Death.

The general condition of the child meanwhile became steadily worse, and at no time after the operation was she in a fit state to attempt further operative measures. There was no vomiting and no special symptoms; the child simply became weaker and weaker. Bile in various forms by the mouth was tried, and also reduction of fats in the diet, but no improvement took place. She went steadily downhill, and died on February 10th. During the post-operative period the wound remained clean, and the temperature and pulse were not raised.

Post-mortem examination revealed a cystic swelling situated in front of the right kidney and behind the second part of the duodenum, which it had pushed forward. The swelling also extended behind the head and the greater part of the body of the pancreas. When injected with formalin to preserve it, it held 140 c.cm., and would probably have taken 20 to 40 c.cm. more. On dissection this swelling was found to consist of a cystic dilatation of the common bile duct, continued into the hepatic ducts and also into the cystic duct, where it joined the common bile duct. The walls of the cyst were about 2 mm. thick.

The whole of the extraduodenal portion of the common bile duct was involved in this cystic dilatation, and it was so closely adherent to the duodenum that no duct could be found between the cyst and the duodenum. The opening from the cyst into the duodenum consisted of a small slit-like orifice at the lower end of the sac. There was no flap of mucous membrane guarding it. After the specimen had been hardened in formalin, the patency of this orifice could not be demonstrated, but the opening into the duodenum appeared to have been very oblique. The pancreatic duct was patent and appeared normal. The gall bladder was collapsed. The liver was enlarged, hard, and definitely cirrhotic. The spleen was also enlarged and presented rather a mottled appearance.

Microscopical Examination.—The cyst wall was composed mainly of dense fibrous tissue. The inner layer was not quite so compact, and in places flattened epithelial cells could be recognized.

The liver showed a coarse cirrhosis of portal distribution; the fibrous tissue in the portal spaces was well formed and contained bile ducts, the larger of which were dilated and all of which had thickened walls. The bile ducts were lined with normal epithelium. The smaller ducts were more numerous than usual, though no actual proliferation was seen. The cirrhotic condition did not spread along the smaller bile capillaries, nor between the liver cells, though here and there detached liver cells were seen in the fibroid area.

The spleen pulp was intersected by strands of fibrous tissue, many of which appeared to have been formed by thickening of the normal trabeculae of the organ. In close relation to them the larger blood vessels lay, and from them more delicate strands of fibrous tissue spread into the spleen pulp. The blood sinuses of the spleen were moderately distended. The Malpighian corpuscles were of normal appearance.

Nearly fifty cases similar to the one described have been recorded, and possibly more might be included, as many observers have omitted doubtful or insufficiently described cases.

McWhorter,¹ in his excellent and comprehensive survey of the literature, states that cases in which there was a definite pathological condition explaining any obstruction, such as congenital atresia, pancreatitis, stone, or inflammatory stricture, were not included in his series, yet in his own case he describes the sac as containing some "bird-shot-like" stones, and the hepatic ducts as being partially filled with similar stones. He has, for instance, excluded Milner's² case, without, it seems to us, sufficient justification.

The condition is looked on by most observers as congenital, the evidence being that Heiliger³ described a similar condition in an almost mature foetus, and from the fact that most of the cases recorded have occurred in children and young subjects. The average age for the onset of symptoms would appear to be between 10 and 14 years: 12 to 14 in Waller's⁴ series, and rather younger in McWhorter's. Of the cases recorded 87 per cent. occurred in females.

In all the recorded cases there has been evidence of obstruction to the outflow of bile from the common duct into the duodenum. This has been shown:

1. *Clinically*, by the symptoms of jaundice, which has occurred in all but three cases, the jaundice either having been present at the time the patient came under observation, or a history of one or more attacks elicited.

2. *Pathologically*, by the fact that in many cases no opening from the common duct has been found at operation or autopsy, although from the patient's history an opening must have been present either at the time or very shortly before. In other cases where an opening has been found, obstruction has been demonstrated by oblique openings, valve-like folds of mucous membrane, etc.

3. *From Post-operative Recovery.*—Cases in which a primary choledocho-duodenostomy has been performed have

recovered; in other words, the obstruction has been overcome by means of a short-circuit.

In cases examined *post mortem*, some observers describe a fold of mucous membrane overlying the distal end of the common bile duct, and preventing bile from reaching the duodenum until the sac (upper portion of the bile duct) has sufficiently dilated to expose the opening. Others describe an abnormally oblique entry of the bile duct into the duodenum. Others, again, suggest that the cause of obstruction may be of the nature of an inflammatory duodenitis involving the duodenal portion of the common bile duct. Together with this obstruction it is assumed that there is a congenital weakness of the walls of the bile duct in its upper part, the cystic dilatation of the duct only involving the extraduodenal portion, and often only the upper two-thirds of the common bile duct.

