Original Articles

OBSERVATIONS ON THE NAKED-EYE MANIFESTATIONS OF CHRONIC INFECTIVE PROCESSES IN THE ABDOMEN

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'READ not to contradict and refute; not to believe and take for granted; nor to find talk and discourse; but to weigh and consider.'—BACON.

It is unfortunate, but true, that many of us in India handle medical and surgical material of intense interest, and yet after years of experience have little to show for it, except official annual returns and mental impressions. It is not wholly our fault, for the pressure of work is immense, change of district, and frequent change of assistants necessitated by the exigencies of service tend to retard good intention. I plead guilty of this failure, yet feel constrained to set forth my mental impressions as they have grown strong by frequent repetition. I also gain encouragement by the knowledge that many respected colleagues over a number of years have confirmed my observations and agreed to the probability of the conclusions drawn from them.

Case records and scientific laboratory reports, I have none. To delve into the records of headquarter hospitals no longer in my district is to call upon others to do the work that I should have done myself. However, if the reader will condone this unscientific approach, maybe what follows will not be without interest as it offers a fresh explanation for some common abdominal diseases, and endeavours to explain others that are still obscure. The reader must take, with me, a very general view and regard the patient, his surroundings, habits and customs as an entity, and all that, against a time background. I wish particularly to emphasize the time factor, for I talk of chronic infections and not of the acute infective processes that are sudden and dramatic, although the chronic may become dramatic at any time, and so obscure the chronic origin. It must also be realized that all diseases have a degree of local variance, and I speak of south-west India, particularly of Malabar and Coimbatore.

I will discuss my observations in the order in which they have impressed themselves upon me.

1. The naked-eye appearance of old-standing inflammation of the appendix and of its effect on the intra-abdominal organs.
2. The naked-eye appearance of old-standing inflammation in the lower abdominal cavity other than that of appendicular origin and its apparent results.
3. The origin and scope of these different infective processes.
4. Deduction.

1. The appendix itself varies in appearance from a sclerosed dry strand of tissue to a long thick turgid œdematous structure. It may be of any shape and in any position, but, whatever its condition may be, there will be found, taking origin from it, a degree of lymphangitis which gives rise to a membrane formation. This membrane is highly vascular in the active stages, tough, and in places unyielding in the later stages; it is loosely applied to the surface of all the viscera that it encounters in its upward path; but it forms strong ligamentous attachments between the viscera. New glandular formation and enlargement of normal existing glands will be found according to the degree of inflammatory activity encountered during the laparotomy. It is not usual for the people of south-west India to resort to hospital for discomfort that does not incapacitate them, so the early stages of this membranous formation are not so commonly seen; what is usually seen is the result of years of a chronic inflammation which has resulted in complications.

I have attempted to sketch the general appearance (figure 1). The whole of the right

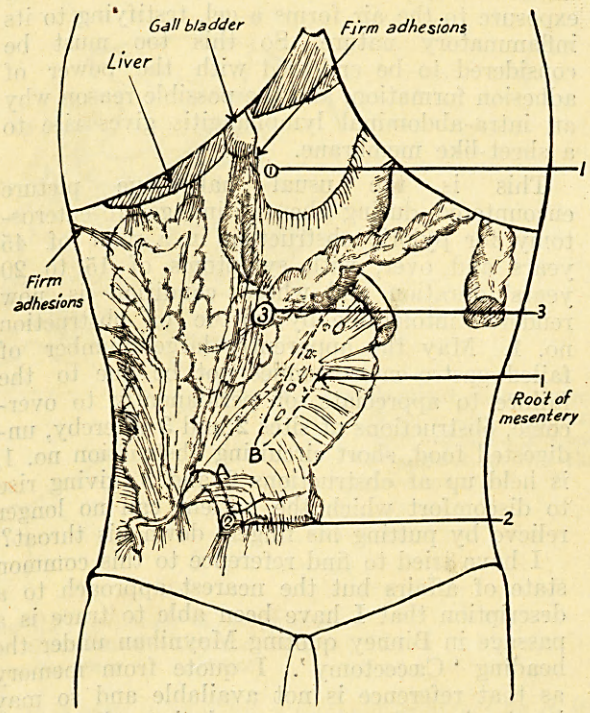

Fig. 1.—Sites marked (1), (2), (3) represent the three main sites of obstruction.

side of the abdomen is invested by a membrane, so tough in places as to appear ligamentous. This although closely applied to the underlying viscera, does not appear to be part of their peritoneal covering, for it can be separated from them to demonstrate its adventitious nature.

The terminal ileum is bound down, often to a degree that renders it unrecognizable until freed by Mayo's scissors. The cæcum ascending colon, hepatic flexure and the first third of the transverse colon are all invested. The pyloric end of the stomach and the first part of the duodenum are firmly bound to the under surface of the liver and gall bladder, and strong bands bind the large gut on the right side of the abdomen to the adjacent para-colic gutter. As this membrane toughens, the pyloro-duodenal junction becomes first fixed, and then kinked. The site of the kink is often the site of a so-called pyloric ulcer. This is obstruction no. 1 (figure 1).

The exaggerated Lane's kink due to the approximation of points A and B in the figure, by sclerosis, in the presence of a bound terminal ileum, is obstruction no. 2 and where the strong bands of membrane cross the junction of the first and second thirds of the transverse colon is obstruction no. 3. It must however be realized that the whole length of gut from site 2 to site 3 is hampered in its activity. It is often turgid and œdematous from lymphatic obstruction, and the exudation escaping from this part, added to the little free fluid usually found in the dependant parts of any abdomen, often amounts to some ounces and this on exposure to the air forms a gel, testifying to its inflammatory nature. So this too must be considered to be endowed with the power of adhesion formation, and the possible reason why an intra-abdominal lymphangitis gives rise to a sheet-like membrane.

