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The Practice of Tropical Medicine from the Consultant's Chair

BY

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"Most mistakes in medicine occur, *not* from not knowing, but from not looking."

—Patrick Manson.

The practice of tropical medicine in London tends to pose rather a different proposition from what it is popularly supposed to be. It does not take long for the consultant to recognise that the *tropical side* is just but one facet of the composite picture. He soon finds himself in a morass of differential diagnosis when faced with the gamut of those diseases which man is heir to. For, wedged in between what is thought to be malaria, amoebic dysentery, sprue and helminthic infections—or what have you—there are possibilities of a more mundane nature which have to be considered.

It becomes a matter of *Experientia docet*—often one is moved to exclaim, "Oh where, oh where have I seen anything like this before?"

More often than not, the consultant is appealed to judge upon rival claims to priority in diagnosis, and he has to decide too in a very short space of time. The patient may be in such a hurry to get away to catch the next train or plane that no time is available to undertake what seem to be the necessary minimum investigations.

Most, when coming from afar, arrive armed with X-ray films and reports of laboratory examinations carried out elsewhere, and it is difficult indeed to decide whether what has been found before is actually decisive or diagnostic. It is always a question whether all the laboratory reports of the discovery of intestinal protozoa are any longer applicable.

But, above all this, I would endeavour to emphasise, as more important than any other evidence, the general appearance of the patient and the impression he makes on one's mind, of his colour, bearing, carriage, expression and how much of the general picture should be attributed to physical or to psychological causes; or as to whether or not there is really some underlying pathology overlaid by introspective neuroses.

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To try to evaluate the interpretation of these various factors and to spot the real cause of the patients' troubles is to feed upon the fund of past experience.

In spite of all the advances of technique in the avenues of scientific diagnoses, I would emphasise the overriding importance of taking a minute, careful history and of getting down the patient's own account of his feelings. The past history of disorders and upsets is of special value in dealing with intestinal diseases. Let us take the diarrhoeas and dysenteries first. Amoebic dysentery has become a very popular complaint and a very important one from the layman's point of view. Nearly every traveller from the tropics is convinced that he has got it and in some crude fashion is familiar with all its textbook possibilities. In my own opinion this impression is really far from the truth. We are all aware of the infinite variety of symptoms which this infection may evoke. However, I remain convinced that, when a genuine sufferer from amoebiasis turns up, there can be little doubt about it.

There is, generally speaking, an "amoebic facies." The myxoid complexion is dark and muddy and the expression anxious. The nature of the diarrhoea, the alternating bouts of constipation, the shifting abdominal pain, the flatulency and general discomfort—all are suggestive. A story of subacute attacks of dysentery with blood and mucus in the stools may be elicited. In spite of this, the loss of weight, as a rule, is not marked (in contrast to bacillary dysentery).

It is, of course, quite possible that your patient may be suffering from amoebic dysentery *without* the ascertainable presence of amoebae or cysts in the faeces. However gloomy this defeatist attitude may appear, I insist that, as a general rule, the appearance of the mucosa, as viewed by proctosigmoidoscopy, is usually suggestive. It is also quite true that *E. histolytica* cysts may be found in the faeces and that, even so, they may not constitute the only factor in the diagnosis.

Regarding the physical examination of the abdomen, this must always be meticulously carried out, as it may produce valuable information. I would stress the importance of bilateral tenderness over the caecum and sigmoid and in particular a sensitive spot in the central sigmoid loop in the left iliac fossa which I have termed "the amoebic point," while some thickening and induration are evident in the

lower section of the large bowel. The lower edge of the liver is occasionally palpable.

Upper abdominal pain and tenderness are not an accompaniment of amoebiasis nor are nausea and vomiting. I make a special point about this, because I have had referred to me at various times as amoebiasis cases of gastric, duodenal ulcer, gastric carcinoma, cholecystitis and gallstones which have been later revealed by skiagraphy.

Of other abdominal diseases which amoebic dysentery may simulate, perhaps the most frequent are ulcerative colitis and diverticulitis. The former steals in like a thief in the night, but the intensity of its onset, the extremely painful colon, the beef-like redness of the mucosa and its vulnerability, together with the "strawberry juice" stools, proclaim its identity. Still, I have seen very acute cases of amoebic dysentery diagnosed and treated as ulcerative colitis, but the *vice versa* is much oftener the case.

