CHAPTER XI

THE INTESTINAL CANAL

THE physiological importance of the intestinal canal is evidenced by the fact that its length is between 25 and 30 feet, along the whole of which absorption may take place; yet the first feature of intestinal disorders which strikes the student is their inaccessibility to examination. Healthy individuals often show considerable variations in the size, length and position of the large intestine; on the other hand, dilatation and redundancy of the colon may be associated with disease. Micro-organisms or their toxins can make their way through the mucous membrane of the intestine into the lymph spaces beneath, and thence into the glands and the circulation, particularly when the mucous membrane is unhealthy, abraded, or ulcerated. The bacteriology and the chemistry of the intestinal contents are now assuming much importance and the examination of the stools is necessary in every complete investigation of a patient. X-ray examination after a barium meal has thrown much light on the nature of the intestinal movements. In the small intestine they are churning in character and result in a to-and-fro or pendulum action, with a peristaltic wave over a short length of intestine at intervals. The residue from the small intestine slowly fills the cæcum and ascending colon to the neighbourhood of the hepatic flexure. At intervals, usually after a meal (gastro-colic reflex), the mass of contents of the cæcum and ascending colon is rapidly passed through the remainder of the colon and evacuated, though usually a small residue remains in the sigmoid until the next mass movement takes place.

Another striking feature about diseases of the intestines is the disproportionate amount of prostration which accompanies them. When a patient is attacked by a slight but sudden diarrhœa or abdominal pain, the feeling of exhaustion, which in some cases may amount almost to collapse, is out of all proportion to the local mischief. This disproportionate degree of prostration or collapse is especially seen in early life, when "diarrhœa" is, mainly on this account, found to be one of the chief causes of death in children under two years of age. Again, among the acute specific fevers fatal collapse and prostration often occur in those in which the chief lesion is in the intestinal canal—in cholera, dysentery, and typhoid fever. This may be due in part to the large vascular bed in the abdominal cavity, and to the extensive surface through which toxins can be absorbed.

PART A. SYMPTOMATOLOGY

§ 301. The cardinal symptoms of intestinal disorder are ABDOMINAL PAIN, DIARRHŒA, CONSTIPATION, and INTESTINAL DISCOMFORT.

ABDOMINAL PAIN is frequently present, especially in the more acute conditions and may be due to many conditions within the abdominal cavity (see § 241). DIARRHŒA and CONSTIPATION are dealt with in Part C.

INTESTINAL DISCOMFORT may be due to colic, peritoneal pain or strangulation, or distension with wind, accompanied by borborygmi. It is a marked feature in COLOSPASM, which occurs in two conditions: (i.) reflex, as from adhesions after appendicitis or operation, with gallstones, and in the early stages of diverticulosis; (ii.) with worry, tense brain work and anxiety, associated with depression (visceral neurosis) (§ 252).

The GENERAL or REMOTE symptoms, such as loss of appetite from toxæmia and discomfort, are sometimes (especially in acute cases) of a very severe character, in view of the profound PROSTRATION which is associated with some intestinal disorders. PYREXIA is not usually a marked feature (see § 239). In the more chronic forms of intestinal disease EMACIATION is apt to ensue from malnutrition. The SALLOW SKIN of intestinal toxæmia is well known. Various NERVOUS DERANGEMENTS of a neurasthenic type are sometimes, as in gastric diseases, associated with disorders of the intestinal canal, consequent partly on mal-assimilation and intestinal toxæmia, and partly, no doubt, arising in a reflex manner. Less troublesome reflex symptoms—*e.g.*, vague pains, itching of the nose, or bad dreams—may be associated with intestinal parasites, constipation and other intestinal conditions.

PART B. PHYSICAL EXAMINATION

§ **302.** The physical investigation of the intestinal canal must be accomplished by an EXAMINATION OF THE ABDOMEN AND OF THE FÆCES. In all cases of abdominal disease a RECTAL EXAMINATION should be made. X-RAY and SIGMOIDOSCOPIC EXAMINATIONS are called for in some cases.

Examination of the Abdomen.—PALPATION and PERCUSSION will enable us to make out any general swelling or local tumour. The tenderness which often accompanies intestinal disorders may also be elicited. A loaded cæcum or descending colon, or the *scybala* present within the colon may be felt; these should not be mistaken for the nodules of cancer or other tumour. Their mobility is a very deceptive feature, and the occasional association of diarrhœa may delude us. Their disappearance after active purgation or a course of enemas, is the only certain method of diagnosis. The reader is referred to § 240 for further details as to examination of the abdomen.

§ **303.** An **Examination of the Stools** is always important, and sometimes absolutely necessary for the diagnosis of intestinal disorders. A great deal of information can also be thus obtained with regard to diseases of the other abdominal viscera. The fæces should be examined *first* as to their physical properties—size, consistence, colour, shape, odour, and reaction; *secondly*, for undigested food and other substances, such as

mucus, gall-stones, or parasites; *thirdly*, for the presence of blood or pus; *fourthly*, a microscopic examination. *Lastly*, culture of the stools is often of great value. One can rarely rely on a patient's statement, even as to the colour and appearance of the stools; they should be inspected by the physician. Early disease of the pancreas and intestinal canal can be detected by thorough investigation. For the technique of these examinations the student should consult pathological text-books.

It is preferable to see the fæces in bulk, the patient having used a night-stool. He should pass urine before going to stool. A large wide-mouthed glass jar, closed at the top by a stopper, is a convenient receptacle for their preservation. Nothing should be added to the motion until the doctor has examined it.

Physical Properties of the Stools.—(1) The *Consistency* is normally solid or semi-solid and the form roughly cylindrical. About four ounces are passed daily on an ordinary diet, but when fat is inadequately absorbed, as in sprue, the bulk is much increased. (*a*) When passed in hard, dry, roundish balls they are known as *scybala*, this condition being due to defective intake of fluid or its excessive absorption by a "greedy" colon. Scybala are generally coated with mucus and sometimes the irritation they cause sets up a spurious diarrhœa which may alternate with constipation. (*b*) *Pencil-like* stools may result from spasm of the anal sphincter, possibly associated with fissure, while *ribbon-like* stools may be produced by colospasm or stricture of the rectum resulting from cancer, syphilis, or gonorrhœa. (*c*) *Uniformly fluid* stools are common in lesions of the small intestine like typhoid, sprue and tuberculous or simple enteritis: in lesions of the large bowel the evacuations are generally more fæcal and slimy.

(2) The *Colour* of the fæces varies normally from light to dark brown, due to stercobilin, chlorophyll and other pigments; the depth of colour affords a fair index of the amount of bile passing into the intestinal canal. As diarrhœa progresses they become lighter in colour. *Pale-coloured stools* may be due to (*a*) obstruction to the entrance of bile into the intestine as in jaundice; (*b*) dilution of the stool as in cholera; (*c*) excess of unabsorbed fat; (*d*) a milk diet. Characteristic naked eye appearances are: (i.) *Clay-coloured* stools, found in obstructive jaundice, and pale, bulky, acid stools occur with steatorrhœa, due either to defective pancreatic secretion or defective fat absorption, as in tropical sprue, non-tropical sprue and cœliac disease; (ii.) *tarry stools*, dark or black coloured, due to blood entering the alimentary canal *high up*, as in duodenal ulcer. Black stools are also seen in patients taking iron, bismuth and charcoal; (iii.) "*red-currant jelly*" or "*strawberry ice*" stools are seen in intussusception. (iv.) *Streaks of blood* may be present with local lesions such as hæmorrhoids, or fresh blood in conditions such as ulcerative colitis, or cancer of the bowel and acute dysentery, when it is generally associated with mucus. *Muco-purulent* stools are also met with in the latter disease. Other characteristic stools are: (v.) the *green* stools of dyspeptic diarrhœa and enteritis of infancy and after calomel; (vi.) the odourless, colourless "rice-water"

stools of cholera, alkaline in reaction and containing flocculi of mucus and epithelium; (vii.) the frothy, acid, yellow stools resulting from excessive carbohydrate fermentation (the gaseous stool characteristic of sprue has a similar origin); (viii.) the soft, brown, offensive, alkaline stool of protein putrefaction; (ix.) the bilious "pea-soup" stools of typhoid.

(3) The *Odour* of the stools is due to skatol and indol, and is largely governed by the amount of meat ingested. A characteristic gangrenous smell is met with in severe ulceration, cancerous, dysenteric or syphilitic. An ammoniacal odour, if present, originates from contamination with urine.

(4) The *Reaction* of the fæces is normally amphoteric; with excess of protein it is alkaline, and of starchy foods and fats distinctly acid. The stool should be tested soon after being passed by moistening red or blue litmus paper with distilled water and rubbing a small portion of the stool on the paper; the colour reaction is seen on the other side. Steatorrhœa, due to pancreatic disease, sprue or cœliac disease, often yields acid-reacting fæces.

VARIOUS SUBSTANCES may be found:—

1. UNDIGESTED PARTICLES OF FOOD, if in excess, are indicative of imperfect digestion (gastric or intestinal), and, unless the food has been excessive, denote especially intestinal or pancreatic disease (see also p. 371 and § 256). In children this feature usually indicates over-feeding. Small, hard concretions, consisting of phosphates and other matter, are sometimes found. By noting those articles of diet (protein, vegetable, fruit, or carbohydrate) which pass for the most part undigested, the physician learns which the patient should reduce.

2. MUCUS in the fæces is often overlooked unless specially sought for. To discover it satisfactorily *water must be added* to the fæces, when any mucus present will be seen floating about like small pieces of jelly. The presence of mucus in small amount is of no consequence; it is usual in constipation. When in quantity, and intimately *mixed with the fæces*, it indicates catarrh of the *small* intestine. When in *isolated masses* it signifies the presence of catarrh of the large bowel. In membranous, or mucous, colitis, *long cylinders* of mucus are passed, sometimes without much fæces. These cylinders are generally swarming with organisms of both coliform and streptococcal types, which infest the colon.

3. BLOOD in the stools may appear either in streaks or in quantity, when from rectum or large bowel. If it comes from the stomach or small intestines, it will have undergone partial digestion and gives to the stools a tarry appearance (melæna). In either case it reddens the water in which the stool is placed, and gives the characteristic spectrum.[1] The *causes* are dealt with below (§ 314). *Occult* blood must be tested for in cases of suspected oozing from an ulcerated surface.

[1] Cases have been recorded where, after standing for some time, the fæces developed on the exposed surface a colour resembling blood, but no blood was detected by the spectroscope. It appears that in certain as yet unknown conditions some pigment is present in the fæces, which on exposure to the air becomes red like blood.

Occult blood test in fæces. The patient should be given a purge three days before the intended investigation, and take a hæmoglobin-free diet—milk and milk puddings, bread and butter, eggs, cheese, potatoes, fruit, tea, coffee or cocoa. Meat, meat extracts, liver or liver extracts, soups, poultry, game, fish, and green vegetables, must not be given. A charcoal biscuit given at the beginning of the diet will indicate when a fæcal specimen may be collected. During this period a soft tooth-brush should be used, lest bleeding from the gums occur. A series of tests over consecutive weeks is of importance where the result is positive. If each is positive the diagnosis is in favour of malignant ulceration ; if the positive result is intermittent it is in favour of simple ulceration.

To a piece of fæces of the size of a walnut, add 5 c.c. each of glacial acetic acid and water, preferably in a large "boiling-tube." Break up the fæces thoroughly with a glass rod, add an equal quantity of ether, and stir well. The ether extracts all the blood pigments, and if it does not rise spontaneously to the surface add water till it does so. Decant the ethereal extract and divide it into two parts. To the first apply the guaiacum test (§ 382). To the other add a quarter of

Fig. 74.—Microscopic Elements of Normal Fæces:

a, muscle fibres ; *b*, connective tissue ; *c*, epithelial cells ; *d*, white blood corpuscles ; *e*, spiral vessels of plants ; *f–h*, vegetable cells ; *i*, plant hairs ; *k*, triple phosphate crystals ; *l*, stone cells. Scattered among these elements are micro-organisms and debris.

its volume of conc. HCl, and shake. The acid hæmatin dissolves in the ether layer at the top, while the acid hæmatoporphyrin dissolves in the watery layer underneath. These pigments may be examined for by the spectroscope (Plate IV). If the bleeding is from the lower colon, the blood is not appreciably altered, and a positive guaiacum reaction and the presence of acid hæmatin is shown. If the blood is from a high level (*e.g.*, stomach or duodenum), only acid hæmatoporphyrin will be present ; but when the bleeding is considerable, some undigested acid hæmatin will be present in addition.

4. Pus always indicates *ulceration* of the rectum or colon, which may be due to ulcerative colitis, dysentery, cancer, tuberculosis or of syphilitic origin (§ 310). Pus is difficult to detect when diarrhœa is present. When in large quantity, pus indicates an abscess bursting into the bowel, such as a pelvic or ischio-rectal abscess.

5. Gall-stones may be found by mixing the stools with water, and passing the mixture through muslin or a fine sieve. Gall-stones sink in water when recently passed, though they float when dried. They are

often friable, and any suspicious particles should be examined under the microscope for cholesterin; see Fig. 86.

6. WORMS, see Tables XVIII and XIX, § 304 and § 316.

7. Various FLY LARVÆ are common; generally they are deposited after defæcation, but sometimes man swallows the eggs and larvæ develop later in the gut, giving rise to intestinal myiasis; gastro-intestinal and toxic symptoms may result. A dose of castor oil is generally effective; if not, thymol, santonin or turpentine may be subsequently administered.

Microscopic Examination of the fæces (Fig. 74) is often necessary to diagnose pathogenic protozoa and helminthic ova. As a routine a loopful of mucus or fæces should be rubbed up on slides, with (1) warm physiological saline (0·9 per cent.); (2) Lugol's iodine solution, and cover slips applied. Smears stained by Gram's method may also be made. NORMALLY, under the microscope, a few undigested starch granules, fat cells and partially digested muscle fibres may be observed, also crystals of fatty acids, oxalate of lime and other calcium salts. Hæmatoidin, phosphates, cholesterol, and Charcot-Leyden crystals are rare. Various bacteria, cocci and yeasts are found, as well as occasional epithelial cells.

1. *Abnormal* constituents to look for first are: ova, segments of tape-worms, flukes and nematodes (§ 304).

2. Amongst the *undigested food products* note any excess of muscle fibres, starch granules and fat. STARCH GRANULES stain blue with iodine, and if their presence is pathological the stools are usually acid and show signs of fermentation (gas bubbles) and the presence of yeasts. With starch indigestion excessive gas is formed in fermentation tubes in the incubator. Where digestion of PROTEIN is defective there is an excess of undigested muscle fibres showing cross striations and frayed ends: the stools are generally brown, offensive and alkaline. Neutral FAT droplets are soluble in ether and stain with Sudan III; fatty acids show up as sheaves of colourless, acicular crystals, while soaps form greasy amorphous masses which dissolve on heating. Normally the total fæcal fat does not exceed 25 per cent. of the dried fæces and the ratio of split to unsplit fat = 3 : 1. Excess of fat amounting to 30–80 per cent. of the dried fæces indicates: (1) defective bile secretion; (2) disease of the pancreas (§ 256) with defective external secretion; (3) intestinal disease interfering with absorption. In (1), as in obstructive jaundice, there is lack of bile salts with resulting defective absorption; the fat, however, is mainly split. In (2), as in chronic pancreatitis, fat may fail to be adequately split owing to the diminution of lipase in the pancreatic juice. In (3) splitting is adequate, the excess of fat resulting from malabsorption; this may occur in tropical sprue (§ 311), idiopathic steatorrhœa, cœliac disease of infants, gastro-colic and gastro-jejuno-colic fistula, and in lymphadenoma, lymphosarcoma and tuberculosis involving the mesenteric lymph glands. To ensure reliable results for fat analysis the patient must be on a normally balanced diet for at least three days previously, and without liquid paraffin.

3. Various *micro-organisms*, such as those of the typhoid, dysentery, tubercle and cholera group, may be found in the fresh stool; and in Shiga, Flexner and Sonne dysentery it is important to make cultures from recently passed mucus at as early a stage of the disease as possible. The bacterial flora, including *B. coli*, streptococci, anærobes, etc., may be modified in certain conditions by diet and purgation as well as by the administration of *B. acidophilus* and antiseptic drugs.

4. *Intestinal sand* consists of fine granules of calcium salts and silica formed around an organic nucleus, or of granules from pears or other fruit. 5. *Charcot-Leyden* crystals are common in amœbiasis, but also occur in ankylostomiasis and mucous colitis.

§ **304. Certain Intestinal Parasites** and their ova, described in Tables XVIII and XIX, may be found in the fæces (Figs. 75 to 81). In addition, the operculated eggs of certain intestinal and liver flukes are met with in tropical countries. Segments of tape-worms often appear in the stools: held between two glass slides and examined with a

Table XVIII.—The Principal Pathogenic Helminths of the Intestine.

For treatment, refer to § 316.

Specific Name.	Chief Characteristics of Worms and Habitat.	Clinical Features.	Ova or Embryo; Chief Characteristics, and Where found.	Animal Hosts, etc.
Cestodes.				
Tænia saginata. (Tapeworm in man.) Fig. 75.	14 to 24 ft. long. Head, 4 suckers, no hooklets. Segments, over 1000, show central stem uterus with 15 to 30 lateral *dichotomous* branches. Fastens itself to mucosa of small intestine in man.	Reflex irritation, digestive or nervous disorders. Segments passed per rectum.	Recognised by segments containing ova discharged in fæces. Ova similar to those of *T. solium*. Cystic stage in beef ($35\mu \times 25\mu$).	Cattle the intermediate hosts. Found in Great Britain. Widespread geographical distribution. Man infected by eating underdone beef.
Tænia solium. (Tapeworm in man.) Fig. 76. (Cysticercosis also in man.)	About 7 to 10 ft. long. Head, 4 suckers, and double row of 26 hooklets. Segments, about 850, show central stem uterus with 7 to 10 lateral *ramifying* branches. Fastens to mucous membrane of small intestine in man.	Ditto—from adult worm. Epilepsy from embryos (Cysticercosis).	Recognised by segments containing ova discharged per rectum. A six-hooked embryo inside ovum which, eaten by pig, bores its way into the flesh. Ova spherical; 35μ in diameter.	Pig the intermediate host. Adult worm takes 3 months to develop in man, who becomes infested by eating "measly pork." Man may also act as intermediate host, ingestion of eggs leading to Cysticercosis.
Diphyllobothrium latum or **Dibothriocephalus latus.** Fig. 77.	16 to 25 ft. long. Head club-shaped, with long lateral grooves. No hooklets or suckers. About 3000 segments; uterus, rosette-shaped. Found in intestinal canal of man.	Occasionally anæmia of pernicious type. Intestinal disorder in children.	Segments containing ova discharged per rectum. Sometimes ova discharged alone; brown shelled with a lid at one end, broadly oval ($70\mu \times 45\mu$).	Ova hatch on reaching water. Swallowed by a *Cyclops* which is eaten by a fish (intermediary host). Chiefly found in Switzerland and other parts of Central Europe, also in U.S.A.
Echinococcus granulosus or **Tænia echinococcus.** (Hydatid cyst in man; tapeworm of dog). Fig. 85.	$\frac{1}{3}$ in. to $\frac{1}{2}$ in. long. Head pointed, with 4 suckers; double row of hooklets. Has 4 segments, the 4th longer than all others. Found in intestinal canal of dogs, wolves or jackals.	Hydatid cysts form in liver, or other organs in man, sheep, cattle and pigs.	Ova found in fæces of dog or wolf. Embryo becomes encysted in various organs, especially liver and lungs.	Man, sheep and cattle are intermediary hosts: Man becomes infected from contaminated food or water, or from contact with dogs to whose coats and mouths ova may be adherent. Found chiefly in Australia, New Zealand, Argentine and Iceland; occasional cases occur in Great Britain and elsewhere.