This is the usual naked-eye picture encountered during the routine gastro-enterostomy for pyloric obstruction in a man of 45 years and over, with symptoms of 15 to 20 years' duration and whose condition is now rendered intolerable by virtue of obstruction no. 1. May the apparently large number of failed gastro-enterostomies not be due to the failure to appreciate, or permanently to overcome, obstructions at sites 2 and 3 whereby, undigested food, short circuiting obstruction no. 1, is held up at obstructions 2 and 3, giving rise to discomfort which the patient can no longer relieve by putting his fingers down his throat?

I have tried to find reference to this common state of affairs but the nearest approach to a description that I have been able to trace is a passage in Binney quoting Moynihan under the heading 'Cæcectomy'. I quote from memory as that reference is not available and so may be forgiven for not reproducing that lucid fluidity of writing that characterizes Moynihan. He says in effect that 'Sometimes the whole of the right side of the abdomen is found turgid and œdematous, affecting the cæcum, ascending colon and the first half of the transverse colon and for this I can think of no effective treatment other than hemi-colectomy'. I think that at some time Lord Moynihan must have recognized what I describe as commonplace in South India, but what is in England, I presume, much less common. I have been tempted to do an ileo-transverse colostomy in such extreme cases, as such appears reasonable, but the patients are poor surgical risks and dislike operation by stages.

I am satisfied that the kinking described as obstruction no. 1 can be, and is, the site of ulceration. Ulceration behind an obstruction is a habit in the alimentary canal. I am also equally satisfied that the thickening behind the kinked, bound obstruction is taken for pyloric ulcer with sclerosis even when there is no ulceration, for I have followed operated cases to the post-mortem room on several occasions and found no ulcer, but with the relaxation of death, a free passage. This error is more likely to occur with spinal and general anæsthetics than with local, although I hasten to add, it is not necessarily bad surgery to perform gastro-enterostomy if there be obstruction even in the absence of ulceration, so long as obstructions 2 and 3 are appreciated.

Now all that is said above will explain another common condition, namely, chronic gastric dilatation. I had one extreme case whose stomach during *x*-ray examination obscured every organ in the abdomen and pelvis. I kept him in hospital for over 10 months, when he died. Autopsy proved that there was no ulcer, but he did have a very tough membrane formation and evidence of long-standing appendicitis. Another such extreme case treated by gastrostomy and the passage of a tube through the pylorus remained happy until the tube came out and could not be replaced, and so he too died. But between the slight and the extreme, many cases of simple chronic gastric dilatation are seen in this part of India, and while painless because free of ulcer, yet give rise to extreme discomfort which the patient tries to relieve by belching and air-swallowing to enable him to belch more freely. (Perhaps the home practitioner may not realize it, but this is classical conduct in the part of India I speak of.) This too, I believe, is the result of the membranous lymphangitis rising from a chronic appendix, thickened into ligamentous attachment between the pyloro-duodenal junction, and the under surface of the liver.

The name volvulus is a loose one. If what I describe below is volvulus of the small intestine, then 4 out of 5 cases of intestinal obstruction (other than hernial strangulations) are in my experience, on the Western side of South India, volvulus of the small intestine.

The condition appears to be this. The appendix shows evidence of chronic inflammation resulting in sclerosis. The membrane described above is marked, and, as the lymphangitis is no longer active, it is now a contracting scar. The appendix may lie across the terminal ileum, either in front or by artifact, behind (the ileum being bound down and dragged down by scar contraction on top of it). The appendix and

cæcum are drawn up towards the root of the mesentery which, in its lower part, is puckered and contracted, more below near the appendix than above which is the root of jejunum. There is a degree of small intestinal obstruction in the lower reaches of the ileum at the site marked obstruction no. 2 in figure 1, while above, the gut is hypertrophied and dilated. As a result there is a longer mesentery to the jejunum than to the ileum, the latter being scarred and puckered by its proximity to the appendix and old-standing lymphangitis at its base. Some day after a big meal, the weight of the upper gut bears it down and to the right, and the base of its mesentery lying over the scarred, fixed, lower mesentery *cum* appendix brings the jejunum towards, and even into, the pelvis. In this way the mesentery makes a half-turn anti-clock-wise that is clear to the eye at operation and is corrected only by complete eventration, when, the fixation of the cæcum *cum* appendix *cum* lower ileum having been relieved, the bowel resumes its normal position, the contents pass on and the patient (temporarily at all events) is cured (figure 2).

A highly situated appendix is at times described as due to failure in descent of the cæcum and ascending colon. This developmental failure cannot be considered a common occurrence as it is not commonly found in abdomens innocent of the pathology I shall shortly attempt to describe.

The appendix is, at times, plastered down on to the ascending colon, and so covered over by this lymphangitic membrane that it looks like one of the longitudinal striæ, and is only demonstrated to be the appendix by the surgeon dissecting it out.

The tip of the appendix may be right up under the liver and attached to it by a ligamentous thickening of this membrane. It may even be so attached to the gall bladder, that, by contracture of this membrane and the adhesion caused by it, the whole of the large gut in the right side of the abdomen is drawn up, so that to effect visualization and removal of the appendix the incision must be extended upwards to the maximum, and woe to the surgeon who has started work through the grid incision.

