tion. Because of the danger of the infant aspirating vomited barium a small quantity of 70 per cent. diodone is a preferable medium. One of the reasons for avoiding radiography after an opaque meal in an infant is over-radiation. Another is that the passage of a gastric tube and aspiration of a large residue two hours after a feed is equally informative.

Differential Diagnosis.—The most difficult condition from which hypertrophic pyloric stenosis of infants has to be differentiated is *cardiochalasia*[1] *of the œsophagus*, often due to a hiatus hernia (see p. 307): a clinical entity that has been summarised pithily as one of " persistent vomiting without bile and with no other signs of hypertrophic pyloric stenosis." The mother sometimes volunteers the key information that the infant does not vomit if it is held in the upright position. In this instance radiology is essential; it shows that the gastric cardia is in a state of continuous relaxation with frequent regurgitation of the barium that has been given.

Other conditions from which hypertrophic pyloric stenosis must be differentiated are (*a*) intracranial hæmorrhage; (*b*) duodenal atresia; (*c*) high intestinal obstruction, e.g. volvulus neonatorum (see Chapter 26). In (*b*) and (*c*) bile will be present in the vomitus.

Lastly, attention is drawn to the fact that in babies under two weeks of age a palpable pylorus is not conclusive evidence of hypertrophic pyloric stenosis (W. S. Craig). The term pylorospasm is a convenient label suggesting a reversible upset in function in which the infant patient empties its stomach by repeated rather than one copious vomit, and in these cases the pyloric lump is not so firm, not so well-defined, and not contracted as that of a patient with hypertrophic pyloric stenosis. The proof that this clinical concept exists is that in a series of cases reported by Craig, answering in all particulars to the designation 'pylorospasm,' the patient stopped vomiting with no more treatment than gastric lavage.

Treatment.—In most centres, for the majority of patients, medical treatment is employed for only two or three days, to prepare the patient for operation. Patients who, on account of complete pyloric obstruction, are admitted in a dehydrated condition should receive sufficient dextrose-saline solution, given intravenously, or subcutaneously with hyaluronidase, or both, to restore the fluid balance. This results in a remarkable improvement in the general condition; the sunken eyes and cheeks and depressed fontanelle fill out; the dry skin and mucous membrane become moist, and the output of urine increases. A gastric tube is passed, and after aspirating and measuring the amount of retained gastric contents, the stomach is washed out with normal saline solution. Eumydrin (atropine methyl-nitrate) 1 : 10,000 of water, freshly made, is given half an hour before each feed, beginning with 0·5 to 1 ml. and increasing to 2·5 ml.

Toxic symptoms (erythema and hyperpyrexia), though less common than when atropine is employed, do occur with eumydrin, and are signs that the dose of the drug should be decreased or discontinued.

Small, frequent feeds of milk (if possible the mother's milk), diluted with 5 per cent. dextrose, are given.

In subacute cases the patient often reaches the age of two months before the symptoms become obvious and if, as a result of medical treatment, the gastric residue decreases in amount, a cure by non-operative means can be anticipated.

In cases complicated by infection, especially of the mouth, because of the increased risk of post-operative gastro-enteritis, a continuance of medical

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[1] Cardiochalasia is the direct opposite of achalasia of the cardia (cardiospasm).

William Stuart McRae Craig, Contemporary. Professor of Pædiatrics and Child Health, University of Leeds.
Elisabeth Svensgaard, Contemporary, Physician, Rigshospitalet, Copenhagen, introduced eumydrin as a therapeutic improvement on atropine in 1936.

the vomiting becomes forcible and projectile, when usually some of the vomitus is ejected through the nose. There is no bile or other yellow colouration of the vomitus: exceptionally a few streaks of blood are observed therein. Commonly, immediately after vomiting the baby is very hungry.

Visible Peristalsis.—After the child has been fed, often peristaltic waves can be seen passing from left to right across the upper abdomen, but protracted watching, perhaps on several occasions, is sometimes required before the phenomenon becomes apparent. Peristaltic waves are unlikely to be seen unless the stomach is fairly full.

The Presence of a Lump.—Usually the hypertrophied pylorus can be palpated mid-way between the umbilicus and the right costal margin. It has been described as feeling like an acorn, or an olive, or the tip of a nose beneath the bed-clothes, and from time to time it can be discerned to harden and then to soften beneath the examining fingers. Without conclusive evidence of the presence of a palpable pylorus the diagnosis of hypertrophic pyloric stenosis is incomplete, and on this account not infrequently prolonged and repeated examinations are required to elicit it. The lump is more likely to become detectable soon after the empty stomach has received an ounce (30 ml.) of milk.

Constipation is present invariably, and when a stool is passed it is small, dry and brown, like that of a rabbit.

Loss of Weight.—One of the most striking signs of infants suffering from hypertrophic pyloric stenosis is loss of weight.

Naturally it is not long before the infant begins to look emaciated and dehydrated. Furthermore, recurring loss of hydrochloric acid causes alkalosis which, if not remedied, will bring about skeletal muscular hypertonicity and respiratory disturbances.

Sometimes a change from the breast to artificial feeding results in a remission. Consequently a series of changes in diet are sometimes made before the diagnosis is established, by which time the infant's condition is pitiable. No baby should ever be taken off the breast because of vomiting (S. E. Keidan).

In premature infants, in whom the condition is not uncommon, the symptomatology is often paradoxical. There is anorexia instead of a voracious appetite; the vomiting is regurgitant rather than projectile, and so frequently is peristalsis visible in these attenuated subjects that its significance is liable to be disregarded. None the less, amidst this sea of bewilderment there is consolation—a hypertrophied pylorus can be felt through the poorly-developed abdominal wall with comparative ease.

Radiography.—A plain radiograph shows the stomach to be full of air with a diminution of intestinal gas. In dehydrated patients the complete absence of intestinal gas is noteworthy. Occasionally (perhaps 1 in 40 cases) an opaque meal is necessary to confirm the diagnosis. Normally the baby's stomach is completely empty in three hours; in congenital hypertrophic pyloric stenosis there is a large gastric residue at the end of that time. Barium emulsion should always be aspirated at the conclusion of this examina-

Saul Eli Keidan, Contemporary. Pædiatrician, Alder Hey Children's Hospital, Liverpool.

treatment is advisable until the infection is controlled, even if operation would otherwise be indicated.

With these two exceptions, if operation is performed in comparatively early cases, the recovery rate in breast-fed babies approaches 100 per cent. Among bottle-fed babies the mortality is appreciable, owing to their greater susceptibility to gastro-enteritis (D. Levi).

Ramstedt's Operation.—*Preliminary Preparation.*—The stomach is washed out several times ; finally, one hour before operation. Immediately before operation gastric aspiration must be performed, and continued throughout the operation. The prevention of chilling is of great importance. To this end the temperature of the operating theatre should be high (80° F., 27° C.) and the infant's body is encased in wool, the upper abdomen alone being accessible.

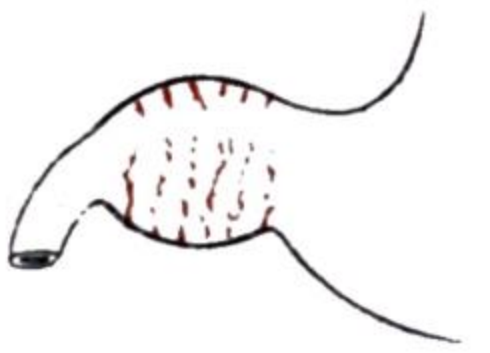

FIG. 509. — The upper part of the hypertrophied pylorus is comparatively bloodless.

Operation.—Local anæsthesia is advisable unless the child is in good condition and the services of an anæsthetist skilled in anæsthesia in infants are available. The abdomen is opened by a grid-iron[1] incision in the upper right quadrant of the abdomen. The hypertrophied pylorus is delivered and rotated so that its superior surface comes into view ; thus the most avascular portion (fig. 509) can be selected for the incision. In order to ascertain the distal limit of the hypertrophied pylorus the surgeon invaginates the duodenum with his index finger. The incision is made through the serosa only from this point along the whole length of the lump (fig. 510). Because the hypertrophied pylorus is of the consistency of an unripe pear, splitting muscle coats can be accomplished with a blunt dissector. On separating the edges with artery forceps, the pyloric mucosa bulges into the cleft which has been made in the muscle (fig. 511). Great care is taken not to penetrate the mucosa,

FIG. 510.—Ramstedt's operation.

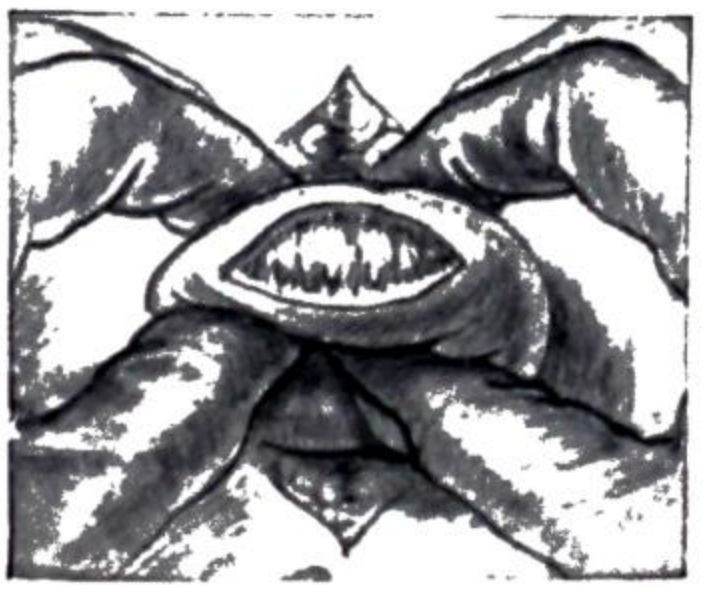

FIG. 511.—Showing the hypertrophied muscle divided and the mucous membrane bulging into the incision.

an accident which is liable to occur while dividing the most distal part of the constricting fibres which are in the vicinity of the duodenal 'fornix' (see fig. 507). In order to be sure that there is no perforation, some air is squeezed from the stomach into the duodenum. If a perforation has occurred, it is closed by a wisp of omentum held in place by three or four interrupted sutures of fine silk (fig. 512). The free entry of air into the duodenum indicates adequate division of the muscle. It is necessary to suture bleeding vessels in muscle in about one-third of patients. Hæmostasis must be meticulous. The abdominal incision is repaired. The edges of the loose skin fall together and can be kept in apposition with strips of adhesvie plaster.

FIG. 512.—Patching a perforation of the mucous membrane. (*After C. E. Welch.*)

After-treatment.—To minimise the risk of cross-infection, and consequent gastro-enteritis, the patient must be segregated. Further infusions of dextrose-saline, and, in patients in poor con-

[1] Transverse through the skin and the anterior rectus sheath, and splitting the rectus muscle vertically.

David Levi, Contemporary. Surgeon, Infants' Hospital, London.
Conrad Ramstedt, Contemporary, Emeritus Chief Surgeon, Rafael Clinic, Münster introduced his operation in 1913. He is now 93 years of age.

dition, plasma or blood transfusion should be given. The feeds must be very small and well-diluted, not more than ℥i (4 ml.) being given at a time ; they are commenced eight hours after the operation unless the mucosa was accidentally opened, when the child is not fed orally for a full forty-eight hours. The majority of patients cease to vomit on the third post-operative day.

Complications:

(*a*) *Post-operative pyrexia* is rather common and is not necessarily of serious consequence. If excessive, tepid sponging is advisable.

(*b*) *Gastro-enteritis* is a troublesome and sometimes fatal complication. It is minimised by thorough medical preparation, very careful after-feeding, and the administration of sulphasuccidine.

(*c*) *Disruption of the wound* is a rare complication, and is more liable to occur in emaciated subjects. The use of the grid-iron incision in these cases has reduced the incidence of this complication.

Hypertrophic pyloric stenosis of adults is not exceedingly rare. While the diagnosis of pyloric stenosis *per se* presents no difficulty, that the condition is not due to ulcer or carcinoma is seldom suspected clinically. In a few instances the X-ray findings are reported by the radiologist as being due to primary hypertrophic stenosis, but as a rule the true nature of the case is only elucidated after partial gastrectomy and microscopical examination of the specimen. Contrary to the corresponding condition in an infant, the myenteric nerve plexus exhibits fibrosis. When a pre-operative diagnosis can be made with assurance, Finney's pyloroplasty or a gastro-jejunostomy will render the patient symptom-free.

CONGENITAL OCCLUSION OF THE DUODENUM

There is a septum, usually complete, across the duodenum (fig. 513). This occurs at the point of fusion of the fore- and mid-gut, and consequently lies in the neighbourhood of the ampulla of Vater. The infant vomits *from birth*, and daily rapidly loses weight. In contradistinction to congenital pyloric stenosis *the vomit contains bile*. Laparotomy should be undertaken without delay. Duodeno-jejunostomy is the best procedure when, as is usually the case, the obstruction is in the second or third part of the duodenum (W. E. Ladd). Lasting cures have also followed gastro-jejunostomy.

FIG. 513.—Congenital septal duodenal obstruction at the commencement of the third part of the duodenum. The gut above is enormously dilated.
(*After W. E. Ladd.*)

FOREIGN BODIES IN THE STOMACH

A variety of ingested foreign bodies reach the stomach. Fortunately, for the most part they are opaque to X-rays. Sharply pointed or large objects are best removed promptly by gastrotomy. Rounded, smaller foreign bodies may be left to pass along the natural passages. Suitable doses of normacol form a gelatinous pabulum, in which the article becomes embedded during its transit along the alimentary tract. That the journey is being accomplished can be verified by periodic examinations under the fluorescent screen (fig. 514).

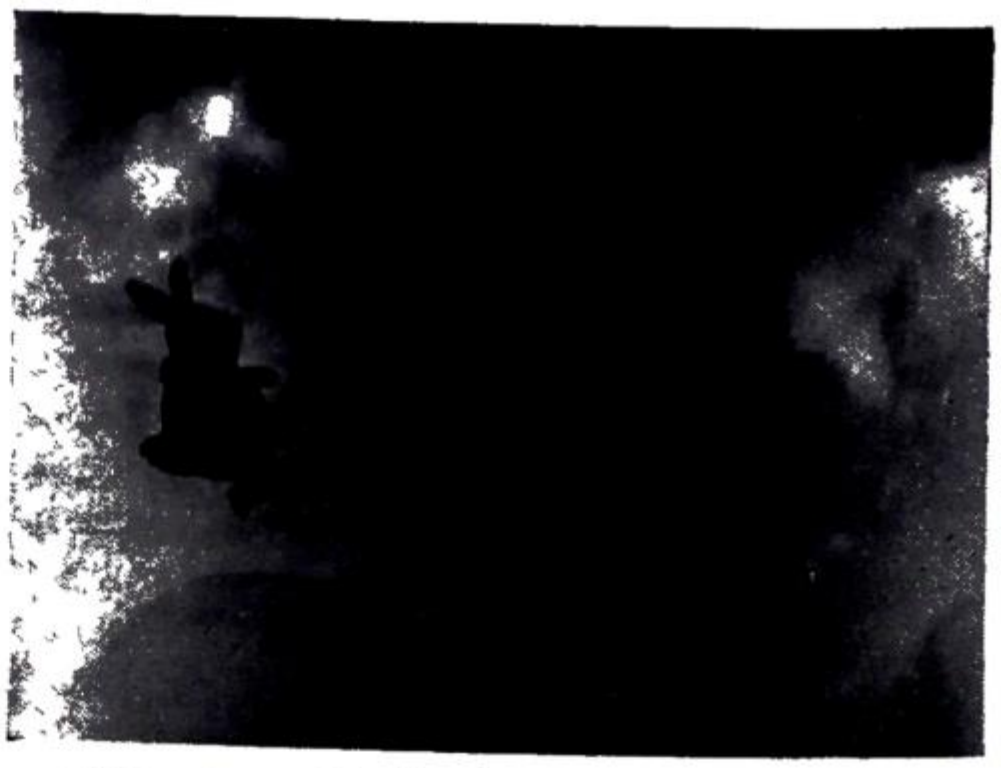

FIG. 514.—Embedded in normacol, this foreign body was passed naturally in three days.

John Miller Turpin Finney, 1863–1942. Surgeon, Johns Hopkins Hospital, Baltimore, U.S.A.
Abraham Vater, 1684–1751. Professor of Anatomy and Botany, Wittenberg.
William E. Ladd, Contemporary. Emeritus Professor of Child Surgery, Harvard University, Boston, U.S.A.

Hair-ball of the Stomach (**Trichobezoar**[1]).—An example of this rare condition is usually to be found on the shelves of a pathological museum. Trichobezoars occur almost exclusively in females, and in 80 per cent. of cases the patient is below thirty years of age. An individual who swallows quantities of hair is often feeble-minded, but this need not necessarily be the case. Trichobezoars can give rise to high intestinal obstruction, gastro-duodenal ulceration leading to hæmatemesis, perforation, peritonitis, or inanition. A trichobezoar shows well on radiography (fig. 515). The treatment is removal of the mass by gastrotomy.

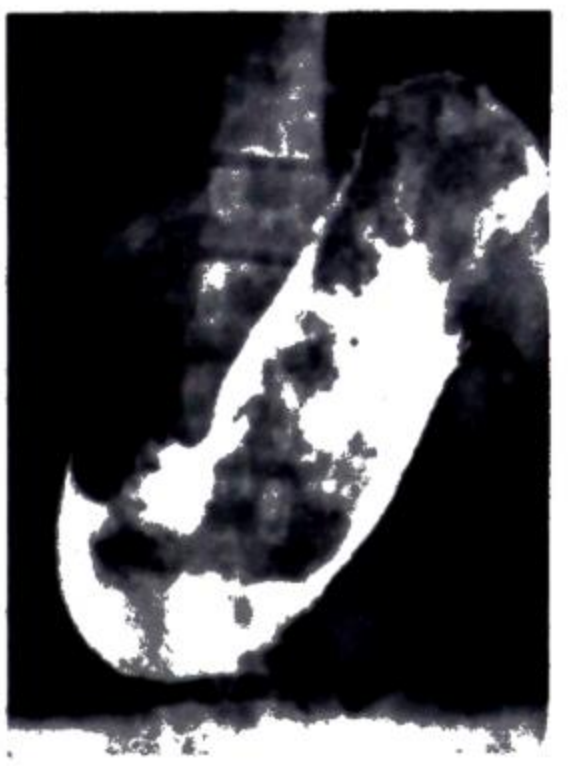

FIG. 515.—Radiograph showing hair-ball in the stomach. *(Dr. P. M. Wine Birmingham.)*

ACUTE DILATATION OF THE STOMACH

The incidence of acute dilatation of the stomach, once a fairly common post-operative complication, has been reduced to rarity by the routine use of transnasal gastric aspiration before, during, and after abdominal operations. It can occur after any operation, or after the application (under an anæsthetic) of a plaster-of-Paris jacket, but the greatest incidence is after operations on the biliary passages and pelvic organs. More rarely, the condition complicates the state of shock, such as might be occasioned by a fractured femur.

Pathology.—In fatal cases the stomach is found to be enormously dilated (fig. 516). The organ is filled with air and dark, watery fluid. Sometimes the dilatation ends at the pylorus ; more often it extends into the duodenum, and in a few instances the dilatation involves the extreme upper end of the jejunum. The gastric mucosa is bespattered with petechial hæmorrhages.

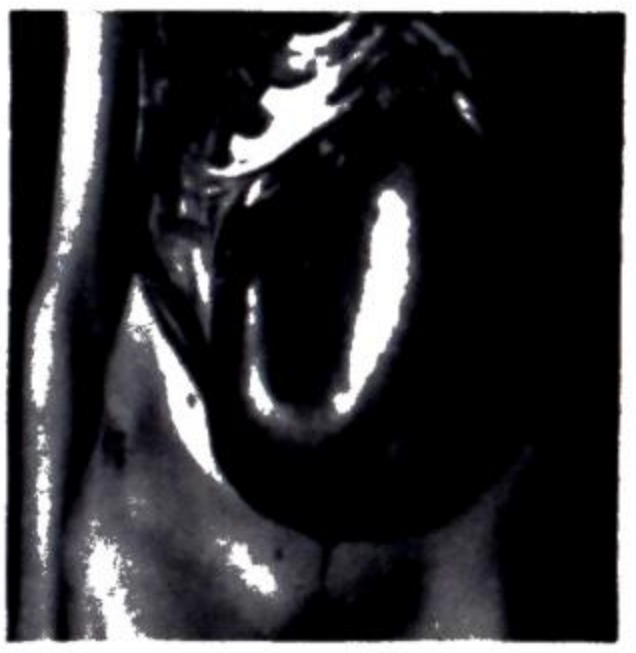

FIG. 516.—Acute dilatation of the stomach seen at necropsy.

Ætiology.—Acute dilatation of the stomach can be looked upon as a local form of paralytic ileus.

Clinical Features.—Signs of acute dilatation of the stomach may come on two or three hours, or as long as two or three days, after an operation or an accident. The first warning sign is often muffled hiccoughs. Soon the pulse-rate rises to 100 or 120 beats per minute and other signs of shock become manifest. Then the patient vomits ; if the contents of the stomach is not aspirated forthwith, as it should be, this is followed by enormous effortless vomits of dark, watery fluid which is characteristic of the condition.

On abdominal examination a fullness in the epigastrium can often be seen, and splashing sometimes can be elicited, but these signs are less evident in obese patients.

Prevention:

(1) Daily auscultation of the abdomen after an abdominal operation and forbidding anything by mouth until normal intestinal sounds can be heard; and (2) daily measurement of the girth of the abdomen at the level of the umbilicus go a long way to preventing this condition and detecting paralytic ileus early.

Treatment.—In a fully established case, prompt action is imperative. The two principles are : (1) To empty the stomach and keep it empty ; (2) to restore fluid balance by administering continuous intravenous normal saline, followed (after chloride loss has been satisfied) by dextrose-saline solution. The stomach should be emptied and kept empty by aspiration through an indwelling Ryle's or other small gastric tube passed, when possible, by way of the nose. In an endeavour to restore the muscular tone of the stomach it is permissible to inject one dose of 50 mg. of calcium pantothenate [2] intramuscularly.

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[1] Bezoars, or masses of foreign material in the stomach or intestines of animals, are relatively common. In some primitive communities a gastric bezoar from a goat is accredited with magical healing properties.

[2] Pantothenic acid is one of the vitamin B complex components. It is a co-enzyme involved in the formation of acetylcholine.

John Alfred Ryle, 1889–1950. Professor of Social Medicine, University of Oxford.

By these measures, unless effective treatment has been delayed unduly, the stomach soon regains its tone. If it does not do so, the possibility of hypopotassæmia (see p. 76) being a contributory cause should receive immediate attention.

GASTRIC TETANY

Gastric tetany, due to alkalæmia, sometimes complicates simple or malignant pyloric obstruction, particularly when there has been long-continued vomiting, or much alkaline medicine has been ingested. The spasms, which are usually confined to the extremities, are accompanied by depressed respiration and cyanosis. The dilated stomach is emptied and kept empty by aspiration. Continuous intravenous dextrose-saline solution should be administered without delay. The blood calcium level must be raised by the administration of calcium (see parathyroid tetany, p. 247). In addition, an acidifying mixture, e.g. ammonium chloride by mouth, is prescribed.

RUPTURE OF THE DUODENUM

Traumatic rupture of the duodenum is a rare accident, usually the result of a blow on the right flank. The rupture may be intra- or extraperitoneal, or both.

Intraperitoneal Rupture.—The tear can usually be sutured.

Extraperitoneal Rupture.—The initial symptoms are often slight, and the condition is overlooked until an abscess forms. When such an abscess is opened, a duodenal fistula results.

Duodenal Fistula.—The most usual causes are as follows :

1. As a complication of partial or complete gastrectomy (see p. 331).
2. Opening an abscess connected with a perforated duodenal ulcer.
3. Traumatic rupture of the duodenum.
4. As a complication of transduodenal choledochotomy.
5. As a complication of right nephrectomy.
6. As a complication of right colectomy.

The duodenal contents well up on to the surface, and unless effective sump drainage is instituted early, pancreatic enzymes digest and excoriate the skin. When the fistula discharges copiously, unless measures are taken to prevent it by intravenous fluid therapy, dehydration, gross electrolyte imbalance and hypoproteinæmia will occur rapidly.

Other necessary precautions are to protect the skin by aluminium paint or barrier cream, and to guard against disruption of the abdominal incision (see Chap. 29). In cases of a small or moderate leak, these measures are followed by lessening of the discharge and spontaneous closure. When the discharge from the fistula is profuse, temporary jejunostomy, by allowing nourishment to be given below the level of the fistula, if performed early, will prevent general deterioration and aid in natural closure of the fistula. Alternatively the advisability of re-operation with an attempt to close the fistula after dissecting the duodenum more distally can receive consideration.

ACUTE VOLVULUS OF THE STOMACH

For axial rotation (fig. 517) to occur there must be considerable congenital or acquired lengthening of the gastric ligaments, particularly of the gastro-hepatic omentum. Torsion of the stomach, which is an extremely rare emergency, can occur in a horizontal or a vertical axis. Usually horizontal rotation is associated either with a diaphragmatic hernia or the lower pouch of an hour-glass stomach. Vertical rotation, which is rarer still, can occur only when extreme gastroptosis (fig. 518) is present. While volvulus of the stomach can occur at any age, in most of the reported cases the patient has been elderly. Almost always the volvulus is preceded by attacks of upper abdominal pain with distension accompanied by retching. Then one day, after a full meal, the volvulus fails to rectify itself, a large resonant swelling appears

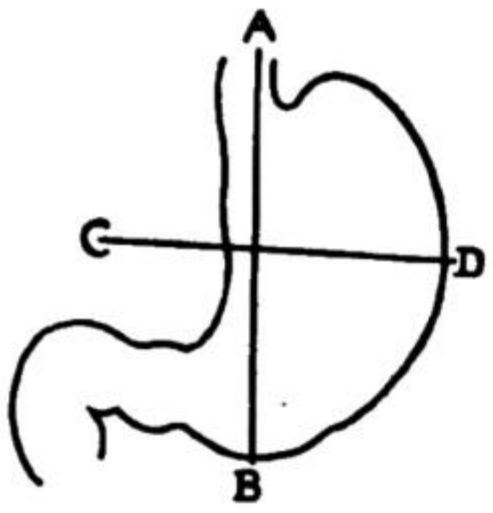

FIG. 517.—A–B, axis of vertical rotation. C–D, axis of horizontal rotation.

FIG. 518.—Profound gastroptosis. (*From an X-ray.*)

in the upper abdomen, there is vomiting, mostly froth, *without bile*, and considerable shock. The fact that (hour-glass stomach excepted) a gastric aspiration tube cannot be passed aids in the diagnosis : this fact, and the complete absence of bile in the vomits at once rules out acute dilatation of the stomach. Radiography reveals an exceptionally large gastric air shadow which, in the case of a diaphragmatic hernia, is often intrathoracic. Early operation with puncture of the stomach to let out the gas, suture of the puncture hole, untwisting of the stomach and the passage of a stomach tube (which remains in position for three or more days) is imperative. If operation is not performed early, the enormously distended stomach, with its blood-supply impaired by reason of the torsion, bursts.

ACUTE PHLEGMONOUS GASTRITIS
(*syn.* ACUTE SUPPURATIVE CELLULITIS OF THE STOMACH)

Both the anterior and posterior walls of the stomach are angry and swollen, and feel like wet blotting-paper : pus is present in its submucosal layer. In its most acute form phlegmonous gastritis invades the walls of the stomach from the cardia to the pylorus. There is a localised variety that, when situated in the distal part of the stomach, has been treated successfully by partial gastrectomy. The absence of characteristic signs and the rarity of the condition makes preoperative diagnosis impossible. Probably in some cases resolution occurs, and the true nature of the condition never comes to light. In the acute generalised variety, the diagnosis of either perforated peptic ulcer or (?) acute pancreatitis is made. At laparotomy the stomach is found to be inflamed and œdematous. If a hollow needle connected to an aspirating syringe is introduced into the submucosa, thick, muddy pus is withdrawn. A small incision is made through the muscle coats only. Drainage is provided to this locality and to the lesser sac. Post-operative treatment consists of the administration of fluids intravenously, complete rest of the stomach, gastric aspiration continuously or at very frequent intervals, and intense antibiotic therapy (penicillin and streptomycin), in which the main hope of recovery lies.

CONDITIONS THAT MIMIC CHRONIC PEPTIC ULCER

1. **Appendicular dyspepsia** is a great imitator of chronic *duodenal* ulcer. Many of the symptoms of appendicular dyspepsia are clearly due to an associated duodenitis, and bleeding from the duodenal mucosa occasionally occurs in these cases. The patient is often cured completely by the removal of the appendix, which in genuine cases is demonstrably diseased, and often contains pus. Most convincing evidence that this condition exists was a case seen by the author in the post-mortem room. In spite of transfusions, a man of twenty-two died of hæmatemesis and melæna. No ulcer could be seen macroscopically in the stomach or duodenum, but there was a retro-cæcal appendix full of pus. Histologically the duodenum was the seat of multiple erosion of its mucous membrane.

2. **Duodenal diverticulum** has been called the diagnostic scapegoat of the upper abdomen (W. W. Davey). In 95 per cent. of cases the presence of a duodenal diverticulum (disclosed during the course of a barium meal) is incidental, the symptoms being due to some other concomitant lesion. These remarks concern a primary or idiopathic diverticulum (fig. 519) arising on the concave side of the *second* part of the duodenum, usually near the ampulla of Vater. This is the common variety. In such cases the sac consists of mucous membrane covered by fibrous tissue, the muscular layers of the gut being completely, or almost completely, absent, which suggests that it is a

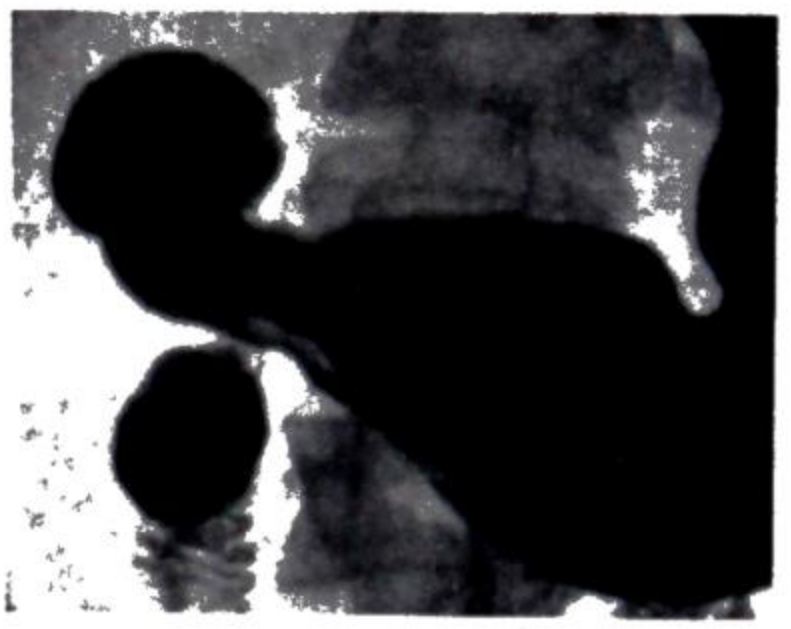

FIG. 519.—Typical duodenal diverticulum.

William Wilkin Davey, Contemporary. Professor of Surgery, University College Hospital, Ibadan, Nigeria.

herniation of mucous membrane related to a defect in the musculature along the course of blood vessels entering or leaving the duodenum.

On rare occasions a diverticulum of the *first* part of the duodenum occurs in connection with a long-standing duodenal ulcer, and the symptoms arise from the ulcer or from pyloric obstruction. On still rarer occasions a diverticulum is found in the *third* part of the duodenum, especially just proximal to the duodeno-jejunal flexure, in which case often the diverticulum is large, and in this instance, and in this instance only, it is frequently the cause of symptoms.

Diagnosis.—From the above remarks it will be clear that in so far as the cause of symptoms is concerned, a diverticulum of the second part of the duodenum must be regarded with scepticism. Culpability should be considered seriously only when (*a*) the diverticulum fails to empty after six hours, and (*b*) when it is palpated during a radiological examination. From time to time cases of perforation of a duodenal diverticulum have been reported.

Treatment.—When the diverticulum is causing symptoms and the relevant lesions have been excluded, excision of the diverticulum is indicated. In cases where the second part of the duodenum is involved the operation is very prone to be followed by a duodenal fistula, therefore drainage down to the vicinity of closure of the amputated sac must always be provided.

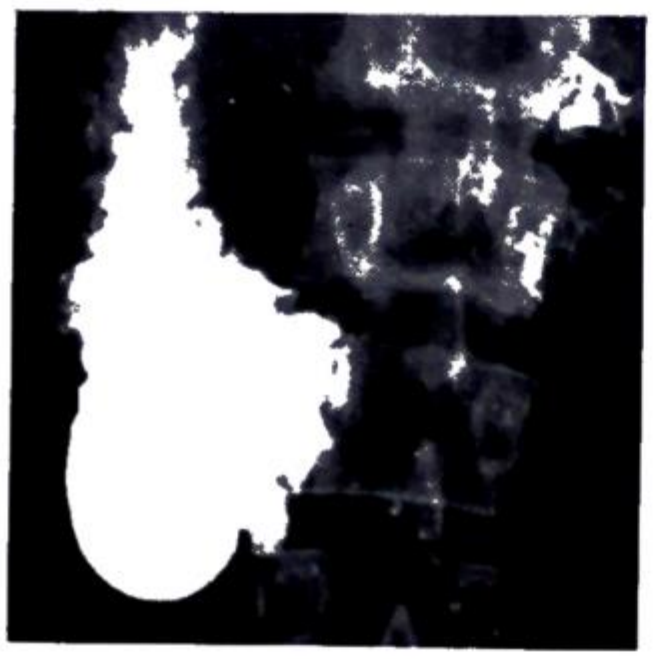

FIG. 520.—Duodenal ileus. Note the abrupt obstruction to the third part of the duodenum.

3. **Chronic duodenal ileus** is a rare condition, producing symptoms akin to those of pyloric stenosis. Women are the usual sufferers. The duodenum is found considerably dilated up to the point where the superior mesenteric vessels cross it (fig. 520).

When the symptoms are persistent, anastomosis of the lowest part of the dilated duodenum to an adjacent loop of jejunum (duodeno-jejunostomy) is the best course (Sir David Wilkie), but the results are often disappointing.

4. **Prolapsing Gastric Mucosa.**—Some deny the very existence of this clinical entity, but reports of series of cases occurring in many parts of the world continue to be published. Hypertrophied gastric mucosa prolapses through the pylorus (fig. 521 (inset)), thereby causing partial pyloric obstruction. This condition does not give rise to pathognomonic symptoms, but a long history of dyspepsia comparable with that of a pyloric ulcer is usual. Prolapsing gastric mucosa should be suspected when the barium meal displays considerable delay (six hours) in emptying, combined with a 'jockey-cap' residue in the duodenal cap (fig. 521), and a display of traceable mucosal patterns as the stomach eventually empties. Partial gastrectomy gives excellent results (L. H. Appleby).

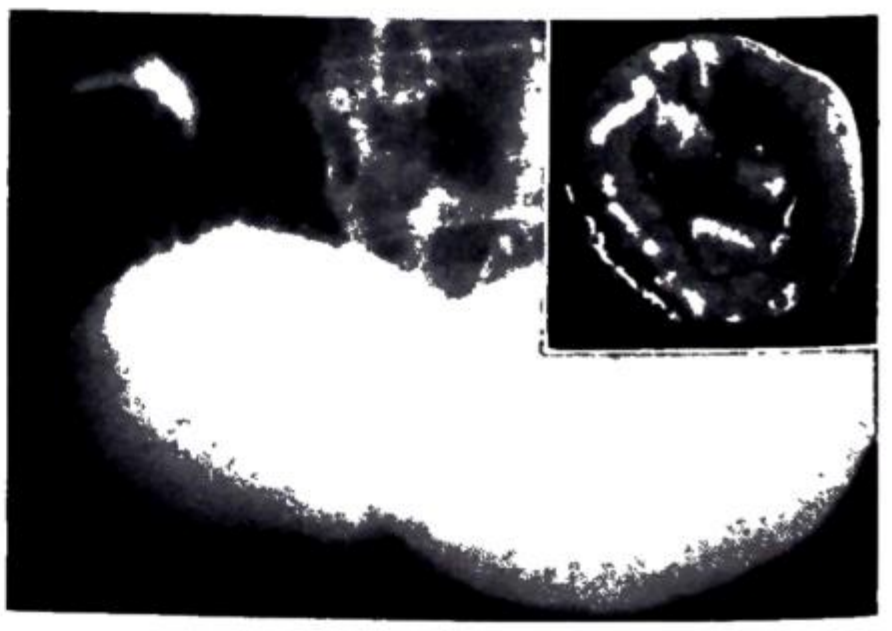

FIG. 521.—Showing the 'jockey-cap' deformity. Inset.—The prolapse viewed from the duodenal aspect of the pylorus. (*Mr. L. H. Appleby, Vancouver.*)

5. **Syphilis of the stomach** produces thickening of the wall and mucosal ulceration, with symptoms and X-ray findings identical with those of peptic ulcer or cancer. The differential diagnosis is particularly difficult because ulcer or cancer of the stomach may be present in a patient with a positive Wassermann reaction (K. A. Meyer).

6. **Tuberculosis of the Stomach.**—The stomach is very seldom affected by tuberculosis, and this has been attributed to the bactericidal action of hydrochloric acid and pepsin. For a concrete diagnosis of gastric tuberculosis, tubercle bacilli must be demonstrated in the lesion.

Sir David Wilkie, 1882–1938. Professor of Clinical Surgery, University of Edinburgh.
Lyon H. Appleby, Contemporary. Chief Surgeon, St. Paul's Hospital, Vancouver, British Columbia.
August von Wassermann, 1866–1925. Director of the Institute for Experimental Therapy, Berlin.
Karl A. Meyer, Contemporary. Senior Surgeon and Medical Superintendent, Cook County Hospital, Chicago.

CONDITIONS THAT MIMIC GASTRIC NEOPLASM

1. **Atrophic Gastritis of Pernicious Anæmia.**—Patients with pernicious anæmia often have symptoms identical with those of carcinoma of the stomach, and their gastric juice shows an absence of HCl. The upper two-thirds of the walls of the stomach are reduced to parchment-like thickness, the change terminating abruptly at the commencement of the pyloric antrum. So striking and so constant is this regional atrophy that the stomach removed at necropsy from a patient who has died from pernicious anæmia can be recognised by naked-eye appearance alone (H. A. Magnus). Microscopically, all that is left of the mucosa is the surface epithelium and a few scattered glands. The oxyntic and peptic cells have disappeared entirely.

See also Gastric Biopsy, p. 353 and Gastric *Adenomatous* Polyposis, p. 349.

2. **Giant rugal hypertrophy of the stomach** (*syn. polyadenome en nappe*) is more common on the continent of Europe than in the British Isles. This is a condition in which gastric rugæ, especially towards the greater curvature, are so enlarged (fig. 522) by hypertrophy of their glands that they have been likened by the French to cerebral convolutions. The importance of this condition is that it is confused with lymphosarcoma or diffuse carcinoma, and so is subjected to unnecessary resection of a large part, or all, of the stomach. The thick folds can be demonstrated radiologically, seen at gastroscopy, and palpated at laparotomy. A test meal usually reveals a high acidity. Actually there is no need for operative treatment, but as a result of a barium meal, not infrequently the condition is reported as being due to an extensive carcinoma of the stomach, and unless the operator is familiar with the feel—so aptly likened in shape and consistency to sausages—he embarks upon a wide resection of the stomach. In cases of doubt, a small incision in the stomach will clinch the diagnosis. Unlike gastric adenomatous polyposis (see p. 349), there is not the slightest evidence that this condition is premalignant, nor is it inflammatory. The ætiology is unknown.

FIG. 522.—Giant rugal hypertrophy of the stomach. (*H. D. Johnson and A. Stansfeld.*) (*British Journal of Surgery.*)

Henry A. Magnus, Contemporary. Director of the Pathology Department, King's College Hospital, London.

CHAPTER XX

THE SPLEEN

HAMILTON BAILEY

Surgical Physiology.—The functions of the spleen are :

1. **Destruction of effete erythrocytes** and possibly of platelets. It is believed that during their sojourn in the spleen the lipoid coverings of ageing erythrocytes are rendered less strong. At least some effete red cells are disintegrated in the spleen, for fragments of them have been observed within the splenic phagocytes. After splenectomy the liver, bone marrow and hæmolymph nodes perform these duties.

2. **Formation of erythrocytes and lymphocytes** during fœtal life, infancy and childhood. In case of need, this function is resumed during adult life.

3. **Maintenance of a Reserve of Erythrocytes.**—Anoxia (*via* the splanchnic nerves) stimulates the spleen to contract, thereby causing reserve red cells idling in its meshwork to be put into circulation as oxygen-carriers. It is questionable if the human spleen is sufficiently contractile for this function to be of the same importance as it is in some animals.

4. **Regulation of iron metabolism and iron storage.**

5. **Phagocytosis of foreign substances** by the reticulo-endothelial cells of the spleen is well illustrated by the high concentration of opaque substance in the spleen in patients who have received an injection of thorium. This function is also evident in the lipoid dystrophies, when phagocytosis of abnormal lipoids is the main cause of splenic enlargement.

6. **Protection against Certain Toxins.**—It would appear that the spleen plays a part in the formation of antibodies. This is borne out by the fact that a splenectomised human being fails to produce antibodies in response to antigens administered intravenously. There is increasing evidence that splenectomised children are less able to combat severe infection than those with a normal spleen. Young splenectomised persons require close observation so that immediate and energetic steps can be taken in cases of pyogenic infection.

7. **Hormone Regulation of the Marrow.**—There is some evidence that the spleen secretes a hormone that inhibits the production of thrombocytes and leucocytes by the bone marrow. W. Dameshek considers excess of this hormone to be the cause of thrombocytopænic purpura and splenic neutropænia.

8. **Splenin A,** a substance elaborated by the spleen and having anti-inflammatory properties, has been isolated (G. Ungar). It is probable that the virtue of cortisone in the treatment of rheumatoid arthritis and other collagen diseases is due to the stimulating effect of the steroid on the production of splenin A by the spleen.

PHYSIOLOGICAL EFFECTS OF SPLENECTOMY

1. **Spleniculi** (accessory spleens), if present, **hypertrophy.**

2. **Bone Marrow Changes its Character.**—Within six months red marrow replaces yellow marrow in many of the long bones (Rendle Short). This accounts for fleeting bone pains ' like rheumatism ' that are sometimes a matter of serious concern to the patient.

3. **Changes in the Blood.**—*Initial changes:*

(*a*) Leucocytosis
(*b*) Erythropenia
(*c*) Diminution in hæmoglobin
(*d*) Increased platelet count.
(*e*) Increased coagulability.

} These changes reach their zenith between the second week and the second month after splenectomy.

A characteristic morphological change occurs in some of the erythrocytes, viz. the presence within them of Howell-Jolly bodies. These are rounded structures usually

William Dameshek, Contemporary. Physician, New England Centre Hospital, Boston, Mass., U.S.A.
Georges Ungar, Contemporary. Director, Department of Pharmacology, U.S. Vitamin Corporation.
Arthur Rendle Short, 1880–1953. Professor of Surgery, University of Bristol.
William Henry Howell, 1860–1945. Professor of Physiology, Johns Hopkins University Medical School, Baltimore.
Justin Jolly, 1870–1953. Professor of Histophysiology, Collège de France, Paris.

occurring singly, and placed eccentrically, that stain in the same way as nuclei. They are thought to be fragmented nuclei.

After two months lymphocytosis is present invariably.

After many months there is moderate eosinophilia, and the mast cells increase in number.

4. **Undue Excretion of Iron by the Kidneys.**—Therefore the patient should take a preparation of iron.

SPLENICULI (*syn.* ACCESSORY SPLEENS)

Instead of a spleen, some fishes are provided with islets of splenic tissue scattered through the cœlom. In man accessory spleens, in common with the main organ, are derived from the dorsal mesogastrium. One or more is present in 25 per cent. of children (J. Jolly), but they are to be found in only 11 per cent. of adults (J. G. Adami). Obviously, then, as maturity is reached accessory spleens tend to disappear. Their usual locations are as follows :

1. In contact with the spleen (fig. 523), usually near the hilum : more than 50 per cent. are found in this situation.

2. Related to the splenic vessels and behind the tail and body of the pancreas—about 30 per cent.

3. The remainder are found in the splenic ligaments, the greater omentum, mesentery, mesocolon, and abutting the left ovary or testis (carried thither during descent of the gonad). From time to time a hypertrophied spleniculus gives rise to an intrascrotal tumour.

FIG. 523.—Spleen with a spleniculus.

When splenectomy has been performed for conditions that are known to be cured by removal of the spleen, return of symptoms after an interval of freedom is the result of hypertrophy of (usually) one accessory spleen. Occasionally a morsel of the spleen left attached to the pedicle during splenectomy continues to grow, and may be said to form a new spleen. Therefore, not only must the whole of the enlarged spleen be removed, but, except in cases of rupture, if the condition of the patient permits, the sites of an accessory spleen should be examined meticulously, and if one or more spleniculi are found, they must be extirpated.

Accessory spleens can be sought pre-operatively by radiography following an intravenous injection of colloid thorium.

Splenosis is a rare condition, and nothing to do with spleniculi. Fragments of splenic tissue, spilled at the time of bygone rupture of the spleen, are found disseminated widely over the peritoneum, having thrived in situations other than those to be expected on developmental grounds. Sometimes there are scores of them. Splenosis does not give rise to symptoms, and is found only at laparotomy or necropsy.

SPECIAL METHODS OF INVESTIGATING THE SPLEEN

1. **Radiography.**—In plain films of the upper abdomen the spleen is visible in whole or part, especially when the stomach and the splenic flexure contain gas, and the patient is not obese. These findings, repeated at intervals, are good evidence that the spleen is unruptured (see p. 374).

John George Adami, 1862–1926. Professor of Pathology, McGill University, Montreal.

2. **Splenic Puncture.**—Provided the following stipulations are adhered to rigorously, splenic puncture can prove a most valuable diagnostic aid.

(*a*) Only definitely palpable spleens should be punctured.

(*b*) No patient with a hæmorrhagic tendency should be subjected to splenic puncture.

(*c*) The puncture should not be performed in the presence of infected splenomegaly, or of a tender spleen.

(*d*) Unconscious patients and children must never undergo splenic puncture (D. O. Ferris).

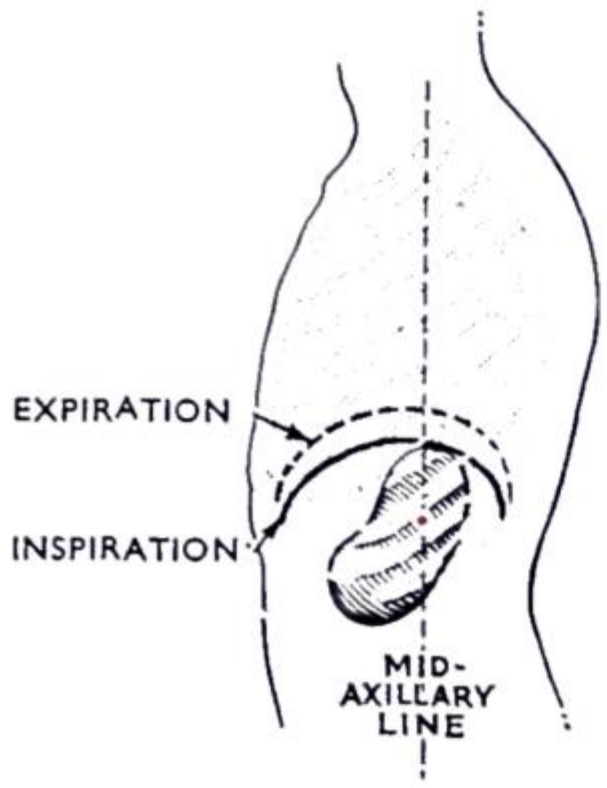

FIG. 524.—The puncture is made in the 9th intercostal space in the mid-axillary line during full inspiration. (*After D. O. Ferris.*)

Technique.—An ordinary lumbar puncture needle equipped with a guard to regulate the depth of the thrust should be employed. With the patient lying on his back, the site of puncture (fig. 524) is infiltrated with local anæsthesia. The puncture must be performed during full inspiration, with the guard set to the required depth. Strong aspiration with the syringe is performed for several seconds. Before the needle is withdrawn the negative pressure must be released. After splenic puncture the patient is kept in bed for six hours, as a precaution against hæmorrhage.

