DYSENTERY AMONG PRISONERS OF WAR: SINGAPORE 1942-45.

A Thesis for the Degree of M.D.

by

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CHAPTER I.

A HISTORY OF THE DYSENTERIES.

"Of Dysentery it may be said that where man is found, some of its forms appear." (Hyres).

References to a disease which was almost certainly dysentery appear in Hindu writings over a thousand years before Christ. A similar condition is described by early Greek and Roman writers. The army of the Philistines was struck down with it. According to Herodotus, Xerxes was defeated by the Greeks in 480 B.C. because his army was destroyed by dysentery and plague. In truth, from the earliest times dysentery appears to have existed in some form all over the world. Hippocrates himself, wrote of it:

> "Dysenteries, when they set in with fever, alvine discharges of a mixed character, or with inflammation of the liver, or of the hypochondrium or of the stomach, such as are painful, with retention of food and thirst, all these are bad: and the more of these symptoms there are, the greater the danger; and the fewer, the more hope is there of recovery. Children from five to ten years of age are the most apt to die of this complaint; the other ages less so. Such dysenteries as are of a beneficial nature, and are attended with blood and

scrapings of the bowels, cease on the seventh or fourteenth or twentieth or thirtieth day, or within that period. In such cases even a pregnant woman may recover and not suffer abortion. All cases of lientary are said to be of bad character when they are continued and protracted, both day and night, and when the dejections are either very crude, or black, soft and foetid; for they occasion thirst and determine the fluids otherwise than to the bladder, give rise to ulcerations in the mouth, redness and ephelis of all colours, and at the same time the belly is in a state of ferment, and has a foul wrinkled appearance externally. This disease is most to be dreaded by old persons; it is formidable to men of middle age, but less so in the other ages. The indications of cure, it is acutely stated, are to determine the fluids to the urine, to relieve the body from its atrophy, and change the colour of the skin. All the other varieties of diarrhoea without fever are of short duration and mild, for they will all cease when washed out, or of their own accord. The discharge may be predicted as about to cease when, upon touching the belly, there is no movement, and flatulence passes without the discharge."

(Adams' translation.)

Following in the footsteps of the father of Medicine, many of the ancient writers have written outstanding descriptions of the disease, but none as brilliant as his. Celsus, referring to it as Tormina, said that it was due to ulcers in the interior of the bowel which caused the patient to suffer continuously from tenesmus and pain near the anus. After Galen the name "dysentery" came to include many kinds of intestinal "fluxes", so that one must not imagine that early and mediaeval writers were necessarily referring to the pathologies which we ourselves think of as dysentery. However, the syndrome of bloody flux and tenesmus plays no small part in the Arabian medical compendiums and in the works of European writers in the Middle Ages.

Medical writers have not been alone in their interest in dysentery. Numerous references to the disease are to be found in the works of chroniclers and historians in the Dark and Middle Ages. Epidemics are recorded as having occurred in France in 534 and 538, in Northern Europe in 760, and among German troops in Hungary in 820. Further epidemics are described in Germany in 1083 and 1113, in England in 1316, on the Ligurian coast in 1330 and in Bordeaux in 1411. An outbreak of the disease is recorded as having occurred at the siège of Bagdad by the Saracens in 1439.

Henry V, as was about three quarters of his army, was struck down by dysentery while advancing against the Dauphin in 1422. Henry's infection appears to have become chronic, as he died of a wasting illness some three months after first becoming ill. Dysentery was one of the principal diseases at the siege of Metz by Charles V in 1552.

Dysentery appears to have been common in the New World early in its history. Bloody diarrhoea is said to have been prevalent among the Indians even before the arrival of Columbus. The English expedition to Puerto Rico at the close of the sixteenth century was largely defeated by dysentery. Out of one thousand men who landed at San Juan in 1598, four hundred died of an illness which was described by one who was present, a physician called Layfield. The symptoms were fever and diarrhoea with as many as sixty to a hundred stools daily with bloody mucus.

During the seventeenth century the clinical entity which today is known as dysentery became more clearly defined. It was in this century that "dysentery root" (ipecacuanha) was introduced. In 1628 a great epidemic of dysentery swept across the Dutch Indies. This was described by Jacobus Bontius who himself suffered from the disease during the siege of Batavia. He describes dysentery as "an ulceration of the intestines with a perpetual purging, at first mucous,

and afterwards bloody, and lastly purulent, with intolerable pain and griping of the belly." No man who has suffered from the disease can fail to be struck by the feeling in this vivid description.

As the century progressed dysentery came to play an increasingly prominent part in medical writings. At this time the disease was spread generally throughout Europe, and epidemics occurred in England, France, the German states, Switzerland and Scandinavia. It was so common in Ireland that it was known as the "Country Disease", and in that country in 1689 at the siege of Dundalk 6000 deaths were attributed to dysentery.

In the following century the North American Colonies (later the United States) began to figure as a pandemic area, and epidemics spreading widely over the country occurred between the years 1749-53, 1773-77 and 1793-98. In the middle of the century Pringle gave an account of dysentery in the British Army and Cleghorn in the Navy. Pringle was impressed by the infectious nature of the disease and regarded the excreta as the vehicle of infection. In a book published in 1767 on an epidemic in Switzerland accurate clinical descriptions were given of the different types of dysentery. At the end of the century and the beginning of the next, the Napoleonic Wars provided numerous opportunities for outbreaks of dysentery as a war disease.

It was during the eighteenth century that several outstanding essays on dysentery as a tropical disease were published. Most of these early articles on dysentery in the tropics were from the West Indies. As early as 1764 Grainger of St. Kitts recognised that there are two fundamentally different types of dysentery - a distinction which was not drawn in the East Indies for a further forty years. In 1788 John Hunter described dysentery in the Army in Jamaica. From his post mortem findings it would appear to have been of the bacillary type. During this century several new suggestions were put forward as the cause of dysentery. One said it was due to obstructed perspiration, another to drenching with sea water, and a third to the combined effects of fatigue and salted provisions. It was not until 1825 that contaminated drinking water was first considered as the medium of infection by McCabe of the West Indies.

In the first half of the nineteenth century the milestones in the history of dysentery are the profound and accurate observations of the tropical clinicians working mainly in India. In 1818 Sir George Ballingall, the last occupant of the Chair of Military Surgery in Edinburgh, clearly distinguished, in a work on dysentery among European troops in India, the two basically different clinical types of the disease. In 1822 Chisholm also divided dysentery into

two types which he called Sporadic or Ideopathic dysentery and Hepatic dysentery respectively. Unfortunately, in the light of modern knowledge even the names of his types seem to have confused the issue somewhat. This author, however, gave an interesting description of his second and more severe type. He speaks of the passage of frequent fluid stools like the washings of raw meat, which, if neglected might have fatal results in six, ten or twenty-four hours. At post mortem the liver was found to be large, and inflamed, and particularly suppurated or 'like rotten cork'. The intestines were extensively inflamed 'more especially the smaller intestine'. This last remark makes one wonder if the condition was dysentery at all. If it was dysentery it would appear to have been a particularly acute bacillary dysentery, probably a Shiga infection.

During the earlier years of the century tropical clinicians displayed a lively if somewhat speculative interest in the connection between hepatic abscess and dysentery. It was early accepted by many workers that there must be some intimate relationship between the two conditions, but their supposition was based mainly on intelligent conjecture, and the subject continued to be disputed for more than three quarters of the century. In 1828 a very great work on Tropical Medicine made its appearance, namely, Sir James Annesley's

"Researches into the Cause, Nature and Treatment of the more Prevalent Diseases of India". Annesley's beautiful volumes contain many artistic but accurate coloured plates, and among them are several excellent illustrations of liver abscess and of the changes in the colon in both forms of dysentery. He sums up the discussion on the subject thus:

> But although dysentery thus frequently supervenes in a more or less immediate manner, to affections of the liver, in some instances appearing as nearly coeval with them, in others seeming to supervene as a remote and contingent affection - it must also be kept in recollection that dysentery may be the primary disease, and that the affection of the liver may be consequent upon it, not as an accidental occurrence, but as a consequence of the morbid action....

However, four years later Sir George Ballingall, whose words carried great authority, wrote in his "Outlines of Military Surgery", "...I think myself entitled to conclude... that hepatic inflammation and dysentery are not so frequently co-existent in India as has been commonly stated and believed".

The debate continued. Twining working in 1835 in Calcutta considered liver abscess to be secondary to the type of dysentery he described, which was obviously amoebic. Parkes, who published in 1846 his "Remarks on Dysentery and

Hepatitis in India", described lesions which were undoubtedly of amoebic origin, and stated that he found liver abscess in 21% of cases in British soldiers, but had never seen one in a native.

In 1845 in a work on the diseases of the liver Budd said that "...in the tropics the endemic prevalence of abscess of the liver is causally connected with the dysentery which is likewise endemic in those regions". Some forty years later, only one year before the matter was proved beyond the shadow of a doubt, Hirsch commented on Budd's theory that though having scanty foundation it was received with applause, but that "...neither statistics nor the due consideration of other matters of experience are in its favour." He then goes on to consider evidence from non-tropical countries, where, he says,

> Severe epidemics of dysentery are far from uncommon, although abscess of the liver from whatever cause counts as one of the rarest of maladies, and as a sequel of dysentery rarer still- if we keep these facts in mind, we shall have to admit that although abscess of the liver is a rather common complication of dysentery, and the two affections may in many cases be even causally related to each other, we are by no means warranted in concluding from the facts as we have them, that dysentery is the

real occasion of hepatic abscess being endemic in the tropics.

Thirty years earlier Sir James Ranald Martin, writing on dysentery in India matched Hirsch's caution if not his circumlocution. He was referring to William Baly, who had said that out of hundreds of cases of dysentery in the Millbank Prison in London not one had been complicated by hepatic abscess. On this Martin comments, cannily,

> These facts would lead to a conclusion that foreign climate mainly and its unnatural influences, and more particularly heat and malaria, produce the difference of result as respect British subjects; but it is most difficult here, as in other diseases to determine what cause and what coincidence.

Martin also described an interesting form of complicated dysentery which he called 'scorbutic dysentery'. From this, he said, he saw forty men die in one night. The symptoms he describes are swollen, loose livid gums with ulcerated sloughing edges; foetid breath; pain and hard swelling in the calves and purple discolouration of the lower extremities. So far it would appear purely scurvy, but he goes on - oedematous swelling of the feet and legs, anasarca ascites and hydrothorax. This would appear to be beri-beri or famine oedema, according to the circumstances.

In justice to Martin it must be added that he was familiar with beri-beri and also with another vitamin B deficiency, namely, 'burning feet'. He had, of course, no means of knowing the cause of these two conditions, but strangely enough he links them in one chapter-heading in his book.

Early in the second half of the nineteenth century a discovery was made in Prague which should have arrested much of the speculation. In 1859 Lambl found living amoebae in the stools of a child dying of diarrhoea. Lambl's report was not generally credited and others were of the opinion that he had merely found degenerated forms of Trichamonas hominis. In 1870-71 Lewis and Cunningham, while working on cholera in India, independently reported that they had found amoebae in choleraic stools. However, in 1875 a great advance was made along the right path. In that year in St. Petersburg Lösch discovered an amoeba in the stools of a peasant from Archangel who was suffering from chronic dysentery. From Lösch's detailed description it is clear that he had discovered the organism which is known to us as Entamoeba histolytica, but the full magnitude of his discovery eluded him. In spite of the fact that by injected fresh stools from this case into the recta of dogs, he produced dysentery with rectal ulceration from which he was able to isolate the amoebae afresh, Lösch thought that the amoeba merely prevented the ulcers from healing and was not itself the cause of the disease.

In 1883 Robert Koch was investigating cholera in Egypt and his findings appeared to confirm the work of Lewis and Cunningham of twelve years earlier, for he found amoebae to be present in sections of bowel from the cholera cases. These amoebae which were found in choleraic material were probably E. coli. Much of the discussion which was provoked by Lösch's historic discovery proved unfruitful because it dealt largely with this harmless organism. It was, of course, not known at the time that dysentery is really several diseases of which only one is caused by an amoeba, or that some amoebae are pathogenic whilst others are not. However, unfortunately, this side-tracking of the main line of the investigation led at least one worker back on to the right path, for the work of Koch inspired Kartulis in Egypt. He found amoebae to be present in the colon at post mortem in 150 cases of dysentery, and sections of the ulcers in some of them, and what is more, in 1887 he found the same organism in the pus of a liver abscess and in the walls of such abscesses. This outstanding advance, which was quickly confirmed by Koch and Gaffky, finally proved the contention which had long been held by clinicians in India, namely, that 'tropical dysentery' and 'tropical abscess' were linked in their aetiology.

In 1890, further confirmation of Kartulis's great

discovery came from America where Osler found amoebae in the pus of the liver abscess of a man who had contracted dysentery in Panama. In the following year Councilman and Lafleur working in Osler's wards in Baltimore, published a paper for the title of which the phrase 'Amoebic Dysentery' was coined. They found that amoebae similar to those described by Lösch were present in many of the dysentery cases in the wards, that many of these cases were of a chronic character, and that such cases did not usually have fever unless complicated by hepatitis. They also indicated that there are two types of amoebae, a harmless and a pathogenic, which they called Amoeba coli and Amoeba dysenteriae respectively. At about the same time Lutz, in Germany, came to similar conclusions, namely, that amoebae were the cause of chronic dysentery, which was often followed by liver abscess, and was a distinct disease from epidemic dysentery. In 1891, also, Dock reported the presence of amoebae in dysentery stools in Texas, and in the following year similar reports came from Italy, Austria, Germany and Batavia.

In 1893 Kruse and Pasquale, working in Egypt, confirmed the findings of Councilman and Lafleur, and gave descriptions of the two types of amoebae. They were able to produce dysentery in cats not only by injecting dysenteric stools into the bowel but also by injecting pus from liver abscesses. In the same year the first accurate description of the mode of infection was published in Germany. The authors were Quincke and Roos, who

described the cystic form of the amoeba. They infected cats both by causing them to swallow the cysts and by injecting vegetative amoebae into the rectum. Finally they drew the logical conclusion that mode of infection in man is by the ingestion of cysts. These authors compared the morphology and movements of the pathogenic and non-pathogenic amoebae, but it was not until ten years later that these organisms acquired their present day names of Entamoeba histolytica and E. coli. These two names, well-known today, made their first appearance in 1903 when Schaudinn's paper on the life-history of human intestinal amoebae was published.

Mention must be made here of the Italians, whose work takes us back to 1879 when Grassi reported having found amoebae in dysenteric stools. Perroncito confirmed this in 1881. However, in 1895 Celli and Fiocca concluded that all dysentery was bacillary. In that year also Casagrandi and Barbagallo carried out a biological and clinical investigation, but came to the conclusion that all amoebae were harmless.

Of French publications those of Sonsino in 1876 and Marchoux in 1899 reported amoebae in dysenteric stools. Marchoux's paper dealt with dysentery in Senegal, and he proved the pathogenicity of the amoebae he found by infecting cats.

In 1901 and 1903, Leonard Rogers reported on cases of amoebic dysentery in India and proved that in liver abscess secondary to this form of dysentery, the amoeba was the only

organism present provided the material for examination was taken before post-operative secondary infection could take place.

In 1901, also, Jaeger reported the occurrence of amoebic dysentery in Germany, and in the following year Jurgens published a paper on amoebic dysentery among German troops who had returned from China, and contrasted this with the epidemic form of dysentery. In 1903, the year that saw the publication of Schaudinn's historic observations, Huber described the four-nucleated cysts of E. histolytica.

Having reached this stage the progress of the investigation appears to have been held up for some years. According to Dobell this set-back was largely due to errors in Schaudinn's work and to the statement of Musgrave and Clegg that 'all amoebae are or may become pathogenic'. These authors, two of several who were working on the problem in the Philippines at that time, were the first to use the term 'amoebiasis'. Unfortunately they supposed that there was but one species of amoeba. Although they devised culture methods, the organism they were working with was a free-living amoeba and not an entamoeba. In 1908 Anderson in the Andaman Islands published some statistics to prove his contention that E. histolytica caused neither dysentery nor liver abscess. In 1910 Allen agreed with Musgrave and Clegg that all amoebae found in the human intestine were pathogenic, and stated that their hosts

would sooner or later develop active amoebiasis.

And so on, conclusion contradicting conclusion until the work of Walker, another worker in the Philippines, was published in 1911. His important findings can be summarised as follows - that free-living amoebae found in the water supply could not be parasitic in man: that only entamobae could be parasitic in man and that the genus was a strict obligatory parasite and contained two species, the pathogenic E. histolytica which formed cysts with four nuclei, and the non-pathogenic E. coli which formed eight-nucleated cysts: that infection by either of these entamoebae must come directly or indirectly from another infected person: that water or uncooked food could only transmit amoebic dysentery when contaminated by faecal matter from a case of amoebic dysentery. He recognised cyst-passers (infected persons who continued to pass cysts after symptoms of amoebic dysentery had passed away) as a danger to the community, and he pointed out the obvious prophylactic measures. In 1913 Walker published a paper jointly with Sellards which cleared up most of the remaining confusion. This reported a series of controlled experiments on human volunteers who were fed with the cysts of various species of amoebae. Only those who received the cysts of E.histolytica developed dysentery. These authors also described the process of encystment - how the large, actively moving vegetative form throws out its food

particles and assumes a smaller precystic form containing one large nucleus, before finally forming the four-nucleated cysts.

Meanwhile as the result of parallel investigations, the knowledge of epidemic dysentery had developed pari passu. In turn, bacteriologists ascribed the disease to a variety of organisms, as naturally the difficulty of isolating the causative organism from the complicated flora of the human intestine was considerable. In 1869 Basch reported having found leptothrix filaments in sections of the bowel wall in a case of dysentery. After that it was fashionable for a period to consider the disease polymicrobic in origin. In 1888 Chantemesse and Widal described an organism which may well have the same as that which was described later by Shiga, although some of the methods by which that organism was later identified were not then known. In the next few years the harmless B. coli was often indicted, and other organisms blamed included B. pyocyaneus, streptococci and B. proteus.

In 1898 the results of Shiga's work on epidemic dysentery in Japan were published. He reported the discovery of distinct species of the typho-coli group of bacilli, and demonstrated that the serum of patients suffering from dysentery agglutinated his bacillus. Not long afterwards Kruse isolated the same bacillus from dysentery cases in Germany. In 1900 Flexner once more found the dysentery bacillus of Shiga in the Philippines

and also another species with the power of fermenting mannite. Flexner's bacillus was shown by Martini and Lentz in 1902 to have distinct agglutinative characters. In 1903 Hiss and Russell isolated a bacillus of the Flexner type from fatal cases of infantile diarrhoea in the United States. This organism which ferments mannite as well as dextrose, they called the Y bacillus. The Bacillus of Sonne dysentery was first isolated in 1900 by Kruse, who called it the lactose-fermenting pseudo-dysentery bacillus. This organism was rediscovered at intervals until 1915 when Sonne gave a detailed description of it in an epidemic in Copenhagen.

Thus in the early years of this century, amoebic and bacillary dysentery were clearly established as separate diseases; but nevertheless, the problem of distinguishing one from the other is often fraught with difficulty to this day. Amoebic dysentery is a disease of tropical and sub-tropical regions: it has rarely occurred in those who have never left temperate zones; although in closed communities in the tropics (such as that to be described later) a large number of cases may be encountered, it is not an epidemic disease, as in most cases there is a considerable lapse of time after infection before symptoms develop. Bacillary dysentery has occurred in all climates. In temperate climates with good sanitation the cases are usually sporadic, although epidemics have occurred in recent years in several European countries and in the United States.

In the days when the sanitary conditions in this country were similar to those which are still to be found in many parts of the tropics, great epidemics occurred. The disease has also occurred in very cold climates as, in the depths of the Russian winter, it struck down many in Napoleon's army on the retreat from Moscow. However, today, the most widespread incidence of bacillary dysentery is in tropical and sub-tropical countries where, in the presence of almost non-existent sanitation the most alarming epidemics sweep through the population.

Speaking of bacillary dysentery Shiga said "it is always a constant companion of war, and it is more fatal to armies than powder or shot." Although the number of cases of amoebic dysentery in certain theatres of the late war was extremely large, bacillary dysentery has always been a more formidable enemy of armies in the field. Mention has already been made of dysentery in the wars of antiquity, in the dark and middle ages and in the Napoleonic Wars. It took part in the Crimean War. 38,108 cases of bacillary dysentery were reported in the South African War. In the 1914-18 War it was responsible for a considerable portion of casualties from all fronts. In Gallipoli and Mesopotamia only 15-20% of the vast number of dysentery cases are said to have been amoebic. In 1917 3.76 per thousand casualties from France were due to bacillary dysentery, and in view of the large number of battle

casualties which occurred in that year this figure must represent quite a considerable number of cases. In the same year the incidence of dysentery reached 486 per thousand casualties in East Africa.

A History of Treatment.

Remedies almost without number have been used in the attempt to provide a specific cure for the dysenteries through the ages. Everything from plain water to strong poisons, from hypnotics to the most active purgatives seems to have had its place. As is so often the case with dangerous diseases for which the remedy was long sought, every panacea which had proved a success in other ailments was tried, sometimes with advantage. Nevertheless the only drug whose history for several centuries has been linked with dysentery is ipecacuanha. An early reference to this drug dates from 1625, when the following passage appeared in "Hakluytus Posthumus, or Purchas his Pilgrimes":

> Igpecaya, or Pigaya is profitable for the blondie Flux:... this roote beaten and put in water all night at the deaw, and in the morning if this water with the same roote beaten and strained be drunke, onely the water, it causeth presently to purge in such sort, that the laske ceaseth altogether.

The 'dysentery root' or 'Brazilian root' was first brought to Europe in 1658 by Piso, and it was used in India for dysentery as early as 1660, but appears to have been forgotten later. In 1688 the root was being used in Paris by a Dutch physician, Helvetius, and the secret of its use was sold by him to Louis XIV as a specific for dysentery. Neglect of its use for dysentery appears to have set in here as in India, but in the West Indies it was introduced into general use by Richard Towne in 1726. James Sims in 1773 appears to have recognised two important facts about the use of this drug; firstly that it was the drug retained and not the emesis which was beneficial, for he advocated smaller doses with opium; and secondly that the drug was of greater value in the chronic form of the disease.

In the disastrous British expedition to Walcheren Island in 1809, many thousands of soldiers lost their lives through disease. In the course of this ill-conceived and mismanaged campaign both the Surgeon-General and the Physician-General of the British Army were dismissed for declining to go to the front to cope with the medical situation. Pringle, who had to deal with the epidemic dysentery which was decimating the force, appears to have used ipecacuanha mainly as an emetic. He did note, however, that the best results were obtained by giving five grains of the root every hour.

In 1828 Annesley was using ipecacuanha in small doses in India. He advocated one to three grains thrice daily, but mentioned that some doctors employed doses of 20 to 30 grains. The following technique for administering large doses of ipecacuanha was described some years later:

> A sinapism to cover the abdomen and immediately after this a drachm dose of laudanum. Half an hour after, when the irritability of the stomach has been diminished, the ipecacuanha is administered, generally in draught, sometimes in pill or bolus, while a semi-recumbent posture is steadily maintained.

Twining, writing on dysentery in Bengal in 1835 recommended ipecacuanha in six grain doses twice daily with gentian to prevent vomiting and five grains of blue pill. He was doubtless dealing mainly with amoebic dysentery, for he regarded ipecacuanha as the best remedy, and gave this warning about the use of salines: "Great caution is necessary in the administration of saline purgatives in all stages of dysentery; even the mildest saline solutions are sometimes apt to cause irritation by carrying away the natural mucus of the intestines." Twining gave cold water per rectum in cases in which there was anuria or much bleeding.

Another author who had to deal mainly with amoebic dysentery was Parkes who published his "Remarks on the Dysentery and Hepatitis of India" in 1846. He found 30 to 60

grains of ipecacuanha much more efficacious than the small dosage previously prescribed. In the next year, however, Baly whose experience in the Millbank Prison must have been entirely of the bacillary type, wrote,"...ipecacuanha has wholly failed in my hands as a specific, or in any way active remedy for the disease."

A Frenchman, Hospel, who published his observations on the treatment of liver abscess in the Army in Algeria in 1852, said that ipecacuanha acted by reducing hepatic congestion. This, he thought, was brought about partly by drawing the blood from the liver to the stomach, and partly by the muscular contractions in the act of vomiting. During the next few years two of his countrymen, Delioux and Savignac, carried out clinical experiments at the Naval Hospitals of Rochefort and Toulon. They concluded that ipecacuanha was as much a specific for dysentery as quinine for malaria, that the vomiting was deleterious rather than beneficial, and that the therapeutic effect was produced by the ipecacuanha absorbed.

Although Parkes had been using large doses of ipecacuanha more than ten years earlier, many authors give the credit for introducing this practice to Scott-Docker who was treating dysentery among British troops in Mauritius in 1858. Out of fifty cases he only had one death. His technique was to use 20 to 30 grains of ipecacuanha after

premedication with opium. In severe cases his dosage was as high as 90 grains. Judging by these results there seems little doubt that these were cases of amoebic dysentery, while in the case of Woodward who recorded his experiences with dysentery in the American Civil War, the type was probably entirely bacillary. He did not have good results with ipecacuanha, and appears only to have used it in the acute stage, or in chronic cases where there was no ulceration.

W. C. Maclean in his "Diseases of Tropical Climates" (1886) recommended half drachm doses of ipecacuanha following opium, and this was repeated every eight hours until the patient recovered. He was aware of the value of ipecacuanha in preventing hepatic complications including liver abscess. At this time the fashion was to use cathartics rather than ipecacuanha. "How this remedy" wrote Maclean, "almost deserving the name of a specific, came to be superseded by calomel and opium in the treatment of dysentery in the East is one of the most curious questions in the history of tropical medicine." Nevertheless in the next few years ipecacuanha was used less and less and salines were taking its place in the routine treatment of dysentery. But the answer to Maclean's 'curious question' was already at hand as Kartulis's first paper on the amoeba in dysentery had been published in the previous year.

Although Sir Patrick Manson used ipecacuanha exclusively for chronic dysentery and liver abscess from 1883 onwards, attention was mainly focused on the saline treatment of dysentery during the last decade of the nineteenth century. The experience of Army Medical Officers in the South African War was that this treatment was far more effective than ipecacuanha except in soldiers who had served in India (thus making it abundantly clear that the common form of the disease in South Africa was bacillary). Soon after the turn of the century ipecacuanha was coming into its own again as the specific treatment for amoebic dysentery. In India it was either given following a sedative or mixed with tannic acid. In the Philippines it was given in pills with a protective coating to prevent it having an action on the gastric mucosa. But the nausea and vomiting were grave disadvantages. In an effort to avoid this it was advised that fluids should be restricted and the patient kept lying still (this must often have been difficult in a patient having frequent stools).

To avoid the emetic action of ipecacuanha had been the object of workers on emetine for nearly a century. Emetine was first isolated from ipecacuanha in 1817 by Pelletier. The importance of this was first thought to be that as emetine was the emetic principle it could be eliminated and the drug used without it. The results of

this ipecacuanha sine emetine were naturally disappointing, and its use was abandoned. Emetine itself does not appear to have been used often during the nineteenth century. Bardsley, a Manchester physician, referred to its use in dysentery in 1829. According to a French authority, Huard, a Frenchman, was using emetine by injection in 1882, and certainly the following passage appeared in Le Dictionnaire Dechambre in 1887:

> L'émétine est le principe actif de l'ipéca et peut le remplacer dans toutes ses applications. Toutefois, l'avantage d'une pareille substitution peut être contestée. L'action émétique a surtout été recherchée. On la détermine en administrant la substance médicamenteuse en injection sous-cutanée ou en ingestion.

But it is doubtful whether the hypodermic injection of emetine was ever used as a practical procedure until 1912 when Rogers, as he puts it himself, 'decided to try if soluble salts of emetine could be safely injected hypodermically.' This alkaloid had, of course, been used by mouth for some years, but by that route it had all the disadvantages of ipecacuanha. In 1911 Vedder had demonstrated that the lethal action of ipecacuanha on amoebae depended on its emetine content, so that at this time Rogers was on the look out for a suitable case on which to try injections of the alkaloid.

He soon found a desperate case of acute amoebic dysentery and injected one sixth of a grain of emetine (equal to 15 grains of ipecacuanha). As this produced neither sickness nor depression he injected a third of a grain and repeated the dose the following morning. In two days the patient was convalescent and had ceased to pass blood or mucus, and she was discharged from hospital a week later. And that is the first recorded case treated by emetine injections.

In 1912 also Rogers reported successes with emetine injections in amoebic hepatitis and liver abscess. The latter condition he treated by aspiration followed by the injection of emetine hydrochloride into the abscess cavity. He compared a series of cases treated with ipecacuanha with a similar series treated with emetine injections, and proved conclusively the great superiority of the latter even when the total dosage was as little as two grains.

During the 1914-18 War a series of experiments by numerous workers was undertaken to ascertain the mode of action of emetine. Extracts made from the stools of patients receiving ipecacuanha or emetine by mouth were found to have no amoebicidal action. Therefore it was thought the active principle only acted on the amoebae within the tissues of the host and that relapses were due to viable amoebae remaining on the surface of the bowel mucosa. Attempts to administer emetine by enema caused severe pain and a flare up of the

infection. Some other form of emetine was therefore sought which would have an action on the amoebae, whether vegetative or cystic, within the bowel in these chronic relapsing cases. As long ago as 1891 Tull Walsh had treated a case in India with the double iodide of emetine and mercury, but it was emetine bismuth iodide that was now introduced. The action of this drug in these chronic cases is due to the fact that it passes through the stomach unchanged and is split up in the intestines. The bismuth salt is converted to the sulphide and the emetine is slowly liberated on the surface of the bowel.

Pursuing the more recent history of the treatment of amoebic dysentery, one comes to the organic arsenicals. The chief of these is Stovarsol which was introduced by Marchoux in 1922. Marchoux reported successes in the treatment of cyst passers, and later it was used in mild cases of amoebiasis. The toxicity of the drug was said to be a disadvantage. Other arsenicals were later introduced: Treparsol in 1925 and Acetarsol in 1928. Carbarsone was originally prepared by Ehrlich, but it was not until 1932 that it was first advocated for chronic amoebiasis by American workers.

Quinoxyl (Chiniofon, Yatren etc.,) was introduced by Mühlens and Menk who reported excellent results in resistant chronic amoebiasis in 1921. These workers recommended that

the drug should be given in the form of pills, but it was later found to be more efficacious when given as a retention enema.

Conessine, the alkaloid from Kurchi bark, was introduced in 1927. It was claimed to be of value in chronic amoebiasis. The whole bark was an old Indian remedy for dysentery.

Rivanol, like acriflavine a derivative of acridine, was first recommended as a retention enema by Urchs and Peter in 1926. It was later used by mouth. The results in amoebic dysentery were not considered of great value by later workers.

Having sketched the history of the principal drugs which were used in the treatment of amoebic dysentery up to the beginning of the year 1942, it is now proposed to trace the history of the treatment of epidemic - later bacillary - dysentery. Prior to the differentiation of the two principal types of dysentery and, in fact, for some time afterwards, remedies which we now know to be more appropriate to one or other type were, of course, used almost at random. It is, however, significant that ipecacuanha which was found to be in use in a tropical zone among the American Indians, fell into disuse after being brought to Europe, and was later to find its chief protagonists in the tropics; while those who preferred other forms of treatment were usually dealing with epidemics.

Warm baths, rubbing the body with various oils and swathing in wet cloths, sudorifics and diaphoretics all featured in the writings of the ancients among whom the principal object was to open the pores and release the humours. Venesection and the application of leeches had their distinguished advocates during the seventeenth, eighteenth, and nineteenth centuries, and, in fact, they were still in vogue as late as 1860. One early writer claims that he has cured many a case of dysentery with nothing more than a cup of warm water every quarter of an hour, and all who have seen much of the disease will endorse the paramount importance of his remedy. Blisters, wines of various kinds, saffron and musk have been recommended; the 'bark' for many years was used to good effect, but here one naturally assumes that the condition being treated was malaria and not what we understand by dysentery. Antimony, especially in the form of James's powders was largely used in this, as it was in so many other diseases, at the end of the eighteenth and the beginning of the nineteenth centuries.

Opium in various guises played its part for centuries in the treatment of dysentery, as did also numerous vegetable astringents, but as early as a hundred and fifty years ago writers were beginning to sound a note of warning about the use of this type of treatment. Harty referring to opium in 1805 says:

> There are now few authors who do not caution us against it, under different circumstances of the disease, and though they in general agree that it must be avoided in the beginning or before evacuations have been employed, they are not so unanimous as to the extent, or the object for which it should be administered.

and from this somewhat opaque reference one must assume that some of his contemporaries advocated its symptomatic use, while others condemned it. Nevertheless opium was being largely used much later than this, especially in India. Woodward in the American Civil War used it freely at first, but as his experience of dysentery enlarged he was more cautious and only used it for pain or sleeplessness, and here advocated the use of a quarter of a grain of morphia and one hundredth of a grain of atropine.

In 1913 Leonard Rogers was of the opinion that opium should be given in the form of an enema in protracted cases of bacillary dysentery with frequent distressing stools, especially if pain was present between stools. He rarely gave it by mouth, and by this route he advocated its being combined with calomel. Up to the latest editions of his works, Manson Bahr strongly condemns the use of opium as a routine measure. In his view it should only be used to enable the patient to rest or withstand the fatigue of a long journey. When given, it should be in the form of morphia

hypodermically. Felsen, however, regards crude opium as the best sedative. He thinks it should only be used as an emergency measure to relieve or allay intestinal spasm, but never to stop diarrhoea.

