post-mortem examination. In either case death is caused by unrelieved intestinal obstruction, and the same occurrence is not infrequently noticed after operations for internal hernia or obstruction by intestinal adhesions in which the mechanical cause of the obstruction has been removed without relieving the obstruction itself.

The explanation of this fact is by no means clear. It is sometimes described as "paralytic ileus"; but however applicable this description may be to some cases of distension with obstruction, following severe abdominal operations, not involving the intestines themselves, it certainly does not fit the cases I have described. So far from being paralysed, the bowels above the point at which strangulation was relieved frequently continue in a state of active visible or palpable peristalsis. It is obvious that the strangulated coil of bowel or the adjacent portions which have been united after resection of the strangulated part in some way oppose an insuperable obstacle to the passage of the intestinal contents, even when propelled by unusually powerful peristalsis, just as a dead or functionally inert segment of bowel caused by mesenteric embolism will cause intestinal obstruction without any mechanical stoppage of its lumen. Why the interposition of a passive but easily distensible segment in the course of the intestinal tube should cause obstruction within the abdomen, and not do so when an equally passive tube of thin rubber is tied into the bowel and brought outside the body, is difficult to explain, but it is certainly a fact. In order to avoid this danger, without sacrificing the great advantages of an immediate restoration of the continuity of the bowel after enterectomy, I have on several occasions successfully resorted to a temporary drainage by means of a small rubber tube sewn into the bowel some inches above the line of section, and brought out through the abdominal wound. For this purpose, in the small intestine, an ordinary rubber catheter of No. 12 to 14 English scale suffices. It is passed through a small incision in the bowel, and fixed by means of a suture which transfixes both the bowel wall and the catheter itself. A second sero-muscular purse-string suture is then inserted in such a manner as to invaginate the first incision, and the bowel is then placed within the abdomen, taking care to avoid kinking.

The tube becomes loose in a few days, and leaves a small fistula which closes without difficulty as soon as the natural function of the united intestine is resumed. I regard this method as far superior to that of merely resecting the gangrenous or damaged loop of bowel and placing drain tubes in each lumen, since this method, even if the patient recovers, demands a difficult and dangerous operation for the subsequent restoration of the continuity of the bowel.

In conclusion, I should like to emphasize once more the following points: Large numbers of patients die every year from intestinal obstruction due entirely to mechanical causes which are remediable by surgical operation and by no other means. The majority of these fatal cases are not operated on until a late stage in the disease, and most of them are in the first instance treated by drugs or applications, internal or external, which cannot possibly give relief. The difficulty of diagnosing the existence of mechanical obstruction is increased by the popular confusion between constipation—that is, temporary suspension of the motor functions of the bowels—and real obstruction—that is, mechanical hindrance at a definite point. The consequences of the latter condition are so serious and so urgent that the question of its existence should be carefully considered in every case of abdominal illness in which pain is associated with vomiting, distension, and absence of recent bowel evacuation. If the possibility of obstruction cannot be excluded after a short period of observation, surgical assistance should be obtained as soon as possible. Obstruction of the bowels within the abdomen is just as much a surgical disease as strangulated hernia. The difficulty of the diagnosis in the former merely adds to its dangers, the greatest of which is delay.

A BILL is being promoted in Parliament which provides for the construction of a crematorium for South London in the parish of Lambeth.

THE late Surgeon-Lieutenant-Colonel H. W. Kiallmark, formerly of the Royal Bucks Hussars, left estate valued at £17,156. He left £1,350 to charitable institutions.

A CASE CONTRIBUTING TO THE STUDY OF FEVER WITHOUT OTHER PHYSICAL SIGNS.

By H. GWYNNE LAWRENCE, M.B.LOND.

THE subject of the following notes was under observation for ten months. For the first six months he presented very little in the way of physical signs, although throughout this period he was the subject of marked fever—at first intermittent, with definite periods of apyrexia, but latterly remittent, and finally continuous.

