ovarian tumour, and it was thought that the intestinal symptoms were due either to twisting of the ovarian pedicle or to pressure on the intestine by a tumour which had become impacted in the pelvis by the force used in bicycling.

The patient was advised to submit to an immediate operation, but declined, refusing to believe in the existence of any tumour. A morphine injection, a turpentine enema, and rectal feeding greatly relieved the symptoms, and next day the bowels acted and the vomiting ceased. The tumour remained extremely tender, and increased to double its size in a week. There was also a considerable amount of griping colicky pains.

On July 8th—twelve days from the onset of symptoms—ovariotomy was performed. During the preparation of the abdomen a large thin-walled cyst was felt to rupture. The tumour was a large multilocular papillomatous cyst arising from the right ovary, with a large solid mass the size of a foetal head, firmly adherent to the bowel, and when separated leaving at one spot a large white plaque the size of a penny.

In making a final examination of the pedicle preliminary to closing the abdomen, a hard lump was felt in a coil of intestine, and when brought into view this turned out to be an annular stricture caused by a solid growth completely surrounding the bowel, and apparently occupying its entire lumen. There were also some glands felt in the mesentery. The patient had been very much collapsed during the operation, and no further measures could have been undertaken at that time.

There were no unfavourable symptoms after this operation but much flatulence, and occasional twisting pain, apparently at the site of the intestinal growth.

On July 20th, that is, twelve days after the ovariectomy, the abdomen was again opened, and some 4 inches of small intestine removed with a V-shaped piece of mesentery containing some enlarged glands. The part removed appeared to be high up in the small bowel, and lay above and to the left of the umbilicus. The bowel was united end to end with the aid of an Allingham's bobbin. There was very severe hæmorrhage from dilated veins in the mesentery, and all the glands could not be removed.

The patient made an uninterrupted recovery. Contrary to expectation the bobbin was passed *per rectum* on the fifth day, and the bowels continued to act naturally. The growth proved to be a cylindrical carcinoma, and the lumen barely admitted the passage of a goose quill.

At the present time, February, 1901, the patient can walk about. No signs of recurrence can be felt from the abdomen or vagina, but there is pain and difficulty in passing flatus, though the bowels act freely with aperients.

CASE OF EXTREME STENOSIS OF THE SMALL INTESTINE IN AN INFANT.

By ERNEST W. HEY GROVES, M.D., B.Sc.LOND.,
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It is probably extremely rare to meet with a case of chronic intestinal obstruction in an infant apart from peritonitis or intussusception, and the recognition of such an occurrence may be of such practical importance that the following case presents both clinical and pathological interest.

G. P., a female infant aged 1 year 8 months, was brought to me suffering from distended abdomen and great emaciation.

History.—There was no history of tubercle or syphilis in the family. She had been a full term child, and was healthy and strong for the first twelve months of her life, during which time she was exclusively breast-fed. Soon after the baby was weaned, in February, 1899, when she was about 1 year old, she began to lose flesh, and was constantly crying, as if in pain. Since the appearance of these symptoms the child became steadily worse until September 3rd, 1899, when her condition was as follows.

Anamnesis.—She was extremely emaciated, weighing 9½ lbs., and constantly crying and moaning. Her face was thin and drawn, showing blue veins very distinctly through the skin. There were no signs of rickets, tubercle, or syphilis, and the dental development was good. The abdomen was much and uniformly distended, measuring 25 inches round at the navel. Through the very thin abdominal walls the coils of intestine could often be seen. The contractions of the intestine were accompanied by a loud gurgling noise and generally caused the child acute distress. The largest visible coil was 2½ fingerbreadths across. The whole abdomen was tympanitic to percussion; there was no tenderness on palpation and no hard masses or tumour could be felt. *Per rectum* nothing abnormal

was felt. The child was admitted to a nursing home for observation and treatment, when the following further facts were discovered.

She took her food greedily and was never sick (her mother, too, said she never remembered the child vomiting). The bowels were opened with great regularity two or three times a day. The stools were soft, but well-formed and rather clay-coloured. She was fed while under my care on dilute peptonised milk and meat juice. This careful feeding made the child more comfortable, so that she sometimes would sit or lie quiet for several hours together, and the abdominal distension and peristalsis were less marked.