Calomel was first introduced early in the seventeenth century by James I's physician, Mayerne. In dysentery it appears to have been first used later in the same century by Andreas Libovius and Robert Boyle. Pringle and Hunter both advocated its use in the eighteenth century. In 1828 Annesley was giving it in heroic dosage. He gave thirty grains of calomel with two grains of opium at night followed by a purge of jalap, castor oil and tartrate of soda in the morning. In chronic cases he continued calomel and opium at night, and in these cases also gave mercury by inunction. In 1841 excessive doses of calomel were condemned by Macpherson, and after that the dosage used became smaller, but in 1860 Morehead was still using it in ten grain doses. In more recent times it has been given in half-grain doses every hour for twelve hours on three consecutive days. Other forms of mercury which have been used are blue pill and the perchloride.

Many clinicians in India seem to have used castor oil at some stage in their treatment of acute dysenteries. Baly used it in doses of one to three drachms in the Millbank Prison, and according to Woodward it was much used in the

American Civil War. Up to modern times it has been a common practice to commence the treatment of the acute case with half an ounce of castor oil - often with the addition of laudanum. Welch and Mascarenhas (1924) and Boase (1925) recorded a reduction in mortality, duration of illness and relapse rate, as compared with cases treated with saline purgatives, in a series of cases treated with repeated small doses of castor oil. A large dose of castor oil was given on the first day (e.g. 2 oz.) and one drachm every hour during the daytime on the second and third days. The death rate in the castor oil group was 2.77% as compared with 8.8% in the saline group.

The principal saline purgatives which are still used in the treatment of dysentery, namely, magnesium and sodium sulphate, were first used in the seventeenth century. The original theory was that a purge at the beginning of the illness swept away the corrupted humours. Donald Munro, writing in 1780 was a strong advocate of the purgative treatment. Above all he recommended the use of saline purges which he repeated on the second, third and fourth day according to the severity of the attack, but he also advised rhubarb. A few years later Rollo endorsed this recommendation, and he persevered with the laxative treatment throughout the illness. After that the salines appear to have been largely replaced by calomel, and in fact Annesley thought they might do harm by

reducing the strength of the patient. Twining also, as has already been mentioned, condemned the use of saline purgatives. These last two writers were probably dealing mainly with amoebic dysentery.

During the American Civil War the saline purgatives returned to favour, the reason being that here it was mainly bacillary dysentery that was being encountered. A similar state of affairs existed in the South African War where the salines were definitely established as the treatment of choice. In other places, such as the Indian jails, where bacillary was the prevailing type of dysentery, the salines maintained their reputation. The routine very widely used up to recent times was to give one drachm of sodium sulphate every two hours for the first twenty-four hours or until the stools became faeculent. Some authors advised larger dosage of sodium sulphate, such as one drachm every hour for the first forty-eight hours.

During the earlier part of this century bismuth in the form of the carbonate or the salicylate was used by many physicians in the treatment of acute dysentery. It was given in drachm doses every three hours. Unfortunately intestinal obstruction with rocks of bismuth and paralytic distension of the small intestine were apt to occur.

Of the absorbents, charcoal was first recommended by

McCabe in 1825, but it does not seem to have been generally used at any time. The dosage was one tablespoonful of powdered charcoal three times daily. Kaolin was introduced much later and was strongly advocated by Austrian and German authors in 1914-15. It was later combined with charcoal in large doses (1½ oz. of each). Colloidal kaolin, or Kaylenol is considered preferable by Manson Bahr.

Rectal injections of various medicaments have long been used in the treatment of dysentery. Annesley (1828) found anodyne enemata useful for tenesmus and he added turpentine if there was much flatulence. Twining (1835), advised cold water enemata in cases with anuria or much blood in the stools, and he also gave rectal injections of lead acetate (10 grains to the ounce). Parkes (1848) gave retention enemata of silver nitrate and nitromuriatic acid in chronic cases. Baly (1847), in chronic cases in the Millbank Prison also used silver nitrate but preferred a few ounces of black wash with laudanum or zinc sulphate. Morehead (1860) thought opium enemata beneficial in relieving tenesmus, but he considered large watery injections disturbing and dangerous. Woodward (1880) gave normal saline at blood heat per rectum. Chevers (1886) condemned the use of large rectal injections as he had seen fatal perforation result. Rogers (1913) advocated large rectal injections (two pints) in acute cases. He used six to ten grains of calcium permanganate to a pint of water. In

chronic bacillary dysentery he recommended astringent retention enemata in the semi-prone or knee elbow position. Silver nitrate and copper sulphate he considered the most valuable astringents.

In recent times the use of rectal injections has been confined to the treatment of chronic cases. Two forms are used, rectal irrigation and retention enemata. Willmore (1918) advocated an 'etherol' retention enema which consisted of 12 ounces of olive oil and six drachms of ether. Camomile (one drachm to eight ounces of warm water) has been used as a soothing retention enema. Manson-Bahr, in many editions of his works right up to 1942, has recommended the use of eusol as a retention enema. He commences with one part of eusol to nine of water and increases the strength over several days until a half and half mixture is reached. Smyly (1930) reported good results with Dakin's solution, the strength increasing from 20% to 50%. Silver gelatose (Albargin) and silver protein (Argyrol and Protargol) in normal saline, bismuth subgallate, suspensions of iodoform in gum, and tannic acid have also been used as retention enemata.

Colonic lavage through an appendicostomy or caecostomy has had its advocates in chronic bacillary dysentery. Gregg (1923) had successful results (in cases which had not responded to other forms of treatment) by resting the large intestine

completely by means of an ileostomy whilst using a caecostomy for daily colonic lavage.

The first to make use of antidysenteric serum therapeutically was Shiga (1902). Although his figures are not considered convincing by statisticians, he claimed to have reduced the mortality from 22% to 7% by the use of his serum. Rosenthal working in Moscow in 1903 and Todd in London in 1904 independently prepared and used therapeutically antitoxins contra the Shiga-Kruse bacillus. Rosenthal claimed that the mortality fell in Moscow from 17.5% to 4.5%. The disadvantage of being unable to identify the causative type of dysentery bacillus early in the attack, led to the introduction of a polyvalent antidysenteric by Ruffer and Willmore at the El Tor quarantine station in Egypt in 1909. During the 1914-18 War a polyvalent serum was the type generally used. Waller (1919) in Mesopotamia and Klein (1919) in France treated over 1300 cases with Lister Institute polyvalent serum which they administered subcutaneously. They gave 120 to 140 cc. divided into three doses eight hours apart. If first administered after the seventh day of the illness serum had less effect. Lornie and Jones (1922) gave 20 to 40 cc. repeated daily for four days. The intravenous route was first employed in very acute or fulminating cases by Manson-Bahr in 1912. The advantage of this route was largely outweighed by the risk of

anaphalaxis. Graham (1918) treated 2500 cases with antidysenteric serum given intravenously followed by 150 to 300 cc. normal saline. The saline was repeated twice daily for two days and thereafter once daily for two days. Beyer (1918) used the intramuscular route for doses of 10 to 20 cc. daily. Lantin (1921) reported favourable results from injecting the serum into the bowel per rectum. Knauer (1926) gave intraperitoneal injections of large amounts (100 to 300cc. daily) of serum in infants and children.

In recent years great improvements have been made in the preparation of a concentrated anti-Shiga serum containing 50,000 international units in less than 10 cc. It is an established fact that serum therapy is less effective in Flexner infection than in Shiga infections (Nolf, 1918-19; Job, 1921; Stawell, 1921) so that the view held at the early part of the recent war was that it was better to have an efficient anti-Shiga serum at hand than a polyvalent serum which only contained small amounts of Shiga-immune bodies. However, the modern anti-Shiga serum has proved to have a purely antitoxic effect and this is somewhat short-lived. In selected cases it was given intravenously with full precautions against anaphylaxis as early as possible (50,000 to 100,000 i.u.) and thereafter was repeated by the subcutaneous route once or twice daily.

That experiment should be made with the sulphonamide group of drugs in the treatment of bacillary dysentery was inevitable. By 1938 it was established that these compounds would inhibit the growth of both the Shiga and Flexner types of B. dysenteriae in vitro. O'Brien (1939) described an unpublished research by Buttle and Parish in which mice experimentally infected with Flexner and Sonne bacilli were treated with sulphanilamide by mouth. In the group in which 1,000 m.l.d. was used half of the treated mice survived, whilst all the untreated controls died.

Welch, Meyer and Smith (1940) reported a series of cases of acute bacillary dysentery in infants in Alabama treated with sulphapyridine. It appears that Smith, who was an interne in a Childrens' Hospital, first used sulphapyridine in desperation, in June 1939, in an apparently moribund infant. The result was astounding. The child, after having been desperately ill for four weeks with acute bacillary dysentery, recovered with such rapidity that it was discharged from hospital one week after commencing the sulphapyridine. After this spectacular result the authors treated twelve other cases, eleven of them with like success.

In 1941 many articles on the chemotherapy of bacillary dysentery were published. Reitler and Marberg in Palestine treated 20 adults with acute bacillary dysentery with sulphapyridine with uniformly good results. The infections were

mainly due to Shiga, Flexner-Y or Sonne strains. 1gm. of sulphapyridine was given four times daily for two to four days. The temperature fell almost immediately and the motions became formed in 48 hours. The same authors stated that this drug was useless in amoebic dysentery. Similar results were obtained by Bell in a small outbreak of Flexner and Sonne dysentery in England. He used a total of only 5 gm. of sulphapyridine per case. Ching, Warr and Witherington had published a preliminary report on the use of sulphathiazole in dysentery in 1940, and in 1941 they published case records of 19 cases of acute bacillary dysentery treated with this drug. Two patients who had already contracted peritonitis died; the remaining 17 recovered extremely rapidly. These cases were in Tennessee. Another series from the Southern States was that of Ravenol and Smith in South Carolina. Their cases were children and they used both sulphapyridine and sulphathiazole with equally outstanding success. Masefield in a Mental Hospital in England reported a series of cases of Flexner dysentery treated with sulphapyridine. All cases were free from infection on the fifth day.

Early in the history of the chemotherapy of bacillary dysentery it was considered that compounds such as sulphapyridine and sulphathiazole were dangerous because they are readily absorbed from the gut and may produce severe toxic affects on

the kidneys if used in full dosage in a patient who is already dehydrated with, perhaps, oliguria. A further disadvantage is that they are gastric irritants and may produce severe vomiting in a gastro-intestinal upset such as bacillary dysentery. For these reasons Marshall and his colleagues in Baltimore commenced their search for a sulphonamide compound which, although efficient as an intestinal antiseptic would be poorly absorbed from the gut. After preparing numerous compounds they chose sulphaguanidine (sulphanilylguanidine) as the most suitable. In 1940 they described the preparation of this drug, its pharmacology and its action in laboratory animals. In the following year they reported a series of clinical tests of sulphaguanidine in dysentery cases. The drug was no less toxic than sulphapyridine, but owing to the small amount absorbed its toxicity could be disregarded. Absorption only took place from the small intestine, and the absorbed fraction was quickly excreted by the kidneys. For this reason, in order to attack an infection in the colon, large quantities of the drug had to be given. The dose recommended was 0.1gm. per kg. of body weight as an initial dose, followed by half that quantity every four hours until the number of stools dropped to four daily, and after that the same dose eight hourly.

Lyon (1941) described the results of the use of sulphaguanidine in a series of 23 cases of bacillary dysentery as compared with a similar number of untreated cases. Improvement was noted in all but five of the treated cases in 24 to 72 hours. Later in the year Lyon, Folsom, Parsons and Sprouse published a study of 259 cases of acute bacillary dysentery in which their results were similar. They added the recommendation that if the sulphaguanidine treatment was not effective in 48 hours, sulphathiazole should be administered for five days. Anderson and Cruikshank (1941) published a series of 41 cases of Flexner dysentery in a mental hospital in England treated with sulphaguanidine, 55 untreated cases serving as controls. Apart from the beneficial effect of the drug in curtailing the acute stage of the disease they found that sulphaguanidine prevented the convalescent carrier state which occurred in about half the untreated group. These authors used smaller doses of sulphaguanidine than those previously used. They gave 3gm. three times a day for two days followed by 2 gm. twice a day for two to five days.

*In January 1942 there appeared in the Lancet a preliminary report from Hamilton Fairley and Boyd on the treatment of bacillary dysentery by sulphaguanidine in the Middle East. In their series the drug was reserved for severe acute or persistent chronic cases. The series included in all 371 cases of which 135 were Shiga infections. The authors commented on the rapid relief of the abdominal pain and tenesmus; the early disappearance of blood from the stools; the fall of the temperature to normal within three days and the fall in the number of stools to one or two daily in five to six days. They emphasised the importance of sulphaguanidine being used early in the disease. They did not include a large number of chronic cases in their series. Only 12 cases had been ill for 21 days or more before the commencement of sulphaguanidine. In this group they recorded three failures and nine successes. In the view of these authors the action of sulphaguanidine is either bacteriostatic or bacteriocidal. While leading to the early cessation of damage in the gut and the immediate decrease in the production of toxin, it appears to exert no effect on the exotoxin already absorbed and circulating in the blood. Therefore they

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* This paper which is included for the sake of completeness could not have reached Malaya before the fall of Singapore as the last ships to arrive docked on February 5th, having taken over two months to come from Britain.

recommended the combined use of sulphaguanidine by mouth and Shiga antitoxin by the intravenous route.

Such, then, is the history of dysentery and of its treatment up to the beginning of 1942. The foregoing represents, more or less, a summary of the total knowledge in that sphere which was available (but not necessarily possessed by) those who were at that point sealed off from civilisation - cut off from the interchange of medical ideas and advances without which modern medicine cannot flourish freely. What ensues was undertaken solely with the object of helping companions in misfortune to survive a prolonged ordeal and not consciously as a contribution to increased medical knowledge.

CHAPTER II.

THE BACKGROUND.

On the 15th of February, 1942, the British Commander in Singapore surrendered to the Japanese with the result that 53,000 white troops (British and Australian) became Prisoners of War in the hands of a largely uncivilised military force. It was in this community that the work to be described was performed. The population varied within wide limits as parties were sent off from time to time to work on such undertakings as the Burma-Siam railway, whilst incoming parties from Sumatra and Java, Burma, etc., composed of British, Australian, American, Dutch and Eurasian troops arrived in the camp at intervals throughout the period of captivity which was over $3\frac{1}{2}$ years.

Immediately after the surrender of Singapore the white troops (with the exception of those in hospital) were ordered to proceed on foot to a peninsula at the North East corner of Singapore Island, known as Changi. No provisions were made for their reception. This area contained a British barracks, but all sewers and water and electricity supplies had been destroyed. The men who were sent to occupy this peninsula were demoralised, defeated troops, with apparently nothing to look forward to but years of imprisonment. Under these circumstances

discipline was naturally strained - in fact in certain respects it completely broke down. Unfortunately this was most marked in the sphere of hygiene. Although discipline and morale were restored in the course of the next two or three weeks by the efforts of British officers - efforts which were aided to an enormous degree by the natural cheerfulness, optimism and decency of the British soldier - the damage done by the short interval in hygiene discipline was considerable. The area had become soiled in many odd corners. What latrines had been made were inadequate and exposed to flies. Many of the men were already developing dysentery on arrival, and some were convalescent. As soon as a man was fit to walk (long before he was fit to march the 14 miles to Changi) the Japanese insisted on his being discharged from the hospitals which remained in Singapore. The result was that many early dysenteric convalescents arrived in the Camp in a state of collapse and often had a recurrence of diarrhoea. Such men arrived late at night. They did not know where the latrines were and were too tired to care.

By the end of February, 1942, all the British Military Hospitals in Singapore had been moved out to Changi. The move, carried out under the orders of an unsympathetic and victorious enemy, was bound to cause hardship among the patients. Few, if any, arrangements had been made for the reception of over

two thousand patients. The so-called fit troops had only just been moved out of the area which was to form the main P. O. W. Hospital. The area in itself, known as Roberts Barracks, was quite suitable, as it contained several concrete barrack blocks close together. Each block had three large barrack-rooms on three separate storeys. These barrack-rooms in "Peace Time" accommodated forty healthy soldiers. In the P. O. W. Hospital it was necessary to put 140 or more patients in each room. At this time there was no water laid on, no sanitation and no electricity. The area, as has already been mentioned, had been widely contaminated. Our engineers were able to fix up an emergency water pipe-line and provide water points at a few places outside the buildings, but it was months before the internal water supply, main drainage, or electricity, were available. Work was immediately put in hand to provide latrines; Otway pits for excreta, for dressings, for cook-house refuse; soakage pits, etc., but such work took some considerable time when one's labour force consisted of tired, undernourished men working in an equatorial climate. Even when the pits were dug, the problem of fly-proofing had to be solved. The only source of timber was the hospital buildings themselves from which doors and other woodwork had to be ruthlessly stripped. Other materials required for the urgent fly-proofing were only provided by destroying parts of our

own buildings. The less essential had to give place to the essential.

When the hospitals first moved into the area the fly menace was already out of hand. Fly destruction was carried out on as large a scale as possible by everyone capable of moving an arm, but it was quite impossible to keep food continually protected from flies. Among the patients who came out from the hospitals in Singapore were some 1500 serious battle casualties. Unfortunately the reduced diet, which at that time consisted almost entirely of 16 oz. of rice a day and some tea, had an adverse effect on many of the more seriously wounded, and in their weak state they were easy victims for the dysentery bacillus. During the latter part of February and the beginning of March 1942, the incidence of dysentery increased to such an extent in the P.O.W. Camp generally and also among patients already in hospital that it was necessary in the middle of March to open a special wing of the hospital to deal with dysentery cases. The epidemic grew so quickly that within a few days of opening the Dysentery Wing it contained over 800 patients, and had to be staffed with the entire R. A. M. C. personnel of the three Field Ambulances belonging to the 18th Division. Although this represented over 300 men the Wing was by no means over-staffed. Most of the patients were bed patients as the large number of admissions each day forced Medical Officers to discharge men from hospital

almost as soon as they could walk. Every drop of water for washing, drinking or ward cleaning, had to be carried from outside. All food had to be brought from cookhouses at a distance. All excreta, waste water, etc., had to be carried down to disposal pits at the furthest possible point. Corpses had to be carried down to the mortuary (fortunately the burials were done by the deceased's own Unit in the main P. O. W. Camp.) Each ward might discharge and receive from fifteen to twenty cases daily, and at that time there was no ward which had not several cases on the Seriously Ill and Dangerously Ill lists, and such cases required constant care. Many of them might be having continuous drip salines or blood transfusions. About half the personnel, of course, were not employed in wards at all, but on outside duties. All rations and stores had to be dragged on a "trailer" (the chassis of a lorry stripped of all non-essentials - such as the engine, there being no petrol in the P. O. W. Camp) from the "ration point" two miles away. Wood for cookhouse fires was brought into camp in a similar fashion. The sanitary squads digging latrines and pits represented a large number of men. Men had to be found for cookhouses for patients and staff, for wood chopping, and for work in the rice-mill where rice was made into flour (these rice-mills had to be designed and made of material available in the camp itself). All this work had to be carried out by semi-starved but ever-

willing R.A.M.C. orderlies, who throughout 3½ years of captivity deserved, with few exceptions, unstinted praise.

The diet in these early days consisted almost entirely of 16 oz. of rice daily. No other method of cooking was possible at first but boiling, and this produced a monotonously unappetising dish which was consumed with difficulty by even the healthy. The only vegetable available was unripe Papaya. After the initial period of a few weeks things improved and for a few months small quantities of meat were supplied, but this was soon replaced by fish which gradually dwindled. Generally speaking the only protein in the diet for 3 years were minute quantities of fish which more often than not was bad. After the first few months it was possible for the camp to buy oil for cooking purposes, and the diet was then made more appetising by part of it being fried. This oil was a Red Palm Oil which was said to be unfit for human consumption as it contained free fatty acids. It was, however, consumed by thousands for 3 years without noticeably harmful results. As time went on the rice ration was gradually reduced until during the last year of captivity it was about 8 oz. per head per day. The vegetable position, on the other hand, was improved by the cultivation of gardens by the prisoners. Towards the end of the period of captivity, the Hospital had between 20 and 30 acres under cultivation.

The diet was seldom augmented by Red Cross supplies.

In the second half of 1942 valuable supplies did arrive on the return of Japanese ships from neutral ports after the repatriation of Allied Diplomatic staffs. The most valuable items were corned beef, Marmite and some drugs. These supplies were mainly reserved for hospital cases. The amount of corned beef, for instance, which it was possible to issue to all ranks in the camp was, speaking from memory, equivalent to three 12 oz. tins per head. The use of this quantity in the cooking was spread out over some months so that the amount it was possible to use at a time was virtually little more than a flavouring agent. In addition to this each man got half a tin of jam and a few other odds and ends. It is perhaps fortunate that it was not realised at the time that no more Red Cross supplies would be available for years.

It was possible, however, to store quite appreciable amounts of corned beef and Marmite for hospital patients. These stores were reserved for the worst cases with such care that it was still possible during the latter part of 1944 to procure a little extra nourishment in the form of corned beef for an extremely serious case of malnutrition. (Of course, Medical Officers' standards of what was considered malnutrition progressed with the passage of time. This will be obvious when one considers that what was regarded as serious malnutrition in

1942 was the general state of everyone in 1945). Those who were responsible for the inclusion of Marmite in that shipment of Red Cross supplies earned the gratitude of all Medical Officers. This valuable foodstuff saved thousands from death, blindness and paralysis, but unfortunately many for whom it was not available perished or were crippled.

No further Red Cross supplies arrived until the end of 1943 when very small quantities of American Red Cross parcels were permitted to enter the camp. These parcels were the normal weekly P. O. W. parcels. The number of parcels was only sufficient to divide each parcel among seven men. The tins of powdered milk in the parcels were removed before the general issue took place. This powdered milk was conserved until 1945 when it was of great value in the treatment of cases of famine oedema. No further Red Cross supplies entered the camp until early in 1945 when a similarly small quantity was issued. In August 1945 after the Japanese had surrendered they released parcels which had been in Singapore since 1942.

The labour conditions for British and Australian Other Ranks were always severe. The type of work they were forced to perform was that usually done in the tropics by coolies. They had to work in the heat of the tropical sun out of doors. Many of them had no hats or protection for their eyes. As time went on the only clothes they had were scanty loin cloths.

During 1942 thousands of men were removed to working camps in the city of Singapore where they worked on reconstructing the city after the raids, the camp in Changi serving as a labour pool and as a hospital. The sick were brought to Changi in Japanese lorries at irregular intervals, and, although many men were seriously ill before being evacuated from the Working Camps, generally speaking at that time men requiring hospital treatment were brought to Changi without too much delay. From the end of 1942 onwards large parties were moved under appalling conditions either by train to Siam, or by ship to Formosa or Japan. The tragic story of those who were taken to Siam to work on the Burma-Siam Railway is already well-known. The survivors - many of them moribund - of the two most disastrous of these parties came back to Changi Hospital at the end of 1943. During 1943 the Japanese commenced the construction of a large aerodrome at Changi, and thousands of prisoners of war were employed as labourers on this work, where they had to work for 10 to 12 hours daily unprotected from the sun. During the latter half of 1944 and the beginning of 1945 the Japanese began once more to establish Working Camps in and around the city of Singapore. The work now was the construction of defensive positions. The conditions of work in Singapore in 1945 were terrible. Men were, of course, far less fit than they had been one, two or three years before. They had one

day off in ten and apart from this day were never in their camps during the hours of daylight. They had to work extremely hard mainly on work which was particularly trying to the nerves, such as tunnelling in clay without rivetting or pit props. The food situation in Singapore had been deteriorating by this time for three years and was much worse here than in other parts of Japanese occupied territory. The conditions in the Singapore working camps by the middle of 1945 were similar to those of the Siam camps in 1943. Seriously ill men were forced to go out and work. Those who were evacuated to hospital were often moribund on arrival. In one camp of 180 men, 120 were so ill that they could not walk, and one unfortunate R.A.M.C. corporal had to care for them as best he could.

In September 1942 an event took place which profoundly affected the incidence of sickness. The Japanese ordered all prisoners of war to sign a document stating that they would not attempt to escape. There were about 20,000 men in Changi at the time and all but three refused to sign this document. The Japanese ordered the entire camp into a small barracks at Selarang (2 miles from Changi) which normally housed one battalion. After strenuous protests had been made they permitted the hospital to remain in Changi (probably because of the difficulty of moving it and not because of the hardship which it would have entailed). However, 17,000 troops were accommodated

in one barrack square and the buildings around it. They had to move at short notice with what they could carry. Latrines had to be dug in the asphalt square, and cookhouses put up in the same place. Barrack rooms which normally housed 40 men now housed 800. Even the roofs of the buildings were covered with men. The whole barrack square was surrounded by machine guns and searchlights. Up to this point the number of cases of dysentery in the hospital had been gradually falling and had reached the low level of 230. A few days after the beginning of the Selarang episode the number of cases had risen to 830. In addition to this mounting toll of sickness the Japanese refused to issue rations and when the food which the troops had been able to carry into Selarang was exhausted there was no more. Under these conditions of duress the senior British officer ordered all prisoners of war to sign the document, and the troops were allowed to return to the camp at Changi.

In August 1943, as a result of the construction of the aerodrome in Changi, the main camp was moved to the area surrounding the Selarang barracks referred to in the previous paragraph. The hospital was once more accommodated in barrack buildings similar to those occupied at Changi. In April 1944 the main camp was closed down and 8,000 prisoners of war were accommodated in Changi gaol which has a peace-time capacity of less than 2,000 Asiatic convicts. Until this point the gaol had

been occupied by 2,000 civilian internees who had just been moved to a camp nearer the city of Singapore. In the following month the main P.O.W. Hospital was moved by road a distance of 22 miles to Kranji, near the southern end of the causeway which connects Singapore Island to Johore. The hospital, which at this time had 2,000 patients, was accommodated in a rubber plantation. The camp consisted of really nothing more than a collection of dilapidated huts made of rush-work sides and roofs on wooden frames. Most of these were on the point of falling down and there was no material available to repair them. The roofs leaked so much that most men preferred to go underneath the floor of the hut when it rained (the huts being built on concrete piles). This procedure was not possible as time went on as it was later necessary to accommodate patients permanently under the huts, owing to the fact that half of the accommodation was taken away and wired off to house a working camp which was constructing fortifications in the area.

An interesting point on the Japanese outlook on sickness is that they refused to give hospital patients as much food as those who were working. The Hospital Staffs officially drew the working scale of rations but it was naturally necessary to pool resources in order to augment the patients' very meagre ration. When the hospital formed part of a huge camp this was no great hardship, but unfortunately in 1944-45 when rations were being

cut drastically every few months the hospital was a self-contained unit at Kranji, and the staff of 300 or so had to carry 2,000 patients. The result was that the R.A.M.C. orderlies were on a much lower ration scale than men in working camps. Most of them, of course, did not have to work as hard as men in working camps, but there was naturally a great deal of hard manual work to be done in a hospital camp of 2,500 living under primitive conditions. The system was that the hospital work was done in the mornings, and in the afternoons the ward staffs were reduced to a bare minimum, while the rest, medical officers and orderlies alike, felled trees for cook-house fires, worked in the vegetable gardens, pulled 3-ton trailers to fetch supplies etc. The deterioration in the men's condition can be judged by the fact that a trailer which 16 men could easily pull in 1944 required 36 men for the same journey with a similar load in 1945.

Owing to the shortage of hospital staff, which was constantly being reduced by the Japanese demanding more workers in their working parties, convalescents and up-patients suffering from chronic disease of long standing had to work within the hospital camp. Only very light work could be done by these men as the number of calories in the diet was so low that they could not get better if they expended much energy. Light ward duties, picking and preparing vegetables (provided they were not

suffering from bowel diseases), tapping rubber trees, making brooms and brushes, and light jobs in the gardens were all suitable work for them. Great benefit was derived by many patients from this compulsory work. In the dysentery wards patients who had suffered for months or even years from diarrhoea which had resisted all treatment, improved remarkably when they were put to work. A man who had had never less than seven bowel actions daily for many months would be reduced to two or three when his body and mind were occupied with other pursuits. This type of occupational therapy was of great value in the non-specific diarrhoeas of which there were large numbers, but was not only useless but harmful to those suffering from bacillary or amoebic dysentery or pellagra.

Clothes were a great problem as time went on. Most patients on admission to hospital during 1944 or 1945 had no clothes at all but a piece of cloth about one foot by two feet which they wore as a loin cloth. They usually had no more than a scrap of torn blanket or a piece of floor carpet to cover themselves with at night. Very few men had hats or boots. Many had no footwear at all. In the hospital, which from May 1944 onwards was in a rubber estate, we ran a rubber factory where sandals were made and every effort was made to provide a man with footwear before he left hospital. To clothe them all was impossible, but by taking into the store all kits of

deceased patients and spare garments from all who had more than one pair of shorts, an attempt was made to give men a pair of shorts or pants before they returned to their working camp. Towards the end most medical officers had one pair of much-patched shorts which they wore for their ward rounds in the mornings. Their shirts had long since fallen to pieces or had provided the patches for the shorts so that professional attire consisted entirely of a pair of shorts and a stethoscope. Less formal attire was worn during the afternoons when doing manual labour.

When Singapore fell in 1942 the Base Medical Stores like everything else, fell into the hands of the enemy, who seized 50% of all stocks on charge. Fortunately large stocks of many important drugs were held (for instance a quarter of a million tablets of M & B. 693 found its way to Changi). Individual Medical Units were not searched immediately, and drugs and instruments from them were conveyed to Changi in their entirety and were there buried until after the Japanese check-up had taken place. But even large stocks of drugs were inadequate to provide for a large population cut off from all sources of supply. All parties leaving the main P.O.W. camp had to be provided with their quota of the bulk stores. Rigid economy was practised throughout the $3\frac{1}{2}$ years with all drugs in short supply, and to foster economy a system of counter-signature

by a senior medical officer was instituted (the doctor in charge of the case is always apt to think his own patient more deserving than problematical future patients). The only drugs supplied by the Japanese in appreciable quantities were quinine sulphate for malaria and magnesium sulphate for dysentery. At a time when there were large numbers of severe amoebic dysenteries the Japanese responded to frequently repeated appeals for emetine by issuing twenty tablets of one third of a grain each. This was the only occasion on which they supplied any anti-amoebic drug. The amount of drugs from Red Cross sources which were permitted to enter the camp were on the whole negligible. Small supplies of Sulphaguanidine arrived in the 1942 Red Cross consignment and again from American sources in 1943. From then onwards Red Cross supplies were not permitted to enter the camp in useful quantities until August 1945. In these circumstances treatment depended entirely on what was available or could be spared from carefully hoarded but rapidly dwindling stocks, or on what could be manufactured from materials inside the camp. Castor oil was made from home-grown seeds; ferrous sulphate was prepared from sulphuric acid and old iron; watery extracts were made from rice polishings which the camp was able to purchase on rare occasions, and from various inedible grasses and leaves for the treatment of deficiency diseases. There was fortunately within the Hospital Camp at

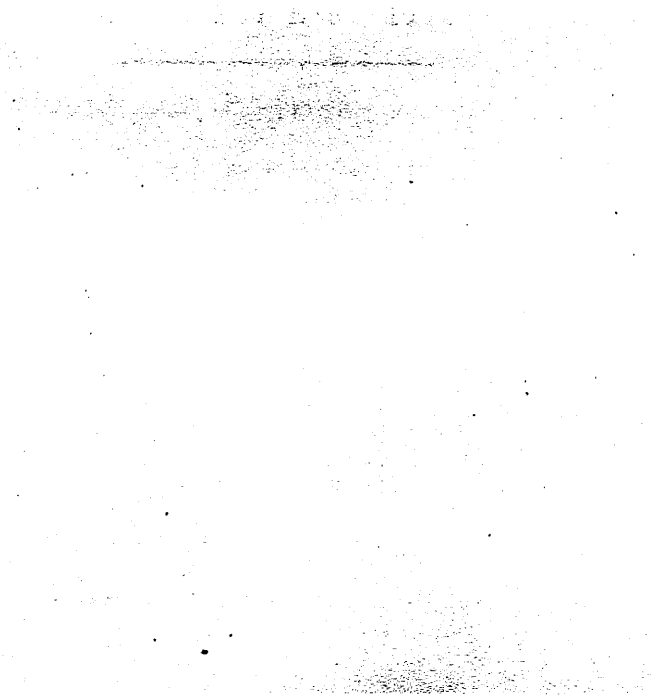
Kranji a small area in which the sub-soil was a good quality china clay (this fact was discovered accidentally when digging latrines) from which it was possible to prepare Kaolin powder which was used extensively in the treatment of bowel and skin diseases. These are but a few of the locally prepared drugs. Economy and improvisions were the order of the day. The principal British and Australian Military Hospitals had contrived to move to Changi the X-ray plants complete with generators, and the entire contents of the operating theatres including operating tables and lighting units. X-ray films had a short life in the hot damp climate and after the first few months screening alone was possible and this was on a very limited scale after the tubes of the larger machines had given out. From 1943 onwards the only radiography possible was a five second exposure on a fluorescent screen which had been fitted to a small portable X-ray plant. Major surgery of every sort was performed by senior British and Australian surgeons, and once inside the operating theatre it was easy to imagine that one was in civilised surroundings as there was nothing to be seen that would have been out of place in a modern hospital.