It seems clear, therefore, that in all recorded cases there has been obstruction to the outflow of bile; that the obstruction is partial and intermittent; that the obstruction tends to progress—all cases not operated upon having died. While most observers take it for granted that the cyst is congenital, does it not seem far more likely that the obstruction is the congenital anomaly, and the cystic dilatation merely secondary to this, comparable to a hydronephrosis produced by chronic partial obstruction to the urinary outflow?

In no case has a diagnosis of this condition been made before operation or autopsy. This is hardly surprising since so few cases have been recorded. In nearly every case the surgeon has been unfamiliar with the condition.

In almost all the cases the symptoms have been similar, consisting of attacks of jaundice, abdominal pain, progressive enlargement of the abdomen, and a cystic swelling on the right side of the abdomen. The swelling has been variously diagnosed as a hydatid cyst of the liver, hydronephrosis, ovarian cyst, etc. The enlargement of the liver and spleen, such as occurred in our case, is by no means universal, although it occurred in Milner's case with very similar *post-mortem* findings. In some cases a biliary cirrhosis of the liver has been described. As regards treatment, where the cyst has been extirpated, its nature not having been realized at the time of operation, the mortality has been 100 per cent. The cases treated by drainage of the cyst alone have, with one or two exceptions, died. In some of these a subsequent choledocho-duodenostomy has been performed with success, but in most cases where it has been attempted it has failed.

The operation of choice is choledocho-duodenostomy at the first operation so as to provide internal drainage of the bile into the duodenum. From the position of the cyst, which lies just behind the duodenum, this should not prove too difficult an operation, provided the patient is not very young and is in good condition. The ease or difficulty of the operation may be determined in part by the thickness of the cyst wall, as in some cases it is quite a substantial structure, while in others it is so delicate that it has actually ruptured at the time of operation.

For a complete bibliography of the condition the reader is referred to the papers of McWhorter and Erik Waller, previously quoted.

We are indebted to Dr. T. H. G. Shore for the preparation and description of histological specimens.

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- ¹ McWhorter: *Archives of Surgery*, vol. 8, 1924, pp. 604-626. ² Milner: *BRITISH MEDICAL JOURNAL*, 1909, i, p. 1275. ³ Heiliger, J.: *Inaug. Dissert.*, Giessen, 1910. ⁴ Erik Waller: *Annals of Surgery*, October, 1917, pp. 446-463.

TREATMENT OF HAEMORRHOIDS BY THE
GALVANO-CAUTERY.*

BY

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THE objections to the ordinary surgical methods for the cure of haemorrhoids are considerable. Among these are the damage to normal tissue, the raw surfaces left and consequent exposure to faecal infection, the lengthened process of the healing of the sloughing mucosa, the prevalence of pelvic carcinoma; the loss of time and interference with occupation; the ordeal of an operation, with

* The substance of a communication to the Harveian Society of London, October 15th, 1925.

its very trying after-effects; the expense of the anaesthetist and nursing home charges, etc.

The method of treatment by injections of a solution of carbolic acid 10 per cent. in water and glycerin—or, as preferred in America, of quinine and urea—has been in use for many years, and has probably been tried by most of us. Unfortunately it is suitable only in simple uncomplicated cases and fails in some 50 per cent.; moreover, it sometimes causes very severe pain, lasting many hours, even in experienced hands.

Treatment with the galvano-cautery is almost as simple, is successful in every class of case, is painless, entirely under control, leaves no raw surface, can be performed on the consulting room couch at a moment's notice, occupies only a few minutes, and does not necessitate lying up or interfere with occupation. I have used this method for over twenty years and have never seen a failure, complication, or recurrence.

The patient, who is advised to come with an empty bowel, lies on the left side with a square of waterproof material tucked under the buttock and a packing of cotton-wool to catch any bleeding. Any growth extruding from the anus is first dealt with; the others, if they cannot be brought down with the rubber-clad finger aided by an expulsive effort of the patient, are exposed through a short speculum. Messrs. Allen and Hanburys have made, at my suggestion, a modification of the one used by the late Mr. Graeme Anderson, lipped and partially cut away at the upper end. Mine has the advantage that it will revolve, allowing any portion of the interior of the bowel wall to drop into the cut-away opening without moving the handle from the posterior buttock groove; two small eyelets to attach hooks from a waist-belt make the speculum self-retaining.

The mucosa is swabbed with a solution of crystal violet and brilliant green (1/500 of each), or of acriflavine (1 in 500). Eight minims of a sterile local anaesthetic, such as 2 per cent. novocain with adrenin and two drops of a 5 per cent. solution of carbolic acid added in a minim measure glass, are injected into the submucous cellular tissue at the base of the pile. I prefer the genuine Record syringe, using an adapter with Schimmel needles; these are so fine that the puncture closes at once. A minute later the pile is punctured to the depth of from one-quarter to one-third of an inch with a bulb-pointed burner (made for me by Messrs. Allen and Hanburys), not platinum, but an iron alloy which does not bend easily when hot and has a higher caloric capacity. A small pile needs only one such puncture, a large one perhaps three or four, especially if it is tough and fibrotic. The cautery bulb-point must go into the vein and be kept there, moving it in and out to prevent it sticking, until the blood is coagulated and bleeding stops. I never treat more than two piles at one sitting.