Diverticulitis may simulate amoebic dysentery very closely and, indeed the two may co-exist. This brings me to the next point. Can one always trust the findings of sigmoidoscopy? One thing I am certain of is that no one, without great experience, can be relied upon to recognise all the varying features of amoebic lesions with certitude. Every suspected amoebic ulcer should be scraped with a biopsy spoon with a view to finding the living entamoebae. Failure to do so may lead to disaster. For instance, a surgeon, who is most expert, diagnosed amoebic dysentery by demonstrating petechial lesions in the sigmoid. The patient also had a choleric facies with an enlarged hard liver, but there was something striking about his dull leaden-like complexion. He also had diarrhoea with peculiar dark bilious stools. Treatment with emetine and emetine bismuth iodide made matters only worse; nevertheless, further investigation revealed a nocturnal haemoglobinuria and that, quite unexpectedly, the real diagnosis turned out to be haemachromatosis. Both mucous and ulcerative colitis are frequently misdiagnosed as amoebic dysentery by relying too much on sigmoidoscopy, but of all the tragedies the greatest is the non-recognition of bowel carcinoma and of mistaking it for amoebiasis. Rarely, it is true, both may be found together. Indurated intrarectal ulcers of amoebic origin may readily be taken for malignant disease in the absence of microscopical investigations. Unfortunately I have found that a barium enema is of little help in making a

diagnosis of amoebiasis, except when the process is confined to the caecum, when there may be filling defects and distortion of the *caput caeci*.

The presence of piles is also not of much assistance, as they are common in different forms of dysentery as well as in malignant disease.

As regards the microscopic recognition of *E. histolytica*, there is usually no difficulty about the active vegetative form, but it is otherwise with the cyst.

At the present time protozoologists are engaged in what might be termed the process of debunking the *Entamoeba histolytica*.

We have now to recognise the existence of *coprozoic* commensals of this species which pass their lives in the faeces, like *E. coli*, and produce four-nucleated cysts. Their presence is harmless in what are known as "symptomless carriers." The large race of *E. histolytica* is potentially always pathogenic, but there also exists a *small* race, also a harmless commensal, also producing *small* four-nucleated cysts and which henceforward should be known as *E. histolytica hartmanni*. To make matters even more complicated, some authorities recognise the existence of a *small* race of genuine *E. histolytica* which, like the larger race, is pathogenic.

At this stage one feels impelled to exclaim:

"Also there are not three incomprehensibles and three others not created, but there is one which was created, but is now incomprehensible."

Moreover, *E. histolytica* is capable of producing a great range of pathological lesions in the large bowel, depending upon the intensity of the infection and the resistance of the tissues.

The deep, angry and sometimes gangrenous ulcerations in the Bantu, resembling a ploughed field, are the result of repeated reinfections and differ from those which obtain in those living under more hygienic conditions, in whom extensive destruction of the tissues does not commonly ensue. In Europeans there may be small pin-point depressions in the mucosa or localised granulations, and there are others in whom no lesions of the bowel can be seen within reach of the sigmoidoscope. The variations in the post-parasite relationship are almost limitless. Different laboratory animals, as is well known, react differently to amoebic infection. The kitten is so susceptible that in this animal *E. histolytica* produces an acute and rapidly fatal disease. The dog, on the other hand, is so tolerant that a "carrier" strain produces no symptoms, but when the diet is changed to salmon the infection becomes acute and deep

amoebic lesions are found at autopsy (Burrows, R. B. (1957), *Amer. J. Hyg.*, 65, 172).

As for extraintestinal amoebiasis, this sometimes creates a state of affairs which may best be termed the "asses' bridge of tropical medicine."

To the non-tropically-minded this combination of hepatic and pulmonary signs offers scope for a variety of diagnoses. It always gives a sense of satisfaction to unravel these puzzles. When confronted with this symptom-complex the discovery of *E. histolytica* cysts in the faeces imparts a great sense of security in rendering the diagnosis a certainty.

This may be illustrated by the story of an official on his return to England after a brief visit to Addis Ababa. He had pyrexia, hepatic pain, cough and pulmonary congestion at the right base, and moreover, a history of a short attack of dysentery, the significance of which had been overlooked. This combination defeated the diagnostic capabilities of a series of physicians who failed to connect his illness with previous residence in a tropical country. The discovery of *E. histolytica* cysts in the faeces, together with the ragged appearance of the rectal mucosa, clinched the diagnosis. If any other proof were needed, it was the almost instantaneous recovery on emetine and emetine bismuth iodide treatment in contradistinction to the lack of response to aureomycin with which he had previously been subjected as a kind of blunderbuss treatment.