NEMATODES.				
Enterobius vermicularis or **Oxyuris vermicularis.** (Threadworm.) Fig. 78.	F. = 8–13 mm.; M. = 2–5 mm. in length. Found in large intestine, chiefly the rectum.	Reflex irritation. Worms tend to migrate at night, and cause itching of anus and genitals.	Worms emerge from anus at night to lay eggs in sticky masses on peri-anal skin. Fingers contaminated by scratching cause reinfection. Ova ($50\mu \times 20\mu$) thin-shelled, planoconvex and contain coiled embryo.	Often trouble children. Found in all countries.
Strongyloides stercoralis.	Only female worms found in intestine—1 in. long. Males develop outside body from rhabditiform larvæ.	Early—dermatitis and lung symptoms. Later—sometimes diarrhœa and urticaria.	Thin-shelled, oval eggs ($60\mu \times 30\mu$) which hatch out free rhabditiform larvæ passed in fæces.	Sexual cycle outside human body. The rhabditiform larvæ differ from ancylostome larvæ in being free and having a short presœsophageal mouth.
Ancylostoma duodenale. (Old world hookworm.) Fig. 81.	M. = 8–10 mm.; F. = 12–18 mm.: buccal cavity large and contains two pairs of ventral teeth; attached to mucosa of jejunum.	Asthenia: mental lethargy; hypochromic anæmia: eosinophilia: œdema: serous effusions.	Ova = $60\mu \times 40\mu$, thin, transparent shell showing 2–8 stages of segmentation. Ova found in fæçes, rhabditiform larvæ rarely.	Man is infected by filariform larvæ in the soil penetrating human skin. Widespread geographical distribution.
Necator americanus.	M. = 7–9 mm.; F. = 9–12 mm. The buccal cavity is small with a less effective biting apparatus.	Ditto.	Ova = $70\mu \times 36\mu$ slightly narrower and longer than **A. duodenale.** Found in fæces.	Ditto.
Ascaris lumbricoides. (Roundworm.) Fig. 79.	M. = 6 in.; F. = 12 in. Inhabits the small intestine of man.	Early—urticaria and ascaris larval pneumonia. Later—produce symptoms by toxic, reflex and mechanical means.	Ova are yellow, elliptical with thick outer shell showing excrescences. ($60\mu \times 45\mu$.)	World-wide distribution; very common in children, also adults. May wander widely in human host.
Trichuris trichiura or **Tricocephalus dispar.** (Whipworm.) Fig. 80.	Length = 1¼ in. found in cæcum; anterior portion thread-like.	Generally nil. May be reflex disturbances, urticaria or verminous appendicitis.	Ova brown, barrel-shaped with terminal knobs. ($50\mu \times 23\mu$.)	Cosmopolitan distribution. Man is infected by swallowing embryonated eggs.

Table XIX.—Somatic Helminthic Infestations of Man.

Specific Name.	Chief Characteristics of Worm and Habitat.	Clinical Features.	Ova or Embryo; Chief Characteristics, and Where found.	Life Cycle and Geographical Distribution.
Trematodes.				
Schistosoma hæmatobium (Vesical Schistosomiasis.) Fig. 107. Syn., Vesical bilharziasis.	M. = 10–15 mm.; F. = 20 mm., female lives mainly in gynæcophoric canal of male in portal veins and pelvic venous plexuses.	Terminal hæmaturia, frequency, perineal, penile and loin pain. Eosinophilia.	Terminal spined ova (120–160μ × 40–60μ) containing a ciliated miracidium passed in urine.	Africa, Syria, Arabia and Mesopotamia. Intermediary host mainly *Bulinus* species of snail: man infected by cercariæ penetrating skin while bathing.
Schistosoma mansoni (Intestinal Schistosomiasis.) Syn., Intestinal bilharziasis.	M. = 12–20 mm.; F. = 12–16 mm. Worms inhabit portal and mesenteric veins.	Schistosomal dysentery, papillomatosis of colon common. Sometimes cirrhosis and splenomegaly. Eosinophilia, anæmia.	Lateral spined ova (140–165μ × 60–70μ) containing a ciliated miracidium passed in fæces.	Africa, South America. Intermediary host mainly *Planorbis* species of snail. Infection ditto.
Schistosoma japonicum (Japanese Schistosomiasis.)	M. = 12–20 mm.; F. = 18–26 mm. Worms inhabit portal and mesenteric veins.	Ditto.	Ova (70 × 100μ–50 × 65μ). Have a lateral knob; passed in fæces.	China and Japan. Intermediary host mainly Katayama and *Oncomelania* species of snail. Infection ditto.
Clonorchis sinensis.[1] (Liver fluke.)	Adult is a spatulate fluke (10–20 mm. × 2–5 mm.). Found in the bile ducts of man.	Anorexia, epigastric pain, diarrhœa, enlarged liver and ascites.	Oval, brownish ova (30μ × 15μ) with an operculum, found in fæces.	Common in Japan and China. Life cycle is through a snail and fish which has to be eaten by man.
Paragonimus westermani. (**Distoma ringeri**) (Lung fluke.)	Adult flukes (7·5 mm.–12·0 mm. × 4–6 mm.). Live in bronchi, where they produce cystic swellings and dilatation.	Cough, hæmoptysis; physical signs like bronchiectasis and broncho-pneumonia. Causes "endemic hæmoptysis" of Japan.	Broad, oval operculated ova (100μ × 60μ). Appear in brown, rusty sputum; sometimes in fæces also.	Ditto.

[1] **Fasciola hepatica,** the common liver fluke of sheep and other mammals, rarely affects man.

T. solium and *E. granulosus* also produce somatic infestations in man (vide Table XVIII p. 372).

NEMATODES.				
Wuchereria bancrofti.	Worms resemble fine cat-gut. M = 30–40 mm. F. = 75–100 mm. Inhabit lymphatics and discharge embryos, appearing in the blood stream at night.	Transient painful red swellings in arms, legs, scrotum, etc., with eosinophilia and occasionally mild fever; later lymphangitis, elephantiasis, chyluria, etc.	Embryos appear in blood at night: possess a loose sheath (230–320μ × 7·5–10μ). In Pacific filaria is non-periodic.	Mosquito (Anopheline, culicine or aëdine) is the intermediary host. In Pacific vector is *Aedes variegatus*; bites in day time.
Wuchereria malayi. (Syn., Filaria malayi.)	Adult worm resembles W. bancrofti. Embryos are nocturnal.	Similar but less severe.	Sheathed embryos (200–250μ × 5–6μ) in blood.	Mosquito (various species of *Mansonioïdes*) is the intermediary host.
Loa loa or **Filaria loa.**	Worms inhabit subcutaneous and retroperitoneal tissues. M= 30–34 mm. F. = 50–70 mm.	Urticaria, painless calabar swellings; worms cross conjunctiva producing conjunctivitis. Marked eosinophilia.	Sheathed embryos (250–300μ × 6–8·5μ) appear in blood from 9 a.m. to 9 p.m., *i.e.*, diurnal periodicity.	Transmitted by mangrove fly (*Chrysops* species) feeding in day time. Occurs in West Africa.
Onchocerca volvulus.[4] (Onchocerciasis.)	Adult male and female worms occur encapsulated in fibrous tissue nodules in subcutaneous tissues.	Subcutaneous nodules. Dermal lesions. Ocular lesions: blindness. Eosinophilia.	Sheathless embryos occur in local lesions and in skin; not found in peripheral blood.	Transmitted by black fly—(*Simulium damnosum*)—in West Africa and Congo Basin.
Dracunculus medinensis. (Guinea-worm.)	Adult female measures 40–80 cm. Inhabits the subcutaneous tissues producing a local ulcer through which embryos are discharged.	Urticaria; anaphylactoid features; local vesicle and ulcer: local abscess, cellulitis, etc.	Larvæ are reflexly ejaculated by female worm; they are filiform, actively motile measuring (500–750μ × 15–25μ).	Larvæ escape into water and undergo development in a water-flea, *Cyclops*; man becomes infected by drinking water containing it. Occurs in India, Africa, Arabia, etc.
Trichinella spiralis. (Syn., Trichina spiralis.)	Adults inhabit the intestine and liberate embryos which migrate to muscles. M. = 1·4–1·6 mm. F. = 3–4 mm.	Gastric and intestinal symptoms followed by fever, eosinophilia and myositis of affected muscles.	Embryos (100μ × 6μ) may be found in laked blood, or identified in piece of muscle removed at biopsy.	Rats act as reservoir hosts. Infection acquired by eating underdone pork in which larvæ are encysted.

[4] *Onchocerca cæcutiens* which causes onchocerciasis in Guatemala and Mexico is transmitted by Simuliid coffee-flies and is probably the same parasite.

hand lens they are identified by the number of lateral branches each side of the central uterine stem. *T. saginata* has fifteen or more, but *T. solium* never exceeds twelve. The cysticercus stage of the latter parasite may involve the muscles and brain : epilepsy may result (§ 723). The segments of *Diphyllobothrium latum*, the tapeworm of Central Europe, possess a rosette-shaped uterus. Eggs of the common round-worm (*Ascaris lumbricoides*) and the thread-worm (*Enterobius vermicularis*, *Oxyuris vermicularis*) are often found in Europe, while ancylostomes, including *Ancylostoma duodenale* and *Necator americanus*, the whip-worm, the thread-worm and another nematode, *Strongyloides stercoralis*, are frequent in patients from abroad. For symptoms and treatment, see § 316. The lateral-spined eggs of *Schistosoma mansoni* occur in the

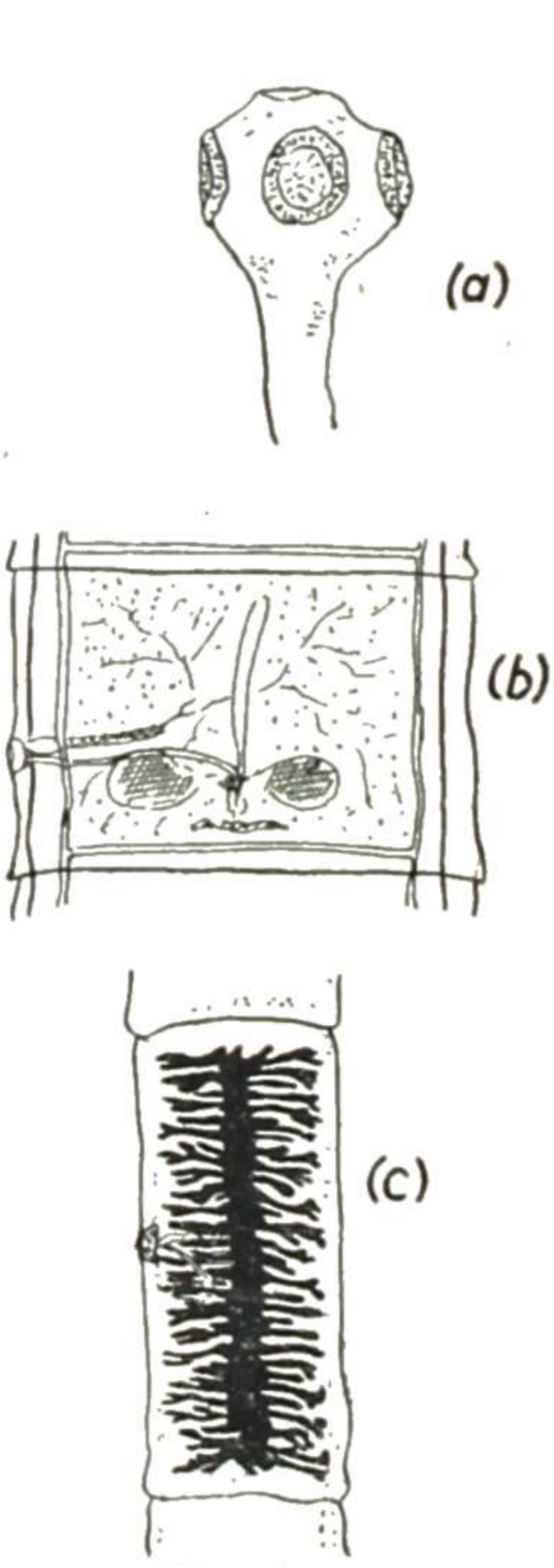

FIG. 75.

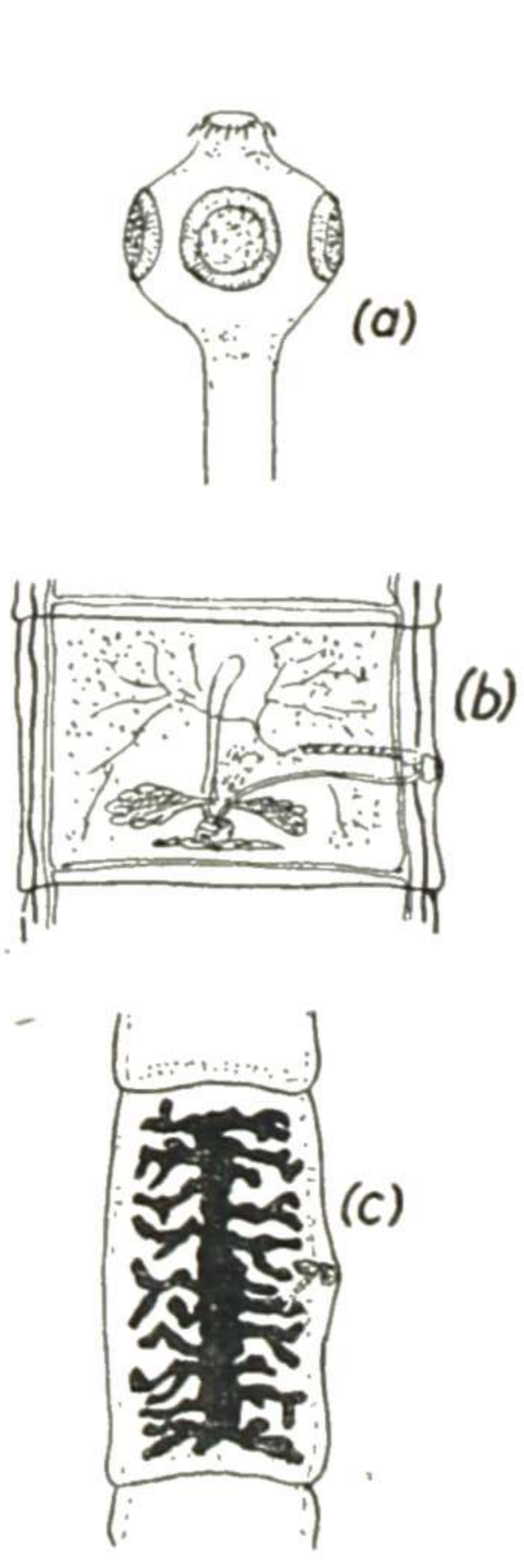

FIG. 76.

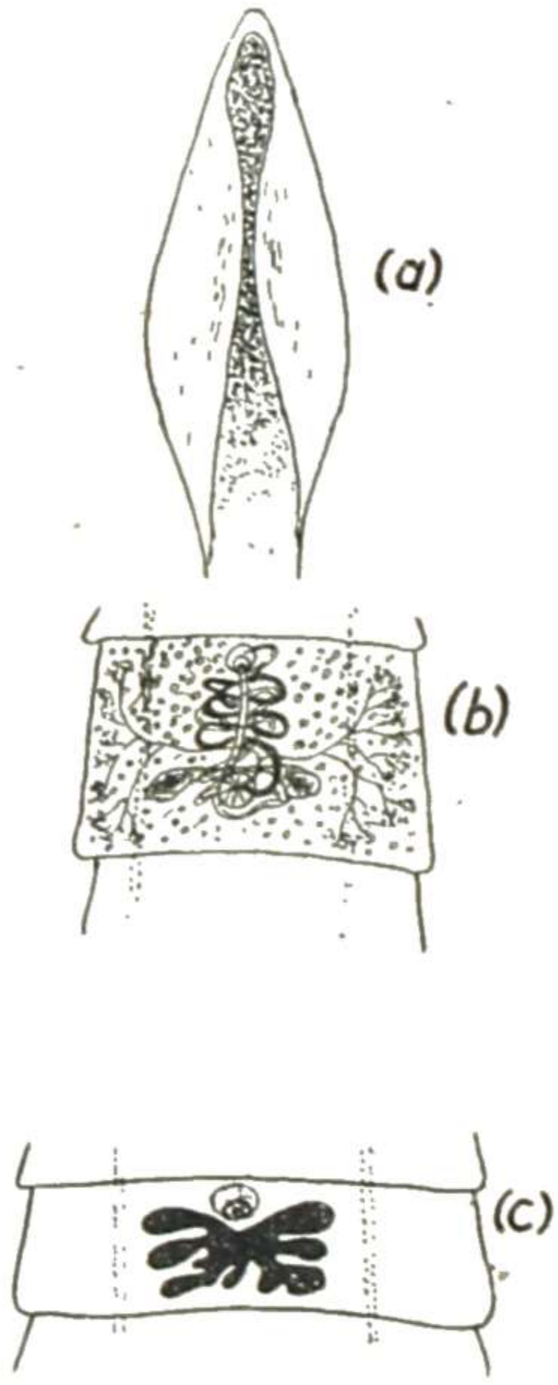

FIG. 77.

FIG. 75.—*Tænia saginata*. (*a*) Head × 10 ; (*b*) maturing segment showing reproductive system × 3 ; (*c*) segment, showing central stem uterus with 15 *to* 30 *lateral dichotomous* branches, × 3.

FIG. 76.—*Tænia solium*. (*a*) Head × 10 ; (*b*) maturing segment showing reproductive system × 3 ; (*c*) segment, showing central uterus with 7 *to* 10 *lateral ramifying* branches, × 3.

FIG. 77.—*Diphyllobothrium latum*. (*a*) Head × 10 ; (*b*) maturing segment showing reproductive system × 3 ; (*c*) segment, showing *rosette-shaped* uterus, × 3.

mucous coating of the stool, and the terminal-spined eggs of *S. hæmatobium* which produce hæmaturia are found in urine, but only occasionally in fæces.

Entamœba histolytica, the cause of amœbic dysentery and tropical liver abscess, must be distinguished from other amœbæ, e.g., *E. coli*, *Iodamœba bütschlii*, *Endolimax nana*, and *Dientamœba fragilis*. Mucus from a warm, freshly-passed stool is mixed with saline and examined microscopically ; diagnosis of *E. histolytica* depends on the presence of an actively motile amœba containing ingested red blood corpuscles. Their cysts occur in the solid fæces and are best demonstrated by mounting in a weak solution of iodine ; spherical and less than 14 μ in diameter, they characteristically have a diffuse glycogen mass, chromidial bodies, and one to four nuclei with central karyosomes.

Various flagellates, including *Giardia intestinalis* (Lamblia), are not uncommon, but unless in large numbers their pathogenicity is doubtful. A ciliate, *Balantidium coli*, gives rise to ulceration of the large bowel which may end fatally.