She became much worse on September 9th, her temperature rising above 100° F. for the first time, and she refused her food. This continued until the 12th, when the child died. It was proposed to perform a laparotomy in the hope of being able to deal with the cause of intestinal obstruction, but the parents' consent to this was delayed until it was too late.

Post-mortem Examination.—On opening the abdomen several very large dark-red coils of intestine presented themselves, which proved to be the jejunum and part of the ileum enormously dilated. In the ileum, 39 inches from the cæcum and 25 inches from the pylorus, the calibre of the gut was suddenly reduced by a hard unyielding stricture three-quarters of an inch long, which only admitted a probe with difficulty. The other parts of the intestine were normal, but the mesenteric glands were slightly enlarged. There was no free fluid or adhesions, or any evidence of inflammatory changes. On opening the bowel in its dilated portion a great quantity of food debris was washed out, including fruit skins, vegetable fibres, a number of unripe black currants, and three cherry stones! This debris had formed a thick felted accumulation immediately above the stricture. There were no recognisable particles below the stricture. The other abdominal viscera were normal. An examination of the other parts was not allowed.

The clinical interest of the case centres round (1) the diagnosis and (2) treatment.

(1) The absence of febrile conditions, tenderness, and the signs of free fluid or hard masses, and the visible mobility of the intestinal coils excluded tuberculous peritonitis. There was no tumour, diarrhoea, or bloody mucoid stools, and the history of gradual onset was unlike intussusception. There were no means of telling whether the condition might not have been due to chronic obstruction by a band, and it was in hope of this that I proposed to open the abdomen. The fact that the illness dated from the time the child was weaned was clearly explained by the mass of debris found above the stricture, the peculiar character of which was explained by her having been allowed to play in a large fruit garden. The extreme emaciation and the regular action of the bowels pointed to the stricture being one of the small intestine, but, on the other hand, the complete absence of vomiting throughout was very remarkable, and left a doubt to the last that there could be any marked stricture of the small intestine.

(2) If it had been possible to diagnose the condition early enough and the child had been kept on a rigid diet of soft digestible food she might have lived for years. In support of this I would refer to a case of a man aged 62, who had a diaphragmatic stricture of the ileum 36 inches from the cæcum, who never showed any sign or symptom of the disease; and also Hudson's case of a boy aged 8, which I refer to below. As regards operative treatment the chances of success in such conditions are infinitely small. The intestine just above the stricture must have been brought out of the wound and opened and a subsequent anastomotic operation attempted.

Pathology.—The condition was almost certainly congenital, and as such is well recognised under three varieties—(1) a diaphragmatic stricture composed of mucous membrane, sub-mucous layer, and circular muscle, (2) a regular contraction of the bowel involving all the coats; and (3) an entire obliteration of the bowel at one or more spots. This last is the commonest variety, and, of course, leads to death with vomiting and wasting in a few days after birth. But as a disease associated with clinical manifestation apart from the newborn any kind of congenital stricture is extremely rare. I can only find one other instance, and that is of a boy, aged 8, who had suffered from a long-standing abdominal illness, and eventually died of peritonitis. He was found to have a stricture 2½-inch long, barely admitting a probe 38 inches above the cæcum.² The commonly-received theory of this congenital occlusion or stenosis is that it arises in connection with the involution of the embryonic vitelline duct. If this duct remains pervious as Meckel's diverticulum the whole way from the intestine to the navel, it causes a faecal fistula. If it only remains partially pervious and attached to the navel by a fibrous cord it often causes intestinal obstruction. If its connection with the umbilicus is obliterated, it remains as a simple diverticulum of the ileum

or an insignificant pouch. If the process of its involution is carried still further, it is supposed that an annular constriction or an actual occlusion of the bowel may take place. And there are some cases in which a fibrous cord or a polypus re-

Fig. 1.—A, appendix vermiformis; B, duodenum; C, colon; D, and E, empty and flaccid gut below stricture; F, stricture; G, hypertrophied and congested gut above stricture; H, rectum.

main attached to the seat of stricture, which bear out this theory; but in cases where such an appendix does not exist as in the above case, and still more so in the cases where the stricture is multiple, we have no knowledge of its causation.

REFERENCES.