Once this state of affairs is acquired it is very doubtful if a patient is materially benefited by removal of an appendix that has itself long since prohibited the ingress of any bowel content, for on slitting it up it is found to be thick-walled, tough, and innocent of any fæcal content or of naked-eye evidence of active inflammation. But it has materially interfered with the free movement of the right half of the large intestine, duodenum and pylorus by virtue of the membranous formation resulting from years of chronic inflammation.

Appendicular gastralgia conveys the idea of a nervous reflex. I do not refer to this form of upper abdominal discomfort secondary to appendicitis, but to gastric or upper abdominal discomfort amounting to pain in the absence of obviously active appendicitis. One knows that a patient treated gently under a local anæsthetic does not greatly complain, but directly an organ is pulled upon he immediately resists or even cries out. In the condition described above with the pyloro-duodenal junction fixed to the liver and gall bladder, the colon fixed to the liver, and hypertrophy of these organs secondary to their hard work in overcoming obstruction, it is easy to imagine a stomach contracting in its length as it goes into peristalsis so pulling on the gall bladder, wave after wave and pull after pull. Naturally, the patient resents this pulling on an organ. So I suggest that constant upper abdominal discomfort after food may be due to the stomach or, alternatively, the colon constantly pulling, as it were, on the gall bladder door bell. It is not unduly difficult, but laborious, to work through these tough

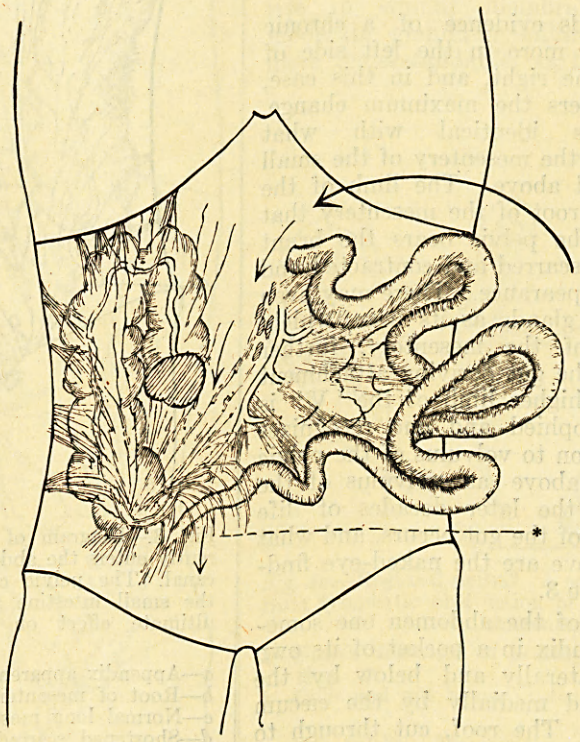

Fig. 2.—Diagram showing the shortening of mesentery of the terminal ileum and the relatively long mesentery to the jejunum predisposing to volvulus.

adhesions, but having done so how are they to be prevented from reforming? Hence, after removing a 'chronic appendix' for a gastralgia (there being no evidence of pathological lesion of the stomach) the patient complains that his old pain is still present, and so it will be, for all time, unless removal of the cause permits of stretching and elongation of the adventitious attachments this possibility I very much doubt, because they are seen so many years after, on re-opening what is diagnosed to be a failed gastro-enterostomy. In explanation of this discrepant diagnosis I advance my second point, which is essentially an attempt to show the similarity between the symptoms usually associated with the macroscopic appearances of abdominal pathology of appendicular origin, and similar appearances which, I suggest, arise from causes other than chronic appendicitis, so :—

2. When what is described above apparently affects both sides of the abdomen, where does the inflammation come from and what is the picture?

One sometimes finds evidence of a chronic inflammatory process more in the left side of the abdomen than the right, and in this case, the pelvic colon suffers the maximum change, and the change is identical with what is seen in the root of the mesentery of the small intestine as described above. The limb of the inverted 'V'-shaped root of the mesentery that extends lower into the pelvis bears the brunt of the change. It is scarred and contracted and has a watered-silk appearance. There may even be a bunch of hard glands as its base, which, invading the root of the mesentery, further shorten it. Above, the gut deriving attachment from the other and higher limb of the 'V' is dilated and hypertrophied and one recognizes the same predisposition to volvulus of the pelvic colon as described above as volvulus of the small intestine. In the later decades of life volvulus of this part of the gut occurs, and what I have described above are the naked-eye findings sketched in figure 3.

On the right side of the abdomen one sometimes finds the appendix in a pocket of its own which is bounded laterally and below by the para-colic gutter and medially by the cæcum and terminal ileum. The roof, cut through to expose it, is formed by a false membrane often of surprising thickness. It is as if the appendix, squeezed out of the way, took no part in the proceedings, and, as if the cæcum contacted the para-colic gutter above it, the lymphangitic membrane jumped the potential gap, missed the appendix, and continued on its upward path towards the pre-aortic glands and upper and side abdomen.

Now, if one holds up the anterior abdominal wall and throws in a light, not infrequently one will see strands of vascular adventitious membrane arising from the internal inguinal ring on one or both sides. At times one even finds that the omentum is adherent here, and when it is peeled off, a broken-down gland wedged in the internal inguinal ring, is easily delivered. The appearance is reminiscent of the fundus of the eye with all vessels radiating from the disc.