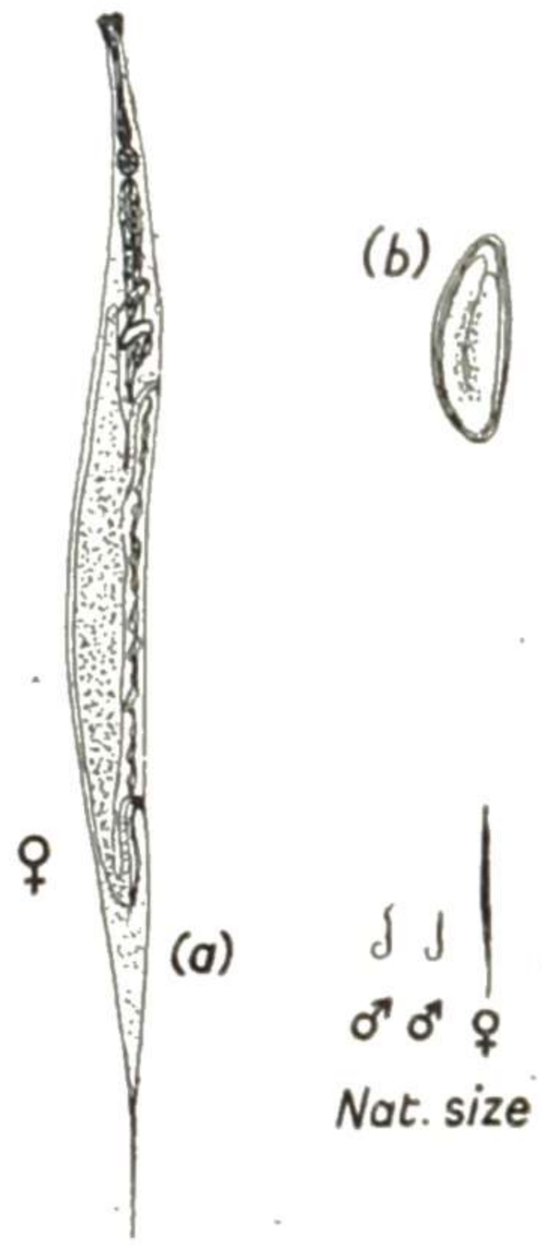

FIG. 78.

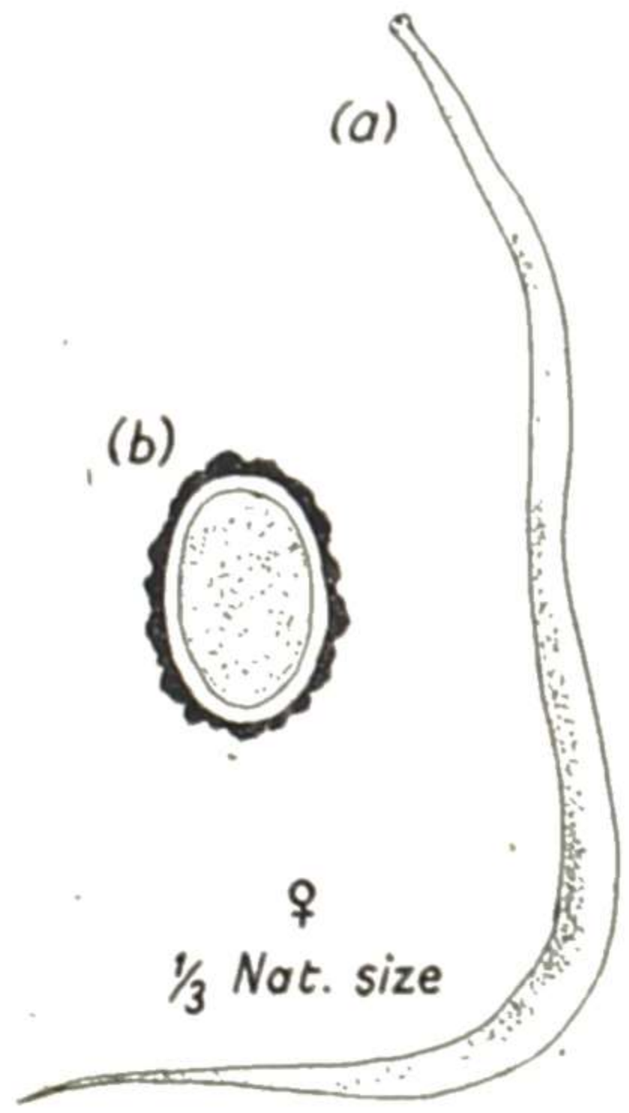

FIG. 79.

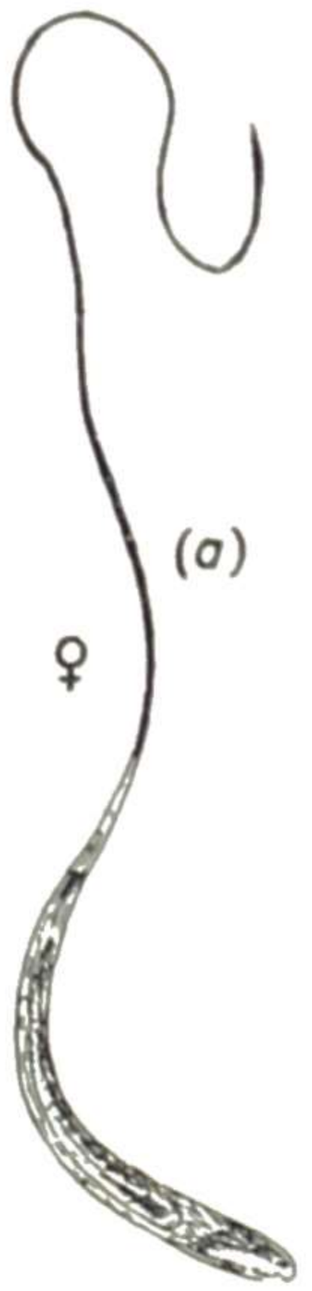

FIG. 80.

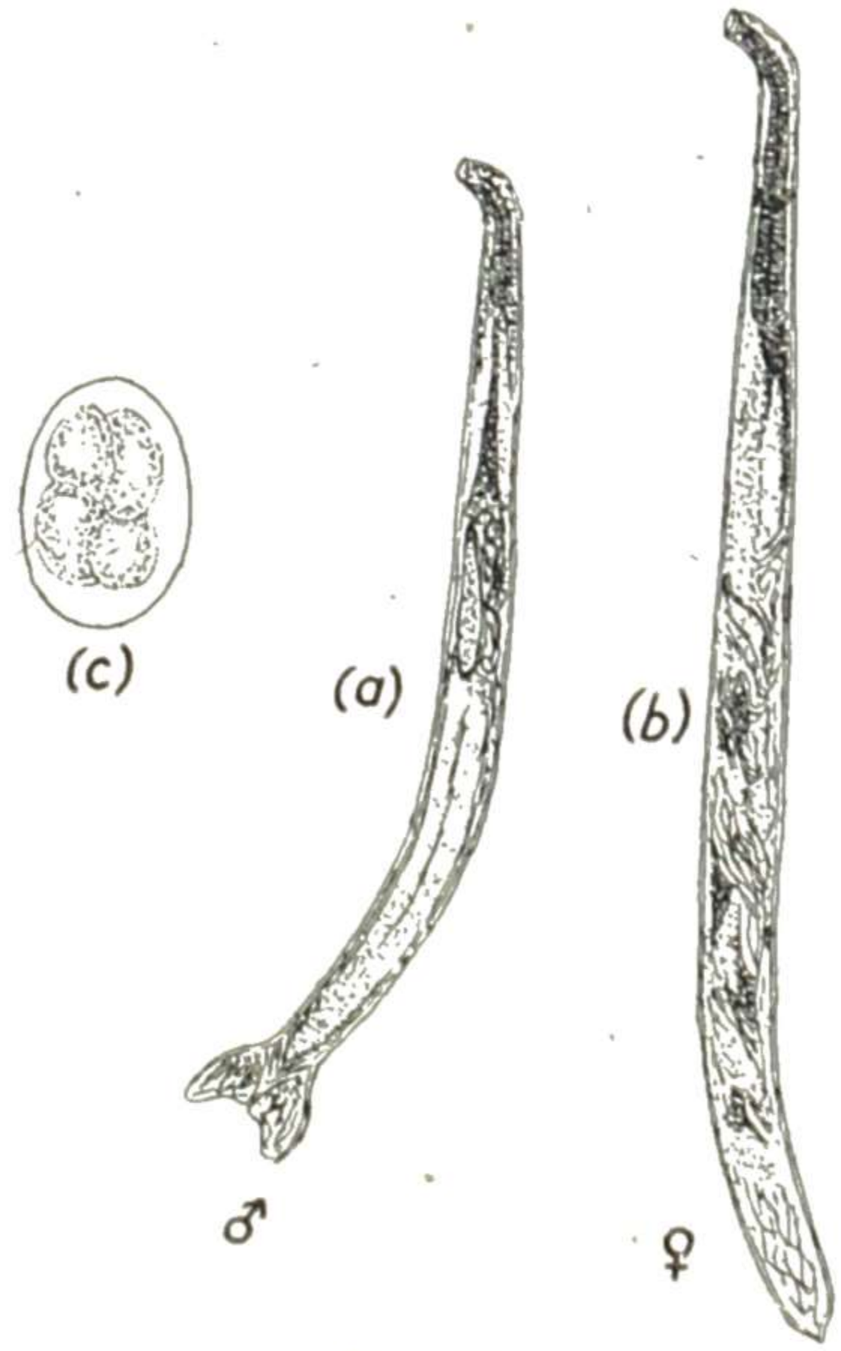

FIG. 81.

FIG. 78.—*Oxyuris vermicularis.*

(*a*) Female × 8, also two male worms and female, natural size;
(*b*) egg × 30.

FIG. 79.—*Ascaris lumbricoides* (Round Worm).

(*a*) Female, one-third natural size;
(*b*) egg × 200.

FIG. 80.—*Trichuris trichiura* (Tricocephalus dispar, "Whip-worm").

(*a*) Female × 30;
(*b*) egg × 170.

FIG. 81.—*Ancylostomum duodenale.*

(*a*) Male × 5;
(*b*) female × 5;
(*c*) egg × 170.

PART C. DISEASES OF THE INTESTINAL CANAL, THEIR DIAGNOSIS, PROGNOSIS, AND TREATMENT

§ 305. Routine Procedure, and Classification.—Ascertain first that the patient's LEADING SYMPTOM is referable to the intestinal canal; and secondly, by inquiries into the HISTORY of the illness, whether it came on *acutely* and suddenly, or gradually in a *chronic* manner. In the History, the following points should be investigated: (i.) duration of the present symptoms, previous diseases and operations; (ii.) residence in

tropical climates; (iii.) fever; (iv.) pain and uncomfortable sensations in the intestine; (v.) tenesmus; (vi.) defæcation; frequency or incontinence of fæces, presence of blood or other abnormalities; (vii.) the appetite; any alteration in weight; (viii.) the presence of gastric symptoms may aid the diagnosis. Intestinal Colic is dealt with under abdominal pain without collapse, in § 246. Next proceed to the PHYSICAL EXAMINATION of the abdomen after the manner set forth in Chapter IX (§ 240). If, in the course of these inquiries, definite disease is suspected in any particular organ, reference should afterwards be made to the appropriate chapter.

A. **Diarrhœa** is the leading symptom:

If *acute*, or attended by choleraic or dysenteric symptoms	turn to	§§ 307–309
If *chronic*	„	§ 310
B. There is **Tenesmus** without diarrhœa	„	§ 312
or Rectal Spasm	„	§ 313
C. **Blood** or some other **alteration in the stools** is the leading feature	„	§§ 314–316
D. **Constipation** is the leading symptom	„	§ 317
E. **Intestinal Flatulence** is the principal feature	„	§ 318
F. **Stoppage in the Bowels** is complete	„	§ 319
G. **Pain in the Left Iliac Fossa** is associated with **Fever** and **Constipation**	„	§ 321

TABLE XX.—CAUSES OF DIARRHŒA.

	Acute.	Chronic.
COMMON.	I. Unsuitable or infected food. II. Water. III. Intestinal parasites. IV. Infantile diarrhœa.	I. Acute causes becoming chronic. II. Local conditions about anus. III. Ulceration, other than ulcerative colitis. IV. Chronic or mucous colitis. V. Ulcerative colitis. VI. Portal obstruction or congestion. VII. Dysenteric diarrhœa. VIII. Nervous diarrhœa.
RARE.	V. Typhoid and toxic blood conditions. VI. "Chill." VII. Acute ulcerative colitis. VIII. Some causes of chronic diarrhœa. IX. Dysentery. X. Cholera.	IX. Amyloid disease. X. Senile diarrhœa. XI. Mineral poisons (*e.g.*, arsenic). XII. Gastrogenous diarrhœa. XIII. Pancreatic disease. XIV. Gastro-colic fistula and gastro-enterostomy. XV. Rare constitutional causes. XVI. Sprue.

§ 306. **Diarrhœa** is the frequent occurrence of loose or liquid motions; it is the *watery consistence* of the stools which is the chief characteristic. A frequent call to stool may arise from some local irritation (see Tenesmus), without any alteration in the consistency or form of the stool. This source of fallacy should be guarded against by careful inquiry.

Examination of the fæces (§ 303) may show the situation of the disease.

Thus, for instance, when the stools are coloured with bile, and contain undigested food, and *small pieces of mucus intimately mixed* with the fæces, catarrh of the small intestine may be suspected. When mucus or "slime" occurs in *larger masses*, in "strings" or "casts," it points to disease of the large intestine.

§ 307. In **Acute Diarrhœa** there is usually a good deal of pain and tenesmus (straining at stool); the tongue is usually furred, there is thirst, and may be vomiting. Profuse vomiting and prostration indicate some violent irritant, or serious organic lesion of the bowel or peritoneum. In profuse diarrhœa the temperature is usually subnormal, and the urine diminished. Scybala retained in the intestines may give rise to attacks of diarrhœa alternating with constipation. The possibility of a "controlling appendix" is also to be considered.

Causes.—I. The **food** taken, and the vessels in which it has been contained and cooked, should be the first questions in all cases of acute diarrhœa coming on suddenly in a healthy person. Collapse and many of the symptoms of cholera can be produced by food cooked in a new copper vessel. One of the irritant poisons may have been introduced into the food accidentally or designedly. This should be borne in mind; and in cases of sudden and unexplained diarrhœa the physician should patiently consider every article taken at every meal during the preceding twenty-four hours, *e.g.*, unripe, over-ripe or decomposing fruit, too much raw vegetable food, meat which has been long in store and has undergone putrefaction. In this variety of acute diarrhœa there may be a considerable degree of intestinal colic (§ 246). The most common infections causing diarrhœa and colic include the bacillary dysenteries (Flexner, Shiga or Sonne); also the *B. enteritidis* (Gærtner) and *B. ærtrycke*, which in hot weather may occur in meat, fish, crabs, mussels, cheese and other milk products. *Staphylococcus aureus* and its toxins can cause acute outbreaks of gastro-enteritis even as an epidemic. The first or diarrhœic stage of *trichinosis* should be considered in pork-eating countries; in cases of acute diarrhœa in which trichinosis is suspected, the worm should be sought in the fæces. The diarrhœa which precedes the intestinal obstruction caused by *intussusception* in children frequently follows a heavy meal of indigestible articles; and diarrhœa is itself a cause of intussusception.

II. The quality of the **water** is often responsible for diarrhœa, acute or chronic. This is frequently the case in malarial districts in the summer and autumn, especially when the temperature is high. Soft water containing much peat from the mountains may be a cause.

III. Of the **intestinal parasites**, worms often cause diarrhœa, especially in children, who may have had uneasy abdominal sensations, night terrors, picking of the nose, itching of the anus, vulvo-vaginitis; but sometimes they are discovered in the stools when there have been no symptoms (§ 316).

LAMBLIA INTESTINALIS (Giardia) is a frequent inhabitant of the small intestine: small numbers of organisms are not pathogenic, but very large numbers can cause recurrent attacks of acute diarrhœa, with mucus and some blood in the stools.

Treatment: mepacrine hydrochloride B.P. (atebrin) 0·1 G. t.d.s. by mouth for five days is a certain cure.

IV. **Infantile Diarrhœa** occurs in at least three well-recognised clinical forms: (i.) Acute Catarrhal or Dyspeptic Diarrhœa; (ii.) Inflammatory Diarrhœa or Entero-colitis; and (iii.) Epidemic Diarrhœa or "summer diarrhœa" (including Infantile Cholera)—mentioned in progressive order of severity.

(i.) In ACUTE CATARRHAL (dyspeptic) DIARRHŒA the stools are offensive, at first yellow, then greenish, slimy and mixed with curds of undigested food. Vomiting may or may not be present. It is usually transient if adequately treated.

(ii.) In INFANTILE INFLAMMATORY DIARRHŒA (Entero-colitis) the stools are green, slimy and often contain mucus and streaks of blood; there is some fever at the beginning, and abdominal distension. The stools vary with the predominant infection; they are acid and frothy in the fermentative type, alkaline and green in the putrefactive variety. The inflammation attacks chiefly the colon; consequently there is tenderness on pressure over the region of the colon. Prostration is great when much vomiting occurs. Adults also are sometimes affected. It lasts only one to three weeks if treated correctly.

(iii.) EPIDEMIC DIARRHŒA ("summer" or "autumnal" diarrhœa of children) is met with chiefly in childhood and infancy in the summer months of the year, and is attended by catarrh of the mucous membrane of the bowel. The *symptoms* of a severe attack are: Watery stools, foul-smelling, of altered colour, containing lumps of mucus; vomiting; acute abdominal pain and tenesmus; prostration, collapse, subnormal temperature with pinched aspect, rapid dehydration and wasting, and often (after a course of a week or so) death from exhaustion. INFANTILE CHOLERA forms about 2 per cent. of "summer diarrhœa" cases. The stools are serous, there is persistent vomiting; collapse rapidly supervenes, the temperature in the rectum is raised as in adult cholera, and death soon follows.

Etiology of Infantile Diarrhœa.—These diseases affect chiefly hand-fed and over-fed children, in warm weather, being probably in part due to dirty feeding-bottles, teats, sour milk, etc. Most of the cases occur in children under six months old. Dietetic errors account for some cases, but the cause of *Epidemic Diarrhœa* is usually an infection. Seasonal, epidemic, and microbic causes have long been suspected on account of its prevalence during the summer and autumn months. It occurs chiefly after hot, dry summers. Flies and dust have been blamed; flies undoubtedly act as carriers of infection. It occurs chiefly in towns, and certain localities have been notorious for recurrent lethal outbreaks in summer and autumn. Adults do not altogether escape; diarrhœa is widely prevalent in the hot, dry summer months in some years; but in children the death-rate is often high. In most cases a virus infection is probably causal: some cases are due to dysentery bacillary infection of

the Shiga, Flexner or Sonne types, or to Morgan's bacillus; in others the food-poisoning bacteria (*B. enteritidis* of Gærtner and *B. ærtrycke*).

