Since the operation he has been perfectly well, and a report received in September, 1905, says that he has gained $3\frac{1}{2}$ st. in weight, has returned to work, and is in all respects quite well.

REMARKS.

The course of events in this case would seem to be quite clear. In December, 1902, a stone became impacted in the common duct; in April, 1903, this stone was passed. As a result of the irritation set up by the stone in the duct an ulcer formed, and this ulcer in healing gave rise to the stricture. The subsequent dilatation of the common duct and hepatic duct behind the stricture was due to the obstruction, which was increased when stones again arrived in the duct from the gall bladder.

Acquired stricture of the common duct is rare. It follows, as a rule, upon ulceration caused by the constant fretting of the wall of the duct by a stone; other causes are said to be typhoid ulceration and syphilis. Cases are recorded by Pye-Smith,¹ Johnson,² Phillips,³ and Pennato.⁴

Fig. 3.—Suture commenced.

Fig. 4.—Suture continued.

Fig. 5.—Suture completed; an opening left for the introduction of the tubes.

Fig. 6.—Tubes introduced, operation completed; note that the lower part of the duct though small in comparison with the upper part is also dilated.

Körte⁵ records the case of a man, aged 40, who had suffered for five months from jaundice, rigors, and remittent fever. On opening the abdomen, the gall bladder was found dilated, and stones were present in the cystic duct. The hepatic and common ducts were dilated. The common duct was opened and found to contain a stone behind a very narrow stricture. The stricture was excised and an end-to-end anastomosis made between the cut ends. A drainage tube was placed in the hepatic duct, and cholecystenterostomy was performed. The patient died on the twelfth day, of haemorrhage from an ulcer on the lesser curvature of the stomach; a large vessel was found eroded. Kehr⁶ also records a case of excision of a stricture of the common duct.

The various plans of treatment that may be adopted are: Excision of the stricture and end-to-end anastomosis; excision of the stricture, closure of the lower cut end of the duct,

and implantation of the upper end into the duodenum, which is "mobilized" to make the approximation easier; excision of the stricture, closure of both ends, and cholecystenterostomy.

The case now recorded is the first in which a plastic operation has been attempted, and the success of the case would seem to warrant the adoption of this method in a future case, as one of easier accomplishment than any other.

The illustrations have been drawn for me by Miss Ethel M. Wright.

REFERENCES.

¹ *Trans. Path. Soc.*, vol. XXIV, p. 250. ² *BRITISH MEDICAL JOURNAL*, 1880, ii, p. 200. ³ *Lancet*, 1887, ii, p. 926. ⁴ *La Clinica Moderna Italiana*, 1900, No. 6. ⁵ *Beit. z. Chir. der Gallenwege*, p. 341. ⁶ *Technik d. Gallenstein-operationen*, pp. 318, 319.

A CASE OF SUPPURATIVE CHOLANGITIS FOLLOWING CHOLELITHIASIS AND CHOLECYSTITIS.*

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WITH PATHOLOGICAL NOTES ON THE CONDITION OF THE LIVER BY

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THE following case is one of somewhat unusual interest, and fortunately of comparative rarity. Suppuration in the substance of the liver is a common occurrence within the tropics, but its more usual etiology and situation in the liver tissues contrast forcibly with the etiology and situation of the suppurative process in the case I now bring to your notice.

I considered it somewhat important that I should bring this case under your notice for two reasons: First, that a knowledge of this case might possibly be of some assistance in diagnosis should a similar case be met with by you; and secondly, that possibly a prophylactic treatment in the early stage, when a diagnosis of cholecystitis or cholelithiasis has been made, might lead to a happier result than ensued in the case under consideration.

W. H. D., aged 27 years, male, was sent up from Manila to Hong Kong on October 12th, 1904, convalescent from an illness characterized by "a sharp febrile reaction, moderate jaundice, nausea, and vomiting, and by a moderate but distinct enlargement of the liver." These symptoms were detailed by Dr. Strong of Manila, who also added that no leucocytosis was present.

Past History.—The patient had resided in Manila for the past four years. During the past three months he had suffered from two similar attacks, but in neither of these attacks was the liver enlarged. He had never suffered from dysentery, but had, however, suffered from diarrhoea now and again—in fact, while in Manila there was always a tendency towards loose motions. There was no history of gall-stone colic. He had never had typhoid fever.

