection, to distinguish between cases presenting the 'typhoid state', and cases sinking from failure of the heart's action and asthenia, after the system has been completely shattered by the effects of the fever. The latter are not suitable cases for bathing; and unless the circulation can be rallied by stimulants, no good can be done by the cold bath treatment.

But in the 'typhoid state' — distinguished by delirium, stupor, twitching of the muscles, dicrotic pulse &c, the bath often does much good; whereas in cases of collapse — characterised by pallor with lividity of the skin, cold extremities, and perhaps an increase of the internal temperature, contemp- -oraneous therewith, and a feeble or imperceptible pulse, the bath cannot be of any use in the treatment, and may hasten the fatal event.

In cases where collapse may be

feared, it is recommended, to give the bath at a higher temperature, or use the graduated bath, or some other means of antipyretic treatment.

Haemorrhage from the bowel, is also advanced as a contraindication to the use of the cold bath. The advocates of the bath maintain however, that the cold bath treatment of enteric fever, does not increase the tendency to intestinal haemorrhage, but rather tends to diminish haemorrhage from the bowels, by contracting the mesenteric arteries, and capillary vessels. They further quote the fact, to support their argument, that dangerous haemorrhage usually occurs in cases where there is ulceration sufficiently deep to implicate an arterial branch of some size, and is never congestive; and consequently is not much under the influence of external conditions. But it is evident that in the event of haemorrhage or peritonitis occurring, the indication is for perfect rest, and that all disturbance of the patient,

for bathing, or any other purpose, ought to be avoided.

Pulmonary congestion and pneumonia, are regarded by some authorities as contraindications to the use of the cold bath. Dr. Bristowe records two cases of fatal collapse of the lungs which suddenly supervened, soon after the administration of a cold bath, during the third week of the disease; the temperature of the patient being in one case 105°. F. and the use of the bath apparently beneficial.[1]

Caution is therefore necessary, when pulmonary complications are present. But the advocates of the cold bath treatment, maintain, that notwithstanding these accidents, if only the bathing be begun early enough, before the pulmonary complications have become developed, the use of the bath may even prevent the occurrence of pulmonary complications. They advise however, that in cases where

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1. British Medical Journal; February 23rd. 1884, page 361.

pulmonary complications already exist, before the baths are given, care must be exercised, and all the circumstances of the case taken into consideration.

The objection of repugnance on the part of the patient, to submit to the treatment, may be overcome, although it is easy to understand, that the bath might be so unskilfully administered, as to become very disagreeable to the patient. It is always unpleasant to the patient, when the bath has to be administered frequently — for example, every four or six hours, which may be required in obstinate cases. But it seems, that the habit of high temperature, may be overcome by the use of the bath, aided by an occasional dose of Quinine, so that as the case progresses, the temperature may be kept in control, by a less frequent repetition of the bath. For some time after the administration of a bath, the temperature continues to fall, in the rectum, on account of the trunk parting with its heat to the extremities; when

the temperature reaches 102.5° or 103° in the rectum, the indication arises, for another bath. If skilfully administered, many patients may be glad to submit to the bath, on account of the relief which they experience, while others are willing to tolerate the immediate discomfort, in order to experience the euphoria which is afterwards promoted. In order to accustom patients, particularly children, to the use of the bath, it is well in many instances to begin with a temperature higher than 70° to 75°, and then gradually diminish the temperature of the bath given: or the graduated bath may be used. The bath must be brought close up to the bed before lifting the patient. Where the repugnance is insuperable, the bath treatment must be intermitted; and also in those cases in which the treatment is contraindicated, some other means of antipyresis must be adopted. It seems that those cases which are most benefited, comprise the acute variety of enteric

fever, in which the temperature rises to 104° F. or upwards, and in which the abdominal symptoms are not marked.

In the more insidious forms, accompanied by deep ulceration, and a temperature not dangerously high, the bath treatment is not so beneficial; and hence the cold bath treatment is not suitable in the case of aged persons. Although the cold bath, is the most generally applicable and efficient remedy of its class, in enteric fever, it is necessary to be acquainted with the chief modifications of the methods adopted for the external application of cold to the body in fevers.

The graduated bath has already been mentioned. In this modification the patient is placed in a bath about 10° lower than the temperature of the body, and cold water is gradually added until the required temperature is obtained.