In the *Treatment of Infantile Diarrhœa* (1) first free the gastro-intestinal tract of all irritant materials; small doses of calomel (gr. $\frac{1}{10}$), or grey powder (gr. $\frac{1}{4}$), or equal parts of lime water and castor oil (F. 64) or 2–5 grs. sodium sulphate in water every two or three hours, until the stools become healthy; (2) a period of starvation for the first 12–24 hours; and later, (3) easily digestible food such as barley-water, diluted fruit juice, apple purée, two-hourly feeds of whey, peptonised milk or diluted condensed milk. In the fermentative type, use only a small amount of sugar, or a non-fermentable sugar such as dextri-maltose or lactose; subsequent additions to the diet must be made very cautiously. When vomiting is troublesome, gastric lavage with saline solution (not bicarbonate) should be used; then give sherry-whey (§ 297. XXII). When diarrhœa is persistent, give phthalyl-sulphathiazole or sulphasuccidine, and combine with astringents. In mild chronic cases, give protein milk, such as sprulac, or milk protein such as soluble casein or casec. In more severe cases colonic lavage with warm normal saline removes irritating material. Albumen or barley-water can be tried, and brandy, arrowroot, and an astringent mixture are useful: (bismuth carbonate 2 gr., calcium carbonate 3 gr., tincture catechu 5 min., glycerin 10 min., water to 60 min.). If used cautiously, nepenthe (♏iii. t.d.s. at 6 months) is valuable. Dehydration is a serious complication and must be combated with water by mouth or saline by rectal, subcutaneous, intraperitoneal or continuous-drip intravenous methods. For collapse give a transfusion of human blood plasma; niketham ide (coramine) and a warm mustard bath are the best stimulants.

A rare form of severe recurrent diarrhœa may occur in children—**Cœliac disease.** **The motions are pale and fatty, the abdomen is distended.** Owing to the continued defective absorption of food, the child does not grow (infantilism) and is often rickety; there is anæmia—micro- or megalocytic—yet after years of protracted illness fatal cases show no recognisable cause at autopsy. Defective fat and carbohydrate absorption appear to be the chief errors; the fats, though split, are not absorbed in the small intestine. The slightest error in diet will bring about a relapse of severe diarrhœa which may last many months. The diagnosis from tuberculous peritonitis may be difficult.

Treatment.—The fats in the diet are largely eliminated; sugar and starches are reduced when there is distension, and a large amount of protein added, as in sprue. Vitamin deficiency must be corrected by suitable additions of vitamins A, C and D. Crude liver extract injections and iron are useful.

V. **Typhoid** and **Toxic Blood States.**—Diarrhœa is usual in typhoid fever, and may occur in measles and the other eruptive fevers (especially at their advent), some cases of Graves' disease, chronic renal disease, uræmia, and pyæmia. Sometimes it appears at the termination of acute illness, as in pneumonia. (And see chronic causes, § 310.)

VI. A **chill** to the surface in some individuals will determine an attack of acute diarrhœa.

VII. **Acute Ulcerative Colitis** is usually of sudden onset, with diarrhœa, and abdominal pain occurring in paroxysms. The motions are dark,

offensive, and contain mucus and blood. There is tenderness over the colon, and its ascending portion is usually distended. The tongue is furred at first, and the breath very offensive. Pyrexia may be present, about 101° to 102°. The commonest complications are exhaustion, anæmia, profuse hæmorrhage; less common are perforation and peritonitis. Sigmoidoscopy shows a uniformly inflamed mucosa with exudate, necrosis of membrane, and often ulceration. Its etiology is unknown, although it resembles the epidemics of so-called *ulcerative colitis in asylums*, in which the organisms of bacillary and sometimes of amœbic dysentery may be found. (And see § 310. V.)

VIII. In cases of acute diarrhœa in which the cause is obscure, reference should be made to the other **Causes** of **Chronic Diarrhœa,** any of which may from time to time give rise to an acute attack. **Dysentery** (§ 308) and **Cholera** (§ 309) are the commonest causes of diarrhœa in tropical climates, and are occasionally met with in this country.

Prognosis of Acute Diarrhœa.—The causes of acute diarrhœa are for the most part removable; and though weakened by the attack, the patient generally makes a good recovery. Acute Epidemic Diarrhœa in children, however, is a more fatal affection, and it leads to a high death-rate in infancy. The prognosis in any given case depends upon (i.) the cause; (ii.) the severity of the symptoms and the evidences of dehydration; (iii.) the state of the hygienic surroundings; and (iv.) the response to treatment. Infantile cholera is usually fatal. Dyspeptic diarrhœa may be cured in a few weeks, but if untreated, is apt to go on to catarrhal or mucous colitis. Without treatment all forms of epidemic diarrhœa, even in adults, are serious. Should symptoms of prostration or collapse ensue, the outlook is bad; but it is only at the two extremes of life that this disease is so grave. Ulcerative colitis is serious; death may occur from complications, exhaustion, anæmia, or relapses.

Treatment of Acute Diarrhœa.—The indications are (*a*) to remove any irritating matters left in the intestinal canal; (*b*) to provide absolute rest, and warmth to the abdomen; and (*c*) to check excessive diarrhœa. (*a*) Thus, simple acute diarrhœa following the eating of bad food is readily arrested by giving castor oil, ½ oz., with tr. opii, ℥x., followed by a simple bismuth salicylate mixture. No food is allowed for a day, but as much water as desired is drunk: half-normal saline by mouth is essential when dehydration is severe. Then arrowroot made with water is given, and a gradual return to ordinary diet, beginning with milk and milk puddings. For some time all irritating skins, seeds, vegetable cellulose and raw fruits may not be taken. Simple dyspeptic diarrhœa in children is cured with grey powder every night and alkaline carbonates by day. After the acute stage is over, if the condition threatens to become chronic, other drugs are used. Opium checks diarrhœa; it can be given in the form of Tr. chloroformi et morphinæ, B.P./'85, 5–10 minims. Catechu, kino, chalk and tannin are excellent astringents. Bismuth carbonate or kaolin (up to 3–4 G. daily) soothe the congested mucous membrane. For

offensive stools give salol, calomel or charcoal. A course of intestinal antiseptics is often useful—e.g. sulphasuccidine or phthalyl-sulphathiazole. For putrefactive diarrhœa, protein foods should be avoided ; *B. acidophilus* helps to implant a healthier intestinal flora. In fermentative diarrhœa carbohydrates must be restricted ; sugar, bread and flour are usually digested ; but rice, tapioca, bananas, root vegetables, especially potatoes, should be avoided for a time, and a diastase preparation taken. In more obstinate cases of diarrhœa colonic irrigations may be required ; saline or permanganate douches, introduced slowly and without pressure, give excellent results in some cases. Where there is much loss of fluid, saline infusions may have to be given to prevent collapse. The treatment of acute is similar to that of chronic ulcerative colitis and is therefore dealt with in § 310. V.

In the tropics **Diarrhœa** is a common complaint ; it may merely indicate some simple intestinal derangement due to bad food, indiscretion of diet or chill such as may follow sleeping under a fan or punkah during the hot weather. On the other hand, many serious intestinal maladies such as cholera, typhoid and sprue may begin with diarrhœa ; the appearance of mucus, pus and blood in the stools, however, indicates that the trouble, which may be due to a number of different causes, probably arises in the large bowel.

The patient, who is living or has lived abroad, complains of severe DIARRHŒA, WITH BLOOD, MUCUS, *and perhaps* PUS *in the stools.* The disease is probably DYSENTERY.

§ **308.** IX. **Dysentery** is a colonic inflammation, often leading to necrosis and ulceration of the mucosa, due to certain specific bacilli, protozoa or helminths, and characterised by the frequent passage of stools containing mucus, blood and pus. Three main types occur : (1) ACUTE and CHRONIC BACILLARY DYSENTERY, due to a number of different micro-organisms ; (2) PROTOZOAL DYSENTERY, due to (*a*) *Entamœba histolytica*, (*b*) *Balantidium coli*, (*c*) malignant tertian malaria and (*d*) kala-azar ; (3) HELMINTHIC DYSENTERY, associated with (*a*) blood flukes : *Schistosoma mansoni*, *S. japonicum*, and (*b*) the intestinal nematode *Œsophagostomum apiostomum*.

(1) **Acute Bacillary Dysentery** has an incubation period of 1 to 7 days and sudden onset with fairly high fever, nausea, vomiting and headache, followed by colicky abdominal pain, tenesmus and the frequent passage of small stools, 5 to 50 times daily. As toxæmia increases the cheeks become flushed, the pulse rapid, the tongue coated. Dehydration produces restlessness, mental confusion, thirst, dry brown tongue, pinched features, sunken eyes, collapsed veins, and in infants a depressed fontanelle. The stools soon lose their fæcal character and consist of odourless, gelatinous mucus mixed with bright red blood, later becoming muco-purulent ; with recovery bile-stained fæcal matter reappears. Localised abdominal pain and tenderness are infrequent in the absence of peritoneal involvement ; and though some rigidity of the abdominal muscles may at first be present, the contracted sigmoid can later be palpated. Arthritis and iritis may occur. Fulminating and severe Shiga cases die of toxæmia or dehydration with subnormal temperature, but with appropriate treatment the average case becomes apyrexial in 7 to 14 days ; renal failure sometimes occurs associated with glomerulo-nephritis from dehydration and toxæmia. Most cases of Flexner and Sonne dysentery recover.

Chronic Bacillary Dysentery may (1) follow an acute attack or (2) be subacute from the onset. In (1) there is a history of acute dysentery from which there has never been complete recovery. Generally there is frequent defæcation, rectal discomfort or tenesmus, muco-pus and blood in the fæces. During exacerbations fever

may recur. Emaciation, asthenia, secondary anæmia and often œdema of the limbs follow. The thickened, spastic descending colon is palpable. During exacerbations dysentery bacilli may be isolated in about 25 per cent. of cases. In (2) the onset is more insidious, and the original attack mild; bouts of diarrhœa with mucoid, bloody stools follow; remissions are common. The disease closely resembles chronic ulcerative colitis.

Etiology.—The specific organisms are Shiga's bacillus, the Flexner Y group (V, W, X, Y and Z strains) and Sonne's bacillus. Shiga dysentery is mainly a tropical malady; the others may occur in Europe, especially in military barracks, prisons, asylums and in certain outbreaks of summer diarrhœa in children. The disease is spread by water and food contaminated by carriers, or by infected flies.

Treatment of Acute and Chronic Bacillary Dysentery. The *sulphonamide compounds* are of the greatest value. Give by mouth sulphaguanidine, 6–8 G., initially followed by 3–4 G. four-hourly until the stools are less than five per day; then eight-hourly until the stools are normal. This drug is effective and safe in patients initially dehydrated; crystals may be seen in the urine but they appear soft and do not produce renal blockage: rarely headache, nausea, mild erythematous or maculopapular rashes, with or without slight fever, may result from sulphaguanidine, but the risks of agranulocytosis or of exfoliative dermatitis seem negligible. Succinyl sulphathiazole 20 G. daily, or phthalyl sulphathiazole 20 G. daily, in divided doses at three-hourly intervals, have been advocated, as also have the absorbable sulphonamides, *e.g.*, sulphadiazine 1 G. four-hourly. All these drugs act by bacteriostasis, and consequently in severe Shiga infections, where there is already much toxæmia, Shiga antitoxin in addition is advisable, *e.g.*, 200,000 I.U. of refined antitoxin intravenously and repeated in 12 hours if indicated. *Dehydration* is treated by liberal fluids by mouth and if necessary by intravenous injections of saline or 5 per cent. dextrose solution. Routine treatment with saline purges is no longer favoured as it appears to be of little real value and leads to further dehydration; gentle colonic lavage with warm physiological saline may help to remove toxic material. In severe cases with circulatory failure serum, plasma or blood *transfusions* may be of value. The *diet* should be fluid with glucose and albumin for the first day or two, then gradually increased but maintaining a low residue; in the later stages a high calorie, high vitamin, low residue diet is required. Symptomatic relief of griping or tenderness may be obtained with warmth to the abdomen, or gentle saline bowel washouts, or injections of morphine. In chronic conditions, as a last resort, cæcostomy or ileostomy may be considered, to rest the bowel.

(2) PROTOZOAL DYSENTERY. (*a*) **Amœbic Dysentery** is characterised by afebrile diarrhœa with several voluminous fœtid stools daily, containing brownish mucus and dark red blood; tenesmus occurs if the rectum be involved. In 10 per cent. of patients the condition is more acute, fever is present and the bowels may act a dozen times in the 24 hours. Palpation reveals a thickened, tender colon; sigmoidoscopy may show typical painless, yellow, amœbic ulcers surrounded by a zone of hyperæmia, and healthy intervening mucosa. *Entamœba histolytica* can be found by examining the fresh material obtained by swabbing or lightly curetting the base of the ulcer. The disease is acquired by swallowing the cysts in contaminated water and food, especially vegetables, infected from convalescent or contact carriers.

Diagnosis.—The actively motile amœba containing red blood corpuscles is found in the mucoid exudate in acute cases, and the cysts in the solid fæces of chronic cases. Ulceration is almost always confined to the colon and often involves the muscular coats. Complications include intestinal hæmorrhage, perforation with peritonitis, retro-colic abscess and post-dysenteric adhesions, and amœboma (a chronic amœbic granuloma) often closely simulating neoplasm. Amœbic hepatitis is associated with fever, enlarged, tender liver and slight leucocytosis, while the formation of liver abscess is suggested by rigors, sweating, shoulder pain, and involvement of the base of the right lung (§ 336). Amœbiasis of the lung is occasionally found; involvement of the brain, spleen and abdominal wall is rare.

Treatment.—Patients require a low-residue diet, and should be kept in bed during treatment with emetine. For intestinal infection, each night emetine bismuth iodide gr. iii, preceded half an hour before by phenobarbitone gr. i, is given orally for ten to twelve days: while each morning (after a preliminary bowel washout with 1 per cent. sodium bicarbonate in water) a retention enema of 250 c.c. of 2½ per cent. chiniofon (B.P.) in water is given slowly and retained for 4–5 hours. Following this combined treatment carbarsone (B.P.) or stovarsol (B.P.) 0·25 gm. is given orally morning and evening for a further ten days. When there is severe ulceration, specific treatment may be preceded by a course of penicillin (30,000 units three-hourly subcut.) and sulphaguanidine (3 G.; three-hourly by mouth) for five to seven days to eliminate secondary infection. Amœbic hepatitis or abscess requires emetine hydrochloride gr. i. intramusc. daily for seven to ten days, while pus, if present in any quantity, may require aspiration: open operation is only performed if secondary infection necessitates it. Emetine is a myocardial poison, producing tachycardia and a lowered blood pressure; its prolonged use may lead to profound asthenia, neuromuscular weakness and heart failure: an increase in diarrhœa, after an initial improvement, may be also a sign of over-dosage. Diodoquin 1·5–2·0 G. daily by mouth for three weeks may be useful in ambulatory treatment of intestinal amœbiasis; it is not absorbed and therefore is useless for hepatitis.

(*b*) **Balantidial Dysentery** occurs in people who handle pigs. There are frequent muco-sanguineous stools and anæmia; many cases remain latent. Ulcers form in the colon and may perforate. Clinically, this condition is indistinguishable from amœbic ulceration, the diagnosis being made by microscopical examination of the stools or of material curetted from the ulcers during sigmoidoscopy. *Balantidium coli*, in vegetative forms or cysts, may occur in the excreta.

Treatment.—Stovarsol, gr. iv. t.i.d. for 1 week, and enemata of methylene blue (1 in 3,000) are advocated.

(*c*) **Malarial Dysentery.**—Malignant tertian malaria (*Plasmodium falciparum*) may manifest itself by severe diarrhœa, with occasionally blood and mucus, producing a syndrome indistinguishable clinically from bacillary dysentery; the condition originates from obstruction in the capillaries with clumps of agglutinated corpuscles infected with parasites. Sigmoidoscopy in the milder cases shows a diffuse or patchy hyperæmia; in severe cases scattered hæmorrhagic areas are seen in the hyperæmic bowel wall, and these may go on to actual necrosis. The *diagnosis* is made by finding the parasites in blood smears and by the associated clinical features, such as splenomegaly, anæmia and fever.

(*d*) **Kala-azar Dysentery.**—In kala-azar, diarrhœa is a not uncommon complication; *Leishmania* may be found in the intestinal villi or polypoid tissue formed in the gut wall. Occasionally a dysenteric-like syndrome with the passage of blood and mucus supervenes, which may prove to be due either to kala-azar itself or to a superadded bacillary dysentery infection.

(3) Helminthic Dysentery.—(*a*) **Schistosomal dysentery,** especially at first, is characterised by diarrhœa or loose motions containing mucus and blood; later there are often solid stools coated with mucus containing the lateral spined ova of *S. mansoni* or the lateral knobbed ova of *S. japonicum*. Occasionally the terminal spined ova of *S. hæmatobium* may be found (Fig. 107), though this species rarely gives rise to dysenteric features. Tenesmus, loss of weight and secondary anæmia may follow: many cases remain latent. Subacute or chronic schistosomal appendicitis, colonic and rectal papillomata, fistulæ, and a periportal cirrhosis of the liver may develop; the latter may be associated with an enlarged spleen (Egyptian splenomegaly). Eosinophilia is common, and the schistosomal complement fixation reaction is of value when it is difficult to find ova. Sigmoidoscopy aids: early minute tubercles are seen and eggs are found on scraping these; later, the pathognomonic schistosomal papillomata, which on sloughing leave punched-out circular ulcers.

Treatment.—Trivalent antimonial compounds are specific. Sodium antimony tartrate intravenously on alternate days for one month is frequently used; give an

initial dose of gr. ½ and increase in successive doses by gr. ½, to a maximum of gr. 2½. Recently a short intensive course of a total dosage of gr. i per 12 lb. body weight (12 mgm./kgm.) divided into 6 doses has been advocated, 3 doses being given at three-hourly intervals on two successive days: each dose is dissolved in 10 c.c. of 5 per cent. dextrose solution and injected not faster than 2 c.c. per minute. Cough, vomiting or rheumatic-like pains may follow administration of this drug. Stibophen, in 6·3 per cent. solution, is also used, doses of 1·5 c.c., 3·5 c.c., and then 5 c.c., being given intravenously on alternate days to a total of 40–60 c.c. Anthiomaline, in 6 per cent. solution intravenously or intramuscularly, is preferred by some in doses of 4 c.c. on alternate days to a total of 40–60 c.c. Miracil in doses of 10 mgm./kgm. twelve-hourly by mouth for 10 doses appears of great value.

(*b*) *Dysentery due to* **Œsophagostomiasis.**—The nematode parasite, *Œsophagostomum apiostomum*, commonly affects man in Northern Nigeria. Its embryos embed themselves in the walls of the colon and become enclosed in fibrous tissue nodules; as they approach maturity they escape into the lumen, often leaving behind an ulcerated area, and finally attach themselves to the mucosal lining of the gut. Diarrhœa with blood and mucus may result, and peritonitis is an occasional complication. The eggs, which resemble ancylostome ova, are passed in the stool. Tetrachlorethylene or carbon tetrachloride cures the disease. (See Ancylostomiasis § 547.)

The patient complains of ACUTE DIARRHŒA, *coming on very suddenly, and attended with severe* COLLAPSE, *abdominal* CRAMPS, *and "rice-water" stools.* The disease is probably CHOLERA.

§ **309.** X. **Cholera** (synonym: Asiatic Cholera) is a disease caused by *Vibrio choleræ*; it begins with urgent vomiting, purging, and colourless evacuations, cramps and a tendency to collapse, and which, if not fatal in the first stage, is attended by secondary fever. The period of incubation is usually three to six days, but it may vary between one and ten. There are three well-marked stages:

(*a*) *Stage of evacuation*, which lasts from two to twelve hours, or longer. The patient is *suddenly* seized with violent vomiting, profuse diarrhœa and later cramp. The stools, after the first few, are colourless and opaque, resembling rice-water, and containing flakes of columnar epithelium and casts of villi, and the *comma-shaped bacillus*. There are severe cramps in the fingers, toes and abdominal muscles, great exhaustion, small and weak pulse, and coldness of the body. (*b*) The *algid stage*, cold stage, or stage of collapse, lasts a few hours to a few days according to the severity of the case. The patient looks like a corpse; the surface temperature falls, and the skin becomes a deadly livid hue; the pulse cannot be felt at the wrist. The temperature is most remarkable, for in the rectum it may be as high as 105° F., while in the axilla it is only 90° F. During this stage the purging ceases, but the vomiting and cramps persist. The mind remains clear. There is suppression of urine and bile. (*c*) *Stage of reaction.*—The pulse returns, the temperature rises, the bile reappears, the urine is scanty and deficient in urea. The temperature goes up, and may be attended by typhoid symptoms. The bowels are confined. There may be erythematous, urticarial and other eruptions upon the skin. This stage is followed by great weakness. Fluid and salt loss is important in this disease. Diarrhœa and vomiting also produce salt depletion, decreased blood volume with increased viscosity of blood and dehydration of the tissues. Polycythæmia and leucocytosis also result from concentration. The blood chemistry shows reduced blood chloride, diminished plasma alkalinity, phosphate retention and increased blood urea, with decreased urinary output. Finally the weakened heart fails to pump the viscous blood through the kidneys and anuria with acidosis results.