3. **Splenoportography** can be carried out under general anæsthesia prior to laparotomy, or under local anæsthesia. The needle is inserted as above. After withdrawal of the stilette of the lumbar puncture needle, blood drips from the lumen; only after this has been demonstrated is the dye-filled syringe connected, and 30–50 ml. of diodine injected *rapidly*. Apnœa, voluntary or controlled, must be maintained during insertion of the needle and throughout the injection of the dye. If this stipulation is not adhered to rigorously a laceration of the spleen as the spleen moves with the diaphragm, and possibly serious hæmorrhage, will result. Splenoportography permits visualisation of the spleen and the portal venous system. The first exposure is made towards the end of the injection, and three or four exposures follow subsequently.

CONGENITAL ABNORMALITIES OF THE SPLEEN

Congenital absence of the spleen is exceedingly rare, and when it occurs it is nearly always associated with a gross congenital cardiac abnormality (usually due to a septal defect) and also the presence of accessory lobes to the lungs, and sometimes lobulation of the liver. The condition can be suspected in a baby with cyanosis due to a cardiac lesion whose blood-count shows an excessive number of normoblasts. If some of these red cells contain Howell-Jolly bodies, the diagnosis is established. Owing to the associated cardiac lesion, the mortality among infants with agenesis of the spleen is very high.

Movable Spleen (*syn.* Wandering Spleen).—The spleen, well supported by its two ligaments (the gastrosplenic and lienorenal), and resting on a third (the costocolic), which acts as a shelf, is the one abdominal organ that maintains its correct anatomical position in general visceroptosis. Movable spleen is a congenital abnormality that occurs more often in women. The condition is of great rarity, and the diagnosis is seldom made correctly in the first instance. Movable spleen is sometimes associated with postural albuminuria owing to pressure on the left renal vein resulting in temporary congestion of the kidney.

Torsion of the splenic pedicle sometimes occurs in the wandering organ. The torsion may be acute or chronic, acute cases presenting the symptoms of an intra-abdominal catastrophe. Chronic torsion may result in atrophy of the spleen, and after a period of indefinite abdominal discomfort all symptoms abate.

Treatment.—A movable spleen giving rise to symptoms should be removed.

RUPTURE OF THE SPLEEN

Usually a ruptured spleen occurs as the result of a traffic or industrial accident of the crushing or run-over type. Blows on the abdomen or the

Deward Olmsted Ferris, Contemporary. Head of the Section of General Surgery, The Mayo Clinic, Rochester, Min., U.S.A.

left lower thorax and falls on to a projecting object are other sources of the violence. Especially when rupture of the spleen occurs as a result of a traffic accident, associated injuries are commonly present. The most frequent are: (1) Fractured rib; (2) Fractured extremity; (3) Injury to the left kidney; (4) Lacerated liver. A number of cases of spontaneous rupture of a normal spleen have been reported, but in all probability in some of these there has been an accident that has been forgotten or suppressed by the patient.

Cases of ruptured spleen may be divided into three groups:

1. **The Patient Succumbs Rapidly, Never Rallying from the Initial Shock.**—This type is comparatively rare in temperate climates; tearing of the splenic vessels and complete avulsion of the spleen from its pedicle[1] gives rise to the symptoms which characterise this group. In countries where malaria is rife, splenic rupture is often rapidly fatal, and advantage has been taken of this knowledge by murderers in China, who achieve their end by digging the victim beneath the left ribs with an implement known as a larang (fig. 525).

2. **Initial shock; recovery from shock; signs of a ruptured spleen** is the usual type seen in surgical practice. After the initial shock has passed off, there are signs which point to an intra-abdominal disaster, and by correlating these signs it is often possible to arrive at a correct pre-operative diagnosis.

FIG. 525. — A larang, which, being interpreted, means 'forbidden.' (*After J. Johnston Abraham.*)

General signs of internal hæmorrhage are inconstant. Perhaps the most helpful is increasing pallor and a rising pulse-rate.

Local Signs.—(*a*) Abdominal rigidity is present in more than 50 per cent. of cases; it is most pronounced in the left upper quadrant.

(*b*) Local tenderness is found constantly.

(*c*) Shifting dullness in the flanks is often present.

Ballance's sign is positive in about 25 per cent. of cases. There is a dull note in both flanks, but on the right side it can be made to shift, whereas on the left it is constant. The interpretation is that there is blood in the peritoneal cavity, but the blood in the neighbourhood of the lacerated spleen has coagulated.

(*d*) Abdominal distension commences about three hours after the accident, and is due to intestinal paresis, an early stage of paralytic ileus.

(*e*) Kehr's sign is pain referred to the left shoulder. There may be hyperæsthesia in this area. This sign is present very often especially if sought a quarter of an hour after elevation of the foot of the bed. Blood in contact with the under-surface of the diaphragm is the cause of the phenomenon.

(*f*) Rectal examination frequently reveals tenderness and sometimes a soft swelling, due to blood or clot in the rectovesical pouch.

(*g*) Very occasionally an umbilical 'black eye' (Cullen's sign) appears. The phenomenon is occasioned by a hæmoperitoneum of some standing from any cause.

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[1] Favoured by adhesion of the spleen to the diaphragm.

Sir Charles Ballance, 1856–1936. Surgeon, St. Thomas's Hospital, London.
Hans Kehr, 1862–1916. Surgeon, Halberstädt, later Berlin. He died of septicæmia contracted during his work.
Thomas Stephen Cullen, 1818–1953. Professor of Gynæcology, Johns Hopkins University, Baltimore, U.S.A.

3. **The Delayed Type of Case.**—After the initial shock has passed off, the symptoms of a *serious* intra-abdominal catastrophe are postponed for a variable period up to fifteen days, or even more. As a rule it is only a matter of minutes to an hour or so, during which time the patient often appears to have recovered from the blow. Thus a Rugby footballer has continued to play after a short rest, only to collapse later from internal hæmorrhage.

Considerable delay of serious intraperitoneal bleeding is explained in one of the following ways :

(*a*) The greater omentum, performing its well-known constabulary duties, shuts off that portion of the general peritoneal cavity in the immediate vicinity of the bleeding (see fig. 655(*c*), p. 502).

(*b*) A subcapsular hæmatoma forms and later bursts.

(*c*) Blood-clot temporarily sealing the rent becomes digested by escaping ferments from the lacerated tail of the pancreas. What happens rather frequently is that a patient with a suspected rupture of the spleen is taken into hospital : the symptoms abate, and in due course he is allowed up. Suddenly he collapses, often in the lavatory while straining at stool. At other times fresh hæmorrhage is heralded by a rising pulse-rate, increasing pallor, advancing to air-hunger and collapse. Such disasters can, and should, be prevented by careful re-examination and radiography, if necessary repeated.

Radiography.—A normal, well-outlined spleen is a reliable negative sign, but radiographs should be repeated if the decision not to operate *pro tem* is taken. Indeed, every patient with a left-sided thoracic injury, especially where a lower rib is fractured, should be kept under radiological observation. When there is delayed rupture, or when the symptoms and signs are atypical, radiography often can help considerably in diagnosis. In decreasing frequency, the radiological signs of ruptured spleen are : (1) Obliteration of the splenic outline ; (2) Obliteration of the psoas shadow ; (3) Indentation of the left side of the stomach air-bubble ; (4) Fracture of one or more lower ribs on the left side (present in 27 per cent. of cases) ; (5) Elevation of the left side of the diaphragm ; (6) The presence of free fluid between gas-filled intestinal coils.

Treatment of Rupture of the Spleen.—Immediate laparotomy and splenectomy is the only reliable course. Blood is mopped up and, after injury to other viscera has been excluded, the abdomen closed completely. When circumstances permit, blood transfusions should be given before, during, and after the operation. In the absence of stored blood, auto-transfusion at the time of the operation is indicated.

Auto-transfusion.—Extravasated blood is collected from the peritoneal cavity by means of a suction apparatus. After mixing it with 3·8 per cent. sodium citrate solution, 2 ounces (60 ml.) in 1 pint of blood (568 ml.), it is strained through several layers of sterile gauze and gravitated into a vein. We have carried out the procedure on numerous occasions, and never with regret.

The results of timely operation for traumatic rupture of the spleen are excellent.

When the organ is damaged by a stab wound or missile penetrating the left pleural cavity, access to the spleen is best obtained by excising the thoracic and diaphragmatic wounds, and enlarging the opening in the diaphragm.

Post-operative Complications.—From time to time the following complications occur after splenectomy for rupture of the spleen.

Acute Dilatation of the Stomach.—This preventable complication should not be allowed to occur. Routine gastric aspiration for at least twenty-four hours post-operatively has banished it.

Left basal atelectasis, sometimes accompanied by left pleural effusion, is particularly common, and is due to bruising of the diaphragm at the time of the accident, or to trauma during splenectomy. Breathing exercises and forced coughing go a long way in preventing atelectasis.

Paralytic ileus (see Chapter 26) is rather common.

Peritoneal effusion is due to concomitant injury of the tail of the pancreas. The effusion lasts three or four weeks, and usually lessens gradually in amount. It is accompanied by slight pyrexia.

Wound disruption is liable to occur where the tail of the pancreas has been wounded, for escaping ferments digest the catgut in the abdominal wound. In order to forestall this complication, unabsorbable sutures should be used to repair the abdominal wall.

Persistent hiccough is the result of irritation of branches of the left phrenic nerve on the under-surface of the left side of the diaphragm.

Transient haematemesis from ligation of the vasa brevia veins.

RUPTURE OF A MALARIAL SPLEEN

As has been mentioned, in tropical countries this is a frequent catastrophe. The delayed type of rupture (following a trivial injury) is also very common, and the patient is admitted with a perisplenic hæmatoma (fig. 526). If splenectomy can be performed before the hæmatoma bursts into the general peritoneal cavity, the prognosis is less grave.

The operation is considerably more difficult than in the case of a ruptured normal spleen. Surgeons with tropical experience have surmounted these difficulties by ligating the splenic vessels as they run along the superior border of the body of the pancreas (see fig. 527), before disturbing the hæmatoma (A. T. Andreasen).

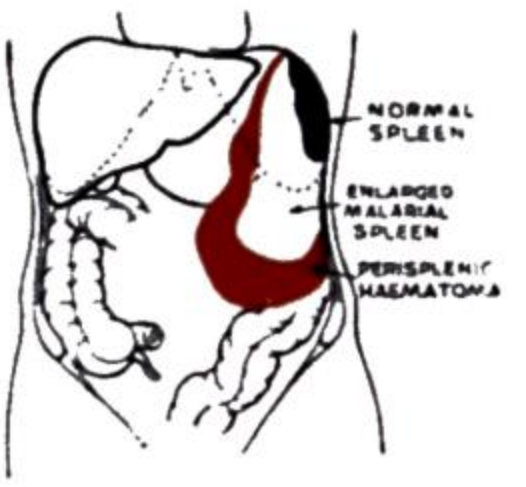

FIG. 526.—Normal spleen, malarial spleen, perisplenic hæmatoma. Note that the splenic contour is lost when the capsule has ruptured.

ANEURYSM OF THE SPLENIC ARTERY

Perhaps due to its tortuosity or to its liability to become arteriosclerotic (frequently arteriosclerosis affects the splenic artery and no other), the splenic artery is more frequently the site of an aneurysm (fig. 527) than any other artery in the abdomen, the abdominal aorta excepted. Even so, the condition is very rare.

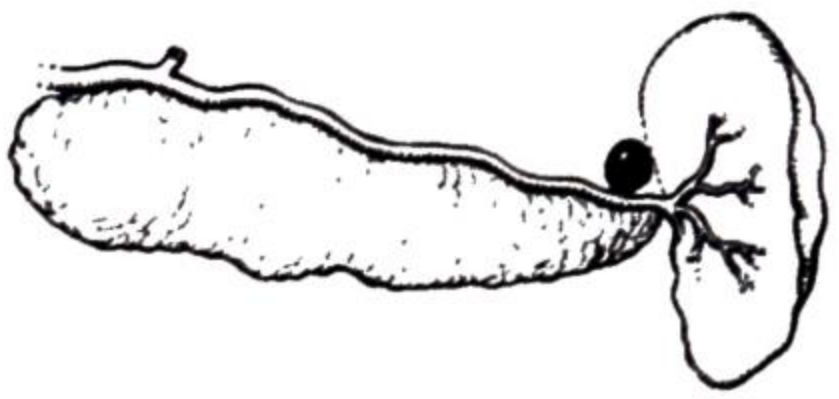

FIG. 527.—Usual position of a splenic aneurysm. (*After O. E. Owen.*)

As a rule the aneurysm is symptomless unless (as occurs rather frequently) it ruptures. Exceptionally it gives rise to upper abdominal pain, in which event the condition has been diagnosed pre-operatively: (*a*) By a bruit over the left hypochondrium; (*b*) By radiography, concentric calcification being present in the walls of many splenic aneurysms of some standing: many aneurysms of the splenic artery, as a rule no more than 1–3 cm. in diameter, have been discovered in the course of routine radiography. An unruptured splenic aneurysm has also been discovered at operation, usually in the course of an operation upon the stomach.

Rupture of the aneurysm occurs somewhat more often in women than in men, and it is noteworthy that approximately 25 per cent. of the female patients are pregnant, and usually the pregnancy is advanced (six to eight months). In most instances rupture occurs into the peritoneal cavity, and the symptoms are precisely those of delayed

Anthony Turner Andreasen, Contemporary. Formerly Professor of Surgery, University of Calcutta.

rupture of the spleen (see p. 374), although in relevant cases ruptured ectopic gestation will receive prior diagnostic consideration. Attention is drawn to the fact that during the stage of perisplenic hæmatoma (as in spontaneous rupture of the spleen, bursting often occurs in two stages) either or both the diagnostic signs set out above frequently are present. Hæmorrhage into the stomach or the retroperitoneal tissues has been recorded.

Treatment.—So dangerous is an aneurysm of the splenic artery that unless the patient is very old or infirm, excision of the sac should be advised in all cases where the lesion is discovered radiologically or at operation for another condition. In cases of rupture of the aneurysm urgent laparotomy and blood transfusion are required. The lesser sac must be opened and both the proximal and distal portions of the splenic artery (respectively entering and leaving the aneurismal sac) must be ligated securely. Proximal ligation alone is useless, for the distal end bleeds furiously. Splenectomy, although often performed, is unnecessary.

Taking into consideration that fatal cases are not often published, rupture of an aneurysm of the splenic artery carries a very high mortality.

INFARCTION OF THE SPLEEN (Fig. 528)

The patient, who is often suffering from subacute infective endocarditis or auricular fibrillation, is seized with agonising pain in the left hypochondrium. In a case seen by us, symptoms mimicked those of a perforated gastric ulcer, but the heart lesion helps the clinician to elucidate the diagnosis. When the embolus is aseptic the symptoms pass off in a few days. Infarct of the spleen has become a problem of air travel, especially in the U.S.A. At a height of between 10,000 and 15,000 ft. patients with sickle-cell anæmia (a disease confined to negroes) frequently sustain a splenic infarct.

FIG. 528.—Infarct of the spleen.

In bacterial endocarditis, when the bacteræmia cannot be subdued by antibiotic therapy, frequently the infection is maintained by an infected infarct or infarcts of the spleen. In such cases splenectomy has been tried, and in a few instances it has proved to be the deciding factor in curing the patient of a condition that is almost assuredly fatal.

PERISPLENITIS

Thickening of the splenic capsule has been noted at necropsy, usually in subjects with advanced hepatic cirrhosis. The best example of perisplenitis occurs in association with multiple serositis (Pick's disease). In this obscure malady the spleen appears as though covered with sugar icing. The covering is hard, structureless, and gives staining reactions similar to those of hyaline cartilage.

CYSTS OF THE SPLEEN

The majority of cysts of the spleen, which are very rare, arise in the lower pole, and in 75 per cent. of cases the patient is between ten and fifty years of age. There are several varieties.

1. *Single, usually unilocular, non-parasitic cysts* are of two varieties: (*a*) *True cysts*, comprise 10 per cent. of the total, and are dermoids or epidermoids. It is difficult to understand how the spleen, which is mesochymal in origin, should contain epithelium. (*b*) *False cysts*, which often reach a large size and are more common than true cysts. They have no lining membrane, and usually are due to past hæmorrhage, and contain blood-stained fluid and cholesterol crystals. Their origin is likely to be an ancient subcapsular hæmorrhage or degeneration of an infarcted area. In a number of cases there is a history of malaria.

2. *Small multiple cysts* are sometimes encountered at necropsy, and occasionally at operation. They have no clinical significance.

3. *Polycystic Spleen.*—Polycystic disease is sometimes confined to the spleen, but is more often associated with congenital cystic kidneys and liver. Occasionally the pancreas is similarly affected.

4. *Hydatid Cyst of the Spleen.*—A number of cases of hydatid cyst of the spleen have been reported.

A cyst of the spleen should be treated by splenectomy.

Friedel Pick, 1867–1926, described Pick's disease in 1896. He afterwards became Professor of Laryngology in Prague.

ENLARGEMENT OF THE SPLEEN

The spleen is a meeting-place of medicine and surgery. The following is a useful table of the causes of enlargement of the organ.

1. Infective	*Bacterial*—	Typhoid and Paratyphoid. Typhus. Anthrax. Tuberculosis.† Septicæmia. Weil's disease. Syphilis. Psittacosis. Abscess of the spleen.‡
	Protozoal—	Malaria. Egyptian splenomegaly.† Kala-azar.
	Parasitic—	Hydatid cyst.*
2. Blood diseases		Myeloid leukæmia. Lymphatic leukæmia.‡ Pernicious anæmia. Polycythæmia. Erythroblastosis fœtalis et infantium. Spherocytosis (acholuric familial jaundice).* Acquired hæmolytic anæmia.* Thrombocytopenic purpura.* Mediterranean anæmia.‡ Neutropenia.†
3. Metabolic		Rickets. Amyloid. Porphyria.‡ Gaucher's disease.*
4. Circulatory	Infarct—	Portal hypertension (see p. 415). Infective endocarditis.‡ Mitral stenosis.
	Occlusion of the portal vein —	Thrombophlebitis.† Neoplastic, e.g. carcinoma of tail of pancreas.‡
5. Collagen Diseases		Still's disease. Felty's syndrome.†
6. Non-parasitic cysts		Congenital.* Acquired.*
7. Neoplastic		Angioma. Primary fibrosarcoma.* Hodgkin's disease.‡ Lymphosarcoma.

* Cured by splenectomy. † Often benefited by splenectomy. ‡ Splenectomy sometimes indicated

In connection with the above Table, the following points should be noted. In thrombocytopænic purpura the spleen, although somewhat enlarged, is seldom palpable. In psittacosis the enlarged spleen can be palpated regularly after the first few days of the illness; this enlargement is helpful in differentiating the condition from other varieties of pneumonia. In portal hypertension the spleen is enlarged secondary to hepatic cirrhosis. The condition is therefore described in Chapter 21, 'The Liver.' Other causes of splenic enlargement of particular surgical importance will be considered in detail in the pages that follow immediately.

Adolph Weil, 1848–1916. Professor of Medicine, Heidelberg. Contracted laryngeal tuberculosis and subsequently resided in Italy.
Sir Frederick Still, 1868–1941. Physician, Hospital for Sick Children, London.
Thomas Hodgkin, 1798–1866. Curator of the Museum, Guy's Hospital, London.

ERYTHROBLASTOSIS FŒTALIS ET INFANTIUM

Icterus neonatorum is noticed in more than half of all newly born infants. It appears from the second to the fifth day after birth, and then fades gradually. This condition requires no treatment.

Icterus *gravis* neonatorum, as its name implies, is a serious malady. The baby is *born* jaundiced. The condition is due to immunisation of the mother to a dominant antigen factor present in the red blood cells of the fœtus inherited from the father. In 90 per cent. of cases the antigen is the rhesus factor. A Rh-negative mother is sensitised by antibodies from a Rh-positive fœtus or, less frequently, by a blood transfusion with Rh-positive blood which may have been given years before the pregnancy. The antibodies of the mother passing through the placenta violently hæmolyse the infant's red corpuscles. The first-born rarely suffers.

A proportion of infants die from erythroblastosis *in utero*; they are extremely anæmic and usually hydropic. Without treatment the mortality among infants with erythroblastosis who are born alive is very high (57 per cent.). Some die of cardiac failure within twenty-four hours; others become increasingly jaundiced, and die comparatively suddenly, about the third day, from fixation of bile-pigments in the basal ganglia (kernicterus).

Diagnosis.—Until comparatively recently the diagnosis was made by the infant being born jaundiced, or becoming jaundiced soon after birth. At the present time Rh-sensitisation of the mother can be detected during pregnancy by an examination of her serum for antibodies. In such cases delivery should be undertaken in hospital, where, if necessary, exchange transfusion can be undertaken, as a rule between four and six hours after birth. In such circumstances the mode of procedure is as follows: the umbilical cord is clamped about 6 in. (15 cm.) from the abdominal wall as soon as possible after delivery. Using a dry syringe and a No. 1 needle, the midwife or the doctor collects 10 ml. of blood from the umbilical vein on the maternal side of the clamp. Half the blood is transferred to a dry test-tube for serological tests, and half into a test-tube containing dry anticoagulant, for hæmoglobin estimation. The criteria upon which the necessity for exchange transfusion is based are:

1. A cord hæmoglobin[1] level of 14·8 G. per cent. (100 per cent. Haldane), or less.
2. A hæmoglobin level of 17·7 G. per cent. or less if the bilirubin content of the cord is 2·8 mg. per 100 ml. or more.

Compatibility Tests.—As a rule Rh-negative blood of the same ABO group as the child is used. When urgency or lack of expert laboratory assistance prevents the necessary tests being performed, Rh-negative group O blood can be used.

Blood for Exchange Transfusion.—In order to provide the maximum number of erythrocytes in exchange for the infant's impoverished blood, blood with a hæmatocrit raised to about 55 per cent. is highly desirable. Either a bottle of fresh blood is taken and centrifuged, or a bottle of blood two to five days old is selected; in either event 250 ml. of supernatent citrate-plasma is removed. For a baby weighing 8½ lb. (3·85 kg.) or under, two bottles of blood thus concentrated will be required.

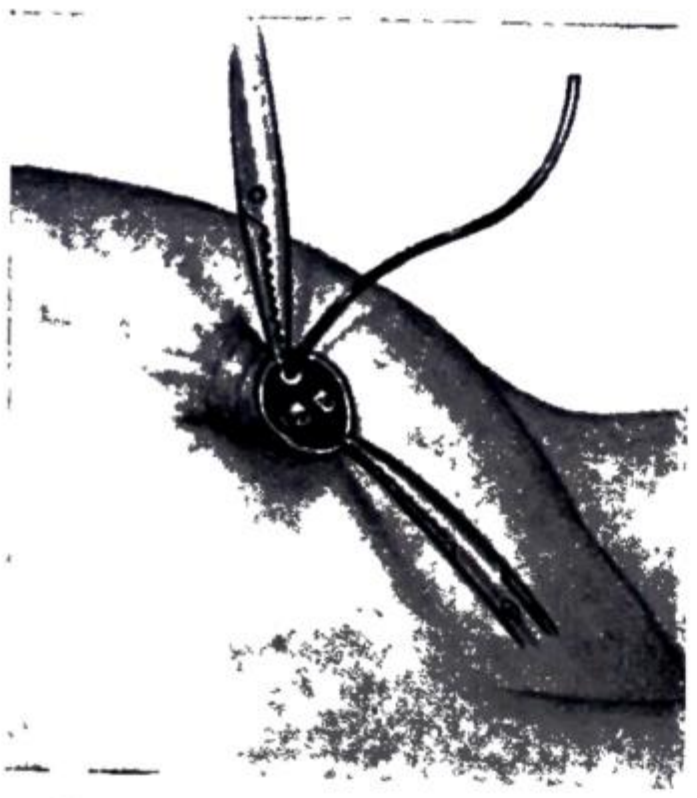

FIG. 529.—Cannulising the umbilical vein with polythene tubing.

Exchange Transfusion.—Usually the umbilical vein is cannulised with a polythene tube (fig. 529). In the rare event of the umbilical vein proving unsatisfactory, the long saphenous vein is substituted. Transfusion is effected by means of a siliconed two-way syringe, with tubing treated similarly. Oxygen should be administered during the transfusion, and subsequently if there is the least sign of cyanosis or respiratory distress. Exchange transfusion performed in this manner has reduced the mortality of erythroblastosis infantium to under 5 per cent.

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[1] There is a great difference between the hæmoglobin content of the cord and that of the peripheral blood.

John Scott Haldane, 1860–1936. Director of the Mining Research Laboratory, Birmingham University.

HÆMOLYTIC ANÆMIA

This disorder, which is due to a defect in the erythrocytes, presents in two forms : (1) congenital (hereditary spherocytosis) and (2) acquired.

1. **Hereditary Spherocytosis** (*syn.* Congenital Hæmolytic Anæmia; Acholuric Familial Jaundice).—Because of the existence of cases demonstrating neither anæmia nor jaundice, the designation hereditary spherocytosis is to be preferred. The defect can be transmitted by either parent as a Mendelian[1] dominant, with the result that males and females are affected equally.

Acholuric jaundice is jaundice without bile in the urine.[2] Spherocytosis is a term that gives a clue to the underlying pathology. The erythrocytes are smaller than normal, and instead of being biconcave, are biconvex. Not all of them are so shaped, but a proportion are, according to the severity of the condition. Biconvex (spheroidal) red cells burst more easily than normal red cells ; indeed, the biconvexity may be said to be the first stage of hæmolysis. Whereas the life of a normal erythrocyte is about one hundred days, that of a red cell in a case of hereditary spherocytosis is about fourteen days. Spheroidal red cells are *not* present in the circulating blood during the crises that characterise this disease (J. L. Emery).

Clinical Features.—Once the disease manifests itself, spontaneous remissions are almost unknown. As a rule the patient is pale and jaundiced, which varies in intensity and at its height is of a daffodil hue. On encountering a case of jaundice answering to this description, one of the first questions to ask the patient or his parent is "Are other members of the family affected similarly "? If the case is one of hereditary spherocytosis the answer is in the affirmative. In established cases lassitude and undue fatigue are seldom absent, but they vary with the amount of hæmolysis that is proceeding.

Sometimes the patient is born jaundiced, or becomes so early in life. In certain families the disease is characterised by severe crises of red blood-cell destruction ; thus, with the onset of a crisis, an erythrocyte count may fall from $4\frac{1}{2}$ millions to $1\frac{1}{2}$ millions in less than a week. Such crises are characterised by the sudden onset of pyrexia, abdominal pain, nausea, vomiting, and extreme pallor, followed by increased jaundice. These crises may be so severe as to cause death in infancy or childhood. Two elder brothers of a patient of mine with this condition died of the disease very early in life. More usually the jaundice, although variable, is very mild, and may not appear until adolescent or even adult life. In adult cases there is often a history of attacks of gall-stone colic ; indeed, 68 per cent. of untreated patients over the age of ten years have pigment stones in the gall-bladder. Every child with gall-stones should be investigated for evidence of hereditary spherocytosis.

On examination the spleen is large, and in thin subjects it can be palpated

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[1] In 1865 Mendel described 'dominant' and 'recessive' traits in hybrids. His work passed unnoticed for thirty-five years.

[2] Although there is excessive breakdown of red cells with transformation of liberated hæmoglobin to bilirubin, the bilirubin so produced is bound chemically to globin, and thus is not excreted by the kidneys.

Gregor Johann Mendel, 1822–1884. An Austrian monk and naturalist.
John Lewis Emery, Contemporary. Pathologist, Children's Hospital, Sheffield.

easily. Sometimes the liver is also palpable. Chronic ulcers of the legs are a common occurrence in adult sufferers.

Hæmatological Investigations.—There are two pathognomonic laboratory tests for this condition, to which may be added a third :

The Fragility Test.—Increased fragility of erythrocytes characterises this disease. Normally, erythrocytes begin to hæmolyse in 0·47 per cent. saline solution. In this condition hæmolysis occurs in 0·6 per cent., or even in solutions which more nearly approximate physiological saline.

The Reticulocyte Count.—To compensate for the loss of erythrocytes by hæmolysis, the bone marrow discharges into the circulation immature red cells, which differ from the adult cells by possessing a reticulum. This cannot be seen in the usual blood films, but can be demonstrated readily by vital stains. Crises are associated with reticulocytopenia, and *hypo*plasia of the erythroid element of the bone marrow. *After* a crisis the reticulocyte count is increased very much.

Fæcal urobilinogen is increased, as most of the urobilinogen is excreted by this route. Although technically difficult, this measurement is the best means of determining the extent of hæmolysis in cases of hereditary spherocytosis.

Treatment.—When a patient is so anæmic as to render blood transfusion advisable, more than ordinary precautions are required. Coombs' test (see p. 81) must always be performed—in *hereditary* spherocytosis it is negative (cf. *acquired* hæmolytic anæmia). While correctly-matched blood is usually well tolerated and the transfused red cells survive in patients whose disease is in remission, during a crisis transfusion increases the amount of hæmolysis. In such circumstances whole blood should be eschewed and a cautious transfusion of packed red cells attempted. As a rule packed red cells are accepted. However, after gravitating a few ounces of packed red cells very slowly, should the expedient seem perilous the risks of ligating the splenic artery, mainly under local anæsthesia, probably are less than chancing a severe hæmolytic reaction induced by unacceptable red cells.

All patients who have hereditary spherocytosis should be treated by splenectomy, for two reasons : (1) the danger of hæmolytic crises ; (2) the eventual development of gall-stones. The rationale of splenectomy is this : spherocytes are trapped in the spleen more readily than normal erythrocytes. Splenectomy removes the red cell 'trap,' with the result that the rate of hæmolysis is reduced sharply.

In the great majority of cases splenectomy can be undertaken as an elective operation during a remission. In juvenile cases the age at which operation is recommended has been decreasing.. If it is not imperative before, between the third and fourth years seems the optimum time, i.e. before gall-stones have had time to form.

At operation (splenectomy) the gall-bladder should be palpated for gall-stones, and if they are present cholecystectomy can be undertaken at a later date.

Following splenectomy, the patient can soon be rendered completely free from anæmia, which does not return. The jaundice disappears, but the

Robin Royston Amos Coombs, Contemporary. Veterinary Surgeon, Department of Pathology, University Cambridge.

tendency to hæmolysis persists ; it has been demonstrated twenty-five years after removal of the spleen. Ulcers of the leg due to this disease heal rapidly. In short (ruptured spleen excepted), in no other condition is splenectomy more triumphant. Owing to the absence of adhesions, the operation can be undertaken easily and expeditiously, which in part accounts for the very low operative mortality.

From time to time cases are encountered when a patient suffering from hereditary spherocytosis is admitted with obstructive jaundice due to pigment stones in the bile duct. In such cases the stones in the common bile duct must be removed by choledochotomy, but extirpation of the gall-bladder and the spleen is deferred until such a time when full recovery from the obstructive jaundice has occurred.

Acquired Hæmolytic Anæmia.—Whereas hereditary spherocytosis is characterised by an abnormality inherent in the erythrocytes, the acquired form is held to be due to abnormal immunological responses. Erythrocytes of the correct ABO group from normal donors are hæmolysed quickly by patients suffering from hæmolytic anæmia. Thus it has been postulated that the disease is acquired by the production of auto-antibodies that attach themselves to normal circulating erythrocytes and bring about their early dissolution. This phenomenon serves as a basis for the Coombs' test which is usually, but not uniformly, positive.

Clinical Features.—An onset commencing in middle or late life is suspicious that the disease is acquired ; the absence of a family history makes it almost certain. The spleen is palpable in 50 per cent. of cases, and in many of the patients with an enlarged spleen, the liver is palpable also. Sometimes there is a generalised enlargement of lymph nodes.

Laboratory Tests.—Examination of the peripheral blood reveals anæmia. Spherocytosis may or may not be present. As has been emphasised, the Coombs' test is nearly always positive, and when it is, this helps to establish the diagnosis.

Treatment.—As in this form of the disease spontaneous remissions are not uncommon, a trial of ACTH or cortisone is warranted. If a sufficiently good response is obtained, splenectomy can be postponed in the hope that the remission will be sustained. An additional advantage of taking this course is that there is some evidence that severe transfusion reactions—always a concern in this disorder—are reduced in number and severity in those who receive steroid therapy (F. A. Coller).

PURPURA

Purpura is a condition of great importance to the surgeon. It forcibly intrudes into his diagnostic arena ; on rare occasions it is the cause of an intussusception, but more frequently subserosal hæmorrhages produce signs similar to those of acute intestinal obstruction ; it may give rise to profound hæmaturia or alarming hæmorrhage from a mucous lining of the body, e.g. epistaxis, hæmatemesis, melæna or, in the case of young females (in whom the condition is particularly common), menorrhagia so severe as to render the

Frederick A. Coller, Contemporary. Professor of Surgery, University of Michigan, Ann Arbor, Michigan, U.S.A.

made after spreading the blood upon a cover slip spread previously with brilliant cresyl blue. The stained platelets can be identified easily.

As a rule the clotting time is normal, but (as a result of deficiency of platelets) clot retraction is delayed or absent—a most significant finding. If the anæmia is out of proportion to the loss of blood, the diagnosis of thrombocytopænic purpura should be questioned.

Sternal Puncture.—The bone marrow shows megalokaryocytosis with absent platelet budding.

The behaviour of the disease in adults is dissimilar to that in children.

In Children.—After one acute episode consisting of cutaneous purpura, often accompanied by epistaxis and bleeding from the oral mucous membrane, the disease undergoes a spontaneous cure in 75 per cent. of cases.

In Adults.—Although the initial attack is seldom as severe as in children, the disease tends to become cyclic, and often the relapses are of increasing severity, uterine or gastro-intestinal hæmorrhages being much in evidence. This difference has an important bearing on treatment.

Treatment :

In Children.—As dreaded intracranial hæmorrhage is exceptional, and for reasons given above, a conservative policy can be followed and, if necessary, continued for three months. No active treatment is given if the symptoms are mild, but should the bleeding be severe or persistent, in addition to maintaining a safe hæmoglobin level a course of ACTH and cortisone often effectively controls the hæmorrhagic manifestations. Such a course lasts two or three weeks. Splenectomy is advised in (1) severe cases ; (2) when relapses occur ; (3) especially in girls approaching the menarche. At least two days' hormone therapy should be given prior to operation. The results of splenectomy are all that can be desired, in spite of the fact that after an initial rise, the low platelet count persists. If, after splenectomy, relapses occur, an overlooked accessory spleen should be suspected.

In Adults.—Steroid therapy, if necessary combined with blood transfusion, should be employed in preparation for splenectomy.

Blood transfusion improves the hæmostatic state for a few hours without necessarily increasing the platelet count. The volume of any one transfusion should not exceed 1 litre, because massive and rapid transfusions often aggravate the thrombocytopænia and the bleeding tendency. For the same reasons repeated transfusions are also contraindicated. Fresh blood is essential, and vein-to-vein transfusion with siliconed syringes and tubes is undoubtedly the best method of blood transfusion in this instance. Some centres hold stocks of platelet concentrates, and transfusion of platelets, repeated as necessary, is a procedure of great promise.

Cortisone Therapy.—Unless the patient already has lost much blood, cortisone therapy frequently so controls the hæmorrhage as to render blood transfusion unnecessary. Its mode of action appears to be that of rendering the capillaries less permeable, and it also results in a reduction of the time of clot retraction, but without a change in the platelet count.

Primary.—*Thrombocytopænic*[1] *purpura* is a relatively frequent hæmatological disorder that manifests itself by hæmorrhages of all grades of severity. At one end of the scale there is merely a tendency to bruise easily; at the other the disease is fulminating in its onset, and disastrous in its consequences.

Ætiology.—For many years the cause of this condition has been disputed. Some authorities maintain that the reduced number of blood-platelets is the result of diminished production by megalokaryocytes[2] of the bone marrow; others regard increased destruction of blood-platelets, particularly by the spleen, as the predominant abnormality.

Clinical Features.—The disease can occur at any age, but it is observed principally in youth; before the age of puberty it is divided evenly between the sexes, but above that age there is a preponderance of five males to one female (Evans). Its various manifestations are as follows:

1. Ecchymoses (see fig. 530) or purpuric patches in the skin and mucous membrane. There is a tendency for these lesions to appear in the more dependent areas, because of the higher intravascular pressure in dependent parts.

2. Sustained bleeding from wounds, which in themselves are often trivial.

3. Bleeding, frequently serious, from mucous membranes—epistaxis, bleeding from the gums and, in women during the period of reproductive life, menorrhagia, are of frequent occurence. Urinary and gastro-intestinal hæmorrhages are less common.

4. Intracranial hæmorrhage occurs rarely, and not until other sites of hæmorrhage have given ample warning of the existence of thrombocytopænic purpura. Nevertheless, intracranial hæmorrhage is the most frequent cause of death in idiopathic thrombocytopænic purpura occurring in adults. This tragic complication can be averted by timely splenectomy.

5. Hæmarthrosis is rare (cf. Hæmophilia, p. 88).

Cerebral Purpura.—Usually the onset is sudden, often with pyrexia. The leading features are thrombocytopænia with cutaneous hæmorrhages and neurological lesions attributable to involvement of cortical cerebral vessels. As a rule the patient lingers for a few weeks; in a few instances partial or complete recovery has occurred.

On Examination.—Except for cutaneous ecchymoses or a positive tourniquet test, usually physical examination is unrevealing. In only a quarter of the cases is the spleen palpable, and in these it extends but little below the costal margin. Pronounced splenic enlargement makes the diagnosis of thrombocytopænic purpura highly improbable.

Blood Examination.—The bleeding time is prolonged. The most striking and important finding is that the blood-platelet count[3] is greatly reduced (usually to below 70,000 per cu. mm.)—in some cases to zero.

The counting of platelets is technically difficult, and the reading is vitiated by profound hæmorrhage, which renders the count relatively high. The count is best

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[1] Thrombocytopænia = poverty of thrombocytes (blood platelets).

[2] Megalokaryocytes (*syn.* megakaryocytes), the giant cells of bone marrow, give origin to blood-platelets.

[3] The normal blood-platelet count is 250,000 to 400,000 per cu. mm.

Lord Evans, Contemporary. Physician, The London Hospital.

Initially 300 mg. of cortisone is given by mouth in divided doses during the first twenty-four hours, 200 mg. during the second twenty-four hours: and 100 to 150 mg. daily thereafter for two or three weeks. In preparing the patient for operation the dose of cortisone is decreased in the immediate five days prior to the operation, substituting an equivalent amount of ACTH. No cortisone is given two days pre-operatively, but it is given immediately post-operatively, and continued for about a week in decreasing doses.

Splenectomy.—In slightly more than half the cases the bleeding tendency is controlled by the above measures, the operation can be converted from an emergency procedure to one of election which, being unhurried, permits a thorough search for accessory spleens.

In the remainder, operation must be undertaken with as little delay as possible. In the majority of these cases splenectomy can be performed with ease, because of the absence of adhesions, but in patients who are very ill ligation of the splenic artery must suffice. During the operation, prior to either of these procedures, the injection of adrenaline into the splenic artery causes a considerable shrinkage of the spleen, and it is said to squeeze out platelets contained in that organ (A. C. Perry).

Splenectomy in primary thrombocytopænic purpura is followed by good results in over 80 per cent. of cases, in spite of little change in the platelet count after an initial rise lasting about a week.

Treatment of Secondary Purpura.—Obviously, if the cause is known and can be removed, considerable faith can be placed in conservative measures. Cortisone and ACTH are effective in producing a remission in most instances. Up to a few years ago it was thought that splenectomy was contraindicated in secondary purpura, but a number of cases of cure by splenectomy have been reported recently. Particularly encouraging have been cases where the purpura is due to splenic tuberculosis and in rare examples of Boeck's sarcoid. If the bleeding is so profuse or prolonged as to tax the resources of the blood bank, splenectomy should not be delayed (A. G. Lowdon). In most instances, however, with steroid therapy it is safe to wait for a remission. Half of the cases of secondary purpura are benefited by splenectomy.

MEDITERRANEAN ANÆMIA (*syn.* COOLEY'S ANÆMIA; THALASSÆMIA[1])

Cooley's anæmia is a hereditary disease due to a defect in pigment anabolism resulting in failure to synthesise an adequate amount of hæmoglobin.

Originally thought to be confined to those who dwelt near the Mediterranean Sea, or who had a Mediterranean heritage, it is now known that this is only partially true, for scattered reports from various parts of the world show that the disease occurs from time to time in families who have no such heritage; for instance, a number of cases have been reported in natives of Thailand. The condition is inherited. In a severely infected infant both parents show characteristic blood changes. While the mild form is asymptomatic and can be detected only by an examination of blood, severe forms can be suspected by the following characteristics:

Clinical Features.—The patient is of short stature and the facies are reminiscent of a congenital syphilitic; the skull is large and bossed, and the bridge of the nose is depressed (see fig. 31, p. 32). In addition the maxillæ are prominent, as also the upper front teeth. There is a mongoloid slant of the eyes. The complexion is

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[1] Thalassæmia—Greek, Thalassa = Sea. (Because it occurs in persons of Mediterranean stock.)

Alan Cecil Perry, Contemporary. Senior Surgeon, The London Hospital.
Andrew Gilchrist Ross Lowdon, Contemporary. Professor of Surgery, University of Durham, Newcastle-upon-Tyne.
Thomas Benton Cooley, 1871–1945. Professor of Paediatrics, Wayne University College of Medicine, Detroit.

muddy; the conjunctivæ have an icteric tinge. Especially in a young child, the abdomen is prominent because of an enlarged spleen and liver. In patients with moderate anæmia these signs are correspondingly less, and in mild cases all are absent.

Blood Examination.—Increased resistance of red cells to hæmolysis in hypotonic saline solution is *the* characteristic finding. It is always present in Mediterranean anæmia, irrespective of the severity or mildness of the disease. In the more severe forms, not only is there a diminished number of erythrocytes and a low hæmoglobin level, but nucleated red cells and other immature blood cells abound.

Radiography of the skeleton shows early rarefaction of all bones and late sclerosis of tubular bones.

Treatment.—In selected cases splenectomy reduces the number of transfusions required, and lengthens the life of the patient which otherwise (in severe cases) is short.

PORPHYRIA, WITH SPECIAL REFERENCE TO THE ABDOMINAL CRISES OF THE DISEASE

Porphyria is a hereditary error of katabolism of hæmoglobin in which porphyrinuria occurs. The abdominal crises, which are characterised by violent intestinal colic with constipation, are liable to be precipitated by the administration of barbiturates, to which these patients have an idiosyncrasy. The patient is anæmic, frequently suffers from photosensitivity, and in advanced stages of the disease neurological or mental symptoms (from damage to the brain) are often present. On examination the spleen will be found to be enlarged. On a number of occasions the splenic enlargement, which is usually well marked, has been overlooked and the abdomen has been opened on the diagnosis of intestinal or appendicular colic, with negative findings. Another manifestation of acute porphyria is spasmodic abdominal pain followed by jaundice. This is due to spasm of the common bile-duct and Oddi's sphincter.

Two methods of establishing the diagnosis are available :

The urine is sometimes normal in colour. Usually it is orange (which is often dismissed as 'concentrated'). If the specimen is left exposed to daylight for a few hours it becomes coloured amber, particularly near the surface, where it is exposed to the air. There are several conclusive laboratory tests for porphyrinuria.

Radiography of the abdomen. Serial X-ray films show areas of intestinal spasm causing short segments of gaseous dilatation of the small and large intestine, especially of the cæcum.

Treatment.—Often there is a striking decrease in the serum sodium level and the patient is improved considerably by infusion of normal saline solution with careful control of electrolytic balance. To relieve the abdominal pain pethidine is the best drug. If a sedative is required, paraldehyde should be given. Acute porphyria is a serious, and often fatal, disease and in every acute attack megimide (a barbiturate antidote) should be administered, irrespective of whether the attack is produced by barbiturates, or not (T. K. With). If the patient can be tided over the crisis, splenectomy is advised, and usually it proves curative.

EGYPTIAN SPLENOMEGALY (*syn.* ENDEMIC HEPATO-SPLENOMEGALY)

Although especially common amongst the fellaheen (peasants) who dwell in the delta of the Nile, this disease is by no means confined to Egypt. It is prevalent in many parts of Africa, and is met with in Asia and South America, particularly Venezuela.

Ætiology.—While not yet proved scientifically, the consensus of opinion is that in 75 per cent. of cases the disease is due to infestation by the *Schistosoma mansoni*, and the remainder by *Schistosoma hæmatobium*.

Pathology.—A diffuse periportal fibrosis of the liver (fig. 531) is the first manifestation. Bilharzia ova in various stages of degeneration can be found in the portal vein and its tributaries. Next enlargement of the spleen occurs, due, it is believed, to bilharzial toxins and the products of disintegrated worms

Ruggiero Oddi, 1845–1906. Surgeon and Anatomist, Rome.
Torben K. With, Contemporary. Svendborg County Hospital, Denmark.
Sir Patrick Manson, 1844-1922. Practised in Hong Kong; later Physician to the Dreadnought Hospital, Greenwich.
Theodor Maximilian Bilharz, 1825-1862. Professor of Zoology, Cairo.

and ova, all of which are filtered by the spleen, causing active hyperæmia and hyperplasia of its reticulo-endothelial elements. The splenic capsule becomes thickened, due to perisplenitis, and the splenic vessels are often tortuous and sclerotic. Diminished splenic contractility causes stagnation

FIG. 531.—Periportal fibrosis of the liver in Egyptian splenomegaly. *(Dr. Halawani, Cairo.)*

of blood in the spleen, and results in its further enlargement. About this time the enlarged liver commences to contract, and the degree of enlargement of the spleen is roughly proportional to the degree of fibrosis of the liver (A. Halawani). Advanced cases are accompanied by ascites.

Clinical Features.—Egyptian splenomegaly is encountered at all ages from ten to seventy years, frequently reaching its zenith in the fourth decade. Ninety per cent. of the patients are males. The course of the disease is characterised by three stages :

1*st Stage*.—There is gradual enlargement of the spleen, hypochromic anæmia, eosinophilia, leucopenia and a relative leucocytosis. The liver becomes moderately enlarged and is tender in 70 per cent. of cases. During this stage there is little or no disturbance of general health.