Cold affusion. In this modification, the patient is seated naked in an empty

bath or tub, and several buckets of water, at a temperature of 40° to 50° F., are poured over the head and chest, from a height of three or more feet, the patient being then hastily dried and restored to bed.

The Cold Pack, in which the patient is enveloped in a cold wet sheet, as recommended by Brand.

Cold Sponging, recommended by Sir Robert Christison, and resorted to by medical men in ordinary practice.

Ice compresses to the abdomen and axillae, are also much used in ordinary private practice, especially where there is much tympanites, intestinal haemorrhage or peritonitis.

Cold applied to the head by an ice bag or irrigating cap, is useful in enteric fever, especially for the relief of headache.

Exposure of the body to cold air, is another method. After sponging, a cradle may be placed over the patient, open at both ends, in order to allow

the surface of the body, to be exposed to the surrounding atmosphere. Bags of ice are suspended, inside the cradle, so that the temperature is reduced thereby. This method is adopted at the London Hospital.

Many other methods, which have not yet obtained a place in medical practice, have been proposed — such as iced water beds, refrigerating envelopes of various kinds, and permanent tepid baths. Iced enemas have been suggested, but are dangerous. Ice may be given by the mouth, to mitigate the fever, and relieve other symptoms.

The antipyretic treatment by means of drugs, is not nearly so satisfactory as the cold bath treatment. Liebermeister however regards antipyretic drugs, as useful adjuncts to the use of the bath, whereas Brand and others, are opposed to their use. Quite an army of new antipyretic remedies have been recently introduced,

such as antipyrin, antifebrin, acetphenetidin, antithermin, Hydrochinon, Salol, Thallin &c, but they have not yet been sufficiently tried, to be generally relied upon in ordinary practice. And a feeling of doubt moreover, is beginning to creep in, as to whether, it is really an advantage, to reduce the temperature in febrile diseases, by means of powerful drugs. Nearly all antipyretic drugs, if given in doses sufficiently large to keep the temperature constantly under a fever heat, affect injuriously many functions of the body.

Those drugs most to be relied on are Quinine, Salicine, Salicylic acid, Salicylate of Soda and Digitalis. They have been well tried in cases of fever, and it is now well known, what effects they will produce.

It is said however, that the cold bath is far superior to these drugs, in relieving the dry tongue, promoting the assimilation of milk, and diminishing the excretion of urea in enteric fever. The headache

stupor, delirium and cerebral symptoms moreover, are more markedly relieved by the use of the bath, than by drugs.
Quinine is probably the drug to be most relied upon; the objections to Salicylate of Soda being its depressing action on the heart, and its tendency to induce delirium and albuminuria. Quinine, except that it may produce symptoms of Cinchonism, in the large doses which it is sometimes necessary to give, in order to control the temperature, does not seem to give rise to injurious results. The quinine may be given in doses of from 15 to 40 grains.
It is best given in suspension, and when large doses are required, they may be divided, and administered every ten minutes until the required amount is given. Should this treatment produce vomiting, a little opium may be added to the quinine. According to Liebermeister, the result to be desired, is a marked remission of the morning temperature, rather than a diminution of the evening exacerbation; he therefore

recommends, that the quinine should be given towards evening. The maximum antipyretic effect is produced, about eight hours after administration.

Quinine is well borne by children; and it seems to be most useful, late in the disease, in cases where the morning remissions are marked. As an anti-pyretic, Salicylate of soda may be given in doses of from 20 to 60 grains.

Kairin ($C_{10}.H_{13}.N.O.$). has been recently introduced as an antipyretic by Dr. Wilhelm Filehne, who among others, has carefully investigated its action. If fresh, and properly prepared, it seems not to produce any disagreeable symptoms. Often the reduction of temperature is the only change produced; it sometimes however, produces rigors, and usually profuse perspiration. Cyanosis and symptoms of collapse, which have been observed as results of its administration, are attributed to the drug being either impure or not recently enough prepared. But it would probably be better not to

give it, in cases where symptoms of cardiac failure, or pulmonary complications were present. It may be given with safety, in doses of from ½ to 15 grains, every hour until the temperature is reduced.