Bacillary dysentery, in the acute stage, now seldom comes under purview in London as in former days. In the past it has come my way to isolate Shiga and Flexner bacilli from the bowel and also from the faeces, and I think the highlight in this respect was from a Colonel who lived in Piccadilly Circus; and there was a period when Sonne infections were not uncommon and produced manifestations which reminded me of the dysentery days of the first world war. Formerly, too, eye and joint complications made their appearance in the consulting room, but now they are never seen. Post-bacillary intestinal cases are still not infrequent. Most of the so-called "tummies" of the Near and Far East are, in fact, due to mild forms of bacillary infection, and the story they relate is quite common amongst air passengers. They all give a similar history of sudden onset, mild fever (occasionally), acute abdominal pain and explosive diarrhoea with mucous discharge. Usually there is mild dehydration with some loss

of weight. More often than not, for some obscure reason, they are considered to be acute cases of amoebic dysentery and treated accordingly. The incubation period is a very short one, while in amoebiasis it is a matter rather of weeks and months. To make exact recognition of the invading organism is by no means easy. It entails the time-consuming process of isolating dysentery bacilli from the faeces or from the rectal mucosa. This is rarely successful and then, of course, there are agglutination tests which are not always reliable, but I think the most important of all is the appearance of the rectal mucosa, which is plum-coloured red, angry and slightly granular. In the exudate there are usually pus and plasma cells.

I shall never forget the picture of a wizened officer who hailed from Bombay, where he had suffered from a very acute attack of febrile dysentery with blood and mucus stools which laid him low; but in spite of all these typical signs and symptoms of bacillary dysentery, he had been subjected to intensive emetine treatment. No wonder he looked and felt so ill. His serum agglutinated Shiga's bacillus in a dilution of 1:1,200.

Another bogey is mucous colitis, nearly always associated with a spastic colon, an intensive nervous temperament combined with a real degree of introspection. The origin of "mucous colitis" is very difficult to ascertain; quite often the basis is chronic constipation.

A spastic colon is usually readily palpable in the left iliac fossa and is slightly sensitive. This contraction is readily confirmed by barium enema, but is not always feasible. To the practised hand, palpation of the colon imparts a distinctive rubbery sensation. Mucus-bearing cells may be seen in the faeces and swollen goblet cells in rectal "scrape" preparations.

Lambliasis, or giardiasis, an infection of the small bowel with *Giardia intestinalis* is by no means an uncommon intruder on the scene. That such a pathological entity exists there is no reasonable doubt. These protozoa may be present in enormous numbers in the faeces, where they are seen in clumps or strings. Strangely, the active trophozoites are by no means always apparent.

The symptom-complex is a tense inflated abdomen with a marked degree of meteorism, a history of explosive diarrhoea and the passage of acrid pale faeces. The steatorrhoea which accompanies this protozoal infection is well-recognised and is apt to be confused with sprue.

Fortunately giardiasis reacts rapidly to mepacrine and chloroquine and this response is diagnostic.

Sprue is by no means so common as it was 30 years ago, but it still turns up from time to time and it is the one important abdominal disease of tropical origin which is most frequently overlooked. This is mainly because laboratory reports on the faeces are particularly unhelpful. Probably my most dramatic and exciting clinical experiences have been with this disorder. Most physicians in this country have never seen it, and to others the very name is unfamiliar. Thus when a sprue patient presents himself the true import of the copious faeces and the strange diarrhoea is overlooked. Another peculiarity which is very misleading is the tendency of the disease to develop in those who have formerly lived in the endemic area. Not infrequently the "latent period" may be seven years or even longer. The symptoms, together with the obvious emaciation and the character of the diarrhoea, are apt to call up visions of gastric carcinoma, pernicious anaemia or gall-bladder disease.

It happened that during the last war there was a call to a hospital in the vicinity of the metropolis, where a middle-aged woman was being prepared for cholecystectomy. Her mouth had suddenly become sore and aphthous ulcers had made their appearance on the tongue. The sister in charge was horrified because I insisted on viewing the stool before making a physical examination. However, there could be little doubt about the diagnosis. The specimen was large, copious and bulky, with froth and bubbles of a peculiar creamy yellow colour and exuding that penetrating fatty acid odour which is quite unlike anything else. I sincerely believe that this physical appearance is much more reliable than elaborate faecal fat estimations. This somewhat crude identification of sprue excreta is of special value in so-called atypical cases, where there are no mouth or tongue symptoms to guide one. The diagnosis proved in this case to be correct and a remarkable recovery ensued. This agreeable issue was confirmed a year later when a florid and buxom female in ARP uniform burst into my room to announce herself as my former sprue patient.