Present Condition.—Temperature 100.5°, pulse 60, good tension and regular. The skin is of a yellowish tinge, and there is distinct jaundice in the fornices conjunctivae and over the sclerotics. Heart sounds normal. Breath sounds somewhat impaired over the right base behind. Liver: Dullness not enlarged upwards, but there is a definite tongue-shaped protrusion of liver substance downwards under the right rectus; this protrusion extends for three fingerbreadths below the costal margin. There is marked tenderness over the fundus of the gall bladder. No calculi can be felt. Spleen: Lower edge reaches two fingerbreadths below the costal margin. It is not tender on pressure. Urine: Specific gravity 1020, reaction acid, no albumen, no sugar, bile is present to a moderate extent. Stools: There is a tenderness to looseness without actual diarrhoea, slight acholia, no calculi or sand discoverable after careful search.

Examination of Blood.—Three smears were made and stained with Leishman's stain. No malarial parasites were present. There was no leucocytosis. A provisional diagnosis of cholecystitis and cholangitis was made, and a careful examination of the stools ordered to be made daily for the presence of "sand."

Treatment.—A light non-farinaceous diet was ordered, together with rest in bed. The following drug treatment was adopted: Hydrarg. cum creta gr. iij. Nocte. Carlsbad salts 1 drachm. Mane. Also: Tinot. podophylli miv; ammon. chlor. gr. xv; acid. nitro-hydrochlor. dil. mxxv; infus. gent. co. ad 5j. T. d. s. Hot fomentations were applied constantly over the whole hepatic area.

Progress.—October 25th. Some pain over the right hypochondrium, not extending through to the back or right shoulder. There is some crepitation on deep inspiration over the right base behind; the condition of the liver remains unaltered, tenderness being slightly increased.

October 26th. Examination of the blood reveals a slight leucocytosis, the increase being confined to the polymorphonuclear corpuscles. The liver exhibits a further enlargement downwards, and is very tender on

* Read before the Hong Kong and China Branch of the British Medical Association.

palpation. The jaundice has diminished slightly. The stools are still somewhat pale. The temperature is normal in the mornings, and rises to 100° or 99.5° in the evenings.

October 30th. Chloroform was administered, and an aspirating needle inserted twice through the right rectus into the enlarged liver substance in a direction upwards and backwards towards the dome of the organ. On each occasion a quantity of dark venous blood, but no pus, was withdrawn.

November 3rd. A rigor occurred at 2 a.m., the temperature rising to 102° .

November 7th. Rigors occur every night now, the temperature rising to 102° and over. The liver is further enlarged, and its dullness is now increased upwards to the lower margin of the fifth rib in the nipple line. It extends four fingerbreadths below the costal margin. Blood examination reveals a marked polymorphonuclear leucocytosis.

November 8th. Under chloroform a needle was inserted: In front through the right rectus in the same direction as before; in the sixth space in the mid-axillary line horizontally inwards; from behind towards the dome of the liver in the line of the scapular angle. No pus was found.

Diagnosis.—Suppurative cholangitis was now diagnosed, and it was surmised that the process was diffused throughout the liver substance with no definite localization of liquid pus suitable for aspiration and drainage.

November 25th. The patient has become markedly cachectic in hue. There is some referred tenderness down the right side of the abdomen towards the right iliac fossa. Rigors are of daily occurrence, generally taking place towards early morning. In a vain endeavour to afford some relief to the patient, chloroform was administered, and a 4-inch vertical incision was made through the middle of the right rectus as advocated by Mayo Robson. The abdomen was opened and the liver found considerably enlarged, the right lobe extending fully 4 in. below the costal margin; there were no superficial adhesions. After packing round the liver with sterilized gauze a needle was inserted in various directions directly into the liver substance. No pus was withdrawn. On the inferior surface of the liver the gall bladder was found to be completely adherent up to its fundus; it was not distended, and no actual gall stones could be felt. There were also adhesions between the under surface of the liver and the hepatic flexure of the colon and duodenum. The wound was united in separate layers with silkworm gut, the skin incision with horsehair, and a collodion scab applied.

After Progress.—December 3rd. Wound dressed; some suppuration at upper portion of wound in one stitch. The temperature the past three mornings has been below normal, rising with a rigor to 102.5° F. in the evening.