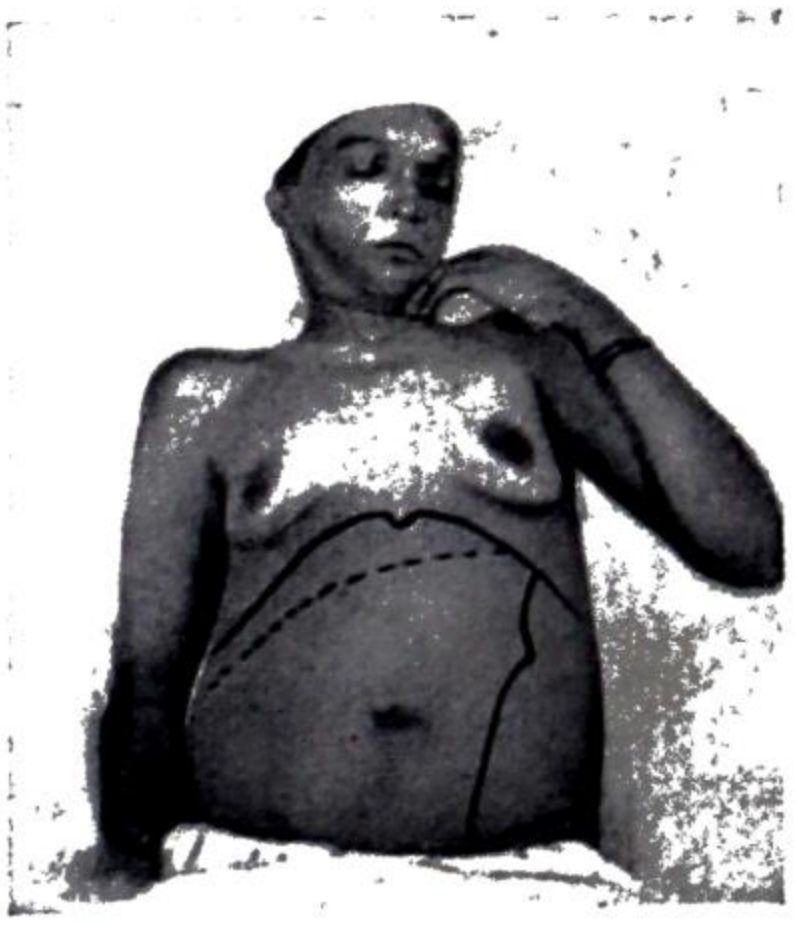

FIG. 532.—Egyptian splenomegaly. Successful splenectomy was performed. *(Owen Richards.)* *(British Journal of Surgery.)*

2nd Stage.—The spleen becomes further enlarged, extending to the pelvic brim (fig. 532). The liver gradually shrinks. The major symptoms at this time are :

Splenic pain or 'heaviness' .	90 per cent.
Weakness . . .	78 per cent.
Spasmodic abdominal pain .	75 per cent.
Diarrhœa (often blood-stained)	60 per cent.
Hæmaturia	38 per cent.
Hæmatemesis . . .	4 per cent.

The patient suffers from lassitude and easily falls a victim to intercurrent bacterial infections.

3*rd Stage*.—The liver atrophies more and more. Ascites supervenes. The superficial abdominal veins sometimes become dilated and tortuous. The general health deteriorates, and usually the patient dies in six to twelve months from the appearance of ascites. As a rule it takes eight to twelve years to pass from the first to the final stage.

Ahmed Halawani, Contemporary. Director of the Institute of Tropical Medicine, Cairo.

Laboratory Findings :

Schistosoma ova in the fæces	55 per cent.
Schistosoma ova in the urine	22 per cent.

Liver function tests reveal a varying degree of hepatic impairment. A hypochromic anæmia is always present.

Treatment.—Many early cases respond to the general treatment of bilharzia mansoni detailed in Chap. 28. In more established cases no substantial reduction in the size of the spleen results from a six-weeks' course of this treatment. It is in the latter class that, after careful pre-operative preparation, splenectomy with pre- and post-operative blood transfusions is undertaken under penicillin cover, and is often curative. The consensus of opinion in Egypt is that splenectomy should be performed in cases where there is no evidence of ascites, liver damage or severe renal insufficiency. Following splenectomy, over 70 per cent. of the patients are able to return to full work. When ascites has supervened, portacaval or splenorenal anastomosis (see p. 423) is the only form of treatment that can arrest the course of the disease.

FELTY'S SYNDROME

A moderate number of patients with chronic rheumatoid arthritis develop mild leukopænia; in a few of these the leukopænia becomes extreme and usually is associated with enlargement of the spleen. This combination is referred to as Felty's syndrome. A remarkable characteristic of this syndrome is that the leukopænia and splenic enlargement are apparently unrelated to the severity of the arthritic changes; indeed, in some instances the arthritis has commenced to improve or has become quiescent by the time the low white-cell count and the splenomegaly become unmistakable. In those cases in which the arthritis is slight but the splenic enlargement and the blood changes are much in evidence, a diagnosis of primary splenic neutropænia is sometimes made.

Clinical Features.—Sufferers from Felty's syndrome are very prone to pyogenic infections of all varieties, infected ulcers around the ankles being particularly frequent. They are fatigued easily, lean, and unable to gain weight. It is impossible to say whether these symptoms should be attributed to the arthritic process or to the leukopænia and splenomegaly. As a rule the degree of splenic enlargement is not great. Enlargement of lymph nodes and generalised brown pigmentation of the skin are sometimes present.

Blood Examination.—The white-cell count shows persistent leukopænia varying between 1,000 and 2,000 per cu. mm. with less than 20 per cent. of neutrophils. The greatest reduction is in the granulocytes. Neutrophils range from slightly below normal to virtually absent. There is almost always an associated anæmia of moderate severity. Achlorhydria is often present.

Sternal Puncture.—The bone marrow shows increased cellularity but a deficiency of mature granulocytes.

Treatment.—The results of splenectomy are variable. Usually there is an improvement in the blood picture with increased neutrophils, but this improvement is not maintained. However, the liability to infections seems to be decreased in many cases and rheumatoid arthritis that had become resistant to cortisone therapy reacts favourably to steroid therapy once more.

The Rôle of the Spleen in Rheumatoid Arthritis.—This opens up an important question regarding rheumatoid arthritis in general. As has been mentioned on p. 370, cortisone is believed to stimulate the output of splenin A, which is an anti-inflammatory factor. It has been shown that the spleen secretes a substance named splenin B that inhibits splenin A. In patients

August Roi Felty, Contemporary. Physician, Hartford Hospital, Hartford, Connecticut.

in whom cortisone is, or has become, non-effective, removing the factory for splenin B often renders the patient responsive to cortisone therapy once more. Work on these lines, which may prove to be an important discovery, is still proceeding.

OCCLUSION OF THE SPLENIC VEIN

Whether due to thrombophlebitis or to neoplastic infiltration of the splenic vein by a carcinoma of the body or tail of the pancreas (see p. 475), occlusion of the splenic vein (which gives rise to congestive splenomegaly) can be diagnosed only with certainty by splenoportography.

When the diagnosis is established, splenectomy is indicated, provided that in the case of a neoplasm, the neoplasm is operable.

GAUCHER'S DISEASE

As mentioned on p. 370, the spleen may take an active part in the storage of abnormal lipoids, as does the remainder of the reticulo-endothelial system. In the case of Gaucher's disease the lipoid in question is a cerebroside, kerasine. Gaucher's disease, which is rare, is characterised by enormous enlargement of the spleen, which may weigh 8 or 9 lb. (3·6 or 4·1 kg.). In the majority of cases the splenic enlargement begins in early childhood, often before the age of twelve, although the patient rarely seeks advice before adult life. Until the splenic enlargement becomes massive the symptoms are few. There is slight anæmia of the chlorotic type, a yellowish-brown discoloration of the skin of the hands and face due to a deposit of hæmosiderin, and a curious conjunctival thickening that help to clinch the clinical diagnosis. Slavonic and Jewish races appear to be more prone to this disease than the rest of humanity. The diagnosis is confirmed by finding Gaucher's cells in the bone marrow (fig. 533).

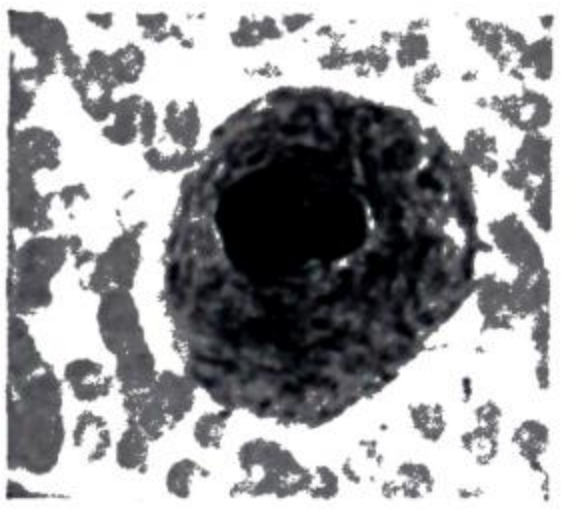

FIG. 533. — Typical Gaucher's cell from bone marrow obtained by sternal puncture. *(Dr. M. L. Thomson, Manchester.)*

Treatment.—Splenectomy rids the patient of a large abdominal swelling, but the operation is difficult because of perisplenitis and friability of the splenic pulp. It is widely held that the results of the operation are unsatisfactory because intercurrent infection, often tuberculosis, carries off the patient within two years, but recent reports show that several patients are in excellent health more than five years after splenectomy for Gaucher's disease.

FIG. 534.—Leukæmia. Showing the enlarged liver and spleen as displayed at necropsy. *(Dr. L. C. Hermitte, Sheffield).*

LYMPHATIC LEUKÆMIA

is of surgical importance in the differential diagnosis of enlarged lymph nodes and splenic enlargement. The diagnosis is made by an examination of the blood. The main treatment (and this is palliative) is deep X-ray therapy, although radioactive chromic phosphate (^{32}P) is now on trial. In the past, splenectomy was tried for this condition, and was found to be useless. Recently it has been suggested that splenectomy is helpful to those undergoing deep X-ray therapy, because after splenectomy the patient is able to retain red blood cells given by transfusion, which otherwise would be destroyed in the spleen. In this condition both the liver and the spleen are greatly enlarged (fig. 534).

Phillipe Charles Ernest Gaucher, 1854–1918. Physician to St. Louis Hospital, Paris.

TUBERCULOSIS OF THE SPLEEN

Tuberculosis of the spleen is not as uncommon as is sometimes believed. It occurs chiefly in adults between twenty and forty years of age. When a patient has splenomegaly with asthenia, loss of weight and an evening temperature, it is well to bear in mind the possibility of the enlargement of the spleen being due to tuberculosis. Too often these signs lead to the erroneous diagnosis of leukæmia or some other disorder for which splenectomy is not indicated. Occasionally tuberculosis of the spleen produces portal hypertension. Another form is cold abscess, which is very rare. Splenic puncture, followed by culture, or guinea-pig inoculation, will yield positive results. A therapeutic test with P.A.S. and streptomycin usually brings about some improvement, and there is less danger of dissemination of tubercle bacilli if splenectomy is undertaken. The operation, which usually is rendered difficult because of adhesions, is contraindicated only if other active tuberculous lesions are present. With this stipulation, the results of splenectomy in tuberculosis of the spleen are excellent (J. Patel).

ABSCESS OF THE SPLEEN

If a splenic embolus is infected, and the primary condition does not prove fatal, a splenic abscess may be expected to follow. Other sources of metastatic abscesses of the spleen are typhoid and paratyphoid fever, osteomyelitis, otitis media and puerperal sepsis. An abscess of the spleen can also occur by direct extension of infection from a diverticulitis or carcinoma of the splenic flexure of the colon. An abscess in the upper pole of the spleen may rupture and form a left subdiaphragmatic abscess. If the abscess is in the lower pole, rupture of it results in diffuse peritonitis. Primary splenic abscess of uncertain origin, containing foul gas and chocolate-coloured pus, occurs rather frequently in natives of Rhodesia.

Treatment.—As a rule, owing to dense adhesions, drainage of the abscess is the only course. Very rarely, it is possible to perform splenectomy with the abscess *in situ*.

NEOPLASMS

New-growths of the spleen are strikingly uncommon. Of the benign tumours, cavernous hæmangiomata and lymphangiomata have been encountered. Of the malignant, a few examples of primary fibrosarcoma have been described. Lymphosarcoma is somewhat more common. Metastases of carcinoma in the spleen are extremely rare. If it is conceded that Hodgkin's disease is a neoplasm, then this is the commonest cause of neoplastic enlargement of the spleen ; indeed, it is sometimes the leading sign of Hodgkin's disease. In primary neoplasms the treatment is splenectomy. In Hodgkin's disease with splenomegaly, splenectomy brings about a remission, sometimes up to three years.

SPLENECTOMY

The main indications for splenectomy are :

1. Rupture.
2. Movable spleen with symptoms.
3. Congenital spherocytosis.
4. Acquired hæmolytic anæmia.
5. Thrombocytopenic purpura.
6. Egyptian splenomegaly.
7. Felty's syndrome.
8. Gaucher's disease.
9. Cysts.
10. Primary new-growths.

From the surgical standpoint the spleen may be said to have two pedicles—the gastro-splenic omentum and the lieno-renal ligament ; the splenic artery and vein lie in the latter (fig. 535).

Technique of Splenectomy.—A left paramedian incision is made and the organ is palpated with special reference to adhesions. Division of adhesions on its convex aspect is effected at the expense of the diaphragm rather than of the splenic capsule. Once the organ is free, a hand passed between the spleen and the diaphragm eviscerates the spleen and rotates the

Jean Patel, Contemporary. Surgeon, Hôpital Tenon, Paris.

pedicle. A pack is placed in the splenic bed, and the splenic artery is identified and divided between ligatures. The splenic veins are then secured as close to the hilum as possible, to avoid injury to the tail of the pancreas.

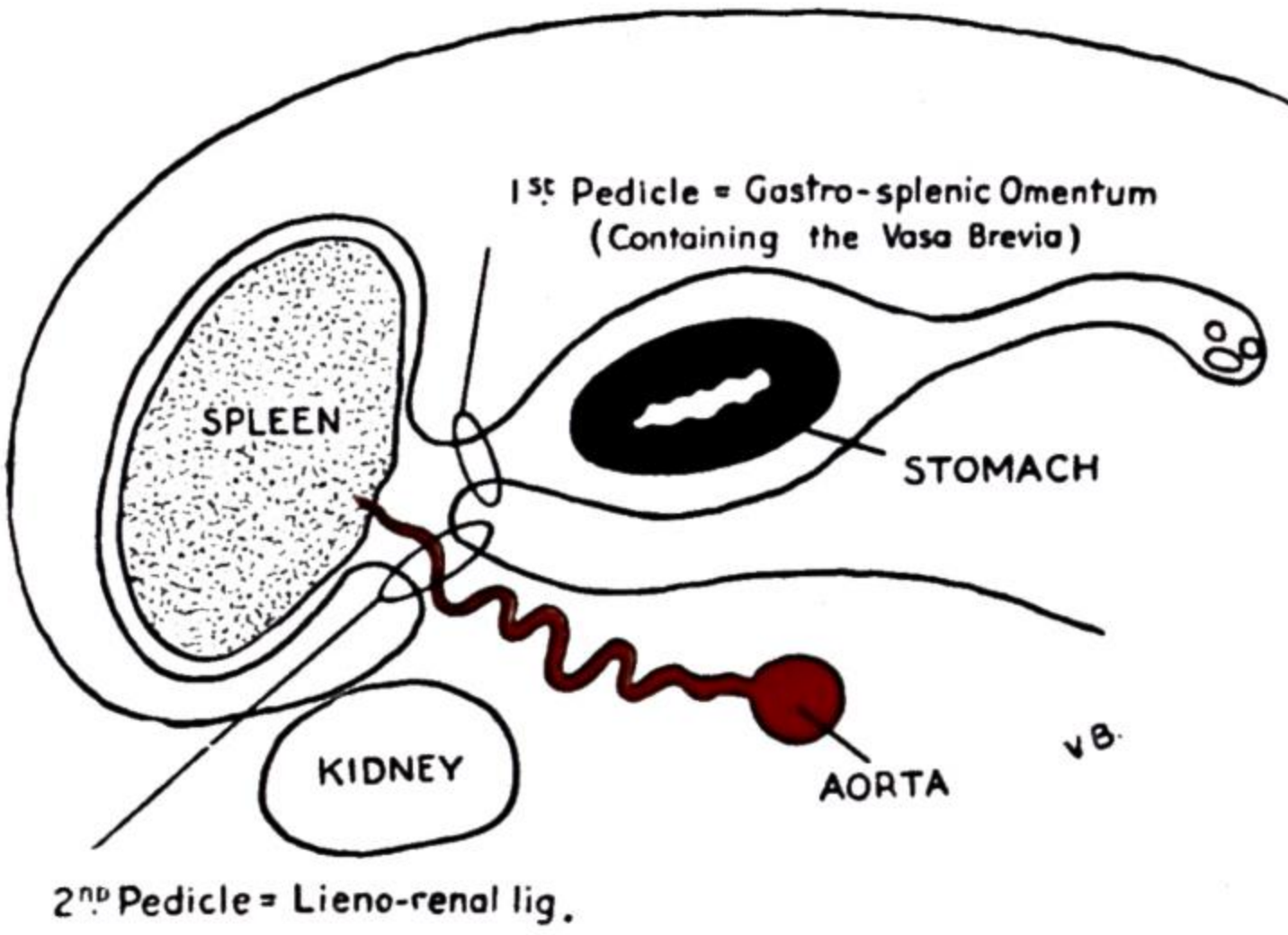

FIG. 535. — The spleen has two pedicles. The splenic vessels lie in the lieno-renal ligament.

Finally, the gastro-splenic omentum, containing the vasa brevia, is divided between ligatures.

The lowest mortality for splenectomy is associated with (1) Traumatic rupture; (2) Hereditary spherocytosis; (3) Idiopathic thrombocytopænic purpura, the reason being that in these conditions frequently the spleen is unencumbered by adhesions, and the operation can be performed easily and expeditiously.

CHAPTER XXI

THE LIVER

HAMILTON BAILEY

Surgical Anatomy.—The dual afferent blood supply, consisting of the hepatic artery and the portal vein, singles out the liver from all other organs. That the portal vein has no valves is a basic fact in the understanding of portal hypertension. On the other hand, not only is the hepatic vein provided with valves, but its main tributaries are peculiar in possessing a coat of unstriated muscle that contracts with histamine and is relaxed by stimulation of the splanchnic nerves, or by adrenaline. To a certain extent blood leaving the liver is controlled by this mechanism. In shock active constriction of the hepatic veins takes place through reflex action. This produces congestion and some degree of stagnant anoxia that, if severe and prolonged, results in hepatic centrilobular degeneration.

There are two sets of capillaries within the liver :

1. Those of the portal system draining the stomach, intestine, spleen, pancreas and biliary tract convey between 75 and 80 per cent. of the afferent blood to the liver, together with the products of digestion from the alimentary canal and the internal secretion, insulin, from the pancreas. No less than one-fifth of the portal blood comes from the spleen.

2. Those of the hepatic artery which transmits oxygen to the liver and, by a delicate pressure arrangement with the hepatic vein, determines the blood flow through the liver lobules. The pressure relationships of the hepatic artery, the portal vein and the hepatic vein testify further as to the unique nature of the circulation through the liver (fig. 536). During exercise the blood flow through the liver falls sharply in favour of other vital organs and the muscles.

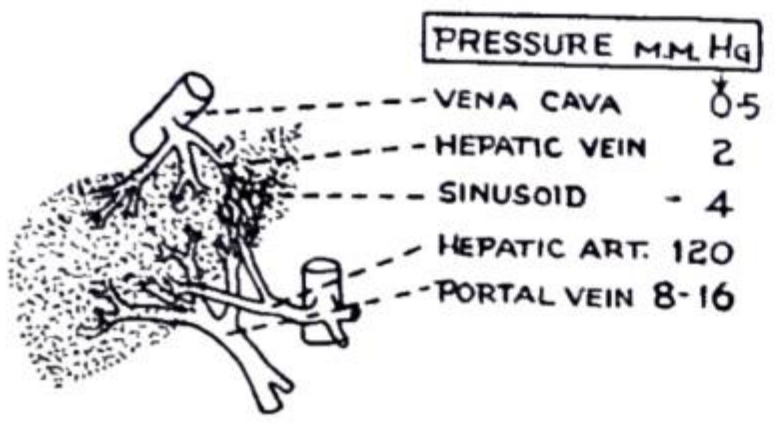

FIG. 536.—Pressure in various components of the hepatic circulation. (*After I. S. Ravdin.*)

Functions of the Liver:

1. The secretion of bile.

2. The final destruction of effete erythrocytes that escape dissolution in the spleen and the excretion in the bile of bilirubin.

3. The importance of the liver as a hæmopoietic organ is demonstrated by the remarkable beneficial effect of liver extract in pernicious anæmia.

4. The formation of fibrinogen and prothrombin.

5. Storage and metabolism of carbohydrates, including the conversion of monosaccharides (e.g. dextrose) into glycogen, and vice versa.

6. Desaturation of fats.

7. Deaminisation of amino-acids, with regulation of the level of plasma proteins.

8. Formation of uric acid.

9. Control of ammonia katabolism. Ammonia, mostly derived from the breakdown of protein in the alimentary canal by bacterial activity, is carried to the liver by the portal blood, and there synthesised into urea and uric acid. The brain also helps to rid the blood of ammonia, which is a poisonous product. Glutamic acid is the only amino-acid oxidised by the brain. Within the cerebral tissue free ammonia becomes linked to glutamic acid to form glutamine, which is then metabolised without ammonia becoming liberated.

10. Destruction of bacteria. Bacteria, especially Gram-positive cocci, have been found repeatedly in samples of portal blood taken at laparotomy for non-infected conditions. It is therefore believed that the liver destroys many bacteria that gain entrance to the body through the portal tributaries.

11. Detoxification of drugs and hormones, e.g. short-acting barbiturates; œstrogens.

Unlike other organs, the liver possesses a remarkable ability to replace lost tissue rapidly and completely by compensatory cellular hypertrophy and hyperplasia. This permits wide resection for a localised hepatic neoplasm, and removal of large portions of doubtfully viable liver tissue in cases of severe trauma. It also allows the formation of a liver abscess containing many pints of pus. Thus, in all these conditions, there is a prospect that, following correct surgical technique, the patient eventually returns to normal health.

LIVER INSUFFICIENCY—HEPATIC COMA

Ætiology.—The most important factor in the production of hepatic coma is ammonia intoxication. While it is improbable that this is the sole cause of the coma it can be regarded as analogous to the high blood urea level of renal insufficiency. When the liver cells are so incapacitated as to render them incapable of synthesising ammonia into relatively harmless urea and uric acid, the blood ammonia rises. A high absorption of protein nitrogen, such as is occasioned by a large amount of blood in the intestinal tract following hæmorrhage from œsophageal varices, frequently engenders hepatic coma in patients with cirrhosis of the liver: it is also often the terminal event in cases of advanced hepatic cirrhosis. Other precipitating factors are intercurrent infection, a surgical operation including paracentesis, the administration of a general anæsthetic and even narcotics.

Unexplained 'liver deaths' following cholecystectomy are nearly always due to accidental ligation of the hepatic artery which can bring about acute liver failure. For the same reason intentional ligation of the hepatic artery for aneurysm is nearly always fatal. Owing to the very high mortality involved, ligation of the hepatic artery as a means of treating portal hypertension has been abandoned.

Clinical Features.—Fœtor hepatis, a sweetish musty odour,[1] is frequently noticeable, sometimes it is so strong as to fill a room. Unusual behaviour and disorientation are the prodromal signs of hepatic coma. Soon there is ataxia and examination reveals a flapping tremor of the outstretched hands reminiscent of the beating of the wings of a hovering bird of prey. Cogwheel rigidity of the limbs and ankle clonus can be elicited as the encephalopathy advances. Plantar reflexes are usually flexor. Excessive salivation is usually present. This, the stage of excitability, lasts a few hours or several days and presents a problem in sedation. Small doses of long-acting barbiturates are the choice, because barbiturates are eliminated by the kidneys.

Unless the precipitating factor can be removed, stupor followed by coma supervenes. Repeated convulsions frequently herald early dissolution.

Ten per cent. of patients undergoing porto-caval shunting suffer from episodal stupor[2] and confusion at some time during their post-operative course.

It should be noted carefully that acute necrosis of the liver often commences with severe abdominal pain that has been mistaken for that due to a perforated viscus. The administration of an anæsthetic in such cases is paramount to the tolling of the bell.

Blood Ammonia Level.—As a rule patients with neurological signs have a

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[1] The probable source of these odoriferous sulphur compounds is methionine liberated by hydrolosis of proteins in the intestine.

[2] Dogs with an Eck's fistula fed exclusively on meat become stuporous (meat intoxication).

Nikolai Vladimirovich Eck, 1848–1908. Surgeon, St. Petersburg (Leningrad).

higher (above 2 micrograms per ml.) level than those with uncomplicated liver disease.

Treatment:

1. Eliminate the source of ammonia. If the gastro-intestinal tract is full of blood, the blood must be removed by all means available (see p. 418). All protein intake by mouth is stopped for a week. If need be plasma[1] can be given intravenously to supply protein in a form that is harmless.

2. A carbohydrate diet of 1,600 calories is supplied daily as dextrose drinks or as 20 per cent. solution of dextrose gravitated through a transnasal intragastric tube by the drip method. In necessary cases the required amount is given as 40 per cent. dextrose gravitated through a polythene tube passed into the vena cava. It is said that a small dose of hydrocortisone added to the concentrated dextrose solution will permit it to be given into a peripheral vein without causing thrombosis.

3. Plasma electrolytes must be measured and any deficiencies rectified. Fluid retention (e.g. ascites) is often associated with a low serum sodium level. Frequently potassium needs supplementation. Occasionally the correction of a deficiency of either sodium or potassium is followed by a return of consciousness.

4. The administration of an oral antibiotic to reduce the enzymic activity of the intestinal flora is highly important. Neomycin is the most powerful agent in this respect. Aureomycin is the next best and is less expensive, but its continued use is liable to result in staphylococcal enterocolitis.

5. Vitamin K and vitamin B complex are required. They are given parenterally.

6. Magnesium sulphate (*NF*) 15 ml. per diem or milk of magnesia is given orally; a daily enema is required if the bowels do not act freely.

7. If it appears likely that the patient will remain unconscious for any length of time, tracheostomy reduces the risk of inhalation of saliva or gastric contents.

8. Oxygen therapy is advisable in the hope that an increased concentration of oxygen will reach the brain.

9. Hæmodialysis has been shown to remove ammonia nitrogen from the blood and the results of the use of Kolff's artificial kidney (see Chapter 31) are encouraging.

Prognosis.—As a result of the above treatment over half the patients in hepatic coma due to cirrhosis of the liver recover. When the coma is due to acute liver necrosis the mortality is very high. Cortisone given in very large doses (1,000 mg. daily) is said to be helpful in these cases.

SPECIAL METHODS OF INVESTIGATING THE LIVER

Liver Function Tests.[2]—Owing to its manifold functions, there is no single test by which the liver can be stated to be functioning normally. Therefore several tests are usually undertaken in each patient, and in some

[1] Two bottles of plasma provide approximately 45 G. of protein.

[2] Tests for the degree of jaundice and the differential diagnosis between obstructive and non-obstructive jaundice are given on p. 449.

instances the individual test must be repeated. No less than 80 per cent. of the liver can be out of action without affecting individual tests; hence, on occasion, the necessity for a battery of them. Among a large number of tests available (including the serum alkaline phosphatase test on p. 449) are the following:

1. **Estimation of serum albumin** is a good general test of the state of liver function. A level below 2·5 G. per 100 ml. indicates that liver function is greatly impaired, and the patient is unfit for operation. A level above 4 G. per 100 ml. is satisfactory.

2. **Bromsulphalein Test.**—The value of this test is diminished in the presence of jaundice, not only because of the technical difficulty of estimating the dye in the presence of bile pigments but also, more importantly, because either intra- or extra-hepatic biliary obstruction may cause increased retention of the dye irrespective of hepato-cellular damage. In the absence of jaundice, it is a most sensitive test of even slight derangement of hepatic function. After an intravenous injection of the dye, not more than 30 per cent. of it should be present in the serum at the end of an hour. An increase in this amount is indicative of liver damage.

3. **Galactose Tolerance Test.**—The liver is capable of converting galactose into glycogen. The patient is given 40 G. of galactose by mouth. Normally the galactose in the blood should not rise above 40 mg. per 100 ml. If it does, or if any is retained in the blood at the end of two hours, the carbohydrate metabolism of the liver is impaired.

4. **Urine Urobilinogen Estimation in the Absence of Jaundice.**—Normally the liver oxidises nearly all the urobilinogen reabsorbed from the intestine, and only very small amounts (below 3 mg.) are excreted in twenty-four hours. Liver damage interferes with this oxidisation; consequently large amounts (from 5 to 300 mg.) of urobilinogen are excreted in the urine, the amount depending on the severity of the liver damage.

5. **Quick's Test.**—This test is only of value if it has been proved that renal function is unimpaired. The principle of the test depends on the administration of sodium benzoate and the collection of urine over a stated period. The hippuric acid which has been formed from the glycine of the patient in this period is precipitated from the urine and its amount determined by weighing.

6. **Thymol Turbidity Tests.**—A saturated solution of thymol buffered at *p*H 7·8 (3 ml.) is added to serum (0·05 ml.). A turbidity usually develops in cases of hepatitis, but results are mainly negative in biliary obstruction. Normal value 0 to 4 units (N. F. Maclagan).

7. **Plasma prothrombin coagulation time** is estimated on two days before and two days after the administration of vitamin K_1. If the initial value is low, this is an indication for pre-operative vitamin K therapy. Where there is little or no response to vitamin K_1 extensive hepato-cellular damage is almost certain. Owing to large reserves a satisfactory response does not exclude considerable liver damage.

Liver biopsy has a limited field of usefulness. It should never be attempted until the presence or absence of a bleeding tendency on the part of the patient has been ascertained by all accepted tests, and if present, remedied. The puncture is carried out on the right side in a manner similar to that described for splenic puncture on the left. Should the liver be greatly enlarged, the puncture can be made beneath the costal margin. There are several patterns of liver biopsy needles. The Silverman needle is as good as any, and better than some. Fig. 537 is self-explanatory. The tubular specimen is placed in a test-tube containing 10 per cent. formalin, which

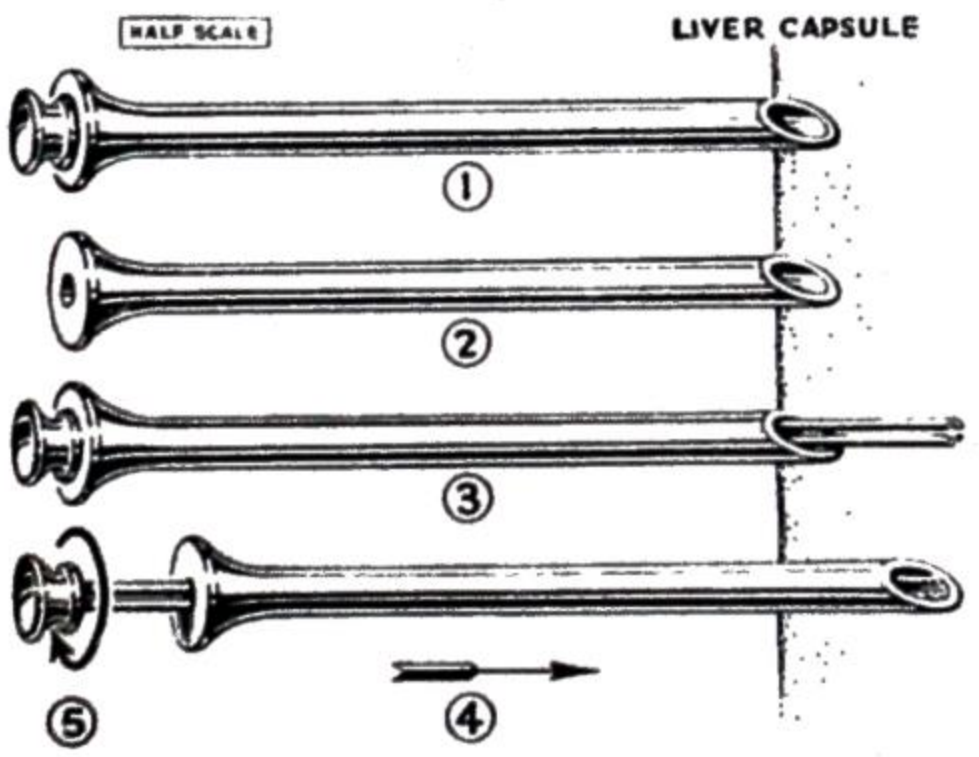

FIG. 537.—Method of obtaining a tubular specimen of the liver for biopsy, using Silverman's needle.

Armand James Quick, Contemporary. Professor of Biochemistry, Marquette University, Milwauki, Wisconsin, U.S.A.
Noel Francis Maclagan, Contemporary. Chemical Pathologist, Westminster Hospital, London.
Irving Silverman, Contemporary. Physician, Brooklyn, New York.

acts as a fixative, and after frozen or paraffin sections have been obtained, an expert pathologist can often pronounce a diagnosis. Failure to obtain a satisfactory specimen occurs in 5 per cent. of cases. When carried out with the stipulation emphasised already, bleeding is an extremely uncommon complication. Much more dangerous is bile peritonitis; a number of fatal cases have been reported from this cause. When obstructive jaundice cannot be excluded, it is safer to perform laparotomy than liver biopsy.

CONGENITAL ABNORMALITIES

Riedel's Lobe.—There is a tongue of liver projecting from the right lobe, which forms a palpable mass beneath the right costal margin. This accessory lobe (fig. 538) must be distinguished from an enlarged, or an unduly mobile, right kidney.

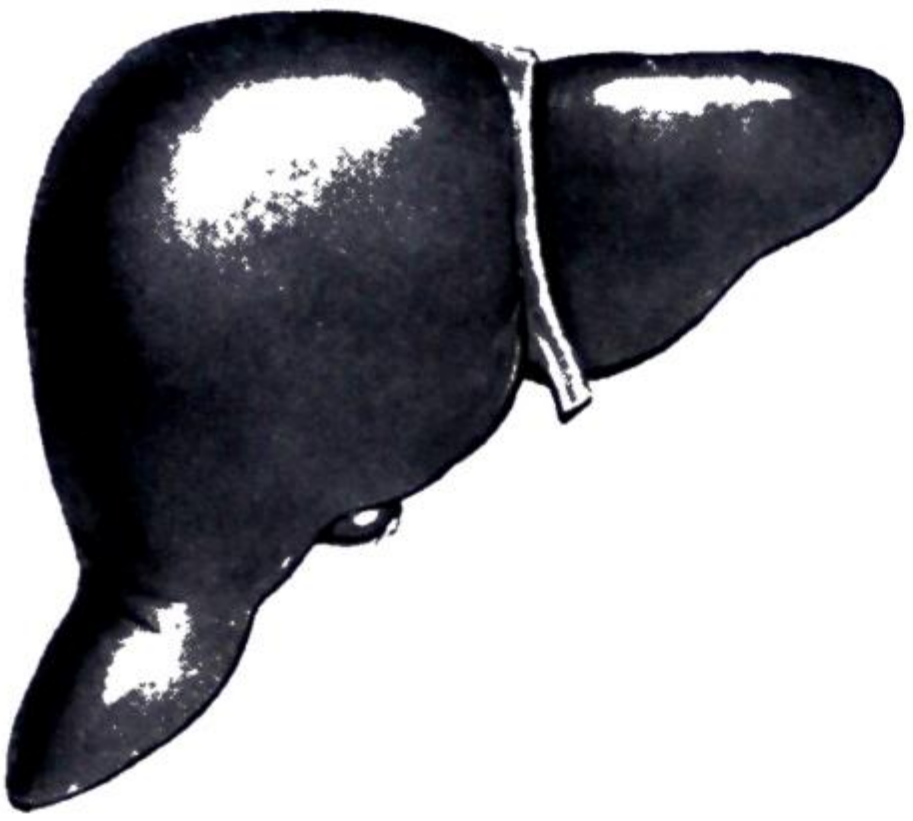

FIG. 538.—Riedel's lobe.

Hepatoptosis (*syn.* **movable liver**) is usually part of a general visceroptosis. There is a congenital form which is due to the absence of certain ligamentous supports of the liver. Almost without exception the patient is a female, and in extreme cases the liver descends below the umbilicus. When it is impossible to control this displacement by a belt, operation has been advised and practised successfully. After scarification of its dome, so as to invite adhesion to the diaphragm, the liver is slung to the abdominal wall and lower costal cartilages.

INJURIES

Rupture of the liver, when extensive, is an extremely grave accident; moreover, in a considerable proportion of cases it is associated with other serious injuries, and consequently the mortality is high. The violence that produces this injury is usually of a crushing type. Tears in the liver are sometimes found at necropsy in subjects who have died from other injuries; small tears, therefore, probably occur frequently, but do not give rise to serious symptoms. Rupture of the right lobe is six times more common than of the left, and usually the tear is on the anterior or the superior surface of the organ. Very often a rib, or ribs, on the right side is fractured. Four clinical types are encountered:

1. There are neither special symptoms nor signs of a ruptured liver. They are those of a hæmoperitoneum, and cannot be distinguished from a ruptured spleen.

2. There are signs of a hæmoperitoneum with localising signs of pain, tenderness, and rigidity in the right upper quadrant.

3. Occasionally, but less commonly than in the case of a ruptured spleen, the symptoms and signs of serious intra-abdominal hæmorrhage are delayed for hours, or even days.

4. A large subcapsular hæmatoma gives rise to a palpable, tender, enlarged liver.

Treatment.—In the first three types a blood transfusion is of cardinal importance. In the fourth type conservative measures should be employed,

Bernhard Riedel, 1846–1916. Professor of Surgery, Jena, Germany.

and often the patient recovers without operation, although during convalescence he may become slightly jaundiced, or periumbilical jaundice may be noticed. In other cases, laparotomy must be undertaken.

Operation.—As soon as the peritoneum is opened, owing to the decrease in intra-abdominal pressure, hæmorrhage from the liver becomes violent. What is more dangerous is that as soon as the anæsthetist has administered a muscular relaxant the bleeding may become greatly increased, therefore it is unwise to inject one of these drugs in cases where a ruptured liver is suspected until the abdomen has been opened. Hæmorrhage from the liver can be controlled by passing a finger into the epiploic foramen (of Winslow) and compressing the hepatic artery and portal vein between the finger and thumb. If the tear extends into the superior surface of the liver, it can be rendered more accessible by dividing the round and falciform ligaments. Should better access to the seat of rupture be required, and a vertical laparotomy incision has been employed, a transverse cut to the right, dividing the rectus muscle, will often provide it. When the rupture is mainly postero-superior, good exposure can be obtained by converting the abdominal incision into an abdomino-thoracic incision. Many ruptures of the liver are clean cut, and do not require excision of tissue; on the other hand, pulped liver and semi-detached portions of doubtful viability must be removed. Failure to débride non-viable liver tissue favours infection and secondary hæmorrhage, a combination of which too often culminates in liver failure (see p. 393). When the tear is clean cut it is repaired by mattress sutures which, if introduced in the manner shown in fig. 539, accomplish their mission in a surprisingly effective manner. Suture is possible in most cases if it is carried out with the largest round-bodied needle available and very thick absorbable sutures. When, after débridement, there is loss of substance, the laceration should be packed with strips of oxycel or suitably shaped pieces of gelfoam, preferably soaked in prothrombin topical. Two or three superficial sutures are passed and tied loosely to keep the packing in place. In extenuating circumstances where speed is essential, the rent can be packed with a roll of ordinary sterile gauze. If this method is used, post-operative antibiotic therapy must be continued until the wound is healed and the gauze must be removed gradually, and not disturbed until the twelfth day, when it is removed little by little by gentle traction under thiopentone anæsthesia in the operating theatre. Only a few feet should be removed each day. Drainage down to the site of rupture must be provided in all cases, because some leakage of bile occurs from torn ductules.

FIG. 539.—Coapting the surfaces of a traumatic cleft in the liver. In this case ribbon catgut is being used. Note the method of tying mattress sutures over a roll of absorbable gauze; this prevents cutting out.

Wounds of the liver occur as a result of gunshot injuries and stab wounds. When, as is commonly the case, the wound of entrance is in the right lower thorax, the thoraco-abdominal approach, excising the wound of entrance, is the best. In some cases the wound of the liver is comparatively small, and no serious bleeding occurs; in such cases, if it is considered that other abdominal organs have escaped injury, conservative treatment is often successful. If the wound is a large one, the same principles of arresting hæmorrhage given above are employed. As one would expect, the mortality is higher when the wound has to be packed.

Jacob Winslow, 1669–1760, a Dane who migrated to Paris, and there established a School of Anatomy.

ANEURYSM OF THE HEPATIC ARTERY

Of over one hundred cases of aneurysm of the hepatic artery, only six were cured by ligation or excision, and many died as the result of attempting these measures. Aneurysmorrhaphy is the only treatment which gives more hope of success.

In this connection, it is instructive to observe that in fifty cases of accidental ligation of the hepatic artery during cholecystectomy, 60 per cent. died as a result of the accident.

ENLARGEMENTS OF THE LIVER

- *General enlargement*
  - With jaundice
    - Regular
      - Virus hepatitis.
      - Cholangitis.[1]
      - Pylephlebitis.[1]
      - Carcinoma of head of pancreas.
    - Irregular
      - Biliary cirrhosis.
      - Late secondary carcinoma.
  - Without jaundice
    - Regular
      - Portal cirrhosis, 1st stage.
      - Chiari's syndrome.
      - Failing heart.
      - Leukæmia.
      - Rickets.
      - Amyloid (examine spleen and urine).
    - Irregular
      - Secondary carcinoma.
      - Gummata.
- *Localised lump*
  - Riedel's lobe.
  - Hydatid cyst.
  - Amœbic abscess.[1]
  - Hepatoma.
  - Secondary carcinoid tumour.
- *Massive irregular enlargement*
  - Polycystic disease (examine kidneys).
  - Cholangio-hepato-adenoma.
  - Secondary melanoma.
  - Late secondary carcinoma.

[1] Intermittent pyrexia with rigors is usually a feature.

VIRUS HEPATITIS

Virus hepatitis, which is due to the virus A, occurs in both sporadic and endemic forms. The virus is excreted in the fæces and is carried, probably, by the house-fly. The 1955 epidemic in Delhi followed floods, so that the city's water supply was contaminated with sewage. Virus hepatitis which is the commonest cause of jaundice in adults is, in the U.S.A. and in some areas of Britain, a notifiable disease. Young persons are attacked more often than the middle-aged and elderly, and the incubation period is fourteen to forty days. As a rule, the condition commences abruptly with nausea and perhaps vomiting, together with general malaise and mild pyrexia. This pre-icteric phase lasts approximately three days, and is followed by mild jaundice. The liver becomes palpable and tender, and the jaundice lasts for two or three weeks. Transient ascites sometimes occurs, especially in middle-aged women: it has been noted quite often in Scandinavian epidemics.

Differential Diagnosis.—Occasionally the disease occurs in a more

severe form, and the jaundice is so profound as to render the differential diagnosis between this condition and extrahepatic biliary obstruction difficult. An alkaline phosphatase estimation (see p. 449) and the flocculation tests, considered together, sometimes solve the problem; frequently they are unhelpful. If biliary obstruction complicated by cholangio-hepatitis cannot be ruled out by biligrafin cholecystography, laparotomy for exploration of the bile-ducts and operative cholangiography is required.

Severe and recurrent virus hepatitis is liable to be followed by portal cirrhosis. The general mortality of virus hepatitis is low; occasionally the disease proves rapidly fatal from acute liver necrosis.

Prophylaxis.—A single injection of gamma globulin often prevents infection (e.g. of the nursing staff).

Treatment.—There is no specific treatment, consequently treatment resolves itself into common-sense principles. The patient must be nursed with full aseptic precautions, and kept in bed until improvement sets in. The patient must be coaxed to eat a reasonably high calorie diet: the amount of fat in which is dictated by the fancy of the patient. Should sleeplessness be troublesome, chloral hydrate (which is free from toxic effect on the liver) is prescribed. If the prothrombin level falls, intramuscular injections of vitamin K_1 are indicated. The bowels are kept open with appropriate doses of magnesium sulphate, which possesses some action as a cholagogue. If there is no bile in the urine at the end of a week, it is permissible to allow the patient up for part of the day, and as soon as thought advisable he should take a restful holiday. Until the patient has felt absolutely well for two months, two things are forbidden—alcohol and strenuous exercise (E. R. Cullinan).

As a rule young adults are free from symptoms, and fit to return to work in seven weeks from the time of the onset of symptoms. In older persons the disease is inclined to last longer. A small proportion of patients have a second visitation of the disease shortly after the first.

The Post-hepatitis Syndrome.—About 5 per cent. of patients do not feel well for about a year. They are easily fatigued, and breathless on exertion. The sight of fatty food often revolts them. Because liver function tests and liver biopsy are negative, too often these symptoms are mistaken for an anxiety state, but tachycardia and brisk reflexes are absent. The post-hepatitis syndrome does not last more than eighteen months.

Acute Yellow Atrophy of the Liver.—From time to time virus hepatitis takes a fulminating course. The patient, who may not exhibit jaundice, passes into a semi-comatose state, with symptoms suggesting meningitis. These are, in point of fact, manifestations of hepatic neuro-encephalopathy. The fœtor hepatis and the fact that there is an epidemic of virus hepatitis in the neighbourhood are the main keys to diagnosis. The majority of victims gradually go downhill, and die. At necropsy the liver is found to be comparatively small, and of a bright yellow colour.

Obstetric acute yellow atrophy is the name given by H. L. Sheehan to a similar condition occurring in women near term. The cause of the condition, which is categorised generally under the heading of toxæmia of pregnancy, has not been elucidated.

From time to time patients with either of the above conditions, after hovering between life and death, eventually recover. However, the liver is grossly scarred (potato liver), and portal hypertension is likely to supervene.

Edward Revill Cullinan, Contemporary. Physician, St. Bartholomew's Hospital, London.
Harold Leeming Sheehan, Contemporary. Professor of Pathology ,The University of Liverpool.

ANEURYSM OF THE HEPATIC ARTERY

Of over one hundred cases of aneurysm of the hepatic artery, only six were cured by ligation or excision, and many died as the result of attempting these measures. Aneurysmorrhaphy is the only treatment which gives more hope of success.

In this connection, it is instructive to observe that in fifty cases of accidental ligation of the hepatic artery during cholecystectomy, 60 per cent. died as a result of the accident.

ENLARGEMENTS OF THE LIVER

- *General enlargement*
  - With jaundice
    - Regular
      - Virus hepatitis.
      - Cholangitis.[1]
      - Pylephlebitis.[1]
      - Carcinoma of head of pancreas.
    - Irregular
      - Biliary cirrhosis.
      - Late secondary carcinoma.
  - Without jaundice
    - Regular
      - Portal cirrhosis, 1st stage.
      - Chiari's syndrome.
      - Failing heart.
      - Leukæmia.
      - Rickets.
      - Amyloid (examine spleen and urine).
    - Irregular
      - Secondary carcinoma.
      - Gummata.
- *Localised lump*
  - Riedel's lobe.
  - Hydatid cyst.
  - Amœbic abscess.[1]
  - Hepatoma.
  - Secondary carcinoid tumour.
- *Massive irregular enlargement*
  - Polycystic disease (examine kidneys).
  - Cholangio-hepato-adenoma.
  - Secondary melanoma.
  - Late secondary carcinoma.

[1] Intermittent pyrexia with rigors is usually a feature.

VIRUS HEPATITIS

Virus hepatitis, which is due to the virus A, occurs in both sporadic and endemic forms. The virus is excreted in the fæces and is carried, probably, by the house-fly. The 1955 epidemic in Delhi followed floods, so that the city's water supply was contaminated with sewage. Virus hepatitis which is the commonest cause of jaundice in adults is, in the U.S.A. and in some areas of Britain, a notifiable disease. Young persons are attacked more often than the middle-aged and elderly, and the incubation period is fourteen to forty days. As a rule, the condition commences abruptly with nausea and perhaps vomiting, together with general malaise and mild pyrexia. This pre-icteric phase lasts approximately three days, and is followed by mild jaundice. The liver becomes palpable and tender, and the jaundice lasts for two or three weeks. Transient ascites sometimes occurs, especially in middle-aged women: it has been noted quite often in Scandinavian epidemics.