On another occasion a sprue victim was unearthed in the ENT department of a London hospital, where he was undergoing treatment for an infected antrum and where tongue and mouth signs had been mistaken for sepsis. On other occasions it has been treated as secondary

syphilis. The microscopic appearance of the faeces is suggestive. Here distinctive features are fatty-acid crystals, fatty globules and cholesterol plaques. There is usually, but not always, some megalocytic anaemia. Personally I place some reliance on proctoscopic inspection where the mucous membrane is lax and lustreless, and quite often colourless faeces may be seen trickling down from the sigmoid, which is really a most distinctive phenomenon. Some importance too must be allotted to the clinical history, especially to the explosive nature of the diarrhoea, the excessive flatulency and the sense of relief and exhaustion which follow the passage of a stool. The diarrhoea is of the early morning variety and the patient is roused from his bed by urgency. In the physical examination, attention is directed to the dough-like sensation on palpating the abdomen, the shrinking of the liver, the harsh dry skin, the soft pulse and low blood pressure. Most striking of all is the sad drawn expression; indeed, to the practised eye the diagnosis is stamped on the forehead. The whole attitude is "spruey" and there are signs of emaciation in the face and neck, with exaggeration of the naso-labial folds.

The subject of fevers and their differentiation is an immense business. Some 30 years ago fever in the tropics was synonymous with malaria. Everybody had it or could "feel it in their bones." The "feva" of the pukka sahib fresh from the torrid plains of India was as renowned as his *chota peg*. It became a matter of great import to try to separate the sheep from the goats. When once one had contracted malaria, rumour had it that it lingered for ever. It racked the wizened frames of veterans of frontier wars long ago as they shivered by the fire and fought their battles over again. Nowadays it is quite exceptional for malaria to be seen in the consulting room, but it behoves one to be watchful lest one should be caught unawares. In order to arrive at a correct diagnosis of malaria, especially in those who have already been subjected to treatment, it is necessary to take a very careful history of the patient's sensations in his own words—the story of the rigor, the shivering, the sweating, the intense burning headache, followed by the drenching sweat which soaks through the bedclothes, should be distinctive of benign tertian, the most common parasite. This dramatic sequence of events is less marked in the subtertian, which is the dominant species in those who come from West Africa, and may not occur at all in the quartan. There are other signs: the spleen, for instance.

Is it always palpable in every case of genuine malaria? The answer must be "not by any means."

In primary cases of benign tertian the infection may be so slight that the spleen may not be enlarged. In acute subtertian cases it may be congested, but not palpable. There are indeed quartan fevers of 10 years' duration without splenomegaly. The technique of palpation is important. The supine position is not the most favourable for distinguishing, either the edge or the notch. For many years I have been accustomed to adopt the lateral position in which the patient reposes on his right side with the left arm extended horizontally. The spleen is thereby pressed forward so that the edge impinges against the index finger. It is this technique which has often made a diagnosis in an obscure case. But again, "when is a spleen not a spleen" may well be asked. The answer is, "When it is an enlarged kidney." This has been a not uncommon error. Of course, the blunt lower pole differs from that of the spleen, but it has been my experience that a polycystic left kidney may be mistaken for it.

This happened once in an officer who had been drawing a pension for malaria for 12 years till it was spotted and the kidney removed. Another paradox may arise in those rare cases of amoebic abscess of the right lobe of the liver with compensatory hypertrophy of the left, which may simulate malarial splenomegaly.

Subtertian may sometimes be very difficult to recognise by even the well-practised. The acute febrile stage with bloodshot eye, bone pains and headache is easily mistaken for influenza and treated accordingly. Sometimes, too, the true nature of the fever remains unrecognised until it is too late. Other mistaken diagnoses spring to mind, such as typhoid or paratyphoid. I have even seen it treated as acute tonsillitis in a young soldier who happened to have a sore throat grafted on to his malaria. On account of a painful and tender liver and distended gall bladder which may accompany subtertian, cholecystitis may be suspected. In difficult cases the sherry-colour of the urine with intensive urobilinuria is confirmatory. In performing blood examinations, thin and thick films must be taken, of which the latter are most helpful, because the infection may be so scanty that the small rings may readily be picked out. In quartan malaria it is often the case that parasites may be few and far between. I have had some dramatic experiences in London when one half-grown

trophozoite was demonstrated after 24 hours' continuous search. Sometimes added assistance can be obtained by finding a melanin-containing leucocyte or basophilic stippling of the erythrocytes.

Common infections most likely to mimic malaria are bacterial affections of the genito-urinary tract. The infecting organism is usually *B. coli*, possibly derived from the bowel. Acute pyelitis and bacilluria are common in women and may produce such paroxysms with rigors as to simulate malaria. In men, ascending infections from the prostate may do the same. I have also seen renal and vesical calculi mistaken for malaria. Infective hepatitis in newly arrived passengers from the tropics may cause chills and rigors and mimic malaria, too, before icterus becomes visible. In these the bile-stained serum is a reliable portent.

To steer a true course the intestinal aspect of malarial infection must not be neglected. In this variety there may be no history of fever, or indeed the temperature may be subnormal, but the patient's story is gastrointestinal, with vomiting, diarrhoea and abdominal pain. I have known of tragic cases where, while at sea, they have been fobbed off as sea sickness. Subtertian malaria should always be at the back of one's mind.