December 4th. A considerable quantity of "sand" was passed in the stool this morning; this was subsequently analysed by the Government analyst, Mr. Franklin, and found to consist of pure phosphate of calcium with some organic matter. Gmelin's test for bile pigment was positive. The passage of this sand in large quantities continued for ten days, accompanied by much tympanites and abdominal pain, the patient having to be kept continually under the influence of morphine. The aspect of the patient was markedly cachectic. The jaundice, which had disappeared except in the fornices conjunctivae, giving place to a very muddy complexion; emaciation was extreme, but the patient took his nourishment well, stimulants being administered freely.

December 23rd. There is now shifting dullness in the flanks; pain shooting through to the right scapula; cough and expectoration of muco-purulent material; there is some friction audible over the base behind and in the mid-axillary line.

December 25th. Toxaemia profound; temperature normal, no evening rise; pulse 100, very feeble.

Result.—The patient died a week after the last note at 11.30 a.m.

REMARKS.

One of the chief features of interest in this case lies in its pathogenesis. The pathogenesis of biliary calculi is still very obscure; experimental research has not as yet effected much in the elucidation of this important problem. Two main theories occupy the field:

1. The chemical theory.
2. The anatomical theory, as it is called, but personally I would prefer to contrast it with the chemical theory and call it the bio-chemical theory.

According to the first—the chemical theory—calculi are formed by the precipitation of cholesterol from special chemical changes in the bile. Cholesterol is dissolved in the bile by alkaline bile salts and soaps of sodium and potassium. If, as the result of nutritional exchange in the tissues, more especially in the nervous system, cholesterol is formed in large amount and is eliminated in the bile, supersaturation occurs and precipitation follows. Further, supersaturation is not even necessary, for the alkaline reaction of the bile may be diminished or changed to acid by a permanent excess of organic acids in the fluids of the body or by their imperfect combustion within the body. These organic acids set free a larger amount of lime from the tissues, and this being eliminated in the bile forms calcium soaps and bile salts of calcium, both of them insoluble. The diminution of glycocholate and taurocholate of sodium and

potassium further contributing to the precipitation of the pigment which they hold in solution.

The chief objections to this theory are those raised by Naunyn. These appear to me to completely negative the theory, namely:

1. The proportion of cholesterol in the bile is constant and is not increased in cholelithiasis; and
2. The bile contains an amount of biliary salts, soaps, and fats greater than is necessary for complete solution of the cholesterol present.

The second theory—the anatomical, or, as I prefer to call it, the bio-chemical theory—holds that calculus formation is due to a special catarrhal condition of the bile passages, whose function it is to separate the cholesterol and the salts of lime which, by reason of this inflammatory process of the mucosa, increase in amount.

According to this theory, then—a theory which in the light of modern pathology seems a reasonable one—calculus formation depends on catarrh of the bile passages.

What is the cause of this catarrh? Researches tend to demonstrate a bacterial origin. It has been proved—

1. That the gall bladder is a favourite habitat of the following organisms: Colon bacilli, staphylococci, streptococci, pneumococci and typhoid bacilli, the latter in Welch's laboratory having been isolated from the gall bladder in pure culture seven years after an attack of typhoid fever.
2. That gall stones can be produced experimentally by injecting micro-organisms into the gall bladder of animals.
3. That gall stones have an association with certain specific fevers, more especially typhoid.

In the case under consideration I have already stated that the patient had always, while resident in Manila, a tendency to looseness of the stools—it is not improbable, therefore, that he suffered from a chronic colitis with subacute attacks, as evidenced by his occasional attacks of diarrhoea. Now, under conditions such as these it is well known that the *Bacillus coli communis*, ordinarily harmless, can become pathogenic, and I venture to think that this organism was perhaps the causa vera of the trouble.

As regards the diagnosis of such a case, I think it will be freely admitted that this is difficult. Gall stones may exist in the gall bladder and cause no symptoms. If this is the case with true calculi, how much more likely is it to be the case when gall stones occur in the form of gravel or sand! In the present case symptoms were caused perhaps from the outset of the disease. Amongst these symptoms one noticed an association which led one at once to suspect the true nature of the case. I refer to—

1. The tenderness over the gall bladder.
2. The tongue-shaped protrusion of liver substance beneath the costal margin, such as is often present in such cases.
3. The presence of jaundice.
4. The nausea and vomiting.
5. The enlargement of the spleen without obvious cause.

With such an association of symptoms I feel sure that few would neglect a careful examination of the stools for calculi or "sand." In this case the search proved negative at first, but continued search cleared up the nature of the case. The jaundice was not intense—it rarely is in cases of cholangitis. The tongue-shaped enlargement of the liver associated with cholelithiasis only occurs when the gall bladder is elongated and brings down with it the adherent right lobe of the liver.