Differential Diagnosis.—Occasionally the disease occurs in a more

nected together, forming a 'canal and cavity' system (H. M. Turnbull). Cultures show a mixed infection in which *Esch. coli* is prominently represented. In rare instances there is localisation of the infection.

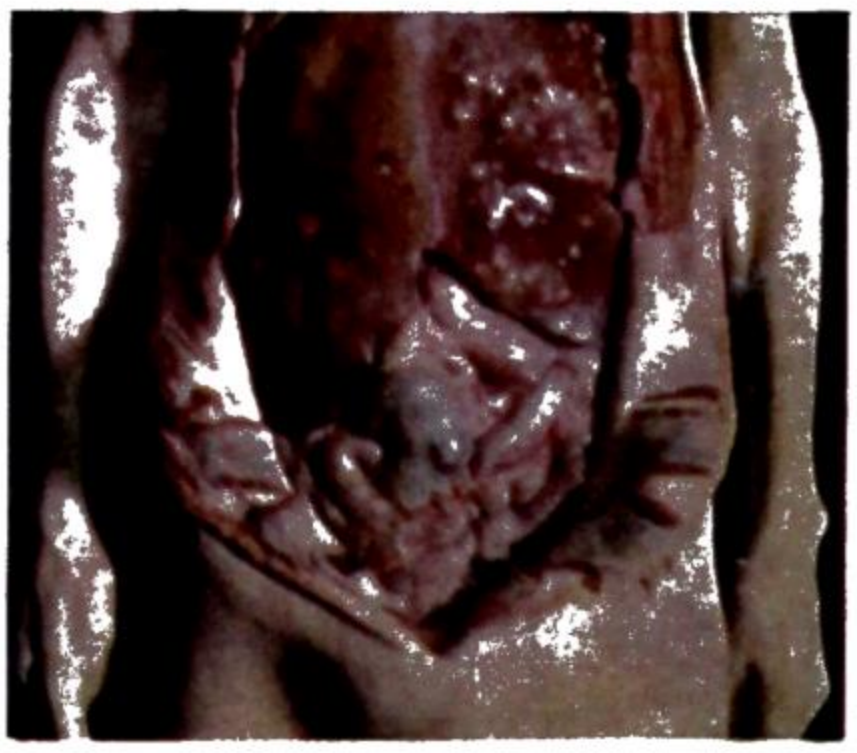

FIG. 540.—Pylephlebitis. Liver riddled with small abscesses.

Clinical Features.—Pylephlebitis is characterised by a hectic temperature; repeated rigors are never absent. In relevant cases this symptom serves to differentiate the condition from a subdiaphragmatic abscess, but it does not exclude it. If tested for, urobilinogen will be found in the urine in every case. Diarrhœa is a frequent accompaniment. Moderate ascites is sometimes present.

Blood culture is positive in 50 per cent. of cases if blood is withdrawn during or immediately after a rigor. The patient soon becomes slightly jaundiced, i.e. the conjunctivæ are icteric when observed in daylight, and the liver is found to be somewhat enlarged and tender.

Prophylaxis.—(*a*) The most important means of preventing pylephlebitis is to administer a broad spectrum antibiotic (tetracycline or chloramphenicol) in cases of fulminating appendicitis and diverticulitis. (*b*) Hæmorrhoids must never be removed in an acutely inflamed state. (*c*) Ligation of the ileocolic vein in addition to appendicectomy is of possible value in those rare cases of early acute appendicitis where pre-operative rigors are in evidence.

Treatment.—As the organisms responsible are mixed, and usually insensitive to penicillin, broad spectrum antibiotics and combinations of antibiotics must be administered and continued for at least ten days after the temperature has become normal. Blood transfusion is often required. If in a protracted case it is considered possible that there is a sizeable abscess within the liver, laparotomy should be performed with a view to draining the abscess.

Prognosis.—When pylephlebitis is fully established the outlook is extremely serious.

IDIOPATHIC PYOGENIC LIVER ABSCESS

A pyogenic liver abscess, solitary in over 50 per cent. of cases, can arise without any obvious cause. The early symptoms do not differ from those of pylephlebitis. The enlarged tender liver can be felt easily in two-thirds of cases. Radiological evidence of hepatic enlargement is present in even a higher proportion. When the possibility of an amœbic abscess can be excluded, laparotomy is the correct course. After careful isolation of the enlarged liver with packs the presence of pus is confirmed by aspiration. If pus is present the needle is left *in situ* and the abscess is drained in a manner similar to that described for amœbic abscess (p. 405).

In most instances the cause of a liver abscess is apparent or can be ascertained, infection reaching the liver in one of several ways, viz:

Herbert Maitland Turnbull, 1875–1955. Professor of Pathology, The London Hospital.

Virus hepatitis following blood transfusion, plasma infusion,[1] and, to a lesser extent, the administration of sera, is a major problem. Virus hepatitis due to blood transfusion is caused by the virus I.H.,[2] while that due to other vehicles is more likely to be caused by virus S.H.[3] One carrier-donor can contaminate the whole consignment of plasma or serum; therefore plasma should not be pooled in large quantities. If a maximum of ten donors is employed, the incidence is reduced.

Syringe jaundice is another variety of virus hepatitis, and in this instance the virus S.H. is responsible. The interior of a syringe becomes contaminated by blood drawn into the syringe from a person suffering from infectious hepatitis. As in this form of virus hepatitis the incubation period is 60 to 135 days, little wonder that neither the patient nor his doctor associates the attack with the bygone injection. It should be known more widely that injection of local anæsthesia for dental operations has recently been proved to be culpable in a large number of instances. It is of paramount importance to reiterate that all syringes should be boiled or autoclaved fresh for each patient, and not sterilised in spirit. It is becoming recognised generally that if it can be proved that virus hepatitis is due to an injection, the doctor or dental surgeon will be held responsible: thus in Italy a doctor was gaoled for five years and fined heavily following the occurrence of virus hepatitis in many of his patients, some of whom died from the disease thus transmitted.

CHOLANGITIS (*syn.* INTERMITTENT HEPATIC FEVER OF CHARCOT)

By cholangitis is meant a state of inflammation of the bile-ducts, but it is the radicles of the biliary tree within the liver, as opposed to the extrahepatic ducts, that should be visualised as being the seat of the infection. Cholangitis can occur as the result of mucoviscidosis (see p. 461) or to obstruction of the common bile-duct by a gall-stone or a stricture. Cholangitis is characterised by pyrexia with rigors and jaundice, and is met with as a complication of obstruction to the common bile-duct (see p. 449).

Suppurative cholangitis is rare, and like pylephlebitis, is characterised by the formation of multiple liver abscess.

PYLEPHLEBITIS (*syn.* PORTAL PYÆMIA)

Pathology.—Pylephlebitis can follow a suppurative disease in any part drained by the portal system, but in over 70 per cent. of cases it arises as a complication of appendicitis. The process commences as a thrombophlebitis of a small vein draining the infected lesion. The thrombus spreads to a larger collecting vessel, and pieces of infected thrombus break off and are swept into the liver, where they lodge and form abscesses. Abscesses so formed (fig. 540) are usually small and multiple, in which case they are con-

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[1] The incidence of virus hepatitis following small pool plasma infusion is 0·12 per cent.; that following transfusion of whole blood 0·16 per cent.

[2] I.H. = Infectious hepatitis.

[3] S.H. = Serum hepatitis.

Jean-Martin Charcot, 1825–1893. Physician to the Hôpital Salpêtrière, Paris.

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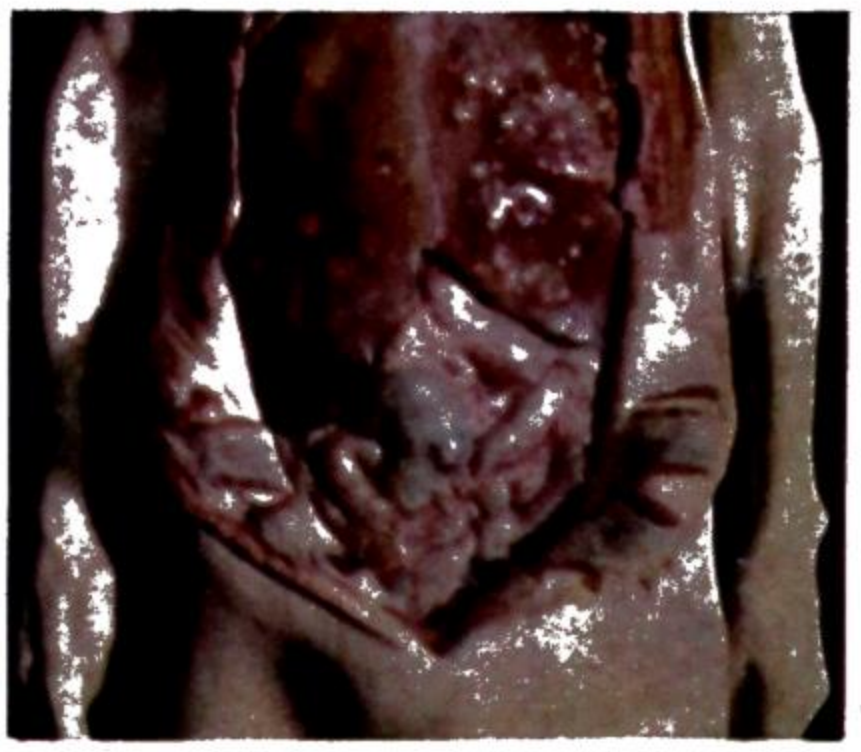

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Treatment.—As the organisms responsible are mixed, and usually insensitive to penicillin, broad spectrum antibiotics and combinations of antibiotics must be administered and continued for at least ten days after the temperature has become normal. Blood transfusion is often required. If in a protracted case it is considered possible that there is a sizeable abscess within the liver, laparotomy should be performed with a view to draining the abscess.

Prognosis.—When pylephlebitis is fully established the outlook is extremely serious.

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In most instances the cause of a liver abscess is apparent or can be ascertained, infection reaching the liver in one of several ways, viz:

Herbert Maitland Turnbull, 1876–1955. Professor of Pathology, The London Hospital.

ABSCESSES OF THE LIVER

Via the Portal Vein
- Suppurative appendicitis.
- Diverticulitis.
- Amœbiasis.
- Infected carcinoma of the colon (especially after resection).
- Ulcerative colitis.
- Actinomycosis of the right iliac fossa.
- Inflamed hæmorrhoids.[1]
- Typhoid or paratyphoid (rare).

Along the Bile Ducts
- Stone impacted in common duct.
- Stricture of a main bile duct.

Via the Hepatic Artery
- Septicæmia and pyæmia.
- Facio-cervical actinomycosis.
- Infection of a hydatid cyst.

From the Umbilicus
- Along the umbilical vein of the new-born.
- Along the para-umbilical veins.

By Direct Extension
- From a subdiaphragmatic abscess.
- From an empyema thoracis.
- From a penetrating wound.

[1] Extremely rare unless hæmorrhoids are removed in an inflamed state.

AMŒBIC LIVER ABSCESS (*syn.* TROPICAL ABSCESS DYSENTERIC ABSCESS)

Amœbic abscess of the liver is one of the terminations of amœbic hepatitis, which in turn is a complication of amœbic dysentery.

Pathology.—Hepatic complications occur in about 8 per cent. of patients with amœbiasis. *Entamœbæ histolyticæ* (fig. 541) pass from a focal lesion in the colonic wall into a radicle of the inferior mesenteric vein and via the portal vein they enter the liver to take up residence there, usually in the upper and posterior portion of the right lobe. The right lobe is so frequently (85 per cent.) the seat of an amœbic abscess because blood from the intestine goes mainly to the right lobe of the liver, while the blood from the spleen goes mainly to the left lobe (Bernard Shaw). In the liver the entamœbæ colonise and live at the expense of the liver cells, causing localised liquefaction necrosis. The amount of liver destruction is proportional to the size of the colony and the resistance of the host. In 70 per cent. of cases the abscess is solitary; in 30 per cent. more than one abscess is present. Characteristic pus from an amœbic liver abscess is chocolate-coloured, or like anchovy sauce, and consists of broken-down liver cells, leucocytes, and red blood cells; nevertheless, in an appreciable number of instances the pus is green, from being admixed with bile. In about half the cases the pus contains staphylococci, streptococci, and *Esch. coli*, as well as *E. histolytica*. In the remainder the pus is sterile, but except in long-standing cases, motile entamœbæ can be demonstrated in the last few drops of pus to be withdrawn, or from a scraping of the abscess wall at operation. Nearly always perihepatitis causes the liver

FIG. 541.—Entamœba histolytica.

Arthur Frederick Bernard Shaw, 1888–1947. Professor of Pathology, University of Durham, Newcastle-upon-Tyne.

to become fixed to the diaphragm or the abdominal wall; consequently the liver may be prevented from enlarging in a downward direction.

Course.—An amœbic abscess of the liver runs a variable course:

1. In early stages of amœbic hepatitis with abscess threatening, resolution often occurs under emetine treatment.

2. When an abscess forms the liver enlarges, most often in an upward direction. It is at this stage that surgical intervention is called for.

3. It may become encapsulated and remain dormant for long periods.

4. Unrecognised and untreated, it may burst into (*a*) the right lung, (*b*) the peritoneal cavity, (*c*) the right pleural cavity, in that order, or, more rarely, into a hollow viscus (fig. 542). Exceptionally the abscess points beneath the skin overlying the liver.

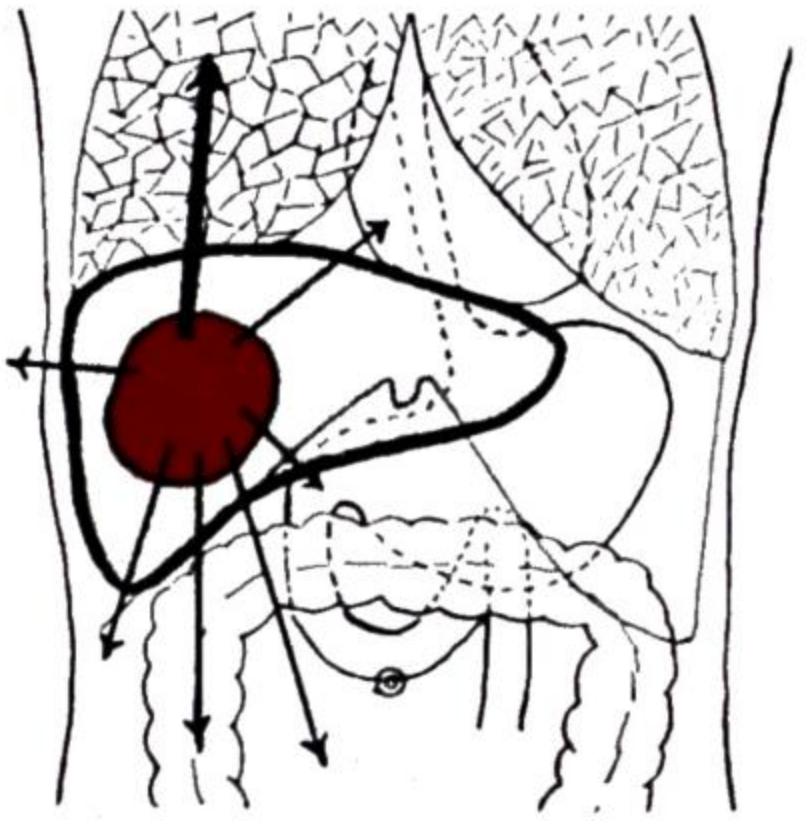

FIG. 542.—Directions in which a tropical liver abscess may burst. (*After Sir Zachary Cope.*)

The bursting of a liver abscess into the lung and the expectoration of a quantity of chocolate-coloured sputum sometimes results in a natural cure.

Bacterial infection is a rather frequent and serious complication of amœbic abscess. While solitary amœbic abscess is more amenable to combined specific drug and surgical treatment, the prognosis in cases of multiple amœbic liver abscesses is extremely poor.

Clinical Features.—White males between twenty and forty years of age are usually affected. Although women suffer from amœbic dysentery equally with men, amœbic abscess of the liver is a rarity in the female sex, possibly because they drink less alcohol. As a rule, the condition develops soon after an attack of amœbic dysentery while the patient is resident in a tropical or sub-tropical country. Less frequently its appearance is delayed, sometimes for many months; exceptionally it has occurred more than thirty years after returning home from the tropics. Occasionally an amœbic abscess develops in a carrier who has not had overt dysentery; indeed, it frequently appears in persons who have had mild diarrhœa not diagnosed as dysentery, and consequently have not had treatment for that condition.

Early Symptoms.—Anæmia, loss of weight, and an earthy complexion are often the first symptoms.

Pyrexia rising to 101° F. (38° C.) or more, at night, with profuse sweating, is nearly always present. Rigors occasionally occur, especially in the early stages.

Pain is constantly present in the liver area, and is occasionally referred to the right shoulder. Jarring increases the pain so that the patient learns to try to support his enlarged liver with his hands when he walks.

Tenderness and *rigidity* in acute cases are comparable to that of acute cholecystitis. In old-standing chronic cases tenderness may be absent.

Enlargement of the liver can often be demonstrated by clinical methods (fig. 543), but it is not unusual for an abscess to be present in a liver which is fixed by perihepatitis, and therefore the liver cannot enlarge in a downward direction.

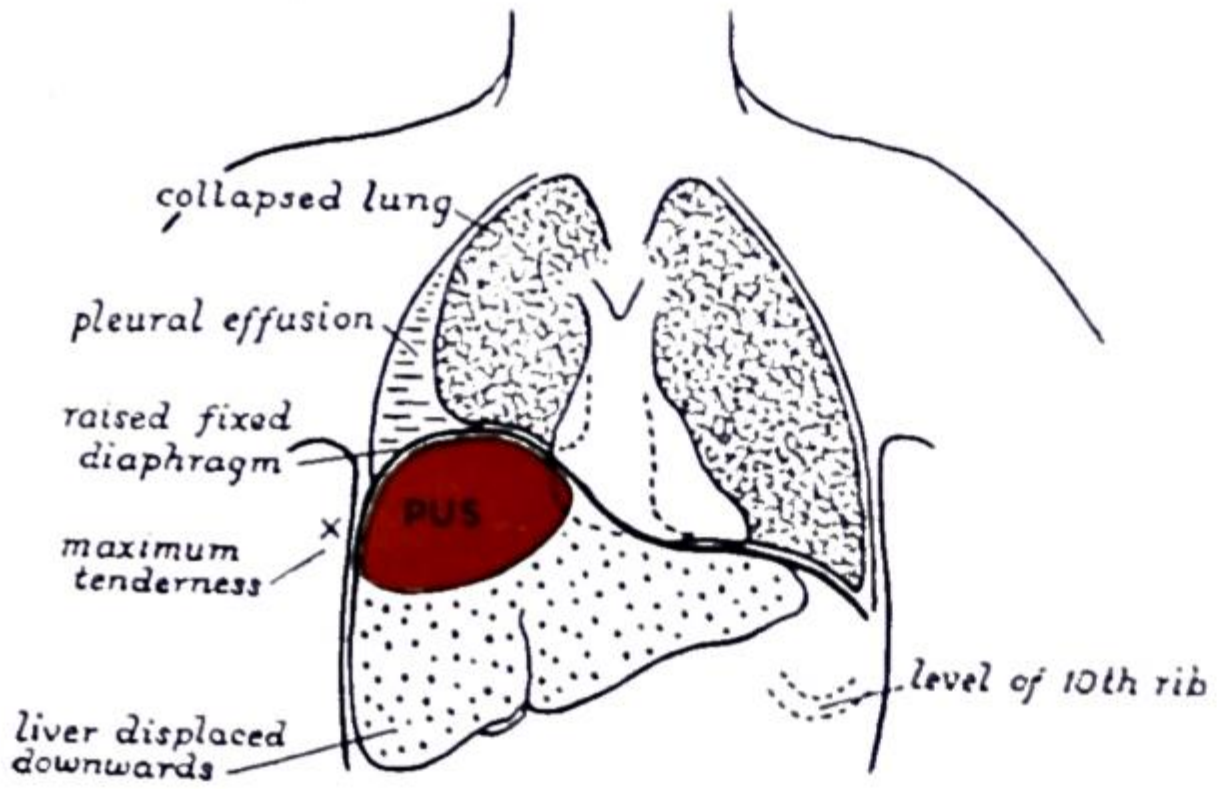

FIG. 543.—The physical signs of a tropical liver abscess (commonest site). *(After A. T. Andreasen.)*

Basal lung signs on the corresponding side can always be elicited in acute cases of hepatic abscess.

Leucocytosis is present in nearly all cases. Polymorphonuclear cells constitute, at the most, only 75 per cent. of the total count.

Examination of the stools for amœbæ should be undertaken, but their absence does not exclude the diagnosis of amœbic abscess of the liver.

Sigmoidoscopy sometimes reveals the characteristic ulcers (p. 535).

Radiography (antero-posterior and lateral positions) often reveals an elevation and fixation of the right cupola of the diaphragm (fig. 544).

Often an absolute diagnosis is possible only on finding typical pus by exploratory aspiration.

Occasionally an amœbic liver abscess does not give rise to symptoms; healed lesions have been found at necropsy and calcified lesions (that may have been an abscess) have been revealed radiologically.

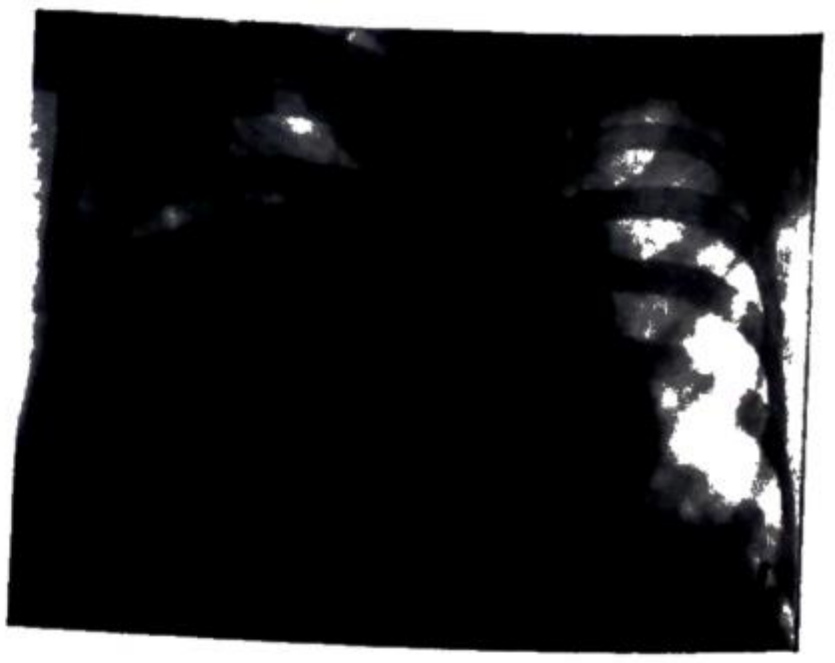

FIG. 544.—Radiograph showing tropical liver abscess. *(Major C. J. Hassett, Nairobi, Kenya.)*

Treatment.—When amœbic hepatitis is even suspected, chloroquine 0·6 G. daily for two days, followed by 0·3 G. daily for twelve to nineteen days, is the treatment of choice. Should there be no response after a few days, it suggests the possibility of secondary infection, and a tetracycline (achromycin or terramycin) in doses of 250 mg. four times each twenty-four hours should be given in addition. When this combination fails to produce the desired result the treatment is stopped, and intramuscular injections of emetine hydrochloride, 1 grain (60 mg.) daily for ten days is substituted. Emetine was the standard remedy for this condition for many years, and very successful it was. Its only drawback is its tendency to cause myocardial toxicity; therefore blood-pressure readings twice daily and an electrocardiograph once weekly should be the rule in patients receiving emetine.

In cases where specific drug therapy proves successful (and often it produces such obvious improvement in amœbiasis as to be diagnostic), provided

there is no contraindication to the re-exhibition of the chosen drug, the course of treatment is repeated after an interval of three weeks.

Aspiration.—When, in spite of specific drug therapy, the temperature does not settle, if the pain persists, and particularly if the presence of a suspected abscess is confirmed radiologically, aspiration should be undertaken. A secondary infected abscess should be treated similarly in the first instance; indeed, such infection can be discovered only by bacteriological examination of the pus.

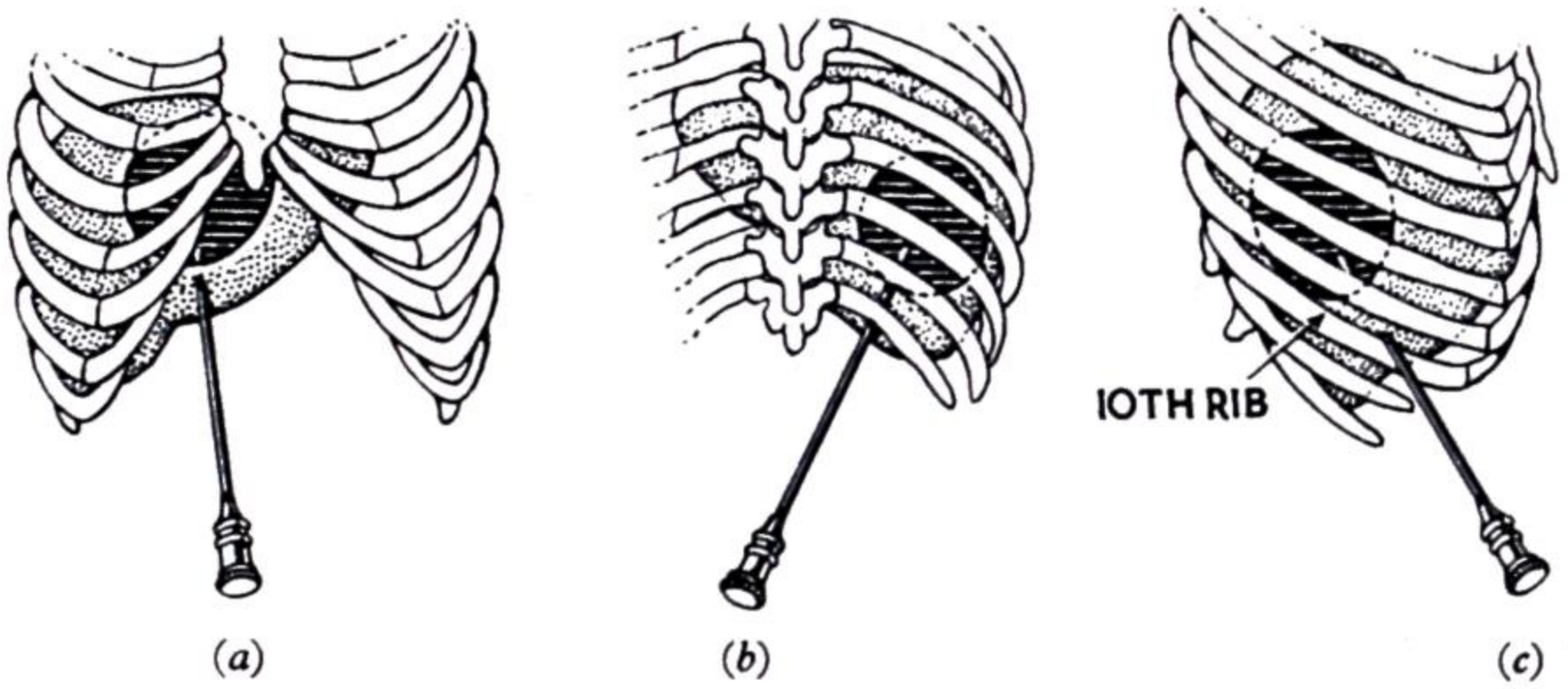

FIG. 545.—Aspiration of an amœbic hepatic abscess: (*a*) in the anterior part of the liver; (*b*) in the posterior part of the liver ; (*c*) located near the dome of the diaphragm.

Technique.—Aspiration must be conducted in the operating theatre. A long needle is necessary, and its bore should be wide, as the pus is usually creamy in consistency. The important technique of percutaneous introduction of the hollow needle (under local anæsthesia) into the abscess cavity in various locations is shown in fig. 545. In some cases the abscess eludes the aspirating needle, in which event laparotomy must be performed, thus allowing the liver to be explored by the aspirating needle more thoroughly. At the same time laparotomy permits the exclusion of a primary carcinoma of the liver which, in coloured races, sometimes closely resembles an amœbic liver abscess in its onset and physical signs, and at times is even accompanied by a low pyrexia. In the case of an amœbic liver abscess the amount of pus aspirated averages half a pint (285 ml.) although sometimes a much greater quantity is obtained. Many surgeons with tropical experience instil 1 G. of emetine hydrochloride into the abscess cavity at the end of the aspiration. Aspiration avoids secondary infection and amœbic infestation of the wound.

After-treatment.—A full course of specific drug therapy should be given after aspiration and each re-aspiration.

Drainage of a Liver Abscess.—A number of surgeons are finding that under antibiotic cover extra-pleural drainage of a posteriorly-placed amœbic abscess of the liver gives quicker and better results than aspiration. French surgeons, in particular, are insistent that open drainage of any amœbic abscess is no longer associated with a higher mortality or morbidity than more conservative measures.

Technique.—A posteriorly-situated abscess is drained extra-pleurally in a manner similar to the drainage of a subdiaphragmatic abscess (see p. 489). When laparotomy is indicated (central and anteriorly-placed abscesses of the right lobe, and all abscesses of the left lobe), packs impregnated with a solution of erythromycin 1:1,000 are so arranged as to isolate the liver. The presence of an abscess having been ascertained

by needling, a trocar and cannula is thrust into the abscess cavity: pus having been evacuated, the cavity is irrigated with saline solution. The opening in the liver is enlarged carefully, and a suitably-sized de Pezzer catheter is inserted and anchored to the liver. An omental barrier is constructed before closing the abdomen.

After-treatment.—Every effort should be made to keep the drainage closed for as long as possible by connecting the tube to a water-sealed bottle. After the operation a full course of specific drug therapy should be given, and an antibiotic should be administered until the abscess ceases to drain.

ACTINOMYCOSIS OF THE LIVER

Actinomycosis produces the well-known 'honey-comb' liver. Actinomyces reach the organ in one of the following ways:

1. From contiguous viscera, e.g. a penetrating peptic ulcer invading the liver—20 per cent.
2. Via the portal vein from actinomycosis of the right iliac fossa—50 per cent.
3. Via the hepatic artery from a more distant primary focus, e.g. facio-cervical actinomycosis—30 per cent.

The disease is slow to develop, and causes swelling of one or other lobe. Gradually the liver tissue is destroyed and replaced by multiple abscesses.

Treatment.—Exploration is essential, and in every case of liver abscess of doubtful origin the pus should be examined for actinomyces. Long-acting penicillin, or other more suitable antibiotic therapy (see p. 203) must be continued for a considerable time. Usually by the time the liver is involved the prognosis is grave, but not hopeless.

TUBERCULOSIS OF THE LIVER

In patients with advanced tuberculosis, the liver is frequently fatty; presumably this is due to the toxicity of pulmonary tuberculosis. In 50 per cent. of necropsies upon patients who have died from tuberculosis, miliary tubercles are present in the liver.

Local tuberculosis of the liver is usually diagnosed at necropsy, but occasionally at laparotomy. There is a large mass containing necrotic material. This lesion is more common in children, and in negroes with little immunity. Less uncommon is a frank tuberculous abscess. In a great majority of cases the lesion is secondary to tuberculosis elsewhere. The symptoms are identical with those of an amœbic abscess. Tuberculosis of the liver responds well to streptomycin, and the timing of aspirating or draining such an abscess should be left to the tuberculosis specialist. That these lesions sometimes resolve can be testified by subsequent visualisation of one or more calcareous areas in the liver on radiography.

HEPATIC SYPHILIS

As far as the liver is concerned, syphilis, always an accomplished actor, can, and does, deceive the clinician, and even the operator. Gummata give rise to rounded masses in the liver. These swellings sometimes simulate closely a liver abscess. Multiple gummata of the liver give signs not unlike secondary carcinoma when examined clinically, although, when displayed to the light of day, they lack the characteristic umbilicated appearance of the latter. Penicillin and bismuth treatment is frequently effective, especially when gummatous lesions predominate. It can also be employed as a therapeutic test to substantiate the diagnosis. Syphilis is incriminated as a cause of hepatic cirrhosis, although this is not so in adults (S. Sherlock).

HYDATID DISEASE OF THE LIVER

Although the parasite can thrive in many parts of the body, in 70 per cent. of cases it does so in the liver. After ingestion, the tænia echinococcus enters this organ through a radicle of the portal vein.

Oscar Michel Benvenuto de Pezzer, 1853–1917, Assistant to Professor Guyon, the Necker Hospital, Paris, invented the de Pezzer catheter in 1885.

Sheila Sherlock, Contemporary. Professor of Medicine, Royal Free Hospital School of Medicine, London.

Source of Infection.—While dogs are the chief mediators of hydatid disease to human beings (fig. 546), it is chewing grasses and eating unwashed raw vegetables on which ova have been deposited that is usually responsible for the infestation in man. Once in the stomach, the ova burrow through the stomach wall to enter the portal system and the liver. Dogs become infected by feeding on offal of infested sheep and, to a lesser extent, cattle

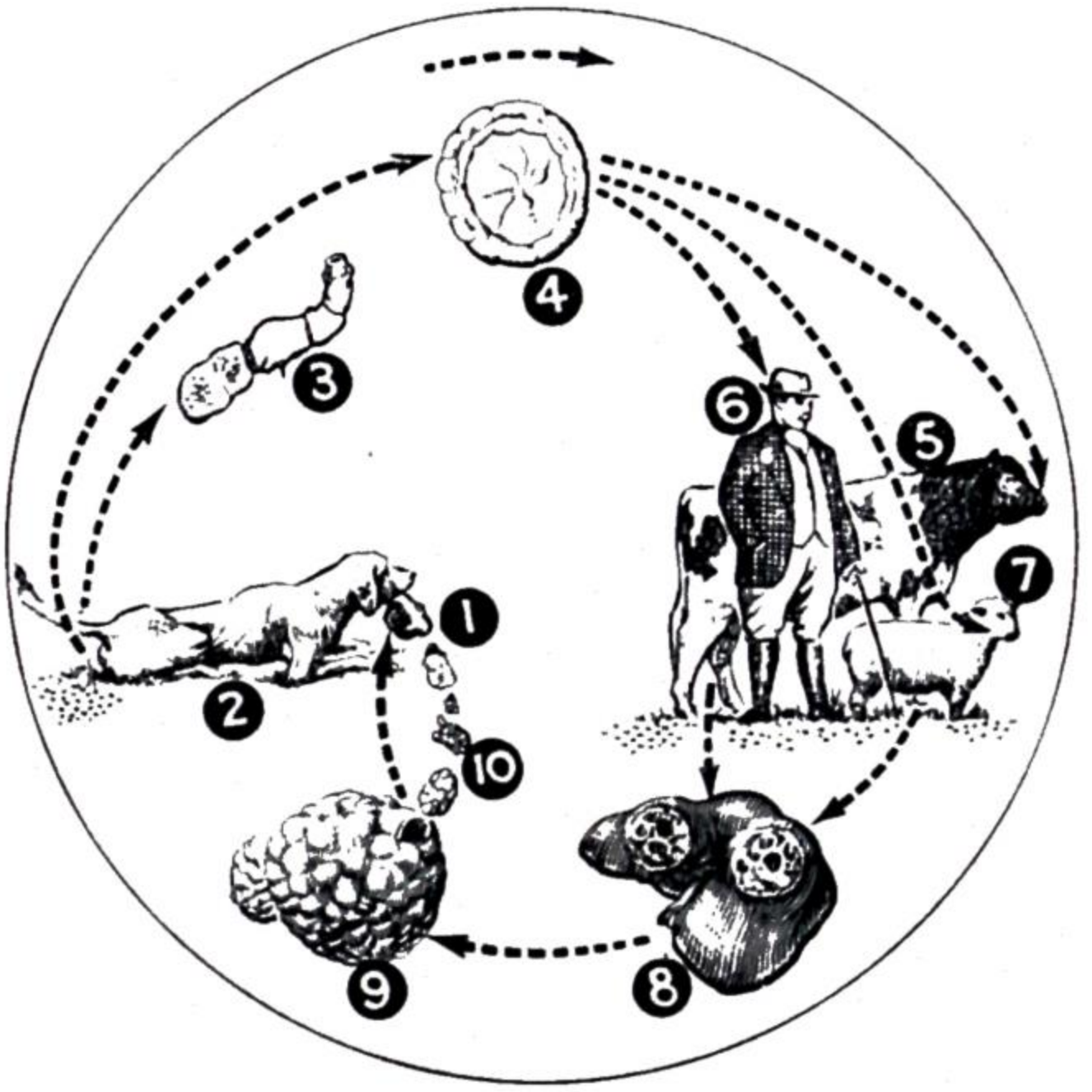

FIG. 546.—The life-cycle of the tænia echinococcus. Offal (1) infected with hydatid cysts is eaten by a dog (2). The *Tænia echinococcus* (3) develops in the dog's intestine. This parasite is made up of a head and three segments, the last of which contains about 500 ova (4). The ova are expelled from the dog's intestine on to grass, vegetables, etc. Cattle (5), human beings (6), or sheep (7) ingest the eggs. The liver (8) is the organ most frequently infested with hydatid cysts, a larval form of *Tænia echinococcus*. Such cysts (9) harbour thousands of heads of the echinococcus (scolices) (10). *(After V. P. Fontana.)*

(V. P. Fontana). As would be expected, the disease is relatively common in the sheep-rearing districts of Australasia and South America, while, for the same reason, in the British Isles, Wales shows the highest incidence.

In other parts of the world where the disease is common the life-cycle can be completed in other animals; thus pigs (not kept in styes) and occasionally horses can take the place of sheep and cattle, while in the frozen North (the disease is common among Red Indians and Esquimaux) the wolf → moose → wolf often maintain the cycle.

Pathology.—A hydatid cyst consists of two layers.

1. *The adventitia*, consisting of fibrous tissue, the result of reaction of the liver to the parasite, is grey in colour and blended intimately with the liver, from which it is inseparable.

2. *The laminated membrane* formed of the parasite itself is whitish and elastic, and contains the hydatid fluid. Indeed, this membrane closely resembles a child's uncoloured balloon filled with water, and unless bacterial infection has occurred it peels readily from the adventitia.

Hydatid fluid is crystal-clear; it registers a specific gravity of 1·005–1·009, contains no albumin, occasionally a trace of sugar, and when not too old, hooklets and scolices.

The only living part of a hydatid cyst is a single layer of cells (germinal epithelium) lining the cyst. This secretes : (*a*) internally : the hydatid fluid ;

Velarde P. Fontana, Contemporary. Professor of Surgical Pathology, Montevideo.

(*b*) externally : the laminated membrane (fig. 547 (*a*)). The laminated membrane is of variable thickness, according to the age of the cyst, and is composed of white hyaline material. The surrounding tissues of the host

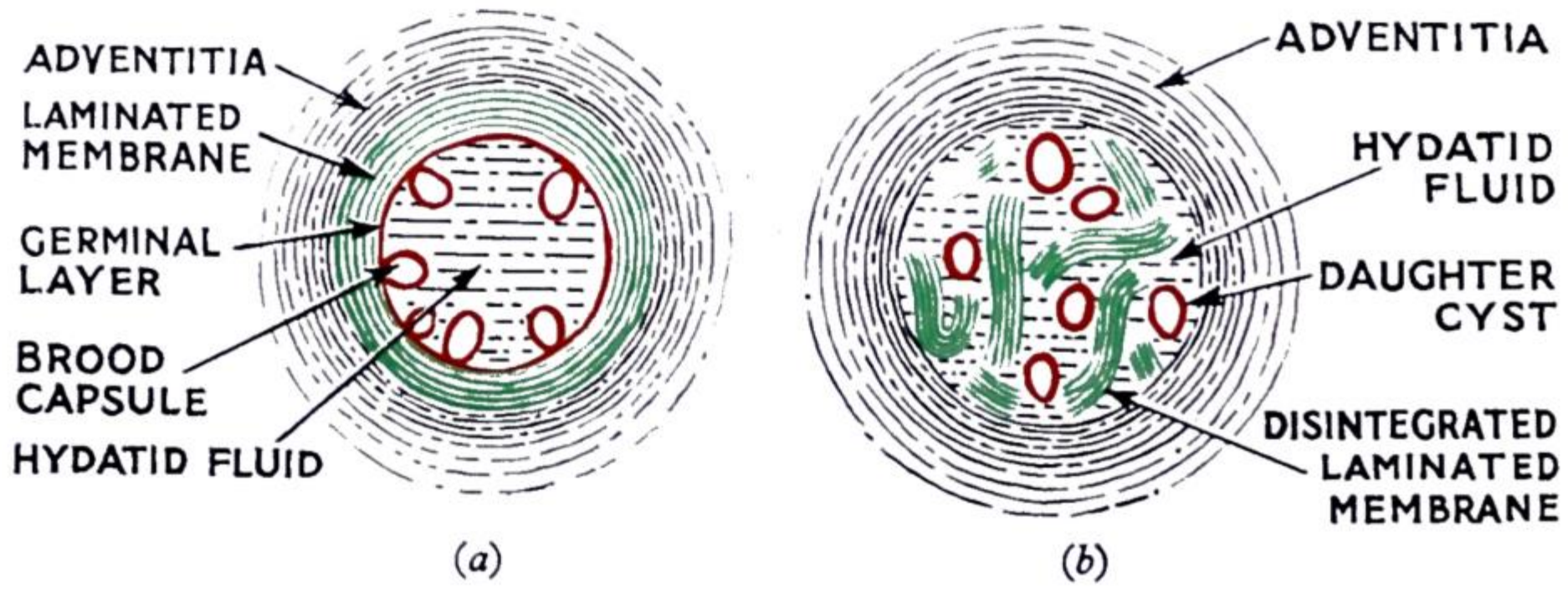

FIG. 547.—(*a*) Typical hydatid cyst.
(*b*) Development of daughter cysts (not common).

react to the presence of the parasite by entombing it in fibrous tissue—the adventitia. Referring again to the germinal epithelium : here develop brood capsules within the cyst, attached by pedicles to its innermost wall. Within the brood capsules, scolices (heads of future worms) develop. Should the laminated membrane become damaged, it disintegrates (Sir Harold Dew), and the brood capsules, becoming free, grow into daughter cysts (fig. 547 (*b*)). In this event the mother cyst ceases to exist as such, the hydatid fluid and its content being confined by the adventitia only.

A hydatid cyst grows very slowly.

Clinical Features.—For a long time, usually for many years after the original infestation, a hydatid cyst remains symptomless (fig. 548). In the course of time, owing to the frequency with which a hydatid cyst of the liver enlarges in a downward direction, a visible and palpable swelling in the upper abdomen is discovered. The size which a hydatid may attain without causing serious disturbance to health would seem to be limited only by the capacity of the peritoneal cavity.

FIG. 548. — Multiple hydatid cysts in the liver. The patient, who had never left England, died after a street accident.

In academic circles undue prominence is accorded to the hydatid thrill, a sign that is rarely present, even in advanced cases. Percussion reveals dullness over the swelling continuous with the liver dullness. When the liver is enlarged upwards by the cyst the diagnosis is much more difficult, and among other conditions a differential diagnosis must be made between hydatid cyst and an amœbic abscess.

Sir Harold Dew, Contemporary. Emeritus Professor of Surgery, Sydney.

Naturally, when a patient hails from a locality where hydatid disease is rife, the diagnosis is simplified. In obscure cases radiography and immunological tests are of great diagnostic assistance.

FIG. 549.—Casoni's test. Positive reaction.

The intradermal test (Casoni's test) is comparable to the tuberculin reaction, and is positive (fig. 549) in 75 per cent. of cases of hydatid disease.

The complement fixation test, although more complicated, is of greater accuracy.

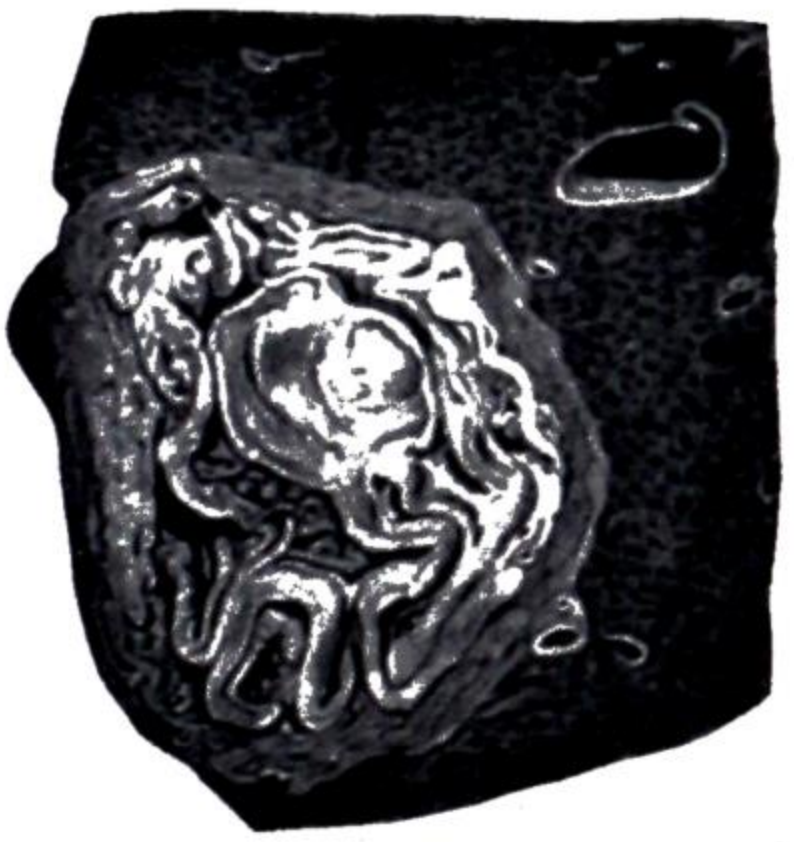

FIG. 550.—An encapsuled hydatid found at necropsy. (*The late K. A. L. Aschoff, Freiburg.*)

A blood-count often, but not invariably, shows an eosinophilia (6 per cent. or more).

Course of the Disease.—1. Occasionally the parasite dies. The fluid is absorbed, and all that remains is an encapsuled, laminated, bile-stained membrane; such is found occasionally at necropsy (fig. 550). In cases of very long standing the walls of the dead parasite calcify.

2. Usually the cyst enlarges gradually, and becomes manifest by its size. It is at this juncture that surgical intervention is indicated.

3. Complications arise. "It is the onset of complications that makes the morbidity not much inferior to that of malignant disease" (J. B. Fortacin).

Complications
- Rupture
  - Into the peritoneal cavity
  - Into the alimentary canal
  - Into the biliary channels
  - Into a pleural cavity
- Suppuration

Rupture into the peritoneum is accompanied by profound shock, and all the signs of diffuse peritonitis. A green discoloration about the umbilicus has been noted in rare instances. As with any case of rupture of a hydatid cyst, anaphylactic phenomena, notably urticaria, are prone to occur. The treatment of intraperitoneal rupture must be immediate, and directed to combating shock and cleaning the peritoneal cavity. Even in those who survive, the ultimate prognosis is poor, for if, as is usual, the cyst contains brood capsules, however meticulous the cleansing of the peritoneal cavity, the disease must tend to become disseminated within the peritoneum

TREATMENT OF HYDATID CYST OF THE LIVER

The only treatment is surgical, for there is no drug which has the slightest effect upon the course of the disease.

Tomasso Casoni, Contemporary. Physician, Ospedale Coloniale, Vittorio Emanuele III, Tripoli
José Blanco y Fortacin, Contemporary Professor of Surgery, Madrid.