Throughout its three and a half years of existence the Singapore P.O.W. Hospital had efficient laboratory facilities directed by experienced pathologists. Post mortem examinations were performed on all fatal cases after the first month or so.

Unfortunately there were certain types of laboratory tests which could not be performed for lack of materials and apparatus. For this reason it was never possible to carry out serological typing of B.Dysenteriae. Microscopic examination of stools of all cases were performed and at first diagnosis depended largely on this. As a diagnostic aid the present writer set up a Sigmoidoscopy Clinic in May 1942, and between then and September 1945 performed close on 6,000 sigmoidoscopic examinations. Most of the work of this clinic was consultative, cases being sent from all parts of the hospital. More will be said of this work, which was carried out in addition to the writer's duties as Medical Officer in charge of dysentery wards.

The foregoing description of the Singapore P.O.W. Hospital may give some rough idea of the conditions under which the work to be presently described was carried out. Unfortunately the exigencies of the situation resulted in the loss of valuable records which had already been collected or prevented the compilation of statistics which might have enhanced the value of the present work. For instance much material which had been collected between 1942 and 1944 was lost when the hospital was moved by the Japanese under chaotic conditions in 1944. The keeping of diaries or personal papers was forbidden, and frequent searches for secret documents were carried out by Japanese guards who could not read even their own language. In these circum-

stances medical records were apt to be taken away as incriminating evidence and therefore had to be hidden. As time went on the shortage of paper itself rendered the keeping of adequate case records impossible. Nevertheless it was possible to preserve a not inconsiderable quantity of material, and the gaps have been largely filled in from memory since returning to this country.



CHAPTER III.

ACUTE BACILLARY DYSENTERY.

The largest epidemic of acute bacillary dysentery to occur in the Singapore P.O.W. Camps was the initial outbreak. The Dysentery Wing of the hospital was opened on 11th March, 1942. Two days later the total number of in-patients was 550. The number rose to 800 during the next two weeks and remained at that level for two months, after which the total gradually fell. From June to August the total was between four and five hundred. During the first five months 10,000 were admitted to the Wing: the deaths numbered about 120 and the chronic cases 100. At the beginning of September the Selarang Episode caused a sharp rise in the number of cases of acute bacillary dysentery. The record total of 830 patients in the Dysentery Wing was then reached. This epidemic was not sustained to the same extent as the previous one and by mid November there were only 290 cases in the Wing including 30 amoebics and 90 chronic bacillaries. At the end of 1942 parties began to arrive from Java, and in these many already dangerously ill with bacillary dysentery on arrival. This caused a fresh epidemic in Singapore during which the largest number of cases in hospital was between 400 and 500. The total number of cases admitted in 1942 was 15,000 and the deaths numbered 245.

During the first half of 1943 the average number of cases

in the Dysentery Wing was 200. When the hospital moved to Selarang in August 1943 there were only 140 cases of dysentery, including amoebics and chronic bacillary cases. At that time acute bacillary dysentery was only occurring as a sporadic disease - only one or two cases a week being admitted. In December 1943 about 100 extremely severe dysentery cases of all types arrived from the Burma-Siam Railway working parties. During the first half of 1944 the total gradually fell and only 40 amoebic cases and 40 chronic bacillary cases were in the hospital when it moved to Kranji at the end of May 1944. From then until September 1945 most of the acute bacillary cases were dealt with in Camp Hospitals at the various Working Camps. At the Main Hospital in Kranji the number of acute bacillary cases in hospital at one time averaged about ten during that period.

All these figures exclude Australian cases, as these cases, apart from those referred to the Sigmoidoscopy Clinic for an opinion, were always treated in a separate Australian Wing.

The severity of the cases in the early epidemic of bacillary dysentery was extremely variable. All types from ambulant cases having a few loose stools daily to choleraic cases in which death occurred in two to four days were seen. It was not possible to estimate the incubation period as the entire community was in more or less constant contact with infection from some source.

Symptoms and Signs.

About 75% of cases of acute bacillary dysentery started with the sudden onset of diarrhoea usually immediately preceded by acute abdominal pain. Of those which had prodromal symptoms, the commonest were fever and headache. The following table indicates the incidence of symptoms which occurred prior to the diarrhoe in 42 uncomplicated fatal cases. The length of the prodromal period varied from 12 hours to 6 days. In the majority of these cases the onset of diarrhoea was during the first or second day, but in no less than six cases no diarrhoea occurred until the fourth day. The second table, which only covers 38 of these cases, shows the day on which diarrhoea first appeared.

Table 1. Incidence of Pre-diarrhoea Symptoms in 42 fatal cases of acute bacillary dysentery.

Symptom.	Cases.
Fever.	21
Headache.	17
Abdominal pain and colic.	10
Vomiting.	7
Pains in back.	7
Pains in legs and muscular pains.	7
Pain behind the eyes.	7
General malaise.	4
General weakness.	4
Nausea.	4
Rigors and shivering.	4
Anorexia.	3
Sore Throat.	2
Pain in left iliac fossa.	2
Dizziness and fainting.	1

Table 2. Day of onset of diarrhoea.	Cases.
1st.	11
2nd.	18
3rd.	1
4th.	6
5th.	1
6th.	1

The majority of cases of acute bacillary dysentery were ushered in by a sudden severe abdominal pain immediately followed by diarrhoea. The site of the pain varied and a study of the post mortem findings brings one to the conclusion that this was influenced by the part of the bowel affected by the infection. In the early stages, and in fact throughout the course of infections in which there was no peritoneal irritation, the pain was referred, as far as one can ascertain, to the anatomical site of the lesion. For instance, pain due to a lesion in the transverse colon was referred to by the patient as a pain across the upper abdomen. The initial diarrhoea was not usually accompanied by the passage of blood and mucus. The time at which blood and mucus first appeared seemed to depend firstly on the amount of faeces present in the bowel at the onset of the illness, and secondly on the site of the initial lesion. For example, in a very severe acute infection affecting the ileum alone, the patient died before any blood and mucus appeared in the stools. Infections affecting the ileum were far from uncommon. This was usually shown by central abdominal or

right iliac fossa pain and abdominal distension.

The pre-diarrhoea pain was of a griping character and was repeated before each stool. It is probable that this type of pain was due to the affected part of the bowel going into spasm as a result of the irritation caused by the bowel contents passing over the inflamed or ulcerated mucous membrane. One has observed this spasm of the bowel while doing sigmoidoscopies, and on these occasions the patients have complained of griping pain and an irresistible desire to pass a motion. In one case in which the sigmoidoscope caused such a contraction of the bowel the resulting griping pain lasted for half an hour.

Although some authors draw a clear distinction between the griping pain prior to defaecation and tenesmus (a painful squeezing, bearing-down sensation occurring during or immediately after defaecation) it was difficult to distinguish one from the other in cases in which the lower bowel was the seat of infection. In these cases the two types of pain were apt to be continuous one with the other. In other cases in which more proximal parts of the bowel were involved, although tenesmus was not present at the beginning of the attack it was usually a prominent feature of the disease by the time blood and mucus appeared in the stool. It seems probable that the presence of tenesmus in cases in which the infection does not affect the rectum is due to the irritation of the rectal and anal canals by the alkaline stools and the

congestion caused by frequent defaecation. In many cases a vicious circle was set up in which the constant straining produced by the tenesmus resulted in anal prolapse which in turn increased the tenesmus. Haemorrhoids were also a very common complication brought on by constant straining. Most cases of chronic dysentery which came later to the Sigmoidoscopy Clinic had some degree of haemorrhoids or prolapse.

The intensity of the diarrhoea as measured by the number of stools a patient might have in each twenty-four hours was not always a true indication of the severity of the case. Some patients would exert will-power and lie in agony resisting the almost irresistible urge to stool, while others would get up at each twinge of pain. As far as possible patients were encouraged to resist the urge for the sheer bodily fatigue of getting in and out of bed was a factor which told against them. In nearly all fatal cases the number of stools decreased before death as the patient became more toxic. In cases in which peritonitis supervened there was always a reduction in the intensity of the diarrhoea. With these exceptions, however, generally speaking the severity of the case was in proportion to the number of stools. Mild cases might have six to twelve stools a day; moderately severe ones from 20 to 30; and severe cases anything from 40 to 60. There were also the very severe cases in which over one hundred stools in 24 hours were

recorded, and, of course, those whom continuous tenesmus kept constantly on the bed-pan passing small quantities of blood and mucus.

Fever was a constant feature of all but the mildest cases. It was often difficult to distinguish between acute bacillary dysentery and malaria as the latter cases sometimes had diarrhoea with blood and mucus, and shivering and rigors occurred in the former. It was of course a rule that all cases with pyrexia should have a blood smear examined for malarial parasites. Temperatures of 105°F. or 106°F. were seen even in uncomplicated cases of bacillary dysentery. The taking of temperatures was only possible as a routine in the early days. Thermometers were irreplaceable, and as time went on it was necessary to limit their use to special cases. During the last one and a half years of captivity there were no thermometers left and one had to rely on one's sense of touch and other signs in estimating pyrexia.

In the early stages of the disease pyrexia was associated with profuse sweating, but in severe cases the skin later became dry and harsh. Severe cases had often a drawn anxious expression and haggard appearance and the worst cases with profound toxaemia exhibited a purplish cyanosis. Great emaciation was produced in quite a short time even in the early days before the effects of chronic starvation were generally noticeable. In such cases the

flesh seemed to fall away from the face with alarming rapidity and in a very severe infection the bones of the face were outlined after only a few days of illness. The abdomen rapidly became scaphoid and the ribs stuck out to such an extent that one often had difficulty in placing a stethoscope flat on the chest. These severe cases suffered a great deal from cramps in the limbs probably as a result of salt loss due to sweating and the frequent alkaline stools. Actual tetany occurred in the later stages of two fatal cases. Dysuria or strangury often became extremely distressing as dehydration increased. Very severe cases often had a characteristic sour, sweetish, decaying smell which appeared to emanate from the serum and necrotic material pouring from the bowel. In time one came to regard this smell as being of grave prognostic significance. In fact, no case in which this sign was observed by the present writer recovered. At post mortem such cases were found to have a very large percentage, if not all, of the mucous membrane of the colon completely destroyed.

In a severe dehydrated case the tongue was dry and furred, and often brown; the lips were dry and cracked. Although the appearance was that of extreme emaciation the skin was not stretched tightly over the prominent bones as in cases of chronic starvation, but was loose and inelastic so that large rubbery folds of skin could be picked up. These folds did not

fall back into position immediately on releasing them. During the height of the illness the expression of the eyes was often tense and anxious, but as toxaemia increased and the patient became apathetic the eyes became vacant and glazed and lacked lustre. The sclerotics were injected and the palpebral conjunctives red even in an anaemic subject. This was accounted for by haemoconcentration. The extent to which this could occur was shown by a case of chronic amoebic dysentery who had a profound anaemia with a haemoglobin of 18%. This patient developed acute bacillary dysentery and in two days the haemoglobin rose to 50%.

On palpation of the abdomen of an early case tenderness was most common in the left iliac fossa where the spastic sausage-like sigmoid colon could be felt. Tenderness in the right iliac fossa in an early case with little or no diarrhoea was at times misleading, and a few such cases had their abdomens opened by surgeons who mistook them for acute appendicitis. But in most cases there was little difficulty as in the early case of dysentery there was seldom rigidity, and later, when there was rigidity it was not worth while opening the abdomen as in all probability the whole caecum and possibly the appendix would be inflamed as a result of the dysenteric infection. Right iliac fossa pain and rigidity was more likely to cause confusion in chronic cases where there might well be a surgical condition

apart from the dysenteric condition. As a general rule in acute dysentery affecting the caecum that part of the bowel could be palpated as a spastic, sausage-like tumour, and the maximum point of tenderness was at this point. (It was observed in three or four cases of chronic dysentery, with easily palpable thickened caecums, who developed acute appendicitis, that the maximum point of tenderness was at the inner border of the caecum). In the acute dysenteries large sections of the bowel, such as the whole of the ascending or transverse colons, were often spastic, sausage-like and tender. In some severe infections the whole course of the large bowel could be clearly palpated. In cases with ileal involvement the tenderness was near the umbilicus. The ileum was never palpable owing to the abdominal distension in these cases.

As cases became more gravely ill and toxaemia became more profound, certain symptoms were regarded as being indicative of a bad prognosis. The most important of these was persistent hiccoughs. This most trying symptom was recorded in 28 out of 202 fatal bacillary cases. Similarly the prognosis was bad in cases with persistent vomiting, and this was recorded in 11 cases in the same series. Purpura was noted in 9 cases.

Stools.

Whilst in many instances the stools in cases of acute bacillary dysentery were characteristic, in many others the

naked eye appearances were quite inconclusive. The character of the stool, naturally, varied with the stage of the disease and the severity of the infection. The initial diarrhoea usually consisted of copious liquid faeces. Most cases were not seen at this stage, but where the condition developed in a patient who was already in hospital, one often noticed small pieces of clear mucus containing flecks of blood clinging to the bottom of the bedpan. Other cases, after perhaps a night of diarrhoea, would pass a fairly large mass of intermingled blood and mucus. Many cases after the intense diarrhoea had pretty well cleared the intestines of faecal material, passed stools which consisted almost entirely of greenish-brown bile containing pledglets of mucus and flecks of blood. Once the disease was well established very little faeces made its appearance in the stools at all, and at this stage the frequent scanty stools were usually nothing more than mucus, which was tinged with a meat-juice colour, and contained flecks and streaks of clotted blood. This mucus was not generally sufficiently viscid to remain in a blob, but would spread out over the bottom of the bedpan. However, on tilting the pan it would cling to the bottom characteristically. In some cases masses of blood-stained mucopus were passed, the intermingled blood, mucus and pus giving a marble effect. Occasionally the stool consisted of mucoid blood, having a red currant jelly

appearance. In severe infections large amounts of blood, either fluid or clotted, were not uncommonly found in the stools during the second and third weeks. In one series of fatal cases 14.5% had had a serious haemorrhage at this stage. Blood in bacillary stools was of a definite red colour, and not foul-smelling. In fact, of bacillary cases in general, it could be said that the stools were seldom offensive and once the early faecal stage was passed they seldom had any smell at all. The exception to this was the type of moribund case in which the sickly, sweetish odour of the stools consisting of pure serum and necrotic mucous membrane, has already been mentioned. Sloughs, varying in size from small particles to long strips of bowel mucosa, were commonly found in the stools of severe cases in the second and third weeks.

Once the height of the attack was passed recognisable faecal material re-appeared in the stools. At first these consisted of liquid faeces which were often of a pale colour and contained a certain amount of intermingled mucus, but frequently in the course of a day or two the stools became firstly semi-solid and then formed. It was common even in cases in which a rapid recovery was being made for a little blood or bile-stained mucus or white stringy mucus to appear at odd intervals for a week or so after clinical recovery.

Microscopic Findings.

The ideal method of obtaining a specimen of stool for microscopic examination was to send a bedpan containing a freshly passed stool direct to the laboratory so that the microscopist could himself select a suitable portion from which to prepare a slide. However, in epidemics during which there was an acute shortage of bedpans it was not always possible to do this, and in these circumstances specimen jars had to be used. There was, perhaps, no objection to this in the diagnosis of bacillary dysentery, although it is now felt that some amoebic cases might have been diagnosed earlier had this necessity not arisen. Later, by having the laboratory near to the Dysentery Wards and by having trained specimen orderlies who could prepare the slides from a portion of the stool indicated by the microscopist, time was saved, and after the epidemics in 1942 it was always possible for the fresh stool to go to the laboratory in the original vessel. In all cases after the very early days of the Dysentery Wing at least one stool was examined microscopically, and in cases in which the diagnosis was in doubt, many stools were examined, and in fact, it was quite common to have every stool which was passed by a particular case during a period of a few days examined, in an effort to make a firm diagnosis. This labour was repaid by finding living entamoebae histolytica which might otherwise never have been discovered.

In the absence of facilities for the culture of serological identification of the causative organism, the diagnosis of bacillary dysentery rested largely upon the microscopic examination of the stools. The presence of a bacillary exudate had to be accepted as diagnostic, although as a clinician one was at times not satisfied that this was sufficient evidence. In a number of cases which proved to be amoebic months or years later a bacillary exudate had been originally reported. This, however, does not disprove the diagnosis of bacillary dysentery on the earlier occasion as a double infection might have been present, or indeed, the amoebic infection might have occurred subsequently. In fact the presence of an amoebic infection in addition to the fatal bacillary process was established in 8.6% of post mortems. Taking a broad view of thousands of cases from this distance, and considering the effects of treatment and sigmoidoscopies on those who recovered and the post mortem reports of those who did not, it does appear that the presence of a bacillary exudate in the stool was on the whole indicative of a bacillary infection. On the other hand a negative finding in that respect could not be accepted in the face of a clinical diagnosis. The author carried out sigmoidoscopy on all cases in a 140 bedded ward of acute dysenteries in the course of two or three days. The exact findings of this investigation unfortunately were not preserved, but the conclusion was that

the vast majority of cases in whom bacillary exudate had been reported showed visible signs of bacillary dysentery, whilst there were also many cases definitely suffering from the disease whose stools had not shown a bacillary exudate.

The microscopic picture of a bacillary exudate was one in which the following points could be observed. Large numbers of red blood corpuscles were usually present. The actual number was greatly variable and they were generally scattered throughout the field or arranged in groups, but without any actual clumping or rouleaux formation. Most frequently there was a preponderance of polymorphonuclear leucocytes, which were usually intact with well defined nuclei. Setting aside the red blood corpuscles these leucocytes represented over 90% of the cells present. Next in importance from the numerical point of view, but of paramount importance diagnostically were the macrophages - large cells whose granular cytoplasm contains vacuoles and greenish globules and sometimes ingested red blood corpuscles. These cells are oval or may have lobes which resemble the pseudopodia of an amoeba, but they are non-motile and the protoplasm is opaque whilst that of the pseudopodia of entamoeba histolytica is clear. The presence of macrophages, coupled with a complete absence of motile bacilli, was regarded as the most important point in the diagnosis of a bacillary exudate. Columnar ephithelial cells, usually disintegrated, might also be present.

Careful, personal inspection of the stools of dysentery cases at regular intervals by the medical officer in charge of the case was regarded as an absolute necessity. It was considered that the stools of all acute cases and chronic cases during an exacerbation, should be inspected daily. With chronic cases during a less active period it was customary to see the stools about three times weekly. Orderlies were trained to observe and recognise the important points in dysentery stools so that if at any time anything of note appeared the medical officer could be summoned to see it before the bedpan was emptied.

Duration.

The duration of the disease varied greatly. The two extremes were the fulminating type of case which had a fatal termination three days after the onset of the illness, and the type which had a prolonged acute stage of four to six weeks which gradually merged into a chronic stage which might last for many months or even years. Between these extremes all variations were found. The vast majority of cases had a sharp acute stage lasting three to six days and thereafter recovered rapidly, and were fit to leave hospital in ten to fourteen days.

Table 3 shows the duration of illness counted from the earliest recorded symptom in 172 fatal cases of bacillary dysentery. From this it will be seen that 39% of the deaths occurred from the tenth to the fifteenth day.

Table 3.

Day of death.	3.	4.	5.	6.	7.	8.	9.	10.	11.	12.	13.	14.	15.	16.
No. of cases.	1.	9.	5.	4.	11.	6.	6.	13.	14.	13.	10.	8.	9.	5.

Day of death.	17.	18.	19.	20.	21.	22.	23.	24.	25.	26.	27.	28.	29.	30.	31.
No. of cases.	5.	8.	5.	3.	4.	3.	8.	3.	3.	2.	0.	4.	1.	2.	2.

Day of death.	32.	33.	34.	35.	36.	37.
No. of cases.	1.	1.	0.	1.	0.	2.

Complications.

A wide range of complications was encountered among the acute bacillary cases. Bedsores were an inevitable complication in emaciated dehydrated cases lying on hard beds with no proper materials for treating the skin. Some of these ulcers became infected with the Klebs-Loeffler bacillus which from mid 1942 onwards was a common infection in all skin ulcers. The commonest complication was haemorrhoids which might or might not be associated with anal prolapse. Arthritis was rare, in fact the present writer only saw one case. This was an arthritis of the hipjoint occurring as a late complication in a bacillary dysentery of some weeks' standing. Several cases of venous thrombosis affecting the legs were seen, and one case in which the upward spreading process affected the external iliac vein. Toxic hepatitis with jaundice was not uncommon, six such cases occurring in a series of 191 fatal cases. Among the severe cases the commonest serious complication, and one which often

spelt disaster, was a haemorrhage from the bowel. In 17 out of 117 fatal cases which went to post mortem severe haemorrhages were recorded. The danger period for haemorrhage was the second and third weeks of the illness; most fatal haemorrhages occurring between the 9th and 20th days. This is the period during which the sloughs are separating from the ulcers in the bowel. Sometimes, however, in cases of fatal haemorrhage no definite source of the bleeding could be identified at post mortem, whilst on the other hand, the author had a case in which an area of freshly separated non-necrotic mucous membrane 6" x 3" was passed without serious haemorrhage occurring.

Peritonitis occurred as a complication of acute bacillary dysentery in two distinct forms. Firstly it occurred by direct spread of the inflammatory process through the bowel wall, and in this way caused either a localised or generalised peritonitis; and secondly it occurred as the sequel of a perforation of the bowel wall. Generalised peritonitis without perforation was found in 10 cases out of 117 post mortems on acute bacillary dysenteries. A further two cases had localised peritonitis without perforation. Perforation producing peritonitis had occurred in seven cases of the same series while an ischio-rectal abscess had resulted from a perforation in two cases.

Peritonitis as a result of the direct spread of the acute inflammatory process through the bowel wall was a comparatively early complication. In most cases with this type of peritonitis

it occurred during the first week or early in the second week of the attack of acute bacillary dysentery. With the onset of peritonitis there was invariably a decrease in the number of stools whilst in some cases there was a sudden cessation of the diarrhoea. This was accompanied by a change in the character of the abdominal pain, the griping intermittent pain being replaced by a constant aching pain. There was increased tenderness of some area of the abdomen with the usual muscular rigidity. Rigidity was not necessarily a sign of peritonitis as it occurred in many severe acute cases in the absence of this complication. It was definitely present in several cases on whom a laparotomy was performed without signs of peritonitis being found. In these cases there was nothing more definite to be seen than a generalised hyperaemia of the affected areas of the bowel. Rigidity was also noted as a definite sign in severe cases which went to post mortem without any trace of peritonitis being discovered.

Perforation was a later complication, its onset varying from two to four weeks after the onset of the attack. Perforation of the bowel in these gravely ill, toxic cases, was a much less definite condition than, for instance, perforation of a peptic ulcer where a patient who has been going about normally is suddenly struck down. Here it was merely a question of a deterioration in the condition of one who was already causing anxiety. The patient seemed more shocked and the

abdominal pain was worse. There was invariably a diminution in the number of stools but not an absolute cessation of the diarrhoea. There was guarding or rigidity of the abdominal muscles, possibly only of one quadrant, but never was the board-like rigidity associated with perforation of a peptic ulcer seen. Perforations of dysenteric ulcers of the bacillary type occurred in all areas of the colon and also in the appendix. In some cases there was more than one perforation. In cases with small intestine involvement, although peritonitis due to direct spread of the inflammation through the ileal wall occurred, definite perforation of the ileum was not seen. In the absence of surgical intervention the result of perforation was either generalised peritonitis with an early fatal issue, or the formation of a localised abscess either intra or extra peritoneal in position. In the latter case there was in time a palpable abdominal tumour, but even here it was difficult to say whether there was a collection of pus or whether it was omentum wrapped round a leaking bowel that one could palpate. In some cases there was subsequent rupture of the abscess into the general peritoneal cavity followed by fatal generalised peritonitis.

Pyaemia with widespread abscess formation was a terminal event in several cases. Pyaemic abscesses were found in these cases in lungs, liver, kidneys and prostate. In two cases

meningitis resulted from pyaemia. Whilst lobar pneumonia occurred as a complication in several cases, bronchopneumonia was a common terminal event. In two fatal cases haemorrhage into the suprarenal bodies was discovered and bilateral necrosis of these organs was found in four cases. Other less common fatal complications were paralytic ileus (two cases), cholecystitis (one case), and acute suppurative parotitis (one case). As one would expect, a large number of dysentery cases of all types were complicated by various deficiency diseases. These cases will be discussed later.

Illustrative Cases.

A typical case of acute bacillary dysentery is that of Pte. S- of the Royal Dutch Forces. He was admitted to hospital immediately on arrival from Java, having had diarrhoea with blood and mucus for three days. He died three days later and during that time he averaged 20 to 30 motions a day with much blood and mucus. He suffered from intractible hiccoughs during the last 48 hours. The post mortem findings of note were as follows:-

Ileum: Necrosis of the mucous membrane in the last 10 cm.

Colon: In caecum and ascending colon were occasional patches of superficial necrosis of the mucous membrane. Throughout the transverse and descending colons, and in the upper half of the sigmoid colon there was confluent necrosis of the

mucous membrane which was wet and swollen and greenish-black in colour. In the lower sigmoid colon and upper rectum there were scattered patches of surviving mucosa. The total amount of destroyed mucous membrane represented three-quarters of the total area.

In the case of Pte. McD. of the Gordon Highlanders, the onset of acute bacillary dysentery followed a long history of diarrhoea. This patient was one of a party which was moved from Singapore to Thailand to work on the Burma-Siam Railway. He had had diarrhoea for six months. The acute bacillary dysentery appeared to have started 10 days before admission to hospital. Most of that time had been spent on the journey back from Thailand lying on the steel floor of an over-crowded railway wagon. About 25 stools per day were being passed with much blood and mucus. Pain had originally been only in the left iliac fossa at the time of defaecation, but on admission there was constant central abdominal and right iliac fossa pain. The patient was extremely dehydrated and emaciated with a temperature of 96°F. and a pulse of 96. The skin over the sacrum had broken down due to lying on the floor of the railway wagon. The abdomen was scaphoid. The whole colon was spastic and could readily be palpated as a sausage-like mass throughout its length. Tenderness was most marked over the caecum (especially at its medial border) and the ascending colon. There was no rigidity of the anterior

abdominal wall. Auscultation of the abdomen revealed absolute silence in the right iliac fossa, whilst peristalsis could be heard on the left side. The stools consisted of foul smelling blood and mucus which on microscopic examination proved to be a mass of blood and pus cells. Later the stools became serous with a sickly sweet smell. The patient died five days after admission to hospital. The relevant post mortem findings were as follows:-

Ileum: Complete destruction of the mucous membrane in the terminal 100 cm.

Colon: The whole colon and rectum showed bacillary dysenteric changes of varying degree. There was no evidence of mucosal repair. The interior of the descending and pelvic colons and rectum was covered with firm greyish-green unseparated sloughs, whilst higher up the bowel were large haemorrhagic bacillary ulcers which were most extensive in the caecum. The mesenteric glands were acutely inflamed.

A third case, that of Capt. S-. of the British Army illustrates the effects of a terminal pyaemia in an acute bacillary infection. This patient was admitted on 18th March, 1943, with a history of only two days diarrhoea (10 stools with blood and mucus daily). There was no previous history of dysentery. During the first few days in hospital the number

of stools daily averaged 25 with much blood and mucus, but on 25th March the stools suddenly fell to three in twenty-four hours. At the same time the patient complained of constant aching pain in the left iliac fossa where there was great tenderness and rigidity of muscles. A laparotomy was performed in the belief that a perforation had occurred. At operation there was no free fluid in the abdomen and no perforation was found. The terminal eight inches of the ileum was bright pink in colour and the wall thickened and swollen. This part of the ileum felt like a doughy sausage. The transverse colon was covered by injected, inflamed peritoneum. The patient died on 31st March, and post mortem findings were as follows:-

Ileum: There was necrosis of mucous membrane which commenced 120 cm. from the ileo-cacal valve and soon became confluent on passing downwards. The Peyers patches were unaffected. In the final 20 cm. ulceration exposing the submucosa covered most of the area.

Colon: There was extensive necrosis with partial sloughing of mucous membrane of the entire bowel with the exception of the sigmoid colon and the rectum, which were unaffected. In the caecum, ascending and descending colons, about 50% of the mucous membrane had sloughed, whilst in the transverse colon the areas of ulceration were even more extensive.

There were scattered pyeamic abscesses in the lungs and kidneys. Almost the whole prostate gland was replaced by one large abscess.

It will be noticed that the acute dysenteric process was present in the ileum in some degree in each of these three illustrative cases. In this they were not altogether exceptions as slightly over 50% of all post mortems on cases of acute bacillary dysentery showed ileal involvement of varying extent. The largest scale on which this was seen was a case of undoubted bacillary dysentery in which 425 cm. of ileum showed patchy necrosis whilst the entire colon was unaffected. This was the only primary bacillary infection in which the disease was limited was limited to the ileum, although there were a further six cases in which the fatal bacillary lesion affected the ileum alone. In these, however, there was in the colon a pre-existing healing or chronic bacillary dysentery or amoebic dysentery.

The post mortem findings in the case of Pte. M--- of the Royal Army Service Corps, who died after an illness of less than one week, are those of acute bacillary dysentery complicated by generalised peritonitis. They were as follows:-

Peritoneal Cavity: Signs of generalised peritonitis with no perforation or localised abscess formation. 100 cc. of turbid fluid in the pelvis. There was fibrinous exudate over the sigmoid colon.

Colon: The mucosa of sigmoid colon was completely necrotic. It was greyish-green in colour, irregular, wet and swollen. In the remainder of the colon, areas of necrosis and areas of oedematous surviving mucosa were roughly equal in extent. In no part of the colon had the pathological process reached the stage of ulceration.

In Lieut. S-. a British officer who died after an illness of nearly three weeks, the findings, those of peritonitis and localised abscess secondary to perforation of the bowel, were as follows:-

Peritoneal Cavity: The omentum was wrapped round the whole length of the colon. The wall of the bowel from the transverse colon to the sigmoid was so friable that it was impossible to separate the omentum from it without tearing. In some places the lumen of the bowel was only separated from the peritoneal cavity by omentum. There was a perforation of the posterior wall of the transverse colon, and a retrocolic abscess shut in by coils of jejuneum had formed.

Colon: The remaining mucous membrane consisted of small necrotic islands on greyish-red submucosa. In a few areas there were deep well-defined ulcers, one of which, in the transverse colon, had perforated, whilst elsewhere there was extensive ill-defined destruction of the bowel wall.

Another case of generalised peritonitis - this time due to spread of the inflammatory process through the wall of the ileum, was that of Sgt. W-. This patient died after an illness of little more than a week. Two days before death the diarrhoea suddenly ceased and thereafter no stools were passed. The post mortem findings were as follows:-

Peritoneal Cavity: Signs of early peritonitis. The coils of the small intestine were distended and congested and covered with a fibrinous exudate. The exudate was thicker over the terminal part of the ileum.

Ileum: The terminal 70 cm. showed confluent necrosis of mucous membrane. There was purple discolouration of the whole wall and infection of the serosa in the lower part.

Colon: Uniform necrosis of mucous membrane from the caecum to the anus. Sloughing of mucous membrane in places. Only one eighth of mucosa had escaped the infection.

Illustrative of the fulminating, choleraic type of case is that of Gunner B. of the Royal Artillery. On 13th August, 1942, he complained of headache and of feeling feverish. Some diarrhoea commenced. When he was admitted to hospital on the 14th August he had had about 20 stools with blood and mucus in the previous 12 hours. By the 16th he was semi-comatous and he died on the 17th, less than 4 complete days after the onset of the illness. Post mortem showed the following points:-

There was 1 oz. of clear fluid in the peritoneal cavity. The whole of the peritoneal surface of the colon was injected. The lymph glands were soft and pink.

Colon: Mucous membrane showed early bacillary necrosis throughout the entire length of the colon. This was most advanced in the caecum and ascending colon, but the sloughs had not separated in any area.

Appendix: One third showed similar changes to those in the colon. The remainder of mucous membrane slightly congested and oedematous.

Diet.

The rations provided so little scope for the suitable dieting of acute dysentery cases that this presented a major problem at all times. It was imperative that the period during which the patient was kept on fluid diet should be limited to absolute necessity, as most patients were grossly under weight even before they contracted dysentery. The importance of this became progressively greater as time went on and malnutrition and deficiency diseases became general.. At most times the only liquids available for fluid diets were water, tea or vegetable water. During the middle of the period of captivity limited amounts of dried milk were available from Red Cross supplies, and it was sometimes possible to obtain a

few eggs from local purchase or from hens kept in the camp. However, supplies of these two commodities were so restricted even at the best of times, that they had to be reserved for the worst cases. Light diets usually consisted of ground rice or sago boiled with water, or plain boiled rice with a sieved vegetable stew. There was also a dish known as 'pap' which consisted of rice boiled with an excess of water until it was mushy. Into this pulped vegetables or a small quantity of red palm oil was mixed. Generally speaking oil was best left out of dysentery diets, and even patients on full diet were not allowed fried dishes. The full diet was composed of rice and vegetables in different forms with occasionally, minute quantities of dried fish.

Treatment.