Amongst the many affections of the spleen, enlargement associated with cholelithiasis is rarely mentioned, and yet such is often the case. If careful blood examination fails to reveal a cause for the enlargement in a case with the already mentioned associated symptoms, and if there is no other obvious cause, I would always regard this sign as strong confirmatory evidence of cholelithiasis. Naunyn gives it as one of the distinguishing signs of stone in the common duct, but he gives it in association with other symptoms which were not present in this case.

With regard to the form of cholelithiasis in this case, while "gall sand" is not uncommon, the formation of actual calculi is by far commoner. If the gravel type is assumed the stones generally consist of bilirubin calcium, and vary in size from a grain of sand to a pea. In this case the rarest type occurred—namely, phosphate of calcium associated with bile pigments—and, as you have seen, the form taken is that of a very fine sand.

As regards the occurrence of suppurative cholangitis, this is fortunately a rare event, though perhaps, if all cases were collected, not so rare as we might be led to suppose. It generally follows suppuration around a calculus or calculi in a branch of the hepatic duct, spread of infection taking place

to the finer ducts. In the case under consideration, I think the sequence of events must have been cholecystitis, simple non-suppurative cholangitis, the formation of sand, then suppurative cholangitis; my chief reason for this being that in the early stages of the disease there was an entire absence of leucocytosis, as evidenced by Dr. Strong's examination and my own. Cholecystitis can occur without known pre-existing disease. This was proved by Maurice Richardson, who, in a paper published in the *American Journal of the Medical Sciences*, stated that in 10 out of 59 cases of operation on the gall bladder acute cholecystitis was present with no history of previous disease.

In this case we have a past history of colitic trouble which I have already noted. The occurrence of spread of infection to and suppuration in the bile ducts is marked by increased enlargement, tenderness of the liver, increased pain, often radiating through to the angle of the scapula, leucocytosis, and progressive emaciation and loss of strength. Such cases generally run a short course, and recovery is said never to take place. That spread of infection from the gall bladder to the bile ducts does not occur oftener is due to the fact that in acute infectious cholecystitis the cystic duct is usually occluded.

With reference to treatment in such a case, the only hope lies in surgical interference—namely, cholecystotomy and drainage. In this case such a procedure proved a physical impossibility owing to the great enlargement of the liver and inaccessibility of the gall bladder. Had such a procedure been possible, it is almost beyond doubt that in this case little relief would have been given owing to the diffuseness of the suppuration in the bile ducts.

With regard to prophylactic treatment, the diet should be carefully regulated, avoiding as much as possible starchy and saccharine foods. The sulphate, or phosphate of soda or Rochelle salts, may be taken in doses of 1 to 2 drachms daily; regular exercise should be indulged in.

If the diagnosis is confirmed on examination of the stools and the patient's condition is not improving, early surgical interference is necessary, and drainage of the gall bladder before adhesions have formed and before the infective process has spread from the gall bladder up the biliary ducts.

Only a limited examination was possible *post mortem*.

The following notes on the condition of the liver are by Dr. Hunter (Government bacteriologist, Hong Kong):

Pathological Appearances.—The liver was generally enlarged, increased in weight and in specific gravity, and its consistence was tough and leathery. It was jaundiced. There was no marked congestion. The general contour of the organ was maintained.

On section of the organ, the following appearance was presented: Studded throughout the entire right hepatic lobe were rounded areas of a yellow colour. These varied in size from a millet seed to a pea. Many had a deeply pigmented centre. Surrounding these areas there was a narrow zone of tissue of a deep brownish colour.

The remaining out surface of the organ was that of a jaundiced liver with commencing interlobular cirrhosis.

An examination of the yellow areas more in detail showed that they were not equally distributed throughout the right lobe. They were most abundant in the neighbourhood of the main portal, hepatic, and biliary channels.

In the intervening hepatic parenchyma they were few and scattered.

These areas were further examined as to their relation to the parenchyma of the liver, to their consistency, and their microscopic and bacterial composition.

They bore no direct relation to the actual hepatic parenchyma. These yellow areas could be picked out *en masse*, leaving a hollow or space with roughened but hard and fibrous walls. The consistence of these masses varied. Some were pultaceous; others were hard and gritty. To touch, the harder masses were like small peas, and could be thrown about without much damage to their form.