From one or both internal inguinal rings the same type of intra-abdominal naked-eye picture as described for the 'chronic appendix' is reproduced, and the same upper abdominal pathology faithfully copied, with fixation of the pylorus,

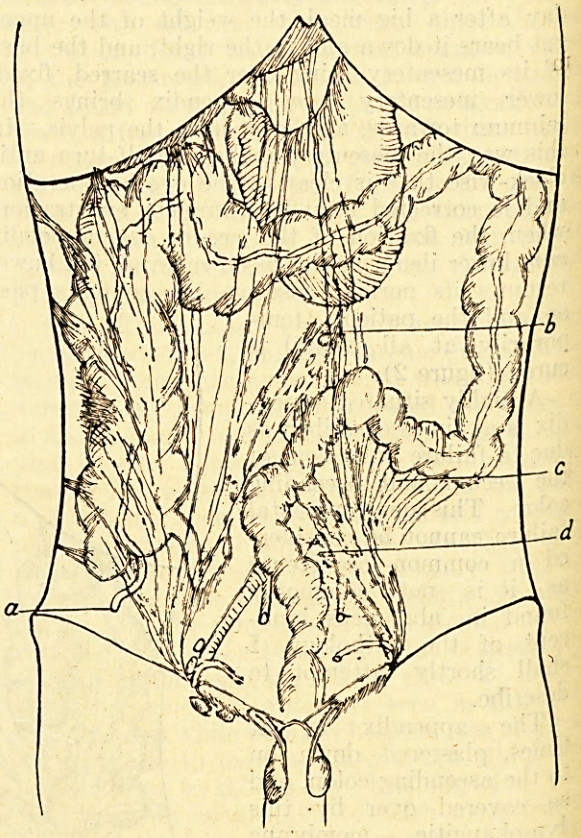

Fig. 3.—Diagram of appearance when infection apparently enters the abdominal cavity through the inguinal canal. The pelvic colon suffers in the same way as the small intestine shown in figure 2, and the same ultimate effect on the whole abdominal cavity is apparent.

a—Appendix apparently normal beneath the membrane.
b—Root of mesentery.
c—Normal long mesentery.
d—Shortened scarred mesentery.

and the picture is as represented in figure 3 with all the signs and symptoms of chronic appendicitis and pyloro-duodenal ulcer or obstruction. That the infection originates at the internal inguinal ring is further supported by the common association of a mass of glands in the inguinal and even femoral regions, or for that matter the tell-tale scar, and also clinically one knows that acute gonorrhœa can simulate intra-abdominal disturbance, temporarily at least.

Now chronic infection arising from this site sometimes shows a state of affairs not seen in

that arising purely from the appendix. It may come from either or both sides. I refer to masses of glands which follow along the spermatic and iliac vessels to the pre-aortic glands and which seem to have a predilection for causing a massive adenopathy in part or all of the root of the mesentery. These glands, the size of pigeon eggs at the root of the mesentery, get smaller as they reach the free margin of gut and, blocking lymph flow render a section of gut œdematous and wooden, so much so that the state of affairs may go on to ulceration of the gut although this is comparatively rare, except in the cæcum. In other words the condition is indistinguishable by the naked eye from the various descriptions of regional ileitis or Crohn's disease, and this may occur anywhere from the pylorus to the cæcum, and again in the lower sigmoid and rectum, in fact in all the sites said to be commonly attacked by Crohn's disease (figures 4 and 4*a*).

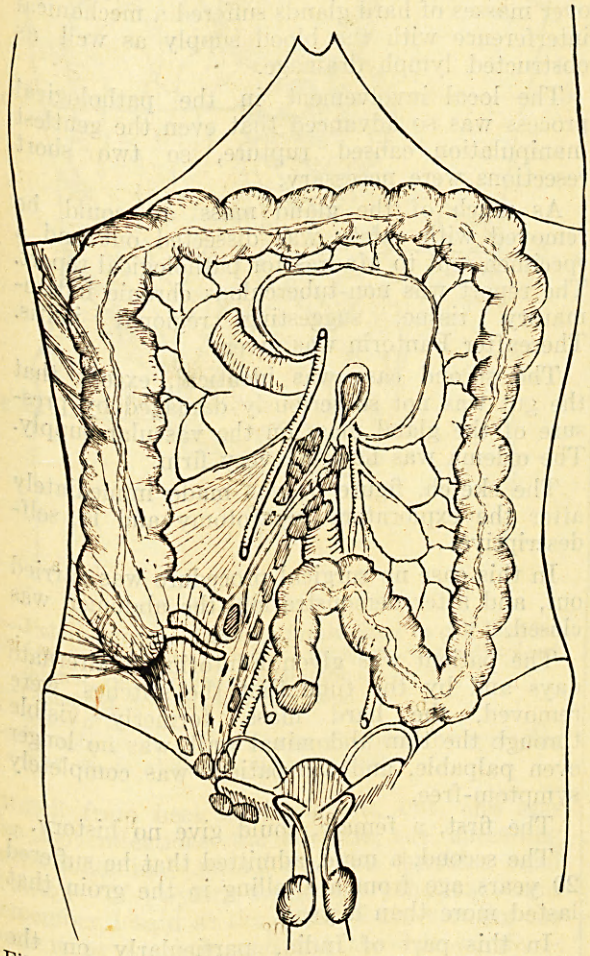

Fig. 4.—Mass of glands in part or whole of the root of mesentery giving rise to what looks like Crohn's disease.