Two points in the treatment of acute bacillary dysentery were considered of greater importance than any drug or diet. These were drink and rest. It was impressed on patients and orderlies by constant re-iteration that vast quantities of water had to be consumed by all acute cases. The patients on either side of a severe case were instructed to see that he kept drinking. A small quantity of salt was usually added to the water, but one had to be careful not to use enough to cause nausea or to deter the patient from drinking. The second point which patients had impressed upon them was the need to resist

the urge to stool. Many patients tired themselves out by getting up and down every few minutes, whilst others with much worse infections conserved their energy by lying still until they could resist the urge no longer. Although these two factors may never have abated the virulence of the infection, it is felt that they frequently enabled patients to resist infections which might have proved fatal without their help. It was repeatedly demonstrated that dehydration and exhaustion were easier to prevent than to cure.

During the first seven or eight months of captivity no form of sulphonamide was available for the treatment of acute bacillary dysentery, so that during the great epidemics of 1942 treatment was mainly by saline purgatives or aperients. Magnesium sulphate or sodium sulphate were the salines mostly used. Some medical officers started with a large dose of the saline and then gave one drachm every two hours, whilst others used a half ounce dose of castor oil as an initial aperient and then followed on with one of the salines every two hours. Calomel was used in doses varying from one sixth grain every half hour to half grain every hour for twelve hours on two consecutive days in one series of cases, but this method certainly had no advantage over the saline treatment. Of all cathartics sodium sulphate was the most comforting to the patients, many of whom showed appreciation of the relief it gave

from the griping pain and tenesmus.

During the first epidemic colonic lavage was used by some medical officers in the treatment of acute cases. This practice was abandoned after a short time as it appeared to increase the incidence of serious haemorrhage. Normal saline washouts were used to some extent but there was a great shortage of common salt, and in a few cases sea-water suitably diluted was used as a substitute. Eusol washouts were used in a dilution of one part in eight parts of water but even this caused a great deal of pain.

Morphia was used freely in all cases with severe bleeding. Cases of dangerous haemorrhage were given maximum doses of the drug repeated every two or four hours and some of these cases seemed to have been saved by this means. Sedatives such as chlorodyne or tincture of opium were very useful in moderate repeated doses to relieve pain or tenesmus at night, and in severe cases morphia was used for this purpose.

There were very small supplies of polyvalent anti-dysenteric serum. This was only used in cases of great severity and one cannot claim any appreciable improvement in these cases. This was possibly due to the fact that owing to the limited stocks most of the cases to whom it was given were probably fulminating Shiga infections, and also that it was withheld too long.

A large number of dehydrated cases were given intravenous normal saline. In cases who were not vomiting the results of

persuading the patient to take saline by mouth were as good, if not better than those obtained from saline infusions. Some haemorrhage cases were given intravenous salines but the results were not good. It was quite obvious that there was no comparison between the use of intravenous salines and blood transfusion in these cases. Blood transfusion in these cases was of such value that one felt justified in taking blood from the patients' semi-starved friends. However, one never took more than half a pint from any one donor.

Blood transfusion by continuous drip was also given to many collapsed, dehydrated, toxic cases. These patients were kept as warm, quiet and still as possible. They were sedated and were not bothered with constant bedpans, but merely had tow or wadding placed under them. The only thing for which they were disturbed was to persuade them to take fluids.

The Sulphonamides.

During the latter half of 1942 most medical officers in charge of dysentery wards had long been dissatisfied with the results of treatment with the drugs so far available. Towards the end of that year it became possible to procure sulphapyridine for the treatment of dysentery cases from a previously inaccessible store. The results of the very first trials of this drug were so promising that it was soon adopted as the treatment of choice for severe cases of acute bacillary dysentery. The

necessity for practising strict economy in the use of sulphapyridine meant that only severe cases could have the benefit of it. Perhaps this very economy was the key to the outstanding success with which its use was met in the Prisoner of War Camps, for economy also dictated the dosage which was restricted to the minimum effective.

In moderately severe cases (with perhaps 20 stools in 24 hours with much blood and mucus) a total of 5 grammes of sulphapyridine was given in two doses of 2 grammes and one of 1 gramme at four-hourly intervals. This small quantity of sulphapyridine spread over no more than eight hours had a most remarkable effect on the vast majority of cases of bacillary dysentery of this degree of severity. In most cases the number of stools in the following 24 hours would drop to somewhere in the region of five, whilst only one or two stools were passed in the second 24 hours. On the third day there were often no motions at all, and thereafter the average was one formed motion daily with perhaps a streak of mucus for a day or two. The other symptoms, the fever, the pain and tenesmus were relieved with a like rapidity. The absorption of the drug appears to have been so rapid that even cases in which vomiting occurred within a quarter of an hour of taking the sulphapyridine responded to the

treatment. Vomiting as a result of taking the drug was not common and this was probably because of the brevity of the course. Where vomiting did occur it was largely overcome by giving a solution of sodium bicarbonate a quarter of an hour before the dose of sulphapyridine. Generally speaking it may be said that most of the few cases which did not respond to this treatment afterwards proved to be amoebic.

For more severe cases the totsl quantity of sulphapyridine used was 8 or 10 grammes given in 2 gramme doses every four hours. Even extremely severe cases having 60 stools or more in the 24 hours (i.e., they were almost continuously on the bedpan passing blood and mucus) with much pain and tenesmus and a variable degree of toxaemia were given a total of only 10 grammes of sulphapyridine, and here again the results were most arresting. Medical officers who up to the introduction of this drug would have regarded such cases as hopeless were astounded at the rapid improvement which followed its use in all early cases. There was a great change in the patient's general condition in a few hours. The number of stools fell rapidly and possibly no more than 10 or 12 stools might be passed in the first 24 hours and one formed stool or none at all by the third or fourth day was by no means unusual even in these severe cases. Of course, the drug had to be given early but not necessarily before the patient became toxic

because many of these cases showed signs of profound toxaemia on admission to hospital. It was, however, imperative to start sulphapyridine before widespread irreversible changes had occurred in the bowel. There were certain cases of this type, who were too far advanced on admission to hospital for it to be possible to save them. Such cases were given blood transfusions by continuous drip, and sometimes intravenous sulphapyridine without success. As already mentioned it was possible after a time to spot these hopeless cases by the smell. It would appear that once a certain area of mucosa is destroyed that the patient cannot recover. These patients were in a condition akin to surgical shock and owing to destruction of bowel mucosa were incapable of absorbing fluid to such an extent that no measures could help them. It has been said that if a person has a third of his skin destroyed by a burn that he cannot recover. It may well be that there is a similar state of affairs in respect of the mucous membrane, and that destruction of a certain proportion of it is always fatal.

It was possible later to compare the efficacy of sulphpyridine with that of sulphaguanidine. Sulphaguanidine was found not to be so effective in cases which were already showing signs of severe toxaemia before treatment was commenced. Its action was not so rapid and larger amounts of the drug had to be used. Some of the cases in which sulphaguanidine was employed became chronic; this was practically unknown with

sulphapyridine. Sulphaguanidine never caused nausea or vomiting, and, in fact, it had an immediate gastric sedative effect. It was, therefore, the drug of choice in all cases in which vomiting was an early symptom. Patients treated with sulphaguanidine had to be kept on a fluid diet slightly longer than those treated with sulphapyridine. With the latter drug it was only necessary to keep patients on strict fluids for 24 hours, or 48 hours at the most, and this was a most important consideration in men who were in a state of gross malnutrition before they were taken ill. To sum up, it may be said the sulphaguanidine was a most efficient remedy but in the cases of acute bacillary dysentery met with in Singapore sulphapyridine was more effective.

The technique adopted for using sulphaguanidine was as follows: The treatment commenced with a saline purge followed occasionally by a bowel washout of normal salt solution. The washout was only used in cases in which motions were mainly faecal. The saline purge usually acted fairly rapidly and immediately after that an initial dose of 0.1 gm. of sulphaguanidine per kilo of body-weight was given. Thereafter 0.05gm.per kilo was given every four hours until the number of stools in the 24 hours dropped to four or five, and after that the same dose was given every eight hours until the stools dropped to two per diem. This meant that in a ten-stone man 14 half-gramme

tablets, crushed and suspended in water, formed the initial dose and half that quantity was given every four hours. During 1945 when the shortage of all drugs was acute it was necessary to ration the use of sulphaguanidine to a total of 30 half-gramme tablets for each case. At that time sulphaguanidine was always used for severe cases except the acutely ill toxic type of case, for whom sulphapyridine was reserved. The modified course of sulphaguanidine consisted of 10 half-gramme tablets as an initial dose followed by 5 tablets every four hours for four doses. The results were not as good with this limited dosage which was dictated by necessity, but they were far better than those obtained by using salines, which for reasons of economy always had to be the treatment of the less severe cases. Some cases which got rather out of hand on salines were given sulphaguanidine later but these never did as well as those in which sulphaguanidine was given early.

At the end of the period under review, namely in July 1945, certain other "sulpha" compounds were issued. These had arrived in Singapore in a consignment of American Red Cross supplies, but had been held up for some months by the Japanese Military Authorities. When it became available sulphadiazine was used in the treatment of several cases of acute bacillary dysentery with results comparable to those already obtained with sulphapyridine. This drug caused no nausea or vomiting at all.

It was used in the same dosage as had previously been adopted with sulphapyridine.

In Tables 4 and 5 an attempt has been made to demonstrate in tabular form the great superiority of sulphapyridine over the salines in the treatment of acute bacillary dysentery. Only fifteen cases are shown in each group, but these cases are typical of very large numbers which were treated by these two methods. It was impossible to preserve records of all cases, but these tables have been prepared from notes which were made by the author in the P.O.W.Camp on consecutive cases. It is not possible actually to compare two groups of cases of similar severity, as taking as we are here, consecutive cases treated by these two methods, the sulphapyridine group is bound to represent more severe infections than the saline group as it was reserved for this type of case. But it was felt when these notes were made that there was more probability of getting groups approximating to each other in severity if one took the saline group from 1942 when nearly all cases were treated by this method, and the sulphapyridine group from later on. The reason the sulphapyridine group mostly had intercurrent diseases is that these cases were drawn from an outbreak of acute bacillary dysentery in the hospital itself. The exceptions to this are cases 10 and 12 in Table 5 which were admitted from working camps, but they also happened to have intercurrent

diseases. They were included because the series was compiled from consecutive cases treated with sulphapyridine in one of the author's dysentery huts. This particular period was selected because the severity of the dysenteric infection more nearly compared with the cases dealt with in the notes which had been compiled for the saline group. Naturally one was more ready to use sulphapyridine for a patient who was already ill from some other cause. The same remark also applies to numbers 9, 13 and 14, where "key" members of the staff were affected.

In one more respect the comparison of these two tables tends to obscure the true superiority of sulphapyridine. The saline group is drawn from 1942 when the effects of malnutrition, privation and conditions of forced labour had only been operating for some months. In 1945, not only had these factors been operating for three years longer, but in addition, repeated attacks of disease had further reduced the man's general condition.

TABLE NO. 4

15 Cases of Acute Bacillary Dysentery treated with Magnesium Sulphate (1942).

STOOLS PER DAY.(+ = Blood and Mucus).

Case No.	Days of illness before treatment.	Days of diarrhoea before treatment.	Stools in previous 24 hours.	DAY OF TREATMENT. 1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	NOTES.
1	3	3	8+	14+	20+	14	8	5	6	3	2	2	4	4	D										Griping pains ++
2	1	¾	14+	22+	20+	20+	18+	12+	10+	3	2	1	0	0	1	D									Shivering, sweating, headache before onset of illness. Tenderness all over abdomen.
3	2	2	11+	11+	12+	8+	5+	3+	5	8	2+	4+	7	7	10	7	7	5	4	4	6	4	2	3	"Belly ache" and griping pains. Tender over sigmoid. Stools averaged 5 until 29th day.
4	3	3	very frequent	continuous+	continuous+	18+	14+	14+	11+	7	15+	5	10	4+	8	4	2	2	1	1	D				Griping pains. collapsed and dehydrated. Tenderness throughout colon.
5	2	2	25+	30+	23+	33+	20+	12+	12+	15	6+	8	8	5	4	D									Griping pains ++
6	5	5	very frequent	38+	19+	11+	4	0	0	4	3	2	1	4	0	1	2	2	D						Tenderness in left iliac fossa.
7	4	4	20+	9+	5+	7+	5+	5	2	3	2	1	1	1	D										Sudden onset of abdominal pain. Appetite poor. Vomited. Diarrhoea soon after. Tenderness in R.I.F. and along transverse colon.
8	2	2	8+	13+	6+	4	4+	0	0	0	1	1	0	D											Ambulent case.
9	4	4	10+	6+	8+	10	8	7	3+	2	1	5+	3	1	D										Slight colic. Otherwise felt well. Temp. 104°F.
10	5	2	12+	4+	12	6+	5	2	2	1	1	2	2	1	1	D									Malaise for 3 days before onset of diarrhoea Colic & tenesmus.
11	3	3	16+	8+	12+	7	3	2	2	1	D														Colic & tenesmus.
12	5	4	9+	5+	6+	11	8	8	3	1	1	1	D												Malaise day before onset of diarrhoea.
13	1	1	10+	15+	11+	20+	9	4	3	0	2	2	1	1	D										Sudden onset of diarrhoea and abdominal discomfort. Tenderness in L.I.F.
14	5	2	23+	18+	22+	15+	13	9	12	5	4	0	1	0	1	4	1	1	1	D					Abdominal pain. Pain behind eyes & whole body aching. Diarrhoea 3 days later. Generalised abdominal tenderness.
15	1	1	15+	10+	14+	11+	8	4	3	3	3	2	1	1	1	D									Pain in epigastrum before defaecation. Sigmoid palpable and tender.

These cases had not previously suffered from dysentery. There were no intercurrent diseases. All cases showed bacillary exudate. The average number of days on fluid diet was 5, the maximum being 8 and the minimum 2.

D = Discharged.

TABLE NO. 5. 15 Cases of Acute Bacillary Dysentery treated with Sulphapyridine (1945)

Case No.	Days of illness before treatment.	Days of diarrhoea before treatment.	Stools in 24 hours before treatment.	STOOLS PER DAY. (+ = Blood & Mucus). DAY OF TREATMENT: During night.	1	2	3	4	5	6	7	8	9	10	11	Previous attacks dysentery	Total Sulphapyridine in grammes	Intercurrent Disease.	
1	1	1	20+	3+	1	1	1	1	D							0	5	Wound of foot.	Sudden onset of fever & diarrhoea. Pain in iliac fossae before defaecation. Patient looks toxic. Sigmoid spastic and tender.
2	1	1	20+	14+	3	1	1	D								1	10	Lung Abscess.	Central abdominal pain. Vomiting++. Tongue furred. Dehydration +. Looks toxic. Sigmoid spastic & tender. Ascending colon tender throughout.
3	1	1	8+	3	0	2	1	D								0	5	Ankylostomiasis.	Severe pain in hypogastrium before defaecation. Tenderness in L.I.F.
4	1	1	18+	5	2	1	1	1	D							B 1 A 2	5	Malaria. Ascariasis. Malnutrition.	Central abdominal pain before defaecation. Emaciation+++ Looks toxic. Anaemia++ Tongue dry & furred. Spleen 2 f. below C.M. Sigmoid spastic & tender. Caecum palpable & spastic.
5	1	1	22+	6+	1	1	1	2	3	1	D					1	8	Beriberi. Malaria.	Pain across lower abdomen before defaecation. Pyrexia+ Patient looks ill. Tongue furred & dry. Tenderness in both iliac fossae. Caecum palpable. Sigmoid palpable.
6	2	2	36+	13+	6	0	2	0	1	0	1	1	0	D		0	8	Malaria. Malnutrition.	Pain in L.I.F. before defaecation. Vomiting++ Looks toxic & ill. Tongue dry & furred. Sigmoid spastic and very tender.
7	1	1	25+	14+	5	0	0	1	0	0	1	D				0	5	S.T.- Malaria.	Severe pain in left side before defaecation. Fever. Nausea. Looks very ill. Pyrexia++ Sigmoid spastic and tender.
8	4	4	15+	12	5	3	1	1	1	3	2	2	2	D		1	5	Malnutrition. Anaemia.	Constant pain in L.I.F. Pain in R.I.F. before defaecation. Looks toxic. Tongue furred & dry. Tenderness in both iliac fossae. Sigmoid spastic and very tender.
9	1	1	10+	5	3	0	0	1	1	1	1	D				4	5	Nil. (Staff)	Severe pain in lower abdomen during defaecation. Fever++ Vomiting++ Flushed. Tongue dry & furred. Pyrexia++ Pulse 120. Tenderness over whole colon from splenic flexure to sigmoid (spastic).
10	10	10	26+	14	7	4	2	1	1	1	D					1	5	Neuritic Beri-beri.	Started with 45 stools in 24 hours with blood & mucus (10 days ago). Pain in L.I.F. Vomiting+.
11	3	3	41+	10+	3	1	2	2	1	1	D					4		Neuritic Beri-beri.	Severe pain in L.I.F. Repeated Vomiting. Rigidity in L.I.F.
12	20	19	25+	7	0	1	1	1	D							1		Beri-beri. Malaria.	Attack started with fever. Following day had 33 stools with much blood and mucus. 10 to 20 stools daily until admission.
13	2	1	18+	7	0	0	1	D									5	Nil. (Staff).	Severe pain in upper abdomen followed by several solid motions. Tenderness in L.I.F.
14	2	2	24+	1	1	1	1	0	D							B 1 A 1	5	Nil. (Staff).	Sudden onset of severe central abdominal pain immediately followed by diarrhoea. Tongue furred & dry. Sigmoid spastic & tender.
15	1	1	6+	5	2	2	1	1	1	D						3	5	Beri-beri.	Severe pain in upper abdomen which works round to L.I.F. before defaecation. Tongue dry & furred. Sigmoid spastic & very tender.

All these cases with the exception of Nos. 9, 13 & 14, were already suffering from other diseases. D = Discharged or transferred to another ward for treatment of intercurrent disease. All cases showed bacillary exudate. All cases were only one day on fluid diet.

In Table 4 the protracted diarrhoea must be discounted to some extent because of the cathartic action of the treatment employed, but this in no way mitigates the adverse effect of the fatigue caused by this diarrhoea. Even as late as the 10th day 50% of the cases still had diarrhoea. Of the cases in Table 5, only two still had mild diarrhoea on the 3rd day. The continued activity of the bowel lesion as shown by the passage of blood and mucus for an average of five days in Table 4 and never later than the night following the commencement of treatment in Table 5 is of great significance. This is a factor which influenced the decision to keep the cases in Table 4 on fluid diet for an average of five days, whilst in all cases in Table 5 it was possible to permit a light diet on the second day. In 1942, when the cases in Table 4 occurred, the Dysentery Wing was overcrowded and the tendency was to discharge patients early; nevertheless the average stay in hospital of the cases in Table 4 (excluding No. 3 who was still in hospital on month after treatment commenced) was 13 days. In Table 5 the average stay in the Dysentery Ward was seven days although the tendency here was to retain a little longer than absolutely necessary for fear of spreading infection in other parts of the hospital.

The conclusion to be drawn from these facts are self-evident, and do not require further emphasis.

Unfortunately sufficient records of cases treated with sulphaguanidine in full dosage are not available to tabulate

the results, so that it is impossible for the purpose of comparison with the foregoing tables. In the following table (Table 6) ten cases treated in 1945 are shown. The severity of nine of these cases is comparable to the cases already shown in Table 4, and generally speaking they are much milder infections than those shown in Table 5. Case No.10 was similar to some of those shown in Table 5. Cases 6, 7 and 8 were transferred particularly early as they were mental cases who were difficult to handle.

TABLE NO. 6.

9 Cases of Acute Bacillary Dysentery treated with Sulphaguanidine (modified course of 15 gm.)

June - July 1945

Case No.	Days of illness before treatment.	Days of diarrhoea before treatment.	Stools in 24 hours before treatment.	STOOLS PER DAY.(+ = Blood & Mucus) DAY OF TREATMENT 1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	No. of previous attacks.	
1	3	3	20+	6	2	4	3	5	6	5	4	4	3	4	2	2	2	D					0	Lab.Report on admission:- "R.B.C's.W.B.C's - Epith cells". Sigmoidoscopy on 14th day:- "Thickening of mucous membrane of rectum & sigmoid.No ulcers or granulation tissue".
2	5	5	20+	5	2	8	6	4	3	4	4	3	3	3	4	3	4	3	4	2	1	D	0	BO 20-30 daily during 5 days before treatment.Lab.report:- "R.B.C's Pus cells and Epith. cells".
3	9	9	8+	3	3	3	3	3	2	3	2	2	1	5	2	1	2	2	D				5	Sigmoidoscopy on 15th day:- "Mucous membrane of rectum & sigmoid thickened and granular.No ulcers seen".Bacillary exudate reported in stool on admission.
4	3	3	20+	3	1	2	1	2	2	1	1	D											0	Stools said to be uncountable before admission.Suffering from oedematous beri-beri.Lab.report:- "R.B.C's +++ Pus cells +".
5	14	14	5+	4	4	3	4	3	3	2	2	2	1	1	1	D							6	Maximum number of stools in one day during 14 days before admission was 20 with much blood and mucus. Sigmoidoscopy on day of admission:- "Signs of acute bacillary dysentery".
6	1	1	6+	2	0	1	D																4	Case from Mental Ward.Bacillary exudate reported.
7	3	3	18+	4	1	D																	2	Case from Mental Ward.Bacillary exudate reported.
8	½	½	8 in 12 hrs +	6	2	1	1	D															0	Case from Mental Ward.Bacillary exudate reported.
9	1	1	20+	4 in 12 hrs	1	1	1	D															0	Very sudden onset.Bacillary exudate reported.
10	7	7	28	17 + in 12 hrs	5+	8+	9	9	6	3	3	4	5	3	5	4 pus	5	3	3	4	5 pus	4 pus	0	Onset one week before admission with 40 to 50 stools in 24 hours.with much blood and mucus.Very ill on admission.Pains in right side of abdomen.Lab.Report:- "Typical Bacillary Exudate". Sigmoidoscopy on 21st day:- "Very extensive chronic bacillary ulceration. Much mucus present".Sigmoidoscopy 1 week later showed masses of granulated tissue

From this table it will be seen that even in this small dose sulphaguanidine was so effective that there was no blood and mucus in the stools in 9 of these 10 cases twenty-four hours after the drug was administered. The diarrhoea was protracted for several days in 5 of these 19 cases, but all but the last were permitted a light diet on the second day. In case No. 2 this caused a recurrence of the diarrhoea. Case No. 10 became chronic and was subsequently successfully treated with sulphadiazine.

The tentative conclusion one can draw from a comparison of Table 6 with Tables 4 and 5 is that sulphaguanidine even when restricted to a total dosage of 15 gm. was more effective than salines in the treatment of acute bacillary dysentery but considerably less effective than sulphapyridine.

CHAPTER IV.

CHRONIC BACILLARY DYSENTERY.

In May 1942 a ward of 100 beds was set aside for the treatment of chronic bacillary dysentery. Later, owing to the fact that the discharge rate of chronic cases was so low it became necessary to increase the capacity of the ward to 120. At its inception 80 cases, whose dysentery had remained active after six or eight weeks' treatment, were transferred from the "acute" wards. These men were mostly more or less emaciated, but although by normal standards they appeared extremely so, they did not compare with the gross degrees of emaciation one was to encounter later. However, even at that early stage, some cases showed signs of vitamin deficiency. Emaciation in chronic dysentery cases was usually associated with postural faults due to weakness. Those who could stand stood with a stoop, with the back bent, the head drooping, the shoulders rounded and the chest concave. Many patients who became emaciated to this degree never regained an upright posture.

The faces of the acute dysentery cases were often anxious, pinched and drawn or even agonised. That of the chronic case was typically solemn with large eyes and drooping mouth. The complexion was sallow and the mucous membrane pale. The hair was often dry and coarse and the nails brittle. The skin of the body was usually dry and often looked dirty owing

to fading sunburn. At a time when most men wore no more than a pair of underpants or shorts, those suffering from chronic dysentery felt the cold so easily that they would don some uppergarment when the atmospheric temperature fell even a few degrees. When the temperature fell below 80°F. many of them would go to bed and cover themselves with blankets.

The abdomen in chronic cases was usually scaphoid but some were pot-bellied. There was generally some part of the colon which was thickened, whilst in a few cases the whole length of the colon was palpable. The sigmoid colon was the most commonly affected and next in order of frequency was the caecum. In many cases the liver edge could be palpated one or two fingers' breadths below the costal margin. In one or two cases in which no malarial parasites could be detected the spleen was enlarged. Some cases had vague masses, which were probably due to old adhesive peritonitis, palpable in the abdomen, and one or two had free fluid (this was quite apart from cases of ascites due to beri-beri).

Complicated cases were extremely common, and the complications encountered presented a wide variety. All types of Vitamin B deficiency were met with at one time or another, and these will be discussed separately. Vitamin A deficiency in the form of dryness and harshness of the skin was common after the first six or eight months. Vitamin C deficiency in

the form of clinical scurvy was never seen but it was thought that less definite forms of the condition were often present in those patients whose vegetable intake had been restricted for long periods owing to the bowel condition. In cases with repeated haemorrhage and delay in blood clotting one felt strongly that a Vitamin K deficiency might be involved. (There was no synthetic Vitamin K in the camp). Other complications included meningitis, encephalitis; pyaemia, pneumonia, pleurisy, pulmonary tuberculosis, bronchial carcinoma, lung abscess; hepatitis, appendicitis, peritonitis (purulent and tuberculous); venous thrombosis, arthritis; infective mononucleosis, polycytaemia vera, various anaemias; malaria, diphtheria and infestations with various parasites. Many cases were bed-ridden and some had bedsores. Tropical ulcers with or without secondary K.L.B. infection were common. Piles and anal fissure were almost general in chronic dysenteries. Of skin conditions, those seen most frequently were scabies, and various types of tinea. These became increasingly common as time went on. Similarly with lice; these were seldom seen in 1942 and 1943, but from the end of the second year onwards they were seen more and more on patients admitted from working camps.

A great deal of labour and time was devoted to distinguishing between amoebic dysentery and chronic bacillary dysentery. The difficulties encountered and the methods employed and devised

in the diagnosis of amoebic dysentery will be described in the section dealing with that disease.

The author sigmoidoscoped all cases of chronic dysentery as a diagnostic procedure, and many cases had this examination repeated as a check on progress and the results of treatment. The bowel condition in cases of chronic bacillary dysentery varied within wide limits, but there are certain definite categories into which most cases can be grouped. The commonest type was that in which large irregular patches of superficial ulceration were seen throughout both rectum and sigmoid. There was usually thick pus adherent to the ulcerated patches. The intervening mucosa in these cases was usually the seat of catarrhal inflammation. Large masses of granulation tissue were often seen in these cases, and pressure with the end of the sigmoidoscope on these masses caused thick pus to ooze from the granulations. In some cases no ulceration was at first seen and the walls of the bowel appeared to be rigid and thickened and covered with chronically inflamed mucosa. However, on pressure with the end of the sigmoidoscope numerous beads of pus would appear from tiny pits and it was then seen that what appeared to be inflamed mucosa was in reality a mass of granulations covering large areas of the bowel wall. In other cases, in which healing was taking place, one would encounter patches of fine granulations without any sign of frank pus, although there

was usually some mucopus adherent to the surface.

Cases of the type just described usually passed much pus, often blood-stained, per rectum each day. The number and character of the stools naturally varied considerably. Commonly from three to five stools were passed daily. They might be of any description from an emulsion of mingled faecal material and pus to hard pieces of formed faeces in a sea of pus. Cases with ulceration low down in the rectum sometimes had no diarrhoea at all. In one case with four large, deep ulcers in the lower rectum there was no history of diarrhoea, and the complaint was, in fact, one of constipation which was often unrelieved for several days.

The second most common type of chronic bacillary ulceration was that with clear cut ulcers of medium depth containing thick white pus. In these the ulcers were either small and round or oval or larger with a serpentine outline. Of the latter the long axis was usually placed more or less longitudinally in the bowel. In cases in which the ulcers were small and round it was often very difficult to distinguish them from amoebic ulcers, especially if the intervening mucosa was of fairly normal colour - as it often was. In bacillary cases, however, there was, it is thought, always sufficient thickening of the intervening mucosa to prevent the submucous blood vessels being visible.

In these cases the stools were usually semi-solid faeces with a comparatively small amount of mucopus. Visible blood seldom appeared in this type of case, but sometimes there was a fairly large amount of mucus, either alone or mingled with faecal material. Four was the average number of stools to be passed each day in most cases.

Around superficial ulcers, where the intervening mucosa was of normal colour, there was often a margin of inflamed mucosa. This finding was common to both bacillary and amoebic cases. It was considered that where the superficial ulcers were large and of irregular outline that the majority of cases were of bacillary origin. However, in cases with small, "punched out" superficial ulcers surrounded by a "red areola" (the phrase the author used to describe this margin of inflamed mucosa) the diagnosis was extremely difficult unless the microscopic findings of scrapings from the ulcers were helpful. Here, as in acute cases, the discovery of macrophages was a definite pointer to a bacillary infection. In other cases, of course, the presence of the entamoeba histolytica clinched the diagnosis in the opposite direction. Unfortunately in many cases of chronic dysentery the contents of the ulcers was pus resulting from secondary infection and no firm diagnosis could be made on the microscopic appearances of scrapings. Often in such cases the sigmoidoscopist had to rely on his experience

to help him to form a diagnosis, and it was sometimes impossible to put into words the reason for his opinion.

There was a third type of case with a dusky red, oedematous mucous membrane with no visible ulceration. These cases usually had four to seven semi-solid or liquid motions, containing much mucus, each day. Every week or two these cases would flare up and would have what was really a short attack of acute dysentery. Sometimes these exacerbations were very severe. One or two cases which had been dragging on for months underwent spontaneous recovery after a severe exacerbation of this sort.

The condition known as pseudo-polyposis was occasionally seen in the rectum or sigmoid of chronic bacillary cases. Polypoid scars often formed round the edges or in the base of deep ulcers in the rectum. This type of scarring although much commoner in amoebic cases was not unknown in bacillary cases. Several cases were seen in which polypoid scarring in deep ulcers had produced a tendency to stricture formation. These cases usually had a history of both bacillary and amoebic dysentery and it was felt that the condition was produced by the latter disease in most cases. However, there were chronic cases of purely bacillary origin in which masses of rigid granulation tissue were causing stemosis of the bowel. These cases will be discussed more fully later.

The varieties of chronic bacillary dysentery described so far were common in 1942 and 1943. At a later date, namely from 1944 onwards, a different type of chronic case became extremely common. These patients sometimes had no history of a definite attack of acute bacillary dysentery. The onset was often insidious and for months on end these patients passed five, six or seven watery stools daily. There was usually a little mucus to be seen but never any frank pus or blood. The stools often contained flagellates and many of these patients were infested with strongyloides stercoralis. The mucous membrane of the rectum and sigmoid colon was not ulcerated, but it was the seat of generalised, mild catarrhal inflammation. It was thickened so that the submucous blood vessels were not visible, and was of a definite orange-red colour which can best be likened to the glowing embers of a fire. Flecks of whitish mucus or a film of liquid faeces might be adherent to the mucosa, and one often encountered pools of watery faeces in the dependent curvatures of the bowel. It appeared in these cases that what one saw through the sigmoidoscope was not a primary condition, but was secondary to a condition higher up - possibly in the small intestine - which resulted in the mucous membrane of the sigmoid colon and rectum being constantly bathed in alkaline liquid faeces. The mucosa of this region is not normally in constant contact with liquid faeces, and it would

appear that whenever such a state of affairs is long continued a catarrhal inflammation of the type described results. A similar condition of the mucous membrane was observed sigmoidoscopically in several cases of pellagra; and, in fact, some of the cases already referred to in this paragraph may have been due to a deficiency of some part of the Vitaman B2 complex, although others were due to small intestine "hurry" caused by parasites.

A condition of post-dysenteric colitis was comparatively common. In this the patient usually complained of having three or four semi-solid stools daily with much clear mucus. Often some of the motions consisted entirely of jelly-like mucus. A common form was that in which the patient had a normal solid motion the first thing in the morning. Soon after this he would be gripped by severe left iliac fossa pain due to the bowel going into spasm. This might last for five or ten minutes and on the spasm being relaxed the patient would pass a bulky loose stool with some mucus. The sigmoidoscopic appearances in these cases were variable. In some the mucous membrane was pale or of normal colour, but slightly thickened, and in others the mucosa was of a deeper pink colour and definitely oedematous. A considerable quantity of clear or whitish opaque mucus was usually seen during the examination. In one type of colitis which was more often seen after amoebic dysentery the mucous

membrane was dry and lax with adjaeent folds adhering to each by means of a film of sticky mucus. Incomplete emptying of the rectum was often seen in post-dysenteric cases. In these one would discover on sigmoidoscopy that the mid or upper rectum was loaded with solid faeces although the patient had gone to stool immediately before the examination. These were difficult cases to examine adequately without a preliminary washout, but it was often found that the mucous membrane was perfectly normal. In this type of case the patient usually passed three or more normal formed stools daily.