Quite recently a Belgian traveller arrived from Lake Kivu (Congo), where he had been studying big game. Back in Brussels, he began to suffer from acute indigestion, diarrhoea and fits of vomiting, which continued for three months, without malaria being suspected. On arrival in the consulting room he presented that peculiar ochreous tint which shines through the sun tan with just that tinge of green which goes to make the subtertian complexion. He had an enlarged, hard spleen, while scanty subtertian rings were found in the blood. The beneficent action of chloroquine completed the picture. There may be some grounds for the popular belief that malaria can persist for years, for it is perfectly true that the quartan parasite may live in the blood for 10 years or even longer, and fatal results have ensued from blood transfusions when this factor has been ignored. On the other hand, the lifespan of the subtertian is short and only exceptionally have I seen relapses recorded after the lapse of one year. In benign tertian I have found malarial rigors with parasitaemia in patients resident in England a year and a half after their return from India in the absence of a previous history of fever.

It is now more than 20 years since a case of blackwater fever occurred in my practice, but once upon a time there were three or four every year. The total number was altogether somewhere about 25. There can be no doubt that this dwindling figure has run parallel to the incidence of subtertian malaria as a whole. I think that the conclusion must be that these explosions and blackwater were to some extent attributable to the large doses of quinine which were popular in those days.

There were some remarkable experiences. I remember especially a fatal attack in a young fly-fisherman in Hampshire. He had returned from Uganda some eight months previously and no one realised that he was infected with malaria at all till he fell into the Test and got thoroughly chilled during a cold spell one June day. When it was realised what he had got, and specialist opinion was sought, it was too late.

Then there is one other experience which has stuck in my mind when one morning two officials from East Africa came to my consulting room. One was a colonel who had just been decorated at the palace and the other had been in the PWD. Both had suffered from subtertian malaria and both were in what I recognise as the "preblackwater state." In both, small numbers of subtertian malaria parasites were present in the blood: in both, the urine contained an excess of urobilin. It was obvious that both should be hospitalised as soon as possible. The first-named was a sensible fellow, came in, was kept warm and carefully dosed and made a rapid recovery; the second refused, as he wished to see a football match at Plymouth. So he took a large dose of quinine, set out on a raw cold afternoon, and within four hours was down with a virulent attack of blackwater, and in another six was dead with anuria. It was recorded that from start to the fatal finish he passed only six ounces of urine.

There are many other causes of fever and I have seen a dengue rash in London and have studied the rheumatic-like after-effects of this disease.

Fevers of the undulant type were on the menu 30 years ago and sometimes produced malaria-like rigors, especially Malta fever (*Br. melitensis*). It was Manson who exclaimed that he had never seen one who had not been previously *drenched* with quinine. Since these times *abortus* fever has taken its place. Even when there is splenomegaly there is little resemblance

to malaria, and the macroscopic agglutination test is always at hand.

There was a time also when kala-azar came into the picture of differential diagnosis, and on one afternoon, I well remember, I had a case of this leishmaniasis and two of malaria in army officers in my room at the same time. To differentiate between them was a delicate task to settle quickly. The much enlarged liver and hard spleen with some oedema of the legs, backed up by the leucopenia and the formol-gel reaction, settled the diagnosis in favour of the former.

Trypanosomiasis too may have to be reckoned with. In those early days it came my way to spot this rare event by recognising the scarlet erythematous circinate rash, once on the shoulder and on other occasions on the chest and back in colonial officials newly arrived from West Africa. Enlargement of the cervical glands is not always present and there may also be splenomegaly. I soon found that the trypanosome is not easy to demonstrate, even in thick films. It is a fact too that the considerable cervical adenitis may lead the diagnostician astray. There have been instances (especially in *T. rhodesiense* infections) which have been treated as lymphadenoma until it was too late. Another case was regarded for months as malaria till a fatal coma ensued; once I had a mixed infection of *T. gambiense* with subtertian malaria (*P. falciparum*).

We all get stuck some time or other on PUO, which is not a diagnosis so much as a prophecy. In a search from head to foot we may chance upon the crux of the matter. It may be teeth; it may be an infected sinus or antrum. It may well be an acute tubercular infiltration of an apex; it may be a mediastinal lymphadenoma, or that extraordinary variety, the Pel-Epstein disease—all have come my way. Endocarditis comes into the picture, as also do post typhoidal cystitis or cholecystitis. In one particularly puzzling case the cause was found to be a small prostatic abscess, and I remember well a medical officer whose case dragged on till an uroselectan injection revealed a congenital double pelvic ureter which contained a hidden pocket of pus. There was one other PUO in a R.N. seaman in the sick bay with a severe intermittent pyrexia of over a month's duration without any ascertainable signs. I was about to give up in despair when I came across a small hard pimple in the perinaeum. On pricking it a head of pus escaped in which tubercle bacilli were demonstrated. It was indeed a

small ischiorectal abscess. Since that time I have succeeded on several occasions in repeating this diagnosis in scrape biopsy preparations obtained by proctoscopy.