In the cæcum the appearance is characteristic. The clinical history and diagnosis will certainly be tuberculous disease of the cæcum, and laparotomy will show a thick vascular membrane investing the terminal ileum, cæcum and appendix, the whole, woody, hard, and intensely œdematous. It almost 'breaks' away from the cellular tissue of the posterior abdominal wall, and when slit up afterwards shows an inch or so of gut with a wall like ulcerated cartilage. Sent to the pathologist for cancer or tubercle, the result is neither. It looks, in fact, like the excised rectum of a non-malignant stricture of that organ. The resistance of the patient, unlike that in cancer and tubercle, is so good that death is rare, so further verification by post mortem is rarely possible, even if local custom permitted it.

When this state of affairs is found, the membranous formation is dense, and if the examining hand puts tension on the ascending and first third of the transverse colon and pylorus, dense thickenings of the membrane stand out as in figure 1. Any glands and tissue excised for biopsy do not disclose the true pathology, but do exclude active tuberculosis and cancer.

More rarely, frank tuberculous adenitis from a tuberculous lesion of the lower limb will give rise to similar membranous formation and moderate glandular enlargement, but this is more understandable, as the primary disease is

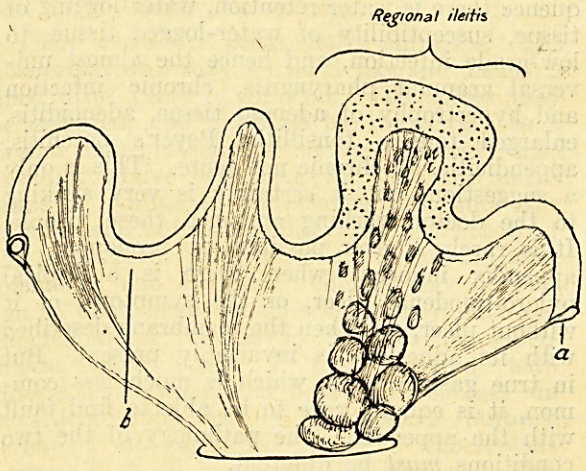

Fig. 4*a*.—Regional ileitis? A segment of intense œdema from lymphatic obstruction of *part* of the base of the mesentery.

a—Simple dilatation of healthy gut above the obstruction.

b—Collapsed gut below obstruction.

recognizable. Mycetoma may show glands in the femoral and inguinal region which on section show characteristic discoloured areas, and so these give rise to no surprise. But my point is this :—Whatever the chronic infective process at work, the naked-eye appearance is similar, in general, namely, a more or less developed membrane formation investing the viscera in its path from the chronic infective focus, upwards, embarassment of movement, lymph stasis and fixation of the viscera, especially the colon, to the side wall of the abdomen. Also fixation of the pylorus and first part of the duodenum to the under surface of the liver and gall bladder, kinking at the site of junction of these latter,

and all the symptoms of chronic appendicitis and pyloric ulcer with obstruction.

Moreover, and in particular, certain specific chronic infective processes may give rise, not only to the above, but to a variety of complications varying from Crohn's disease, to hydronephrosis from obstruction to the ureters where they enter the bladder.

3. *The origin of these different infective processes.* From the frequency with which people in this part of India suffer from chronic appendicitis, I suggest the following, more evident on the west coast where the disease is commonest. No one article of diet or vitamin can be blamed, nor hookworms nor water, but, I do think, adenitis can be blamed.

Diet in that part of India is almost devoid of protein. It is true many claim to be meat eaters, but on questioning them it is found that they get very little meat, or fish either. They get even less fish in the monsoon except a local dried variety, but few, however, will eat that.

Might it not be that the simple absence of protein from the diet means the absence of sufficient urea, that natural diuretic. In consequence there is water retention, water-logging of tissue, susceptibility of water-logged tissue to low-grade infection, and hence the almost universal granular pharyngitis, chronic infection and hypertrophy of adenoid tissue, adenoiditis, enlarged tonsils, tonsillitis, Peyer's patchitis, appendicitis, all chronic not acute. This is only a suggestion, but it certainly is very striking to the doctor working amongst these people. It is rarely if ever possible to pronounce an appendix innocent when there is a typical pyloro-duodenal ulcer, or the symptoms of it without ulcer, and then the membrane described with its adhesions is invariably present. But in true gastric ulcer, which is much less common, it is equally rare to be able to find fault with the appendix. The pathology of the two conditions *must* be different.

Can this membrane and glandular hypertrophy be attributed to chronic amœbiasis? I doubt it, for the splenic flexure escapes so consistently, and why only the right half of the abdomen? Chronic inflammation of the appendix must bear the blame for all the diseases above described as attributable to it, but not for the others.

What is the origin of the infection described as entering the abdomen by the internal inguinal ring and possibly the femoral canal, giving rise to anything attributable to chronic appendicitis, and, in addition, to much more, for instance, 'regional ileitis', tuberculosis of the cæcum that is not tuberculous, scarring of the mesentery and intestinal obstruction.

Climatic bubo heads the list, the tell-tale scar or history of bubo is the rule rather than the exception, although the history is not easy to obtain. A climatic bubo that failed to break down is soon forgotten, especially if it occurred 30 years previously.

Occasionally on opening an abdomen for chronic obstruction, one finds a mass of glands occupying part or whole of the root of the mesentery. The segment of hard œdematous bowel overlying the mass may be so great as to present a danger to life if removal be attempted, yet this mass is apparently cleared by treatment with Fouadin or Fantorin, and the patient is relieved of his symptoms. This seems further clinical evidence incriminating climatic bubo.