Pain, although a fairly constant feature of cases of chronic bacillary dysentery, appeared often to be disproportionate to the severity of the sigmoidoscopic findings. This, of course, will be readily understood as the amount of bowel visible through the sigmoidoscope is but a small proportion of the whole. In general, however, it may be said that pain in chronic bacillary dysentery varied according to the state of the mucous membrane, but that the affected part was not always accessible to the sigmoidoscope. Nevertheless in fairness to that instrument, it must be maintained that the percentage of cases of chronic bacillary dysentery which showed a normal mucous membrane in both rectum and sigmoid was exceedingly small. Deep ulcers in the rectum usually caused a dull aching pain in the perineum, which might be constant or last for half

an hour or so after defaecation. Pain due to ulcers in other areas was usually before defaecation, but in most cases, especially where there was a generally inflamed mucosa, there were griping pains and tenesmus during defaecation. Pain due to spasm of the colon was very common in chronic cases and was often bad enough to make the patient feel faint during defaecation or for a variable period after the act was completed. On spasm of this nature being relieved there was often a further urge to stool and this cycle might be repeated three or four times during one hour in the night or early morning, and thereafter there might be no further stools for twenty-four hours.

If it was difficult to provide a suitable diet for a comparatively short time in cases of acute bacillary dysentery, it was many times more difficult to diet chronic and amoebic dysentery cases over long periods. The ideal, of course, would have been to supply these cases with a low residue high protein diet with additional vitamins. Protein was almost non-existent in the rations supplied by the Japanese for long periods. It was possible to make up for this lack for a minority of cases during most of 1942 and 1943 with supplies of tinned foodstuffs - mainly corned beef - which had either been brought into the camp at its commencement, or had later arrived in the Red Cross supplies. These tinned foods were

reserved especially for such cases as chronic dysenteries with gross malnutrition, but at no time was there sufficient for them to be used for any but the most severe cases. The state of affairs as regards tinned and powdered milk was similar. For a short time in 1942 there was some block chocolate "emergency ration" available for the worst cases. Eggs were at most times unobtainable, but there was a period of some months at the end of 1942 and the early part of 1943 when they could be purchased from outside the camp. The hospital at one time owned a few hens but they, like everyone else, were underfed, and never laid well and ultimately succumbed to an outbreak of "fowl cholera". Generally speaking it was never possible to compensate for the protein loss in patients who were constantly passing large amounts of pus and mucus and there was usually a slow but steady deterioration in these cases. It was, however, surprising how many survived two or three years of chronic dysentery.

The supply of an adequate vitamin intake was indeed a problem. Green vegetables in large quantities were naturally contra-indicated, but it was found that they did not increase the diarrhoea unduly if taken in moderate portions provided no stalks were eaten. At times it was possible to buy from local sources outside the camp an edible legume, known as towgay or katjang-idjo*, which was rich in the vitamin B2 complex and

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* Phaseolus aureus.

was suitable for chronic dysenteries in the form of soup or as a purée. There was another legume which was unpalatable and at normal times not considered edible. These were known as "black beans"* and from them was produced a nauseating soup which was of some value as a source of the B2 complex. Synthetic vitamins were never available in large enough quantities even to treat all the severe cases of deficiency disease, so naturally they could not be used as a routine in chronic dysentery cases. The extracts made in the camp from rice polishings and inedible green leaves will be described in detail later.

Most of the time the general bulk of the ration was in the form of polished rice. This, of course, is an ideal low residue foodstuff, but in the absence of other sources of thiamin it could not be given in large quantities, even when it was plentiful, without producing beri-beri. On the rare occasions when undermilled rice was supplied, although the risk of beri-beri was less, there was apt to be an increase in the diarrhoea. For a short time in 1943 maize was issued in lieu of part of the rice ration. This was very difficult to prepare for chronic dysentery cases. It was so hard it could not be ground in the mills which had already been made

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* Centrosema pubescens

for rice-grinding. Soon after new and heavier machines had been made in the camp the maize ration ceased and never returned. Boiling the grain whole never made it soft unless it had previously been prepared, as in hominy, by soaking in a strong alkaline solution; and this tended to destroy the thiamin.

For another short period soya beans were supplied to replace part of the rice ration. These also were difficult to grind, but after the technical difficulties involved had been surmounted a coarse flour was produced on a small scale. This was cooked in the form of a gruel and given to the worst chronic dysentery cases. Soya beans boiled whole never became sufficiently soft to be of much value even to healthy individuals in whom they often caused diarrhoea. In chronic dysentery cases the beans were passed right through the alimentary canal unchanged in those who did not masticate them properly. Another preparation of soya beans, which was more palatable, was that which is known as tempé in Java. In preparing this the beans are first of all soaked and then dehusked (a very laborious process). Next, by innumerable washings the loose husks are removed, and the beans are then spread out in trays and sprinkled with the spores of a fungus (Rhizopus niger) which is capable of softening the hard cellulose of which the cell-walls of the bean are composed. After forty-eight hours

the beans are enveloped in a meshwork of grey fungus which forms a firm cake which can be fried or boiled. Many considered this tempé a delicious dish and it was said by some who had probably forgotten the flavour of civilised foods to be like sweetbreads. Others likened it to chicken. Unfortunately soya beans were never plentiful and there was only sufficient labour available to prepare tempé on a small scale. The result was that this, like most "extras", had to be reserved for the worst cases.

Sweet potato, small amounts of which were issued in the rations from time to time, was quite a good food for chronic dysentery cases when well boiled and made into a purée. This tuber was grown on a fairly large scale in the hospital camp at Kranji, but the yield was never very great. Another root-vegetable which was grown extensively in P.O.W. gardens as a "filler" food was tapioca root, but this was not very suitable for chronic dysenteries as it contained a large proportion of fibre and it was often not possible to boil it really soft. However, for the want of something better it was used after having been put through a mincing machine. It was possible to purchase ground nuts in small quantities for a short time at the end of 1942. These were a valuable addition to the diet of patients suffering from deficiency disease, and for dysentery cases they were husked and ground and made into peanut butter

or soup. Sago flour and tapioca flour were occasionally available for purchase but, whilst the former was eminently suitable for dysenteries, the latter unfortunately always contained so much sand that it did more harm than good.

That many methods of treatment were tried for the chronic bacillary cases between May and November 1942 is proof that none of them met with outstanding success. Mild aperients and astringents were usually unsuccessful. Kaolin was used in massive dosage but this seemed to increase the tendency to beri-beri, probably because the small quantity of thiamin in the diet was adsorbed by the kaolin. Native remedies such as infusions from the leaves of the guava tree proved quite useless. In cases with long continued diarrhoea sedatives were occasionally used to give some respite to the patient. Occasionally this resulted in improvement, but as a rule there was a recrudescence of the diarrhoea on discontinuing the sedative. Appendicostomy was performed on three cases during 1942, but this procedure resulted in no great improvement in the local or general condition.

Retention enemata were employed with definite advantage in many cases. This line of treatment produced best results in those with few motions daily as they were able to hold the enemata longer. Those whose bowels were irritable were given either tincture of opium or liquor morphinae by mouth a quarter

to half an hour before having the rectal infusion. In most cases it was an advantage to wash the bowel out with a sodium bicarbonate solution before giving the medication, but in some this made it more difficult to hold the retention enema. The retention enema was never more than eight ounces, was at blood heat, and was run in very slowly, usually with the patient in the genupectoral position. The patient was instructed to stay in this position for fifteen minutes after being given the enema and after that to lie on the right side for half an hour. After that he could lie on the back with the lower end of the bed raised on blocks. Men who had difficulty in retaining eight ounces were given smaller amounts - some as small as two ounces - and then gradually worked up to eight ounces. All solutions were begun at a weak concentration and worked up to the optimum therapeutic concentration by easy stages. Patients were encouraged to retain their retention enemata for as long as possible, and to this end competitions in which men vied with each other to see who could hold his enema the longer, were commonly observed in the ward. After a little practice many patients would retain an enema all day. Self-discipline played a great part in this line of treatment, and a course of retention enemata frequently gave a man more control over his bowel and reduced the diarrhoea. This often improved the general condition as it meant that longer time was allowed for

absorption from the bowel, but the sigmoidoscopic picture was sometimes disappointing in such cases as there might be no comparable local improvement.

For granulations oozing pus or superficial ulceration the most valuable type of retention enema was normal saline. Once the pus was diminished a suspension of iodoform in an emulsifying base was found to help these cases. Wherever there was oedematous mucous membrane, or a friable mucous membrane that was inclined to bleed, a solution of silver protein, starting at a concentration of 1:400 and working up to 1:100 was of definite value. Eusol even in weak solutions, such as one in eight, always caused a great deal of pain and generally seemed to do more harm than good. The author has sigmoidoscoped cases which had just previously voided a eusol enema. In these cases the mucous membrane was so inflamed and oedematous that it resembled acute dysentery. Tannic acid was tried in weak solutions but the results were not good.

In November 1942 the author started using "sulpha" drugs in the treatment of chronic bacillary dysentery. The compounds used were sulphanilamide (for one case only) and sulphapyridine. In July, August and September 1945 sulphadiazine was used also. All cases treated by chemotherapy were of long standing and had been under treatment by other methods without improvement. They were severe cases with extensive lesions visible through the

sigmoidoscope and the results of treatment by the sulphonamides were checked by sigmoidoscopy. Between the end of 1942 and September 1945 fifty cases of chronic bacillary dysentery were treated by this method. In 90% there was complete recovery both clinically and sigmoidoscopically. Two cases required a further short course of sulphapyridine before recovery was achieved and two relapsed later and finally cleared up once more on the same treatment. One case only, in the series, could be labelled as a failure. This case showed no improvement at all at the end of the course of sulphapyridine, and finally recovered after a course of iodoform emulsion retention enemata.

Illustrative Cases.

Cpl. I.B., S.S.V.F. had been a resident in Malaya for some years.
Previous Dysentery: In 1940. Amoebic and Bacillary.
In May 1942. Bacillary.
From 30th June 1942 to 28th November 1942 the number of stools daily averaged three. Stools consisted of solid faeces with large amounts of pus.
Sigmoidoscopy on 18th August 1942 showed extensive irregular ulceration of the rectum and lower sigmoid. Ulcers were discharging thick pus. Sigmoidoscopy was repeated in October after numerous methods of treatment had been tried and it was found that the condition was unchanged.
Sulphanilamide (1 gramme thrice daily) was given by mouth from 26th November to 12th December 1942. On the third day of treatment the number of stools fell to one, and remained at one daily until the patient was discharged from hospital on 23rd January 1943.
Sigmoidoscopy on 11th December 1942 showed that all ulcers had healed. There was no pus present. Sigmoidoscopy was repeated on 8th January 1943, when the mucous membrane was still normal. This patient remained well until December 1943 when he contracted

acute bacillary dysentery when working on the Burma-Siam Railway. He was treated as an acute case and made a rapid recovery with no recurrence of the chronic condition. Sigmoidoscopy after this acute attack showed the mucous membrane to be normal once more. After that he remained fit.

Sgn. W., Royal Signals, had had four attacks of acute bacillary dysentery in 1942 (in April, July, August and September). He was re-admitted to hospital in October 1942 having five or six stools with much pus daily. Sigmoidoscopy on admission showed extensive superficial ulceration of the rectum and sigmoid. Ulcers were discharging pus. Between then and February 1943 numerous methods of treatment were tried, but sigmoidoscopy in December 1942 and January and February 1943 showed no improvement in the condition of the bowel. Sulphapyridine (1 gramme every four hours) was given for nine days. Sigmoidoscopy performed a few days after the end of this course of treatment showed the mucous membrane of the rectum and sigmoid to be normal . The patient remained well.

Pte. P., of the Dutch Army, had an attack of acute bacillary dysentery while in Java in August 1942. After that he continued to pass three or four stools containing pus daily until February 1943 when he was admitted to the hospital in Changi. Sigmoidoscopy on admission showed large superficial ulcers, discharging pus, in the rectum and sigmoid. The intervening mucous membrane showed signs of chronic inflammation. Treatment by retention enemata of iodoform emulsion left the condition unchanged, and sulphapyridine (1 gramme every four hours) was given for one week. The daily number of stools fell to one after two days' treatment and no further pus was seen. Sigmoidoscopy performed one day after the completion of the course of sulphapyridine showed the mucous membrane of the rectum and sigmoid to be normal.

These are but a few typical examples of several cases treated successfully early in 1943. In the following case a shorter course of sulphapyridine was employed.

Pte. H., Suffolk Reg., had previous attacks of dysentery in April, June and October 1942. From October 1942 to February 1943 the daily number of stools was five or six. Sigmoidoscopy in February showed an inflamed mucous membrane with extensive ulceration discharging pus. One gramme of sulphapyridine was given four hourly for five days and on the sixth day sigmoidoscopy showed: "All ulcers completely healed except one or two which are obviously healing in the lower rectum. No pus present." Patient was discharged and reported back one month later having averaged one or two normal stools daily in the interval. He was sigmoidoscoped and it was found that the mucous membrane of rectum and sigmoid was normal. The patient remained well.

By the middle of 1943, after twelve or fifteen cases had been treated, it was concluded that the minimum effective dose of sulphapyridine was one gramme thrice daily for one week, and although this routine was adopted as the treatment of choice for chronic bacillary dysentery, reasons of economy forced one to reserve it for severe cases who had failed to respond to other methods of treatment. This in itself has the effect of underlining the outstanding success with which this line of treatment was met. The efficacy of sulphapyridine in chronic bacillary dysentery was not limited to cases resulting from any one epidemic, and it was, generally speaking, found to be equally effective throughout the three years following its introduction in cases which had started in widely separated areas such as Java, Sumatra, Malaya and Siam.

The following illustrations are drawn from cases which are typical of many which were treated with the "Sulphapyridine Routine".

Pte. W., of the Leicester Regt., contracted dysentery in Taiping, Malaya, in January 1942. Two months later he was still passing blood and mucus and having six stools daily. He continued to have from four to eight motions daily for fifteen months. The stool consisted at times of pure pus. On admission to hospital in Singapore at the beginning of April 1943 he was very thin and, although a tall man, weighed 8 stone 12 lbs. The sigmoid colon was thickened and tender, the liver was enlarged, the edge being palpable three fingers'-breadths below the costal margin. His vision was failing and the opthalmologist reported a retrobulbar neuritis of the Vitamin B2 deficiency type. Sigmoidoscopy performed on 7th April 1943 showed: "Numerous large superficial ulcers containing pus scattered throughout rectum and sigmoid. Ulcers surrounded by a red areola. Intervening mucosa normal. Micro:- Numerous pus cells and R.B.C's. Macrophages - a few. No organisms". During the next month a diligent search was made for entamoebae histolytiae in the stools and the deficiency disease was treated with Marmite. Sigmoidoscopy was repeated on 7th May and the condition was found to be unchanged. Sulphapyridine (1 gramme three times a day for 7 days) was started on 9th May. Sigmoidoscopy on 19th May showed: "At 8 cm. on the right wall is a red scar with some mucopus adherent. Ulcers all healed. Mucous membrane in other areas normal. Micro:- A few pus cells, epithelial cells and one or two macrophages present". Stool examinations continued to show an occasional pus cell until 28th May. After that stools were all normal. Sigmoidoscopy was repeated on 11th June, and the mucous membrane of rectum and sigmoid was then found to be normal. The patient was discharged from hospital on 4th August 1943. At that time he was having one normal stool daily. The liver edge was palpable one finger's-breadth below the costal margin, there were no ocular symptoms and the vision was normal. The weight was then 10 stone. The patient remained well.

Sgt. K., of the Dutch Army was first treated for dysentery in Java in 1943. During that year he had attacks of dysentery about every two months. The first three attacks were treated as bacillary dysentery, and it was then assumed that the infection was an amoebic one and the next two attacks were treated as such (there was no microscope in the camp, in which this treatment was carried out). The patient continued to have diarrhoea until October 1944 when he was admitted to the Singapore P.O.W. Hospital at Kranji. He was then having

five stools with blood and pus daily. During the next week or two twenty stools were examined microscopically without any evidence of an amoebic infection being discovered. Sigmoidoscopy was performed on 15th November 1945 and the report was:- "On the middle valve of Houston there is a mass of granulation tissue discharging thick pus. In the upper rectum and lower sigmoid there are several small ulcers with an irregular outline. These ulcers contain pus and on this being sponged away punctate haemorrhage appear. Micro: Pus cells +++. R.B.C's. ++. Macrophages +." Sulphapyridine (1 gramme thrice daily) was given for one week. After two days' treatment the daily number of stools fell to one and no more pus or blood was seen.
Three more sigmoidoscopies were performed and the results were as follows:-

29th Nov:- Granulation tissue on valve of Houston almost healed. Other ulcers completely healed.
6th Dec:- Granulation tissue now healed but new epithelium at this point bleeds readily.
27th Dec:- Mucous membrane of rectum and sigmoid normal.

The patient was then transferred to a surgical ward for treatment of a tropical ulcer and was there until September 1945 having had no further diarrhoea.

Pte. Z., also of the Dutch Army, had repeated attacks of dysentery while in Sumatra from 1943 onwards. In all he had had six attacks which were treated in the Camp Hospital, but he had almost continuous diarrhoea with much pus apart from these exacerbations. He averaged five or six stools daily. He came to Singapore in November 1944 and was admitted to Kranji Hospital at the beginning of December. Sigmoidoscopy on 6th December showed:- "Extensive ulceration of rectum and sigmoid. Ulcers are irregular in outline and contain pus. In the lower rectum they are of medium depth and higher up they are superficial. Intervening mucosa is red and oedematous. Micro: Pus cells +++. R.B.C's. ++. Macrophages +."
Sulphapyridine (1 gramme three times a day for seven days) was given, and the stools dropped to one daily and no more pus was seen after the third day. Sigmoidoscopy was repeated two weeks later and the mucous membrane of rectum and sigmoid was found to be normal. The patient's general condition was very poor, but he gradually improved and had no further diarrhoea.

Chief Officer R. of the British Merchant Navy had had amoebic dysentery in 1938 for which he had had a full course of treatment. He had had no further illnesses until 1942 when he had had an attack of acute bacillary dysentery in which, he stated, he had 50 stools with blood and mucus in one day. He had no further dysentery until 1945, but had a very large number of attacks of malaria. In January 1945 he had an extremely severe attack of acute bacillary dysentery in which he had up to 100 stools in 24 hours. He was treated with sulphaguanidine in the Camp Hospital at a Working Camp in Singapore. He continued to have diarrhoea and averaged seven stools daily until he was admitted to Kranji Hospital on 7th March 1945. He was emaciated but had oedema of feet due to beri-beri. The descending and sigmoid colons were palpable and very tender. Microscopic examination of stool showed: "Mucus. Pus cells ++. Macrophages".
Sigmoidoscopy on 14th March: "Catarrhal inflammation of rectum and sigmoid. Large irregular patches of superficial ulceration. Mucus, but no pus seen".
Sulphapyridine (1 gramme three times a day) was commenced on 17th March and continued for one week. Stools dropped to one daily on sixth day and after that there was no further diarrhoea. The patient then had a further relapse of malaria and after that a further sigmoidoscopy was performed. This showed the mucous membrane of the rectum and sigmoid to be normal. There was no further diarrhoea.

At the end of the series sulphadiazine became available and a few cases of chronic bacillary dysentery were treated with this drug, employing the same routine as had been used for sulphapyridine. The results were quite as good as those obtained with sulphapyridine.

Seaman A. of the Dutch Navy was ill with acute dysentery in June 1945 at a time when the shortage of sulphapyridine and sulphaguanidine was most acute. Although his was a severe infection which had been going on for a week with a maximum of 40 to 50 stools in 24 hours, it was only possible to use a modified course of 15 grammes of sulphaguanidine during the acute phase. The number of stools settled slowly to four or five daily - with pus occasionally

and sigmoidoscopy a month later showed very extensive chronic bacillary ulceration. (See Plate No. 7). Sigmoidoscopy was repeated after a course of retention enemata, and the report was, "In the mid and upper rectum are masses of granulation tissue from which beads of pus can be expressed. In addition there is extensive irregular ulceration, with pus adherent, in lower rectum and lower sigmoid. Intervening mucosa red and oedemated. Micro:- A few R.B.C's, W.B.C's +++. Epith. cells +. Macrophages +." The patient was then given one gramme of sulphadiazine thrice daily for one week. Number of stools fell to one daily on the third day and remained at that level.
Sigmoidoscopy one week after completion of the course of sulphadiazine showed: "All ulcers healed. Mucous membrane of rectum and sigmoid now normal. A trace of mucus present".
The patient was sent out to work on the hospital gardens and remained fit.

Sulphaguanidine, the only other member of the sulphonamide group in the camp, was never used by the author in the treatment of chronic bacillary dysentery. There was no existing evidence in favour of its efficacy in chronic cases, and it was not considered that an experiment would be a profitable expenditure of a drug which was always in very short supply, as a certain number of acute cases which had been treated with sulphaguanidine in full dosage had become chronic. In one case, an acute case of moderate severity (circa BO 30/24 hours), sulphaguanidine had been started at an early stage and 96 half-gramme tablets had to be given before the daily number of stools fell to five. Three weeks later the patient was still having three or four motions with much pus daily, and sigmoidoscopy showed that there were such large masses of

granulation tissue in the rectum that it was impossible to enter the sigmoid. These granulations exuded beads of thick pus from numerous pinhole openings on pressure with the end of the sigmoidoscope. It was thought at the time that there must be an underlying amoebic infection, but nearly thirty stools were examined during the next two months and nothing to suggest amoebic dysentery was ever found although macrophages were often present. It was nearly four months after the original acute attack before the rectal mucosa was completely healed in this case.

In concluding the section dealing with chronic bacillary dysentery it may be said that where the treatment of these cases presented a great problem in 1942, from 1943 onwards one had no doubt what the treatment should be. Generally speaking, the only limiting factor in their treatment was the need to conserve the dwindling stocks of sulphapyridine. Right up to the end, however, strict economy and rigid control of the drug had done their work so well that there were still a few tablets left when a small quantity of sulphadiazine became available in July 1945. It may well have been that had the latter drug been available earlier one might have obtained results which

which surpassed those obtained with sulphapyridine, but it is difficult to see how the improvement could have been very considerable.

CHAPTER V.

AMOEBIASIS.

Throughout the three and a half years of captivity there was a steady increase in the incidence of amoebiasis in Singapore. Originally it was considered that those whose only tropical existence had been on Singapore Island could not have been infected with the entamoeba histolytica, but during 1944 and 1945 there were many proved cases who had not only never been "off the island" but whose life had been spent for two or three years prior to infection entirely in the Prisoner of War Hospital either as patients or staff.

The position with regard to amoebic dysentery was the reverse of that which obtained with chronic bacillary dysentery. During 1942 there was an apparently adequate stock of specific drugs. Emetine, E.B.I. and Stovarsol were all available in fairly large amounts, and all cases which were diagnosed during that year had the same course of treatment, with one notable exception, which they would have been given in normal civilised conditions at that time. The exception was the quinoxyl compounds. These unfortunately were never available in the Prisoner of War Camp. Had they been so, perhaps many cases who received full courses of treatment with emetine followed by E.B.I. and one of the arsenical compounds would not have relapsed. As it was, a high

percentage of such cases ultimately became chronic and continued so after repeated courses of treatment followed by a short improvement and a subsequent relapse had uselessly reduced the stocks of anti-amoebic drugs to a dangerously low level.

During the whole period of captivity the Japanese medical authorities only once responded to frequent representations for an issue of specific drugs for the treatment of amoebic dysentery. This unique response consisted of a tube of twenty one-third grain tablets of emetine. Apart from this total of $6\frac{2}{3}$ grains of emetine (sufficient only for one case), the only other response to the repeated requests for drugs for the treatment of at least one hundred untreated cases of amoebic dysentery who were in the camp at that time was a smile and a shrug. Small quantities of emetine were purchased from time to time from native "black market" sources. The main supplies of specific drugs for the treatment of amoebic dysentery were those which had been originally taken into the camp in 1942. Unfortunately the economy practised with the drugs in 1942 and early 1943 was not as rigid as it might have been. In 1942 amoebic dysentery was a comparatively rare disease in the Prisoner of War Camp. In 1944-45 it was a comparatively common disease. The stock of E.B.I. was completely exhausted in 1943. During the whole

of 1944 and seven months of 1945 the stocks of emetine and stovarsol had dwindled to such a level that what remained had to be reserved with inflexible determination for cases in which the activity of the infection represented a danger to life, and withheld from those whose survival was unlikely because of intercurrent disease. As it is, it can be said that no man died for the want of emetine and that the author still had 4 grains of emetine in reserve when the Japanese surrendered.

The actual number of cases of amoebiasis diagnosed and treated in the main Prisoner of War Hospital in Singapore between February 1942 and September 1945 cannot be stated, but it is estimated that the number is certainly not less than 600. No estimate can be made of the incubation period of the disease from the material available.

Symptoms.

One's impression of the symptoms of cases of amoebiasis encountered in the Prisoner of War Hospital in Singapore is that they were variable in the extreme. One nevertheless carries in one's mind a picture of a few distinct types which will be described.

Firstly, and by far most commonly, there was the man who had intermittent diarrhoea. He might have a history of having been in hospital with five or six attacks of "dysentery" and

between these attacks there was usually a story of odd attacks of diarrhoea. But he was quite as likely never to have had anything sufficiently definite to be labelled "dysentery". There might be nothing more than a story of being easily "upset"; or of odd nights of diarrhoea. Often the patient was conscious of loud abdominal rumblings during the evening, and on going to bed there was difficulty in sleeping because of discomfort due to flatulent distension. On these occasions the latter part of the night might be spent in walking to and from the latrine. And in the morning all might be well again and the incident forgotten until there was a recurrence perhaps two weeks later. These men often found that a "chill" might bring on an attack. Perhaps in the heat of the early part of the night they might fall asleep with no covering on the abdomen, and later, when the temperature had fallen, wake up shivering. This was often sufficient to bring on quite a severe attack of diarrhoea. A "cold" day (i.e., one on which the temperature fell to about 78°F. - at 76°F., which was the lowest recorded, one felt extremely cold in Singapore) one always expected a general increase in the number of stools in the "Amoebic" Ward.

A subdivision of this type was the individual who had lived in the tropics for years, who often wore a woollen belt round the abdomen on the hottest days, and who had not known a

day free of diarrhoea for years. These men usually took a lot of seasoning in their food, and on most days would have to meet two or three sudden demands from the lower bowel. Experience had taught them that they could not safely temporise with these demands, and it was their custom to abandon whatever they were doing and dash without ceremony to the nearest place of shelter.

The second most common type of case encountered in Singapore was that in which the patient was having his first definite attack of dysentery. In these cases there was often no previous history of diarrhoea at all. The general appearance of the patient and the clinical signs were indistinguishable from those found in cases of acute bacillary dysentery. Even the naked eye appearances of the stools sometimes were not unlike those in a bacillary infection.

Constipation, either alternating with diarrhoea, or without any reference to diarrhoea, was occasionally the complaint of the patient suffering from amoebiasis. Sometimes in such cases there was a complaint of pain in the rectum after defaecation. In other cases a great deal of flatus was associated with constipation.

Another type of case was that in which the patient complained of no disordered action of the bowels at all. This was quite a common type. The complaint might be of "indigestion" or of "flatulence" or vague abdominal pains or attacks of colic.

Sometimes it was merely a loss of appetite or a general feeling of being "run down" and losing weight.

In some cases there was no complaint at all and the patient was unaware that anything was wrong with him until vegetative or cystic entamoebae histolyticae were discovered at a routine stool examination such as all food handlers were subjected to periodically.

Some cases who were symptomless, or at least had no symptoms of a bowel infection, had very extensive lesions in their bowels. From several cases of hepatic amoebiasis of various degrees it was impossible to elicit a history of bowel symptoms.

In the following table (Table 7) the incidence of the principal complaints in 150 consecutive cases of proven intestinal amoebiasis treated in the Prisoner of War Hospital is shown. Only one complaint, the "presenting" or dominant complaint, has been included for each case.

TABLE 7.

150 Cases of Amoebiasis.

Principal Complaints.	No. of Cases.
Intermittent Diarrhoea.	76
First attack of Diarrhoea.	25
Alternating Constipation and Diarrhoea	3
Constipation alone.	3
Passage of Flatus without Diarrhoea.	1
Vague abdominal pains.	10
Colic.	11

Table 7 Contd.	No. of Cases.
Griping pains during defaecation.	4
Rectal pain after defaecation	3
Central abdominal pain.	1
Loss of Appetite.	1
Loss of Weight.	4
Fever.	4
Rectal Bleeding.	1
No symptoms (Routine examination of stools)	3

It will be seen from Table 7 that in this series of cases of intestinal amoebiasis, diarrhoea was the principal complaint in only 69.3% of cases. Pain referred to some part of the bowel was complained of in 19.3% of cases. It is felt that "loss of weight" might have featured more prominently in other surroundings. In a community in which everyone was steadily losing weight, loss of weight was not likely to be mentioned unless very gross.

The incidence of constipation in these cases of amoebiasis is perhaps surprising. The classical "alternating constipation and diarrhoea" only occurred in 2% of cases and constipation alone was the complaint in the same number of cases.

Diarrhoea, as the commonest symptom of amoebiasis, is a term which is used to indicate anything from the passage of a few loose stools a day to a condition of severe dysentery with very frequent stools. The wide variation in the number of stools in cases of active intestinal amoebiasis is shown in Table 8 in which the number of stools being passed daily on

admission to hospital is recorded for a series of 100 cases. It will be seen that the variation is from 1 to 40 stools in 24 hours. The number of cases in which each number of stools was recorded is shown in the right hand column.

TABLE 8.

100 Cases of Intestinal Amoebiasis.

No. of Stools in 24 hours.	No. of Cases.
1	5
2	6
3	10
4	16
5	11
6	4
7	10
8	8
9	4
10	11
11	1
12	4
13	2
14	1
20	2
22	2
30	1
35	1
40	1

Summary.

Stools in 24 hours.	Percentage of Cases.
Below 3	11%
3 - 10	74%
11 - 20	10%
Above 20	5%

The character of the stools in amoebic dysentery varied within wide limits. The classical comparison with anchovy sauce was not frequently observed in the cases seen by the author in Singapore. In the series of 150 cases referred to in Table 7 only one was recorded as having "anchovy sauce stools". In general terms, a comparison between bacillary and amoebic dysenteries in Singapore would lead one to say that the amoebic stools were usually more bulky and had a larger faecal element. Very often in the cases which had intermittent attacks of diarrhoea the stool was mainly faecal with some mucus. In these cases a fairly common type of stool was one composed of a smooth faecal paste. Pus was commonly passed per rectum even in cases with no diarrhoea.

On the whole, amoebic stools contained more blood than bacillary stools. When blood was mixed intimately with pus to form a brownish pink emulsion the classical anchovy sauce stool resulted. More common than this was a mixture of altered blood and faeces which resulted in a dark chocolate-coloured stool of "sauce-like" consistency and which was particularly foul-smelling. In fact on examining stools or doing sigmoidoscopies one might make a provisional diagnosis with some degree of accuracy from this smell, alone when present. In nearly every such case the provisional diagnosis was later confirmed. In acute dysenteric cases the stools often were mistaken,

macroscopically, for bacillary stools, but in amoebic cases there was often more blood in the stools, and in some cases they consisted of what appeared to be pure blood. One such case literally bled to death in a few hours as what appeared to be pure venous blood poured from the rectum although no actual bleeding point could be found by sigmoidoscopy or at post mortem.

In many cases the stool consisted of liquid faeces of normal colour, with no visible blood, mucus or pus. In others an occasional fleck of blood might be observed in the liquid faeces. In chronic cases with constipation fairly large amounts of pure pus or blood-stained pus might be passed after a hard stool. It was fairly common for amoebic cases to have a constipated motion the first thing in the morning and immediately after to have an attack of colic followed by a copious loose motion containing mucus.

In Table 7 101 cases had diarrhoea. The naked-eye appearance of the stools in these cases are tabulated below (Table 9).

TABLE 9.

150 Cases of Amoebiasis.

	No. of Cases.
Blood and mucus.	53
Blood but no mucus.	13
Mucus but no blood.	11
Pus.	13
No blood, mucus or pus.	6.
Total number of cases with diarrhoea	101

The second commonest symptom in cases of amoebiasis was flatulence in some form. This point is not brought out in Table 7 because it was a common secondary symptom but seldom the principal complaint. It might take the form of frequent eructations after meals, especially in cases with a minor hepatic complication; or embarrassingly loud abdominal rumblings while at rest in the evenings; or the passage of surprisingly large volumes of flatus per rectum. This last was often accompanied by a "leak" of mucus.

Pain was not only a common dominant symptom as indicated in Table 7, but lesser degrees of it were also almost the rule in cases with severe diarrhoea. Colicky pains before defaecation, griping during the act and a dull sickening pain referred to some part of the colon after defaecation, were the common forms in most if not all of the severe chronic cases which had to remain without specific treatment throughout 1944 and 1945. As in bacillary dysentery, the pain appeared to be referred to the anatomical site of the lesion in the colon, i.e., right iliac fossa pain for caecal lesions, across the upper abdomen for lesions in the transverse colon, and, most commonly, left iliac fossa pain for sigmoid lesions. Pain due to rectal lesions was felt deep in the perineum and this type of pain often lasted for long periods after defaecation. Tenesmus was by no means uncommon in amoebic cases, and most of the untreated

chronic cases referred to above suffered agonies from this if left without analgesics.

Gross loss of weight, which even in that community was considered excessive, was the rule with the severe chronic cases who were deprived of specific treatment by circumstances. Emaciation in these cases was often masked by generalised oedema due to beri-beri or protein deficiency, both of which were common complications of chronic dysentery in 1944 and 1945.

The clinical signs in amoebiasis were both vague and variable. There was no sign which was sufficiently definite or common to be regarded as even suggestive, let alone diagnostic of amoebic dysentery. The only sign common to a large number of cases was an abdominal tumour in the region of some part of the bowel, but even this was only found in a minority of cases. The liver was palpable in some cases and tender in only a few.