The *Diagnosis* is easy in severe cases on account of the extreme suddenness and severity of the symptoms. The copious colourless evacuations are characteristic of cholera. Conditions which resemble it are acute poisoning by arsenic, croton oil, and other irritants, ptomaine poisoning, and certain cases of malignant malaria. The identification of the bacillus renders the diagnosis certain.

Etiology.—The disease occurs in great epidemics, but has not visited this country, except sporadically, since 1865–6–7. In India it is endemic. As regards age, none are exempt. All epidemics in this country have occurred in the autumn and the end of the summer. The exciting cause is the specific organism, which must be introduced into the alimentary canal. As with typhoid, the disease is usually communicated by drinking water contaminated by the evacuations from the bowels and stomach, and requires the same preventive measures (§ 522 *et seq.*). It may be conveyed in other ways, as by flies, through *want of cleanliness*. One attack does not confer immunity.

Prognosis—The earlier cases of an epidemic are the most fatal. The mortality rate varies from 30–70 per cent. in different epidemics. Aged and debilitated people, young children and alcoholics do badly. New methods of treatment have reduced the mortality by half. In the reaction stage uræmic coma, hyperpyrexia, or the typhoid state may cause death. *Untoward Symptoms* are blood in the evacuations, long stage of collapse, restlessness, extreme cyanosis, and absence of the pulse at wrist. Favourable signs are a perceptible pulse in the algid stage, the early occurrence of reaction, cessation of cramp, secretion of urine, and the occurrence of sleep. *Complications* include pneumonia, occurring in the reaction stage, bronchitis, pleurisy, parotitis, bed-sores, inflammation of the pharynx, genitals, or bladder, corneal ulcers and gangrene of the fingers, toes, scrotum or penis.

There are two rare *varieties:* (1) Choleraic diarrhœa, or "cholerine"—resembling autumnal diarrhœa occurring during an epidemic of cholera. (2) Dry cholera or cholera sicca, where there has been no vomiting or diarrhœa, the patient dying of collapse before these have had time to develop. At autopsy the intestines contain much fluid.

Treatment.—Prophylactic vaccine gives immunity for several months. Tinct. opii (♏ 20.) may be given at the onset of the preliminary diarrhœa, but never after the characteristic colourless evacuations have set in. Rest in bed, warmth, and fluid farinaceous diet are essential; animal protein in soups and jellies is harmful. Kaolin may be given in massive doses. Sulphaguanidine by mouth, 6–8 G. initially followed by 3–4 G. four-hourly, has recently been advocated. Injections of hypertonic or isotonic saline have been used with excellent results. When collapse appears, saline injections by rectum are useful so long as the systolic blood-pressure is above 70 mms.; below that point they are not absorbed, and an intravenous injection should be administered of sufficient amount (3 to 5 pints) to raise the blood pressure, and ensure excretion by the kidneys. The hypertonic solution contains sod. chlor. gr. 120, calc. chlor. gr. 4, potass. chlor. gr. 6, to a pint of sterile water, and is given at the rate of 4 ounces per minute. Nikethamide, sips of tea and coffee, act as stimulants. Potassium permanganate gr. 2 is given by the mouth every half-hour as an oxidising agent to destroy the toxins of the cholera bacillus.

In milder cases treatment with essential oils is good, the mixture consisting of ol. anisi, ol. cajuputi, ol. juniperi-āā ♏ 5, spt. æther. ♏ 30, ac. sulph. arom. ♏ 15: Dose ♏ 30 in water every ¼ hour for 16–28 doses.

§ 310. Chronic Diarrhœa.—The term chronic diarrhœa signifies the occurrence of frequent *loose* evacuations, say three or more in the twenty-four hours, extending over a period of weeks, months, or even years (as in Sprue). It is usually, though not necessarily, attended by tenesmus. The stools should be examined (§ 303) and the anus and rectum carefully inspected. Tenesmus points to disease of the rectum.

I. Chronic Diarrhœa may be due to some of the same causes as **Acute Diarrhœa** (*q.v.*). In children think of worms or bad feeding; and in adults, errors in diet, carbohydrate dyspepsia, ulceration, and chronic irritant poisoning.

II. **Fissure of the Anus,** slight ulcers or abrasions, or even an inflamed

pile, may cause a chronic diarrhœa. Actually there is underlying retention of stool (constipation), and the diarrhœa is "false diarrhœa."

III. **Ulceration of some part of the Intestinal Canal** is a not infrequent cause of diarrhœa in England; it is well to mention here the ulcerating lesions which may affect the intestine. (1) Ulcer of the lower part of the ileum may be due to tuberculosis, typhoid fever or Crohn's disease. (2) Ulcer of the cæcum may arise from the pressure of inspissated fæces or some foreign body—*e.g.*, a tooth-brush bristle—which has been swallowed. (3) Ulcer of the appendix may similarly arise from foreign bodies or as part of appendicitis (§ 247). (4) Ulcer of the rectum is generally of malignant, gonococcal or syphilitic origin; it is attended by the passage of blood and pus, and stricture may result. Mainly in tropical areas, lymphogranuloma inguinale can cause chronic ulceration and stricture formation. (5) Ulcers of the large intestine and rectum occur in dysentery. These may contract on healing and produce stricture. (6) Cancer of the bowel may produce ulcer, the most frequent situation being the sigmoid. (7) Simple ulcerative colitis (§ 310. V). (8) Ulceration may follow prolonged constipation with atony of the colon. (9) A submucous streptococcal infection may cause chronic diarrhœa with precipitate stools, as may chronic nephritis, severe anæmia, and other wasting diseases.

The commonest causes of ulceration in this country are Ulcerative Colitis, Cancer, Tubercle, Regional Ileitis, Syphilis, and in tropical climates Dysentery (§ 308).

1. Ulcerative Colitis, (§ 307. VII and § 310. V) causes one of the most intractable forms of chronic diarrhœa.

2. Intestinal Cancer presents the following features: (i.) The patient is usually over fifty; (ii.) diarrhœa and anæmia due to hæmorrhage are common if the disease is in the cæcum or ascending colon: obstructive symptoms and diarrhœa alternating with constipation if in the descending or sigmoid colon; (iii.) paroxysmal abdominal pain is frequent; (iv.) tenesmus indicates a lesion in the rectum; (v.) a tumour may be palpable through the abdominal wall, or by rectal examination. It is most difficult of access when in the lower sigmoid colon; and may then (vi.) be within reach of the sigmoidoscope. (vii.) Cancerous cachexia often accompanies. (viii.) Pyrexia and leucocytosis may be due to ulceration. (ix.) The stools vary; they may contain blood in considerable quantity, but invariably occult blood. (x.) X-ray with a barium enema may show a characteristic filling defect. (Fig. 82.) And see § 320.

3. Tuberculosis of the lungs may be attended by diarrhœa, even without ulceration of the bowel. Multiple ulcers due to tuberculosis may be found in the lower ileum, and less commonly in the rectum, where the symptoms mimic ulcerative colitis (see ulcerative proctitis, § 314. VIII.) Tuberculous ulceration of the bowel is recognised by (i.) evidences of tuberculosis in the lungs or other parts of the body; (ii.) the presence of night sweats and intermittent pyrexia; (iii.) the stools are watery, and

there is rarely any pain ; (iv.) tubercle bacilli may be demonstrated in the stools by appropriate staining methods. Relief is generally effected by quinine and opium internally, combined with appropriate dietary ; if these fail, recourse may be had to iron, opium, and lead.

4. Regional Ileitis (Crohn's disease), usually of the terminal ileum, due to chronic non-specific inflammation, may cause symptoms simulating appendicitis, or ulcerative colitis with much diarrhœa. Secondary abscesses may form. Young people are usually affected, and the symptoms are progressive and recurring. With X-ray there is filling defect of the ileum or cæcum. Excision is the best treatment ; sometimes in two stages, with a short-circuit of the affected part of the bowel first.

5. In Syphilitic Ulceration of the rectum (i.) the motions often consist largely of pus and blood ; (ii.) great pain and tenesmus are usual, combined with (iii.) other evidences and a history of syphilis. Stricture occurs in the later stages. (iv.) Opium and antisyphilitic treatment are here of great value to check the diarrhœa.

IV. **Chronic** or **Mucous Colitis** (Syn. : Intestinal Dyspepsia) is much more common in women. It can cause many years of ill-health, and needs much patience in treatment. The *Symptoms* vary, but in the main are : (i.) Irregular attacks of diarrhœa alternating with obstinate constipation, brought on by slight dietetic errors, by nervous causes, or by the misuse of aperients. (ii.) During the attacks of diarrhœa, mucus may be passed in masses, shreds or casts several inches long : the stools often contain intestinal sand but not blood. (iii.) The stools may show carbohydrate fermentation. (iv.) There is abdominal discomfort, and a good deal of flatus passed per rectum ; the flatulence may cause insomnia. (v.) B. coli may infect the urinary tract. (vi.) Nervous prostration, ready fatigue and loss of weight are almost always present and may be the presenting symptoms. (vii.) Examination may reveal a distended cæcum while spasmodic contraction of the descending colon may be felt. The sigmoidoscope aids diagnosis in obscure cases.

Diagnosis.—The disease is distinguished from ulcerative colitis by the presence of blood in this latter condition, and from carcinoma coli by the length of history and the type of stool. Sometimes neurasthenia is accompanied by marked colonic dyspepsia, in which case treatment of the colon may aid recovery.

Treatment.—Sometimes many of the symptoms of early colitis are due to excess of purgatives and enemas to which the patient has resorted in the mistaken belief that she suffers from constipation. In such cases, no strong purgatives should be used ; relief is obtained by prescribing liquid paraffin or petroleum-agar, belladonna or eumydrin, codeine or kaolin. A non-irritating diet is essential ; all seeds, skins and stringy foods must be forbidden ; forbid or curtail foods which are not digested —in some, fruit and vegetables, owing to their cellulose ; in others, starchy or protein foods. The patient may benefit from diet consisting entirely of milk for a time. A diet deficient in vitamins can lead to atrophy of the intestinal mucous membrane ; vitamins (especially Vit. B) must be restored to the diet in such cases. Fruit juices and purées are given to provide the vitamins which are lacking. B. acidophilus with

lactose aids the development of a healthy intestinal flora. Taka-diastase is useful when undigested starch appears in excessive quantities in the stool. Adequate warm clothing covering the abdomen is essential, and when visceroptosis is also present, a suitable abdominal belt aids. Occasionally lavage of the colon with saline or weak potassium permanganate is of help, when carried out by an expert. In other cases an intestinal autogenous vaccine and abdominal diathermy are useful. The psychological aspect must receive attention: often an unhappy environment at home or at work needs correction: fatigue and overwork should be avoided, and at all times, the patient must be discouraged from examining his stools.

V. **Chronic Ulcerative Colitis** may follow the acute variety (§ 307. VII), but much more often its onset is insidious, with (1) apyrexial diarrhœa, which at first is not regarded seriously, and lower abdominal pain and flatulence: (2) then follow blood and muco-pus, mixed with fæces, (3) rectal discomfort and tenesmus, (4) loss of weight, (5) secondary anæmia and fever may develop. (6) Hypochlorhydria is common. The condition often begins as a granular proctitis which tends to ascend to the pelvic and descending colon and in extreme cases the cæcum. The *Diagnosis* depends on: (1) Sigmoidoscopy in a well-established case shows a uniformly inflamed, granular mucosa bleeding readily on pressure, with miliary or larger-sized ulcers; the lumen of the rectum and large bowel is narrowed, its walls rigid and thickened so that ballooning with air becomes painful and difficult. (2) X-ray reveals a shortened, tubular bowel with complete loss of haustration; if deep ulcers exist the outline of the colon is feathery and moth-eaten in appearance. *Prognosis.*—Intermissions are common, apparent recovery being followed by relapses extending over many years; many of the gravest cases recover eventually. Complications include hæmorrhage, perforation, stricture, polyposis, malignancy and arthritis.

The specific *cause* is unknown. *Treatment.*—Keep the patient strictly in bed until the condition has healed. First the abdominal discomfort, fever and diarrhœa disappear, later the pus, blood and mucus are absent from the stools, but healing is not complete until confirmed by sigmoidoscopic examination. The diet must be ample and varied, but restrict foods with vegetable residues: supplementary vitamins, especially vitamins B and C, should be given. A mixture containing tinct. opii ℥ 10, tinct. belladonnæ ℥ 7½ with bismuth carbonate gr. 10 is often most helpful in controlling diarrhœa. Courses of colonic lavage on alternate days, for 10–14 days, are given—first, sodium bicarbonate solution (60 gr. to 1 pint) or normal saline is used to remove as much fæces and mucus as possible: this is followed by chiniofon B.P. (yatren 1 per cent.), tannic acid (1/500), protargol or albargin (1/1,000) or potassium permanganate (1/5,000) for their astringent and antiseptic effects. Hurst obtained good results with polyvalent dysenteric serum given intravenously in doses of 50–100 c.c., diluted with 150 c.c. saline daily for 7–10 days. Long courses of sulphasuccidine or phthalylsulphathiazole (G. 1–2 q.i.d. for many weeks or months) until the diarrhœa ceases, are often of great value. Septic foci must be suitably dealt with, but there is little evidence that other sera or vaccines are of any help. When anæmia is present, one or more blood transfusions are often of remarkable value. In long-standing or obstinate cases, especially with polyposis, ileostomy may be performed, as this allows the fæces to be drained and permits lavage of the whole colon through the artificial opening. If this fails, colectomy may have to be resorted to. To prevent relapses, the stools must be kept soft with liquid paraffin, warm clothing used to prevent chill, and foods with irritating residues avoided over a period of many years.

VI. **Obstruction in the Portal Circulation** produces diarrhœa, due to the congestion of the intestinal wall. It is recognised by: (i.) A previous history of heart disease, or of intemperance and alcoholic dyspepsia; (ii.) other signs of liver or cardiac disease;

(iii.) other evidences of portal obstruction, such as ascites, piles, and a large spleen (§ 260); (iv.) there is little or no pain, and the stools are liquid and dark, occasionally bloody. The *Treatment* requires caution, because the diarrhœa and hæmorrhage of themselves relieve the condition by diminishing the venous engorgement. (i.) If the diarrhœa has not lasted long, a dose of calomel will relieve the portal congestion, and so cure the diarrhœa. (ii.) Magnesium sulphate, gr. 20, with alum and dilute sulphuric acid, are recommended. Bismuth and opium, with caution, are the most useful for checking the diarrhœa.

VII. **Dysenteric Diarrhœa** is a sequel of dysentery, which may perhaps have been contracted abroad many years previously (see § 308).

VIII. **Nervous Diarrhœa** may continue for years; it has the following characteristics: (i.) The motions are often quite healthy. There is usually no pain or tenesmus. The diarrhœa is recurring or intermittent, occurring in the early morning, or when the patient is "nervous." Sometimes it follows each meal (*lienteric* diarrhœa). (ii.) Diet has little or no influence; the *attacks are determined* by mental emotion or bodily fatigue. The administration of nux vomica, belladonna, and bromides is more efficacious than astringents. Arsenic (℥ ii. Fowler's solution), with meals, is said to be a specific for lienteric diarrhœa.

The crises of LOCOMOTOR ATAXY sometimes take the form of acute diarrhœa, with or without pain. In HYSTERIA acute attacks of diarrhœa, with noisy borborygmi, may occur, determined in the same way as other hysterical attacks.

The *rarer* cases of chronic diarrhœa are:

IX. **Amyloid Disease** of the intestines gives rise to a most intractable form of chronic diarrhœa. Indeed, this is the common mode of death in amyloid disease of the viscera. The characteristics here are: (i.) A history of long-standing purulent discharge, or of syphilis; (ii.) great pallor of the skin, accompanied by evidences of lardaceous disease in the spleen, liver, and kidney; (iii.) the stools are generally liquid and extremely offensive, sometimes attended by hæmorrhage. The *Treatment* is very unsatisfactory. Opium does no harm, even when there is amyloid disease of the kidney, as there is no tendency to uræmia.

X. **Senile Diarrhœa** occurs in persons over sixty or seventy, and is very chronic in its course, but the patient suffers very little. Careful examination for organic disease should be made before concluding that the condition is simple senile diarrhœa. Most remedies fail to relieve it; it may exist for years without emaciation or danger to life.

XI. **Mineral Poisons,** and especially arsenic and antimony, in small and continued doses, may cause persistent diarrhœa.

XII. **Gastrogenous diarrhœa** may occur in cases of achylia gastrica, even when it is not associated with pernicious anæmia. It ceases when 30 to 60 drops of dilute hydrochloric acid are taken in a tumblerful of water with meals.

XIII. **Pancreatic Disease** has been associated with diarrhœa which resists treatment until pancreatic extracts are administered.

XIV. **Gastro-colic Fistula** and **Gastro-enterostomy** occasionally produce persistent diarrhœa and, especially in the case of the former, gross emaciation. If symptomatic treatment, together with that of the associated anæmia, does not relieve, operation must be considered.

XV. Other *causes* are hyperthyroidism, Addison's disease, excessive tobacco smoking in susceptible persons, chronic renal disease, anaphylaxis, and exhausting or wasting diseases of any kind.

§ **311.** XVI. **Sprue.**—Psilosis or sprue is a tropical disease of unknown etiology associated with derangement of the gastro-intestinal tract, characterised by deficient absorption of fat, glucose, certain vitamins and calcium; the secretion of the intrinsic hæmatopoietic factor is often also defective.

Signs and Symptoms.—(i.) Apyrexial morning diarrhœa with bulky, acid, pale, frothy, fatty stools. (ii.) Inflammatory lesions of the mouth; the tongue is tender, shows patches of inflammation, and later becomes pale and atrophic with disappear-

ance of the papillæ. Aphthous ulcers may involve the lingual or buccal mucosa. (iii.) Anæmia, which may be severe; this is almost invariably megalocytic in type with an increase in the average diameter of the corpuscles; it closely resembles pernicious anæmia and responds in the same manner to liver extract therapy and to folic acid. (iv.) Asthenia with low blood pressure. (v.) Emaciation and wasting; the skin becomes dry and wrinkled and sometimes brown pigmentation occurs over the forehead and malar eminences. (vi.) Intestinal flatulence; occasionally vomiting and dyspepsia. (vii.) In advanced cases neuritis, œdema of the feet, cramp and tetany may occur occasionally. (viii.) Physical examination shows a distended abdomen with thin abdominal parietes.