As I write this article, I have been sufficiently fortunate to have 2 such cases admitted into hospital. These cases illustrate my point. Both were classical of high intestinal obstruction.

The first came under the care of my colleague Dr. Vadamalayan. Early operation was clearly necessary. On opening the abdomen two short lengths of jejunum were seen to be involved in an intense œdema, and the gut being stretched over masses of hard glands suffered a mechanical interference with the blood supply as well as obstructed lymph drainage.

The local involvement in the pathological process was so advanced that even the gentlest manipulation caused rupture, so two short resections were necessary.

As much of the gland mass as could be removed with safety was dissected out and a specimen sent to Madras for pathological report. The report was non-tuberculous chronic inflammatory tissue, suggesting regional ileitis. Thereafter Fantorin was given.

The second case was identical, except that the gut was not so seriously damaged by pressure of the gland mass on the vascular supply. The œdema was however very firm.

The sketch, figure 5, was made immediately after the exploratory laparotomy and is self-descriptive.

In this case no surgical procedure was carried out, and after demonstration, the abdomen was closed.

The patient was given Fantorin on alternate days and by the time that the stitches were removed, the hard mass, formerly visible through the thin abdominal wall, was no longer even palpable, and the patient was completely symptom-free.

The first, a female, could give no history.

The second, a male, admitted that he suffered 20 years ago from a swelling in the groin that lasted more than a year.

In this part of India, particularly on the coast, lymphogranuloma seems almost endemic. One meets with it in the neck, axilla, tonsil, floor of the mouth, bladder, lower ureters, rectum, penis, inguinal glands, in fact one is not surprised at any fresh manifestation. Many of these extraneous lesions are variously diagnosed as tuberculosis, cancer, or gummata and are treated for years with the usual remedies,

but are cured in as many weeks by Fouadin. If this external manifestations of lympho-granuloma be so varied, why should lympho-granuloma be excluded as a possible cause of intra-abdominal manifestation ?

In paddy-growing areas men get cracked heels and chronic lymphadenitis from tuberculosis, mycetoma, and many unknown infections, all through their feet. May not such cases of chronic lymphadenitis of femoral and later also of inguinal glands, also be contributive to the prevalence of pyloro-duodenal ulcer and of all the lesions above described? It seems a long

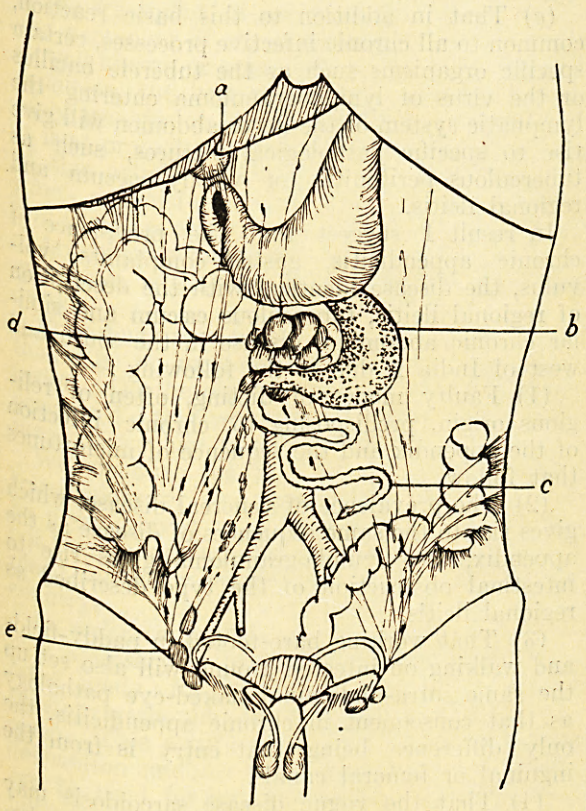

Fig. 5.

a—Pyloro-duodenal junction firmly fixed to gall bladder.
b—Duodenum and upper jejunum hypertrophied, dilated and intensely œdematous.
c—Collapsed gut below obstruction.
d—Mass of glands.
e—Lymphadenitis giving rise to membrane by spreading over whole abdomen on both sides.

stretch from heel to duodenum, but, one sees the foot infections, the adenitis, the spreading intra-abdominal pathology, the hitching and fixation of pyloro-duodenal junction, and remember I said at the outset I am viewing the patient as a whole and not through a micro-scope.

This statement prompts me to make a further suggestion. If masses of glands be found in the abdomen, and if macroscopically at least, they appear to derive their origin from the internal abdominal or femoral ring, why should not the virus of infection extend to the media-stinum and answer the tuberculosis specialist's query of what is that gland mass in the radio-gram which is so unlike pulmonary tuberculosis?

One often sees a mediastinum showing massive 'glands' but no evidence of tuberculosis any-where in the body, and no fever nor toxic quickening of the pulse. If the inner third of the lung field be covered, the outer two-thirds are seen to be clear. What then has drained into the mediastinal glands and from where? Certainly not from the lung tissue. There may be no evidence that the infective process travelled down from the deep cervical lymph glands, but, knowing that similar gland masses are stimulated to growth in the abdominal cavity, why not suggest that the same pathology may repeat itself in the mediastinum? This observation may escape the chest specialist for he seldom has the chance of seeing inside the abdomen. Neither does the abdominal surgeon think in terms of mediastinal glands and venereal diseases.