The local anaesthetic gives confidence, and I use it because, also, I have many times noticed patients flinch a little during the burning. It is advisable to blow away gently any steam or smoke which shows itself.

The speculum is then withdrawn, the interior of the bowel is lubricated (I prefer the old gall and opium ointment), and the pile mass pushed up through the sphincters. A good-sized piece of cotton-wool is wedged over the anus, into the buttock fold, and the patient can go back to his occupation, perfectly comfortable. He should return in a week. By that time there will be little or nothing to feel of the pile or piles so treated, and more can be done. The patient will say that, beyond a little bleeding after a motion, or a little sanious discharge, he has had no discomfort. Piles so treated rarely come down afterwards, and no slough is noticeable. The flaccid and loosened mucosa is fixed back by the cicatricial contraction which takes place and gradually shrinks all round; the sphincters regain tone and shut with a snap as they should do.

The small nodular, very painful swelling which appears suddenly during strain at the anal orifice and distresses so many of us in active life is for convenience called a pile; but it is not of the nature of the looped varicose vein which constitutes the ordinary haemorrhoid. It is a clot due to the accidental rupture of a small vessel into the subcutaneous connective tissue. It cannot be replaced; whereas the ordinary submucous pile is not painful and can be pushed back. The cautery is useless in this case. The clot can be cleared out through a half-inch incision into the skin, under infiltration anaesthesia; this gives immediate relief, and if the cavity be swabbed out with flavine the cut heals by first intention. If the cavity has previously become septic it must, of course, be drained. The alternative is rest in bed, a hypodermic injection of morphine, and hot fomentations.

British Medical Association.

PROCEEDINGS OF SECTIONS AT THE ANNUAL MEETING, BATH, 1925.

SECTION OF SURGERY.

Sir BERKELEY MOYNIHAN, Bt., K.C.M.G., C.B., M.S.,
F.R.C.S., President.

DISCUSSION ON ACUTE INTESTINAL OBSTRUCTION.

OPENING PAPERS.

I.—Sir WILLIAM TAYLOR, K.B.E., C.B., M.D., LL.D.,
Regius Professor of Surgery, Dublin University.

THE TREATMENT OF PRIMARY ACUTE INTESTINAL OBSTRUCTION.

THE surgery of the acute abdomen has advanced so satisfactorily during the past twenty-five years for every condition, with the sole exception of acute intestinal obstruction, that one is compelled to ask why the mortality attending the treatment of this condition should be not one whit better to-day than it was at the beginning of the century. The reason for this appalling state of affairs is not difficult to discern.

It is solely due to what, in truth, may be described as the ignorance and carelessness, both of which are inexcusable and are indeed nothing short of criminal, of the general practitioners or physicians who see these unfortunate cases in the earlier stages of their illness. Instead of recognizing that purgation and enemas can do no good, but, on the other hand, may do an infinity of harm, apart from the delay in surgical treatment their administration produces, these practitioners are continually laying themselves open to an action at law for malpractice, or, worse still, they run the risk of being placed in the dock and charged with manslaughter.

If the truth were generally known, the mortality attending the surgical treatment of primary acute intestinal obstruction the result of intra-abdominal conditions is nearer to 60 per cent. than the 35 to 40 per cent. usually stated—a truly shocking state of affairs. I may say that I almost get a rigor when I receive a telephonic message to ask me to go to hospital, either public or private, to operate upon a case of acute intestinal obstruction. Physicians and general practitioners have all come to recognize that the only satisfactory treatment for all other acute intra-abdominal conditions is early surgical intervention; but why they cannot be got to come to the same conclusion with regard to the treatment of acute intestinal obstruction passes my comprehension.

For over twenty years I have been accustomed to teach that, apart from acute intussusception, three stages can be recognized in these cases, and that the treatment must necessarily vary according to the stage in which one finds the patient.

The first stage is that in which the patient is seen early (within twenty-four hours). His general condition is good and there is but little intestinal distension. The second stage is that in which the patient is not seen until later—two, three, or four days. His general condition is good, but there is considerable intestinal distension and severe vomiting which may or may not be stercoraceous. The third stage is that in which the general condition of the patient is bad. His pulse is feeble and perhaps intermittent, and vomiting is almost continuous and is certainly stercoraceous. The abdomen is greatly distended, and the patient presents the appearance of one profoundly poisoned by the absorption of toxins, whether the result of bacterial activity, or a proteose intoxication, or a combination of both conditions.