Diagnosis.—Sprue has to be diagnosed from pernicious anæmia, chronic pancreatitis, carcinoma of the pancreas and stomach, gastro-colic fistula, tuberculous enteritis and lymphadenoma involving the mesenteric lymph glands. Biochemical and radiological investigations often assist. Sprue shows a high fæcal fat which is adequately split, and low gastric acidity; with the aid of histamine, however, 78 per cent. secrete acid. Blood analysis often shows lowered calcium, slight increase in plasma bilirubin and delayed or low glucose tolerance curve, due to malabsorption of glucose; blood sugar curves following the intravenous injection of glucose show sluggish utilisation of glucose.

Prognosis.—Sprue is a very chronic disease, with spontaneous remissions and exacerbations. It particularly affects Europeans or those of mixed European blood, and until recently often terminated fatally if the patient remained in the tropics.

Treatment.—Patients are put to bed for one or two months and given a high protein, low fat, low carbohydrate diet (see § 297. XVII), in which the ratios of these different food constituents are 1 : 0·3 : 1·3; red meat is the main source of protein, or alternatively a defatted, dried milk known as "sprulac" may be substituted: the diet is graded from 600 to 3,000 calories. Crude liver extract, equivalent to 1½ lb. of whole liver daily should be given orally or intramuscularly; in severe cases, where large doses of liver are so beneficial, oral and intramuscular liver extract therapy may be combined. Recently remarkable initial results have been obtained with folic acid 20 mgm. daily for a week, followed by a daily maintenance dose of 5 mgm., but liver may be required to complete the response. Suitable diet with liver extract has made it possible to restore the majority of sprue cases to normal health in two months and to permit of their return to the tropics. It is, however, difficult to be certain that a permanent cure has been obtained in sprue; relapses may follow indiscretions in diet such as taking spiced and sugary foods or excess of carbohydrate and fat. Chill is especially to be avoided after return to England. In most cases a maintenance dose of liver extract is not required, but where repeated relapses have occurred it is advisable to give the patient liver extract by mouth in dosage equivalent to ½ lb. of whole liver daily for a period of 6 months. Nicotinic acid, 50 mgm. t.i.d., is indicated when oral symptoms are severe, riboflavin 3 mgm. daily for angular stomatitis, acid hydrochlor. dil. ℥ 30–60 t.i.d. with meals for achlorhydria, and calcium lactate gr. 30 t.i.d. for calcium deficiency.

§ 312. **Tenesmus** literally means straining at stool (*τείνω*, to strain or stretch); but in its widest sense it may be taken to mean any local rectal sensation of "bearing down" which results either in constant desire to go to stool, or straining when at stool. The latter may lead to prolapse of the rectum, especially in children. Diarrhœa is always attended by more or less tenesmus, but tenesmus is not always attended by diarrhœa. (1) Ascertain if the tenesmus is accompanied by diarrhœa—*i.e.*, are the motions frequent and liquid? If so, refer to the section on Diarrhœa, § 306. (2) Examine the motions; note their consistence and any abnormal constituents such as mucus and blood. (3) Search for

any local anal or rectal condition such as fissures, piles, polypi, or ulcers. All the pelvic organs should also be thoroughly investigated, especially in women, in whom the symptom is commoner than in men.

Causes.—Tenesmus (not necessarily accompanied by diarrhœa) may arise from four groups of causes:

1. Various conditions of the ANUS—pruritus, eczema, or fissure—may be overlooked for a long time. Piles also, if internal, may be difficult to detect, even by the examining finger, but streaks of bright blood will appear in the motions from time to time.

2. Various RECTAL CONDITIONS, especially carcinoma, simple ulceration (proctitis) or (rarely) stricture. The former are attended by pus or blood, or both. The latter (usually of syphilitic origin) is attended by tape-like stools. In the aged, always suspect cancer of the rectum. Prolonged use of purgatives, or constant use of enemas may result in straining at stool and prolapse of the rectum. An impacted fish-bone is a rare cause.

3. PRESSURE on, or irritation of, THE RECTUM FROM WITHOUT, such as may be caused by chronic congestion, retroversion or other disease of the uterus. Ischio-rectal abscess, pelvic hæmatocœle, and various ovarian and Fallopian tube lesions in women, and congestion or new growth of the prostate in men, are common causes. Any bladder disease, such as stone—a frequent cause of tenesmus in children, and apt to result in prolapse of the rectum—or new growths or chronic cystitis may cause this distressing condition. Menstruation and the later stages of pregnancy may be attended by a certain amount of tenesmus.

4. In HYSTERICAL AND NERVOUS SUBJECTS any fright or other emotion may at once determine tenesmus, which the patient calls "diarrhœa." In tabes dorsalis the "rectal crises" may take the form of tenesmus.

Treatment.—The indications are (1) the removal of the cause, the treatment of piles and other causal conditions being described elsewhere; (2) the relief of local congestion or irritation of the rectum. In any case, morphia, bismuth, belladonna, or cocaine or allied drugs in the form either of suppositories or ointments inserted by an applicator, will relieve the distress from which the patient suffers.

§ **313.** In **Proctalgia Fugax** the patient has recurrent severe cramp-like pain in the rectum and perineum, which usually wakens him up at night; it may last three to ten minutes or even longer. Rectal examination is negative. The pain is regarded as due to rectal or anal spasm, but some medical sufferers regard it as an allergic symptom or attribute it to venous engorgement. In women it occurs near the menstrual period.

Treatment.—The pain is usually relieved by a small meal which excites a gastro-colic reflex; otherwise insert suppos. bism. subgall. (B.P.C.) or inject the rectum with air or warm enemata. Sometimes attacks cease when the patient gives up smoking.

§ **314. Blood in the Stools** is met with, as we have seen, in dysentery and some cases of simple diarrhœa; it may occur in other conditions. The presence of blood in the stools may be recognised by the reddening of the water in which the stool is placed, or by the spectroscope (§ 303). Clinically, blood in the stools may present two widely different characters:

(*a*) When the blood is of *bright crimson colour* and is on the surface of the stool, it indicates either that the bleeding comes from the rectum or the lower part of the large bowel; or, if it comes from the upper part of the intestinal canal, that it is too large in amount to be acted upon by the intestinal secretion. (*b*) *Melæna* (*tar-coloured stools*) is met when hæmorrhage in moderate quantity has taken place in the stomach or the upper part of the alimentary tract, in which case the digestive fluids of the stomach and intestine acting on the blood give it a tarry colour. The causes of these two conditions are to some extent interchangeable, for what will produce a large hæmorrhage at one time may at another produce only a little. Bleeding, even if small in quantity, should never be neglected; often slight intermittent bleeding is the first sign of a malignant growth somewhere in the gastro-intestinal tract.

(*a*) **Bright Red Blood** may be due to lesions of the lower part of the alimentary canal. Of these several causes are referable to the anus or rectum, and are discovered on local examination or by proctoscopy.

I. Hæmorrhoids, or Piles, are undoubtedly the commonest cause of blood in the stools. The blood is generally met in streaks only, but the quantity may at other times be very large (§ 315).

II. Prolapse of the rectal mucous membrane, either acute or chronic, with contraction of the anal sphincter, may cause the appearance of bright blood, usually after a motion.

III. Fissure of the Anus may also produce streaks of blood. It is not infrequent, and is recognised by the excruciating pain during and after defæcation. The irritation it causes may give rise to a variety of false diarrhœa. The fissure can always be seen by *careful* examination. There may be a history of trauma.

IV. In Carcinoma of the Rectum or Colon the blood is usually mixed with the stool, and may be intermittent. A sudden and very severe hæmorrhage from the rectum may be the first sign of carcinoma. An innocent adenoma may cause hæmorrhage; it should be looked on as a precancerous condition and removed. Careful digital and sigmoidoscopic examination should be made.

V. A discharge of blood-stained mucus, coming on somewhat suddenly in an infant, is highly suggestive of Intestinal Intussusception, which is one of the causes of acute obstruction (§ 319).

VI. Rectal Polypi are met chiefly in children.

VII. Typhoid and Tuberculous Ulceration sometimes produce profuse discharges of bright red blood, coming from the lower end of the small intestine. Other evidences of these affections are present.

VIII. **Proctitis** may be simple, traumatic or infective (gonorrhœal, syphilitic, tuberculous or pyogenic). *Simple proctitis* occurs in association with chronic constipation or the repeated use of soapy enemata. *Ulcerative proctitis* is usually present in the upper part of the rectum, and may spread to the sigmoid and cause ulcerative colitis. It can only be diagnosed with certainty by proctoscopic examination. Lympho-granuloma inguinale is a cause of ulcerative proctitis and rectal stricture (90 per cent. of cases are women): and see § 310. III.

The leading *symptoms* are tenesmus and painful defæcation, sometimes discharge of blood and mucus *after* the passage of normally formed stools. *Treatment* is by avoiding hard vegetable residues in the diet, keeping the stools soft and the use of mild astringent retention enemas.

IX. Ulcerative colitis (§ 307. VII) occasionally causes severe hæmorrhage, more usually small repeated hæmorrhages.

X. Schistosoma Mansoni and hamatobium infestations cause a spurious dysentery with polypoid masses in the rectum. They are described in § 308 (3). *Schistosoma japonicum*, a third species, gives rise to Katayama disease or Schistosomiasis of the Far East.

XI. Various General Blood Conditions may give rise to hæmorrhage coming from the rectum or elsewhere in the alimentary canal in varying amount. This occurs in purpura, primary thrombocytopenia, scurvy, agranulocytosis, hæmorrhagic forms of the specific fevers, acute yellow atrophy of the liver, and leukæmia.

(*b*) **Melæna** (*tarry stools*) is met when bleeding takes place in moderate quantity from the stomach, or high up in the alimentary tract. 60 c.c. of blood can cause a tarry stool. Its causes are :

1. When coming from the Stomach, it may be associated with profuse *hæmatemesis* (§ 272) ; the commonest causes of hæmatemesis are peptic ulcer and hepatic cirrhosis. Melæna occurs often without hæmatemesis in duodenal ulcer.

2. Portal Obstruction (§ 260) is one of the most frequent causes of melæna, especially that form due to peri-portal cirrhosis of the liver. It may also occur with advanced cardiac disease. In either case the hæmorrhage is a natural safety-valve, and relieves the engorged state of the portal circulation.

3. Cancerous, Tuberculous, and other Ulcerations of the small intestine (see §§ 307 and 310), Crohn's disease, lardaceous disease of the bowel, mesenteric thrombosis or embolism, may produce melæna.

4. The General Blood Conditions above named, when the hæmorrhage is small in amount, are attended by tarry instead of bright red stools. *Melæna neonatorum* is a rare condition in which there is a passage of blood in new-born children (see § 551. VII).

5. Ancylostomiasis is a possible cause of melæna in Egypt and other foreign countries (§ 547), but generally it is only revealed by tests for occult blood.

The *Treatment* of melæna should be directed to the cause, but the general principles are those laid down for hæmatemesis (§ 272). Turpentine (10 minims capsule), lead acetate, and opium are recommended. Worms are dealt with in § 316. For melæna neonatorum, see § 551. VII.

§ **315. Hæmorrhoids,** or Piles are varicose rectal veins. This varicosity forms a swelling of variable size, which may be altogether within the anus (internal piles), or partly internal and partly external. Internal piles may in some cases be seen, when the patient "bears down," as small purple swellings protruding through the sphincter.

Symptoms.—(1) Streaks of *bright red* blood occur in the stools, usually dripping after the bowel has acted; sometimes as much as ¼ pint of blood may be passed at a time. (2) There is pain on defæcation,

the pain continuing for some time after the passage of a stool. When a pile becomes inflamed, or strangulated by the sphincter, severe pain and discomfort are experienced, and the patient may have to remain in bed. Pain may be referred to other parts of the body—*e.g.*, to the testicles or bladder. (3) Constipation nearly always accompanies piles, due partly to mechanical obstruction, and partly to the pain caused by defæcation. (4) Pruritus is often troublesome. (5) In severe cases constitutional symptoms develop due to severe anæmia.

Etiology.—(1) Portal obstruction is itself a cause of piles, and in all cases we should seek for the other symptoms of this lesion (§ 260). (2) Habitual constipation is undoubtedly the most common cause of hæmorrhoids, particularly in women, who in early life are so apt to contract this habit. (3) Alcohol causes portal congestion, and thus becomes a cause of piles. (4) Sedentary occupations and deficient exercise. (5) Various local conditions, such as sitting on a cold seat or soft cushions which constrict the inferior hæmorrhoidal veins, uterine displacements, pregnancy, carcinoma of the rectum, pelvic and other tumours.

Prognosis.—Hæmorrhoids are not serious, but may be extremely troublesome, by the constant loss of blood, by their liability to repeated attacks of inflammation and thrombosis, and by the pain they cause.

Treatment.—Much may be done by three simple means: (1) The avoidance of alcohol (especially malt liquors) and sugar; (2) keeping the piles scrupulously clean, and (3) the bowels regularly and loosely open. Prolapsed piles must be replaced at once. Rich food, wines and other causes of hepatic congestion must be forbidden. Confect. sulph. or sennæ, with an occasional cholagogue at night is good; paraffin is apt to cause the piles to descend. Local applications should be simple. The old-fashioned gall and opium ointment is now replaced by hamamelis, with bismuth, morphia, or cocaine for the pain, if necessary, or calamine powder on a pad of lint: Suppos. bismuth subgallate co. B.P.C. is very useful. Liquid hazeline is excellent, and is best applied on a strip of lint inserted within the anus, and left there; or a suppository may be employed, containing gr. 1 to 3 of hamamelis, and morphia gr. $\frac{1}{8}$, if requisite. Inflamed piles are very painful, and are best treated by warm hip-baths, frequent bathing, sitting over hot water in a bidet, warm fomentations with opium, belladonna or cocaine. Surgical removal is sometimes called for, but a cure may be obtained by a perivenous injection of the subcutaneous tissues around the pile. Use 5 c.c. of a solution containing phenol 20 gr., menthol 1 gr., almond oil to 1 fluid oz. Thrombosis is caused, and healing by scar. A strangulated pile may be incised radially under local anæsthesia, and the clot evacuated: this effects a cure.

A PERI-ANAL HÆMATOMA may be mistaken for a pile. It causes a local swelling and pruritus and is best treated by simple incision and evacuation of the clot. Gas-gangrene infection is rare, but is invariably fatal.

§ 316. **Intestinal Worms** often cause no symptoms. They are common in children, in inmates of mental hospitals and in adults who come from the tropics. The mor-

phology, symptoms and habitat of various parasitic helminths are described in Tables XVIII and XIX, p. 371 *et seq.* Thread worms (Fig. 78) and round worms (Fig. 79) are the most common in Britain.

Symptoms may result from reflex disturbances, mechanical action, helminthic toxins or anaphylaxis. They include (1) abdominal pain, sometimes paroxysmal in character, (2) capricious or ravenous appetite associated with (3) loss of weight, (4) irregularity of the bowels or diarrhœa, (5) such reflex disturbances as grinding of the teeth at night, enuresis, strabismus and even convulsions. (6) Erythema, urticaria and eosinophilia result from helminthic toxins or anaphylaxis. Skin hypersensitiveness to helminthic protein is manifest by rapid wheal formation following intradermal injection of saline extracts of the different parasites. The round worm, *Ascaris lumbricoides*, may produce helminthic pneumonia during the first week of infection owing to embryos traversing the lungs. Later, after the worms reach the small intestine, the above-mentioned symptoms may appear and occasionally severe manifestations such as: perforation of the bowel with generalised peritonitis or localised abscess with a fistula from which the worm is discharged, intestinal obstruction from masses of worms impacted near the ileo-cæcal valve, obstructive jaundice due to worms obstructing the common bile duct, or cholecystitis, liver abscess or œdema of the glottis associated with worms in these regions. Thread worms (*Enterobius vermicularis* or *Oxyuris vermicularis*) inhabit the colon and migrate through the anus at night, producing pruritus and eczema ani, bladder irritability, sometimes vulvitis and vaginal discharge, and even catarrhal appendicitis. Whip worms (*Trichuris trichiura* or *Trichocephalus dispar*) inhabit the colon, appendix and terminal ileum: the eggs have characteristic knobs. Reflex symptoms may appear and rarely appendicitis and peritonitis. *Strongyloides stercoralis* is a common tropical parasite, the females living in the jejunum and duodenum, occasionally invading the bile ducts and stomach: the eggs hatch out rhabiditiform larvæ which are found in the fæces. Dermatitis and lung symptoms may appear a few days after exposure; later, in severe cases, there is epigastric discomfort and diarrhœa. Urticaria, œdema and occult blood may be present. Ancylostomes are dealt with in § 547. *Heterophyes heterophyes* is a minute intestinal fluke infesting man in Egypt; by means of a sucker it clings to the mucosa and causes indigestion and diarrhœa. *Diagnosis* depends on finding the eggs in the stools, or the parasites themselves after straining the fæces through muslin.

Three different tapeworms may inhabit the intestine of man—*Tænia saginata*, the beef tapeworm; *Tænia solium*, the pork tapeworm; and *Diphyllobothrium latum* (*Dibothriocephalus latus*), the broad-fish tapeworm which undergoes development first in the water flea, *Cyclops*, and later in fish, man becoming infected by eating underdone fish. *Symptoms* may be absent or there may be (1) gastro-intestinal symptoms; (2) reflex disturbances, especially in children and occasionally (3) megalocytic anæmia occurs with *D. latum*. The cysticercus stage of *T. solium* is occasionally found in man, especially in India, producing encapsuled nodules in the muscles, subcutaneous tissues and organs, including the brain; in the latter case epilepsy may result several years after infection, symptoms being associated with the death of the cysts (§ 723).

Diagnosis of the cysticercus stage of *T. solium* is made by biopsy of a cyst, or X-ray examination may show calcified cysts in muscle tissue. Eosinophilia, skin hypersensitiveness to *Tænia* antigen and positive complement fixation reactions may be obtained, but are frequently negative. The prognosis is bad if the brain be involved. The cyst stage of *Echinococcus granulosus*, the small tapeworm of the dog, may affect man, especially involving the liver (see § 347) and lungs (see § 140).

Treatment.—Round worm: santonin 2-grain doses to a child of 3 years and upwards; for an adult, gr. 5. with calomel gr. 2 on three consecutive nights followed by magnesium sulphate ½ oz. in the morning. Oil of chenopodium (1½ c.c.), tetrachlorethylene (4 c.c.) or carbon tetrachloride (3 c.c.) are also specific remedies and are particularly effective where ancylostomiasis co-exists. Children infected with *threadworms* should wear drawers and gloves at night to prevent scratching; the nails should be cut

short ; a mercurial ointment may be applied to the anus. The hands should be scrubbed and the buttocks cleaned with antiseptic soap on rising and whenever there is any chance of their being soiled by contact with contaminated clothing, skin or excreta. Gentian violet gr. ½ t.i.d., a.c., may be given in enteric-coated tablets for 3 weeks. The migrating gravid females are best dealt with by enemata, given at night as soon as anal itching appears ; 4 oz. of hypertonic saline (1 oz. to 1 pint) or cold quassia are effective ; the injection should be given with a baby's Higginson's syringe, and courses are given until the disease is eradicated. In intractable cases a retention enema of hexylresorcinol (1 in 2,000) should be used on alternate days for 10 doses. *Whip-worm* infestations are treated with thymol and oil of chenopodium, by no means always successfully. *Strongyloides stercoralis* is treated with gentian violet given as an enteric coated pill (½ grain) thrice daily after food for 10 days. With the intestinal fluke, *Heterophyes heterophyes* tetrachlorethylene, beta-napthol or thymol is effective. The treatment of the *tapeworm* infestations has three stages : (1) starvation for 12 hours —overnight ; (2) the administration of an anthelmintic ; (3) purgation. (1) Give no food or drink for 12 hours : this empties the small intestine. (2) Next morning at 7 a.m. give extractum filicis liquidum 30 minims in gelatine capsules or emulsion ; this is repeated three or four times during the next hour ; in severe cases 30 minims of oil of turpentine may be given with the last dose. (3) Sodium or magnesium sulphate (½ oz.) is given at 10 a.m. and all the motions sieved against a black background to identify the head ; if this is not removed recurrence is inevitable. Castor oil must never be given with filix mas, as the active principle, filicic acid, is soluble in it and dangerous toxic effects result. Other anthelmintics include tetrachlorethylene, 4 c.c. for adults, followed by a saline purge in 3 hours, also kousso or pelletierine. Surgical treatment may be necessary in *hydatid* of the liver and lungs, and rarely for intestinal perforation or obstruction in ascaris infection.