Helminthic infections play a considerable part in this kind of practice, and of all worms, the whipworm is perhaps the most common and interesting because it gives rise to a considerable eosinophilia in the blood which is only out-distanced by *Loa loa*, common in those who come from S. Nigeria.

This is undoubtedly the most frequent member of the filariasis group in the consulting room, especially when it gives rise to Calabar swellings which may be taken for angioneurotic oedema. Lymphoedema of the legs has also been seen in this infection. Of other species of filaria, the most frequent is *D. perstans*, which does not cause any trouble really, but allergic swellings on the arms and legs which I have seen, in the absence of any *Loa loa*, may have some connection with this parasite. In recent years *Onchocerca volvulus* has turned up from Sierra Leone and the Congo. In some the lichenoid skin on the back may suggest this infection, and ocular manifestations with pannus have been twice recorded in Europeans in whom sight was restored by corneal grafting. *W. bancrofti* is rare, but the non-periodic pacific form has been seen in Fijian soldiers; lymphangitis and elephantiasis in Tahitians, but mostly these manifestations are examples of elephantiasis nostras, Milroy's or Meige's disease.

The eggs of *Ascaris* are not an uncommon finding, especially in the faeces of children. In them round worms appear to be one of the chief causes of abdominal pain.

The ancylostome usually causes no alarming symptoms, but sometimes someone turns up with accompanying anaemia. But the chief role it plays is in simulating gastric or duodenal ulcers. The pain, which becomes apparent after food, is referred to the epigastrium. Ancylostome infection may, moreover, give rise to occult blood in the faeces. This combination may prove to be a real danger, for it is on record that a patient had been diagnosed as a duodenal ulcer and gastrectomy performed before it was discovered that he was heavily infected with ancylostomes, but by then he had lost a large portion of his stomach!

Tapeworms, particularly *T. saginata*, are frequently diagnosed by the appearance of its eggs in the faeces in the absence of proglottides. *Hymenolepis nana* has been seen very rarely

nowadays, as also *T. solium* and *Diphyllobothrium*. The eggs of *Heterophyes heterophyes* have once been found in an officer from Egypt with diarrhoea.

Of the *Bilharzia* worms, the most frequent is *Schistosoma mansoni*. More usually the eggs have been demonstrated, not so much in the faeces, as in biopsy preparations from the rectum. In heavy *S. haematobium* infections it has been my experience to find terminal-spined eggs in the rectal mucosa as well as in the urine. The eggs of *S. intercalatum* from the Congo have been seen once.

Strongyloides stercoralis larvae are commonly found in diarrhoeic faeces and do not appear to give rise to any special symptoms, but the aberrant intradermal migrations resembling *Larva migrans* have sometimes to be recognised.

The skin plays an important part in tropical medicine, so it is necessary to keep one's eyes *skinned* when patients arrive by sea. Many of the curious rashes are due to scabies contracted from ships' blankets; often they arrived labelled "prickly heat" or impetigo.

Urticaria of various types may be an expression of helminthic infection, but usually it has a nervous basis. Since the introduction of the antihistamines it has not been such a bug-bear. Psoriasis turns up in various guises and may be taken for syphilis or ringworm. Surprisingly, psoriasis of the nails is comparatively common and sometimes difficult to distinguish from that particularly pernicious fungus infection; usually psoriasis rashes are benefited by tropical climates.

Eczematous eruptions and seborrhoea are usually aggravated by heat, but nowadays yield to cortisone therapy. Of the fungous skin diseases, tinea circinata is the most common; the rings may fuse and cover large areas of the body. Dhobie itch (*Epidermophyton floccosum*) in the crutch often occurs and is apt to recur, however efficient the treatment, and has to be differentiated from intertrigo.

Fungous infection of the feet, or athlete's foot (*Trichophyton interdigitale*), has become surprisingly frequent in recent years and is often accompanied by extensive allergic manifestations and may give rise to lymphangitis and even lymphoedema of the legs, which may simulate elephantiasis and may therefore be mistaken for filariasis.

The vestiges of the prickly heat rash in the mammary regions and in the belt area are not uncommon.