- ¹ Treves's *Intestinal Obstruction*. ² Hudson, *Pathological Society's Transactions*, 1899, p. 99.

NOTES ON THE ANATOMY AND SURGERY OF MECKEL'S DIVERTICULUM.

By GEORGE A. CLARKSON, F.R.C.S.ENG.,
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OF all the foetal relics which are only occasionally found in the human body there is none, I suppose, with which we are more familiar than with Meckel's diverticulum. Its anatomy, like that of other functionless and useless relics, is subject to considerable variation. Representing as it does the vitello-intestinal duct, which in the early days of development connects the yolk sac to the bowel, in an ordinary way it undergoes complete and absolute atrophy, no trace of it remaining at all.

When atrophy, however, does not occur or is only partial, we may find one of the following conditions:

1. The duct may remain patent throughout its whole length. I had the opportunity of seeing a few years ago an infant in whom this condition was present. There was a small opening at the umbilicus leading into a distinct canal, down which a probe could be easily passed, and which occasionally discharged faecal material.

2. The portion near the intestine may persist, the other part either disappearing entirely or being represented by a fibrous cord extending from the apex of the diverticulum to the umbilicus.

3. The duct may be entirely converted into a fibrous cord attached to the intestine at that point from which it would originally rise.

The anatomy of the diverticulum is of some interest. Statistics show that it occurs in rather over 2 per cent. of all bodies examined, and it is said that it occurs rather more frequently in males than females. It is usually found within the last three feet of the ileum, but the exact position varies within wide limits. It is rare to find it more than 10 feet from the caecum, but 7 cases have been recorded where it arose from the duodenum, and more than twice that number from the jejunum. I have myself reported an instance of a well-developed diverticulum arising from the jejunum 2 feet from the pylorus.¹

The usual length is from 1 to 3 inches, and its origin is most often from that side of the intestine away from the mesentery. Rarely it springs from the mesenteric side, and may then lie between the layers of the mesentery. A process of the mesentery in some cases passes on to it; in others its vascular supply is derived from branches of the vessels supplying that portion of gut which it joins. Its structure resembles that of the adjoining bowel, but its coats are apt to be rather thinner. The size of the opening of the diverticulum into the bowel necessarily varies considerably. Where the communication is small or valvular, the formation of a faecal concretion is favoured. Foreign bodies have been known to lodge in the diverticulum and there cause ulceration.

Surgically the important point is with regard to the fibrous cord, the outer extremity of which may be attached to the umbilicus or to the abdominal wall below it. Generally, owing to the mobility of the ileum, it undergoes stretching and thinning till it breaks, the umbilical portion completely disappearing. The proximal portion is then peculiarly apt to form some fresh attachment, to one of the viscera it may be, to another portion of intestine, or to the mesentery. It is these adhesions that constitute the danger from the diverticulum. They are a fertile source of internal strangulation if they be present.

That strangulation from a Meckel's diverticulum is not a particularly rare occurrence is obvious from the fact that most surgeons of experience have come across cases, and that cases of it are not infrequently reported. In dealing with a case of internal strangulation from this cause it is important to ascertain whether a pervious tube or a simple fibrous band is being dealt with.

REFERENCE.

- ¹ *Journal of Anatomy and Physiology*, vol. xxvi.

THE LETTSOMIAN LECTURES

ON

DISEASES AND DISORDERS OF THE HEART AND ARTERIES IN MIDDLE AND ADVANCED LIFE.*

Delivered before the Medical Society of London.

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Hospital for Consumption, Brompton.

LECTURE II.

DIAGNOSIS AND PATHOLOGY.

As I have already pointed out, the causes of cardio-vascular disease in the second half of life are very often, indeed usually, complex. It follows therefore that if we desire, as we do most particularly, to discover the effects of each pathogenetic influence as distinguished from the others, we must begin our study with the simplest, or purest, or most definite of all, and proceed from it towards those which are more difficult, as well as to combinations of causes. It is easy to adopt this method in our present inquiry.

TOBACCO HEART.

We have in tobacco a single distinct influence at work—one that is universally acknowledged to affect the heart and vessels, and the physiological action of which is understood; one, further, that can be removed, perhaps not without some

* A few passages are omitted from the text of the lecture as delivered.