Operation.—The cyst is exposed by an incision that gives the best access. Abdominal packs are arranged meticulously so as to isolate that portion of the liver containing the cyst from the peritoneal cavity ; finally, a black pack, wrung out in 2 per cent. formalin, is tucked around the exposed liver—*black* so that daughter cysts and scolices will show up against the background ; *formalin* because this antiseptic kills the parasites. The cyst is aspirated, and a suitable quantity of 10 per cent. formalin solution is injected so as to render the cyst about three-quarters full. An incision is made through the liver overlying the cyst, and the adventitia is opened. This brings the rubbery laminated membrane into view. Very gently the laminated membrane is grasped with ovum forceps and separated from the adventitia with a finger. The aim should be to separate the laminated membrane and deliver the cyst intact; usually this can be accomplished in cysts uncomplicated by the effects of past infection. In complicated cases it is sometimes necessary to remove the laminated membrane piecemeal. When the intact cyst can be enucleated, the resulting cavity in the liver can be closed completely. In less favourable circumstances it is advisable to drain the cavity, particularly when infection is present, when the contents of the cyst are bile stained, or when there is uncertainty of its complete removal. No attempt should be made to remove the adventitia. I have seen the liver irreparably split during an ineffectual attempt to excise an adventitia in the belief that it was the laminated membrane; the presence of daughter cysts (see p. 408) and consequent difficulty in emptying the cyst satisfactorily should at once call to mind the fact that a laminated membrane is likely to be non-existent. In all cases full precautions must be taken to prevent spilling the contents of the cyst into the peritoneal cavity or the layers of the abdominal wall, for only if these measures are taken can dissemination of the disease be prevented.

NON-PARASITIC CYSTS OF THE LIVER

A solitary traumatic cyst usually follows abdominal trauma and the symptoms are delayed weeks or months after the injury. The clinical manifestations include abdominal pain, frequently referred to the shoulder, a lump that is usually palpable, and less frequently jaundice. In most instances the cyst is located in the right lobe and contains blood and bile. Drainage is followed by excellent results.

A localised collection of cysts, one or several being larger than the others, is probably the result of sequestration of bile ducts during fœtal life. Usually excision of the mass can be performed comparatively easily in the same manner as a neoplasm is excised (see p. 425).

Congenital polycystic disease of the liver is rare, and a third of the cases are associated with congenital polycystic kidneys. Slow, irregular enlargement of the whole liver becoming manifest in adult life is the only clinical feature. There is no reason why Rovsing's operation (see Chapter 32) should not be performed in necessary cases. Associated polycystic disease of the kidneys carries a greater threat to life than polycystic disease of the liver.

CHIARI'S SYNDROME

Chiari's syndrome results from obstruction to the hepatic veins. Neoplastic encroachment from other organs accounts for the majority of cases. Other causes are congenital obstruction and thrombosis occurring in the veins, usually due to extension of inflammation from other organs.

Clinical Features.—*In acute cases*, nausea, vomiting and severe pain, due to rapid enlargement of the liver as a result of congestion, are often followed by death from hypotensive shock.

A less sudden onset is characterised by rapidly accumulating ascites and signs of portal hypertension with a developing collateral circulation. Signs of hepatic insufficiency are usually pronounced, and early death and hepatic coma is the rule. The simultaneous development of œdema of the legs testifies to involvement of the inferior vena cava in the occlusive process.

Chronic cases closely resemble portal cirrhosis which, indeed, may develop. Liver function tests indicate severe hepatic parenchymal damage, and rarely does the patient survive more than a few months. A few patients who do not die from hepatic coma are carried off by hæmorrhage from œsophageal varices, mesenteric infarction, or inter-current infection. In short, Chiari's syndrome, when the occlusion is, or becomes, complete, must be looked upon as a harbinger of death.

Hans Chiari, 1851–1916. Professor of Pathological Anatomy, Prague.

PRIMARY BILIARY CIRRHOSIS (*syn.* HANOT'S CIRRHOSIS)

Biliary cirrhosis is considered to be rare, but probably lack of familiarity with it prevents its recognition.

Pathology.—The surface of the liver is coarsely bosselated (fig. 551). On section fibrosis is situated mainly around the ductules of the biliary tree.

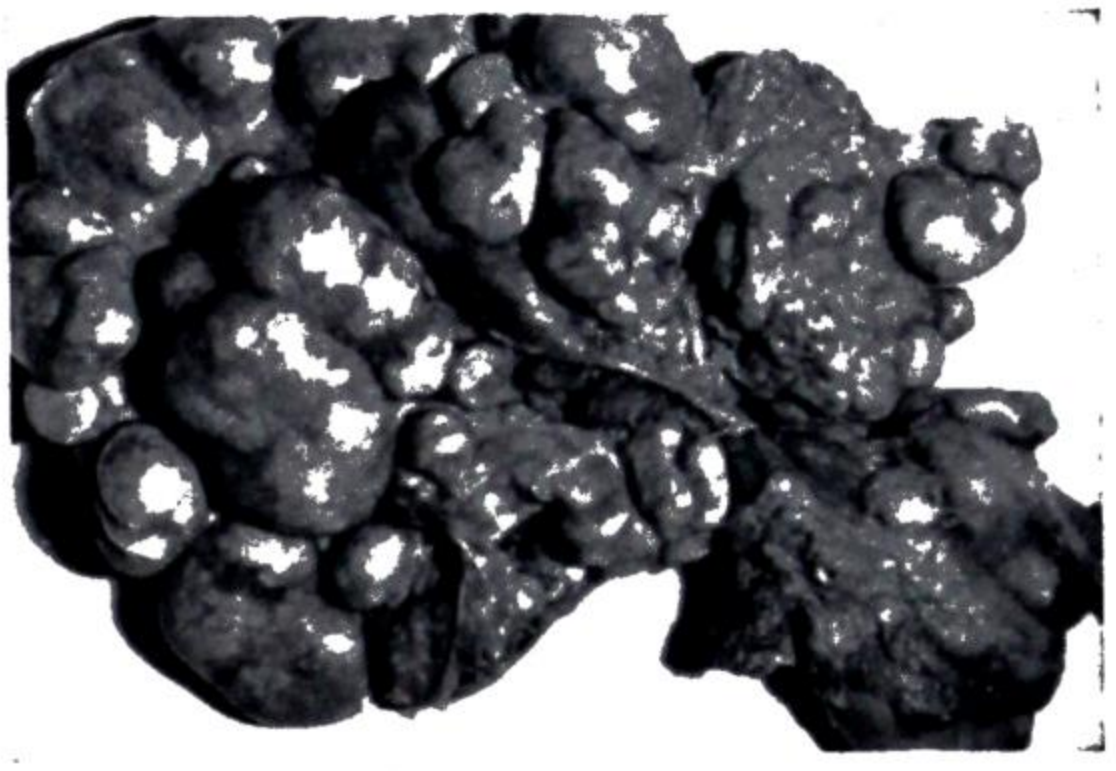

Fig. 551.—Primary biliary (Hanot's) cirrhosis. Note the coarse lobulation. (*Dr. J. M. Craig, Boston, Mass., et al.*) (*American Journal of Diseases of Children.*)

Ætiology is often obscure, but mucoviscidosis of the intrahepatic ducts associated with fibrocystic disease of the pancreas (see p. 461) is a proven cause, and a sweat test is invaluable to differentiate biliary cirrhosis from this cause from all other varieties of cirrhosis of the liver. Three stages are recognised (I. W. MacPhee).

Clinical Features.

Stage 1 is that of obstructive jaundice. It may persist for months or years with varying intensity. The liver is palpable in the middle line, is very hard, has a sharp margin, and it is irregular and not tender. The obstruction to the biliary tree is intrahepatic, but there is no reliable biochemical or other test to help to distinguish intrahepatic from extrahepatic obstructive jaundice. Consequently laparotomy and exploration of the bile-ducts is often undertaken with negative results. Cholangiography helps to disprove extrahepatic obstruction.

Stage 2. Portal hypertension develops. During the whole of the stage, and the next, jaundice persists. If the patient survives repeated hæmatemeses, eventually he or she (it is somewhat more common in women) passes into:

Stage 3. Hepatic failure.

The progress of this disease, though often slow, is relentless and irreversible. The first stage can last many years. Once portal hypertension has developed fully, stage 3 is not far distant.

Treatment.—Only symptomatic treatment is possible. Corticotrophin (ACTH) is sometimes followed by rapid re-establishment of bile flow for a time. When signs of portal hypertension develop, a portacaval shunt is probably unjustified in cases of *Hanot's* cirrhosis, as it will have no effect on the jaundice and eventual liver failure.

SECONDARY BILIARY CIRRHOSIS

The commonest cause in children is congenital atresia of the bile-ducts (p. 432), irremediable by operative means. The jaundiced child soon shows signs of hepatomegaly, splenomegaly, ascites, and varices. Most survive only a few months; a few up to three or more years. In adults the condition can arise following post-operative stricture of the common bile-duct, or possibly from obstruction of the bile-duct due to some other cause.

Victor Charles Hanot, 1844–1896. Physician, Hôpital Saint-Antoine, Paris.
Ian Weir MacPhee, Contemporary. Senior Lecturer in Surgery, University of Liverpool.

There is no biochemical test that will help to distinguish extra- from intra-hepatic biliary obstruction, and as a rule, laparotomy with exploration of bile-ducts and cholangiography is necessary to distinguish primary from secondary biliary cirrhosis.

PORTAL CIRRHOSIS (*syn.* LAËNNEC'S CIRRHOSIS; HOB-NAIL' LIVER)

Portal cirrhosis is essentially a chronic inflammation of the liver accompanied by fibrosis around the radicles of the portal vein.

Ætiology.—It is convenient to divide the ætiological factors into those occurring in infancy and childhood, and those occurring in adult life. It will be understood that some of the factors appertaining in early life take years to manifest themselves as portal cirrhosis.

Causes in Infants and Children:

1. *Nutritional.* (*a*) Infantile portal cirrhosis seen in India occurs mainly in high-caste Hindu vegetarians. It often progresses rapidly to an early fatal termination. It is suggested that the condition is due to a susceptibility of protein-deficient children to massive necrosis of the liver during viral hepatitis. (*b*) Kwashiorkor in Africa, and fatty disease of the liver in West Indies is characterised by an enlarged liver and peripheral nutritional œdema. Patients with this disorder develop cirrhosis, if at all, only during early adult life. (*c*) Galactosæmia. Patients with this inborn error of metabolism who continue to take lactose invariably develop portal cirrhosis.

2. *Infective.*—A history of severe jaundice in early infancy is obtainable in a number of instances. Virus hepatitis is believed to play a part in the production of portal cirrhosis.

3. *Cardiac Cirrhosis.*—Chronic venous congestion of the liver due to tricuspid incompetence or constrictive pericarditis is the cause.

4. *Erythroblastosis.*—Isolated instances have occurred.

Causes in the Adult:

1. *Alcohol.*—Over-indulgence in alcoholic beverages is, as is well known, a leading cause.

2. *Virus Hepatitis.*—The exact percentage of cases in which portal cirrhosis is an aftermath of virus hepatitis is debatable, and it varies in different localities.

3. *Infestation of the liver by schistosomiasis* undoubtedly is a principal cause of the condition in districts where schistosomiasis is rife.

4. *Nutritional Deficiency.*—In the tropics this appears to be a major factor. Its mode of action is as follows: Some toxic agents and mild infections, harmless to the well-nourished, are liable to have a deleterious effect on the liver of protein-deficient individuals. Therefore protein deficiency is indirectly responsible for the production of portal cirrhosis.

5. *Unknown.*—In many instances the cause is obscure.

Pathology.—As a result of widespread parenchymal destruction and overgrowth of fibrous tissue, the tiny radicles of the portal and hepatic veins become compressed and distorted (fig. 552). Arterioles resist this compression for a longer time than veins. As a consequence the cirrhotic liver (fig. 553) becomes dependent upon the hepatic artery for the major portion of its blood supply, and in the desperate effort to maintain the circulation through the liver in these adverse circumstances, extensive intrahepatic communications develop between branches of the hepatic artery and the portal vein, and between the tributaries of the portal vein and the hepatic vein.

Nature's intrahepatic shunts, combined with the opening up of the better-known extrahepatic porto-systemic communications, divert a large portion of

René Théophile Hyacinthe Laënnec, 1781–1826, Professor of Medicine, Collège de France, invented the stethoscope in 1819.

the portal blood past the hepatic parenchyma into the systemic venous system. Thus the remaining hepatic cells are deprived of their fair share of portal blood.

Clinical Features.

First Stage.—The early stages of portal cirrhosis are quiet and long. The liver hypertrophies. In some cases there are repeated attacks of slight jaundice with epigastric pain and vomiting. At each attack areas of the liver are destroyed and replaced by fibrous tissue, while the remainder hypertrophies. Hepatomegaly without splenomegaly is rarely due to portal cirrhosis.

FIG. 552.—A, Cast of branches of a normal portal venule; B, Those of the branches of a portal venule of a cirrhotic liver, showing their attenuation and distortion. (*After I. S. Ravdin.*)

FIG. 553.—In portal cirrhosis, as the liver contracts it becomes *finely* lobulated, giving it a hob-nail appearance.

Second Stage.—The liver commences to contract. The spleen remains enlarged, and usually increases in size *pari passu* with the increase in portal hypertension, of which portal cirrhosis is the principal cause in adults. About this time, especially in alcoholic subjects, there often develops:

Spider nævi,[1] which are usually located around the face, neck, shoulders and the upper arms. Histologically they prove to be an overgrown end-artery with branching arterioles ⟶

Pulsations can be detected with a magnifying glass.

Palmar erythema is less commonly encountered than the foregoing. The hands are warm, and the palms are bright red, more especially in the region here depicted ⟶

Third Stage.—Bleeding from œsophageal varices (see p. 416) occurs in a high proportion of cases. Not infrequently the superficial veins radiating from the umbilicus enlarge, forming a Caput Medusæ. Progressive ascites occurs (fig. 554). Testicular atrophy is common in portal cirrhosis. It is no doubt due to increase of œstrogens in the circulation consequent upon

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[1] An older Miss Muffet
Decided to rough it
And lived upon whisky and gin.
Red hands and a spider
Developed outside her—
Such are the wages of sin.
(*W. B. Bean.*)

William Bennett Bean, Contemporary. Professor of Internal Medicine, Iowa City University Hospitals, Iowa.
Medusa, one of three Gorgons whose fine hair was turned into snakes (Greek mythology).

FIG. 554.—Portal cirrhosis with ascites.

the inability of the failing liver to neutralise these substances, as also is the gynæcomazia which occasionally accompanies this disease.

In women there is a tendency to masculinisation.

Fourth Stage.—Once hepatic decompensation has occurred (if the patient escapes death from torrential hæmorrhage from œsophageal varices), liver failure, like a sword of Damocles, sooner or later falls upon the sufferer from this disease.

Liver Function Tests.—A most useful biochemical method is the bromsulphalein retention test. Additional useful biochemical indications are the constant presence of urobilinogen in the urine and a slightly raised serum bilirubin, globulin, or thymol turbidity level. If the results are equivocal, recourse must be made to:

Liver biopsy is usually necessary only for the diagnosis of a well-compensated case to confirm the diagnosis and assess prognosis.

Treatment.—Medical treatment can do much for portal cirrhosis, but nothing for portal hypertension.

Hepato-lenticular degeneration (*syn.* Wilson's disease) is an uncommon condition that is confined to children and young adults. It is believed to be hereditary. In infants the symptoms are those of portal cirrhosis. In adolescents a coarse tremor is commonly an initial symptom. As the disease progresses, muscular rigidity dominates the picture. Concurrently, inability to close the mouth, accompanied by a fixed grin and drooling, is a distressing symptom. Liver function tests give varying results. Liver biopsy shows portal cirrhosis. A fatal termination ensues in six months to two years.

In cases developing during the third decade (the disease seldom manifests itself later) evidence of portal hypertension is often conspicuous and, in spite of other manifestations of the disease, the advisability of performing portacaval shunt sometimes arises.

PORTAL HYPERTENSION

Ætiology.—The obstruction can be (1) Pre-hepatic; (2) Intra-hepatic; (3) Post-hepatic.

1. **Pre-hepatic.**—About 20 per cent. of patients belong to this group, most of whom are children. The obstruction arises in one of two ways:

(*a*) *There is congenital absence of the portal vein* or an extension of the normal post-natal obliterative process of the umbilical vein and ductus venosus (fig. 555).

(*b*) *Thrombosis of the portal vein* (fig. 556) is commonly due to propagation of a thrombus along the umbilical vein as a result of omphalitis of the new-born (see Chapter 29).

In many cases of pre-hepatic portal obstruction the vein, as such, is absent,

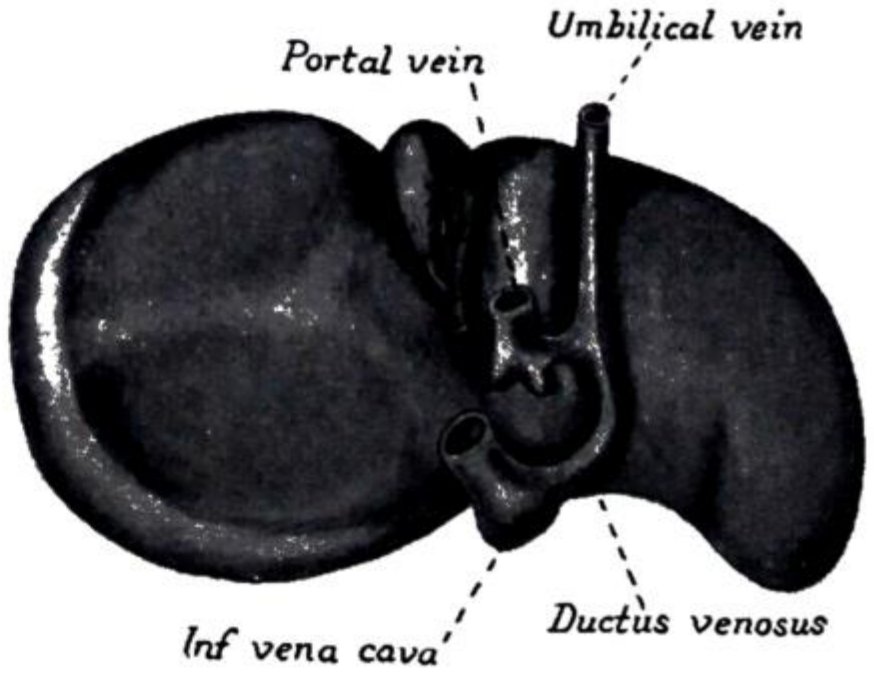

FIG. 555.—Hilum of the liver of a full-term fœtus. (*After R. E. Gross.*)

Damocles, a Greek who was feasted with a sword suspended above his head by a single hair (Greek mythology).
Samuel Alexander Kinnier Wilson, 1877–1937. Neurologist, King's College Hospital, London.

and in its stead there is a cavernous hæmangioma (cavernoma). Whether this cavernoma is due to recanalisation following thrombosis, or to a congenital abnormality, is unsettled.

2. **Intra-hepatic** is by far the most common variety. It accounts for nearly 80 per cent. of all cases. The cause is cirrhosis of the liver, in particular by areas of regeneration in cases of compensated portal cirrhosis.

3. **Post-hepatic** is extremely rare. The Chiari syndrome has been discussed on p. 410. Post-hepatic obstruction is also caused by constrictive pericarditis and tricuspid valvular incompetence.

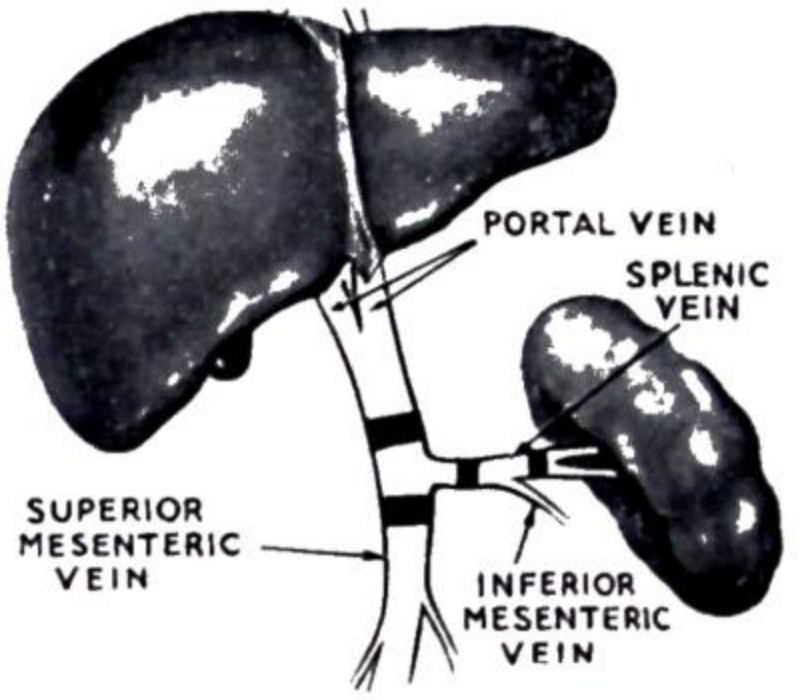

FIG. 556. — Sites of obstruction. The blocks in the veins represent possible sites of thrombus formation. (*After Sir James Learmonth.*)

COLLATERAL CIRCULATION

When there is obstruction of blood flow to or from the liver, Nature endeavours to relieve the obstruction by opening up normally insignificant anastomotic channels.

(*a*) *When the obstruction is pre-hepatic*, collaterals between the portal vein distal to the obstruction and the portal vein proximal to the obstruction enlarge. Thus almost unheard of venæ comitantes of the hepatic artery and the venæ comitantes of the portal vein wax proud. Depending upon the site of the obstruction, some of the collaterals between the portal and systemic

ANASTOMOSES BETWEEN THE PORTAL AND SYSTEMIC VENOUS SYSTEMS

	Site of Anastomosis	*Portal Vessels*	*Systemic Vessels*	*Signs and Symptoms*
1	Plexus around lower end of œsophagus.	Œsophageal branches of left gastric vein.	Lower systemic œsophageal veins.	Hæmatemesis or mclæna.
2	Around umbilicus.	Para-umbilical veins (accompany the round ligament of the liver).	Superficial veins of the anterior abdominal wall.	Caput Medusæ.
3	Plexuses around lower third of rectum and anal canal.	Superior hæmorrhoidal vein.	Middle and inferior hæmorrhoidal veins.	Bleeding internal hæmorrhoids.
4	Veins of Sappey.	Portal venules on the bare area of the liver.	Subdiaphragmatic veins.	Silent.
5	Venæ vasa brevia.	Superior pole of the spleen.	Subdiaphragmatic veins.	Silent.
6	Extraperitoneal surfaces of the colon.	Tributaries of the inferior mesenteric vein.	Retroperitoneal veins.	Silent.

Marie Philibert Sappey, 1810–1896. Professor of Anatomy, Faculty of Medicine, Paris.

venous systems on p. 415, notably the œsophageal plexus, also become dilated.

(*b*) *When the obstruction is intrahepatic*, first *intrahepatic* communications between the portal and systemic venous channels that are, as yet, unobliterated, expand. When this process of intrahepatic shunting becomes insufficient to meet the demands, *extrahepatic* anastomotic channels between the portal and systemic venous systems become engorged, dilate, and so an increasing proportion of the obstructed portal venous blood by-passes the liver.

Although, theoretically, dilated ano-rectal veins should be a common accompaniment of portal hypertension, in practice the relationship between them appears to be casual (A. I. Macpherson). So frequently do œsophageal varices accompany portal hypertension, and so menacing is the hæmorrhage to which they give rise, that it has been considered wise to describe in all their aspects œsophageal 'piles', as they are sometimes called, before proceeding further with the important subject of portal hypertension.

ŒSOPHAGEAL VARICES

Although called 'œsophageal,' it is important to realise that these varices extend well into the stomach. Œsophageal varices (fig. 557) are the most important collaterals of the portal circulation. At the same time they are by far the most dangerous. Sometimes, as intra-abdominal pressure rises, i.e. when straining at stool or during heavy lifting, they burst, or what occurs

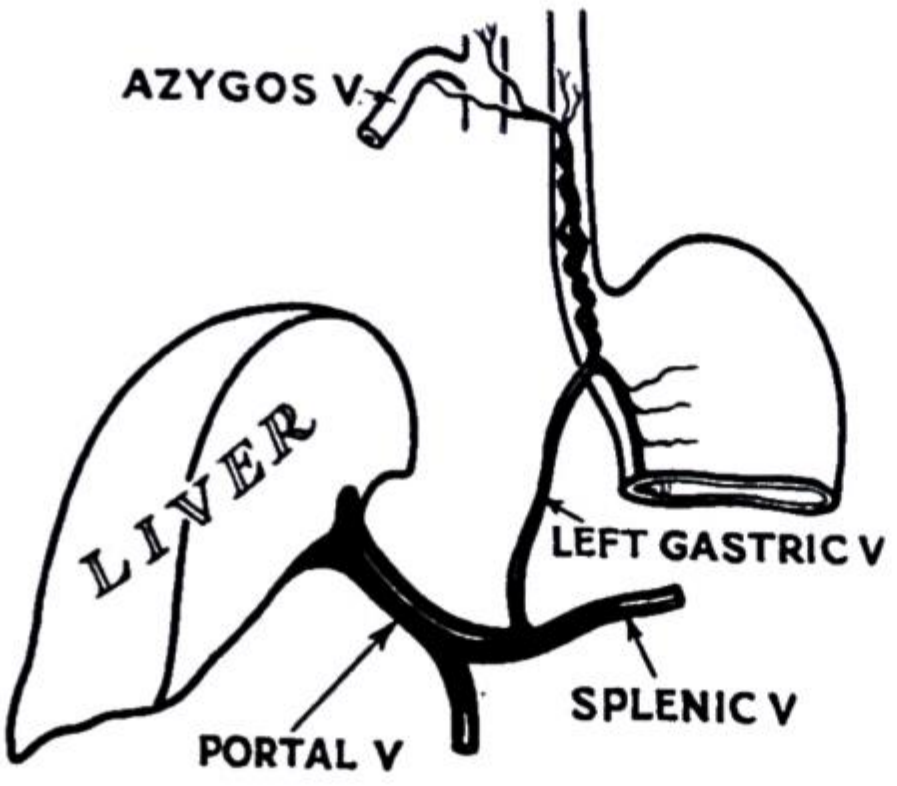

FIG. 557.—Œsophageal varices. FIG. 558.—Barium swallow displaying filling defects due to œsophageal varices. (*Mr. A. H. Hunt, London.*)

more frequently is that the overlying mucosa becomes abraded by a rough bolus. Bleeding can occur slowly or catastrophically. Prior to catastrophic hæmorrhage the presence of œsophageal varices can be demonstrated by:

(*a*) *Radiology* after a barium swallow (fig. 558). In only 40 per cent. of cases can even well-developed œsophageal collaterals be revealed by this method. The varicosities can be rendered radiologically apparent more often by swallowing lipiodol (J. F. Brailsford).

(*b*) *Œsophagoscopy* is more reliable, but great care must be exercised not

Archibald Ian Stewart Macpherson, Contemporary. Surgeon, Royal Infirmary, Edinburgh.
James Frederick Brailsford, Contemporary. Emeritus Director of Radiological Studies in Living Anatomy University of Birmingham.

to abrade the overlying mucous membrane. To this end, once tortuous veins are seen, the œsophagoscope is not advanced, but withdrawn.

Clinical Features.—If the patient is known to have hepatic cirrhosis, it should be assumed that the hæmorrhage is coming from œsophageal varicose veins, although it is by no means a curiosity for a patient with cirrhosis of the liver to suffer also from a gastric or duodenal ulcer. However, hæmorrhage from the œsophagus is often preceded by one or two effortless regurgitations of blood, before blood is vomited. Radiography and œsophagoscopy in the presence of severe hæmorrhage is entirely out of place, for not only is it waste of valuable time, but so often it is inconclusive. Liver function tests, especially the bromsulphalein test, if positive, greatly favour hepatic cirrhosis as the cause of the hæmorrhage.

The presence of engorged œsophageal varices is one of the greatest indications that, if feasible, a portacaval shunt should be carried out without undue delay. So often catastrophic hæmorrhage comes like a thief in the night while the patient is doing well under medical treatment. The very fact that he is doing well implies thsat regeneration of areas of the liver is in progress Regeneration nearly alway entails increased pressure on the intrahepatic. portal network, and so another patient has a massive hæmorrhage which could have been averted by a timely portacaval shunt.

Treatment of Massive Hæmorrhage from Œsophageal Varices:

1. **Purely Conservative Treatment.**—Rest, sedation, and replacement of blood will carry approximately half the patients who are bleeding actively past the emergency period. Pituitrin, which causes lowering of the portal blood pressure, is a valuable adjunct. To obtain the maximum effect, 20 units of obstetric pituitrin diluted in 200 ml. of isotonic saline solution is given intravenously over a period of twenty minutes. Constriction of the splanchnic arterioles is the most logical explanation of the diminution of the portal blood pressure following the administration of pituitrin.

For the prothrombin deficiency consequent upon liver damage, the administration of vitamin K_1 is an absolute necessity.

2. **Tamponade** is required when the foregoing measures fail after a short trial, or they are unlikely to succeed. A child's small balloon tied firmly to the end of a gastric aspiration tube can be employed, but the Sengstaken trilumen tube (fig. 559) is better. The œsophageal and gastric balloons are inflated and moderate tension applied by strapping the tube to the cheek. While the Sengstaken tube causes œsophageal hæmorrhage to cease in every case, and is therefore a diagnostic as well as a therapeutic measure, it has its drawbacks. Chief of these is the inspiration of saliva into the lungs, which must be guarded against carefully. Mechanical respiratory difficulty has been known to cause ulceration and rupture of the œsophagus if the balloon

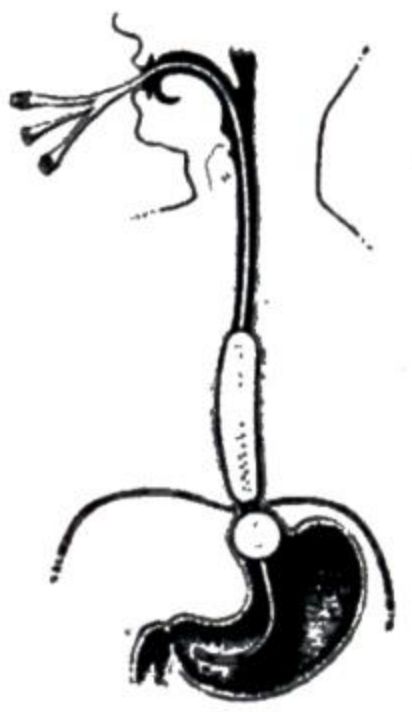

FIG. 559.—The Sengstaken trilumen tube with gastric and œsophageal balloons *in situ*.

Robert William Sengstaken, Contemporary. Surgeon, Garden City, New York.

is left in more than seventy-two hours. However, the greatest danger is renewed hæmorrhage when the balloon is deflated and withdrawn. Therefore, after twenty-four to forty-eight hours the balloon should be deflated, but left in place for four hours. If bleeding recommences there is but one real chance of saving the patient's life:

3. **Urgent Ligation of Œsophageal Varices.**—This is undertaken most rapidly and with least disturbance to the patient by the abdominal route. The lower end of the œsophagus is approached through a midline incision, and after the xyphoid process has been excised, the steps of the operation are precisely those of Heller's operation (see p. 314). The balloon is not deflated until the œsophagus and the first inch of the stomach have been opened. Two or three bunches of veins, bearing a remarkable resemblance to internal hæmorrhoids, present, and each is under-run by a continuous locking suture. The œsophagus is closed in two layers, the outer being of closely-placed unabsorbable, interrupted sutures. The abdomen is then closed, and blood transfusion is continued as long as necessary.

4. **Evacuation of Blood from the Alimentary Tract.**—As soon as possible the alimentary tract must be cleared of blood; otherwise encephalopathies and hepatic coma, due to the absorption of protein metabolites, will follow. All unclotted blood must be aspirated from the stomach, and hourly doses of milk of magnesia must be given by mouth or through the indwelling tube, followed by enemas and colonic wash-outs until there is every reason to believe that all blood has been evacuated.

5. **Diet** is similar to that recommended for hepatic coma (see p. 394).

It should be understood that ligation of œsophageal varices is not an operation for portal hypertension, but an emergency measure that, if successful, will give three weeks' grace to prepare the patient for an elective portacaval shunt.

Portal Hypertension *au Froid*

By this is meant that the patient has portal hypertension, but is not bleeding obviously or dangerously. First let us consider how pre-hepatic portal obstruction differs from intrahepatic portal obstruction.

FIG. 560.—Enlargement of the spleen in pre-hepatic portal obstruction.

The patient with pre-hepatic portal obstruction is nearly always young, and often a child. The most usual cause for advice being sought is listlessness due to anæmia. On examination the liver is impalpable, but the spleen is enlarged obviously (fig. 560). The anæmia is usually due to oozing from œsophageal varices, but sometimes the major factor is hypersplenism,[1] in

[1] Hypersplenism = increased activity of the spleen in the destruction of erythrocytes, platelets, and often leucocytes as well. Cf. Splenomegaly = enlargement of the spleen.

Positioning and Incision.—Fixation of the patient on the operating table in a position akin to that adopted at the start of a hornpipe dance (fig. 565), meets all contingencies likely to be encountered in respect to positioning. In particular, when a portacaval anastomosis proves to be impracticable, and access is required to the contralateral side in order to perform a spleno-renal shunt, the right oblique incision can be extended to expose the spleen (fig. 566).

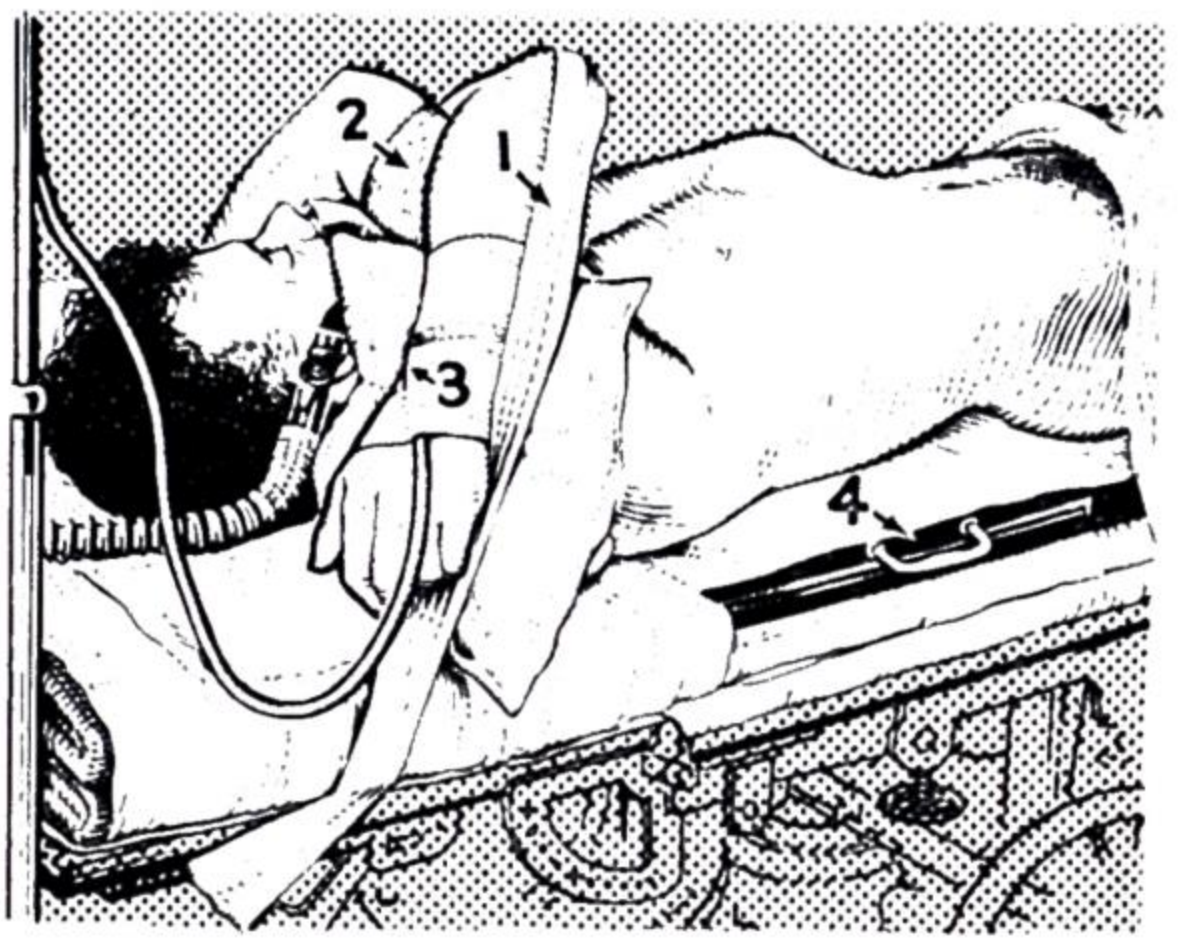

FIG. 565.—Hornpipe position. 1, Arms fixed in flexion with elastic adhesive strapping; 2, Blood-pressure cuff on right upper arm; 3, Needle of intravenous apparatus in a vein on the dorsum of the left hand; 4, Cassette tunnel for venography. (*After A. H. Hunt et al.*)

After the abdomen has been opened, ascitic fluid, if present, is evacuated. The liver is examined, and when necessary the following investigation is undertaken:

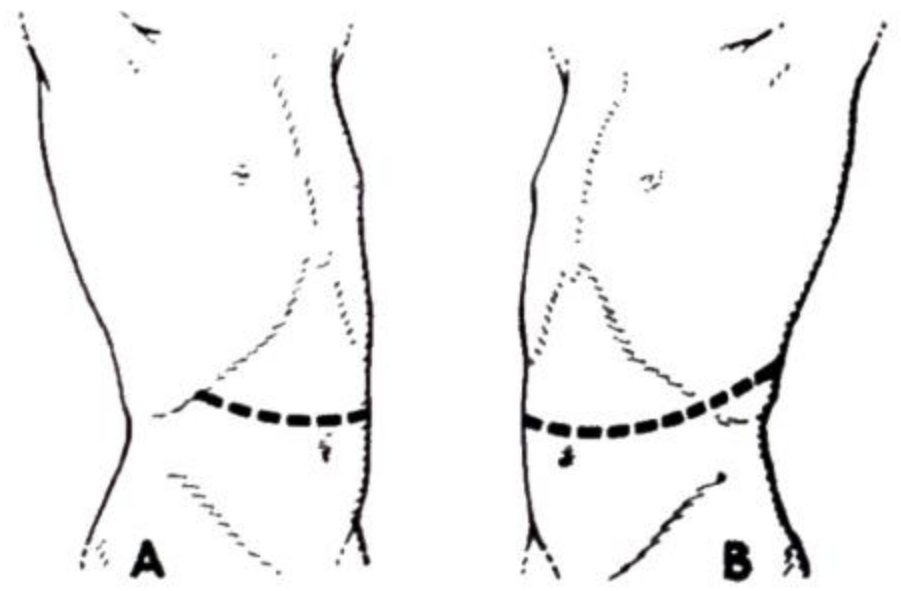

FIG. 566.—Oblique incision (A) for portacaval anastomosis. Should this prove impossible, (B) extension to the left for spleno-renal anastomosis. (*After A. H. Hunt et al.*)

Portovenography.—When the spleno-portographic films are inconclusive, or have not been obtainable because the spleen has been removed previously, the site of the block can be demonstrated by portography after the abdomen has been opened. Thirty ml. of a 35 per cent. solution of diodone is injected into a suitable vein in the gastro-hepatic omentum, or into the superior mesenteric vein. A radiograph is then taken with a portable machine during the injection of the last few ml., and with the help of the venograph thus obtained, usually the site of the block can be determined accurately.

1. **Splenectomy *per se*.**—When the obstruction is limited to the splenic vein—a rare event—splenectomy is likely to be beneficial, but subsequent bleeding from œsophageal varices will be obviated only when the venous obstruction is limited to the distal end of the splenic vein, or when the left gastric vein opens directly into the portal vein (fig. 567). Indisputable evidence of hypersplenism is a strong indication or splenectomy, if necessary, in addition to a portal-systemic shunting operation.

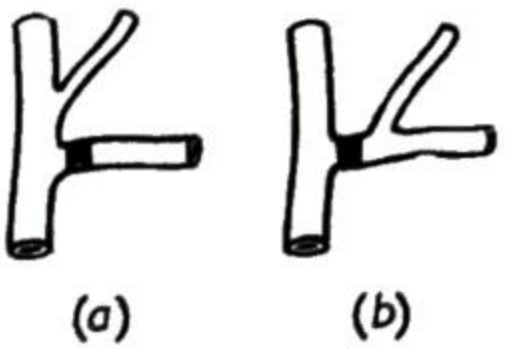

FIG. 567.—Left gastric vein opening into (*a*) the portal vein, (*b*) the splenic vein proximal to the obstruction.

2. **Portacaval Anastomosis.**—Unless the portal vein is represented by a cavernous hæmangioma (which renders a portacaval anastomosis impossible) a portacaval shunt is nearly always the operation of choice.

End-to-side Portacaval Anastomosis.—After incising the peritoneum over its lateral border, the second part of the duodenum is mobilised towards the middle line, together with the common bile duct, until the portal vein and the inferior vena cava are exposed at the place they run side by side (fig. 568). The portal vein is freed from the common bile duct and ligated close to the portal fissure. A vein clamp having been placed on it about ¾ inch (18 mm.) below the ligature, the portal vein is divided below the ligature. Blalock's angled vena cava clamp, shown in the lower part of fig. 568 inset, is so placed on the inferior vena cava that it occludes only half, or little more than half, of the lumen of the vein.

FIG. 568.—Portacaval anastomosis.
(After J. Auvert.)

The distal end of the portal vein is anastomosed to the inferior vena cava, after cutting an oval window in the latter. Using a continuous everting suture of fine silk, one half of the circumference is completed with one suture and the other half by another, so as to bring intima to intima and avoid a purse-string effect with consequent narrowing. Local bathing with heparin solution during the operation helps to prevent subsequent thrombosis.

In many cases, when the anastomosis is completed, the turbulence set up by the inflow of portal blood can be seen through the thin wall of the inferior vena cava (R. Milnes Walker).

The mortality of this operation is about 8 per cent., the main cause of death being hepatic coma (see p. 393).

3. Spleno-renal anastomosis carries a lower mortality than portacaval anastomosis, but the anastomosis between vessels of smaller calibre is more likely to become occluded by clot or progressive fibrosis, and so be effective only for a short time. Because it is an operation of less severity than a portacaval shunt, spleno-renal anastomosis is suited particularly to young children. After an interval, should it prove to be insufficient or ineffectual, it does not preclude portacaval anastomosis being performed at a later date.

End-to-side Spleno-renal Anastomosis.—The tributaries of the splenic vein leaving the hilum of the spleen are separated carefully, with a view to selecting the longest of adequate calibre. This vein is dissected, ligated, and divided as close to

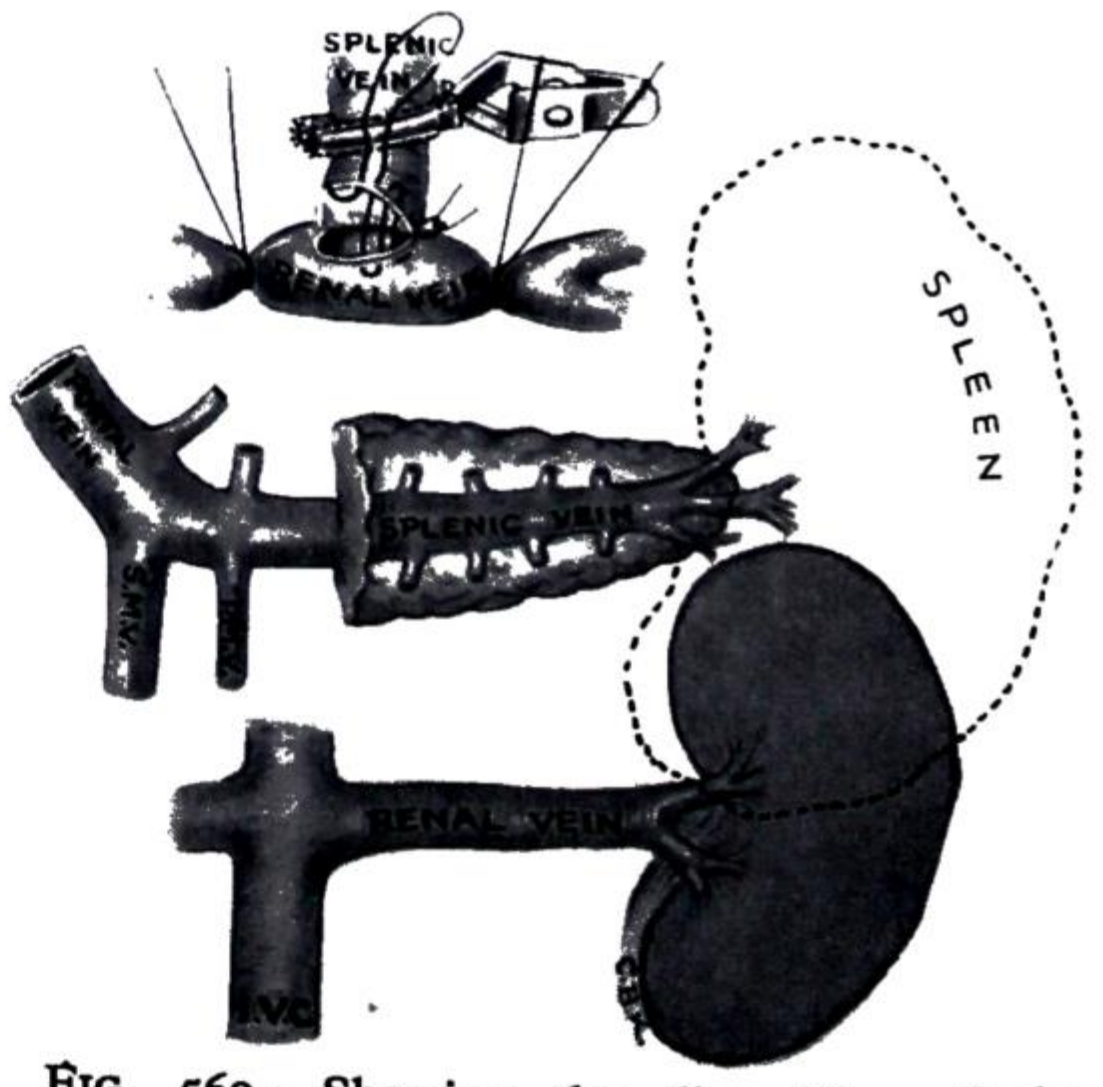

FIG. 569.—Showing the disposition of the splenic and left renal veins. Inset: end-to-side anastomosis being performed. *(After L. M. Roussellot.*

Alfred Blalock, Contemporary. Surgeon-in-Chief, Johns Hopkins Hospital, Baltimore.
Robert Milnes Walker, Contemporary. Professor of Surgery, University of Bristol.

the spleen as possible, the proximal end being occluded for the time being in a rubber-covered bulldog or Blalock's vein clamp. Splenectomy is performed. The difficult task of freeing the splenic vein from the back of the pancreas by tying and dividing the numerous pancreatic tributaries (fig. 569) is completed for a distance of 3 inches (7·5 cm.) (A. H. Blakemore), so as to permit the mouth of the splenic vein to be brought to the renal vein without tension. The renal vein is cleared of surrounding fat, and the method of occluding it temporarily, shown in fig. 569 inset, will not damage the renal parenchyma, whereas if the artery is occluded with the vein, irreversible changes occur. The anastomosis is completed with everting sutures, as has been described. When the splenic vein is too short to reach the renal vein without tension, a vein graft from the superficial femoral vein has been used to bridge the gap with success. In all cases the operation area must be drained, for seepage of ferments from the pancreas is liable to occur.