Leprosy, in its manifold manifestations, may reveal itself when least expected. I have seen it mistaken for seborrhoea, psoriasis and fungous disease. There was one particular case which I can never forget in a young engineer from Malaya. As far as I could learn he had never been in actual contact with leprotic cases and had only visited a leprosarium once in performance of his duties. On routine examination a rash on both legs was evident and which had up to then been treated as intractable seborrhoea. On enquiry he stated that recently both legs had become "wooden." There was no doubt about the diagnosis when leprosy bacilli were demonstrated in biopsy snip preparations as well as in the nasal mucus. Unfortunately both eyes became subsequently affected, but he has since made an astonishing recovery on sulphone treatment. Perhaps the most remarkable experience in leprosy diagnosis was in a dustman of a rural county council who was referred by the M.O.H. in 1942 as a case of intractable seborrhoea of the cheek and nose which recently had become ulcerated and exuded an ichorous discharge in which *M. leprae* were abundant, and they were also demonstrated in scrapings from the surrounding skin. Madarosis and blepharitis were present, as well as thickening of the right ulnar nerve. He had in his youth served as a soldier in India and S. Africa 38 years previously, so that the incubation of the disease, until its true nature became manifest, was a very long one. Fortunately, perhaps, he died of pneumonia seven years afterwards.

One of the most unintelligible neurotic states is leprophilia. One young man who had served in a clerical capacity in a leprosarium in Nigeria had learned to simulate leprotic anaesthesia so perfectly that he had fooled most of the experts, but eventually he was debunked by a well-known neurologist who applied a red hot knitting needle to the soles of his feet, when the anaesthesia disappeared very quickly.

Oriental sore (*Leishmania tropica*) turns up in a variety of disguises. It may simulate many other conditions such as lupus, syphilis and even rodent-ulcer; especially is this the case when it attacks the pinna of the ear, but when it occurs on the nose (Tapir nose) it may be taken for rhinophyma and so on.

It is sometimes very difficult indeed to find the L.D. bodies and there have been occasions when it has taken hours of careful scrutiny to discover one typical parasite. A sore on the lip may look uncannily like a syphilitic chancre.

An amusing situation arose when a young officer from the N.W. Frontier of India arrived with such a sore, accompanied by his fiancée and two elderly worldly-wise aunts who had firmly forbidden him to kiss their ward till it had been proved that it was not just what they had suspected. The sequel was that I was invited to the wedding, gave the inevitable present, but was ill-rewarded when I never in return received my well-earned fee!

The perinaeum is not the most usual location for dermal leishmaniasis, but it is perfectly true that once I was able to demonstrate L.D. bodies in a swelling which was situated within an inch of the anal margin for a great authority on rectal surgery. There is a lupus-like form of oriental sore usually situated on the cheeks or forehead with apple-jelly nodules and which is almost an exact replica of tubercular lupus. It is difficult to diagnose because the contained leishmania are few and far between.

Septic sores on the face and chin may somewhat resemble oriental sores, and I remember one in particular where the origin was a dental abscess with a fistula which had tracked down to the chin.

Tropical Eosinophilia (pulmonary eosinophilosis).—This has become a popular disease and is frequently being diagnosed in tropical residents; often it is a cause of *faux mieux*. Long before the discovery of this disease by Weingarten in 1943 I had been puzzled by the extremely high eosinophilias (60 to 80 per cent.) in the blood of some tropical cases which were referred to me. In these cells the granules appeared to be larger and more refractile than those found normally in an eosinophile response; most of these cases were not really ill and there was nothing to connect them with any kind of pulmonary affection. I now realise that it would have been more satisfactory to have had an X-ray examination of the lungs. As the result of recent investigations by Danaraj in Singapore, it appears very likely that pulmonary eosinophilia may be a manifestation of "visceral larva migrans." The response to tetrazan therapy indicates that it is probably of filarial origin.

Space does not permit a discussion upon all the endocrine disorders and the vitamin deficiencies which have entered into this purview of 35 years' practice in the metropolis, but I would like to refer briefly to the thyroid.

There have been occasions when the visit to my consulting room was prompted by intestinal

disorders due to this gland. The first was in a lady from India who was afflicted by "dysentery" and constipation. Although she was comparatively young, well-made and still handsome, I was struck by her somewhat shuffling, stumbling gait. She was rather hesitant in speech and slurred her s's and r's. The dysentery was due to stercoral ulcers in the rectum and the real diagnosis was myxoedema. She made a wonderful recovery on thyroid extract. An interesting observation was made by the matron of the Hospital for Tropical Diseases, who confirmed my diagnosis by watching her *step out of the lift*. She said it reminded her of her early days in the London hospital many years ago at a time when myxoedema was common.

The second was the exact opposite. This time it was a medical officer returned from Colonial service. His complaint was diarrhoea and abdominal discomfort associated with hyperexcitability. His hands showed a fine tremor, his eyes were startling and prominent, but no enlargement of the thyroid gland could be found.

However, a flash diagnosis of exophthalmic goitre was confirmed by X-rays, which showed clearly that the enlarged gland was substernal.