One occasionally does an exploratory lapa-rotomy in a case of ascites from a medical ward if the patient be young, thinking that it is tuberculous and that the release of fluid and the inclusion of air will both establish diagnosis and be of therapeutic value.

These cases are occasionally surprises. The abdominal wall may be lined like the pleura is lined after years of effusion. The spleen may be enlarged, and attached by its margin to a similar lining membrane, quite different from anything above described, for here the mem-brane is hyaline in appearance and lines the parietes and not the mobile viscera. The liver also taking its part, is enlarged, or if later, contracted and cirrhotic. Glands may be pre-sent in abundance but on section they have not the naked-eye diagnostic features of tuber-culosis. Such cases I have treated with Patterson's buttons, and because of success am an ardent supporter of Patterson's buttons. Perhaps I am not dealing with a tuberculous peritonitis at all but with a sarcoidosis. This introduces a fresh name.

Recently a highly-educated patient on full duty up to the day of admission came to the hospital with all the classical signs of high intestinal obstruction. He did not look as if he had suffered a long illness, his appearance was against a diagnosis of tuberculosis or cancer, but examination showed a protuberant abdomen and the presence of free fluid. Through this free fluid solid masses could everywhere be 'ballotted', the condition was considered hope-less but to satisfy conscience, the abdomen was opened to see if anything could be done.

Free fluid, innocent of any blood staining, escaped and the abdominal contents were as firmly fixed as if set in plaster of Paris. It did not look like tuberculosis of the abdomen, and if cancer, how did the patient survive so long? Exploration was impossible as everything was fixed. A small mass of this tissue was

broken off—not cut—it did not bleed. It proved to be a piece of omentum. Next day the patient died. Unfortunately Indian ideas and customs precluded a post mortem. So further investigation could not be made. The pathologist's report from General Hospital, Madras, was 'sarcoids', not cancer nor tuberculosis.

Now I am sure that pathological report was right. Very little attention has been paid to this disease, but the similarity between the manifestation of sarcoidosis as described, and the various manifestations of lymphogranuloma are very striking to the clinical observer. Of the microscopic differences, I know nothing, but for the benefit of those who see less of this condition than, I believe, we in the south-west India see, I will make a brief description.

Said to have been described by Jonathan Hutchinson in 1869, re-described by Besnier in 1899, more concisely described and in more detail by Boeck in 1899, ocular manifestations were described by Harfordt in 1909. Cutaneous manifestation reported from 1915 onwards, and since, practically every organ of the body has been found liable to suffer.

In an article in the *American Journal of Roentgenology* of April 1941 by Donald S. King, sarcoidosis of the spleen is described in which the spleen weighed 52 oz. and in the same article Teuben Schutz, pathologist, describes sarcoids in the lung, lymph nodes, and liver.

Sarcoidosis is a disseminated disease and the lesions are granulomatous, to all naked-eye appearance like lymphogranuloma in that there is gross lymphatic alteration and an œdema of wooden hardness.

The common occurrence of sarcoid-like diseases in this part of India in association with regional ileitis which looks like local sarcoids or alternatively lymphogranuloma, and the extreme frequency and variety of manifestations in which lymphogranuloma is met with is so striking, that it should be voiced even if only to stimulate the pathologist to classify these diseases and put them in their places. Clinically they appear to be one and the same disease.

Now and then a doctor hits on a new cure for cirrhosis of the liver. I cannot help thinking that he may have had the luck to run up against a small series of sarcoidoses, for this strange disease seems capable of a degree of resolution.

But since I am in my third section of this article dealing with origin of the infective foci, I put it as possible—indeed from a clinical point of view probable—that all forms of regional ileitis, woody cæcum, ano-rectal syndrome and so forth, together with sarcoids intra-abdominal and thoracic adenopathy associated therewith are none other than manifestations of the virus of lymphogranuloma inguinale (*alias* climatic bubo) mostly acquired but not infrequently congenital.

4. *Deduction.*

The reader will have appreciated the general trend of ideas :—

(*a*) That in the abdomen, any low-grade infection over a period of time will give rise to permanent mechanical interference with the intra-abdominal organs, resulting in anything from pyloro-duodenal ulcer, to volvulus, and should therefore be eradicated early.

(*b*) That the same basic picture is reproduced whatever the exciting cause, whether the origin be in the appendix, or inguinal, or femoral canals.

(*c*) That in addition to this basic reaction, common to all chronic infective processes, certain specific organisms such as the tubercle bacillus or the virus of lymphogranuloma entering the lymphatic system of the lower abdomen will give rise to specific pathological pictures, such as tuberculous peritonitis, or woody cæcum and regional ileitis.

In result I suggest that the prevalence of chronic appendicitis, gastric complaints, volvulus, the disease identical with the description of regional ileitis, tuberculous cæcum and similar chronic abdominal disease in the south and west of India is due to the following :—

(1) Faulty modes of dieting, often of religious origin, predisposing to chronic infection of the appendix and the sequence of misfortunes that follow.

(2) The prevalence of venereal disease, which gives rise to the same sequences of disease as the appendix, also lymphogranuloma gives rise to intestinal obstruction of the type described as regional ileitis.

(3) That working bare-footed in paddy-fields and walking on infected ground will also set up the same intra-abdominal naked-eye pathology as that consequent in chronic appendicitis, the only difference being that entry is from the inguinal or femoral canals.

(4) That the vague disease sarcoidosis may possibly also be a late manifestation of lymphogranuloma.