Portal-systemic Shunting : Post-operative Treatment.—Oxygen is given for the first twenty-four to forty-eight hours, and during that time the patient is supported with 5 per cent. dextrose solution intravenously, until the oral intake is sufficient, careful watch being kept on the electrolytic balance. Any major shunt, by by-passing portal blood from the liver, increases the liability to hepatic coma and neuro-encephalopathy. The main *late complication* is the sudden development of neuro-encephalopathy (see p. 393), which occurs most often soon after the patient has returned home. Usually there is a quick response to a protein-free diet. In most instances a gradual return to a normal diet can be permitted ; in a few, attacks of episodic stupor dictate that a supervised dietitic régime must continue. At all times ammonia-containing medicine must be avoided rigorously. Serial post-operative liver function tests show that diversion of the portal blood-stream from the liver does not cause greater deterioration in hepatic function than that occurring in comparable controls.

4. When the patient cannot be rendered fit for a portal-systemic shunting procedure, rather than abandon hope, sometimes it is desirable to make an effort to supplement the collateral circulation by other means. A number of remarkable results have followed the ingenious device of:

Promoting a Subdiaphragmatic Collateral Circulation.—Through an upper laparotomy incision the superior surface of the liver and the inferior surface of the diaphragm are abraded with coarse sandpaper. Magnesium trisilicate powder (talc) is distributed over the abraded liver surface, and vascular adhesions between the diaphragm and the liver, as well as the diaphragm and the spleen, result. In the circumstances defined, when a major shunting operation is out of the question, this method of treatment, because of its simplicity, is appealing.

NEOPLASMS OF THE LIVER

Benign:

Hæmangioma occurs more commonly in the liver than in any other internal organ, and usually the neoplasm is of the cavernous type. As a rule, a hæmangioma of the liver is solitary, and is found either at necropsy or incidentally at operation. Exceptionally, it becomes large enough to cause symptoms, in which event laparotomy is required to establish the diagnosis. The compressibility of the tumour makes the diagnosis unmistakable, and on no account should biopsy be performed, for the resulting hæmorrhage is terrific. The ideal treatment is to excise the tumour with a shell of normal liver tissue around it, but if the surgeon feels that this would be a hazardous undertaking, he can take comfort in the knowledge that the size of the tumour can be reduced by X-ray therapy.

Hepato-adenomata are composed of hepatic cells. Frequently multiple, they are distinguished with difficulty from the nodular hyperplasia of cirrhosis. They seldom grow to a large size and there is no evidence that they become malignant. Their removal is meddlesome, and unnecessary.

Cholangio-adenomata originate from the bile-ducts and form small subcapsular masses. They are found at operation or necropsy, and can simulate closely metastatic deposits, which should not be assumed without biopsy. The same remarks regarding extirpation as were expressed for hepato-adenomata hold good for these neoplasms.

Cholangio-hepato-adenoma originates from both hepatic cells and bile-ducts,

Arthur Hendley Blakemore, Contemporary. Associate Professor of Clinical Surgery, Columbia University, New York.

Treatment.—Laparotomy should be performed when the patient's general condition is good and the swelling is solitary. If, at operation, the tumour is found to be firm in consistency, indicating that it is a relatively benign hepatoma, even if it is of considerable dimensions excision should be carried out. In this connection the tumour may be found on laparotomy to be a primary carcinoma of the gall-bladder invading the liver; this, too, is sometimes remediable by resection.

Resection of a Tumour of the Liver.—In order to conserve blood, the hepatic artery and portal vein can be clamped lightly, and released every five minutes. Another very important fact to know is that branches of the hepatic artery and the portal vein ramify through the liver in the portal canals: each of these canals is surrounded by a prolongation of Glisson's capsule which, when encountered with a blunt instrument, gives a sense of resistance. Thus, if liver tissue is divided by employing a fine-pointed hæmostat using gentle strokes very slowly (Sir Heneage Ogilvie), it is possible to divide liver tissue, but spare the vessels, which can be clamped and ligated, or coagulated with diathermy, before division. The resection completed, the resulting raw surfaces of the cut liver are cared for in the same manner as described for rupture of the liver (see p. 396). A few cases have been reported where practically the whole of the right lobe of the liver has been excised with success. The left lobe of the liver can be resected after securing the left hepatic artery, the left branch of the portal vein and the left hepatic duct. Preceding resection of the liver, full antibiotic therapy must be given, owing to the danger of clostridial infection.

SECONDARY NEOPLASMS OF THE LIVER

Secondary Carcinoma.—As is well known, the liver is a favourite site for carcinomatous metastases. Characteristically, secondary growths in the liver, owing to degeneration of cells in the centre, are umbilicated. As a rule, carcinomatous deposits in the liver are multiple (fig. 571). Occasionally at laparotomy a secondary growth in the liver is found to be apparently solitary and accessible. In such circumstances, provided the primary growth can be or has been resected, excision of that part of the liver containing the neoplasm sometimes results in a long-lasting survival.

Secondary carcinoid (argentaffin) tumours (see p. 544 and Chapter XXVIII) are rare; where they occur they often grow to a large size.

FIG. 571.—Secondary carcinoma of the liver. (*University of Liverpool Pathological Museum.*)

Secondary melanoma occurs in the liver with unfailing regularity unless the primary growth, regional lymph nodes, and intervening lymphatic vessels can be excised satisfactorily. As many as fifteen years have elapsed between the removal of the primary growth and the appearance of the secondary (see also p. 133).

Secondary Sarcoma.—The liver is not an uncommon site for secondary deposits in cases of sarcoma, but the lungs show a higher incidence.

Francis Glisson, 1597–1677. Regius Professor of Physic, Cambridge.
Sir Heneage Ogilvie, Contemporary. Consulting Surgeon, Guy's Hospital, London.

CHAPTER XXII

THE GALL-BLADDER AND BILE DUCTS

HAMILTON BAILEY

Embryology.—During the fourth week of fœtal life the hepatic diverticulum arises from the ventral wall of the foregut near its opening into the yolk sac. As this diverticulum elongates into a stalk to form the choledochus, a lateral bud is given off, viz. ——————→ which is destined to become the gall-bladder and cystic duct. In very early fœtal life the gall-bladder and the bile ducts are hollow, but, owing to cellular hyperplasia, their lumina become filled, and for a short time these structures are solid cords. However, in a matter of a few weeks, as a result of the formation and coalescence of vacuoles within the ductal lumina, they become recanalised.

During the second month, the human gall-bladder is intrahepatic, but because of atrophy of the overlying liver, brought about by mechanical pressure from the distended gall-bladder, three-quarters of its circumference becomes extrahepatic.

Surgical Physiology.—The healthy gall-bladder has several functions:

1. **Concentration of bile** is the most important. The gall-bladder is capable of concentrating hepatic bile four to ten times; this it does through its blood-vessels and, to a lesser extent, its lymphatics. Water, sodium chloride, and sodium bicarbonate are absorbed rapidly by the mucous membrane of the gall-bladder, with the result that hepatic bile which entered the gall-bladder alkaline, is rendered slightly acid. Bile pigments and cholesterol are not absorbed, consequently their concentration is increased. Very little calcium and bile salts are absorbed, so these constituents are concentrated also. Cholecystography depends upon the concentration within the gall-bladder of a radio-opaque drug excreted by the liver.

2. **Reservoir for Bile.**—When the gall-bladder is full and there is no specific stimulus to evoke its partial emptying (the gall-bladder never empties completely) the sphincter of Oddi relaxes, and bile trickles into the duodenum. When fat-containing chyle reaches the duodenum the gall-bladder contracts, with the result that concentrated bile is ejected into the duodenum when it is most required for digestion of fat. This response is mediated by the hormone cholecystokinin which, after its release by the duodenal mucosa into the blood-stream, causes the musculature of the gall-bladder to contract. At the same time reciprocal relaxation of Oddi's sphincter occurs.

3. **Cholesterol excretion** by the gall-bladder has not been proved, but pathological evidence of the gall-bladder epithelium choked with crystalline cholesterol (see p. 437) is at least suggestive that normally the gall-bladder excretes cholesterol into the bile.

4. **Secretion of Mucin.**—About 20 ml. is secreted by the gall-bladder each twenty-four hours.

Surgical Anatomy:

The adult gall-bladder is pear-shaped, and 3 to 5 inches (7·5 to 12.5 cm.) long, with a capacity of about 1½ oz. (45 ml.), but capable of fifty-fold distension. For purposes of description, the gall-bladder is divided into a fundus (which can be palpated when the gall-bladder is distended), a body, and a narrow neck which terminates in the cystic duct. At the junction of the neck with the cystic duct, the neck forms a pouch, called Hartmann's pouch (fig. 572); this is a common site for a solitary stone to lodge. Hartmann's pouch, because it overlaps and partially hides the cystic duct, is the best guide to that structure. The muscle fibres in the walls of the gall-bladder are not arranged in layers, but in a criss-cross manner. They are particularly well developed

Ruggiero Oddi, 1845–1906. Surgeon and Anatomist, Rome.
Robert Hartmann, 1831–1893. Professor of Anatomy, Berlin.

in the neck. The mucous membrane is thin, because it contains no glands; instead there are indentations of the mucosa that sink into the muscle coat; these are the crypts of Luschka.

The cystic duct is usually 1 inch (2·5 cm.) in length, but because of its S-shaped course and the concertinaing effect produced by its contained valve, its length is reduced to 1·5 cm. That half of the cystic duct nearer the gall-bladder contains the spiral valve of Heister; the lower half is bereft of a valve. In the valve-containing portion of the duct the muscle fibres are predominantly in circular bundles, and internal projections of these muscle fibres account for the Heisterian valve, which makes the passage of calculi, as well as a probe, more difficult. When the region of the cystic duct has been dissected, it will be found that the cystic duct forms the base of Calot's triangle. The safest place to ligate the cystic artery is close to the gall-bladder wall at the apex of this triangle (fig. 573). The cystic lymph node, which frequently overlies this artery in this position, is a good guide to the cystic artery.

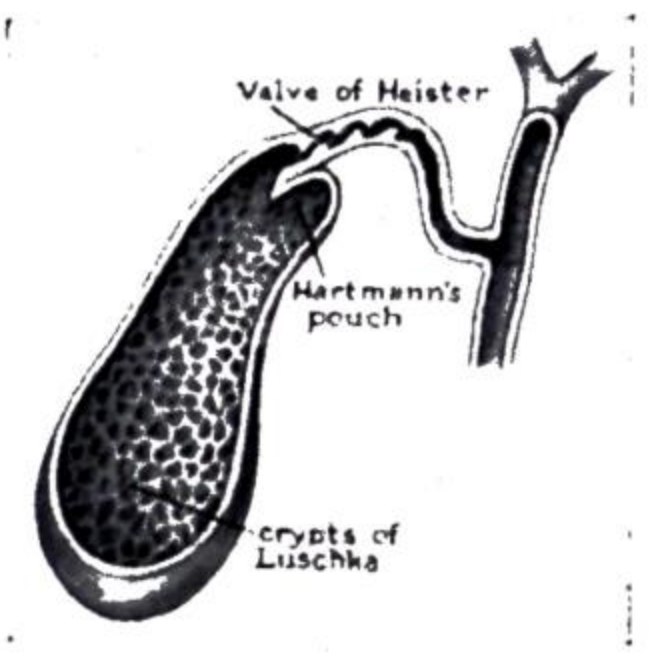

FIG. 572.—Sagittal section of the gall-bladder and the cystic duct.

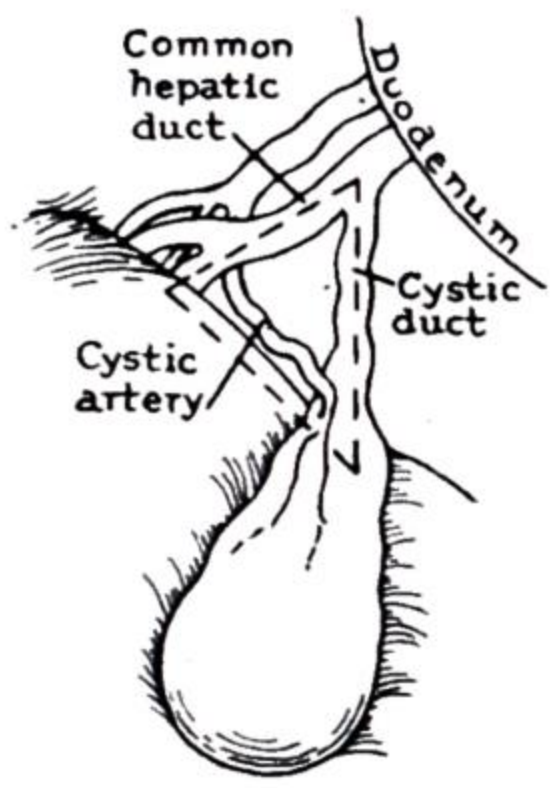

FIG. 573.—Calot's triangle. (*After J. A. Sterling.*)

The common hepatic duct is usually less than 1 inch (2·5 cm.) long, and is formed by the union of the right and left hepatic ducts: it is not exceptional for the right hepatic duct to be duplicated.

The common bile duct, which is 3 to 4 inches (7·5 to 10 cm.) long, is formed by the junction of the cystic and the common hepatic ducts. It is divided into four parts (fig. 574):

1. *The supraduodenal portion* is about 1 inch (2·5 cm.) long and runs in the free edge of the lesser omentum.

2. *The retroduodenal portion* can usually be exposed with ease, seeing that the first part of the duodenum is comparatively mobile.

3. *The infraduodenal portion* usually lies in a groove, but at times in a tunnel in the posterior surface of the pancreas. It is surrounded by arcades between the superior and inferior pancreatico-duodenal arteries, and the right edge of the inferior vena cava lies not far distant behind it.

4. *The intraduodenal portion* passes obliquely through the wall of the second part of the duodenum to open on the summit of the duodenal papilla, which is surrounded by the sphincter of Oddi. Near its opening it is often joined by the main duct of the pancreas (duct of Wirsung), but the presence of an ampulla of Vater[1] (an ampulla indicates a dilatation), contrary to anatomical teaching, is a rarity (J. A. Sterling). The arrangements of the exit are shown in fig. 575.

FIG. 574.—The four divisions of the common bile duct.

(*a*) 67 per cent. (*b*) 30 per cent. (*c*) 3 per cent.

FIG. 575.—(*a*) Common orifice occupying one-third to two-thirds of the length of the papilla. (*b*) Two orifices surrounded by the same sphincter. (*c*) An ampulla present.

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[1] So universally is the term 'ampulla of Vater' employed to indicate the region of the stoma of the common bile duct and the duct of Wirsung that it would be confusing to disturb this convenient designation.

Hubert Luschka, 1820–1875. Professor of Anatomy, Tübingen.
Lorenz Heister, 1683–1758. Professor of Anatomy and Botany, Helmstädt, Germany.
Jean Francois Calot, 1861–1944. Surgeon, Hôpital Rothschild, Berck-sur-Mer, France.
Johann Wirsung, Prosector at Padua, was murdered when entering his house at night in 1643.
Abraham Vater, 1684–1751. Professor of Anatomy and Botany, Wittenberg.
Julian Alexander Sterling, Contemporary. Surgeon, Albert Einstein Medical Centre, Philadelphia.

Aberrations in the Arterial Supply of the Gall-bladder.—The cystic artery is a branch of the right hepatic artery. Usually the cystic artery is given off *behind* the common hepatic duct, and then runs along the cystic duct to the gall-bladder (fig. 576a). Occasionally there is an accessory cystic artery that arises from the gastro-duodenal artery (fig. 576b). In some instances (25 out of 161 cases, E. R. Flint) the right hepatic artery

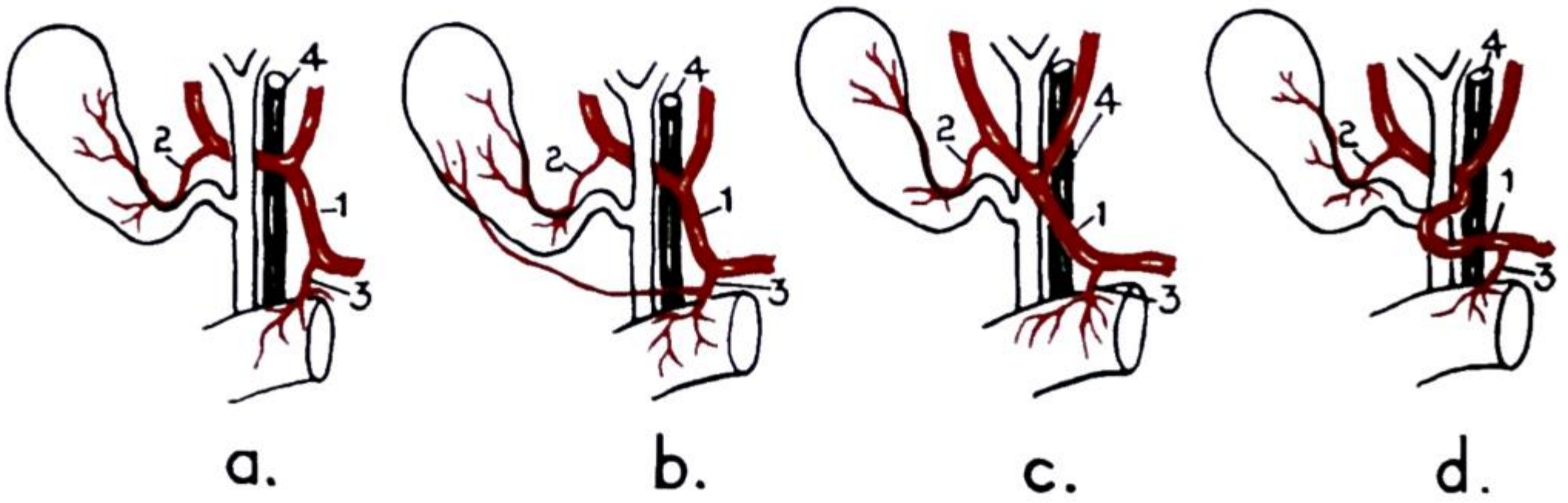


FIG. 576.—a, The usual arrangement of the arterial supply to the gall-bladder; b, c, and d, variants. 1. Hepatic artery. 2. Cystic artery. 3. Gastro-duodenal artery. 4. Portal vein.

and/or the cystic artery cross in front of the common bile duct and the cystic duct (fig. 576c), in which case the right hepatic artery is in an extremely vulnerable position. Should it be ligated, necrosis of much of the right lobe of the liver is liable to ensue. The right hepatic artery in its anterior position may compress the common bile duct. The most dangerous anomaly of all is when the main trunk of the hepatic artery takes a tortuous course in front of the origin of the cystic duct (fig. 576d). Ligation of the main trunk of the hepatic artery results in death in more than 50 per cent. of cases.

ABNORMALITIES OF THE GALL-BLADDER AND THE BILE DUCTS

Anomalies of the biliary tract are found in 10 per cent. of necropsies.

Absence of the Gall-bladder.—The gall-bladder is normally absent in twenty-six species of animals, including the elephant, horse, goat, and rat. Occasionally the gall-bladder is absent in man. Stones have been found in the common bile duct in nearly 50 per cent. of cases of agenesis of the gall-bladder.

The Phrygian cap[1] (fig. 577(*a*)) is the commonest abnormality of the gall-bladder. It is present in 2 to 6 per cent. of cholecystographies, and unless the abnormality is known, it will be mistaken for a pathological deformity of the organ. The probable cause is failure of the caudal portion of the embryological diverticulum to become tubular. Lithiasis develops in a higher percentage of gall-bladders thus complicated than of normal gall-bladders.

Floating Gall-bladder.—Normally one-third of the circumference of the gall-bladder lies in a shallow bed on the under-surface of the liver. Occasionally the gall-bladder has a mesentery, which simplifies cholecystectomy but renders the organ liable to undergo torsion (see p. 435).

Double Gall-bladder.—On rare occasions, due to bifurcation of the cystic bud, the gall-bladder is double. One of the twins may be intrahepatic (fig. 577(*b*)). Sometimes a pre-operative diagnosis can be made by cholecystography.

Absence of the Cystic Duct.—The gall-bladder opens directly into the common bile duct (fig. 577(*c*)). Injury of the common duct is particularly liable to occur when

[1] The Phrygian cap refers to hats worn by peoples of Phrygia, an ancient country of Asia. The cap was rather like a liberté cap.

Ethelbert Rest Flint, 1880–1956. Surgeon, General Infirmary, Leeds.

cholecystectomy is performed in a patient with this abnormality. The only way of avoiding the accident is to divide the neck of the gall-bladder and close the distal end accurately with sutures; a ligature around the termination of the gall-bladder should be avoided.

Abnormally Long Cystic Duct.—Sometimes the cystic duct is of considerable length, and opens into the common duct near the pancreas (fig. 577(*d*)). A particular

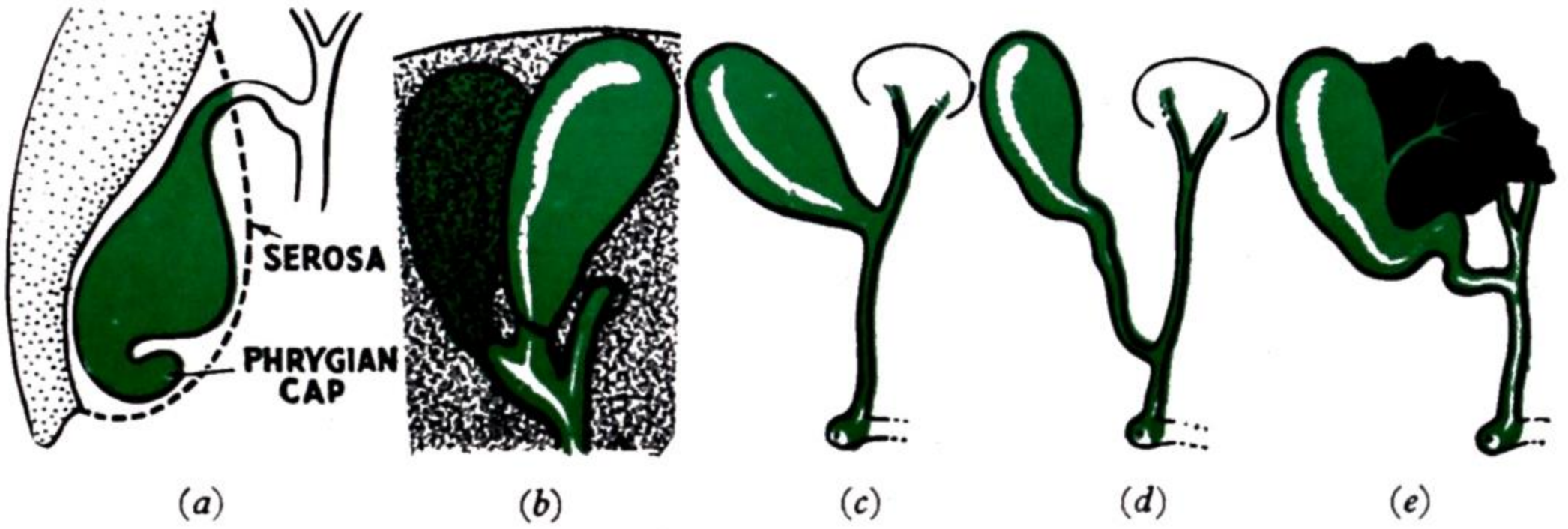


FIG. 577.—Some anatomical anomalies of the gall-bladder and bile passages.

misfortune of this type of abnormality is that a portion of the cystic duct is left behind during cholecystectomy, and as a consequence symptoms return, or are unrelieved.

An accessory cholecystohepatic duct may open into the gall-bladder (fig. 577(*e*)) and cause leakage after cholecystectomy. If drainage has been provided this leakage, although inconvenient, eventually ceases. Not so in the less common, but more dangerous, anomaly where the right hepatic duct terminates in the gall-bladder. In such cases cholecystostomy or partial cholecystectomy should be performed, so as to preserve the anomalous duct and maintain a free exit for its contents along the anatomical channels.

CHOLECYSTOGRAPHY AND CHOLANGIOGRAPHY

Radiography, after the administration of an iodine-containing opaque medium that is secreted in the bile, is necessary to establish the diagnosis of chronic cholecystitis, non-opaque cholelithiasis, and other abnormalities and diseases of the gall-bladder and bile ducts. Three methods of undertaking this examination are available—oral, intravenous, and operative. In the last, the medium is injected directly into a bile duct, and is used to demonstrate pathology or normality of the bile passages (and possibly pancreatic duct).

Oral or intravenous cholecystography should not be attempted in the presence of jaundice, because of the danger of increasing liver damage by the dye; even if this were not so, serum bilirubin levels of above 3 mg. per 100 ml. are incompatible with satisfactory visualisation. Consequently the method is of little value until the jaundice has cleared.

Oral cholecystography is the method in general use for endeavouring to visualise the gall-bladder, and it remains the most satisfactory method in so far as the gall-bladder is concerned.

Telepaque (Bayers Products Ltd.) is a contrast medium containing approximately 66 per cent. of iodine. If cholecystography is to be undertaken at 9 a.m., which is desirable, the last meal should be partaken between 9 and 10 p.m. the previous evening, and must be *fat free*. After this meal six telepaque tablets are swallowed

whole with water. No food of any kind is allowed until the radiological examination has been made, but the patient is permitted to drink water. A saline enema can be given with advantage before the cholecystography, to evacuate gas from the colon. After two or three films have been exposed, the patient is given a meal of eggs and bacon, bread and butter, and some cream in tea or coffee. One hour later another film is taken, in order to ascertain if the gall-bladder contracts after the fatty meal. The oral administration of sodium sulphate during cholecystography improves the visualisation of calculi in a poorly-functioning gall-bladder. When the gall-bladder is visualised only faintly, if a teaspoonful of sodium sulphate in 50 ml. of iced water is administered orally, improvement in delineation often follows. Telepaque gives excellent shadows of a functioning gall-bladder (figs. 578, 579, 580) and in some cases

FIG. 578.—A normal cholecystograph.

FIG. 579.—Same after a fatty meal.

FIG. 580.—Non-opaque stones in the gall-bladder rendered visible by the medium during cholecystography.

the bile ducts are visualised with the standard dose. If the dose is increased to ten or even twelve tablets, the bile ducts are more likely to be demonstrated. Telepaque is contraindicated in all states in which renal function is impaired. It is valueless in disorders of the gastro-intestinal tract, e.g. vomiting or diarrhœa, that interfere with absorption of the contrast medium.

A reliable sign of obstructive cholecystitis is failure of the gall-bladder to cast a shadow in any of the serial radiographs. It is, of course, essential to be certain that the patient has ingested the dye. Distortion of the gall-bladder or the presence of gall-stones otherwise radio-translucent (fig. 580) are frequently demonstrated by cholecystography. *A negative cholecystograph does not exclude gall-stones*, for small calculi may not cast a shadow, nor cause a filling defect in the dye-filled gall-bladder.

Failure of the gall-bladder to contract after a fatty meal suggests chronic cholecystitis, as the muscular wall has become fibrotic.

Intravenous Cholangiography (biligraphin[1]**).**—Whereas preparations used for oral cholecystography contain two atoms of iodine in the molecule, those employed for intravenous cholangiography contain six. When injected intravenously, biligraphin appears in the bile in a few minutes, achieving a concentration 30 to 100 times that of the blood, thus permitting radiological visualisation of the bile passages: here lies its peculiar and inestimable value (fig. 581). For the gall-bladder, it is inferior to oral cholecystography; nevertheless, it is indicated for that purpose when the patient is liable to vomit, or when absorption from the alimentary tract is impaired by diarrhœa. Another clear advantage of intravenous cholecystography is when early confirmation or exclusion of acute gall-bladder disease is required

[1] Cholografin (U.S.A.).

urgently for purposes of differential diagnosis. On the other hand, although no deaths have been reported after its use, it has given rise to severe reactions, the leading symptoms of which are: feeble or imperceptible pulse, stertorous respirations, cyanosis, and often incontinence of urine. The treatment is to administer nikethamide (coramine), anatazoline (antistin), and

FIG. 581.—Stones in the common bile duct visualised after intravenous cholangiography. Note the opaque medium in the duodenum, revealing that bile is entering the duodenum freely. (*Dr. M. Israelski, Leamington Spa.*)

oxygen. A supplementary small ampoule is supplied with each package by the manufacturers. The contents of this is injected into a vein the day before (or, in urgent cases, an hour before) the examination, in order to ascertain if any reaction is likely to occur. It is most desirable to give the patient an enema one or two hours before the examination.

Technique of Intravenous Cholangiography.—Biligraphin (Schering) is supplied in ampoules of 40 ml., but with a view to avoiding reactions, it is recommended that only 20 ml. (which is usually adequate) be used. The medium is given intravenously very slowly, ten minutes being taken over the injection. This contrast medium produces adequate radiological shadows of the biliary passages, independent of the state of the gall-bladder. Thus, in a cholecystectomised patient the biliary tree is visualised ten to forty minutes after the injection. When the initial radiographs do not give satisfactory contrast shadows the latter ones can be intensified by giving an injection of morphine, which causes spasm of the sphincter of Oddi. Later in the chapter several references are made to intravenous cholangiography.

Cholangiography on the Operating Table.—A cassette tunnel is placed beneath the patient (see fig. 565, p. 421) at a level that will include the entire biliary tract. Rotation of the liver is obtained by inserting a hand between the liver and the diaphragm, aided by suitable forceps applied to the gall-bladder, thus exposing the cystic and common bile ducts. The cystic duct is nicked, and a ureteric catheter size 4, 5, or 6 F (whichever fits the most snugly) is introduced through this tiny incision into the common bile duct. A syringe containing 20 ml. of diodone and a hollow needle, that fits the ureteric catheter accurately, are used. One to 2 ml. of diodone is injected down the catheter to ascertain that there is no leakage. All instruments having been removed from the field of operation, 10 ml. of diodone is injected rapidly into the duct (fig. 582). Some surgeons prefer other media, such as neo-hydriol, in which case the aqueous solution should be used, as the viscous preparation will not flow freely along the ducts. At the conclusion of the injection a film is exposed. While the film is being developed appendicectomy is performed, which should not take more than five minutes.

FIG. 582.—Operative cholangiography. Method of introducing the medium.

By radiographs it has been shown that in about 20 per cent. of cases the medium flows along the duct of Wirsung. When the common bile duct and the duct of Wir-

sung have separate orifices, after leaving the common bile duct the medium is observed 'puddling' in the duodenum, and from thence some of it may be demonstrated flowing into the duct of Wirsung. Reflux of bile into the duct of Wirsung must therefore be looked upon as a common physiological process.

A normal cholangiograph (fig. 583) is sufficient evidence that exploration of the common bile duct is unnecessary. When the medium fails to pass into the duodenum, and especially if the ducts concerned are dilated (figs. 584, 585), choledochostomy and exploration of the interior of the common bile duct is the first consideration.

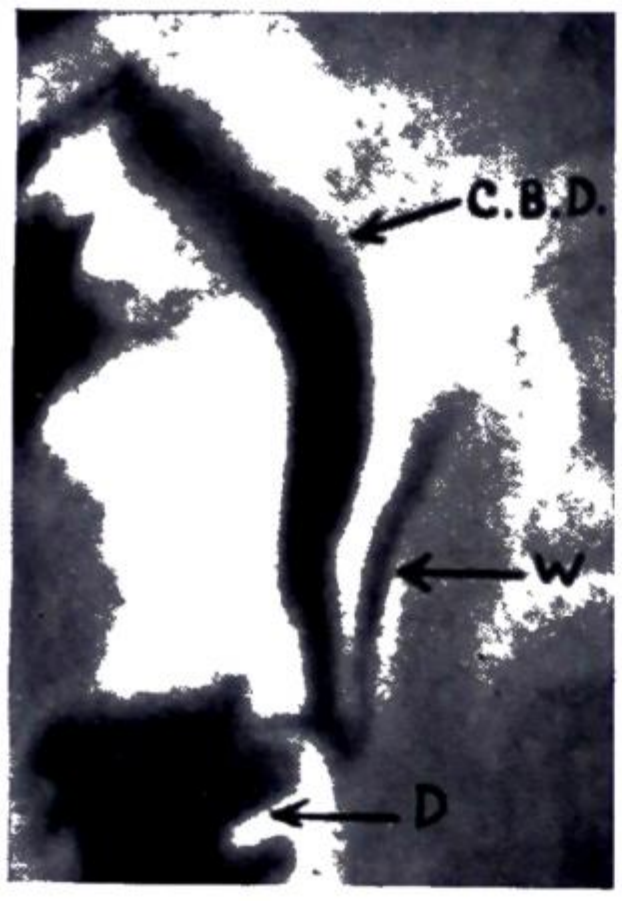


FIG. 583

FIG. 584

FIG. 585

Typical operative cholangiographs. From left to right : FIG. 583.—Normal cholangiograph. FIG. 584.—Spasm of the sphincter of Oddi. Dilatation of the common bile duct. No medium enters the duodenum. FIG. 585.—Showing calculus at the lower end of the common bile duct (inset, the calculus).

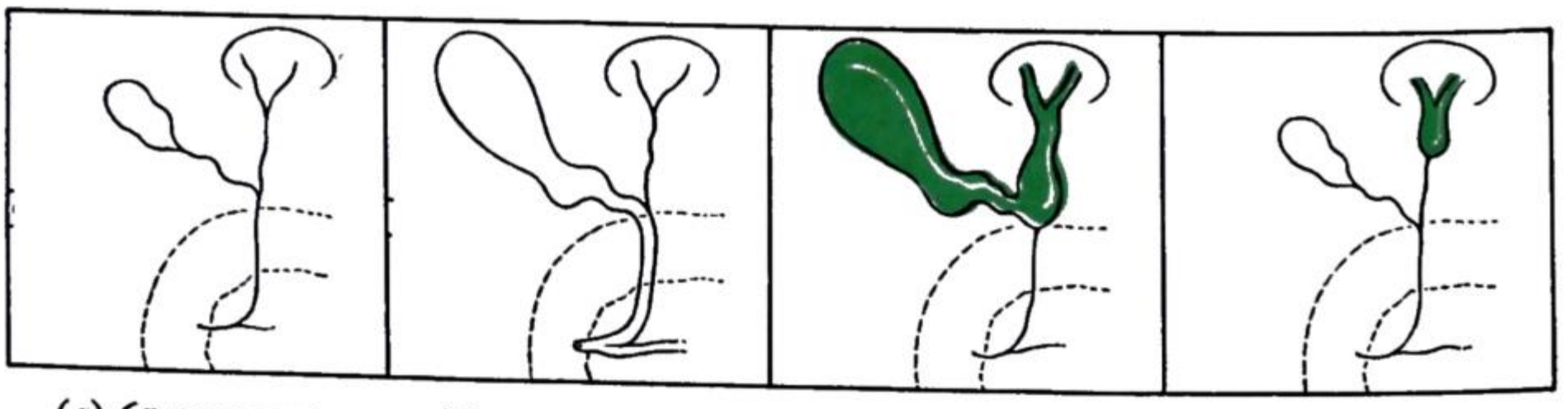
CBD = common bile duct; W = duct of Wirsung; D = medium in the duodenum.

After choledocholithotomy more medium can be injected into the common bile-duct via a T-tube placed in the duct. By this means cholangiography will help to verify that no stone has been overlooked. Misleading artefacts or indifferent clarity of the pictures are due to (*a*) interposition of opaque objects, (*b*) extravasation of opaque material, and (*c*) the presence of air within the ducts, all of which can be obviated by correct technique.

CONGENITAL ATRESIA OF THE BILE DUCTS

Ætiology.—Much the most satisfactory explanation is that recanalisation fails to occur in fœtal life. (See Embryology, p. 426.)

Pathology.—One of the anomalies shown in fig. 586 is present. As a rule the collapsed bile ducts have no lumen; occasionally, however, there is a tiny lumen



(*a*) 61 per cent. (*b*) 14 per cent. (*c*) 4 per cent. (*d*) 8 per cent.

FIG. 586.—Symposium of the more usual types of congenital obstruction to the bile ducts.

blocked with epithelial débris, and perhaps biliary sand. At necropsy the liver, which is greatly enlarged and stained dark green, exhibits varying degrees of biliary cirrhosis proportional to the time the patient has survived. It is a surprising fact that a few

infants possessing extrahepatic ducts with no lumina whatever have lived to be eight to ten months old.

Clinical Features.—Sometimes a slight icteric tinge is present at birth, more usually jaundice appears within two or three days thereof; occasionally it is delayed for up to several weeks. Unless the atresia can be remedied the jaundice becomes deeper and deeper; the urine is bile-laden and dark brown, and even tears and the saliva are yellow. From birth the stools are white or clay-coloured; after two weeks they may become faintly yellow. The clinician must be aware of the fact that in profound jaundice a small amount of bile pigments is excreted by the intestines. Gradually the liver becomes larger and larger, and on palpation feels unduly hard. Nutrition is fairly well maintained, especially if the baby is given feeds containing but little fat. Unrelieved, usually death results in three to six months.

Differential Diagnosis.--The condition has been confused with (*a*) erythroblastosis fœtalis, which can be eliminated by an examination of the blood; (*b*) congenital spherocytosis, which can also be eliminated by appropriate tests (see p. 379); (*c*) jaundice of hæmolytic sepsis; (*d*) congenital syphilis; (*e*) homologous serum hepatitis (serum jaundice), which can occur in patients who have had blood-transfusion or plasma infusion, and (*f*) virus hepatitis. It should be noted that in none of the foregoing conditions does the infant evacuate colourless stools. The serum bilirubin is raised from 3 to 22 mg. per cent. and the serum alkaline phosphatase is usually moderately elevated. If duodenal aspiration can be performed and the presence of the tip of the tube in the duodenum can be confirmed radiologically, the absence of bile in the aspirate is an accurate aid to diagnosis. The correct diagnosis is arrived at by process of elimination, and every effort should be made to achieve this end during the first three weeks of life.

Treatment.—(*a*) *Medical* is occasionally successful when the ducts are blocked by epithelial débris.[1] Cholagogues are given; orally 250 mg. of desiccated bile salts with each feed, and intravenously 5 ml. of 20 per cent. dehydrocholic acid three times a week.

(*b*) *Operative*.—Pre-operative measures include the correction of dehydration, if present, and one or two small blood-transfusions. Vitamin K_1 is always necessary to combat prothrombin lack in patients with obstructive jaundice. Terramycin should be administered before and after operation. At laparotomy one of the conditions shown in fig. 586 will be displayed.

Operation.—The anæsthetic of choice is open ether. As in all cases of obstructive jaundice, the use of muscle relaxants must be forbidden. A midline incision affords adequate exposure, but the enlarged liver makes displaying the minute ducts difficult. Operative cholangiography can be employed to ascertain the site of the stricture when this is not apparent. In (*a*) and (*b*), even when the ducts appear to be fibrous cords, it is always well worth while ascertaining whether a tiny lumen is present in the main duct. After introducing a hollow needle into the gall-bladder, or a duct, syringing sometimes has proved successful in freeing obstructed ducts of inspissated material. With this exception, the operative measure should be as follows:

Gall-bladder present. Cystic duct communicated with main ducts. Atresia lower end of common bile duct.	Cholecystojejunostomy + jejunojejunostomy (see fig. 636, p. 478).
Gall-bladder functionless or absent. Atresia lower end of common bile duct.	Choledochoduodenostomy (see fig. 608, p. 452).
Common hepatic duct alone present, and contains bile.	Hepaticodochojejunostomy + jejunojejunostomy (see p. 454).
Extrahepatic ducts impervious.	Longmire's operation (see p. 454).

In suitable cases the first of the procedures listed would appear to be ideal. It is certainly the most facile of execution. Unfortunately late stenosis of the cystic duct

[1] Probably cases belonging to this category are examples of mucoviscidosis (see p. 461).

often mars the final outcome. It should therefore be performed only when the cystic duct is greatly distended.

Congenital choledochus cyst (*syn.* congenital idiopathic dilatation of the common bile duct) is a rare condition affecting females four times as commonly as males. Contrary to congenital atresia of the bile ducts, the symptoms and signs seldom become manifest before the age of six months. Indeed, in only 50 per cent. of cases do symptoms appear before the age of twenty years. The main pathological feature is striking enlargement of the whole or part of the common bile duct. Contrary to expectation, only occasionally does the distension involve the cystic and hepatic ducts, and the gall-bladder is very seldom distended. Sometimes the choledochus cyst contains as much as 1 to 2 litres. The cyst wall shows inflammatory changes with an absence of its lining epithelium. The ætiology is unknown: the part of the common bile duct below the cyst is either perfectly normal or reduced in diameter, but never is there complete obstruction to the flow of bile.

Clinical Features.—There are attacks of jaundice of the obstructive type. Between the attacks the patient's colour is normal and the general health good. Usually the attacks are accompanied by upper abdominal pain and pyrexia due to infection. In 90 per cent. of cases a swelling is detected in the upper abdomen. Untreated, the condition ultimately proves fatal, due to ascending cholangitis, biliary cirrhosis, or diffuse peritonitis following rupture of the cyst.

Treatment is choledochoduodenostomy, preferably in one stage. The results of the operation, if performed before irremediable complications have set in, are excellent. The treatment of peritonitis due to rupture of a choledochus cyst is drainage of the cyst and the peritoneal cavity. When performed early, operation for ruptured choledochus cyst sometimes saves the patient's life.

TRAUMATIC RUPTURE

Rupture of the gall-bladder or the bile passages is a rare abdominal injury, usually the result of a run-over accident. In many instances the signs are identical with those of rupture of the small intestine. Within a few hours of the accident jaundice appears in 65 per cent. of cases. The advent of unmistakable jaundice should arouse suspicion of an injury to the extrahepatic biliary tract (Sir Gordon Gordon-Taylor). When the abdomen is opened bile is found within the peritoneal cavity.

Rupture of the Gall-bladder.—Usually the tear is small and the hole, if near the fundus, can be used for cholecystostomy: elsewhere the tear can be sewn up and deliberate cholecystostomy performed. When the tear is extensive, cholecystectomy is indicated.

Partial rupture of the common hepatic duct or the common bile duct is treated by the insertion of a T-tube, which should be left in place for at least fourteen days.

Complete rupture of the common hepatic duct, treated by suture, always results in an intractible stricture. If a polythene tube is inserted into the proximal end of the duct, the duct can be implanted into a loop of jejunum, the limbs of which are united by lateral anastomosis.

Complete rupture of the common bile duct can be treated by partial suture around a T-tube or by ligation of both ends, and cholecystjejunostomy.

The best methods of treating these injuries are represented in fig. 587.

FIG. 587.—A symposium of methods of treating early complete tears of the biliary tract.

Penetrating injury of the gall-bladder occurs in about 1 per cent. of abdominal wounds. When the injury is extensive, cholecystectomy is indicated. More often

Sir Gordon Gordon-Taylor, Contemporary. Consulting Surgeon, The Middlesex Hospital, London.

the wound in the gall-bladder is such that it is possible to suture the defect and leave a drain in the peritoneal cavity. During the Korean war this was found to be highly satisfactory, and the conservative operation carried a low mortality.

Accidental injury of a major bile duct during cholecystectomy is by no means a rarity. While it can occur during the course of any cholecystectomy, it does so particularly in four sets of circumstances:

1. During emergency cholecystectomy, when the ducts are œdematous and difficult to define.

2. During the control of profuse bleeding from the cystic or right hepatic artery by the blind application of hæmostats.

3. In cases of extreme shortness of the cystic duct or its absence; severance of an anomalous right hepatic artery opening into the gall-bladder is also a cause.

4. Traction on the cystic duct (fig. 588).

If the accident is recognised at the time of the operation, the duct should be repaired according to the principles set out above. More often the damage goes unrecognised until early in the post-operative period, when the patient becomes jaundiced. This is followed in a few days by a profuse discharge of bile through the wound, and the development of a biliary fistula (see p. 454). Sometimes the damage is such that the common bile duct, though narrowed, can function, but after a varying interval—possibly years—symptoms of stricture of the duct (see p. 453) become manifest.

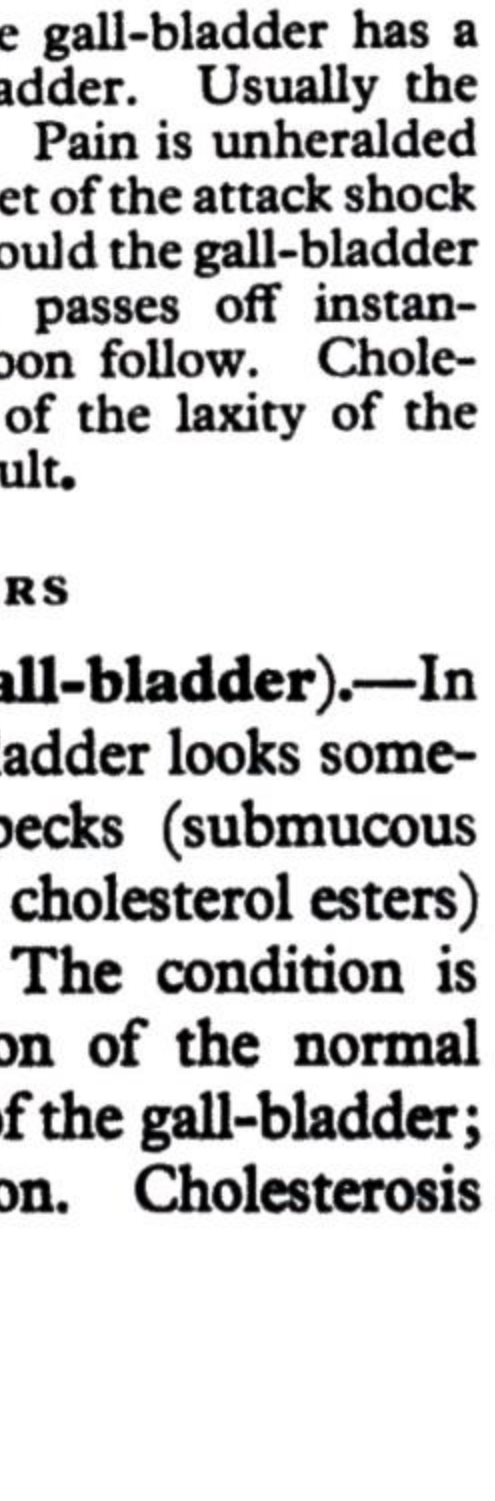


FIG. 588.—One method by which the common bile duct may be severed in cholecystectomy. The accident is more common when the gall-bladder is removed from its fundus towards the cystic duct.

TORSION OF THE GALL-BLADDER

Torsion occurs occasionally in those rare instances where the gall-bladder has a comparatively long mesentery—the so-called 'floating' gall-bladder. Usually the patient is a female over sixty years of age. Pain is unheralded and agonising, and immediately after the onset of the attack shock is pronounced and vomiting is frequent. Should the gall-bladder become gangrenous or rupture, the pain passes off instantaneously, but signs of diffuse peritonitis soon follow. Cholecystectomy is the treatment, and, because of the laxity of the parts concerned, it is unlikely to prove difficult.

FIG. 589.—Cholesterosis of the gall-bladder.

METABOLIC DISORDERS

Cholesterosis (*syn.* **Strawberry Gall-bladder**).—In the fresh state the interior of the gall-bladder looks something like a strawberry; the yellow specks (submucous aggregations of cholesterol crystals and cholesterol esters) correspond to the seeds (fig. 589). The condition is believed to be due to an exaggeration of the normal excretion of cholesterol by the mucosa of the gall-bladder; infection plays no part in its production. Cholesterosis

per se is symptomless, and cannot be diagnosed by cholecystography. The symptoms, if any, are generally due to associated gall-stones, cholecystitis, or spasm of the sphincter of Oddi. In a high percentage of cases cholesterosis is accompanied by a cholesterol calculus, and it is logical to suppose that the cholesterosis is the precursor of the calculus.