In Table 10, which is an abstract of the same series of cases referred to in Table 7, it will be seen that only a small percentage of cases had parts of the colon which were sufficiently thickened to be palpable. Hepatic signs are also included in the table.

TABLE 10.

150 Cases of Amoebiasis.

Colon thickened or tender.	No. of Cases.
Sigmoid Colon.	22
Ascending Colon.	13
Caecum.	7
Descending Colon.	2
Transverse Colon.	1
Liver enlarged.	9
Liver tender.	3

Even in the absence of complications pyrexia was a common sign in cases of acute amoebic dysentery. The temperature was not usually so high, however, as in cases of acute bacillary dysentery.

The microscopic appearances of the stool varied greatly with the stage of the disease. In some there might be no exudate at all and the search for "E.H." cysts was almost as laborious as looking for the proverbial needle in a haystack. In cases of acute amoebic dysentery the stool consisted almost entirely of blood and an exudate which was literally swarming with actively moving vegetative entamoebae histolyticae. The recognition of even a lone, sluggish vegetative "E.histolytica" was comparatively easy to the experienced observer but to explain the reasons for one's certainty that the particular specimen was an "E.histolytica" was often more difficult.

The importance attached to the freshness of stools for examination and the selection of a suitable part by the

microscopist has already been referred to in the section on bacillary dysentery. Out-patients suspected of amoebiasis were always made to attend at the dysentery wards and pass a stool into a bedpan which was immediately taken to the laboratory. In the chronic dysentery ward where most cases of amoebiasis were diagnosed it was found necessary to have a microscopist permanently on duty or on call so that difficult cases might have every stool which was passed examined immediately.

A suitable piece of exudate having been picked up in a platinum loop, it was thoroughly mixed with a drop of warmed normal saline on a slide and a cover slip was applied. The slide was always first examined under a 2/3" objective because amoebae and cysts could often be seen as points of bright light with this power. On turning on the "high-power" the first thing that usually struck one in an amoebic exudate was the myriads of motile bacilli which often appeared to make the whole field quiver. Red blood cells were usually present in large numbers, and were either clumped or arranged in rouleaux. Pus cells were also usually present in large numbers and these were usually scattered throughout the field. Damaged epithelial cells were commonly present. The presence of macrophages, although rare in this type of exudate, was not unknown. Charcot-Leyden crystals were commonly seen in amoebic and

and indefinite exudates. Ones impression is that, generally speaking, they were commoner in exudates in which there were not excessive numbers of red blood corpuscles. Although these crystals could not be regarded as proof of anything but, perhaps, excessive destruction of eosinophils, their presence is undoubtedly a useful piece of evidence in favour of a diagnosis of amoebiasis.

An exudate such as that which has just been described might contain E.histolyticae in sufficient numbers to make prolonged search unnecessary, but more often than not the arduous task of searching field after field until the entire slide had been scrutinized had to be undertaken. The active, vegetative form of the organism was usually three to four times the diameter of a red blood corpuscle in size, but in many the minuta form, which was half this size or less, was seen. the pseudopodia were usually clear and colourless although occasionally they had a greenish tinge. The endoplasm on the other hand was finely granular and greyish in colour. It contained vacuoles and very commonly ingested red blood corpuscles; but it was often difficult to see the nucleus clearly in actively moving amoebae. In more sluggish examples the nucleus with its dotted outline of granules of chromatin and the central spot or karyosome was more readily observed. The much smaller, inactive, precystic form of the organism was commonly seen.

The search for cysts of E.histolytica in the faeces of a patient suspected of amoebiasis but passing normal stools was a process which required great patience. (Fortunately the microscopists who worked with the author were mostly Anglo-Indian Assistant-Surgeons of the Indian Medical Department who were well endowed with patience and if they were bored with tedium they never showed it). It was felt that the discovery of cysts in these cases was made easier by giving the patient a brisk purge. In cases of difficulty the faeces were emulsified in normal saline and centrifuged, or an attempt was made to concentrate the cysts by sedimentation. Slides were usually examined unstained in the first instance and then stained with Weigerts iodine and re-examined. All stages, from the uninuclear precystic form to the mature, four-nucleated cyst were recognised. Under a low-power objective the unstained cyst was often seen as a minute, bright point of light when the microscope was not quite in focus. These cysts varied greatly in size from those which were a shade smaller than a red blood corpuscle to those which were two or nearly three times that size. They stained well with iodine and were then seen to have a very thin cell-wall, four distinct nuclei, some of which might not be visible until one had focussed up and down as all might not be in the same plane. Chromatin bars, usually two in number, were commonly, but not invariably, visible in the E.H. cysts observed.

The microscopic examination of stools of patients suspected of intestinal amoebiasis was carried out by experienced and thorough microscopists. In the early stages the author dispensed with the services of one or two individuals who were considered not to be sufficiently painstaking. Nevertheless it soon became obvious that it was quite futile to examine one or even two or three stools from a suspected case and, on these proving negative, to pronounce that the case was not one of amoebiasis.

At the end of 1942 the author surveyed the cases of proven amoebic dysentery which were at that time in hospital, in order to form some idea of what was the minimum number of negative microscopy reports which could be admitted as evidence against a diagnosis of amoebiasis. This survey covered 50 cases (5 cases which were diagnosed sigmoidoscopically were not included), and in these the average number of careful microscopic examinations of the stools which were performed before E.histolyticae were discovered was 6.5.

TABLE 11.

50 Cases of Amoebiasis diagnosed microscopically.

No. of examinations before discovery of E.H.	1	2	3	4	5	6	7	10	11	12	15	16	17	20
No. of Cases.	6	6	7	6	2	4	4	3	2	4	2	2	1	1

It will be seen from Table 11 that only 50% of these cases were diagnosed in four or less examinations, and that over 15 cases out of 50 required ten or more examinations. The number of microscopic examinations of stools required in one case in the series before E.histolytica was discovered was twenty, but even this compared quite favourably with a fatal case (the patient actually died of pulmonary tuberculosis) whose stools were meticulously scrutinised on no less than thirty-nine occasions without E.histolytica being found. At post mortem, however, typical amoebic ulcers were demonstrated in the colon.

The results shown in Table 11 were checked later on over a much larger series of cases. The exact figures are not now available, but the average number of microscopic examinations of stools required in the later series was 7, which confirms that the small series of cases which provided the material for Table 11 was fairly typical.

Diagnosis.

With such a state of affairs as has just been described it was obvious that some further aids to diagnosis had to be sought. The usual method of resorting to specific treatment such as emetine injections and noting the response in cases in which the clinician had despaired of obtaining a firm diagnosis

by more accurate methods was not one which could be used in more than a few cases owing to the shortage of specific drugs. For the same reason "provocative" emetine could not be used. Sigmoidoscopy was, of course, the principal aid to diagnosis, and after the first few months of captivity, all cases of suspected or proved amoebiasis were sigmoidoscoped. Nevertheless, valuable as the results of the examination were, there remained a large number of cases in which the sigmoidoscopic appearances were equivocal. Out of 530 cases of intestinal amoebiasis, for instance, 76 were found on sigmoidoscopy to have a normal mucous membrane in both the rectum and the lower sigmoid colon. It was comparatively easy for the experienced sigmoidoscopist to recognise "acute amoebic ulceration", but in many of the more chronic cases (which, after all, were those in which difficulty in diagnosis was more likely to be experienced) the ulceration was of such a nature that no dogmatic assertion could be made as to its cause. Of course, if vegetative or cystic forms of E.histolytica could be found in scrapings from ulcers the diagnosis was clinched at once, but a survey of a series of 100 consecutive sigmoidoscopies in which vegetative forms of the organism were actually found in such scrapings, will itself show how varied and inconclusive the sigmoidoscopic appearances could be (see Table 12).

TABLE 12.

100 Consecutive Sigmoidoscopies in which Vegetative E.H. were found in Scrapings.

Sigmoidoscopy Findings.	No. of Cases (%)
Mucous membrane of Rectum & Sigmoid normal.	1
Mucous membrane normal but much mucus present.	1
Generalised inflammation of mucous membrane.	8
Patchy inflammation of mucous membrane.	5
Submucous Haemorrhages - Punctate.	3
Submucous Haemorrhages - Larger.	1
*Inclusion Polyps.	1
Polypoid Stricture.	1
Ulcers.	
Discrete Superficial Ulcers.	9
Irregular Superficial Ulcers.	5
Punched out Ulcers.	41
Oval Ulcers.	9
Crescentic Ulcers.	1
Deep Ulcers with punched out edges.	8
Deep Ulcers with Polypoid edges.	4
Deep Ulcers with undermined edges.	3
Deep Ulcers connected by tunnels.	1
**Acute Amoebic Ulceration.	23
***"Teed-up" Ulcers.	9
Ulcer surrounding bowel with tendency to stricture formation.	4
Pinhead Ulcers.	7
Punctate Haemorrhages surrounding Ulcers.	9
****Red Areola surrounding ulcers.	34
Masses of Granulation tissue oozing pus.	1

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*Polypi containing a core of inspissated faeces, usually with several apertures the size of a pinhead. It is thought that these polypi are caused by the mucous membrane closing over faecal debris in an undermined ulcer.

**Acute amoebic ulceration: very superficial, minute, discrete ulcers which by confluence might form patches of irregular ulceration with many bleeding points. Greyish-white mucopus was usually adherent to parts of the ulcerated area. The surrounding mucosa was generally inflamed. Streaks of dark blood and mucus were often adherent to the area, and

occasionally streaks of thin, yellowish pus and serum covered the surface. Areas of acute amoebic ulceration were frequently extensive and were inclined to bleed copiously during the examination. Scrapings almost invariably contained vegetative E.histolylicae in large numbers.(See Plate 1A).

*** "Teed-up" ulcers:- By this phrase the author meant a type of ulcer which resembled a golf ball on a sand tee. These ulcers were on papules of mucous membrane with sloping sides. Although the ulcer was actually a crater in the papule, the sides of which were undermined, it was usually full of white pus, hence the "golf ball" appearance. (See Plate 2).

**** "Red Areola":- A zone of inflamed mucosa around an ulcer when the intervening mucous membrane was of normal colour. (See Plate 2).

In one respect Table 12 may give an erroneous impression. There was only one case in which the mucous membrane of the rectum and lower sigmoid colon was normal. This is fairly natural as the table only contains cases in which vegetative E.histolytica was found in material obtained during the sigmoidoscopy. In this case the organism was found in a smear of mucofaeces which was adherent to the bowel wall.

It will be seen that 8 cases out of 100 had generalised inflammation of the mucous membrane, whilst only 5 cases had patchy inflammation which in itself was considered more suggestive of an amoebic infection. The cases in the series were mostly men who had had amoebic dysentery for a long time, and the probability is that the generalised catarrhal inflammation was caused by the irritation of chronic diarrhoea.

Punctate submucous haemorrhages, which were only reported in 3 cases, were generally considered by the author to be highly suggestive of an amoebic infection. Of the ulcers, the second group, namely, "irregular superficial ulcers" (this does not include the picture described as "acute amoebic ulceration") were of a type more suggestive to the author of a bacillary infection. Nevertheless vegetative E.histolyticae were present in the scrapings. This type of ulceration was seen in 5 cases out of this series of 100 and also in 5.8% of the case in the larger series (Table 13). The remainder of the ulcers were consistent with a naked-eye diagnosis of amoebic dysentery, but even in the case of the predominant "punched-out" ulcer it was often difficult to state categorically the nature of the infection. If was, of course, a definite point in favour of an amoebic infection if the intervening mucosa was absolutely normal apart from the red areola.

For the sake of comparison with Table 12 which is composed from cases all of which were sufficiently active for vegetative E.histolytica to be found in scrapings taken at the present sigmoidoscopy, the results of sigmoidoscopy on 530 cases of amoebiasis have been tabulated to form Table 13.

TABLE 13.

530 Sigmoidoscopies on Cases of Intestinal Amoebiasis.

Sigmoidoscopic Findings.	No. of Cases.	%	Table 12.
Mucous membrane of Rectum & Sigmoid normal.	76	14.3	1
Mucous membrane normal but much mucus present.	31	5.8	1
Generalised inflammation of mucous membrane.	43	8.1	8
Patchy inflammation of mucous membrane.	46	8.6	5
Loose folds of mucous membrane stuck together. with dry mucus.	13	2.4	
Submucous Haemorrhages (a) Punctate.	19	3.5	3
(b) Larger.	28	5.2	1
Mucous membrane stippled with minute depressions.	2	0.3	
Submucous nodules (millet-seed bodies).	5	0.9	
Pigmented scars.	14	2.6	
Inclusion Polyps.	9	1.7	1
Polypoid Stricture.	7	1.3	1
Ulcers:-			
Discrete Superficial Ulcers.	49	9.2	9
Irregular Patches of Superficial Ulceration.	31	5.8	5
Punched-out Ulcers.	95	17.9	41
Oval Ulcers.	14	2.6	9
Crescentic Ulcers.	1	0.2	
Deep Ulcers.	40	7.5	8
Deep Ulcers with polypoid edges.	19	3.5	4
Polypoid mucous membrane with ulcers or polyps.	7	1.3	
Deep Ulcers with undermined edges.	24	4.5	3
Deep Ulcers connected by tunnels.	5	0.9	1
Acute Amoebic Ulceration.	48	9.0	23
"Teed-up" Ulcers.	14	2.6	9
Ulcer surrounding bowel wall with tendency to stricture formation.	10	1.9	4
Pinhead Ulcers.	32	6.0	9
Rectangular Ulcers.	1	0.2	
Punctate haemorrhages surrounding ulcers.	8	1.5	9
Ulcers surrounded by Red Areola.	99	18.6	34
Amoeboma.	4	0.7	
Masses of granulations oozing pus.	9	1.7	1

Table 13 indicates in some measure how difficult it often was to make a diagnosis from sigmoidoscopic findings unless E.histolytica, in some form, was found in the material taken from the bowel. As time went on one's opinion altered as, on one hand, vegetative forms were repeatedly found in a type of ulceration which had not previously been thought to be of amoebic origin; and, on the other hand, a lack of response to specific treatment was noted in a severe type which one had thought to be definitely amoebic. But when considering an absence of a favourable response to treatment one had to bear in mind the fact that numerous cases of undoubted amoebic ulceration resisted repeated full courses of specific treatment. However, it must be confessed, that cases of a type which the author was courageous enough in 1942 to call definitely amoebic on the sigmoidoscopic appearances alone, were by 1945 (after some 5,000 sigmoidoscopies had been performed) regarded as snares for the unwary.

The large incidence of normal findings in this series has already been mentioned. The importance of generalised inflammation of the mucosa is possibly exaggerated not only for the reasons already mentioned, but also because some of these cases had already started their treatment. A course of retention enemata often caused an appearance similar to catarrhal inflammation of the mucosa. A state of patchy

inflammation with minute ulcers or submucous haemorrhages on the inflamed patches was considered highly suggestive of an amoebic infection. This appeared to be a common mode of onset of the disease.

Of submucous haemorrhages, the punctate form was regarded as probable evidence in favour of an amoebic condition. Larger submucous haemorrhages, it will be noticed, were more common in this series, but these were never so confidently assessed, as large submucous haemorrhages were often seen in cases which were probably of bacillary origin. A mucous membrane stippled with minute depressions, it was felt, was probably a condition of scarring due to previous amoebic ulceration of the type which results from the rupture of submucous nodules. Pigmented scars (usually of a deep red colour) resulted from both amoebic and bacillary ulceration, but they appeared to last much longer in some amoebic cases.

"Inclusion polyps" have been defined in a footnote to Table 12. The polypoid stricture was probably a residual sign of an ulcer which had encircled the bowel. These usually appeared to be inactive. Most of these cases were watched over a period of months or years and some were dilated with bougies through the sigmoidoscope (or if low down without that instrument).

Of the ulcers, the first two groups were present in a percentage remarkably close to the figures shown in Table 12.

So also does the aggregate of the deep ulcers when expressed as a percentage closely follow the figures in Table 12. Punched-out ulcers, acute amoebic ulceration and "Teed-up" ulcers (see definition in footnote to Table 12) were relatively far more common in the more active series shown in Table 12. This is just what one would expect. The same applies to the "red areola" and punctate haemorrhages surrounding ulcers, both of which the author regarded as signs of the activity of the infection. Pinhead ulcers which were tiny ulcers, usually containing beads of white pus, were often a later stage of the "millet-seed body" and probably resulted from the rupture of the submucous nodule. In other cases, however, the pinhead ulcers were found in the middle of patches of inflamed mucosa. It certainly was a very early stage of the infection.

By amoeboma is meant a fungating tumour-like mass resembling a neoplasm. These were sometimes mistaken for carcinomata of the rectum. The last group of cases, namely, those having masses of granulating tissue oozing pus, were indistinguishable from chronic bacillary dysentery. This picture was probably the result of a secondary infection.

It will be seen from the above survey of sigmoidoscopies that the picture presented by cases of amoebic dysentery was not always clear cut and unmistakable. For this reason, early in

1943 the author, casting around for some other aid to diagnosis, decided to investigate the pH of stools of patients suffering from both types of dysentery using a series of normal controls. It was realised that the results of this investigation would only apply to subjects on diets similar to the low protein, high carbohydrate diet which was that of the Prisoner of War Camp. The results were at first promising but after doing a fairly large series of cases the results were inconclusive, and the most that could be said for this method was that it indicated certain cases as requiring further investigation by other means.

The technique employed was to make a watery suspension of the stool, a drop of which was then tested on a white tile with the Universal Indicator made by the British Drug Houses, a bottle of which was in the Camp. The following series of cases were tested in the initial experiment: 100 cases of intestinal amoebiasis including both active and quiescent cases: 100 cases of bacillary dysentery: 100 "normal" controls (R.A.M.C. personnel).

The results were as follows: The vast majority of stools of persons living on the Prisoner of War rations were definitely alkaline. A certain number of stools which were alkaline immediately they were passed became acid in two hours. All stools which were acid either immediately or in two hours

were those of persons who were actively suffering from amoebic dysentery; or who had suffered from that disease; or who were later proved to be suffering from it; or in whom there was strong presumptive evidence such as the combination of Charcot-Leyden crystals in the stools and typical amoebic ulcers in the rectum or sigmoid. On the other hand very far from all cases of active amoebic dysentery had acid stools, and even those in whom this reaction was found did not constantly pass stools which were or which became acid.

Out of 100 cases of intestinal amoebiasis the stools in 22 had a pH of less than 7 immediately they were passed, whilst a further 6 which had been alkaline at that examination were acid two hours later. In the remaining 72 cases the stools were alkaline in two hours and still alkaline twenty-four hours later. Vegetative E.histolytica were present in many stools which were, and remained, alkaline.

100 cases known to be suffering from bacillary dysentery were tested. The stools in all cases were alkaline on being passed and still alkaline two hours and twenty-four hours later.

100 apparently healthy controls were tested. In 98 the stools were alkaline immediately, in two hours and in twenty-four hours. In the remaining two the stools, having been alkaline immediately, became acid in two hours. These two cases were then investigated. Both were quite well and neither

had any history of diarrhoea. In one, cystic forms of E. histolytica were found in the stools after repeated microscopic examinations. The second proved negative, but was found on sigmoidoscopy to have discrete superficial ulcers scattered throughout both the rectum and the lower sigmoid colon. These ulcers contained white muco-pus and were surrounded by a red areola. The intervening mucous membrane was normal. No E. histolyticae were found in the scrapings at repeated examinations, but the ulcers completely cleared up on emetine-E.B.I.-Stovarsol treatment.

The practical application of the information obtained from the experiment was not as great as had been hoped. As long as the "Indicator" lasted the stools of cases of suspected amoebiasis in whom microscopy or sigmoidoscopy had proved inconclusive were tested. A pH of less than 7 immediately or in two hours was regarded as an important piece of evidence in favour of a diagnosis of amoebiasis, and linked with some other finding which in itself was inconclusive such as Charcot-Leyden crystals in the stools, or ulcers suggestive of amoebic infection in which the organism could not be found, one felt sufficiently confident of the diagnosis to venture some scarce specific treatment on the case. A pH of more than 7 was regarded as a valueless negative finding and the test was carried out on subsequent stools. Quite often a case would produce an acid

stool (or one which became acid in two hours) at the fourth or fifth test. In one case which was proved to be a mixed bacillary and amoebic infection 9 alkaline stools were followed by one which became acid in two hours. In several cases of this kind vegetative E.histolyticae were observed in both acid and alkaline stools, but bacillary exudates were never observed in acid stools.

Complications.

Most of the intercurrent diseases which have already been mentioned in the section on chronic bacillary dysentery complicated cases of amoebic dysentery also. Acute bacillary dysentery was a common and dangerous complication in cases of chronic amoebic dysentery.

Of the true complications of amoebic dysentery, the commonest was hepatic amoebiasis. Amoebic hepatitis was comparatively common and occurred in many cases in which there was no history of a definite attack of dysentery, although there was invariably a history of diarrhoea. The onset of this complication, if acute, was accompanied occasionally by rigors but more commonly merely by fever and pain at the right costal margin and in the epigastrium. It was more often subacute with general malaise, an occasional rise in temperature, dull pain at the costal margin, loss of appetite and flatulence. On examination there was rarely any appreciable jaundice. The

tongue was furred and the complexion was sallow. The liver was commonly enlarged downwards being as a rule two or three fingers' breadths below the costal margin. The liver was usually tender.

Amoebic liver abscess was a rare disease in the Singapore Prisoner of War Camp. Only one case was diagnosed during life and successfully treated surgically. This case had no history of dysentery or even diarrhoea. No abnormality was found in the stools or sigmoidoscopically. In addition to this there were two fatal cases of amoebic liver abscess, one of which had the following interesting history.

> Pte.P., a British soldier, had escaped while working on the Burma Railway in Siam. For some weeks he wandered about in jungle, living in various native villages. He was then re-captured and held by the Japanese Military Police in their camp in Siam, and while there was well treated and made to work in the cookhouse. While there, in December 1942, he had an attack of severe diarrhoea which the patient himself thought was due to over-eating after his long period of semi-starvation. Early in 1943 he was moved to Singapore and placed in solitary confinement in Outram Road Gaol on a diet of small quantities of rice and water. At the end of September 1943, after he had been seriously ill in his cell for some weeks without medical attention, he was moved to the Prisoner of War Hospital at Changi. He had had no diarrhoea since the attack in December 1942, but he had had severe abdominal cramps for two months before admission to hospital. On arrival at Changi he was grossly emaciated and was found to be suffering from purpura, oedematous and neuritic beri-beri, exfoliative dermatitis, tropical ulcers and pressure sores. He was placed in a General Medical Ward. He had no diarrhoea while in hospital and did not have a stool examined. The temperature was normal throughout. On 24th October 1943 the patient complained of pain in right side of the chest. On 5th November 1943 diminished breath sounds at the base of the right lung were recorded. The patient died on 15th November 1943.

Post mortem Report:
Peritoneal Cavity: Yellow pus in right para-colic gutter. Omentum adherent to caecum and ascending colon. Yellow pus leaking from abscess on upper and outer surface of the right lobe of liver in mid-axillary line.

Colon: Very extensive amoebic ulcers of caecum and splenic flexure. Ulcers at splenic flexure 1½ inches long with darkly pigmented necrotic bases, and undermined edges. Between the large ulcers were small button-like ulcers. Intervening mucosa was normal.

Liver: No adhesions over abscess. Abscess in upper lateral portion of right lobe contained 250 c.c. of yellow pus. Walls ragged with well defined fibrous capsule except at point of rupture.

Microscopy of Pus: R.B.C.'s. leucocytes. Liver cells. Charcot-Leyden crystals. No E.H. seen.

Fatal Cases:

There were 29 fatal cases of amoebic dysentery in the main Prisoner of War Hospital in Singapore in the three and a half years under review. 11 cases died as a result of a super-added acute bacillary dysentery. Of the remaining 18 cases none died of uncomplicated amoebic dysentery. The principal complications which could be regarded as the cause of death in these cases are recorded below (Table 14).

TABLE 14.

Cause of Death in 18 cases of Amoebic Dysentery.

Complication.	No. of Cases.
Liver Abscess.	2
Perforation of the bowel.	1
Pulmonary Tuberculosis.	4
Lung Abscess.	1

Cardiac Failure	(a) Following Laparotomy	1)	
	(b) Due to Beri-beri	1)	3
	(c) Whilst under Anaesthetic	1)	
Malnutrition and Deficiency Disease			2
Necrosis of Adrenals			1
Faucial Diphtheria			1
Lobar Pneumonia			1
Polycythaemia Vera			1

In 5 cases the records at present available do not state which parts of the bowel were found to be affected by amoebic ulceration at post mortem, but in the remaining 24 fatal cases the distribution of ulcers is indicated in the following table (Table 15).

TABLE 15.

24 Fatal Cases of Amoebic Dysentery.

Position of Amoebic Ulcers.	No. of Cases.
Ileum	2
Caecum	6
Ascending Colon	9
Transverse Colon	8
Descending Colon	6
Sigmoid Colon	8
Rectum	9

Cases of Interest.

Dvr.G., R.A.S.C. died in a Surgical Ward where a diagnosis of carcinoma of the rectum had been made. He had had only one attack of diarrhoea one month previous to admission to hospital. This attack had lasted 12 days and during the attack he had the sensation of "something coming down" at the anus. He had lost a great deal of weight fairly rapidly and complained of pain in the hypogastrium and much flatulence. On admission he was running a temperature. He had no diarrhoea while in hospital. On examination a swelling was found over the symphysis pubis and a constricting mass could be felt per rectum. The swelling over the symphysis pubis gradually disappeared. The patient died five weeks after admission to hospital.

Post Mortem Report: A large pelvic abscess was present. There were many adhesions in the pelvis. In the colon there was marked evidence of chronic amoebic dysentery. In the rectum a faecolith had ulcerated through the bowel wall, become covered with epithelium and later adherent to the sacrum with the formation of inflammatory fibrous tissue.

Pte.D., Gordon Highlanders, was admitted to hospital suffering from B.T. malaria on returning to Singapore from Siam where he had been working on the Burma-Siam Railway. There was no history of previous diarrhoea, but the patient had had diarrhoea for two days before admission. He was admitted on 1st January 1944. On 4th January blood and mucus appeared in the stools and vegetative forms of E.histolytica were found in the first specimen examined. He was transferred to the Dysentery Ward. He had 12 stools containing much blood during the first 24 hours in that ward. The patient was pale but not in pain. The temperature was 100.2°F. and the pulse 100 per minute. The spleen was very large. The patient was in the middle of a course of treatment for malaria. Emetine (gr.i by injection daily) was started on 4th January 1944 and continued for six days. Thereafter Stovarsol (gr.iv twice daily) was started. On 9th January 1944 early signs of lobar pneumonia were observed and sulphapyridine treatment was commenced. The blood count at that date was : R.B.C. 2,200,000 per cu mm. Haemaglobin 49%.
On 15th January 1944 patient developed progressive oedema which quickly progressed to a condition of gross anasarca with ascites and hydrothorax. The patient died on 22nd January 1944.

Post Mortem Report:

Lungs:- 1. Hydrothorax. 2. Pleurisy and lobar pneumonia.

Heart:- Pericardial transudate.

Abdomen:- 16 oz. free ascitic fluid.
Spleen:- 4 times normal size.
Ileum:- In the last three feet were 5 typical amoebic ulcers with undermined edges, varying in size from 0.5 cm. to 3.0 cm. These ulcers contained "Dyak Hair" sloughs.

Caecum and Colon:- There were about twenty typical amoebic ulcers varying in size from a few millimetres to one 10 cm. long in the caecum. Ulceration was predominant in the caecum, splenic flexure and adjacent transverse colon. There was no ulceration in the rectum or sigmoid colon.

Gnr. H., Royal Artillery, was admitted at 3 a.m. on 20th December 1943 immediately on arrival from Siam. He had had diarrhoea almost continuously for six months while in Siam. He was having four to six stools daily during the last few weeks, and the stools contained much blood. He had been under treatment for malaria for 6 days prior to admission (i.e., in crowded freight wagon on train). On admission the patient complained of severe pain in the back (lower thoracic and lumbar regions). There was no history suggestive of haemophilia. On examination the temperature was normal. The patient was extremely emaciated with large ecchymotic patches of purpura scattered over the body. There was marked tenderness in the left iliac fossa. A blood slide was negative for malarial parasites. The stool contained much blood, and numerous vegetative forms of E.histolytica were present. By 11 a.m. the patient had had six stools containing large amounts of blood since admission. Pain in the back was very severe. The patient was given 1 grain of emetine and $\frac{1}{4}$ grain of morphia, and a large haematoma formed at the site of the injection. At 4 p.m. the patient was passing large quantities of blood per rectum and a proctoscope was passed, but no definite bleeding point could be seen. Morphia (gr.$\frac{1}{4}$) was repeated. The patient continued to pass blood per rectum almost continuously until he died at 5.30 p.m. During the last 20 minutes what appeared to be pure venous blood poured from the rectum.

Post Mortem Report:-

Heart:- Normal, but no blood present in chambers or in great vessels.

Abdomen:- 8 oz. of free blood-stained fluid.
Numerous large subperitoneal haemorrhages on colon, ileum and mesenteries. These were especially extensive on the posterior abdominal wall where retroperitoneal haemorrhages formed large clots extending down into the pelvic fossa.
The spleen appeared normal.
In the ileum there was free blood in the lumen. There were several patches of submucous haemorrhage.
The colon and rectum were full of blood.

No faecal material was present. Small, typical, apparently early, amoebic ulcers were scattered throughout the caecum, lower pelvic colon and upper rectum.
There were numerous large submucous haemorrhages in the colon. In the floor of a few amoebic ulcers were thrombosed vessels.

The conclusion reached about the case was that the patient was suffering from acute amoebic dysentery and a purpura haemorrhagica which, it was thought, was secondary to a deficiency of Vitamin K.

Treatment.

In 1942 and early 1943 the treatment of amoebiasis was more or less orthodox. The routine treatment was a course of ten one-grain injections of emetine followed by emetine-bismuth iodide (3 grains daily for ten days). This was followed in most cases by one of the organic arsenicals (stovarsol, carbarsone, amibarson, treparsol). The routine course of stovarsol consisted of four grains twice daily for ten days. The quantities of drugs of the quinoxyl group in the camp was so small from the beginning that they could never be used as a routine measure. About half a dozen selected cases were treated with yatren enemata. As a substitute, retention enemata of quinine (1:1000) was used without outstanding success.

The immediate results of the emetine - E.B.I. - stovarsol routine were apparently good in the majority of cases, but the

relapse rate taken over the next three and a half years was high. Apart from this there was an appreciable proportion of cases in which this line of treatment could be regarded as a failure even when judged by the immediate response. These failures fell into three groups. First, there were those cases which recovered clinically but continued to pass cysts of E.histolytica. Second, there were those cases in which there was a marked symptomatic improvement but in which the sigmoidoscopic picture did not return to normal. In these cases the stools sometimes continued to contain pus and mucus, but E.histolytica was seldom found. Finally there was a small group in which the symptomatic improvement was slight or absent, and in which there was no local improvement in the parts of the bowel accessible to the sigmoidoscope.

There was a limited quantity of novarsenobenzene (N.A.B.) in the camp, and as the number of cases of syphilis was small and there was little prospect of its incidence increasing, the author suggested that a solution of 10% N.A.B. should be used as a retention enema in some of these resistant cases of amoebic dysentery. This, of course, was an extravagant treatment, but some of the cases which had failed to respond to the routine treatment were seriously ill. In one case 80% of the mucous membrane of the rectum was actively ulcerated after no less than three courses of the emetine - E.B.I. - stovarsol routine.

Slides made from scrapings of this ulcerated surface were swarming in vegetative forms of E.histolytica (so many were on the slide that they literally had not room to move). After two ounces of 10% N.A.B. had been run in to the rectum daily for six days the sigmoidoscopic picture had completely changed. All pus and blood had disappeared. Most of the ulcerated area had already healed and the remainder was healing. No. E. histolytica could be found. Unfortunately there was only sufficient N.A.B. to treat a very few cases along these lines but the treatment was equally successful in all.

By the middle of 1943 only 40 grains of emetine remained in the camp. All the E.B.I. was already expended and 500 four-grain tablets of stovarsol remained. This situation was one which caused grave concern. There were at that time between forty and fifty cases of intestinal amoebiasis in hospital, and who could say how long it might be before further supplies of these drugs arrived? (Actually it was two years). It was therefore decided at this point that emetine must be reserved for the dangerously ill, and even for such cases the quantity used would have to be limited to the absolute minimum required for saving life. At first eight injections, each of half a grain was regarded as the minimum, but ultimately it was found that a total of $2\frac{1}{2}$ grains was sufficient to arrest a process which was apparently endangering life. In some cases it was

found that stovarsol was more efficacious in arresting a dangerous exacerbation than emetine. These were mainly cases who had had several courses of emetine when that drug was more plentiful. During the second half of 1943 it was also decided to limit the use of stovarsol to severe cases and as time went by without fresh supplies arriving, one's standards of the degree of severity which justified its use became progressively stricter. Finally the position was reached when this drug also had to be reserved for the dangerously ill.

Only small stocks of powdered ipecacuanha were available in the camp. This, combined with tannic acid, was used in the treatment of a dozen or 20 cases in 1943. This drug was found to be quite effective in cases of moderate severity but the number of cases was too small to draw conclusions. The dosage employed was that recommended by Rogers - 20 grains of ipecacuanha three times a day combined with 10 grains of tannic acid. Most patients were able to retain this mixture without a previous sedative, but they all experienced nausea and rather dreaded the treatment.