The patient, aged 62 years, fifteen years ago resided in India for five years without any detriment. Two years ago he had an attack of tachycardia which passed off in a short period, and for three years he had suffered from an annual febrile attack believed to be influenza. No evidence was obtainable of alcoholic or dietetic excesses. The only other important detail upon which he laid much stress was the fact that all his life any form of aperient upset him profoundly, as did also enemata. For the first six months symptoms other than could be attributed to the fever were completely absent. During the last four clinical signs of cirrhosis of the liver appeared.

In the earliest days a bad night would be followed by a tongue thickly coated with a brown moist fur, the breath would be "heavy," the skin dry and hot, the face slightly yellow tinted, and occasionally some flatulent distension of the colon could be recognized.

The temperature rose at 6 p.m. daily to 102° or rather higher, and was at the normal line again at 6 a.m. This continued for a period of from ten to fourteen days, when a similar period of apyrexia occurred, and the patient became comfortable, and the tongue cleaned to a great extent, but not absolutely.

Conspicuous features at the height of the fever were the facts that the pulse was little disturbed, and rarely exceeded 62 in frequency, and that the respirations remained at 20 to 24. The skin was hot and dry, the bowels acted well, or only required gentle stimulation, and no after symptoms, for example, headache, etc., were experienced.

Blood examinations showed marked leucopenia. The red cells were practically normal in amount, with some deterioration in haemoglobin; the white cells were 2,300 per c.cm.

Agglutination tests to enteric and Malta fever were negative, as was also Wassermann's reaction. Blood cultures proved negative on all occasions.

Treatment was directed against pyorrhoea alveolaris, which was present, but only temporary improvement of this condition occurred, and the course of the main disease was absolutely uninfluenced. This state continued for six months. The liver became gradually more and more displaced upwards by the abdominal distension, the upper margin eventually reaching the fourth space in the vertical nipple line. During this period occasionally a slimy mucous stool, offensive in character, was passed, and an occasional attack, of short duration, of tachycardia occurred.

In the apyrexial period the appetite was good, the general nutrition improved, and there was a complete absence of symptoms.

At the sixth month ascites began to develop, and the abdominal distension was a continuous feature. The base of the right lung showed signs of compression, the skin became yellowish in tint, and showed loss of elasticity. The evidence at this period, from a clinical point of view, suggested the cardinal signs of cirrhosis of the liver—jaundice, ascites, and a liver apparently (by x-ray examination) of even contour, but rather smaller than normal. The spleen could be felt at the rib margin, and some discomfort was experienced over the splenic area. At this stage the blood cultures again proved negative, and the leucocyte count amounted to 1,716 per c.cm. The differential count was:

Polymorphonuclear ...	...	...	41 per cent.
Mononuclear ...	...	...	25 "
Lymphocytes ...	...	...	32.62 "
Eosinophiles ...	...	...	1.38 "
Red cells (per c.cm.) ...	...	...	3,800,000

For a short interval acute pain was experienced at the

point of the left shoulder, in the region supplied by the fourth cervical nerve.

For the last two months the temperature was constantly raised, generally reaching 103° at 6 p.m. The pulse was little affected as regards frequency until cardiac weakness appeared in the last few weeks.

Faecal examinations showed an unusual number of microbic elements and an unusual type of coliform bacillus,

In the early period of disease.

In later period when symptoms of cirrhosis of the liver existed.

but the food elements were digested. In the later periods fats were not digested and bile was deficient. Urobilin was in excess in the urine.

Intestinal antiseptics—for example, salol, paraffin, etc.—were administered from the start, but produced no benefit.

Necropsy.

A *post-mortem* examination by Dr. Spilsbury showed the following condition:

External.

There was some oedema of the lower extremities and scrotum. The abdomen was rather distended.

Internal.

Heart. All cavities dilated; muscle showed microscopically marked brown atrophy. Lungs: Partly compressed by diaphragm; they also showed passive congestion and emphysema. The peritoneum contained 3 to 5 pints of clear ascitic fluid. Liver: of about normal size, but increased weight. There was some surface irregularity, but little thickening of capsule. On section, the organ was very firm and dry. The cut surface had a mottled appearance, reddish and grey areas alternating. The organ did not appear greasy. No fibrous network could be detected with certainty. The gall bladder was normal, and contained pale and watery bile; the bile ducts were normal. Microscopically the organ was traversed by numerous broad, and in places interlacing, areas in which the liver cells were undergoing atrophy, and many had disappeared, the wide spaces between the cells being occupied by greatly dilated blood capillaries, which are not easily seen, owing to *post-mortem* changes.