Cholecystectomy for cholesterosis *per se* is liable to prove disappointing, for so often the symptoms are due to another lesion.

Polyposis of the Gall-bladder (fig. 590).—Cholecystography shows negative shadows in a functioning gall-bladder. The shadows, which are adjacent to the wall of the gall-bladder, remain constant in position and in relation to one another in all films of the series. Radiologically the condition is indistinguishable from the less common condition of papillomata of the gall-bladder (see p. 456). Histologically, polyposis is similar to the cholesterol-laden projections of the strawberry gall-bladder, but the lesions are much less numerous and are relatively gigantic. The treatment is cholecystectomy.

FIG. 590.—Polyposis of the gall-bladder. (*Mr. D. J. Oakland, Birmingham.*) (*British Journal of Surgery.*)

CHOLELITHIASIS

Gall-stones vary in composition. Sometimes they consist entirely of cholesterol, or of calcium bilirubinate. More often they are made up of alternating layers of cholesterol and calcium bilirubinate, or cholesterol and calcium carbonate and phosphate. Gall-stones are conveniently classified into metabolic stones, inflammatory stones, and ductal stones.

1. **Metabolic Stones:**

(*a*) **Cholesterol stone** is solitary, and as a consequence is oval or rounded in shape. It becomes a relatively large stone (fig. 591), often measuring half an inch (1·25 cm.) in diameter, but larger specimens are not uncommon. In its absolutely pure state, it is very pale yellow in colour and, like tallow, it is feebly translucent. More often bile pigments are deposited upon it, and its smooth surface becomes roughened thereby. On section its crystalline interior will be found to radiate

FIG. 591.—Solitary gall-stone from a gall-bladder (actual size). It is unfaceted.

Cultures of the interior of these stones are sterile. Cholesterol stone, which is not uncommon, tends to occupy Hartmann's pouch. The normal ratio of bile salts to cholesterol in the gall-bladder is 25:1. The critical level for the precipitation of cholesterol is 13:1.

(*b*) **Calcium bilirubinate stones** are comparatively uncommon in the gall-bladder, but are rather frequent in the common bile duct. Consisting of bilirubin and calcium bilirubinate, they are small, multiple, and friable, and so dark green in colour that they look almost black. Pure bilirubin stones are soft, putty-like masses. On section all pigment stones are amorphous.

On p. 379 attention has been directed to the frequency with which these stones are associated with hereditary spherocytosis; indeed, their presence in the gall-bladder may be the only evidence of mild forms of that disorder, in which case the patient should always be investigated from this standpoint.

2. **Inflammatory Stones :**

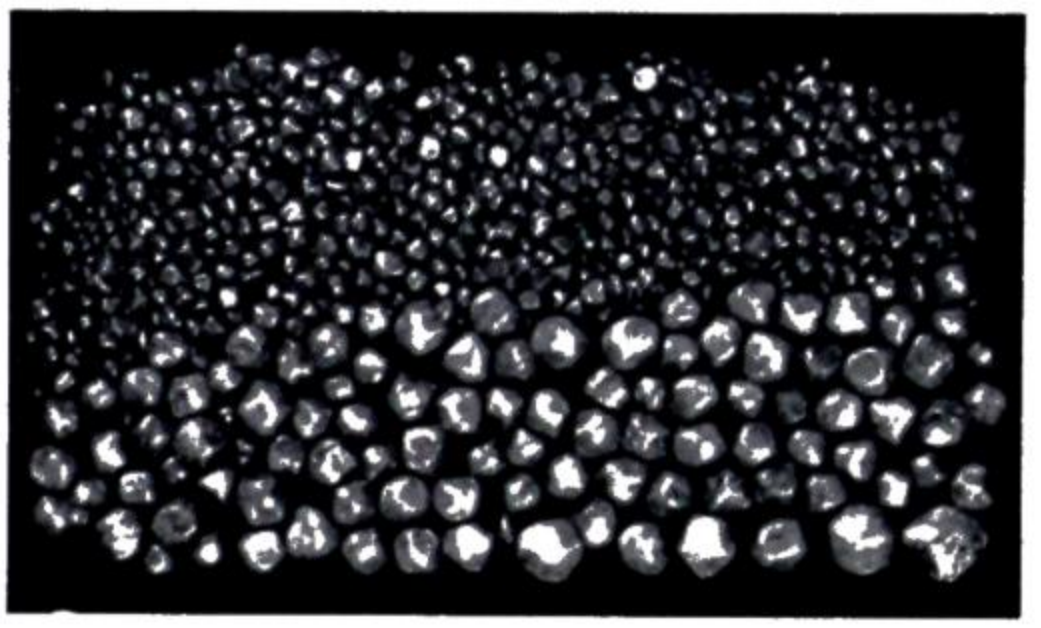


FIG. 592.—Stones from the gall-bladder showing two generations of calculi (half scale).

(*a*) **'Mixed' stones** constitute the majority (80 per cent.) of gall-stones found at operation. Always multiple, by mutual pressure or by friction one against the other, they become faceted. Dozens or hundreds of such stones are often present, frequently the gall-bladder being packed to capacity. Sometimes the stones, all of the same shape, can be segregated into two or more sets of different sizes (fig. 592), which suggests that one generation of calculi succeeds another after recurring attacks of cholecystitis. Inflammatory stones are frequently referred to as 'mixed' stones because on section each is found to be laminated  The central nucleus consists of epithelial débris and perhaps bacteria, or, in other instances, of cholesterol and organic matter, which suggests that cholesterol polypi have been broken off and have acted as nidi. Whatever the composition of the central core, alternating layers of cholesterol and calcium carbonate and/or calcium bilirubinate are deposited upon it. Although there is much truth in Moynihan's aphorism that " A gall-stone is a tombstone to the memory of the germ which lies within it," it is probable that cholecystitis which initiates calculus formation is sometimes chemical, and due to regurgitation of an excessive amount of pancreatic juice into the gall-bladder. Analyses of bile from gall-bladders aspirated at operation prior to cholecystectomy frequently reveal the presence of one or more pancreatic ferments.

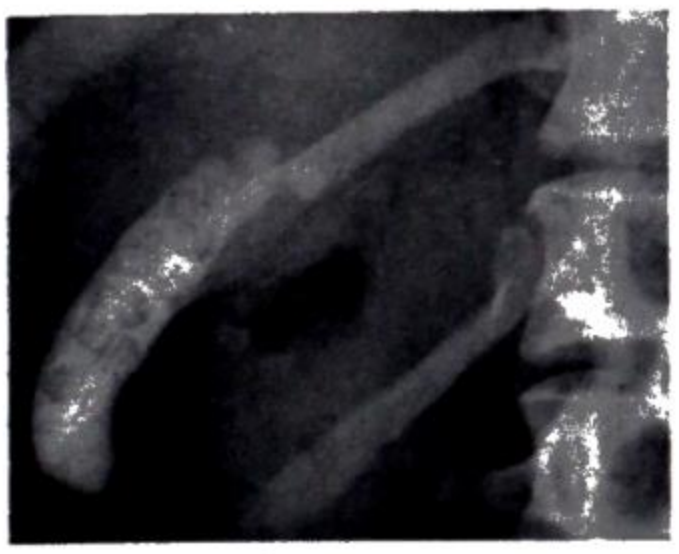


FIG. 593.—Plain radiograph showing a gall-bladder filled with a thick emulsion of calcium salts. Gall-stones are present also.

(*b*) **Calcium carbonate in the gall-bladder** is revealed in a plain radiograph (fig. 593) more clearly than if a normal gall-bladder had been visualised by cholecystography. In this condition, the cause and mechanism of which is unknown, the gall-bladder becomes filled with a mixture of calcium carbonate and phosphate of the consistency of toothpaste. Usually this condition is found in conjunction with chronic infected cholecystitis and cystic duct obstruction, and there is copious growth of organisms from the emulsion.

3. **Ductal stones** have either migrated from the gall-bladder or developed in the bile ducts, the ratio of frequency of each variety is about equal.

(*a*) **Primary ductal stones** have been called by Aschoff 'static' stones, because they develop only when there is stasis in the duct. Primary ductal

Lord Moynihan, 1865–1936. Professor of Surgery, University of Leeds.
Karl Albert Ludwig Aschoff, 1866–1942. Professor of Pathological Anatomy, Freiburg.

FIG. 594.—Biliary sand (calcium bilirubinate) removed from the common duct by choledochostomy.

stones are composed of calcium bilirubinate, and often take the form of, or are present in conjunction with, biliary sand (fig. 594) or mud. Found most often in the common bile duct but also in the common hepatic duct, this form of calculus is associated with a high incidence of bacterial infection.

(*b*) **A secondary ductal stone** is one that has migrated from the gall-bladder, and in two-thirds of such cases the stone that has migrated is solitary. Should its sojourn in the common bile duct be more than transitory, it receives an additional coating of calcium bilirubinate, becomes larger and, even if previously faceted, ovoid in shape. Because the presence of a stone impedes the flow of the bile, the duct above the site of obstruction dilates, and in the dilated portion other stones may form or, what is more usual, biliary mud or gravel is precipitated.

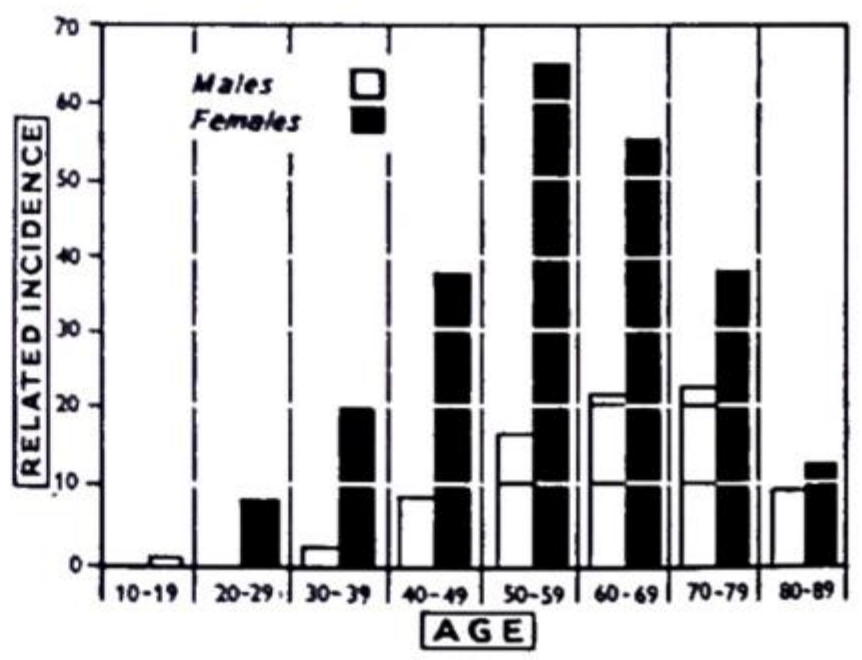


FIG. 595.—Incidence of gall-stones by age and sex *at the time of seeking relief*. (*After G. Horn.*)

Clinical Features.—A fat, fertile, flatulent female of fifty is the classical sufferer from gall-stones (fig. 595). Useful as is this clinical memorandum, it should be tempered with the knowledge that cholelithiasis occurs in both sexes, quite often at a much earlier age—even in childhood—and is not uncommon in the evening of life.

1. **Silent Gall-stones.**—It is possible for a calculus or calculi to be present in the gall-bladder and give rise to no symptoms during a long lifetime. At least 8 per cent. of necropsy subjects of over fifty years of age have gall-stones in the gall-bladder that have not contributed to the cause of death.

2. **Dyspepsia.**—Apart from gall-stone colic, stones in the gall-bladder often give rise to reflex dyspepsia, which is discussed under the heading of chronic cholecystitis (see p. 444). This gall-bladder dyspepsia can continue for a long time—perhaps for years—until one day a comparatively small stone enters the cystic duct, or a larger one occludes the neck of the gall-bladder, in which case there results:

3. **Gall-stone Colic.**—Suddenly the patient experiences excruciating pain in the epigastrium and right hypochondrium. The pain shoots to the back or between the shoulder-blades. In severe cases it ‘ doubles her up,’ and she rolls in agony on the floor. The attack, which lasts for upwards of two hours and is usually accompanied by vomiting and retching, often passes off as suddenly as it came. In most cases heat somewhat relieves the pain, and in old-standing cases a brown pigmentation of the skin over the right hypochondrium bespeaks frequent hot applications.

Physical Signs.—Deep tenderness in the right hypochondrium can often be elicited during the stage of the inaugural symptoms.

It is impossible to examine the abdomen satisfactorily during an attack of colic. Soon after the attack, usually there is rigidity and tenderness in the right hypochondrium; exceptionally a tender, enlarged gall-bladder can be palpated.

Murphy's Sign.—If continuous gentle pressure is exerted over the right hypochondrium (fig. 596) while the patient takes a deep breath, there is a 'catch in the breath' just before the zenith of the inspiration.

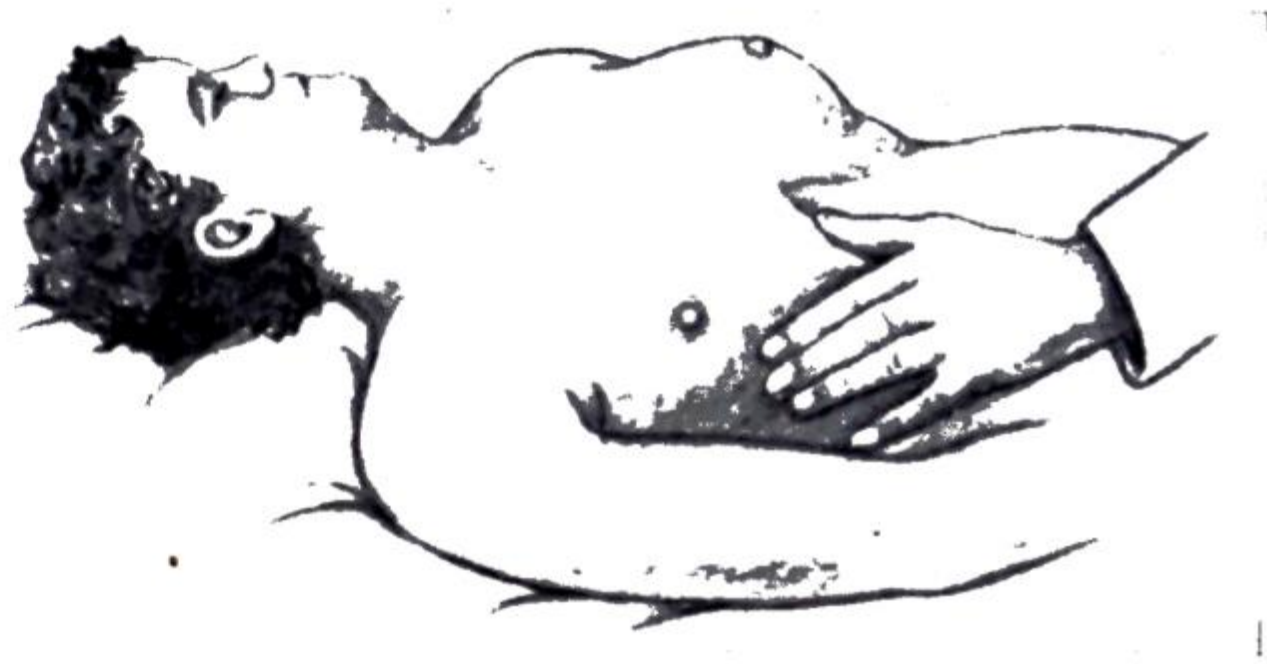


FIG. 596.—Murphy's sign (Moynihan's method).

Jaundice follows an attack of gall-stone colic in 18 per cent. of cases. In 58 per cent. of these the jaundice is due to a stone obstructing the common bile duct; in the remainder it is caused by associated hepatitis or cholangitis.

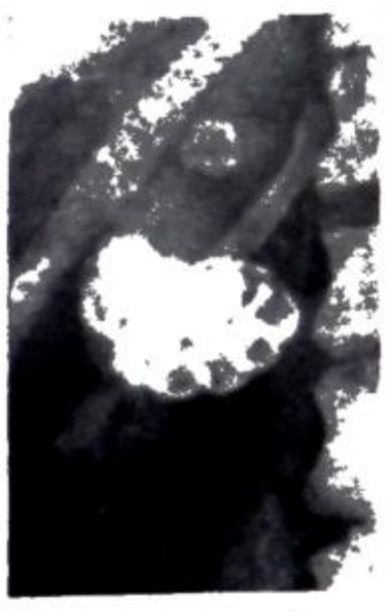


FIG. 597.—Plain radiograph showing stones in the gall-bladder and one in the cystic duct.

Radiography.—It should be noted especially that because the function of the liver is depressed, cholecystography should not be undertaken for at least three or four days after an attack of gall-stone colic, and only then if the patient has been free from jaundice for a similar length of time. On the other hand, there is no objection to having a plain radiograph taken of the upper abdomen. In 25 per cent. of cases gall-stones in the gall-bladder are sufficiently opaque to cast an X-ray shadow (fig. 597).

Treatment:

(*a*) **Of Gall-stone Colic.**—On p. 442 it is explained why morphine should never be given in these cases. The most effective analgesic is pethidine intravenously, which can be repeated in an hour, if necessary, but the second and subsequent doses should be given intramuscularly.

There is no question that small calculi can be discharged from the gall-bladder and passed *per via naturalis* through the relaxed sphincter of Oddi. Instances have occurred in which at laparotomy the gall-bladder and bile ducts were found to be completely emptied of stones demonstrated radiologically a few days prior to operation. In this connection it must be known that the administration of olive oil beguiles the uninitiated—'gall-stones' which on analysis are shown to be merely concretions of soap, are passed in the stools. The so-called treatment of gall-stones by olive oil is practised by charlatans.

(*b*) *Subsequent Treatment.*—Once a patient has had gall-stone colic, no question arises as to the treatment that should be recommended: it is cholecystectomy, unless the patient is very old or otherwise enfeebled, when cholecystostomy can be substituted. In either case it is essential to examine the common bile duct[1] by all means available for the presence of additional calculi and, if such are present, to remove them.

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[1] Necropsy studies show that in 20 per cent. of subjects who have stones in the gall-bladder there is a stone or stones in the common bile duct also.

John B. Murphy, 1857–1916. Professor of Surgery, North-western University, Chicago.

When 'silent' gall-stones are found by radiology, provided there is no contraindication to operation, the patient should be advised to undergo cholecystectomy, for in all probability later in life the operation will become imperative, and furthermore there is a small, but definite, risk of carcinoma developing in a gall-bladder containing calculi. Should gall-stones be found during the course of another abdominal operation, cholecystectomy should be performed, provided it does not add appreciably to the risk. If it is likely to do so, it is better to perform cholecystostomy, which adds little to the shock and operating time, than to leave the stones *in situ*.

CHOLECYSTITIS

Ætiology.—Cultures from excised gall-bladders and their contents show:
Wall.—65 per cent. of cultures positive.
Contents.—35 per cent. of cultures positive.

Thus it must be assumed that in over 40 per cent. of cases the inflammation is chemical, which undoubtedly it is in early cases of obstructive cholecystitis due to a calculus occluding the neck of the gall-bladder. The incidence of positive bacteriological findings vary with age, ranging up to 100 per cent. in those over seventy years of age. The commonest infecting organisms are *Esch. coli*, *streptococci*, *Salmonella paratyphi B*, and *staphylococci*, in that order. The most usual path of infection is from the liver to the gall-bladder via lymphatics, a conclusion based upon the greater ease of culturing organisms from the cystic lymph node than from the gall-bladder (Evarts Graham).

ACUTE CHOLECYSTITIS

Acute Obstructive Cholecystitis.—The contents of the gall-bladder cannot escape, for reasons shown in fig. 598. When the contents of the gall-bladder is sterile, the imprisoned bile is absorbed, but mucus continues to be secreted, giving rise to a mucocele which, if the gall-bladder is thin-walled, is translucent. Untreated, a mucocele often becomes infected and an empyema of the gall-bladder results. Frequently, when the impaction described occurs, the gall-bladder is already the seat of chronic cholecystitis and the ensuing exacerbation of infection causes the thickened gall-bladder to become intensely inflamed, with its mucous membrane swollen, and occasionally gangrenous in places. There is no doubt that the incidence of gangrene has been exaggerated through mistaken interpretation of discoloration from hæmorrhage. The subsequent events are:

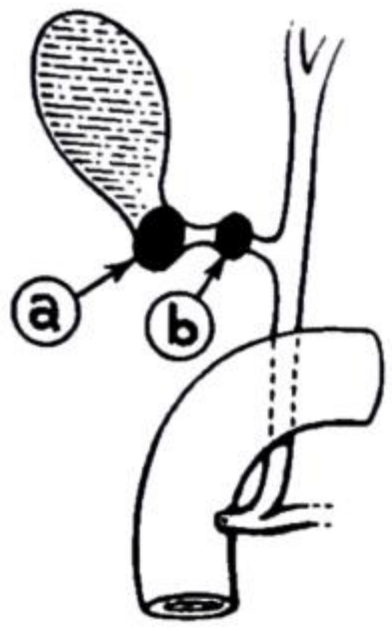

FIG. 598.—The sites of impaction of a gall-stone in acute obstructive cholecystitis: (*a*) In Hartmann's pouch. (*b*) In the cystic duct.

1. When a certain degree of distension of the gall-bladder has been reached, the mucous membrane tends to be lifted from the sides of a stone wedged in the neck of the gall-bladder; as a consequence, there is a good chance of the stone falling back into the commodious gall-bladder proper, viz.→

Evarts Ambrose Graham, 1883–1957. Professor of Surgery, Washington University, St. Louis, Missouri, U.S.A.

and the mucoid or muco-purulent contents of the bladder escape by way of the cystic duct.

2. Less frequently the impaction persists and an empyema of the gall-bladder results.

3. On rare occasions the distended inflamed gall-bladder perforates. An infected gall-bladder seldom bursts, and it does not do so early, suddenly, or without warning (Sir Heneage Ogilvie). Doubtless the infrequency of perforation is due to the thickened walls of an organ that has long been the seat of chronic cholecystitis. Perforation is favoured by cathartics and the ingestion of solid food, but with the patient confined strictly to bed and on a fluid diet it is most unusual.

Perforation of the Gall-bladder.—The site of perforation is either at the fundus, which is farthest away from the blood supply, or, less commonly, at the neck from pressure necrosis of an impacted calculus. The sequelæ of perforation of the gall-bladder are:

(*a*) Local abscess. On account of the present, and probably past, attacks of cholecystitis, there are adhesions between the gall-bladder, the great omentum and the parietal peritoneum. Such protective barriers are responsible for the fact that when an infected obstructed gall-bladder perforates the usual outcome is a local abscess.

(*b*) Perforation into the general peritoneal cavity is uncommon, and the patient is usually a man. Diffuse peritonitis supervenes readily and rapidly. This catastrophe occurs in only 0·5 per cent. of cases undergoing conservative treatment for acute cholecystitis.

(*c*) Occasionally perforation into a neighbouring hollow viscus (usually the duodenum) takes place resulting in rapid amelioration of the acute symptoms.

Internal Fistulæ of the Gall-bladder (p. 455).

Acute non-obstructive cholecystitis is less frequent than the obstructive variety. All grades of acute inflammation, from catarrhal to gangrenous cholecystitis, occur. Especially in fulminating cases, *Cl. welchii* and other anaerobic organisms may be implicated. Typhoid and paratyphoid cholecystitis can occur as a complication of these fevers. Perforation is exceptional, save in the case of acute typhoid cholecystitis (see p. 443).

Clinical Features of Acute Cholecystitis.—The onset is sudden and pain, agonising in the obstructive variety—more gradual in the non-obstructive—is located mainly in the right hypochondrium. Severe nausea and vomiting are features in the early stages. Pyrexia, sometimes to 101° F. (38° C.) or more, is usual; occasionally fulminating cases are ushered in by rigors. On the second or third day of the attack transient slight jaundice [1] is not unusual.

On examination tenderness and rigidity are found in the right hypochondrium. If the patient can be persuaded to relax, particularly in obstructive cases, a mass consisting of the inflamed gall-bladder with attached omentum can be felt. In cases of mucocele of the gall-bladder before infection has occurred pyrexia is absent, and, following an attack of gall-stone colic, a piriform swelling can be palpated in the right hypochondrium.

When abdominal rigidity is a notable feature, the signs closely mimic those of acute appendicitis, an important difference being that the maximum rigidity is inclined to

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[1] Stones are found in the extrahepatic biliary passages in about 15 per cent. of patients admitted with acute cholecystitis. In patients over sixty years of age this percentage is higher.

Sir Heneage Ogilvie, Contemporary. Consulting Surgeon, Guy's Hospital, London.
William H. Welch, 1850–1934. Pathologist, Johns Hopkins University, Baltimore, U.S.A.

be higher, that is nearer the costal margin. The differential diagnosis between high retrocæcal acute appendicitis and acute cholecystitis is often difficult (see Chapter 27). Acute pyelitis and other acute inflammations of the right kidney must also be excluded. When the pain is severe, and rigidity is slight or lacking, acute pancreatitis must receive diagnostic consideration. A serum amylase estimation is never out of place in cases where there is even a slight doubt as to the correctness of the diagnosis of acute cholecystitis. In necessary cases an electrocardiograph should be taken to differentiate acute cholecystitis from coronary thrombosis.

Boas's sign, if positive, is sometimes most helpful in distinguishing acute cholecystitis from other conditions. There is an area of hyperæsthesia between the ninth and the eleventh ribs posteriorly on the right side.

Radiology.—A plain radiograph in an adult is nearly always negative. Exceptionally, a gall-stone or stones are visualised. In children with this condition opaque calculi are demonstrated frequently (I. Forshall). In a few fulminating cases gas can be demonstrated within the gall-bladder. In such cases the organism responsible is almost always the *Cl. welchii*, and the patient is usually a diabetic. When gas is visualised, operation should be undertaken without delay. When it is imperative to differentiate acute cholecystitis from other intra-abdominal catastrophies, intravenous (biligraphin) cholecystography is often most helpful. Demonstration of the gall-bladder and the bile ducts offers good evidence against the diagnosis of cholecystitis. The presence of visualisation of the bile ducts and non-visualisation of the gall-bladder is compatible with the diagnosis of acute cholecystitis. Failure to visualise any part of the biliary tree is likely to be due to liver disease, or to obstruction of the common bile duct.

Treatment:

Conservative Treatment followed by Cholecystectomy.—Experience shows that in more than 90 per cent. of cases the symptoms of acute cholecystitis subside with conservative measures. Non-operative treatment is based upon four principles:

1. *Rest to the inflamed gall-bladder* and the biliary and pancreatic systems by gastric aspiration for three to five days. Nothing is given by mouth. Fluid and electrolyte balance are maintained by continuous intravenous dextrose-saline solution, with the usual precautions.

2. *Sedation.*—Morphine is contraindicated because it increases the spasm of the sphincter of Oddi. Pethidine[1] (B.P.C.) is free from this objection, and 2 ml. (34 minims) can be given intravenously. For repeated administration, it is safer to give the drug intramuscularly, as intravenous pethidine is not without danger in patients with parlous liver function.

3. *An anticholinergic drug* is given to reduce gastric and pancreatic secretion, and therefore loss of electrolytes by aspiration, and to relax the sphincter of Oddi. Probanthine is probably the most satisfactory for this purpose. (For further details see p. 466.)

4. **Antibiotics** of the tetracycline group (aureomycin or terramycin) reach the interior of the gall-bladder via the blood-stream even in the presence of occlusion of the cystic duct. In the initial stages doses of 1 to 1·5 G. are given intravenously *bis die*, but because of the liability of repeated injections

[1] Known in the U.S.A. as Demerol.

Ismar Boas, 1858–1939. Gastro-enterologist, Berlin.
Isabella Forshall, Contemporary. Surgeon, Royal Liverpool Children's Hospital, Liverpool.

to cause thrombophlebitis, oral administration should be commenced as soon as the patient is permitted to take fluids by mouth.

When the temperature, pulse, and other physical signs show that the inflammation is subsiding (usually by the third day), after the bowels have been emptied, e.g. by a glycerine suppository, the gastric aspiration tube is removed, and commencing with flavoured dextrose drinks, fat-free clear fluids are given for one day, and a soft fat-free diet on the next. A full fat-free diet is then given, and the patient encouraged to walk. No fats should be given during the convalescent phase. Eight to ten weeks after the acute symptoms have subsided cholecystectomy is carried out.

Conservative treatment is not advised when there is uncertainty about the diagnosis, e.g. when early high retrocæcal appendicitis or a leaking duodenal ulcer cannot be excluded.

Conservative treatment must be abandoned: (1) If, after a period of twenty-four to thirty-six hours, the pulse-rate and temperature are not falling, the pain persists, and the physical signs point to an empyema of the gall-bladder, operation should be undertaken forthwith. In such circumstances it is usually advisable to limit the operation to cholecystostomy[1] with removal of an impacted stone, if such be present—a dependable, safe operative procedure. (2) On the other hand, when the absence of pyrexia and the presence of a large piriform swelling in the right hypochondrium make the diagnosis of mucocele of the gall-bladder probable, after excretory pyelography has been performed to exclude a right hydronephrosis, operation is best carried out in a matter of hours in order (*a*) to avoid perforation; (*b*) to obviate the transference of a mucocele into an empyema of the gall-bladder. In these circumstances most operators would favour cholecystectomy.

When a gall-bladder perforates into the general peritoneal cavity, urgent drainage of the peritoneal cavity and the gall-bladder is imperative; after such measures, performed promptly, 60 per cent. of the patients recover. When operation is delayed the mortality approaches 100 per cent.

Typhoid fever is comparatively rare except in tropical countries. Because of the danger of perforation in acute typhoid cholecystitis (see p. 537), very early operation must always be advised.

(*b*) *Routine Early Operation.*—Some surgeons, especially in the U.S.A., advocate urgent operation as a routine measure in cases of acute cholecystitis; their number is decreasing. It must be stressed that such early operation does not preclude proper pre-operative preparation, and when necessary up to twelve hours may be required to restore fluid and electrolytic balance. Provided the operation is undertaken within forty-eight hours of the onset of the attack, excellent results are obtained, but no better than those that accrue from the delayed method described above, which is associated with fewer operative injuries to the main ducts.

It is not proposed here to enter into a controversy on the merits and demerits of early and delayed operation for acute cholecystitis, but to state categorically that the most dangerous period for operation is between the third and tenth day of the attack. Should operation become imperative during this period, when the ducts are often obscured by œdema and the liver function is at a low ebb, it is safer to perform a simple cholecystostomy than a difficult cholecystectomy.

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[1] Elective cholecystectomy is still indicated in due course, even if cholecystostomy has relieved all symptoms.

CHRONIC CHOLECYSTITIS

Chronic cholecystitis can, and frequently does, occur apart from gall-stones, although should the infection persist, gall-stones will almost certainly form in the infected gall-bladder. Conversely, a sterile metabolic gall-stone, particularly a solitary cholesterol stone, by temporarily occluding the cystic duct, favours the development of both chronic and acute-on-chronic cholecystitis.

Pathology.—When the gall-bladder is chronically infected it loses its normal bluish translucency and elasticity and becomes thickened and opaque; its mucous membrane, in particular, shows evidence of chronic inflammation. Variable adhesions are found about the gall-bladder; sometimes these are exceedingly dense.

Typhoid Carriers.[1]—Typhoid carriers harbour living *Bact. typhosum* in their gall-bladders. These are discharged into the alimentary tract from time to time, and eliminated through the fæces. Cholecystectomy in such cases rids the patient of his infection and the community of a potential danger.

Clinical Features.—While the disease is most common in middle life, often the original infection dates back to adolescence, or even childhood. Females are affected more commonly than males (3 : 1). Gall-bladder dyspepsia in some respects simulates that of chronic peptic ulcer; it differs from it in the following particulars.

Periodicity, so frequently present in cases of chronic peptic ulcer, is lacking.

Pain (epigastric, right subcostal, or at the angle of the scapula) is sometimes associated with the flatulent dyspepsia. Food does not relieve the pain as it does in duodenal ulcer; indeed, a heavy meal is regretted almost immediately.

Nausea is frequent; so is heartburn.

Flatulence.—The patient feels distended soon after meals, and women find it necessary to loosen their corsets. Belching is a regular accompaniment.

It is probable that cholecystitis brings about pyloric spasm, and in order to overcome that spasm the patient swallows air. Here, at least, is an explanation of the belching.

Sometimes there is associated hyperchlorhydria, which accounts for the heartburn; at other times the test-meal is normal or, what is quite frequent, it shows the presence of *hypo*chlorhydria.

Chronic cholecystitis without gall-stone colic is often overlooked, for the remaining symptoms are extremely varied. For instance, pseudo-anginal attacks sometimes have their origin in a gas-laden stomach secondary to a diseased gall-bladder (C. Miller).

Differential diagnosis between œsophageal hiatus hernia and cholecystitis is sometimes extremely difficult, and always requires radiological assistance. Concomitant chronic pancreatitis should be considered in atypical cases.

Radiography.—A reliable sign of *obstructive* cholecystitis is failure of the gall-bladder to cast a shadow in any of the serial radiographs. It is, of course, essential to be certain that the patient has ingested the contrast medium. Failure of the gall-bladder to contract normally after a fatty meal suggests *non-obstructive* cholecystitis.

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[1] 'Typhoid Mary,' a cook-general who passed *Salmonella typhi* in her fæces and urine, was responsible for nearly a score of epidemics of typhoid in and around New York City.

Charles Miller, 1875–1939. Physician, University College Hospital, London.

Treatment.—In cases of obstructive cholecystitis, cholecystectomy is indicated. For many patients suffering from non-obstructive cholecystitis, medical treatment, which consists of a careful dietetic régime, cholinergic drugs, antibiotic therapy based upon the result of culture of duodenal aspirate and endocrine treatment for those who also suffer from hypothyroidism, together with regular exercise for those of sedentary habit, and possibly spa treatment, is often successful. If, as frequently happens, relapses occur, or the patient cannot or will not adhere to the régime, cholecystectomy is advised. In order to eliminate carriers, cholecystectomy is an absolute necessity in cases of typhoid or paratyphoid cholecystitis.

THE DISEASED GALL-BLADDER IN RELATION TO CARDIAC DISORDERS

While gall-bladder disease sometimes mimics coronary or anginal symptomatology, it is possible for afferent impulses from a diseased gall-bladder to affect adversely a poorly vascularised myocardium, and thus produce angina pectoris or heart-block. Cardiac pain experienced at rest, and particularly after meals or at night, suggests the possibility of an exciting stimulus from a diseased gall-bladder. Patients with myocardial disease, especially those suffering from angina pectoris, are often benefited by cholecystectomy, provided the gall-bladder is unquestionably diseased. Recent myocardial infarction or decompensation of a valvular lesion are definite contraindications to operation.

INTRAMURAL DIVERTICULOSIS OF THE GALL-BLADDER

The mucous membrane of the gall-bladder herniates through the muscle layers in many places, the condition being a deepening of the normal lacunæ of Luschka. Diverticulosis of the gall-bladder can be demonstrated by cholecystography, especially after the gall-bladder contracts following a fatty meal. Unlike colonic diverticulosis, which is not necessarily a harmful state, diverticulosis of the gall-bladder always must be regarded as a dangerous condition. The symptoms are indistinguishable from those of chronic cholecystitis.

At operation, first of all a cause of increased intraluminar pressure should be eliminated by palpation of the ducts and the head of the pancreas, and then by cholangiography. In cases where there is organic obstruction or spasm of the sphincter of Oddi, that must be remedied. In other cases cholecystectomy alone should be carried out, and invariably the gall-bladder is found to be the seat of chronic cholecystitis; occasionally gall-stones are present.

LAPAROTOMY FOR CHOLECYSTITIS AND GALL-STONES

A right paramedian incision is satisfactory when the patient is slender and has a narrow subcostal angle; when the patient is stout and has a wide subcostal angle, a transverse, Mayo Robson's, or Kocher's incision is preferable (fig. 599). After the peritoneum has been opened, the gall-

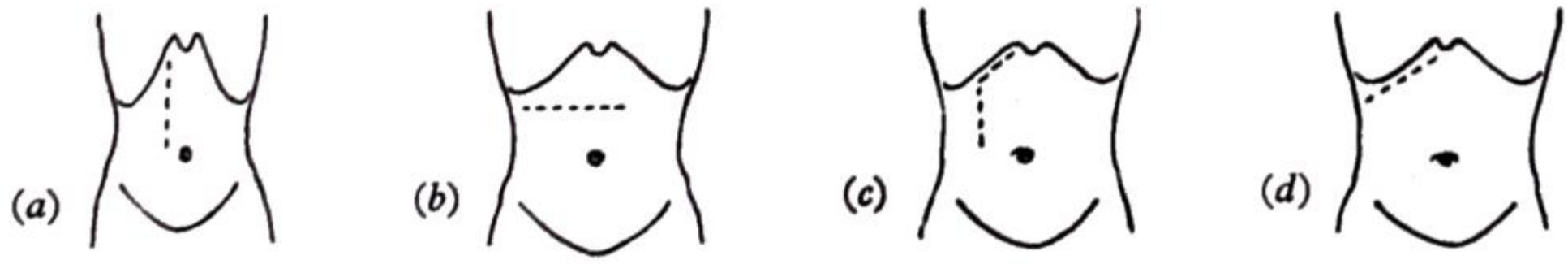

FIG. 599.—Incisions suitable for displaying the gall-bladder: (*a*) Paramedian; (*b*) Transverse; (*c*) Mayo Robson's; (*d*) Kocher's.

bladder is inspected and palpated. The field is isolated with packs. Much of the fluid content of a gall-bladder not packed with stones can be aspirated, and the fluid preserved for bacteriological examination. The fundus of the gall-bladder is then grasped with Denis Browne forceps, thereby

Sir Arthur William Mayo Robson, 1853–1933. Surgeon, General Infirmary, Leeds.
Theodore Kocher, 1841–1917. Professor of Clinical Surgery, University of Berne.
Denis Browne, Contemporary. Surgeon, Hospital for Sick Children, Great Ormond Street, London.

sealing the puncture. The forceps act as an excellent tractor if cholecystectomy is undertaken. After re-palpating the collapsed gall-bladder, should small calculi be felt therein, or particularly if the patient gives a history of jaundice, the cystic and the common bile ducts are palpated for a stone. If this examination is negative and obstruction to the common bile duct is suspected, when facilities exist, operative cholangiography (see p. 431) is most desirable.

Standard Cholecystectomy.—Elevation of the 'bridge' of the operating table in order to arch the back, and thus help to display the environs of the junction of the cystic and common bile ducts, is not recommended as a routine measure because it is liable to strain the spinal ligaments and so cause backache afterwards. On the other hand, it is better to employ this device than to administer muscle relaxants to a patient with impaired liver function. It has been shown that the unbridled use of these agents in large series of cholecystectomies is associated with a raised mortality rate. The liver is rotated by traction on the gall-bladder by the aforementioned forceps, and by dissection the junction of the cystic, the common hepatic, and the common bile ducts is displayed (fig. 600). Removal of the gall-bladder should never be commenced until all three ducts have been exposed clearly. The cystic duct is then divided between ligatures. The cystic artery is ligated and severed. From below, upwards, the gall-bladder is dissected from its bed in the liver, and as the dissection proceeds, so the peritoneum is coapted (fig. 601) over the raw area on the under-surface of the liver. The gall-bladder having been removed and hæmostasis assured, the abdominal wall is closed, leaving a soft rubber drain down to the site of the severed cystic duct. Drainage is advisable on account of the bile which oozes from the gall-bladder bed for a few days.

FIG. 600.—Cholecystectomy. The junction of the cystic, common hepatic, and common bile ducts has been displayed and the cystic artery has been clamped and divided.
FIG. 601.—Standard cholecystectomy. Freeing the gall-bladder from its bed and approximating the peritoneal reflections.

Thorek's modified cholecystectomy is particularly useful when the gall-bladder is deeply buried in the liver, but it can be employed as an alternative to standard cholecystectomy. The object is to prevent bile seeping from liver tissue bereft of its natural covering (the gall-bladder bed). The cystic duct and

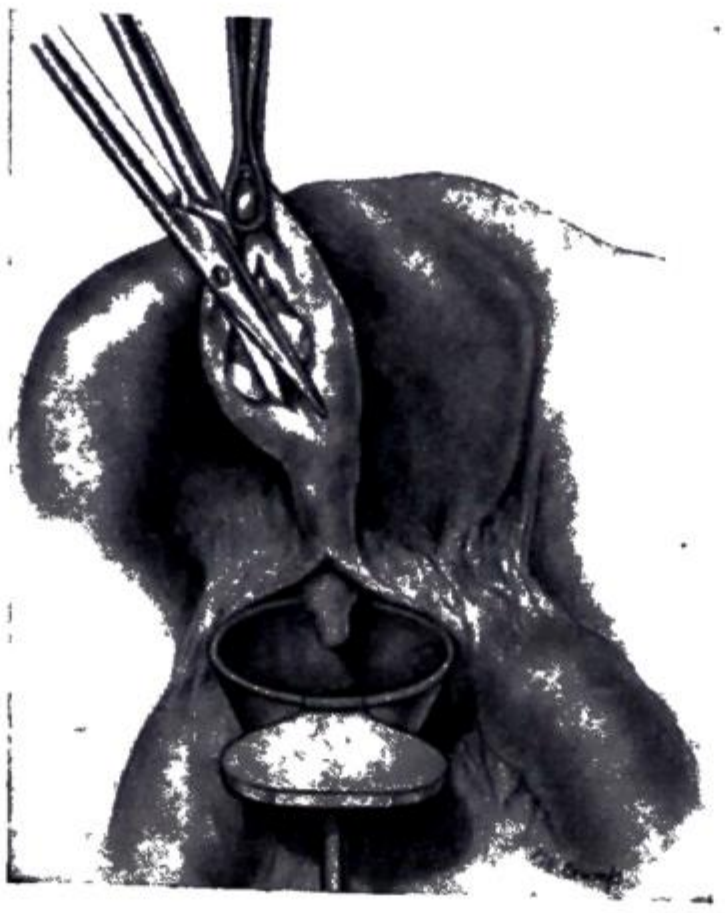

FIG. 602.—The gall-bladder is split open and its contents evacuated into a receptacle. FIG. 603.—That part of the gall-bladder which remains attached to the liver is electrocoagulated.

Max Thorek, Contemporary. Surgeon, Cook County Hospital, Chicago.

the cystic artery are divided in the usual manner. The gall-bladder is slit along its length and the contents evacuated (fig. 602). The lateral walls of the gall-bladder are then excised with a diathermy needle, leaving that part of the gall-bladder attached to the liver *in situ*. The latter is coagulated with the diathermy current, using a ball or button electrode (fig. 603), and afterwards covered with a detached piece of the greater omentum or falciform ligament of the liver. The abdomen can be closed without drainage, for there is no seepage of bile because the gall-bladder bed in the liver has not been opened.

Diathermy Dissection of the Gall-bladder.—Some surgeons remove the whole gall-bladder by diathermy dissection, and if the bed of the liver is dry, close the wound.

Cholecystostomy.—The gall-bladder having been displayed through a suitable incision, its fundus is isolated by packs. Two stay sutures are inserted on either side of the fundus, in order to steady the organ, the fluid contents of which is aspirated. The fundus is opened and stones are removed from the interior by Desjardins' forceps, aided, if necessary, by a finger milking up a stone or stones from Hartmann's pouch. Minute calculi are often dislodged by strips of dry gauze passed into the interior. A ¼-in. (6-mm.) drainage tube is passed into the gall-bladder, and there retained by a transfixion stitch. The opening in the gall-bladder is closed about the tube. The tube is brought through a portion of greater omentum, which is anchored to the gall-bladder by the original stay sutures. Depending upon the incision in the abdominal wall that has been used, the tube is brought through the incision, or through a special stab incision. The abdominal incision is closed, and the tube is joined by a glass connection to more tubing, which leads to a sterile bottle. The tube is removed in about ten days, and provided there is no obstruction to the ducts, the biliary fistula heals within a week.

STONE IN THE COMMON BILE DUCT

The bile ducts, as opposed to the gall-bladder, have little or no muscle in their walls; therefore they are unable to expel stones. When the gall-bladder is chronically inflamed, and especially if it is full of stones, the flushing action on the common bile duct by contractions of this little reservoir is feeble; when the gall-bladder is fibrotic, it is nil. In such circumstances, and after cholecystectomy, the chances of any but a tiny calculus being swept into the duodenum by a flow of bile during a period of relaxation of Oddi's sphincter are slender.

Clinical Features.—It is possible for a gall-stone to be present in the common bile duct for months or even years without disabling the patient; conversely, serious symptoms are liable to supervene at any time. Usually there is a long history of gall-bladder dyspepsia, not infrequently punctuated by episodes of biliary colic.

Pain.—There is no fundamental difference between the biliary colic described already (p. 438) and that occasioned by a stone becoming impacted in the common bile duct. In the latter instance the pain is likely to be prolonged, severe, and difficult to relieve, and is followed by jaundice of varying degree.

Jaundice.—Within forty-eight hours the pain is followed by jaundice, the tinge of which ranges from pale lemon to bright orange, and varies from day to day. Even when the yellow tinge is no longer perceptible, the serum bilirubin estimation remains elevated for several days.

Abdominal Examination.—Usually, soon after the attack of colic, tenderness can be elicited in the epigastrium. As a rule the gall-bladder is impalpable.

Courvoisier's Law states that if in a jaundiced patient the gall-bladder is palpably enlarged, it is *not* a case of stone impacted in the common bile duct, because previous

Ludwig T. Courvoisier, 1843–1918. Professor of Surgery, Basle.

cholecystitis must have rendered the gall-bladder fibrotic. There are several exceptions to this law, notably (*a*) double impaction, where one stone is arrested in the cystic and another in the common bile duct, and (*b*) a pancreatic calculus causing obturation at the ampulla of Vater.

Sometimes it happens that after an attack of biliary colic, followed by fleeting jaundice, the patient remains well for many months. More often than not, after a varying interval, another similar attack occurs, and it is sometimes presumed that another gall-stone has entered the common bile duct. But this is not necessarily the case; it is quite probable that the same stone has again caused some obstruction, perhaps a little farther down the duct.

Impaction of a stone (fig. 604) can occur in the supraduodenal or retroduodenal portions of the common duct (which are the usual sites), or at the ampulla of Vater or, very rarely, in the common hepatic duct. When

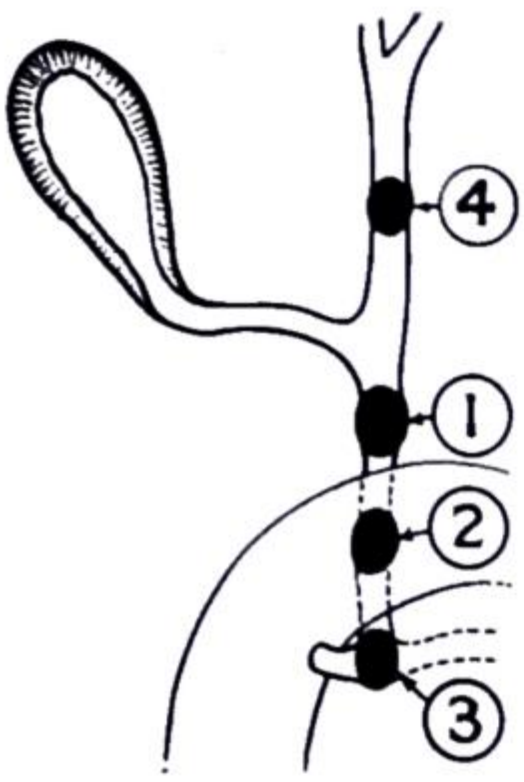

FIG. 604.—1. Stone impacted in the supraduodenal portion of the common bile duct (commonest site).
2. Stone impacted in the retroduodenal portion of the common bile duct (second most common site).
3. Stone impacted in the ampulla of Vater (10 per cent. of cases).
4. Stone impacted in the common hepatic duct (very rare).
Note that the gall-bladder is usually thick-walled and contracted because of chronic cholecystitis.

impaction takes place severe biliary colic occurs, and the colic continues at varying intervals. With each episode of pain the jaundice deepens. Gradually the pain lessens, and eventually it passes off completely, but the jaundice persists. Nevertheless, unlike other types of jaundice, from which it must be distinguished, it varies in intensity from day to day. This variation is due to exacerbations of the concomitant cholangitis and not to a ball-valve action of the impacted calculus. As bile salts accumulate in the blood, the skin commences to itch, sometimes intolerably. Pyrexia occurs in one-third of cases. It is usually mild, although there may be one or two ' spikes ' of elevated temperature towards the end of a series of attacks of biliary colic. The patient loses weight.