It is therefore necessary that each patient be viewed in association with his habits and environment, the result of chronic low-grade infection *plus* time be given due consideration, and the possible value of Fouadin or its substitutes be kept in mind.

I hope in due course to lay some pathological evidence on the table to support these clinical observations, but the life of a district officer is too full and varied for that attention to detail so necessary before arrival at any conclusion. In the rush of life records are apt to be few, while mental impressions are apt to be fallacious.

However, others may be in a position to confirm, refute or follow up some of the above suggestions, and once the extreme prevalence of lymphogranuloma becomes appreciated, perhaps some of the Indian manufacturing

(*Concluded on opposite page*)

A TRANSFUSION SET FOR USE IN HOSPITALS OR IN THE FIELD

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BLOOD, plasma and serum transfusions are now universally accepted as being an essential part in the treatment of hæmorrhage, shock, burns and a number of chronic diseases. What is not so well known or appreciated is that, in an acute condition, the best results are obtained by the early transfusion of the appropriate substance. It is at present impossible, and beyond the scope of this article to lay down the exact time at which a transfusion should be given. Allowing that there are other essential and routine methods of treatment which must be employed, our own experience is that we are constantly regretting delay.

Providing reasonable care is taken, there should be no mortality resulting from a transfusion. On the other hand, we have seen many patients die that would in our opinion have been saved if we had not delayed.

We visualize the time when in certain cases transfusions of the appropriate substance will be given almost as a routine and to prevent, not treat, shock and other conditions, and would plead for their wider and earlier use in this country.

In order to provide for prompt transfusions, it is necessary to have a complete set of apparatus ready for immediate use. Transfusion substances should also be ready or within easy reach. This article is concerned only with the description and use of a transfusion set which can be used for any substance, and fulfils the following conditions :—

(1) Portability.
(2) To be ready for immediate use.
(3) Be capable of being used under any conditions.
(4) Conform to the special conditions required by the Army on active service.
(5) To provide for transfusions, using one or more bottles of one or different solutions.

In the set described, in order to provide for Army requirements the number of spares may be considered excessive for a civil hospital. We would, however, point out that our experience shows there is no harm in having an adequate supply of spares, and it is imperative that the set should be looked after by a responsible person and kept under lock and key until required, otherwise various items of the equipment are certain to disappear.

The set is contained in tow boxes, an upper metal one designed as a water sterilizer, and a lower wooden one, containing items not requiring sterilization, and spares.

Contents of upper box (metal)

This box contains all the items required for a single or double bottle transfusion.

(1) Metal transfusion can fitted with lid (for use as a bowl, or small sterilizer), filter funnel, and hanging hooks. The can is for use in case the simple method of transfusion is desired, or in the event of blood clotting in the bottle.

(2) Bottle fitted with blood-withdrawing cork and tubing, and containing blood-withdrawing component (rubber tubing 10 inches with glass window, metal adaptor and intravenous needle). A piece of thread is tied to one end of the component, the other end hangs outside the bottle, providing for easy withdrawal. The bottle, wrapped in a towel with the ends well projecting to prevent trauma to the glass, is placed in the metal transfusion can and covered by the lid. If space is available, the filter funnel is placed inside the can, otherwise elsewhere in the box.

(3) Bottle fitted with blood-giving cork and tubing and metal hanging cage wrapped in a towel.

(4) Blood-giving component, single bottle (figure 1) consisting of—

(*a*) Drop regulator;
(*b*) Rubber tubing 3/16 inch and two glass connections;
(*c*) Intravenous needle and metal adaptor;
(*d*) Pinch cocks 2;
(*e*) Monel metal gauze filter.

(5) Double bottle component (figure 2) consisting of—

(*a*) Metal Y piece;
(*b*) Rubber tubing, 2 pieces;
(*c*) Pinch cock, one.

Both components are wrapped in a towel.

(6) Glass filter—for use with wool or beads. A spare drop regulator is provided for this purpose and, if required, is inserted above the drop regulator in use.

(7) Set of instruments in bag consisting of—

(*a*) Sharp pointed scissors 5 inches ..	1
(*b*) Dissecting forceps	1
(*c*) Mosquito forceps	2
(*d*) Aneurysm needle	1
(*e*) Blunt hooks	2
(*f*) Scalpel 1½ inches	1
(*g*) Infusion cannula	1
(*h*) Half circle cutting needles no. 12 ..	2
(*i*) Linen thread—thin, or any suture material desired, feet	20

(8) Blood matching set in bag consisting of—

(*a*) Microscope slides	4
(*b*) Test-tubes 2 × ½ inch	2
(*c*) Hypodermic syringe, 2 c.cm.	1
(*d*) Hypodermic needles	2

Contents of lower box (wooden)

(Containing items permanently sterile or not requiring sterilization)

1. Sodium citrate, 4 per cent, ampoules of 80 c.cm.	8
2. Sodium citrate tablets, gm. 1, packets of 12	12
3. Bandages, loose-wove uncompressed, 2½ inches × 6 yards	4
4. Wool, 2-oz. packets	2
5. Gauze—surgical, loose-wove—unmedicated, 25 inches wide × 3 yards, packet ..	1
6. Ampoules procain, 2 per cent, 1 c.cm. ..	6
7. Knife, glass cutting	1
8. Tape—narrow, yards	18

(Continued from previous page)

chemists will endeavour to produce a cheap substitute for Fouadin or Fantorin in response to demand, for these two drugs are of immense value in this part of India, but expensive and difficult to obtain, especially in time of war.