§ 317. **Constipation** is insufficient action of the bowels, delay in the passage of the contents of the intestine, causing hard, dry fæces (scybala), independent of organic disease within or outside the intestinal canal. This source of fallacy must be carefully excluded before diagnosing a case as one of simple constipation. *Causes.*—(1) The usual cause is insufficient or incomplete movement of the mass of contents collected in the proximal colon. (2) In about a fourth of cases the delay is in the sigmoid colon and rectum (dyschezia). (3) A third form occurs when spasm of the colon prevents the mass movement from forwarding the contents through the region of the spasm (spastic constipation). (4) In elderly patients and those with feeble musculature there may be delay or absence of initiation of the mass movement. (5) A rarer type is due to lack of residue from too complete absorption of water and ingested food. A simple test for delay consists in giving a tablespoonful of powdered charcoal at night ; it should normally have completely disappeared from the stools before seventy-two hours.

The *Symptoms* which accompany or result from constipation are sufficiently familiar—at first headache, languor, and depression, followed by a furred, coated tongue, dyspepsia, sallow or pigmented skin, anæmia, sleeplessness, and eruptions, usually of an urticarial or erythematous nature. The temperature may rise a degree or so in certain conditions from temporary constipation, and even up to 102° F. The retention of hard fæcal masses may give rise to an alternating diarrhœa, which leads to error in diagnosis. Habitual constipation may give rise to hæmorrhoids,

and even to a distended ulcerated colon or atony of the colon. In women, in whom the condition is more common than in men, chronic constipation aggravates any pelvic disease. In both sexes varicose veins, œdema of the legs, sciatica, especially on the left side, and numbness of the legs may occur; these are more usually associated with diverticulitis (§ 321). In some cases there may ensue ptosis of part of the intestine, leading to delay of the bowel contents; then follows prolonged ill-health due to the toxæmia of INTESTINAL STASIS.

For purposes of treatment we may consider the Causes of simple or uncomplicated cases of constipation under three headings:

(*a*) **Errors of Diet.**

(i.) Too bland food—*e.g.*, no vègetables, no food with coarse residue.
(ii.) Too rough and irritating food, in certain cases of spasm.
(iii.) Too dry food—*e.g.*, deficient fluid ingesta.
(iv.) Too little or poor food, deficiency of vitamins.

(*b*) **Causes of Defective Peristalsis,** other than errors of diet.

(i.) Sedentary habits.
(ii.) Depressing emotions, anxiety, worry, etc., cause sympathetic inhibition, hence spasm, as in "spastic colon."
(iii.) Old age and other conditions with poor general tone, such as anæmia.
(iv.) Prolonged disregard of the calls of nature, with dilatation of rectum and pelvic colon consequent on blunted sensation.
(v.) Weak abdominal muscles.
(vi.) Atony of the colon, with or without colitis.
(vii.) Some febrile states.
(viii.) Endocrine disorder, especially deficient activity of thyroid and pituitary.
(ix.) Reflex spasm, as in catarrh of the colon or uterine disease.
(x.) Disease of brain or cord, such as tabes and cerebral tumour.
(xi.) Drugs, such as opium, iron, lead.

(*c*) **Deficiency of Bile,** or **Intestinal Secretions.**

(i.) Functional inactivity of the liver.
(ii.) Profuse vomiting.
(iii.) Excessive loss of fluid by skin or kidneys.
(iv.) Astringents, such as chalk or catechu. Hard waters also act in this way.

Diagnosis.—As chronic constipation may lead to the troublesome consequences mentioned above, we must first *find the cause*. With the patient lying down and the muscles well relaxed, examine the abdomen to see if the colon be distended or loaded; place one hand at the back, and press it forwards between the iliac crest and the last rib to meet the other hand, which is placed flat on the anterior abdominal wall. Make a rectal examination. An X-ray examination assists in deciding the presence or absence of mechanical obstruction, and the position of chief delay in the passage of the intestinal contents. Having excluded local causes by a thorough examination, we should consider the various causes above mentioned.

The *Treatment* of constipation comes under the following headings.

(1) *Dietetic.*—Increase the amount of fluid taken—*e.g.*, by sipping a tumbler of cold water slowly whilst dressing in the morning and undressing

at night. Avoid large quantities of milk or hard water. To provide bulk and stimulus, where there is no spasm, but chiefly dyschezia, foods to be eaten include oatmeal, wholemeal, National or brown bread, green and raw vegetables, onions, figs, prunes, and ripe fruits (see § 297. V). A teaspoonful or tablespoonful of salad-oil at mealtimes aids this diet. Where there is colonic spasm, give smooth food with little residue. (2) *Lubricants* may be used for short periods; paraffin, plain or in emulsion, bassorin, psyllium seeds and agar-agar preparations, provide bulk without irritating material, an important point in cases of spasm. (3) Inculcate *regular habits*. As stated above, in about one-fourth of the cases of simple constipation the delay is in the sigmoid and the upper rectum; hence it is important, even when there is no inclination to go to stool, that an attempt should be made at a regular hour daily, for 10 minutes by the watch, trying at regular intervals but being careful not to strain so hard as to cause pain in the abdomen or in the head. The squatting position aids, and lessens strain. If there is no result, a glycerine suppository should be inserted, and after waiting 10–15 minutes another effort made. That failing, a soap and water enema is given on the second day. Psychotherapy is useful in some cases. (4) *Active exercise* is advisable except when uterine or ovarian disease or colonic spasm is present; many systematic exercises are now taught which strengthen the abdominal and pelvic muscles. (5) *Electricity* is used in various ways; some forms relax spasm, others stimulate to healthier muscular action. Abdominal *massage* is useful; gently "rolling" the abdominal wall, or rolling a 7-pound shot-ball over the abdomen in the direction of the hands of the clock. (6) With signs of *endocrine deficiency*, as of the thyroid or pituitary, extracts of these glands greatly aid constipation. Bile extracts are efficacious in other cases.

(7) *Drugs*.—To avoid prolonged use of drugs give methodical trial of the measures above mentioned. For occasional constipation, senna with the evening meal and a seidlitz-powder in the morning are the most harmless. Calomel or other mercurial preparations should not be given habitually, but may be taken once a week for a few weeks. Phenolphthalein is an excellent preparation for temporary use. Cascara, aloes and senna may be used frequently. A useful vegetable pill is pil. colocynth co., pil. rhei co., āā gr. I., ext. hyoscyami, gr. ½; one or two at bedtime. Another good formula is tr. nuc. vom., tr. belladonna, āā ℥ 5; tr. hyoscyam. ℥ 10, ext. casc. sag. liq. ad ℥ 60. Nux vomica in small doses promotes peristalsis; belladonna is especially useful to relax spasm of the colon, and in simple dyschezia. Jalap, elaterium, scammony and gamboge are useful for drastic purgation. Salines given daily for some weeks will often re-establish the functions of a torpid intestine (F. 46). These may be given in the form of the mineral waters, such as Carlsbad, or their equivalents, which contain 20–60 grs. of sulphate of soda, sometimes with alkalies. They are best given on an empty stomach (F. 51 and 90 are also useful). An excellent aperient for children is cascara and malt mixed together in the proportion of 10 to 20 ℥ of the

ext. casc. sagrad. liq. to the teaspoonful of malt. (8) *Enemata* are used in conditions of atony of the descending and pelvic colon, and dilated rectum—1 or 2 pints of plain water may be given, at gradually longer intervals. Half an ounce of glycerine is an effective enema, but it should not be used longer than a few weeks, for it tends to irritate the rectum. In cases of very prolonged constipation, $\frac{1}{8}$ to $\frac{1}{4}$ pint of olive oil may be given every night. If this be injected very slowly, it is retained, and after a course of one or two weeks the bowel often resumes its functions. (9) *Lumbar sympathectomy*, including the removal of the presacral plexus, may succeed in very obstinate cases, where these measures fail, and where evacuations occur at long intervals or only with enemata.

Colon irrigation with normal saline is often necessary where hard masses can be felt in the cæcum. One or two pints at a time, at body temperature, are introduced slowly under a pressure of not more than two feet, and are immediately evacuated. This is repeated until the washing is returned clear. It is best preceded by injection of 3 fl. oz. of warm olive oil to be retained for a few hours. Carried out daily for a week, then on alternate days, and later once a week, this is very effective in clearing the colon of accumulated fæces. Gradually the bowel resumes its normal functioning. The only type of case in which this is not very satisfactory is that in which there is considerable ptosis and as a result the whole of the saline is not evacuated at once. The repeated calls to stool are annoying, and frequently this type of patient complains of depression and increase of toxic symptoms.

Hirschsprung's Disease (megacolon) is a condition of atony and dilatation of the colon of congenital origin: it is ten times more frequent in boys than girls. The cause is developmental or an abnormality of the neuro-muscular mechanism of the bowel due to overaction of the sympathetic nervous system. There are two types—(*a*) pelvi-rectal and (*b*) anal.

Symptoms.—There is obstinate constipation from the first weeks of life, and subsequently gross abdominal distension, tympanites with visible peristalsis, auto-intoxication and emaciation. If early childhood is survived, complications such as peritonitis and intestinal obstruction may ensue. The disease is often fatal in the absence of treatment.

The *Diagnosis* can be made only by the history and obvious signs of a distended colon. A barium enema shows enormously dilated and redundant bowel. A similar condition may be acquired by prolonged bad habits.

Treatment.—Attend to the diet and try the effect of liquid paraffin and large enemata combined with prostigmin 5–10 mgm. t.i.d.: otherwise regular dilatation of the anus with the fingers or an obturator. Spinal anæsthesia—which may have to be repeated—has given promising results. Lumbar sympathectomy, with removal on both sides of the greater and lesser splanchnic nerves, the first four lumbar sympathetic ganglia, and the pre-aortic plexus has also helped, but must be performed before permanent damage has been done.

§ 318. Intestinal Flatulence may be due to fermentation of carbohydrates, especially in the colon, but more often to interference with the absorption of air which has been swallowed with food or drink. Anything which causes congestion of the intestinal veins will delay the absorption of the intestinal gases, and give rise to distension and flatulence, such as heart failure and pulmonary disease, portal congestion and local venous block, as in volvulus and intestinal obstruction.

Symptoms.—There is a sense of fullness with discomfort which may be painful, relief being obtained by loosening the clothing, eructation

or passing flatus. Breathing may become embarrassed and palpitation or irregularity of the heart occur.

Treatment consists in reducing the vegetables and cellulose of the diet, and giving charcoal biscuits and carminatives. When constipation is present, Gregory's powder is most useful. Pituitary extract or prostigmin may be used, by injection: to relieve spasm, give trasentin, tablets of phenobarbitone, or atropine.

The patient complains of SUDDEN STOPPAGE OF THE BOWELS *with inability to pass even flatus,* ABDOMINAL PAIN, *and* VOMITING *which gradually becomes stercoraceous; there is increasing abdominal distension, and a tendency to* SHOCK. The case is one of ACUTE INTESTINAL OBSTRUCTION.

§ 319. Acute Intestinal Obstruction is one of the most serious medical or surgical emergencies.

The *Symptoms* common to all forms of acute obstruction are—(1) complete constipation, not even flatus being passed. Absolute constipation can be assumed only when flatus cannot be passed even after repeated enemata. (2) Pain may be acute at first, and referred to the umbilicus, though later it may be superseded by colicky pains, owing to the peristalsis of the bowel trying to overcome the obstruction. There is not usually much tenderness. (3) Vomiting is a prominent symptom from the onset. It is copious and projectile, first of food, then bile, and later material which is alkaline to litmus and finally fæcal. It comes on earlier, is more urgent, and becomes more rapidly stercoraceous in proportion as the obstruction has taken place high up in the intestines. (4) Abdominal distension is generally present; it is more in the flanks with obstruction to the colon, and more central with obstruction to the small intestine. (5) Peristalsis may be visible. (6) Constitutional symptoms gradually supervene, with prostration and a thready, *rapid pulse.* These also are more urgent when the small intestine is involved. The urine is diminished in proportion as the obstruction is near the stomach, for then the vomiting is more urgent. (7) Tetany can occur in high obstruction of the small intestine.

Diagnosis of Acute Intestinal Obstruction.—When summoned to a case presenting these three symptoms—stoppage of the bowels, vomiting, and acute abdominal pain—the first step is to identify the case as one of acute obstruction. In *colic* (renal, hepatic, or intestinal) all of these three symptoms may be present, but the patient's general condition is not so serious. Moreover, the position of the pain in renal and hepatic colic is characteristic (see § 246). In *acute peritonitis* there is great tenderness over the abdomen, thoracic respiration, and some fever (see also § 244). But when there is *perforation* into the *peritoneum* shock is present, at first without fever, and perforation is diagnosed with difficulty only by (i.) the pain is constant and there is local tenderness; (ii.) the passage of wind by the bowel; (iii.) the collapse being much greater even than that in acute obstruction; and (iv.) a possible history of the condition which has resulted in perforation or rupture (consult also § 243). It is

sometimes impossible to diagnose between these two conditions, and an exploratory operation should be undertaken without delay.

Causes of Intestinal Obstruction.—It is of some importance to ascertain the cause, for the prognosis and treatment differ somewhat in each case. (*a*) In *acute* intestinal obstruction, in which the symptoms come on *suddenly* in a person previously healthy, there are four *common* causes: (I) External hernia; (II) intussusception; (III) internal strangulation; (IV) paralytic ileus. (*b*) Sometimes, however, acute will supervene on chronic obstruction, and the *common causes of chronic obstruction* (§ 320) are four in number: (I) Malignant stricture of the bowel; (II) simple stricture; (III) pressure of a tumour; and (IV) diverticulitis.

Features special to the several causes of acute intestinal obstruction.

I. External Hernia is known by the presence of a tumour in the femoral, inguinal, or umbilical region. No impulse on coughing is present. Obturator hernia is very rare, and is usually only discovered at operation.

II. Acute Intussusception, or invagination of the bowel, is the commonest cause in childhood. True intussusception is always from the bowel above into the part below, and in more than half of the cases the lower part of the ileum becomes invaginated into the cæcum. In a third of the cases some other part of the ileum, and in about one-eighth some part of the colon, is implicated. The invaginated portion slowly sloughs, the two edges may be welded together, the slough may pass about the eighth or tenth day; thus spontaneous recovery may occur, though this is rare. Death from perforation and collapse is more usual unless the case is dealt with surgically. Intussusception is known by (i.) severe tenesmus; (ii.) a rectal discharge of *blood and mucus* with a red jelly appearance; (iii.) a sausage-shaped tumour may be felt, altering in position, on palpating the abdomen, and in extreme cases the invaginated portion of bowel is felt *per rectum*; and (iv.) the patient is usually a previously healthy boy under two years of age.

III. Internal Hernia or Strangulation—*e.g.*, by bands of adhesion—is known by (i.) the urgency of the symptoms; (ii.) the patient is an adult, with (iii.) a history of old peritonitis or previous operation. Volvulus (or twisting of the bowel) may be indistinguishable from the preceding—indeed, it practically results in strangulation—but (i.) it occurs in men over forty, usually with a history of chronic constipation; (ii.) abdominal distension may be great; (iii.) sometimes a tumour is felt over the sigmoid flexure, the usual site of volvulus.

Internal strangulation may also arise from (1) adhesion of the end of the appendix vermiformis through which a knuckle of the bowel gets nipped. (2) Adhesions of the bowel. (3) Congenital deficiencies in the mesentery or bowel, or the foramen of Winslow.

IV. **Paralytic ileus** is one of the most dreaded complications following a surgical operation. It may complicate pneumonia. It may be defined as a condition of "intestinal inertia," in which the intestine is incapable of muscular action, and becomes distended with gas. Its pathology is

not settled; it may be caused by injury to the wall of the gut, by interference with its blood supply or the nervous visceral connections.

Symptoms.—A mild form is met with after any major abdominal operation, and is manifested by constipation, gas and windy spasms; there may be some contraction of the sphincters. This occurs after the first 24 hours, with moderate distension, usually relieved at the end of the 3rd day by an enema. True paralytic ileus sets in about the 2nd or 3rd day, with pain, vomiting, distension and absolute constipation. The pain is a dull ache, not colicky, and there is complete cessation of peristalsis. Vomiting is persistent and copious. It may last 3 to 4 days and then clear up, but if unrelieved death takes place about the 3rd, 4th or 5th day.

It must be *diagnosed* from mechanical adhesive obstruction. The latter occurs later, from the 3rd to the 7th day. It has the same insidious onset; vomiting becomes progressive, and the material is alkaline to litmus, but there is usually colicky pain and peristalsis may be detected.

Treatment.—At the end of 24 hours ox-bile or turpentine enemata should be given and a flatus tube left in. Nothing should be given by mouth; the stomach should be kept empty by continuous aspiration, and the fluid intake kept up by a continuous glucose-saline infusion intravenously. Heat to the abdomen and morphia (gr. $\frac{1}{12}$ – $\frac{1}{8}$ four-hourly for five or more doses) are beneficial. If these are unsuccessful, carbachol subcutaneously (0·25–0·50 mgm.) or prostigmin 1 c.c., repeated two or three times, and followed by a glycerine enema often helps. Should these methods fail, a spinal anæsthetic should be given and if no result follows, operation is necessary. Only in desperate cases should ileostomy be performed.

The **rarer causes** of acute obstruction are three in number:

V. Impaction in the Bowel of a large Gall-stone. A large gall-stone escapes from the gall-bladder by ulceration into the bowel. The obstruction is high up in the small intestine, and consequently (1) the pain and constitutional symptoms are of extreme severity, and of very sudden onset. (2) The patient is usually a woman at or beyond middle age. (3) There may be a history of biliary colic, and in all cases there is a history of localised peritonitis some weeks or months before the seizure. (4) The symptoms may intermit, from the stone shifting its position.

VI. Obstruction of the bowel may sometimes be due to an Extravasation of Blood into the coats of the intestine. It occurs only in purpura, hæmophilia, and other blood disorders. Such cases are recognised by evidences of hæmorrhage in other positions—melæna, epistaxis, purpura, or a history of urticaria or angioneurotic œdema.

VII. Among the still rarer causes of obstruction may be mentioned masses of round worms (Trousseau), impaction of too much cellulose, orange-peel, etc., hair-balls, concretions of ammonio-phosphate of magnesium (a frequent cause in horses, though rare in man), and other foreign bodies in the intestine.

Clinical Investigation and Diagnosis of the Cause of Obstruction.—If the case occurs in a child, and there is a history of sudden onset, it is almost certainly intussusception; in an old person suspect growth, impacted fæces, diverticulitis, or volvulus; in a young adult suspect strangulation or hernia. If the vomiting comes on early and is urgent,

it points to a tight constriction *high up* in the intestinal tract. So also after the onset of obstruction high up there may be a movement of the bowels. If the distension is chiefly in the centre of the abdomen, the obstruction is probably above the ileo-cæcal valve; if it is chiefly in the flanks, the obstruction is below the valve; if more in the right than in the left flank, the obstruction is probably in the splenic flexure.