In the meshes of this interlacing network were islands of liver cells which exhibited marked hypertrophic changes, the cells being of large size and staining deeply, and some having two nuclei apiece. These changes were not very regularly disposed, but the atrophic areas appeared to be chiefly in the central, the hypertrophic chiefly in the peripheral, zones of the lobules. All the liver cells contained some excess of pigment. Fatty infiltration was exhibited by some of the hypertrophied cells, but the atrophied cells contained no fat. There was a little recent fibrous thickening of the capsule, but none of the portal areas, except around some of the larger bile ducts; there was, however, some thickening of the

walls of the hepatic vein tributaries. There were no myxomatous degenerations in the organ, practically no round-celled infiltration of it, and nothing to suggest an infective process.

There was no true cirrhosis or biliary cirrhosis, but a widespread atrophy associated with processes of compensatory hypertrophy—a condition of subacute hepatitis, probably of toxic origin.

The spleen was much enlarged—about 20 oz. in weight. Microscopically it showed advanced passive congestion. Smears from the spleen were examined for micro-organisms and malarial parasites, but without success. The blood in the organ showed, however, very marked anaemic changes of the red corpuscles with numerous nucleated red cells. The kidneys were somewhat enlarged but healthy; the other abdominal organs were normal.

The clinical features of a marked rise of temperature, remittent in type, and a definite leucopenia with evidences of gastro-intestinal catarrh, made diagnosis uncertain during the early stages owing to the conspicuous fever present.

The acute destruction of liver cells and the condition of acute cirrhosis makes it of particular interest pathologically. Investigations of a most thorough nature by Dr. Horder and Dr. Matthews failed to reveal any bacterial infection in the blood during the various stages, and possibly we should be right, in view of the slight constitutional disturbance, apart from temperature, in regarding the poison as being of a chemical nature.

THE SIGNIFICANCE OF FREQUENCY AND TENESMUS IN ACUTE CYSTITIS.

By OLIVER HEATH, M.A., M.B., B.C.CANTAB.,

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THE main idea contained in the following may prove of interest, since its application in practice as an aid to vaccine treatment was followed in seven days' time by an absolute and permanent cure of an acute cystitis of just over one month's duration; and, further, since this result was obtained without the use of any drug, urinary antiseptic, or mechanical washings at any time during the course of the infection. The value of the method is not considered proven by the one case described; but since the writer is essentially an analytical pathologist, a series of cases is not easily obtained by him, and it must therefore be left to those who have the material at hand, and who may consider it worthy of serious trial, to put it to the test of experience.

At the outset it may be as well to state definitely that the term "acute cystitis" is used as applicable only to cases with both pus and bacteria in the urine, and does not here include bacteriuria without pus.

The history was as follows: A previously healthy man, a medical man, acquired gonorrhoea. A stained film of the discharge showed that this was caused by a mixed infection with the gonococcus, a *Staphylococcus albus* and a Gram-positive bacillus of the *xerosis* (or pseudo-diphtheria) type. There was no treatment other than care as to cleanliness, and the discharge disappeared in about three weeks. During the two months which followed there were two or three recurrences of a gleet character, which were not examined microscopically, but which cleared up; at the end of that time the patient began to realize that he could not hold his urine for longer than about two hours in the day, and had to get up once or twice in the night to micturate—that is to say, the infection had spread up into the bladder. This condition was allowed to continue untreated, owing to the great faith the patient put in his own unaided opsonic and phagocytic powers. However, these did not prove sufficient, and, after about one month of great discomfort, advice was sought from the writer, with a view to having a vaccine made.

Examination suggested that the condition was one of cystitis, with probably some post-urethral trouble, though no actual urethral discharge could be demonstrated macroscopically or microscopically. The urine contained a