The acute shortage of specific drugs which has been described forced one to the decision during the latter part of 1943 that the future routine treatment of amoebiasis must take some form as yet untried. That form would have to be dictated largely by availability and a survey of the medical stores

showed that acriflavine, sodium arsenite and liquor arsenicalis were available in large enough quantities to make a trial worth while. It was realised that these drugs could at best only be palliatives, and that the most one could hope to do was to keep the growing numbers of "amoebics" alive for a length of time which it was always hoped would only be another three months. As it was, 151 British * and considerably smaller numbers of Australian and Dutch cases survived the following two years without specific treatment.

The most successful of these improvised treatments was acriflavine as a retention enema. At the end of 1943 it was associated with a short course of stovarsol in the more severe cases, but later it was usually used alone. To begin with acriflavine was tried by mouth but the results did not compare with those obtained by enemata.

The best results were obtained with acriflavine enemata when the desired strength of the dye was made up in a solution of normal saline. Patients were able to retain stronger solutions of acriflavine in saline much longer than when it was merely dissolved in water. The technique employed was as follows:- The solution was sufficiently warm to be blood heat when it arrived in the bowel. It was run into the rectum very slowly with the patient on his left side. Only 8 oz. were

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* This is the actual number of names on a list entitled "Personnel suffering from Amoebic Dysentery who have not received Specific Treatment" which the author submitted to the British Military authorities in October 1945.

employed as a maximum (much less at first in those who experienced difficulty in retaining the enema). Immediately after receiving the enema the patient adopted the genu-pectoral position. Many patients preferred to receive the enema in this position. After fifteen minutes the patient lay on his right side for a further fifteen minutes and after that lay on his back with the foot of the bed raised. After about an hour in all he usually found he could sit up without losing the enema and many could walk about. For the first two days of the course the strength of the solution was one part of acriflavine in one thousand parts of normal saline. After that a strength of 1:750 was given for two days, and finally a solution of 1:500 was given for six days. By this system of gradual increase in the strength of the solution and, if necessary, a gradual increase in quantity, but never exceeding eight ounces, most patients were able to retain the enema for several hours, and many until it was completely absorbed.

The result of this treatment in some cases was an apparent cure. In many cases it converted patients suffering from active amoebic dysentery into comparatively healthy cyst-passers. Generally speaking, a course of acriflavine retention enemata did not cure amoebic dysentery but it enabled acute cases to master the infection temporarily, and less acute cases to become quiescent.

The following sigmoidoscopy reports, typical of many, are from the same case before and after a course of acriflavine retention enemata.

> Sigmoidoscopy 20:6:45:- "Acute amoebic ulceration in lower rectum where there are large superficial ulcers. Ulcers of medium depth on the inferior valve of Houston. Mucous membrane of upper rectum and lower sigmoid normal. Microscopy:- Numerous vegetative E.histolytica."
>
> Sigmoidoscopy 4:7:45:- (after course of acriflavine retention enemata). "Now no sign of acute amoebic ulceration. In the lower rectum there is a group of 3 ulcers. These are punched-out, fairly superficial and are surrounded by a red areola. They vary from 3 m. to 1 cm. in diameter. Microscopy:- No E.H. found."
> (See Plates 5A and 5B).

This was a case of severe recurrent amoebic dysentery. The former sigmoidoscopy report was during a very acute exacerbation. In such cases the patient was given sedatives to help him to retain his enemata during the first few days.

There was nothing dramatic about the results of treatment by acriflavine and normal saline enemata, but by using this method occasionally supplemented by a short course of stovarsol by mouth, one was able to keep many severe cases of amoebic dysentery in check for long periods.

Because it was available in comparatively large quantities an attempt was made to utilise inorganic arsenic in the treatment of amoebiasis. The results were far from being disappointing because it was not expected to be of much value. However it certainly played a part in curtailing exacerbations in some

cases. Sodium arsenite was used as a retention enema in a concentration of 1 in 10,000 in cases of moderate severity. In some cases it appeared to have the effect of converting an active infection to the stage of cyst-passing. Liquor arsenicalis was given by mouth in graduated dosage commencing at 5 minims thrice daily and working up to three times that amount. It was not used until mid 1945 and at that time everyone was on the border line of clinical vitamin B1 deficiency. After one week's treatment with liquor arsenicalis all patients developed oedema of face and legs and its use was abandoned.

The rôle of the sulphonamides in the treatment of intestinal amoebiasis was entirely as an auxillary. Early in the sulphonamide treatment of chronic bacillary dysentery (i.e., during the first half of 1943) it was noticed that certain cases which were treated with sulphapyridine improved and subsequently relapsed. These were usually later found to be amoebic cases. Similarly when an acute amoebic case was treated with sulphapyridine in error there was not just a lack of response but a marked improvement followed by a relapse. At that time it appeared to the author that there was in most cases of amoebic dysentery an element of secondary infection which was susceptible to sulphapyridine whilst the primary amoebic infection was untouched. As a result of this observation sulphapyridine was

utilised in the first place to deal with the secondary infection in cases in which this remained after specific treatment (these cases usually had considerable amounts of pus visible sigmoidoscopically); and later to help economise with specific drugs by giving sulphapyridine early and thus dealing with the secondary infection concurrently with the amoebic infection. However as the necessity for economising with sulphapyridine was itself becoming pressing this policy could not be pursued. But much later when the stocks of emetine and stovarsol were almost exhausted, sulphapyridine and sulphaguanidine both played valuable parts in arresting some serious exacerbations in amoebic cases. Their use became a definite step in the treatment of such relapses. When sedatives and acriflavine retention enemata had failed either sulphapyridine or sulphaguanidine was tried before making inroads into the small remaining stocks of emetine or stovarsol. The use of one of the sulphonamides in this way often made it unnecessary to use specific drugs at all or made it possible to use a smaller quantity. In support of this policy it must be pointed out that one could never use sufficient of the specific drug in any one case to effect a cure, so that all one was doing in most cases during those last two years was to damp down the effects of a flare up. One was therefore justified in using any means to save specific drugs even although one was only tiding the patient over a bad patch, knowing full well that

he would probably relapse again. After all, during the whole of that two years one had only sufficient emetine to give one full course to each of four cases.

Taken all in all, the various improvised methods (and the use of sulphonamides must be included in that term as there was no existing evidence of their value in amoebiasis by the end of 1941) of treating amoebiasis were of great value. This is clearly demonstrated by the fact that of the 151 British cases of amoebic dysentery who had been diagnosed during the last two years of captivity, only 25 were sufficiently ill to be in hospital in September 1945. In fairness, however, it must be admitted that the disease is one in which spontaneous remission of the symptoms is not uncommon. For example, a patient who was slowly dying of Polycythaemia Vera contracted amoebic dysentery at the end of 1943. He had a short fairly brisk attack, received no treatment as the use of specific drugs was not considered justified and acriflavine retention enemata caused needless distres and were discontinued. At post mortem three months later there was no sign of active amoebiasis, although there were scars which appeared to have been caused by small amoebic ulcers in the caecum and at the hepatic flexure.

Strangely enough, the "amoebic" patients who were most seriously ill at the end of the period of captivity were mainly those who had been diagnosed in 1942 or early 1943 and who had

several courses of the emetine - E.B.I. - stovarsol routine. These men were extremely emaciated; they were very prone to contract a wide variety of deficiency diseases and, in fact, without exception, they had oedematous beri-beri. Day in, day out, for years they had diarrhoea with at least four motions containing pus and blood, and every few weeks they flared up with an acute exacerbation with anything from ten to twenty stools in the day with much blood. These men suffered a great deal of pain every day for years, but, generally speaking, they remained good natured and cheerful, and most of them were constantly busy doing some useful work with their hands. It was inevitable, of course, that some of these men should be pre-occupied with their colons, but while they were in captivity they showed little sign of psycho-neurosis. Sigmoidoscopically most of these cases of long-continued activity, showed gross lesions. The following reports are typical of several cases which had had active intestinal amoebiasis for long periods.

i. (Duration 2 years): "Very extensive deep ulceration replacing most (80%) of the mucous membrane of the rectum and lower sigmoid. Micro: Pus cells +++. Sluggish vegetative E.histolytica."

ii. (Duration 3 years): "Very extensive amoebic ulceration throughout rectum and lower sigmoid. Ulcers contain pus, are of medium depth and bleed very readily. Ulcers are surrounded by a wide zone of inflamed mucosa. In some areas these zones are continuous with each other producing a generalised inflammation. About 50% of total area is ulcerated. Micro: Numerous active vegetative E.histolytica."

It was in the management of such cases as these that the greatest flexibility of therapeutics was required. A line of treatment which was of great help in one exacerbation might be useless in the next. One was constantly ringing the therapeutic changes. One relapse might be easily controlled by acriflavine enemata and two months later they might be useless in the same case and sulphonamides had to be used. After that the patient might have a few weeks or months respite and then suddenly without warning a dangerous exacerbation which called for a short course of stovarsol, or even a few grains of emetine might occur.

Emetine or E.B.I. was used exclusively for the treatment of hepatic amoebiasis in 1942 and 1943. Later, with emetine in such short supply, stovarsol was used in amoebic hepatitis with excellent results and without any toxic manifestations. The only case of hepatic abscess which was successfully treated by open operation occurred in May 1945. It was considered in this case that a larger expenditure of emetine than usual was justified, and one grain was injected for six consecutive days. This was followed by four grains of stovarsol twice daily for seven days. This patient made an uninterrupted recovery, and was perfectly fit when last seen four months later.

Cyst-passers were originally treated with E.B.I. Generally speaking this type of case was resistant to treatment. At a

later stage stovarsol was used with quite as good results as those obtained with E.B.I. During 1944 and 1945 symptomless cyst-passers were given no treatment at all but were discharged from hospital after careful instruction in hygiene, with orders to report to their medical officers immediately there was a recurrence of symptoms.

In all, out of hundreds treated with organic arsenicals, the author only saw two cases of idiosyncrasy to those drugs. In one there was irritation of the palms of the hands very soon after one tablet of stovarsol was taken and in a few hours a generalised morbiliform rash. This happened in this case every time an attempt was made to use an organic arsenical. In the other case the reaction was more delayed and took the form of a generalised erythema with some desquamation but the condition was not sufficiently severe to be termed an exfoliative dermatitis. Both cases recovered spontaneously on discontinuing the arsenical.

Of the cases of intestinal amoebiasis encountered in Singapore in 1942 and 1943 it may be said that the results of treatment by drugs which up to 1941 were considered specific for the disease were often disappointing. This state of affairs surprised clinicians in the Prisoner of War Camp who, at the beginning of the period of captivity, welcomed a diagnosis of amoebiasis as something which would be readily amenable to specific treatment, for that is the impression one obtains from

standard works on Tropical Medicine. As time went on and one encountered more and more cases which resisted emetine, E.B.I. and stovarsol one began to wonder if tropical physicians whose published works are based on their experience of tropical diseases in hospitals in Britain were not, perhaps, dealing with a different problem from those who have to treat these diseases in their endemic areas. Presumably, in normal times, these resistant cases would have been sent to a temperate climate. In the Prisoner of War Camp they remained with us.

Plate 1A

Acute Amoebic Ulceration showing very superficial irregular ulceration with many bleeding points. Dark blood and mucus is adherent to the surface. The two light patches are superimposed mucopus. The intervening mucosa is generally inflamed.

Plate 1B.

The same case four days later. Ulceration now has depth (1 mm.) and punched out edges can now be defined. The ulcer contains pus. There is still a patch of acute amoebic ulceration to the left. The ulcer is surrounded by a zone of inflamed mucosa but the mucous membrane to the right is returning to normal, although still somewhat congested.

Plate 2.

Chronic Amoebic Ulcer of the "teed-up" type. The edges of the ulcer are under-mined. The ulcer is surrounded by a red areola and contains pus. The intervening mucosa is normal.

Plate 3.

"Teed-up" ulcers are seen to the left. On the right is a patch of early amoebic ulceration with inspissated blood and mucus adherent. The mucosa surrounding this shows subacute inflammation.

CHRONIC AMOEBIASIS.

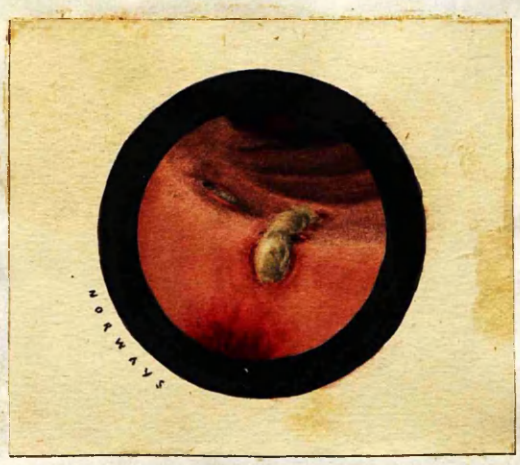


Plate 4.

A case of long standing at present having a recrudescence of the symptoms. At the lower margin of the picture is a patch of acute amoebic ulceration. In the middle and towards ten o'clock are chronic ulcers, the larger being 2 mm. in depth. The lower edge of the large ulcer is inflamed and there are bleeding points on the right-hand edge. The ulcers contain pus. The intervening mucosa in the upper part of the picture and in the region of the chronic ulcers is normal.

CHRONIC AMOEBIC DYSENTERY.

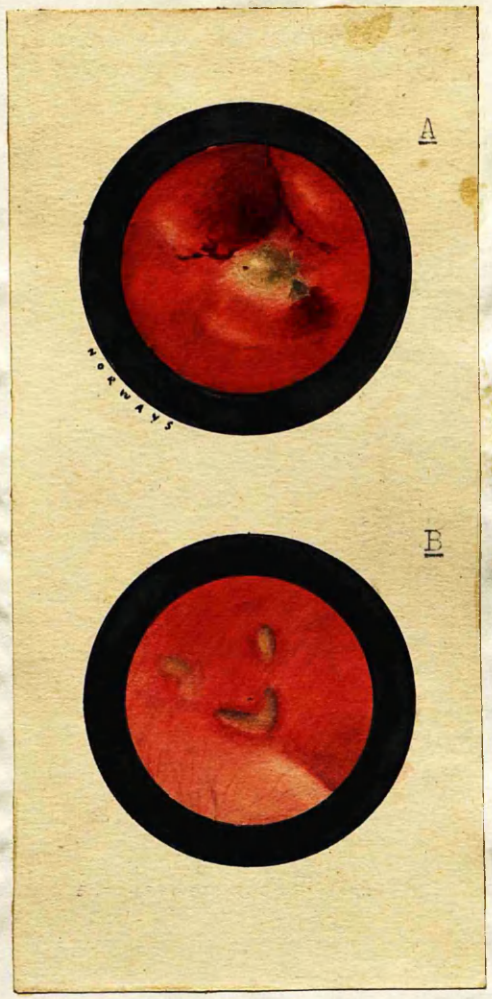


Plate 5.

A. Before treatment.

Recrudescence of acute amoebic ulceration in a chronic case of long standing. Mucous membrane is generally inflamed. There is a patch of irregular superficial ulceration with multiple haemorrhages. Dark blood with mucus is adherent to the area. In the centre is a "blob" of mucopus superimposed on the inflamed area.

B. After treatment.

The same area after treatment by 1:500 acriflavine retention enemata for 10 days. Ulcers have now localised, 3 clear-cut ulcers having formed. Depth of ulcers no more than 1 mm. Ulcers contain mucopus. Inflammation is also becoming localised. Mucous membrane to the top of picture still somewhat inflamed, but that to the left and below is returning to normal.

CHAPTER VI.

NON-SPECIFIC DIARRHOEA, FLAGELLATES AND PARASITES.

With the change over from European to Asiatic diet at the commencement of their imprisonment most people in the Prisoner of War Camp had an attack of diarrhoea which varied from a few loose stools during one night, to two or three weeks during which four or five loose stools containing undigested food were passed daily. This appears to have been due to an alteration in the flora and fauna of the bowel brought about by the revolutionary change in the diet. Precisely the same thing happened in September 1945 when a return to European diet with a higher proportion of protein and fats was made. Here, of course, the unaccustomed richness of the diet played a part, but it is probable that an altered intestinal population was in part responsible.

Diarrhoea per se was always common in the Prisoner of War Camp, but it was far commoner among those who returned to Singapore after working on the Burma-Siam Railway. These people had been living in jungle country with a largely polluted water supply and on very poor rations for many months and then, immediately before they returned to Singapore, a great improvement in their rations, especially in the quantity of protein. When

they returned to Singapore they returned to much poorer rations once more. Of course, many of them had some form of dysentery or malaria which accounted for the diarrhoea, but in at least fifty per cent of those suffering from diarrhoea no specific cause could be found. An infestation with Strongyloides stercoralis was exceedingly common in these cases.

Diarrhoea unconnected with dysentery became increasingly common in Singapore during 1944 and 1945. This may have been a form of deficiency disease (possibly a deficiency of part of the Vitamin B2 complex). In origin it appeared to be due to a disordered action of the small intestine and the effect on the colon appeared to be a secondary one. In time the mucous membrane of the lower colon became thickened and inflamed as a result of being constantly bathed with alkaline liquid faeces. In these cases also there was commonly an infestation with Strongyloides stercoralis or, less commonly, Ancylostoma duodenalis.

Various flagellates were commonly found in the stools of these diarrhoea cases, but whether the flagellates were secondary to the diarrhoea or vice versa was not clear. Certainly Giardia lamblia appeared to play a very definite part in causing or prolonging diarrhoea. Trichamonas hominis and Chilomastix mesnili were common also in diarrhoeac stools but these always appeared to be harmless.

Entamoeba coli was a common inhabitant of the stools of

those suffering from diarrhoea, and this organism was never seen in a normal stool. Equally well cystic forms of E.coli were uncommon in those who had never had a prolonged bowel upset. Iodamoeba Butchlii was rarely seen and appeared to be of no significance.

Tape worms were exceedingly rare in Singapore and the author only saw two cases, both infested with Taenia saginata.

From 1942 onwards, infestation with Ascaris lumbricoides was exceedingly common and it became progessively so as time went on, until in 1945 it was almost the exception to find a patient in whose stools Ascaris ova were not present. The presence of these very annoying parasites was often not suspected by the host and on occasion an apparently healthy man would be alarmed by a round worm emerging without warning from the mouth, nose or anus. The author only once encountered an ascaris worm while doing a sigmoidoscopy. This worm retreated before the sigmoidoscope but was finally caught with a pair of forceps and extracted.

Symptoms, Signs and Treatment.

Strongyloides Stercoralis:

General weakness and loss of weight associated with anaemia and an intractible diarrhoea were the usual signs of

infestations with this parasite in Singapore. Diarrhoea consisting of four to six watery stools daily was common and this often caused a profound degree of emaciation. In two fatal cases no other cause of death could be found. The changes found at post mortem in these cases were mainly those of malnutrition, but the wall of the duodenum and jejunum was oedematous and showed patchy hyperaemia. Mucus scraped off the mucous membrane of the jejunum showed numerous adult female strongyloides in addition to larvae and ova.

Strongyloides infestation was remarkably resistant to treatment. Gentian violet in enteric coated capsules was quite useless. Brilliant green in similar capsules was more effective but very little of this dye was available. Intravenous tartar emetic in dosage graduated from $\frac{1}{4}$ to $2\frac{1}{4}$ grains was the most useful form of treatment but it had unpleasant side effects. The drug was given two or three times a week until a total of 20 grains had been injected.

Ancylostoma Duodenalis:

This parasite was much less common than strongyloides. It caused little or no diarrhoea and little wasting. Anaemia and dyspepsia were the usual symptoms but many cases were symptomless.

The usual methods of treatment were used. Carbon tetarchloride was more effective than oil of chenopodium. Hexyl

resorcinol was most effective.

Ascaris Lumbricoides:

Infestation with this worm was often symptomless. The common symptoms were merely vague abdominal discomfort associated with flatulence. Some cases had attacks of colic followed by short bouts of diarrhoea. Generally speaking, however, diarrhoea was not commonly associated with ascariasis. Cough at night was a fairly common symptom. Loss of weight in excess of that which was general was not usually found in those infested with this parasite, although some patients who harboured many worms were grossly emaciated. It was usual for the infestation to be multiple, some six or seven worms being common, however, some cases in which no ova had ever been found in the stools, passed a single male worm. Cases also occurred in which the ova in the stools were unfertilised and on treatment being instituted a single female was passed.

There was one fatal case. This patient was grossly emaciated and became jaundiced and finally had a subacute intestinal obstruction. At post mortem 49 ascaris worms were found in the bowel, some forty of them, being entangled with each other, had formed a ball which was obstructing the small intestine. One worm had entered the common bile duct. It had then, apparently, doubled back on itself in an attempt to get out and had become firmly jammed and had died in situ.

The treatment which was most successful in the author's hands was santonin and calomel. Oil of chenopodin was disappointing in ascariasis.

Giardia Lamblia:

Many cases suffering from chronic diarrhoea were infested with this flagellate. These cases were not as a rule grossly emaciated but they were often pale and flabby and lacked energy. They commonly passed six or seven watery stools daily for months without remission. Excessive flatulence and anorexia were common symptoms.

In the author's hands the use of atebrin proved quite useless in some of these cases. The most effective remedy was stovarsol but it could seldom be spared for cases of giardiasis.

Treatment of non-dysenteric diarrhoea had often to be purely symptomatic. Sedatives were not generally employed although they sometimes played a part in an attempt to break the habit in cases of "habitual" diarrhoea. Bismuth was not available. In 1944-45 kaolin was available in large quantities in Kranji where it was "mined" within the camp. This local supply was separated from gritty particles by frequent washing and sedimentation and then sterilised by heat. It was most effective in large dosage (one teaspoonful of the powder every two hours) but one had to avoid causing constipation. Some cases relapsed as soon as the kaolin was discontinued but in

many others a lasting improvement resulted.

A course of retention enemata often helped cases of chronic diarrhoea in which no specific cause could be found. It did not seem to make much difference what chemical was employed as long as the patient was able to retain the enema for long periods. The object was really to discipline the patient's bowel but the virtue of the medicament in the enema was impressed on the patient. If the patient co-operated there was often a great improvement, and a man who had had never less than six stools a day for months might come down to three.

Mention has already been made of the effectiveness of work as a therapeutic agent in cases of non-specific diarrhoea. The type of work which was most effective was light work, such as hoeing or weeding, in the hospital garden. At Kranji there were 30 acres under cultivation with new crops coming on constantly all the year round, so there was always work to do. Some cases improved immediately they went out to work whilst others improved gradually over some weeks.

No cholera was seen in Singapore although parties working on the Burma-Siam Railway were decimated by this disease. In December 1943 when parties of seriously ill men were arriving in the Singapore Prisoner of War Hospital from Siam, a patient was admitted in a moribund state. (Many of these

unfortunate men were in a dying condition when taken off the trains and loaded into lorries. Some were found to be dead on arrival at Changi). This patient was extremely collapsed, dehydrated and cyanosed. No history could be obtained. The temperature was 95°F. and the pulse rate 140. He was having frequent stools which consisted of a white milky material. No cholera vibrios could be found in the stool. The patient died a few hours after admission. The significant post mortem findings were as follows:-

Extreme dehydration with a marked lack of body fluids.

Spleen: Gametocytes of malignant tertian malaria present.

Ileum: There was congestion of the peritoneal surface of the last 10 feet. This became more acute in the terminal 3 feet where the colour was a dull purplish-red. The content of the ileum was a yellow watery fluid containing flecks of mucus. The mucosa was acutely inflamed with petechial haemorrhages in several places, but there was no loss of surface. A few inches above the ileo-caecal valve was an area of patchy necrosis. The necrotic patches were about one inch in diameter with grey sloughs adherent.

The Peyer's patches were normal.

The rest of the alimentary canal was normal.

The mesenteric lymph glands were hyperaemic.

No cholera vibrios were found in the contents of the ileum. In this case it was possible to culture both this

material and material from the gall-bladder, but no colonies of cholera vibrios grew in the culture. In view of this one was forced to make the non-committal diagnosis of acute enteritis. No similar cases occurred which shows that the condition could not have been very infectious as the patient had been in an overcrowded railway freight wagon for seven days before he was admitted to hospital.

CHAPTER VII.

SIGMOIDOSCOPIES.

In May 1942 the author set up a Sigmoidoscopic Clinic primarily for cases from the Chronic Dysentery Ward, but within a short time cases were being sent for an opinion not only from the whole Dysentery Wing (which at that time consisted of ten large wards) but also from the entire hospital. Out patients were also referred to the clinic from all parts of the Prisoner of War Camp. Thus, starting from small beginnings the scope of the clinic grew until the author found himself doing fifteen to twenty sigmoidoscopies in a morning session. There were at least three sessions a week during the first year. 2,000 cases were examined between May 1942 and February 1943. During the second year the number of cases dropped off and during the last year and a half of captivity only one session was held weekly, with ten to fifteen cases in a session. In all, between five and six thousand sigmoidoscopies were performed by the author in three and a half years.

In all the sites occupied by the Prisoner of War Hospital the Sigmoidoscopy clinic was located in an annexe to the Chronic Dysentery Ward. It was felt that it was important that patients should not be taken to the operating theatre, and that they should not be encouraged to regard what was really a

simple diagnostic procedure as an operation. Many chronic cases in which a diagnosis could not be established readily were sigmoidoscoped several times; and amoebic and chronic bacillary cases often had repeated sigmoidoscopies during their treatment. In these circumstances it was essential that all the patients in the dysentery wards should view sigmoidoscopy lightly. Patients may have felt apprehensive before their first examination but those who came back several times usually regarded the sigmoidoscopy as a trivial occurrence.

Once the routine was established and the orderlies trained, it was a fairly simple matter to deal with fifteen to twenty cases in a session of four hours. As sterilisation had to be done on a wood-fire this often took longer than the examination itself. One great labour-saving device was to have a stenographer at hand for taking notes. The author always had a pathologist or trained microscopist at work beside him, so that not only were scrapings examined when they were fresh, but also the author could look at a slide of interest or take part in the search for amoebae if necessary. It was also a great advantage in many cases to get an immediate verbal report while the sigmoidoscope was still in situ, so that further specimens could be obtained in a suspicious case if the first proved negative.

No preparation of the patient was required in the vast majority of cases. In the author's opinion it is a mistake to give either purgatives or bowel washouts before a sigmoidoscopy as both of these cause some alteration in the appearance of the mucous membrane and also make the bowel more irritable. Bowel washouts on the one hand remove some of the pathlogical appearances it is necessary to see, such as mucus, pus and blood, or the contents of ulcers, and on the other hand they make the normal look abnormal. A normal mucous membrane looked more red and slightly oedematous, and secreted excessive mucus, after a washout of water or even normal saline, the least irritating of all solutions. Also, if a non-irritating washout was given there was often as much difficulty in eliminating that as there was originally in voiding the faeces, so that one was apt to encounter veritable floods of washout solution when doing the sigmoidoscopy. Apart from all this it was an advantage to see to what extent the bowel was capable of emptying itself unaided and what was the character of the material which remained.

All that was required in most cases was that the patient should defaecate before coming for sigmoidoscopy. After this most patients were clear but if anything was still adherent to the bowel wall, sponging with swabs of cotton wool, either dry or dipped in normal saline was usually sufficient to give a

good view of the mucosa. (The author always worked with a pair of bronchoscopy forceps which could be used for holding swabs, for taking specimens either by scraping or by biopsy, or for snapping off polypi). Cases who were unable to defaecate before the examination, and other cases who might require a washout, were always seen before they were sent to have this, so that at least a glimpse was obtained of the mucous membrane in its natural state. In this way one was able to discount the alteration due to the washout. Besides, those who had been unable to defaecate before were often able to do so after the instrument had been passed.

Sigmoid.

Most cases were examined in the genu-pectoral position and only those who were not strong enough to adopt this posture were placed in the left lateral position. Very weak patients were, of course, examined in their own beds. With the patient in the genu-pectoral position it was seldom necessary to inflate the bowel with air, and it was usually possible to pass the sigmoidoscope into the lower sigmoid colon (i.e., a distance of 25 to 30 cm. from the anus) without using the bellows at all. With patients in the lateral position, however, it was always necessary to inflate. In the author's opinion it is very important to perform the examination whenever possible

without inflation and any expenditure of time and patience was considered worth while to avoid it. The two factors which contribute most to the patient's discomfort during sigmoidoscopy are inflation with air and the effects of a bowel washout. In those who had had a washout the bowel was irritable, and tight contractions in front of the sigmoidoscope were common. Thus one had to use the bellows more often in the minority of cases who had had a washout than the majority who had had no preparation. Inflation was sometimes used as a method of helping to get the instrument through a stenosed part of the bowel. For this purpose also the author had a set of graduated bougies made in the camp. These were three inches longer than the sigmoidoscope and by their use it was possible to dilate strictures at any point in the rectum or lower sigmoid through the sigmoidoscope.

The normal mucous membrane as seen with the electric sigmoidoscope is of light pink colour, smooth and glistening. In the lower rectum it is granular and submucous vessels cannot be seen, but in the rest of the rectum and in the lower sigmoid colon submucous blood vessels can be easily seen. The mucosa is of a lighter colour in the sigmoid colon than in the rectum. After an enema or after straining at stool the mucosa is red and may be thickened so that the submucous vessels are either invisible or not so easily seen.

Sigmoidoscopy was seldom performed on cases of acute bacillary dysentery because as a rule no purpose could be served by the examination. Sigmoidoscopy in acute bacillary dysentery was an extremely painful procedure and there was in these cases a considerable risk of starting a dangerous haemorrhage. In the few acute bacillary cases on which sigmoidoscopy was performed the mucosa was grossly swollen and of a deep dusky crimson colour with a greyish film over the entire surface. Blood and mucus were present in variable quantities. At a later stage patches of ulceration covered by grey sloughs might be seen, and later still, after the sloughs had separated these appeared as clear areas of irregular superficial ulceration, often with fine granulations on the floor. In a healing case the sites of the ulcers were often deep red and irregular whilst the remainder of the mucous membrane was returning to a normal colour but had a granularity of the surface. In cases in which the infection had been aborted with sulphonamides the mucosa did not return to normal as rapidly as the symptoms subsided. In these cases the oedema of the mucous membrane subsided rapidly and the greyish film covering the surface and the blood and mucus disappeared, but the mucous membrane remained of a deep red colour with a roughened, dry surface for some days.

The sigmoidoscopic appearances of chronic bacillary dysentery have been referred to in the section dealing with that disease. The principal types, arranged in order of frequency were as follows:-

1. Large irregular patches of superficial ulceration with adherent and generalised inflammation of the intervening mucosa. In this type of case large masses of granulation tissue from which pus could often be expressed were commonly seen.
2. Clear cut round, or oval ulcers containing white pus. The intervening mucosa was often normal in colour in these cases. It was frequently very difficult to decide from the sigmoidoscopic appearances whether one was dealing with a bacillary or an amoebic infection. In bacillary cases the mucosa was usually slightly thickened so that the sub-mucous vessels could not be seen. Serpentine ulcers were sometimes seen in these cases.
3. Dusky red, oedematous mucosa with no visible ulceration and much mucus present.

Occasionally one encountered cases in which there was a "latticed" appearance of the mucous membrane. In these there was a series of large depressed areas between which was a system of intersecting ridges which were sometimes of a deep red colour. In some cases these ridges resembled the roots of a

tree from which the soil had been eroded. As these were usually inactive and what was seen was merely scarring due to previous extensive ulceration it was impossible to prove its origin. However, there seemed no doubt that this picture was the result of severe acute bacillary dysentery. There was invariably a history of a serious attack of acute dysentery.

Table 12 (page 157.) and Table 13 (page 160.) indicate the variety of sigmoidoscopic appearances which were found in cases of amoebiasis. The typical pictures arranged in order of frequency were as follows:-

1. Round or oval punched-out ulcers, containing pus and surrounded by a red areola. These ulcers were sometimes, but not usually, raised above the surface of the surrounding mucosa. The intervening mucous membrane was, as a rule, quite normal.
2. Deep ulcers - usually in the form of two or three deep, round ulcers in the lower rectum, or solitary ulcers on the valves of Houston. When active, these ulcers contained a mixture of blood, mucus and pus and had undermined edges along which might be seen tiny bleeding points. The base or the edges of an active ulcer would bleed copiously with the slightest trauma. When inactive deep ulcers were often surrounded by polypoid scarring. A puckered scar with irregular concentric

ridges might be seen long after a deep ulcer healed. The intervening mucosa was usually normal.

3. Acute amoebic ulceration. This is described in detail in the footnote (**) to Table 12 on page 157. This type of ulceration was relatively more common than deep ulcers in active cases passing vegetative E. histolytica. Also, it was seen more commonly in 1944 and 1945 than in the earlier years.

4. Pinhead ulcers. These were minute ulcers containing flecks of whitish or yellow pus (the pinhead). They were always multiple and often numerous. If, of course, the patient had had an enema before being examined this appearance was not seen at all. In these circumstances one merely saw a series of minute depressions or craters. The intervening mucosa was sometimes normal, but in other cases the pinhead ulcers occurred in patches of inflamed mucous membrane. In yet others each little ulcer had a tiny red areola. Punctate submucous haemorrhages were not uncommon in the normal intervening mucosa.