Atypical Cases:

1. **Jaundice is the First Symptom.**—In about 3 per cent. of individuals jaundice is the first and only symptom. A short cystic duct without Heister's valve or an absent cystic duct (see fig. 577, p. 429) favours this confusing symptomatology. After other causes have been eliminated as far as possible, laparotomy, if necessary with operative cholangiography, is essential to establish the diagnosis.

2. **Stone in the Common Bile Duct without Jaundice.**—A stone or stones may lie dormant in the common bile duct, giving rise only to vague indigestion and perhaps an occasional tinge of jaundice. Twice as many unsuspected common duct calculi are found during operations upon the gall-bladder in patients over sixty years of age as in those under that age. Intravenous cholangiography is valuable in demonstrating these unsuspected calculi.

3. **Jaundice from Pressure of a Stone on the Duct from Without.**—Jaundice can arise from a large calculus in Hartmann's pouch pressing upon the common bile duct.

Differential Diagnosis.—Calculous biliary obstruction is the most frequent cause of jaundice encountered in surgical practice (fig. 605). It is important to differentiate a stone in the common duct from virus hepatitis (see p. 398) and carcinoma of the ampulla of Vater (see p. 475). Blood is almost always present in the stools in carcinoma of the ampulla, while virus hepatitis severe enough to produce jaundice results in positive liver deficiency tests.

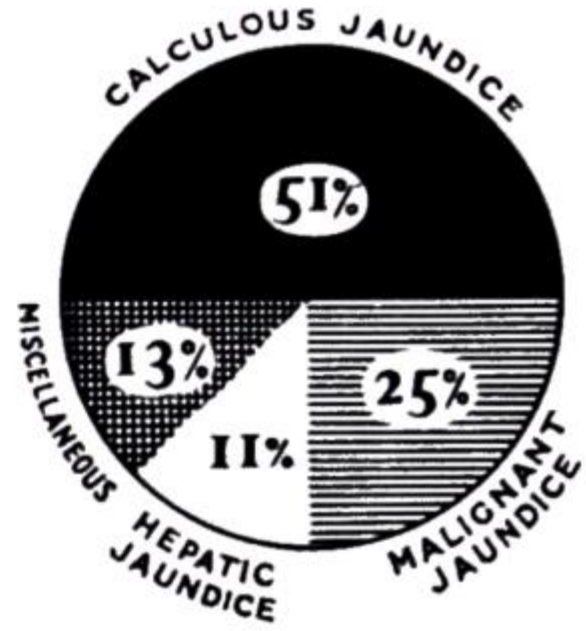

FIG. 605.—The relative frequency of conditions causing jaundice met with in surgical practice. (*After John Bruce.*)

Laboratory Investigations:

Serum bilirubin estimation (normal 0·5 mg. per 100 ml.) is the most valuable of all laboratory investigations for confirming the diagnosis of calculous biliary obstruction, provided it is undertaken frequently. By this test the variations in the depth of the jaundice can be registered with far greater precision than is possible with naked eye observations.

Serum alkaline phosphatase (normal value 3 to 13 King-Armstrong (K–A) units). Values below 20 favour hepatogenous jaundice; those above 35, obstructive jaundice.

Complications.—If the obstruction is not relieved, one of three dangerous complications ensues:

1. **Liver Function becomes increasingly Impaired.**—When liver function becomes seriously depressed, pigment excretion ceases; resorption of pigments from the stagnant bile within the dilated, obstructed ducts occurs, but the mucous membrane of the duct continues to secrete mucus, thus the ducts above the impacted calculus become distended with '*white bile*.' The finding at operation of white bile[1] is of grave, but not necessarily fatal, significance. Timely liver function tests (see p. 394) are often of paramount importance in determining latent liver damage.

2. **Suppurative Cholangitis Supervenes.**—When there is stagnation of bile, infection is prone to occur. Suppurative cholangitis is ushered in by a rigor, and later the temperature chart reveals sudden elevations, precipitate descents, and complete intermissions, known colloquially as the 'steeple chart.' Suppurative cholangitis is described on p. 401. Fortunately this complication, common in days gone by, is, as a result of timely surgical intervention and antibiotic therapy, now comparatively infrequent.

3. **The gall-stone ulcerates through the wall of the common bile duct,** and diffuse peritonitis supervenes. This form of bile peritonitis, occurring in a subject with grossly impaired liver function and infected bile, is rarely amenable to operative or any kind of treatment, although operation should be attempted. At

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[1] A new synthetic cholagogue, zanchol (G. D. Searle and Co. Ltd.), one 250 mg. tablet three or four times a day administered orally after the common bile duct has been drained, stimulates the flow of bile so effectively that bile just tinged yellow soon becomes a brilliant green hue.

Earl Judson King, Contemporary. Professor of Chemical Pathology, University of London (Post-Graduate Medical School).
Arthur Riley Armstrong, Contemporary. Director of Laboratories, Mountain Sanatorium, Hamilton, Ontario, Canada.

operation the perforation may be minute and difficult to find. Sometimes the perforation occurs into the retroperitoneal tissues. The treatment, which is exceptionally urgent, is to drain the common bile duct, and also the general peritoneal cavity or the retroperitoneal tissues, as the case may be. Lastly,

4. On rare occasions (as subsequently revealed at necropsy) a stone in the common bile duct ulcerates into the duodenum, and a natural cure follows.

MANAGEMENT OF CALCULOUS BILIARY OBSTRUCTION

To commence with, conservative treatment, which includes a high intake of dextrose to build up the store of liver glycogen, and also protect the liver, together with the exhibition of pethidine when severe pain occurs, is instituted.

The Jaundice Abates.—As a result of the above measures, the depth of the jaundice may diminish, the raised bilirubin level becomes constantly lower, and the stools become increasingly coloured. Such a train of events sometimes culminates in complete disappearance of the jaundice, and implies disimpaction of the stone, or possibly the passage of a small stone into the duodenum. In such circumstances intravenous cholangiography can be undertaken safely, and if the liver function tests are satisfactory laparotomy can be undertaken with only the same precautions as for gall-stones in the gall-bladder.

The Jaundice does not Abate.—At any time during conservative treatment if, over a period of days, the jaundice is found to be increasing rather than decreasing, and particularly when any degree of jaundice is accompanied by pyrexia, operation should be planned to take place within two or three days. Jaundiced patients tend to ooze from a cut surface. This tendency to bleed is due to a diminution of prothrombin in the blood, resulting from failure of absorption of vitamin K_1 from the alimentary canal, which in turn is consequent upon diminution of bile salts in the alimentary canal. To raise the prothrombin level of the blood, injections of vitamin K_1 100 mg. are given intravenously.[1] Should urgency forbid the delay of awaiting 80 per cent. of normal prothrombin activity, a transfusion of *fresh* blood must be given before the operation is commenced. Antibiotic therapy (terramycin, which is secreted in the bile) is also given. In a jaundiced patient, the length of the operation should vary inversely with the depth of the jaundice. In these circumstances local anæsthesia is the safest, but in any case muscular relaxants, which are dangerous in the presence of impaired liver function, must be forbidden. In patients who are deeply jaundiced, or who show signs of suppurative cholangitis, operation should be limited to draining the common bile duct (choledochostomy), removing a stone or stones only if they are readily accessible. If it is impracticable to remove an impacted calculus, a biliary fistula will probably result, and when the jaundice abates a further operation, commonly, but not necessarily (see p. 453), will have to be undertaken.

Choledocholithotomy.—If a stone, or stones, is present in the common bile duct, its removal should have priority over cholecystectomy if that

[1] K_1 for intravenous use—mephyton (Sharpe and Dohme Ltd.).

operation is contemplated. Unless the stone is readily palpable in the common bile duct, or has been demonstrated by pre-operative intravenous cholangiography, operative cholangiography (see p. 431) is most desirable.

When it has been decided to explore the common bile duct, and that duct has been exposed, J. Garlock advises that the surgeon should move to the left side of the operating table, and carry out the rest of the manœuvres from that side. First, the duodenum should be mobilised completely. The surgeon now holds the duodenum and the contiguous head of the pancreas between the left index and middle fingers behind and the thumb in front. Second, he passes a probe down the common duct from above, so that the instrument can act as a guide for the examining fingers of the left hand. In this way calculi can be palpated even in the lowest portion of the duct.

Supraduodenal Choledochostomy.—Most stones in the common bile duct can be removed by this route. If, as is often the case, a stone can be felt, an attempt is made to manœuvre it into a position midway between the entrance of the cystic duct and the superior border of the duodenum.

Direct Choledocholithotomy.—When the stone is situated in, or can be manipulated into, the above desired position, the stone having been steadied between the finger and thumb, the peritoneum overlying the duct is incised and dissected from the duct, which is opened longitudinally directly on to the stone, enabling it to be removed by a Moynihan's malleable scoop or Desjardins' gall-stone forceps. The interior of the duct is then explored upwards and downwards with the scoop for further stones.

Indirect Choledocholithotomy.—When the stone cannot be felt, or cannot be manipulated into the optimum position just described, after incising the peritoneum overlying the supraduodenal portion of the common bile duct, a length of the underlying structure is displayed. When there are numerous adhesions, and especially when the gall-bladder has been removed at a previous operation, it is sometimes difficult to be certain whether the structure in question is the common bile duct or the portal vein. Aspiration through a fine hypodermic needle connected to a syringe will settle this point. As soon as about ¾ inch (2 cm.) of the common bile duct has been exposed, the duct is transfixed by two stay sutures and a longitudinal incision into the duct is made between them. Escaping bile is mopped up or removed by suction. Through this opening it may be possible to identify the stone and remove it with a scoop or forceps (fig. 606). After removal of the stone or stones by either of the above methods the common bile duct is cleared of any mud or grit by introducing and removing strips of ribbon gauze, followed by irrigation with saline solution. The sphincter of Oddi must always be dilated with graduated bougies (Bakes') up to the diameter of 8 or 9 mm. Drainage of the common bile duct should always follow; it is best carried out by means of a T-tube (fig. 607). The horizontal limbs are passed into the duct, which is closed about the vertical limb.

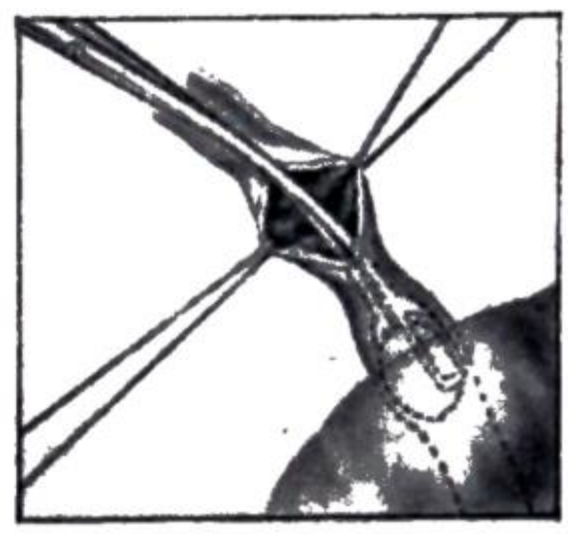

FIG. 606.—Choledochotomy. The stone has been seized with Desjardins' forceps.

FIG. 607.—T-tube for draining the common bile duct.

The vertical limb of the tube is brought out through a small incision just to the left of the middle line, the better to avoid the tube being removed (by mistake) when the peritoneal drain is withdrawn. Only when a post-operative cholangiograph via the tube shows that the medium enters the duodenum normally, is the T-tube removed.

Choledochoduodenostomy is certainly indicated for recurrence of stone in the common bile duct. So good are the results that this measure

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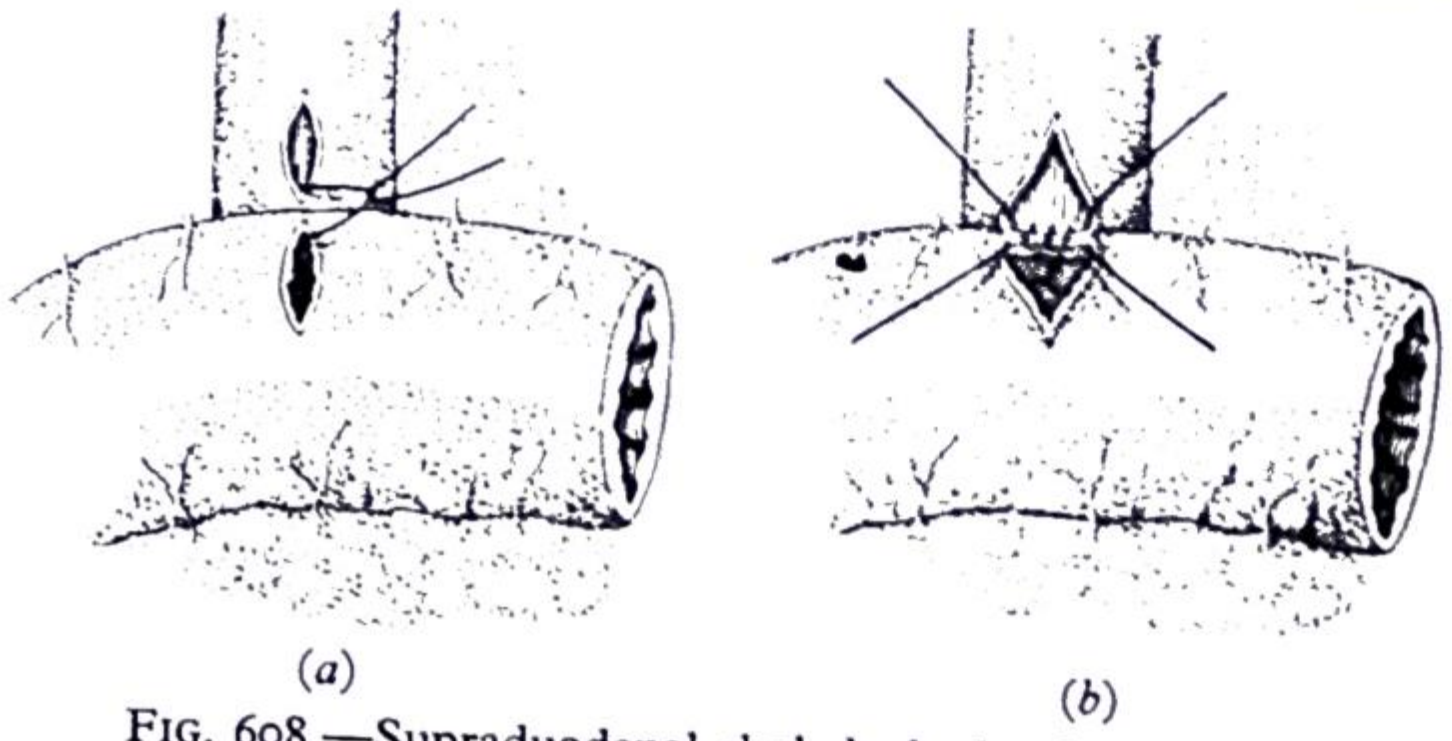
Abel Desjardins, Contemporary. Consulting Surgeon, Dispensaire Henri de Rothschild, Paris.
Johann Bakes, 1871–1930. Chief Surgeon, Landeskrankenhauses, Brünn.

is recommended in addition to choledocholithotomy at a first operation for common duct stones when such calculi are associated with much mud or sand, and particularly when the duct is thickened as well as dilated (J. Hosford).

Operation.—After the common bile duct has been explored, and it is decided to perform choledochoduodenostomy, a vertical incision is made in the duodenum. The edges of the common duct are held apart by stay sutures, and the anastomosis is carried out with interrupted sutures of fine catgut. The first suture joins the lower end of the incision in the duct to the upper end of the incision in the duodenum (fig. 608(*a*)). Sutures are then placed on each side alternately, from above downwards, each being tied after it has been inserted, and so arranged that the cut edges are inverted (fig. 608(*b*)). The stoma is about 2 cm. in length and six or seven sutures are placed on each side. A few reinforcing sutures are placed here and there. A corrugated rubber drain is introduced down to the site of the anastomosis.



(*a*) (*b*)

FIG. 608.—Supraduodenal choledochoduodenostomy. (*After J. Hosford.*)

The transduodenal route is indicated when a stone is found to be wedged near the ampulla of Vater and it cannot be retrieved from above.

Operation.—The anterior wall of the already mobilised second part of the duodenum is incised. The contents of the duodenum, if any, are evacuated, preferably by suction. Occasionally a stone can be seen in the ampulla of Vater (fig. 609), and can be removed easily by a scoop. At others sphincterotomy must be performed. Bleeding is controlled, and the duodenum is repaired as directed on p. 468.

FIG. 609.—The transduodenal approach to a stone impacted in the ampulla of Vater.

A strong case can be made out for the employment of this route when the stone is situated in the lower third of the common bile duct. The diameter of the bile duct increases from the liver down to the ampulla of Vater. Here the sphincter of Oddi produces a normal constriction. Sphincterotomy abolishes this constriction and allows free egress of stones from any part of the main biliary tree into the duodenum. (It is estimated that 7 per cent. of patients with cholelithiasis have intrahepatic calculi in addition.) Furthermore, the post-cholecystectomy syndrome is prevented by this method. Lastly, external drainage of the common bile duct is unnecessary.

★ ★ ★

In the majority of cases, when a stone or stones can be removed from the common bile duct with comparative ease, cholecystectomy can be carried out in addition. In other circumstances additional cholecystostomy can be performed or the diseased gall-bladder can be left for removal on a future occasion. After choledocholithotomy stones rarely re-form in the common bile duct, unless it remains seriously infected or a stricture develops.

John Hosford, Contemporary. Surgeon, St. Bartholomew's Hospital, London.

RECURRENT AND RESIDUAL STONES IN THE COMMON BILE DUCT

In a few instances a new calculus forms after choledocholithotomy; more often the 'new' stone is one overlooked at the first operation. When the fact that a stone in the common bile duct has been overlooked is recognised in the early post-operative period by cholangiography, or it is considered safer to drain the common bile duct without removing any or all the stones —as is not infrequently advisable in cases of profound obstructive jaundice— provided the stone is not causing complete obstruction, the following conservative measures are indicated. The patient receives 3 to 4 G. of bile salts per day by mouth, in divided doses, or better zanchol (see p. 449). In a very high percentage of cases the shadows disappear in eight to thirty weeks. If, at the end of the latter period, the shadow persists, the stone must be removed by a second operation. Other authors recommend the introduction of solvents down the T-tube; warm chloroform appears to be the most effective. Nitroglycerine tablets by mouth cause decrease in the tone of the choledochal sphincter, and are worth trying.

The results of choledocholithotomy for recurrent calculi are not as good as might be expected. Re-formation of stones requiring a third operation occur in a quarter of the cases, and symptoms persist in rather more than another quarter (E. S. R. Hughes). This is a strong indication for employment of the transduodenal route for removing recurrent choledochal calculi. The advantages of approaching the stone through the duodenum are a field comparatively free from adhesions and, above all, being enabled to perform sphincterotomy during the course of the operation.

ASIATIC CHOLANGIO-HEPATITIS

Obstruction to the common bile duct due initially to infestation by liver flukes is frequently encountered in the Far East: in these countries it is more common than inflammatory disease of the gall-bladder alone. The patient presents with pain, jaundice, and intermittent pyrexia. The urine is tea-coloured. Clay-coloured stools are less frequently observed. Secondary infection of the obstructed ducts has almost always occurred by the time the patient seeks relief. Laparotomy should be undertaken as soon as the patient can be rendered fit to undergo the operation.

Operation.—The gall-bladder, which in this condition is an innocent bystander, should be left intact, as it is a useful safety valve (F. E. Stock). Palpation of the ducts often reveals the presence of calculi, and it is not unusual for the whole of the accessible system to feel solid with impacted stones. On opening the common bile duct thick, purulent bile, often containing adult clonorchis, gushes forth, and it is removed by suction. The gall-bladder is emptied by gentle pressure. The common bile duct is cleared of stones and mud by scoop and forceps, aided by irrigation with normal saline solution. Choledocho-duodenostomy at the initial exploratory operation gives better results than T-tube drainage: the new wide stoma allows the stones to pass into the duodenum, and prevents recurrence. Pre- and post-operative antibiotics, the latter being guided by the bacteriological report, help to overcome the infection.

STRICTURE OF THE COMMON BILE DUCT

Ninety per cent. of acquired strictures of the common bile duct, excluding those of the ampulla of Vater, are man-made (see p. 435). The symptoms to which such a stricture gives rise are (*a*) identical with those due to obturation of the common bile duct by a calculus; (*b*) an external biliary fistula that refuses to close, or discharges intermittently.

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Francis E. Stock, Contemporary. Professor of Surgery, Hong Kong.

To remedy the condition it is imperative to reconstruct the common bile duct. Various operative procedures are employed, according to the site of the stricture and the preference of the operator.

1. **Choledochoduodenostomy** (see p. 451).—When the stricture is lower in the bile duct than the superior border of the duodenum, this satisfactory operation is applicable.

2. **End-to-end Anastomosis.**—Painstaking efforts are made to identify, freshen, and approximate the two (unstrictured) ends of the duct. If necessary, in order to facilitate approximation, the duodenum is displaced medially and the duct is dissected from the pancreas. The ends of the duct are united and, when possible, a T-tube is introduced below the anastomosis (fig. 610); alternatively, the anastomosis is made over a plastic tube which, in a large percentage of cases, is eventually passed *per via naturalis*. It is advisable to have opaque material incorporated in the tube, so as to be able to identify it radiologically. Should it fail to pass after a year or eighteen months, the tube should be removed, because it becomes encrusted with bile pigments.

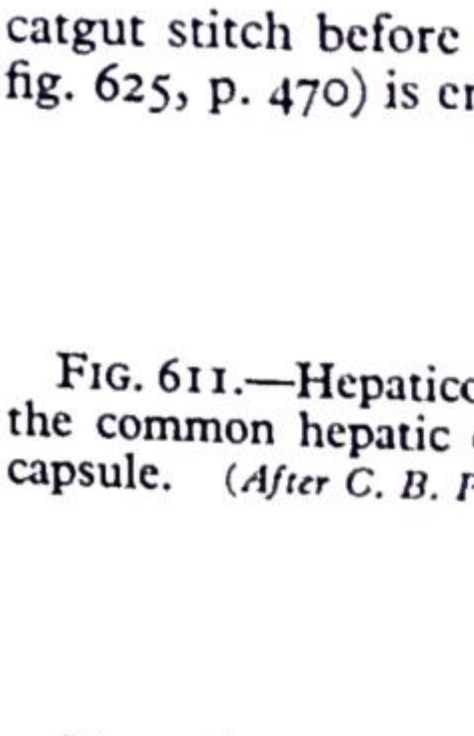

FIG. 610.—Reconstruction of the common bile duct by end-to-end anastomosis.

3. **Hepaticojejunostomy.**—When the loss of tissue is too great to bring the two ends of the severed duct together, a loop of jejunum can be bisected and the distal end brought through the transverse mesocolon to the hilum of the liver, where its mouth is sutured to the capsule of the liver around the open stump of the common hepatic duct (fig. 611). A rubber tube is placed in the duct and retained there by a catgut stitch before the anastomosis is completed. A Roux-en-Y anastomosis (see fig. 625, p. 470) is employed to restore the continuity of the jejunum.

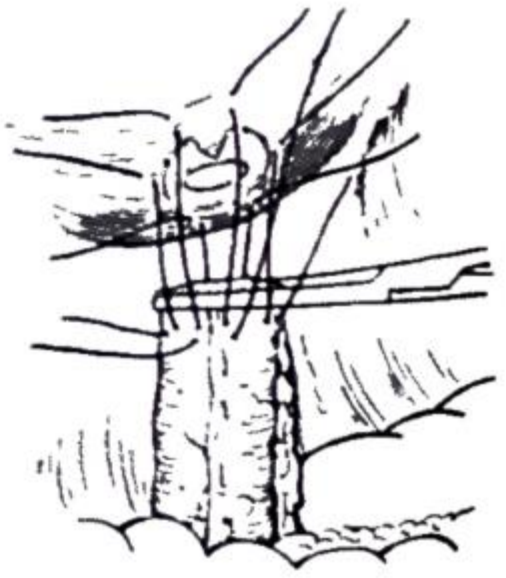

FIG. 611.—Hepaticojejunostomy. Note the method by which the common hepatic duct is held open by sutures to the liver capsule. (*After C. B. Puestow.*)

Hepaticodochojejunostomy (Longmire's Operation).—When scar tissue prevents identification of even the proximal ends of the hepatic ducts, the greater part of the left lobe of the liver is resected and the largest bile duct therein chosen for a similar anastomosis to that just described.

In a series at the Mayo Clinic the stricture was dealt with as follows:

Anastomosis between the hepatic duct and the common bile duct	18
Anastomosis between the hepatic duct or ducts and the duodenum	32
Anastomosis between the common bile duct and the duodenum	20
Anastomosis between the hepatic duct and the jejunum	15
Establishment of an external fistula was all that could be done in	6

BILIARY FISTULÆ

(*a*) **External Biliary Fistula.**—Especially in days gone by, gall-stones and bile were sometimes discharged with the pus after a superficial abscess of the abdominal wall had been incised, or had burst. Spontaneous extrusion of gall-stones through the umbilicus has been reported. At the present time nearly all external biliary fistulæ follow operations upon the biliary tract. When the neck of the gall-bladder or the cystic duct is occluded by a calculus (fig. 612, A), the discharge is mucus. Should a stone be occluding the common bile duct, and cholecystostomy is performed, bile will continue to be discharged externally (fig. 612, B). If cholecystectomy has

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William Polk Longmire, Contemporary. Professor of Surgery, University of California School of Medicine, Los Angeles.
The Mayo Clinic, Rochester, Minnesota, was founded by the Mayos—father and sons—in 1889.

been performed in these circumstances, again a biliary fistula (fig. 612, C) will result. By far the most common cause of a biliary fistula is injury to the common bile duct during cholecystectomy (fig. 612, D). A leakage after cholecystjejunostomy (fig. 612 E) or cholecystduodenostomy also will give rise to a fistula discharging bile.

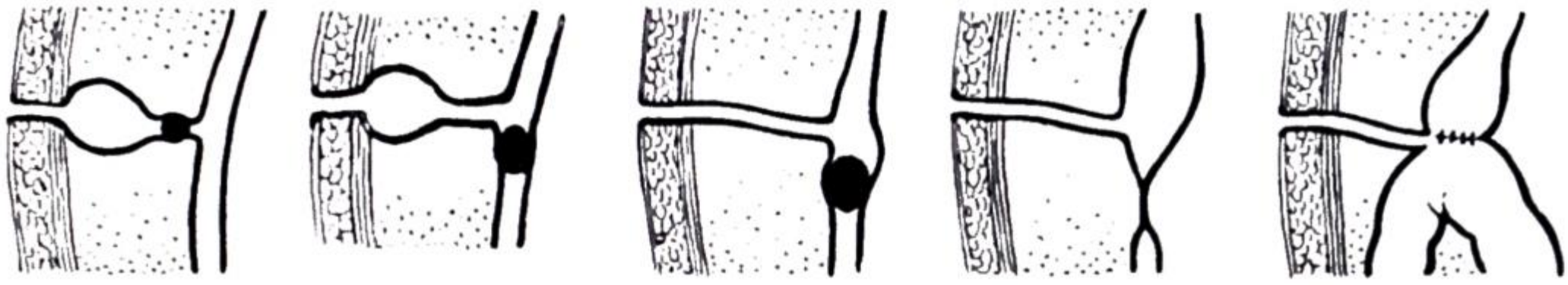

FIG. 612.—Underlying causes of an external fistula of the gall-bladder or the bile ducts. (*After J. J. Byrne.*)

Other causes are division of an aberrent right hepatic duct or a hepaticocystic duct (see p. 429).

After investigating, by retrograde cholangiography, the origin of the leak in the case of a fistula discharging bile, the continuity of the main bile duct must be restored (see p. 454) or, in the case of a mucous fistula, the gall-bladder or its stump must be removed.

(*b*) **Internal biliary fistula** is due to a stone ulcerating through the wall of the gall-bladder or the common duct into a hollow viscus, viz. :

1. *Into the stomach*—the patient may vomit the stones.

2. *Into the duodenum*—a gall-stone in the neck of the gall-bladder sometimes ulcerates into the duodenum; this is the commonest internal biliary fistula. Should the gall-stone be more than 1 inch (2·5 cm.) in diameter, it usually causes intestinal obstruction (see Chapter 26).

3. *Into the colon*—as a rule no untoward complications ensue, but a large stone occasionally becomes impacted in the pelvic colon or the rectum.

While an internal biliary fistula is generally regarded as a most fortunate happening, it is extremely doubtful if the patient should be sent on her way rejoicing without closing the fistula and removing the gall-bladder. Carcinoma of the gall-bladder frequently develops when a fistulous communication between it and a hollow viscus is permitted to remain for years.

BILIARY DYSKINESIA[1]

Hypertonicity of the sphincter of Oddi due to vagotomy occurs frequently in cases of duodenal ulcer; animal experiments show that the application of acid to the duodenal mucous membrane provokes spasm of the sphincter. Therefore it is always necessary to exclude peptic ulcer when spasm of Oddi's sphincter is demonstrated by intravenous cholangiography. Apart from this secondary phenomenon, cases of distension of the common bile duct and the gall-bladder without an organic lesion is due, presumably, to achalasia[2] of the sphincter of Oddi. The condition is comparatively rare, and morbid changes in the gall-bladder are absent. The symptoms to which it gives rise are mainly those of minor biliary colic. Cholecystography reveals a large gall-bladder, slow to empty, while intravenous cholangiography demonstrates obstruction at the duodenal papilla. *Antispasmodics relieve the symptoms.*

Too often the condition is first thought of after cholecystectomy, which greatly aggravates the condition. In the majority of cases the gall-bladder, except that it is large, shows no pathological change.

Treatment.—Prolonged medical treatment consisting of antispasmodic drugs and careful dieting sometimes renders the patient symptom-free. In resistant cases, sphincterotomy is indicated: cholecystjejunostomy also gives good results.

SYMPTOMS PERSISTING AFTER CHOLECYSTECTOMY

In 15 per cent. of all cases, cholecystectomy fails to relieve the symptoms for which the operation was performed. The heterogenous causes of this

[1] Biliary dyskinesia = functional spasm of the sphincter of Oddi.
[2] Achalasia = failure (of a sphincter) to relax.

disappointing sequel are now frequently lumped together under the all-embracing term ' the post-cholecystectomy syndrome, 'whereas, in point of fact, if this term is used it should be reserved for a condition due entirely to the loss of the gall-bladder. The list of causes is rather a long one:

1. The original diagnosis was in error (e.g. the symptoms were, and are, due to a diaphragmatic hiatus hernia).

2. Persistent symptoms are much more frequent after the gall-bladder has been removed for *early* chronic cholecystitis without gall-stones than for more obvious pathological lesions. Cholecystectomy in such circumstances creates a physiological disturbance (see Physiology, p. 426), and it seems probable that the loss of a functioning gall-bladder induces the sphincter of Oddi to go into spasm for long periods.

3. The cause of the original symptoms was biliary dyskinesia, which persists.

4. *Hypo*tonia, of the sphincter, which allows reflux along the ducts, is also a cause of failure of cholecystectomy. As a rule, hypotonia was present before the gall-bladder was removed.

5. In some patients fibrosis of the sphincter of Oddi is the cause of the symptoms, which differ from dyskinesia only in the fact that they are unrelieved by antispasmodics.

6. Should the original symptoms have been due to pancreatitis, removal of a comparatively normal gall-bladder often aggravates those symptoms.

7. A stone in the common bile duct escaped detection at the original operation.

8. When a comparatively long stump of a cystic duct has been left behind (fig. 613), as frequently happens following cholecystectomy where the cystic duct joins the common bile duct lower than usual (see fig. 557(*d*)), it is sufficient to act adversely on the pristine rhythm of the sphincter of Oddi, or certainly to harbour infection, if such was present.

FIG. 613.—Stump of cystic duct remaining after cholecystectomy.

9. Operative damage to the common bile duct occurred, resulting in stricture of that duct.

Treatment.—Cholangiography is extremely valuable in determining the cause which, if possible, is removed, e.g. a stump of the cystic duct must be excised; a stone or stones removed from the common bile duct. If *hyper*tonia, or fibrosis of the sphincter of Oddi, can be demonstrated, sphincterotomy is performed. In cases of *hypo*tonia of the sphincter P. Mallet-Guy carries out right splanchnicectomy. When no lesion is found, subdiaphragmatic vagotomy has led to an improvement (George Crile Jnr.).

NEOPLASMS OF THE GALL-BLADDER AND BILE DUCTS

Papillomata of the Gall-bladder.—True multiple villous papillomata are exceedingly uncommon, as opposed to polyposis of the gall-bladder, which is devoid of villous formation. As a rule there is no concomitant infection or gall-stones. The symptoms are those of chronic cholecystitis, and the radiological diagnosis is precisely that of polyposis (see p. 436). Papillomatosis of the gall-bladder is a pre-carcinomatous condition, and cholecystectomy is most certainly indicated.

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George Crile Jnr., Contemporary. Chief Surgeon, Cleveland Clinic, Cleveland, Ohio, U.S.A.

Carcinoma of the gall-bladder ranks sixth among gastro-intestinal carcinomata, the ratio being 3 females to 1 male, and the average age of the patient is about sixty. It is estimated that if the gall-bladder is not removed, 3 to 8 per cent. of all patients with gall-stones will develop carcinoma of the gall-bladder. On this account, perhaps cholecystectomy in elderly patients with gall-stones should be considered as necessary as extirpation of a solitary adenoma of the thyroid. In 25 per cent. of cases the development of acute cholecystitis is the first intimation that the patient has carcinoma of the gall-bladder; therefore, confronted with a patient over sixty years of age suffering from acute cholecystitis the 1:4 chance that she has a carcinoma of the gall-bladder should be recalled automatically. Apart from the development of acute cholecystitis, symptoms of carcinoma of the gall-bladder appear only when the disease has extended beyond the confines of the gall-bladder (fig. 614); consequently, by the time laparotomy is performed the growth is often inoperable. The signal lymph node of Lund, which lies at the junction of the cystic and common bile ducts is the first to become involved.

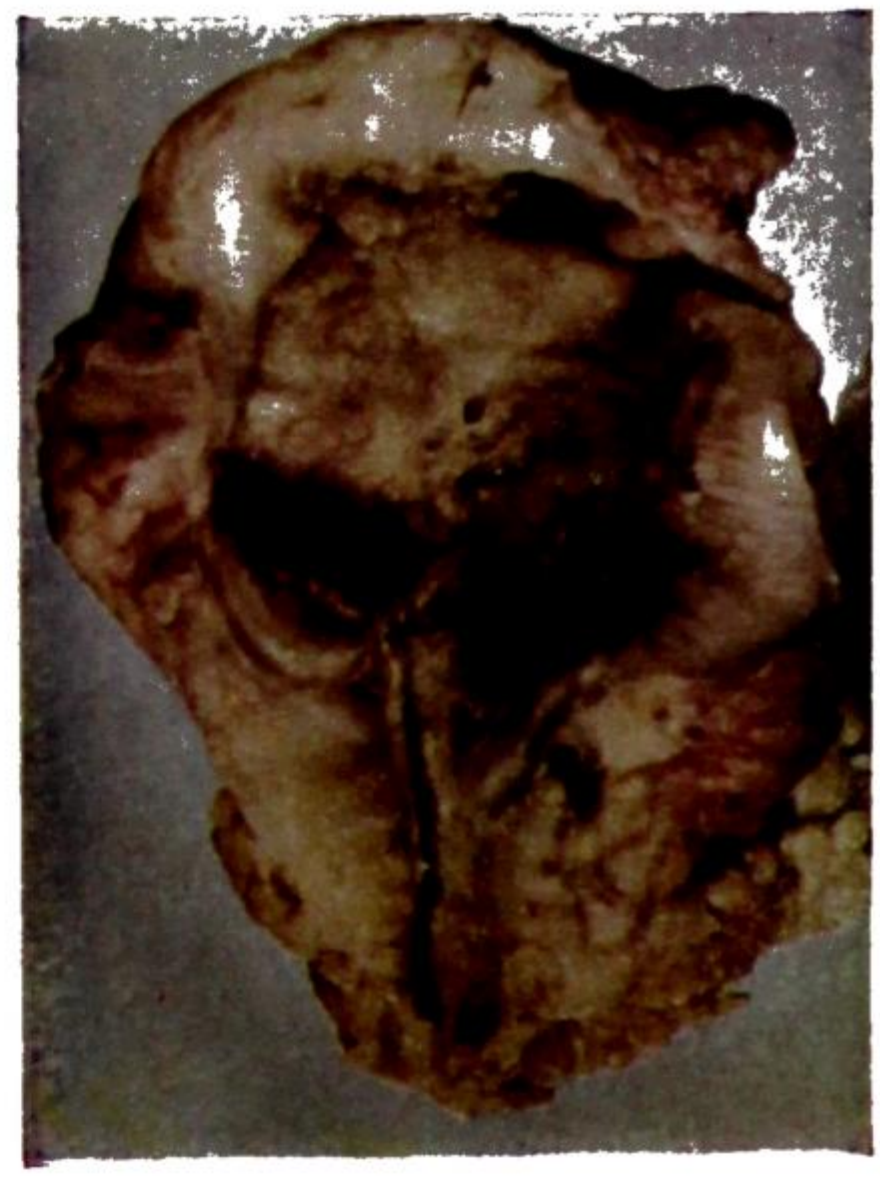

FIG. 614.—Carcinoma of the gall-bladder resected, together with a wedge of the liver which was involved. The patient was an artist, aged 72, who was alive and well three years later, and who drew some of the pictures for this book.

Treatment.—Except in very early cases, where cholecystectomy suffices, a radical operation should be attempted, if feasible. The gall-bladder is removed with a shell of healthy adjacent liver. The gastro-hepatic omentum is excised, and the lymphatics in juxtaposition to the portal vein and hepatic artery are dissected as far as the hilum of the liver.

Prognosis.—The prognosis of carcinoma of the gall-bladder is very bad, as attempts at resection are possible in less than 10 per cent. of cases.

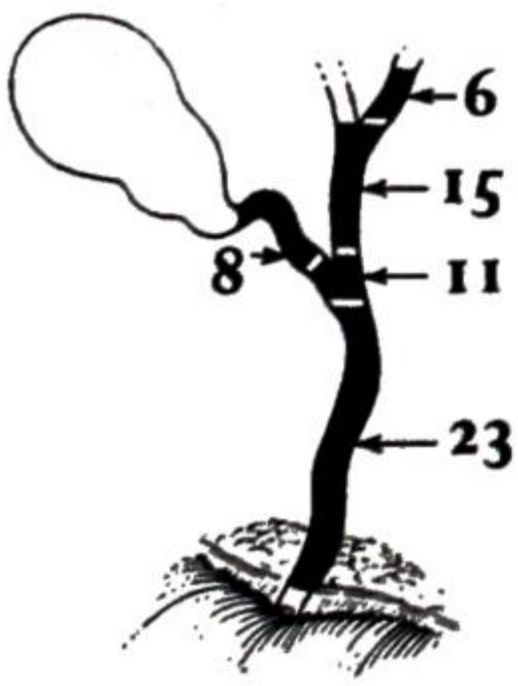

FIG. 615.—Site of carcinoma of the bile ducts in 63 cases. (*After K. Kuwayti et al.*)

Carcinoma of the Major Bile Ducts, excluding the Ampulla of Vater.—The patient is usually between sixty and seventy years of age, but there are numerous exceptions; the youngest patient with this condition reported in the literature was a twenty-three-year-old woman.

Pathology.—In 90 per cent. of cases the growth is nodular; in 10 per cent. it is papilliferous. The middle and lower segments of the common bile duct are involved most often (fig. 615). The primary lesion is often small, but because of its strategic location, it causes symptoms and death early in the course of its development. In 70 per cent. of cases metastases in the liver or the regional lymph nodes, or both, are found at laparotomy. In 40 per cent. of cases there are concomitant gall-stones. Invariably the liver is the seat of cholangiohepatitis: at necropsy it is usually intense green in colour.

Clinical Features.—The onset is sudden in 90 per cent.

Frederick Bates Lund, 1866–1950. Surgeon, Boston City Hospital, Boston, Mass.

of cases. The leading symptoms are obstructive jaundice and rapid loss of weight; in 50 per cent. of cases the jaundice is painless. As in other varieties of obstructive jaundice, pruritus is a distressing feature. Frequently anorexia is present. Usually the liver is enlarged, and the gall-bladder is palpable in 75 per cent. of cases, but sometimes, as a result of recanalisation consequent upon ulceration of the growth, the distended gall-bladder, initially palpable, becomes impalpable. As a rule the disease is rapidly fatal, death occurring on an average fourteen weeks after the onset of symptoms. Hepatic failure with coma is often a terminal event; sometimes this is preceded by cholangitis, with a high temperature and rigors.

Treatment.—Early laparotomy affords the patient a slender chance of prolonged survival. In about 15 per cent. of cases the growth is found to be resectable. In a number of the remainder a palliative procedure such as choledochoduodenostomy is possible, and symptomatic relief, particularly of the jaundice, is afforded thereby.

Carcinoma of the Ampulla of Vater (see p. 475).

CHAPTER XXIII

THE PANCREAS

HAMILTON BAILEY

Embryology.—The pancreas is developed from two buds of that part of the foregut which becomes the duodenum, one bud passing into the layers of the ventral and the other into the dorsal mesentery. The ventral bud arises from the base of the diverticulum that forms the liver (fig. 616, A), and forms the head and the uncinate

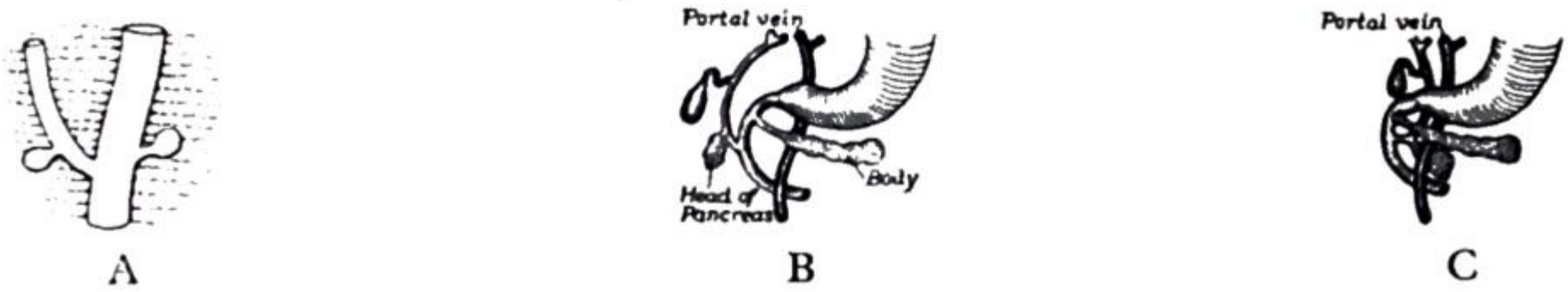

FIG. 616.—A, Primary pancreatic buds from the foregut ; B, Later stage before rotation ; C, After rotation, but before fusion : embyro fifth week.

process of the pancreas ; its duct becomes the duct of Wirsung. The remainder of the pancreas is formed from the dorsal bud, which arises nearer the pylorus than its fellow, its duct being the future duct of Santorini (fig. 616, B). With rotation of the stomach, the two pancreatic anlages close on the portal vein (or superior mesenteric vessels) like a book on a bookmark (fig. 616, C). Having come into contact, the two anlages fuse, and in so doing the duct of Wirsung becomes the main duct of both embryological components.

Surgical Anatomy.—Every student is familiar with the head, neck, body, and tail of the pancreas. It is necessary to recall that from the lower and left part of the head projects the uncinate process (fig. 617), upon which rest the superior mesenteric vessels. The posterior surface of the neck is grooved deeply by these vessels, and more superiorly by the portal vein. In spite of the intimate contact of the portal vein with the pancreas, unless it is bound down by inflammatory adhesions, in neoplastic infiltration it can be separated from the pancreas comparatively easily. In so far as its blood supply is concerned, the two embryological divisions of the pancreas retain their pristine independent nutrient vessels. Thus the head is supplied by the pancreatico-duodenal arteries while the body and the tail are nourished mainly by branches of the splenic artery. The distal part of the common bile duct lies in a deep sulcus between the second part of the duodenum and the head of the pancreas. To examine the pancreas properly by direct palpation, the organ must be mobilised by dividing the peritoneum (*a*) along the right margin of the duodenum and (*b*) along the lower border of the pancreas, the inferior pancreatico-duodenal artery being divided between ligatures (A. O. Whipple).

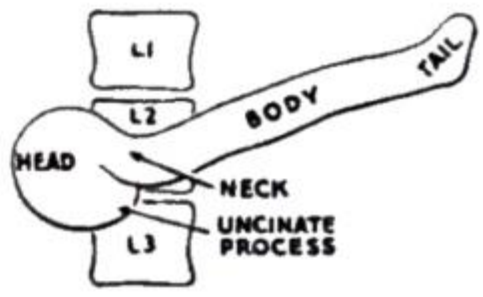

FIG. 617. — The various parts of the pancreas and its relationship to the lumbar vertebræ. (*After Rodney Smith.*)

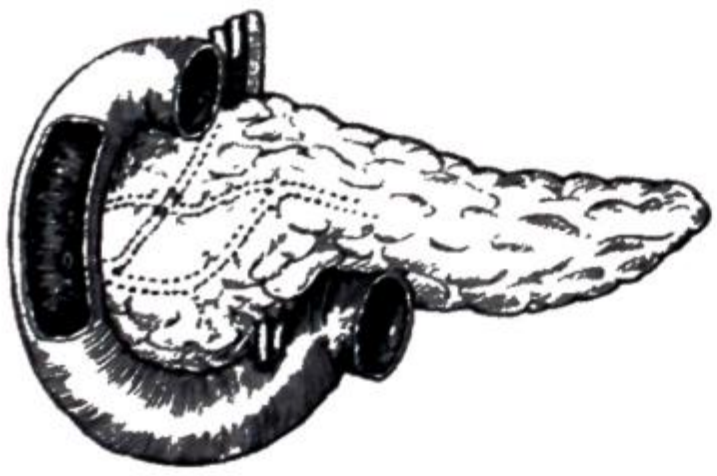

FIG. 618.—The most constant arrangement of the pancreatic ducts. (*After Rienhoff and Pickrell.*)

The ducts of the pancreas are subject to anatomical variations, but the most usual arrangement is shown in fig. 618 ; the duct of Santorini has a separate orifice about

Johann Wirsung, Prosector at Padua, was murdered when entering his house at night in 1643.
Giovanni Domenico Santorini, 1681–1737. Professor of Anatomy, Venice.
Allen Oldfather Whipple, Contemporary. Emeritus Professor of Surgery, Columbia University, New York City.