Before leaving this part of my story I would like to refer readers of this essay to the paper by myself and my very valued assistant—Mr. W. J. Muggleton—on "Rectal Biopsy as an Aid to Diagnosis" (*Lancet*, 1957, 1, 763). This is a very simple method within the reach of all and it may be of some interest that many of the diagnoses described above were verified by this method. By this method too it has been possible to demonstrate non-pathogenic amoebae besides *E. histolytica* and its cysts, as well as the ova of *Ascaris lumbricoides*, *Taenia saginata*, *Schistosoma haematobium*, *S. mansoni*, *Oxyuris vermicularis*, with adults and ova—also occasionally tubercle bacilli.

THE LIGHTER SIDE

The professional and serious side of consulting practice is embellished and enlightened by amusing incidents.

Once a particularly stout pair appeared for examination. The man weighed over 20 stone. Although the exterior appearances were all against it, to my surprise he was found to be suffering from pulmonary tuberculosis with pleural effusion. When this had been settled he enquired about his lady companion, who had such an overlay of abdominal fat that the lower portion hung down like an apron which dangled in front of her thighs, impeded her move-

ments and obscured the outlines of her figure. He begged if it might be possible to remove her mass of redundant adipose tissue. I replied that considerable risks might be involved in such a serious operation. Six months later they reappeared. They had been to Paris. The lady now had a passable figure with the outlines of a waist, but she was anxious to describe the sequel. "Imagine my surprise," said she, "when I found a parcel on my table, bearing a Paris postmark, just before Christmas. There were many wrappings of tissue paper to be unravelled before I came to a brown envelope on which was pinned my surgeon's card wishing me the compliments of the season—in French, of course. Inside I found what appeared to be a pair of 'pigskin' gloves which bore the inscription, 'These gloves have been made out of your own skin!'"

My colleague was holding a clinic in the ward where one of my patients, protected by a screen, was slightly delirious with pneumonia and was uttering some loud but not always very complimentary remarks. My colleague, who was annoyed at this interruption, poked his ruddy countenance over the screen, exclaiming, "Stop that noise, can't you?" "Hello, old frosty face," cried my naughty patient.

There happened to be a poet handy who composed an appropriate hymn which ran as follows:

"Lo, he comes the stairs ascending
Attendant angels in his train.
Thousand, thousand lads proclaiming,
'Frosty face is here again.'"

It was the same wit who was undergoing treatment for amoebic dysentery and who embellished his bed sheet with the following prayer:

"Keep peace in our tum, O Lord,
Make and keep me clean within."

About this time one of my colleagues was palpating the abdomen for a suspected enlarged spleen. It was a fine, sunny August afternoon and he was facing the window overlooking the Euston Road. "What do you make of it, sir?" said the house physician. "Well," he replied, "if to-morrow is anything like to-day, it should be a good day for the grouse."

A high ranking admiral also happened to be one of his patients and, going on his rounds, entered the sick room to find it empty. "Sister," said he in a loud voice, "where is the admiral?" "I think that he must be in the lavatory, sir," she replied. "What *can* he be doing there?" In distinct naval accents a voice boomed from nearby, "Doctor, I give you two guesses."

NOT LOST, BUT GONE BEFORE

It is an invariable practice to ask every prospective patient with an abdominal complaint to bring with him a specimen of his faeces in a container for microscopical examination. There was one young man who turned up without one and, when interrogated, replied, "I am sorry, but it has been stolen!" He explained that having mislaid the carton, he had used a cigarette tin instead. It was a wet day, so he donned his mackintosh and in one of the side pockets reposed the tin. Whilst sitting in the tube he became conscious that the man in the next seat was busy pinching the tin, which he naturally thought contained its legitimate contents. "Didn't you try to stop him?" said I. "No fear; it was too good a joke. The blighter could have it, but I should like to have seen his face when the tin was opened."

THE ROYAL PEARL

A sporting peer who was attached to Buckingham Palace wished to rid himself of a tapeworm acquired from eating underdone beefsteaks.

He stipulated that he would come into the Clinic for treatment for one night on the understanding that he could dine first at White's Club and undergo the treatment without further preparation the next morning. Luckily and somewhat unexpectedly the worm was passed, complete with head.

When he came to say farewell, my assistant addressed him as follows: "My Lord, I have found that you have been eating oysters." "My God, young man, and what has that got to do with you?" replied he.

"It very much has," was the answer, "because I think you might like to see this fine pearl I have found in your lordship's excreta."

Immediately the blustering manner changed as he replied meekly and quietly, "Do you think it would be possible for me to keep this beautiful pearl which I have passed *per vias naturales*, because I will have it made into a tiepin which I shall then ask my royal master to accept?"



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