5. Patchy inflammation of the mucous membrane without visible ulceration.

6. Submucous haemorrhages without visible ulceration. The submucous haemorrhage varied in size from a minute speck to 2 cm. The intervening mucosa was usually quite normal.

7. Teed-up ulcers (see footnote*** to Table 12 on page158.)

8. Extensive deep ulceration with thick pus adherent. This occasionally involved from 50% to 80% of the mucosa. Sometimes this type was seen as a ring encircling the bowel for a distance of one or two inches.

In cases of chronic, non-specific diarrhoea and of diarrhoea of long standing due to flagellates or strongyloides the rectum and sigmoid colon was usually found to be the seat of a generalised mild catarrhal inflammation with an increased redness and slight thickening of the mucous membrane.

The coloured illustrations in this section and in the section on Amoebiasis illustrate some of the points mentioned above. The author was fortunate in having an accomplished professional artist as a patient at Kranji, and when convalescent this artist attended the sigmoidoscopy clinic with the purpose of preparing these pictures from cases demonstrated to him. The pictures in the section on Amoebiasis were completed in the camp, whilst those at the end of this section were completed in this country from sketches and notes made in Singapore. Of the plates in this section, the first four (Plates 6, 7, 8, & 9) are from cases of chronic bacillary dysentery, and the remainder are from cases of amoebic dysentery.

Plate 10, which shows a stricture of the bowel situated in the lower sigmoid colon, is from a case with a history of

both amoebic and bacillary dysentery. The X-ray plate (Plate 11) is from the same case. This plate, taken in December 1942, was the last to be taken in the Prisoner of War Hospital. The film had already deteriorated in the hot, damp climate before being exposed, and keeping the plate in the same climate for a further three years caused further damage. However, it does still clearly show the extent of the bowel which can be examined with the sigmoidoscope. The author frequently passed the instrument up to this stricture for the purpose of dilatation and finally passed the sigmoidoscope through the dilated stricture into the bowel above. Most cases with this type of polypoid stricture seen by the author had a history of both amoebic and bacillary dysentery. That being so, it is not possible to state categorically which type of ulceration was responsible. A few cases had either had an amoebic dysentery alone, or had a history highly suggestive of amoebic dysentery. Two cases with a stricture like a diaphragm, with an irregular aperture, obstructing the rectum (one in the lower rectum and one in the mid rectum) had a history of bacillary dysentery, but none of an amoebic infection. In addition to these a number of chronic bacillary cases were seen in which a mass of rigid granulation tissue was encircling the bowel and causing a condition of stenosis. Several of these were observed over long periods and in all the granulations finally healed without any tendency to stricture

formation. On the other hand a number of amoebic cases were seen in which deep anular or spiral ulcers were encircling the bowel. This type of ulcer had a tendency to polypoid scarring and stenosis.

Four cases of amoeboma of the rectum were seen by the author. In these a fungating, tumour-like mass was protruding into the lumen of the rectum. In three of these the tumour disappeared on specific anti-amoebic treatment. The fourth improved but the rectum remained partially obstructed. The area was resected and an end-to-end anastomosis was successfully performed by Colonel Julian Taylor.

CHRONIC BACILLARY DYSENTERY.

Plate 6.

Masses of granulation tissue with beads of pus appearing on pressure.

Plate 7.

Irregular superficial ulceration with pus adherent.

Plates 6 and 7 are from the same case. The sigmoidoscopy report was as follows:- In the mid and upper rectum are masses of red granulation tissue from which beads of pus can be expressed. There is also irregular superficial ulceration with pus adherent in lower rectum and lower sigmoid. Intervening mucosa red and oedematous.

Micro:- A few R.B.C's. W.B.C's ++. Epithelial cells +. Macrophages +.

Plates 8 and 9 are from the same case. The sigmoidoscopy report read:- Several large irregular ulcers containing pus scattered throughout rectum and lower sigmoid. Some ulcers are superficial, others of medium depth. Extensive granulation tissue also present - mainly surrounding ulcers. Granulation tissue red. Intervening mucosa light in colour but thickened. Micro:- R.B.C's +. Pus cells +++. Epith. cells + .

Plate 8.

A large irregular ulcer of medium depth and containing pus. Granulation tissue in the lower part of the picture.

Plate 9.

A large patch of superficial ulceration with adherent pus is seen on a bend in the bowel. Granulations in the foreground.

DYSENTERIC STENOSIS.

Plate 10.

Polypoid stricture in the lower sigmoid colon.

Sigmoidoscopy report:- At 10" there is a ring of polypoid mucous membrane with stricture formation. This stricture could be inflated slightly but not sufficiently to pass the instrument. As far as could be seen through the stricture the bowel was empty above this point, and there appeared to be small red polypoid patches with normal mucous membrane intervening. Micro:- Mucus +. Pus cells +. R.B.C.'s +. Macrophages +.

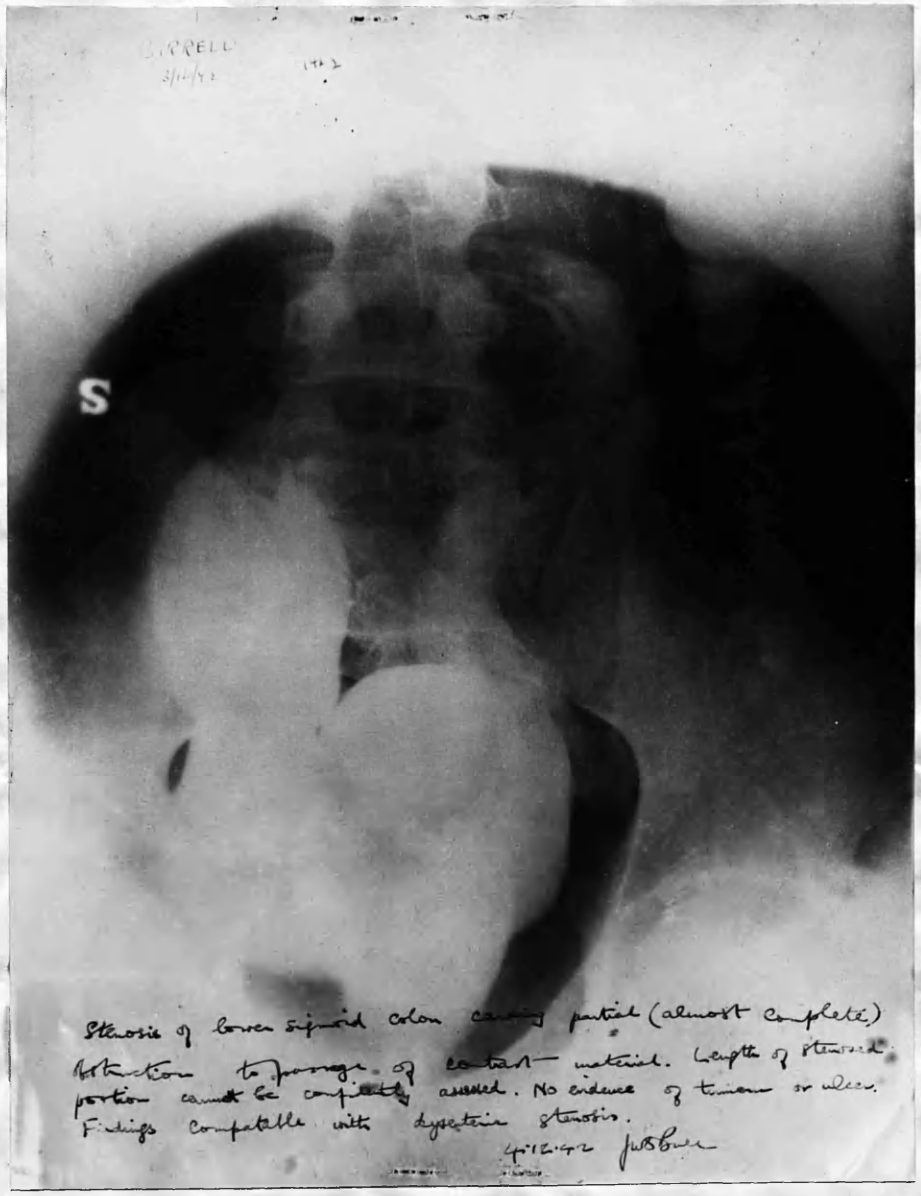


Plate 11.

X-ray of barium enema in same case as Plate 10. X-ray report reads: "Stenosis of lower sigmoid colon causing partial (almost complete) obstruction to passage of contrast material. Length of stenosed portion cannot be confidently assessed. No evidence of tumour or ulcer. Findings compatible with dysenteric stenosis."

Explanatory note to plates 10 and 11.

The patient referred to on pages 214 and 215 had a history of three attacks of amoebic dysentery and two attacks of bacillary dysentery. The last attack (bacillary) was six weeks before the sigmoidoscopy. One week before the sigmoidoscopy (report on page 214) this patient had a partial intestinal obstruction which was finally relieved with enemata.

AMOEBIC DYSENTERY.

Plate 12.

"Millet-seed" bodies, or yellow sub-endothelial nodules surrounded by a red areola. These nodules are probably on the point of rupturing. The intervening mucosa is normal.

Plate 13.

An active, punched-out ulcer, containing pus and surrounded by a red areola. This was discovered in a symptomless case.
This case was a member of the staff whose stool was found to be acid.

Plate 14.

A bridge of mucous membrane which has resulted from the healing of amoebic ulcers connected by tunnelled mucous membrane. This bridge was excised and healed perfectly.

CHRONIC AMOEBIC ULCERATION.

Plate 15.

Large deep amoebic ulcer in the lower rectum.

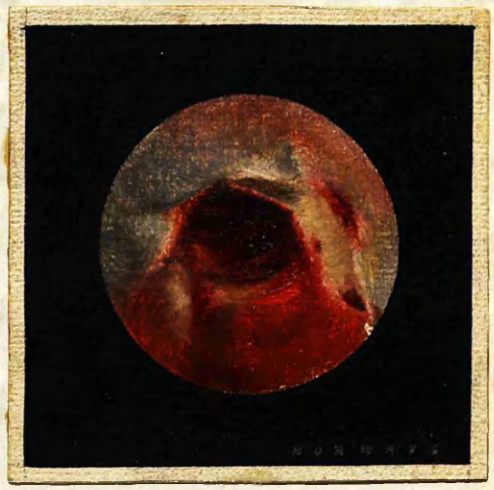


Plate 16.

Stenosis due to deep amoebic ulceration at recto-sigmoid junction.

Plates 16 and 17 are from the same case. The sigmoidoscopy report was as follows:-

In the lower rectum is a very large deep amoebic ulcer encircling the bowel for a distance of 2.5 cm. This tapers off into more superficial ulcers which are scattered throughout the rectum. 50% of the rectum is ulcerated. At the recto-sigmoid junction there is a large deep ulcer encircling the bowel, similar to that in the lower rectum. The lumen of the bowel was considerably reduced at this point and the sigmoidoscope could not pass this ulcer.
Microscopy:- Veg. E.histolytica ++. R.B.C's +++.

CHAPTER VIII.

VITAMIN B DEFICIENCIES IN DYSENTERY CASES.

Conditions due to a deficiency of some part of the Vitamin B complex were exceedingly common among dysentery cases. During the three and a half years of captivity the predominant type of deficiency disease varied from time to time. Even the form in which one particular deficiency presented itself changed. The first cases of avitaminosis occurred less than two months after the fall of Singapore.

Beri-beri.

This was the earliest deficiency disease to occur. The first cases occurred early in April 1942. The very first cases were men who had been heavy drinkers before becoming Prisoners of War. The early cases were of the neuritic type, but cardiac and oedematous beri-beri were both common shortly after. A few cases of encephalopathic beri-beri occurred in April and May 1942. At the end of 1942 and in 1943 oedematous and cardiac beri-beri were common. At the end of 1943 the cardiac variety was common among those who had returned from the Burma-Siam Railway. Oedematous beri-beri was extremely common in 1945.

Treatment: Thiamin either by injection or in tablets was extremely scarce until the middle of 1945. Rice polishings

were purchased by the hospital whenever available. These were not suitable in large amounts for dysentery cases, so the author had a watery extract made in a strength of 1-lb. to the pint. A small amount of acid was added to aid the extraction of thiamin. It was estimated by biological assay that this extract contained 2000 international units of Vitamin B1 and 200 mgm. of nicotinic acid in each pint.

The first case to be treated with rice polishings extract was a case of chronic bacillary dysentery suffering from oedematous beri-beri with gross anasarca, ascites and hydrothorax. This patient was given two pints of rice polishings extract daily. The urine output steadily rose to the fifth day when 300 ounces of urine was passed. The patient then steadily recovered.

There were very many cases of beri-beri in 1945, but no rice polishings were permitted to enter the camp. In the middle of 1945 thiamin in tablet form and in ampoules for injection arrived. Most cases re-acted to these but some grossly oedematous cases in the dysentery ward failed to respond. They were given as much protein as possible including milk, and also plasma transfusions, but this did not reduce the oedema. Finally some rice polishings arrived and the author had the watery extract already described made once more. The results from giving two pints of this a day were as

dramatic as those originally obtained. As these patients had failed to respond to large doses of chemically pure thiamin it appeared to the author that Vitamin B1 in its natural form contains some essential substance which is lacking in the synthesised product.

In 1945 when the author was attempting to treat amoebic dysentery with inorganic arsenic all cases who were given a course of liquor arsenicalis developed oedema, first of the face and later of the legs. At that time beri-beri was very common and oedema was invariably the first sign of it. It appeared to the author that arsenic prevented absorption of the small quantity of thiamin which was present in the diet.

The following incident is an indication of how close everyone was to clinical beri-beri in August 1945. When the relieving forces were expected, the Japanese increased the patients' rice ration from 200 grammes per head to 800 grammes per head daily. It was realised by British Medical Officers that such an increase would inevitably cause beri-beri, so only 350 grammes per head was issued in the patients' diet. This increase caused oedema in 50% of the dysentery patients who had not so far shown signs of beri-beri.

Ariboflavinosis.

Clinical manifestations of deficiency of riboflavine made their first appearance among dysentery patients in

July 1942 in the form of angular stomatitis, atrophic glossitis and scrotal dermatitis. Cases of this type were always present in the Prisoner of War Camp from then onwards. Secondary infections of these lesions were extremely common. The commonest and most dangerous complication was infection with the Klebs-Loeffler bacillus.

In October 1942 affections of the eyes due to aribo-flavinosis were first seen. The earlier form was "granular cornea" and slightly later retrobulbar neuritis became common. Many cases of both these types occurred in the dysentery wards during the first half of 1943 and sporadic cases went on occurring until the end of captivity. A large number of men had their eyesight permanently impaired by these conditions and blindness resulted in not a few.

Treatment: Marmite was the treatment of choice. This was in limited supply and a local product was prepared to conserve it. This was "green leaf extract" which was made from various inedible leaves and grasses which grew in the camp. The most effective was that prepared from wild passion fruit leaves (Passiflora foetida). Other sources of riboflavine which were available on occasion were groundnuts, towgay, soya beans and black beans (see paragraph on diet in section dealing with Chronic Bacillary Dysentery).

Deficiency of Nicotinic Acid.

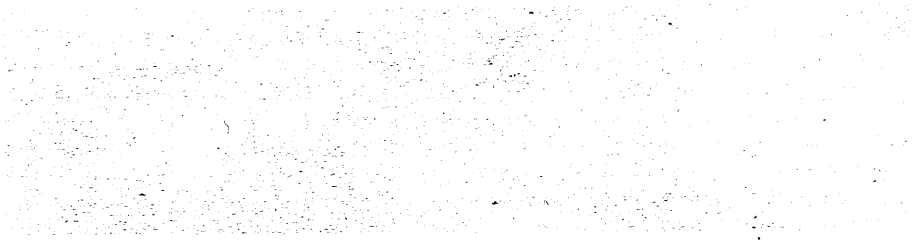
Cases of typical skin pellaga were fairly common in Singapore from late 1942 onwards, but this disease was never more common among dysentery cases than it was in the remainder of the community. Many cases of diarrhoea which appeared to be due to a deficiency of some part of the Vitamin B2 complex occurred, but in these there were usually no skin changes. Mental symptoms of pellagra were uncommon in Singapore.

Nervous manifestations of deficiency of nicotinic acid were very common late in 1942 and during 1943. The commonest form among dysentery cases was "burning" or "painful feet". This was a very trying condition with a burning pain deep inside the feet, worse at night or when the feet became warm. The feet, and sometimes the legs, became spastic and some cases showed a positive Babinski sign. A more serious condition with a permanent crippling due to an upper motor neuron lesion was Spastic Diplegia. Of this type about 30 cases occurred in the Singapore Prisoner of War Camp, but none among dysentery cases although some of these cases later developed dysentery.

Treatment: Nicotinic acid itself was only available in minute quantities. There were larger stocks of Coramine and Nikethoamide and these preparations were used for the worst cases. Marmite was of great value and was used for severe cases

whenever possible. Rice polishings and green leaf extracts were routine methods of treatment.

As time went on a condition of avitaminosis was the state of everyone in the Prisoner of War Camp. Some people, whenever they were overworked or below par physically, would invariably develop signs of beri-beri and others under the same conditions would show signs of ariboflavinosis. Although those with one variety might later develop the other, it did seem as if certain people had a low sympton threshold for one deficiency and others for another; so that the first sign of deficiency disease was the same in one particular person time after time. Also, as time went on, the dosage of vitamins required to stop symptoms became progressively greater.



CHAPTER IX.

A BRIEF REVIEW OF SOME RECENT RELEVANT LITERATURE.

Bacillary Dysentery.

The use of succinylsulphathiazole in bacillary dysentery was described in America soon after those in Singapore were cut off from medical literature, when Poth, Chenoweth and Knotts (1942) reported its successful use in 20 cases of this disease. The dosage employed was 0.25 to 1.0 gramme per kilogram of body weight. They found the drug equally effective in both acute and chronic cases. No toxic effects were reported.

Lyon (1943) gave a general account of the use of sulphaguanidine in bacillary dysentery. The drug, he thought, had completely revolutionised the treatment of acute bacillary dysentery. A footnote was added on 14 cases treated with succinylsulphathiazole.

Scadding (1944) published a comparison of different sulphonamides in the treatment of mild bacillary dysentery in the Middle East theatre of war. He referred to the varying and opposed opinions of various authors and other clinicians of the effect of different sulphonamides on bacillary dysentery. He quoted Pauley (1942) as suggesting that sulphapyridine has definite advantages over sulphaguanidine, and Bulmer and Priest (1943) as referring to sulphaguanidine as "a specific drug in the treatment of acute, subacute and chronic bacillary dysentery".

Scadding treated 358 mild cases with sulphaguanidine, sulphapyridine or sulphanilamide and found these three drugs equally effective. The only advantage claimed for sulphaguanidine was that it seldom caused unpleasant side effects. Of severe cases he said "in not an inconsiderable number of severe cases the response to sulphaguanidine has proved disappointing, frequent small stools of blood and mucus, pain and fever persisting after long courses of 150 grammes or more." In severe Shiga infections he thought that there was no doubt of the value of concentrated antitoxic serum. In several severe cases, after no dramatic improvement resulted from long courses of sulphaguanidine he gave sulphanilamide or sulphathiazole with good results. He regarded the danger of renal disorders as a grave defect of sulphapyridine of which he gave a total of 19 grammes per case.

Amoebiasis.

In the Army Medical Department Bulletin No.21 (March 1943) the treatment of Amoebic Dysentery recommended was a short course of emetine injections followed by E.B.I. and then stovarsol. Concurrently with the course of E.B.I. a course of chiniofon(quinoxyl) retention enemata was advised. The results of this routine were said to be very good and it was suggested that relapses only occurred occasionally.

Leishman and Kelsall (1944) found that one in four of cases of diarrhoea among soldiers admitted to a General Hospital in India was due to amoebiasis. In their series of 333 cases of amoebiasis the relapse rate was 45%. Their treatment consisted of a course of emetine (6 to 12 grains by injection) followed by an organic arsenical by mouth for ten days. 80 - 90% of primary attacks responded to treatment in 48 hours, but in subsequent attacks there was a progressively delayed response until finally no response to treatment occurred. As they had no E.B.I. they used kurchi-bismuth-iodide but found it quite ineffective. Quinoxyl retention enemata sometimes achieved results in less chronic cases but they did not find them to be curative in cases of long standing. When limited supplies of E.B.I. arrived they treated 13 continuously relapsing cases with E.B.I. by mouth concurrently with quinoxyl retention enemata. Six of these cases relapsed within 21 days.

Manson-Bahr (1944) once more re-iterated his confidence in the efficacy of E.B.I. by mouth associated with quinoxyl retention enemata in the vast majority of cases of amoebiasis. He emphasised the danger of repeated courses of emetine hypodermically which tends to produce an emetine-fast strain of E.histolytica.

Adams (1945) believed until 1943 that the elimination of gut-infection with E.histolytica was a matter of simple

routine. His method was a few injections of emetine followed by a "blunderbuss" assault consisting of auremetine, stovarsol and bismuth subnitrite by mouth and retention enemata of chiniofon. He only recollected one case seen in Britain which did not respond. However, in 1943 30 cases arrived from the Indian and Burma theatres of war. These cases proved largely refractory to the routine treatment and some actively relapsed within a few days of its completion. Repetition of the treatment proved successful in some cases but a residue were still infected. Sulphaguanidine and sulphasuxidine were tried both by mouth and rectally. Temporary amelioration was obtained but there was no evidence of specific action on the causative organism.

Hargreaves (1945) referred to a new approach to the treatment of chronic amoebic dysentery on which he had read a paper to the Royal Society of Tropical Medicine and Hygiene in 1944. In this new line of treatment specific anti-amoebic treatment was preceded by an attack on the secondary infecting bacteria with penicillin and succinylsulphathiazole. He claimed that this produced an improvement in severe refractory cases of amoebiasis and made them more amenable to specific anti-amoebic treatment. He found no evidence clinically of emetine resistance.

Cropper (1945) discussed the value of sigmoidoscopy in

amoebic dysentery. He found that 40% of acute cases passing vegetative E.histolytica showed from one to three classical ulcers $\frac{1}{2}$" in diameter between 3 and 6 inches from the anus, but, he said, the commonest amoebic lesion is the pin-point crater, found in colonies at the same position.

In September 1945 the Army Medical Department issued a Bulletin on the treatment of amoebic dysentery which differed from that issued in 1943 in that a preliminary course of penicillin and sulphasuxidine was recommended for intractable cases which resisted the ordinary routine treatment.

Morton (1946) analysed 1,000 sigmoidoscopies, out of which there were 215 cases of amoebiasis, 52 of bacillary dysentery and 40 of lambliasis. In 20% of the 215 amoebic cases the sigmoidoscopic appearances were normal. Amoebic ulcers were present in 56 cases. Raised crateriform pits were present in 169 of the amoebic cases and depressed pits in 126 cases. 5 cases of amoeboma were seen. This author mentioned ten cases of amoebic liver abscess and 60 cases of amoebic hepatitis among his 1,000 cases which were all cases invalided to this country from abroad.

In Morton's cases the sigmoidoscopic appearances of chronic bacillary dysentery were (1) Generalised hyperaemia with excess of mucus. (2) Generalised hyperaemia with tubular stenosis and (3) superficial oval or circular ulcers with a normal mucosa intervening.

SUMMARY

SUMMARY.

The history of dysentery and of its treatment from the earliest times to the end of 1941 has been reviewed.

The conditions of life of Allied Prisoners of War in the hands of the Japanese in Singapore from February 1942 to September 1945 have been described. The incidence of dysentery in this community has been estimated.

Acute Bacillary Dysentery.

(1) The survey covers some 15,000 cases of acute bacillary dysentery. 202 fatal cases have been reviewed.

(2) In most cases of severe acute bacillary dysentery (75%) the onset was sudden with acute abdominal pain immediately followed by diarrhoea. In 25% there was a prodromal period varying from 12 hours to 6 days.

(3) The ileum was involved in 50% of fatal cases.

(4) The symptoms and signs have been discussed.

(5) In severe cases persistent hiccoughs and persistent vomiting were of grave prognostic significance.

(6) 39% of fatal cases died between the 10th and 15th days.

(7) The complications which were encountered have been described.

(8) Fatal haemorrhage occurred during the 2nd and 3rd weeks in 17 cases out of a series of 117.

(9) Two forms of peritonitis occurred (a) by direct spread of the infection through the bowel wall and (b) secondary to perforation. Sudden cessation of diarrhoea always accompanied the onset of peritonitis.

(10) The appearance of the stools of acute cases and the microscopic picture of the bacillary exudate have been described in detail.

(11) The paramount importance of copious fluids and adequate rest has been stressed in the treatment of acute bacillary dysentery.

(12) Treatment by saline cathartics and by sulphonamides has been compared.

(13) Sulphapyridine was used in a total dosage of 5 grammes for cases of moderate severity and 10 grammes for very severe toxic cases.

(14) Sulphaguanidine was used at first in a dosage of 0.1 gramme per kilo of body weight followed by 0.05 grammes per kilo every four hours. It was later used in a total dosage of 15 grammes in cases of moderate severity.

Chronic Bacillary Dysentery.

(1) Some hundreds of cases of chronic bacillary dysentery were seen.

(2) The appearance of the patient and the symptoms and signs have been described and complications enumerated.

(3) All cases were sigmoidoscoped as a diagnostic procedure and as a check on treatment. The sigmoidoscopic appearances have been described.

(4) The condition of post-dysenteric colitis was not uncommon.

(5) The difficulties of dieting dysentery cases in a Prisoner of War Camp and some methods of supplying an increased vitamin intake have been described.

(6) Methods of treatment have been described. The disappointing results obtained with most methods of treatment in severe cases have been mentioned.

(7) Sulphonamides were used in 50 intractable cases with a 90% recovery rate. Sulphapyridine was chiefly used, in a dosage of 1 gramme thrice daily for one week.

Amoebiasis.

(1) Approximately 600 cases of amoebiasis occurred.

(2) The symptoms were extremely variable. Four typical varieties of cases have been described. The principal symptoms in 150 cases have been tabulated and analysed.

(3) The daily number of stools in 100 cases have been tabulated.

(4) The macroscopic and microscopic appearances of amoebic stools have been described in detail.

(5) The methods of examining for vegetative and cystic forms of E.histolytica which were used have been described. It was found to be quite inadequate in many cases to examine two or three stools. The average number of careful microscopic examinations required was found to be 6.5. 39 exhaustive but fruitless searches were carried out in one fatal case which was found to have amoebic ulcers at post mortem.

(6) The value of the sigmoidoscope as a diagnostic aid has been discussed, and the sigmoidoscopic appearances in 100 active cases tabulated. A second similar table includes 530 cases of amoebiasis.

(7) The reaction of the stools in cases of amoebiasis, bacillary dysentery and in normal persons was investigated. All acid stools were from cases of amoebiasis or cases which were later found to be suffering from amoebiasis.

(8) The complications of amoebiasis which occurred have been discussed. The cause of death in 29 fatal cases has been indicated.

(9) The position of amoebic ulceration in 24 fatal cases has been tabulated. The ileum was involved in 2 cases.

(10) The results of treatment by various methods have been assessed. During the first year and a half the treatment consisted of full courses of emetine followed by E.B.I. and then stovarsol. The immediate results were good but the relapse rate was high.

(11) Improvised methods of treatment included acriflavine retention enemata, sulphonamides and inorganic arsenicals.

Non-specific Diarrhoea, Flagellates and Parasites.

The symptoms, signs and treatment of infestation with various parasites which were common in Singapore have been described. These included -

(a) Strongyloides.
(b) Ancylostoma duodenalis.
(c) Ascaris lumbricoides.
(d) Giardia lamblia.

Sigmoidoscopies.

(1) 5,000 to 6,000 Sigmoidoscopies were performed.

(2) The importance of examining without the patient having had a purgative or an enema has been stressed.

(3) The normal sigmoidoscopic appearances have been described.

(4) The sigmoidoscopic appearances of different types of chronic bacillary dysentery and of amoebic dysentery have been described in detail.

(5) The plates were drawn from actual cases demonstrated to the artist in the Prisoner of War Camp.

Deficiency disease due to deficiencies of some part of the Vitamin B complex was an extremely common complication of dysentery in the Singapore Prisoner of War Camp. The various improvised treatments which were used have been briefly described.

Some recent relevant literature on bacillary and amoebic dysentery has been summarised.

CONCLUSIONS.

CONCLUSIONS.

It must be emphasised that no attempt was made at any time to prepare a brief for this or that form of treatment. No system of untreated controls or comparison between severe cases treated with a form of treatment known to be inferior and those treated with some other treatment the superiority of which it was desired to demonstrate, was thinkable in the circumstances. The attention of most Medical Officers during their captivity in Japanese hands was focused exclusively on their immediate surroundings. They gave what little help they could to their fellow captives, but, without being unduly pessimistic, it seemed unlikely to reasonable men that they would ever be in a position to present a report of what had transpired to any audience outside the Prisoner of War Camp. The sudden collapse of the Japanese in August 1945 was even more unexpected to those who most benefited by it than it was to the outside world. It came at a time when the food situation in Singapore was worsening so rapidly that it was obvious that a further reduction of the rations, while inevitable, would cause disaster on a scale not yet experienced. At the beginning of August 1945 it appeared that at least one more year of war had to run its course and it was equally evident that

life could not be supported for that length of time on rations at or below the level they had then reached.

However, although no attempt was made to prove any contention in the work described in the foregoing report, it may be permissible to draw certain tentative conclusions:-

1. In the severe cases of acute bacillary dysentery encountered in Singapore sulphonamides were more effective than any form of treatment previously used. Of the sulphonamides used, sulphapyridine was the most effective in severe toxic cases. No cases of renal disorder due to the use of this drug occurred probably because of the small total dosage employed and the insistence on a high fluid intake.

2. In established cases of chronic bacillary dysentery sulphapyridine was a most effective drug. This treatment resulted in extremely rapid healing of the ulcerative condition in the bowel as shown by sigmoidoscopy and by complete clinical recovery.

3. The sigmoidoscopic appearances of chronic bacillary dysentery were extremely variable. The appearance which the author regarded as being most suggestive of this condition was one of superficial irregular ulceration, with pus adherent, the intervening mucosa being red and oedematous. In association with this, masses of granulation tissue oozing pus were common. Other appearances in order of frequency were clear-cut round or oval ulcers, containing pus, with an intervening mucosa which might be of fairly normal colour, but which was almost invariably slightly thickened; and a dusky red, oedematous mucosa with excess of mucus, but without ulceration.

4. It is recommended by most authors that at least three stools should be examined for E.histolytica before a negative can be accepted, and this is the established practice in many hospitals in this country. Experience in Singapore would appear to suggest that this is quite inadequate as the average number of careful microscopic examinations required to establish a diagnosis was 6.5.

5. As an aid to diagnosis the pH of stools was investigated. A pH of less than 7, either immediately the stool was passed or two hours later, was found to be evidence in favour of a diagnosis of amoebiasis. While it is realised that these results probably only apply to persons on a high carbohydrate low protein and fat diet, estimation of the pH of stools as an aid to diagnosis would appear to be sufficiently promising to warrant further investigations with patients on a normal European diet.

6. 29 cases of amoebiasis were fatal and in each of these some complication was present. That 11 fatal cases had a superadded bacillary infection indicates acute bacillary dysentery is an extremely dangerous complication in cases of chronic intestinal amoebiasis.

7. It is considered important that sigmoidoscopy should be performed whenever possible without a preliminary enema being given. It was found that preparation of this sort invariably caused some alteration in the sigmoidoscopic appearances.

8. After doing more than 5,000 sigmoidoscopies the author finds it impossible to be dogmatic on the sigmoidoscopic appearances of intestinal amoebiasis. In 14.3% of sigmoidoscopies on cases of amoebiasis the sigmoidoscope revealed a normal mucous membrane. In the absence of ulcers the signs most suggestive of amoebiasis were patchy inflammation of the mucosa and punctate submucous haemorrhages. The commonest form of amoebic ulcer was the punched-out ulcer, containing pus and surrounded by a red areola. The intervening mucosa was usually perfectly normal. Deep ulcers of various types were the next most common. A condition of acute amoebic ulceration was very common, especially in 1944 and 1945 when the general resistance to disease was lowered. This type occurred quite frequently during exacerbations in chronic cases of long standing. The teed-up ulcer was invariably of amoebic origin. A red areola surrounding an ulcer, or a series of minute bleeding points along its edge were signs of activity.

9. Many cases of intestinal amoebiasis proved resistant to or relapsed after repeated courses of emetine followed by E.B.I. and then by stovarsol or other organic arsenical.

As an improvised non-specific treatment retention enemata of acriflavine in normal saline were of definite value in holding the infection in check. Sulphaguanidine and sulphapyridine were used in the treatment of amoebiasis and found to be an adjuvent to specific anti-amoebic drugs. They were also of value in the absence of specific treatment in checking exacerbations but the effects were not lasting. This suggests that the sulphonamides have a definite place in the treatment of amoebiasis in neutralising the effects of secondary infection in chronic cases.

10. Only very small amounts of emetine were necessary to arrest dangerous amoebic dysentery in most cases. Frequently a total of 2½ grains was sufficient. In some cases who had had repeated courses of emetine a short course of stovarsol was more effective in arresting a dangerous relapse than emetine. This suggests that it is possible to produce an emetine-fast strain of E.histolytica by giving repeated courses of emetine. On the other hand the fact that the majority of relapses in cases who had previously had repeated courses were arrested by 2½ grains of emetine suggests that the production of an emetine-fast strain is not a common phenomenon.

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