Microscopically, the softer masses were composed of fibrin, hepatic duct epithelium, pus, corpuscles, and a variable amount of bilirubin pigment.

The harder masses were made up of a hyalin-looking material, in which traces of fibrin, pus, corpuscles, etc., could be seen. In the meshes of this material appreciable quantities of bilirubin calcium were found.

Bacteriologically, nothing definite could be obtained, owing to the fact that an examination could not be made until about twenty-four hours after death. *B. coli communis* was found in large numbers. With this organism, however, numerous other extraneous bacteria were present.

The zones, of a deep brownish colour, immediately surrounding the yellow areas, presented the following micro-

scopic appearance. The areas of tissue nearest to the yellow masses were composed of young proliferating connective tissue and hepatic duct epithelium. The changes found were typical as regards a chronic inflammation of the walls of the hepatic duct and its ramifications. Immediately outside this zone of newly-formed tissue, a narrow band of degenerated hepatic parenchyma was found. Large quantities of bile pigment were deposited in this narrow band of newly-formed tissue and degenerated liver cells.

Apart from these changes, the microscopic examination of the liver substance showed marked interlobular increase of connective tissue and the presence of pronounced paracholia.

PORTAL PYAEMIA AND PYLEPHLEBITIS.

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I.—GASTRO-INTESTINAL ULCERATION AS A FACTOR IN SUPPURATIVE PYLEPHLEBITIS.

GASTRO-INTESTINAL ulceration is usually held to be the most potent cause of suppurative pylephlebitis, especially when that ulceration is in the neighbourhood of the appendix. Yet diseases in which gastro-intestinal ulceration is most extensive are not those which lead to suppurative pylephlebitis. If such ulceration were really the direct cause, surely we should expect to find this complication frequently in enteric fever and dysentery, for example. As a matter of fact it is extremely rare in these diseases. In the *post-mortem* register of St. Bartholomew's Hospital I have not been able to find a single instance of pylephlebitis in enteric fever; and Dr. J. H. Bryant has kindly informed me that a search through the records of Guy's Hospital (which reach back to 1828) was equally fruitless.

Hugue¹ found that hepatic suppuration of any kind was very rare in enteric fever, and that if it occurred there was usually colon typhoid. With multiple abscesses there was usually pylephlebitis. He adds the interesting statement that this complication is more common in those who have already suffered from some lesion of the liver. Osler² has only seen one case of suppurative pylephlebitis in enteric fever, and here there was also an empyema of the gall bladder, with a sinus running back from the cystic duct which ended close to the suppurating branches of the portal vein. Romberg³ saw 87 necropsies out of 677 cases of enteric fever at Leipzig, and in only one was there pylephlebitis. Here, besides healing ulcers in the ileum, there was a suppurating phlegmon in the mesentery. Lannois⁴ saw one instance out of 71 necropsies of enteric fever. In this there was definite ulceration of the appendix.

It should be noted that in two of the above cases suppuration other than the typical intestinal ulceration occurred, and that in the third the appendix was involved—a point at which the portal vein is specially liable to invasion.

Again, though single abscess follows dysentery, multiple abscesses with suppurative pylephlebitis is a rare sequel. Marotte⁵ records a case, and Bryant's⁶ statistics give an example. I have only found one instance of it recorded at St. Bartholomew's Hospital. I am, of course, excluding cases where septic infection followed operation on a tropical abscess. We may conclude that it is decidedly rare for pyogenic organisms to invade the portal vein from the surfaces denuded by dysenteric ulceration. It has been suggested, moreover, by Councilman and Lafleur⁷ that even the *Amoeba coli* does not usually reach the liver by the portal vein, but through the peritoneal cavity. Councilman found circumscribed masses on the walls of a tropical abscess, which proved to be the persistent remains of the portal system.

Considering the liability of the gastro-intestinal tract to ulceration, and the varied nature of the microbes inhabiting that tract, it is at first sight surprising that portal pyaemia is not a more frequent occurrence. Elsewhere⁸ I have urged that "a collection of pus outside the gastro-intestinal tract is the real exciting cause of portal pyaemia. Ulceration of the tract is of importance as leading to this accumulation." This would explain why the region of the appendix is so frequently the focus, for nowhere else are "the conditions more favourable to . . . ulceration by faecal concretions or foreign bodies and to the production of such an accumulation of pus" within the portal zone.