Dr. Riess found, that much larger doses of from 30 to 34 grains might be given.

The effects of the administration of Kairin, are transient, and the temperature soon rises again, often to a greater height than before.

Digitalis in large doses, is strongly* recommended by Liebermeister — 12 to 24 grains, in divided doses, in from 24 to 36 hours, in the form of powders or pills, are to be given, in cases where cardiac weakness does not constitute a contraindication. In such doses, no doubt Digitalis powerfully reduces the temperature, but it is probably dangerous, and is not as a rule resorted to in this country in such large doses.

The above named drugs, then, may be used in order to render a smaller number of baths sufficient, in

conducting the antipyretic treatment of enteric fever; and most general practitioners, administer quinine, in cases where the elevation of temperature is obstinate, in order to keep it as far as possible under control.

The last indication, is to sustain the vital powers, by appropriate food and stimulants; but in doing so to avoid exciting congestion, or increasing the work of the already overtasked glandular organs.

The diet in enteric fever, should consist entirely of fluids or semi-solid articles of diet, being at the same time nutritious and easily digested. And, in spite of certain theoretical objections, which have been advanced against it, most practitioners find that milk, is 'par excellence' the diet in enteric fever. Beef tea, seems sometimes to increase the diarrhoea. Therefore, if it be desired to give beef-tea, it had better be thickened with a little arrowroot. Veal or chicken-broth

may be given with vermicelli, or arrowroot, also meat essences, meat jellies, custards, sago and other farinacea, may be found useful at various stages of the disease.

But milk is the one diet, on which many practitioners rely, in treating enteric fever; and many regard it as a favourable circumstance, that the patient is able to take as much as two quarts of milk per diem.

As a rule alcoholic stimulants are not required, at an early stage of the disease. But later in the disease; and particularly, where there are symptoms of failure of the action of the heart; it seems evident, that a little wine or brandy, may prove most beneficial: although there are some who attempt to treat enteric fever, entirely without the use of alcoholic stimulants.

Carbonate of ammonia, is not a suitable stimulant, to administer in enteric fever, on account of the intestinal lesions.

The patient's nervous and muscular power, must be conserved. As soon as the disease has declared itself, the patient should be confined to bed, and all bodily and mental exertion intermitted. He ought to be provided with a bed-pan, to prevent the necessity of getting out of bed; as well on account of the danger of causing rupture of the denuded peritoneum in the later stages of the disease, as in order to conserve nervous and muscular force.

If delirium should be violent, the less restraint exercised to prevent the patient from getting out of bed, or otherwise injuring himself, the better. Gentleness and firmness, will often be more efficacious, in ruling the patient, and less exhausting and distressing to him, than physical force. Sometimes, however, it may be necessary to fold sheets, and pass them over the chest and extremities of the patient, fastening them to the sides of the bed, in order to prevent him from getting out.

During convalescence, patients recovering from enteric fever, must exercise great care. If diarrhoea should be persistent during convalescence, probably there may be an atonic condition of some of the intestinal ulcers. In such cases, acetate of lead, sulphate of copper, or nitrate of silver must be given, as already indicated, under the treatment of symptoms. The patient must, at the same time be confined to bed.
But, it should be remembered, as pointed out by Trousseau, that vomiting and purging may be of purely nervous origin; especially in cases which have been subjected to a too rigorous regimen. In such cases these symptoms, may be at once removed by giving solid food.
The liability to perforation, haemorrhage or a relapse, when convalescence appears to be progressing favourably, must be kept in remembrance. And it ought to be a never-failing rule, to continue to take the temperature

every evening, for at least two weeks, after the commencement of convalescence.

With regard to the diet; no solid food should be taken, for at least seven days after convalescence has commenced; and not until much later even than this, if there should be any intestinal disturbance. The diet during early convalescence, should consist of, milk, eggs, custards, farinaceous puddings of various kinds, fingers of bread and butter &c. And it is better, to try a little white fish, before giving butcher's meat. All purgatives must be avoided, and a teaspoonful or two of castor oil, administered, if constipation should be obstinate. But it is rare that even this is required. Malt liquors are apt to derange the bowels; and they certainly are not necessary in the treatment, during convalescence after enteric fever. It may be useful to administer tonics of various kinds, in combination with mineral acids.