When called to such a case, first examine for swelling in the positions of external herniæ. If the abdomen be distended, and presents visible waves of peristalsis, inquire as to the causes of chronic obstruction (*infra*), as the case is probably an acute supervening upon a chronic obstruction. Always *examine per rectum*, for in acute intussusception the invaginated part of the bowel may be felt *per rectum*, and there may be a discharge of blood and mucus; or a stricture or other cause of chronic obstruction may thus be discovered. Next inquire into the past history—*e.g.*, for peritonitis (as this is a cause of internal strangulation), or for appendicitis or hepatic colic. Then examine the abdomen by palpation and percussion for tumour or tenderness. If the abdomen is distended only on one side, the site of the obstruction may be localised.

Prognosis.—The prognosis of obstruction of the bowels is always very serious. Death occurs in the natural course either from (1) gangrene and rupture of the bowel, or (2) exhaustion and collapse. At the present day the prognosis almost entirely depends upon the *stage at which the case comes under notice*, and the treatment adopted. All the acute cases require early surgical interference, and a surgeon should be summoned at once. As regards the *Causes*, obstruction from a gall-stone is perhaps the most serious, then intussusception, then internal strangulation. Among the gradual causes, carcinoma of the bowel gives the gravest prognosis, and paralytic ileus the most favourable. Cases in which the obstruction is high up are less favourable than those in the large bowel.

Treatment.—Acute intestinal obstruction is one of those serious conditions that demand the resources of both a physician and a surgeon, who should jointly undertake the management of a case. The indications are (1) to ascertain the cause; (2) to endeavour to remove the obstruction; and (3) in the meantime to support the strength and relieve the pain by controlling the peristalsis upon which it depends. Enemata of soapy water to which olive oil, glycerine or oxgall is added may be given in all cases; purgatives by mouth should be avoided. Warmth is applied to the abdomen in the form of hot fomentations, turpentine, belladonna, or opium stupes. The question of the administration of opium is debated (see Appendicitis), but, generally speaking, for the relief of the pain opium may be given as soon as the diagnosis is certain. The diet should consist of fluids, such as iced milk, beef-tea, and stimulants, given in small quantities, and frequently.

In *external hernia*, after a warm bath, it is best to proceed at once to operation. In *intussusception* some mild cases have a tendency to

spontaneous recovery. Some surgeons recommend that an attempt should be made to reduce it by injections of warm saline or olive oil, but it is better to proceed at once to laparotomy. In *internal strangulation* or twisting it is best to operate without delay if an injection does not relieve and we are certain of the diagnosis. In cases of recovery without operation there has probably been a simple volvulus. But death almost always occurs in cases of internal strangulation if unrelieved. Manipulation, and inflating the bowel by means of bellows are dangerous procedures. In *impacted gall-stone*, the progress is so rapid towards a fatal issue that operation, if undertaken, must be immediate. The same remark applies to other foreign substances in the intestine.

The patient complains of CONSTIPATION *progressively increasing,* ABDOMINAL PAIN, *and from time to time* VOMITING; *there is general ill-health.* The case is one of CHRONIC INTESTINAL OBSTRUCTION.

§ 320. In **Chronic Intestinal Obstruction** (1) the abdominal pain is generalised, intermittent, and of increasing severity. (2) There is constipation, or a history of alternate constipation and diarrhœa culminating in complete stoppage; and (3) abdominal distension in most cases, and peristalsis in some, may be visible. The chief causes are four in number:

I. MALIGNANT STRICTURE by new growth in the wall of the bowel—*e.g.*, cancer. Its most common situations are the colon, especially the sigmoid flexure, and the rectum. This cause of obstruction may be recognised by (1) the presence of a tumour or stricture which may be felt on examination *per rectum*, and the distension of the abdomen being mostly in the flanks. When the tumour is situated higher up than the sigmoid flexure, it may generally be felt through the abdominal wall; and when situated in the sigmoid flexure, it may be inspected by a sigmoidoscope. (2) When the sigmoid flexure or rectum is affected, the illness is often preceded by sciatica on the left side. (3) There are cancerous cachexia, the age of the patient, and perhaps hæmorrhage, fœtid discharge and often ascites to aid in the diagnosis. (4) X-ray with barium meal and barium enema combined with air insufflation will show a filling defect (Fig. 82).

II. SIMPLE—*i.e.*, NON-MALIGNANT STRICTURE of the intestine may arise in consequence of amœbic dysentery, syphilitic, or other ulceration, either in the colon or in the rectum. An ulcer alone is capable of producing symptoms of obstruction. This cause is recognised by a previous history of dysentery (perhaps only a mild attack), and residence in a tropical climate; or a history of syphilis, with a rectal discharge. Syphilitic stricture is rare, except between the sigmoid flexure and the anus.

III. PRESSURE ON THE BOWEL by a tumour, tuberculous or other adhesions, or an enlargement of some viscus such as the uterus. This cause is recognised by the physical signs of tumour or enlargement respectively.

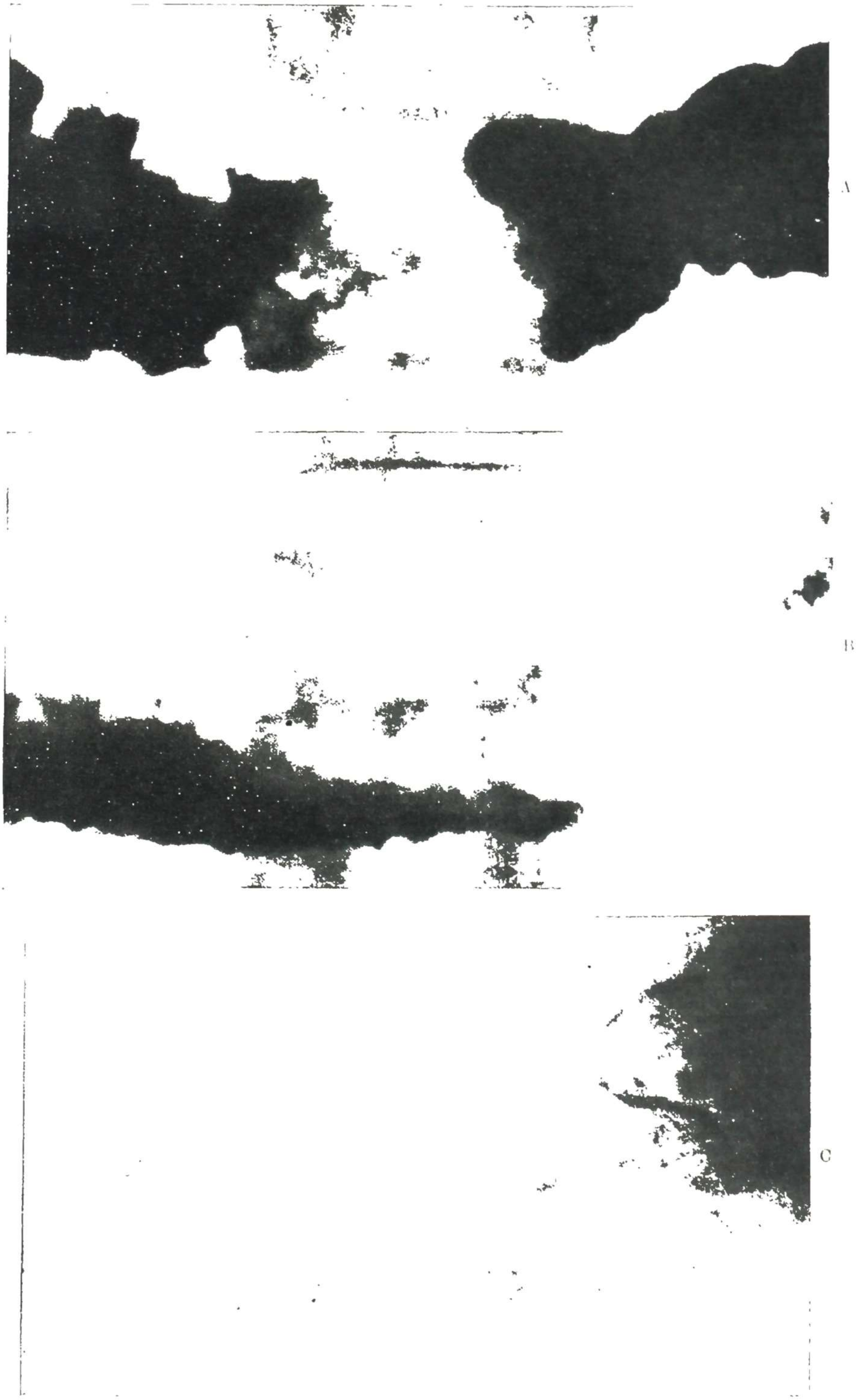

FIG. 82.—BARIUM ENEMA.—A. Carcinoma of transverse colon; could possibly be confused with a filling defect caused by pressure from spine. B. The strial picture. Compression brings the filling defect clear of the spine. C. Oblique view after air inflation, showing the absence of relaxation of the indurated part.

Rarer Causes of chronic intestinal obstruction are:

IV. CHRONIC PERITONITIS (§ 250) causes a matting together of the intestines, and intestinal obstruction may result. Cancerous peritonitis is attended by much pain and the effusion of much fluid; but in tuberculous peritonitis (§ 557) there are mostly adhesions, less pain, and less fluid. Localised peritonitis occurs as a result of diverticulitis, usually in the left lower abdomen.

V. CHRONIC INTUSSUSCEPTION is thus known: (1) It occurs usually in children; (2) tenesmus is present; (3) a tumour may be felt similar to that met with in acute intussusception; and (4) there is usually no great distension (see also Acute Intussusception, above).

VI. HIRSCHSPRUNG'S DISEASE (§ 317).

Prognosis of Chronic Intestinal Obstruction.—In all forms the symptoms of acute obstruction are apt at any time to supervene, from impaction of fæces above the narrowing lumen of the gut, but apart from this the prospect differs considerably in the various causes. A cancerous stricture is the most, diverticulitis the least, serious. Syphilitic stricture may be relieved by arsenic and iodides; dysenteric stricture is irremediable. The course of a tumour varies with its nature. Chronic intussusception *may* spontaneously resolve, the invaginated part sloughing off and being passed by the rectum, but the outlook is always grave.

Treatment of Chronic Intestinal Obstruction.—In most cases surgery is ultimately necessary, but at first the treatment consists in watching the patient until a diagnosis can be formed with as much accuracy as possible, giving digestible food, preferably such as leaves but little residue, and relieving pain by opium and external applications (hot fomentations with turpentine or opium). For simple *stricture of the rectum* gradual dilatation by bougies may be tried. In *chronic intussusception* operation is advisable. In *cancerous stricture* where radical removal is impossible life may be prolonged by colostomy; the longer the operation is delayed, the worse is the prognosis. It should never be delayed until vomiting has begun. Deep X-ray therapy sometimes aids.

A man of middle age or over who has suffered from FLATULENCE *and* IRREGULARITY OF THE BOWELS—*usually constipation—for some time, has an attack of* PAIN IN THE LEFT ILIAC FOSSA *with some fever; the disease may be* DIVERTICULITIS.

§ 321. **Diverticulitis** of the colon is not an uncommon complication of diverticulosis. Patients are usually above middle age and often obese. For some years there may have been irregularity of the bowels, with a tendency to constipation, and at intervals attacks of diarrhœa which are ascribed to indigestible foods. Sometimes there is mucus and occasionally even blood. There is (i.) some degree of fever and often leucocytosis; (ii.) tenderness and rigidity in the left iliac fossa; (iii.) sometimes a local mass is felt, due to simple inflammation or abscess formation; (iv.) irritation of the bladder with frequency of micturition; (v.) there may be occasional rigors; and (vi.) cystitis may accompany.

For *Diagnosis* of these cases, see also § 319. Diverticulitis has to be diagnosed from *cancer* of the colon and *appendicitis*. X-ray after a barium enema shows a contracted and irregular lumen of the bowel in the sigmoid region and diverticula scattered throughout the rest of the colon. Complications are: local peritonitis from peri-diverticulitis, perforation with general peritonitis, or obstruction.

Treatment: (*a*) during the acute attack, consists of bed, fluid and semi-solid diet, poultices or stupes to the abdomen, liquid paraffin by

FIG. 83.—DIVERTICULITIS OF SIGMOID.

mouth and by injection into the bowel. (*b*) Between the attacks, the diet should be fuller, but must contain nothing hard or indigestible; liquid paraffin should be taken by mouth two or three times a day. A course of intestinal douches with normal saline, given at low pressure, may be necessary, to clear away the accumulation of fæces and soothe the bowel. The rectal injection of 3 fl. oz. of warm olive oil two or three times a week, the patient lying on the left side and retaining it as long as possible, is of considerable value.

CHAPTER XII

THE LIVER

THE liver is the largest gland in the body and the fact that it can contain a fourth of the blood in the body shows its importance in the control of the circulation. All the blood passing from the stomach and intestines circulates through the liver, after which it joins the general circulation considerably altered in its composition. The pancreas and the liver work in close co-operation, the pancreatic internal secretion passing direct to the liver. The liver aids in preparing proteins, carbohydrates and fats for the tissues. Other important functions of the liver are: the manufacture and the storage of glycogen; the secretion of bile, containing bile salts and pigments; a detoxicating action by arresting poisons and bacteria absorbed from the intestinal tract, and converting certain noxious chemical substances—indol and skatol—into innocuous compounds; the elaboration of the products of protein metabolism into urea and uric acid, the storage of the anti-anæmic factor against pernicious anæmia, and the production of prothrombin and fibrinogen, both necessary for blood clotting. The Küpffer cells form part of the reticulo-endothelial system. The liver has considerable reserve and much power of regeneration.

PART A. SYMPTOMATOLOGY.

The symptoms due to disorders of the liver are not so clearly defined as those of cardiac or pulmonary diseases. The cardinal symptoms of *structural* disease of the liver are PAIN IN THE HEPATIC REGION, JAUNDICE, and a group of symptoms due to PORTAL OBSTRUCTION, which includes Ascites. When the liver cells become gradually destroyed, as in hepatitis, serious disturbance of the general health ensues, and in the later stages of that and of some other hepatic disorders LETHARGY passing into COMA supervenes. Functional derangement of the liver is attended by DEPRESSION and vague DIGESTIVE DISTURBANCES.

§ **325. Pain and Tenderness over the Liver** occur in PERIHEPATITIS and any other condition in which the capsule is involved or stretched, as in heart failure. The pain may radiate upwards towards the right scapula. The onset of pain in the course of a liver complaint may therefore be of considerable importance; for example, in hydatid of the liver the natural course of which is painless, it would point to a danger of rupture of the cyst. When the upper surface of the liver is involved, the pain is very often *referred to the right shoulder*; it is, indeed, a symptom of phrenic (diaphragmatic) irritation. The most severe form of pain, however, is that which occurs with the passage of GALL-STONES (*biliary colic*). Pain may be completely absent in hepatic disorder. There is,

however, in many cases of disease or enlargement of the liver a feeling of weight or fullness in the right hypochondrium, accompanied by an inability to lie on the left side.

Hepatic pain may be *simulated* by Pleurodynia (rheumatism of the intercostal muscles), Intercostal Neuralgia, Pleurisy, Dyspepsia and various gastric conditions, and by Intestinal or Renal Colic.

§ **326. Jaundice** is the term applied to the yellow pigmentation of the skin and other tissues due to the non-elimination of bile. It appears first in the blood then in the urine, in which increased urobilin, bile pigments and salts may be detected (§ 383), next in the conjunctivæ, then in the skin uniformly.

FALLACIES.—The yellow coloration of the conjunctivæ differentiates jaundice from all similar pigmentations of the skin. (1) Excess of *subconjunctival fat* may simulate jaundice, but this is readily distinguished by its unequal distribution. (2) The *sallowness* of the skin in anæmia is distinguished by the absence of bile in the urine and of yellowness of the conjunctivæ. (3) The *cachexia* of carcinoma, malaria, tuberculosis, and certain other forms of visceral disease, is differentiated in the same way. (4) The *bronzing* of the skin in Addison's disease is hardly likely to be mistaken for jaundice. (5) Long-continued mepacrine administration colours the skin yellow. (6) *Santonin* and *rhubarb*, administered internally, colour the urine, but do not give the reactions for bile in that fluid. (7) Carotinæmia (§ 653) may be mistaken.

Jaundice is classified as follows : (*a*) Obstructive ; (*b*) Toxic or infective ; and (*c*) Hæmolytic. Pure instances of these varieties are rare ; even with obstructive jaundice, damage to liver cells follows.

(*a*) Clinically, **Obstructive Jaundice** is distinguished by the colour of the stools, which are pale, slate or clay-coloured, due to the absence of bile in the intestinal canal. There is increased intestinal putrefaction and steatorrhœa, due to the increase of soaps and fatty acids in the stools. The urine is high-coloured and contains bile. The blood gives an immediate direct van den Bergh reaction (§ 331), the icteric index (§ 331) is raised to 10–15 ; the bile salts and cholesterol in the blood are also increased and the coagulation time is prolonged. Pruritus is severe. Leucocytosis is present in advanced obstructive jaundice, but not in mild cases unless accompanied by inflammation or suppuration.

Obstructive jaundice may be produced in four ways :

I. FOREIGN BODIES within the bile-duct, such as (1) gall-stones and inspissated bile ; (2) hydatids, round worms, Fasciola and other parasites ; (3) foreign bodies from the bowel.

II. INFLAMMATION of the bile-ducts, usually spreading from the duodenum.

III. STRICTURE, or obliteration of the duct owing to (1) congenital absence ; (2) perihepatitis ; (3) cicatrisation after ulcer of the duodenum ; (4) ulceration of the bile-duct, which may produce obstruction by the swelling around, or lead to stricture ; and (5) chronic pancreatitis.

IV. TUMOURS pressing on the duct, such as (1) cancer and other tumours of the liver ; (2) enlargement of the glands in the transverse

fissure of the liver; (3) tumours of the stomach, pancreas, kidney, great omentum; (4) fæcal masses in the intestines; and occasionally (5) tumours growing from the walls of the ducts.

(*b*) In **Toxic** or **Infective Jaundice** some bile usually reaches the intestine, so that the stools are not always clay-coloured, and severe damage to the liver may occur without marked jaundice. The blood in toxic jaundice gives an indirect or a biphasic van den Bergh reaction (§ 331). The urine contains excess urobilin, increased amino-acids and ammonium salts. Pruritus is not severe. This form of jaundice follows damage to the liver cells, which become inefficient, thus favouring retention of bile pigment and salts in the blood. The damage may be mild and recovery be complete, or severe, as in acute yellow atrophy of the liver, or partial recovery may follow, leaving some degree of cirrhosis. The causes of toxic jaundice are: (1) bacterial or protozoal poisons such as occur in infective hepatitis, pneumonia, syphilis, septicæmia, typhus, typhoid, relapsing fever, malaria, spirochætosis ictero-hæmorrhagica, yellow and other tropical fevers; (2) chemical poisons such as trinitrotoluol, tetrachlorethane (poisons affecting munition workers), phosphorus, arsenobenzol derivatives, nitrobenzene, cinchophen B.P. (atophan), dinitrophenol, ether and chloroform; (3) toxæmias as in pregnancy; (4) chronic heart disease with congestion.

(*c*) In **Hæmolytic Jaundice** (1) the fæces are of normal colour; usually there is no bilirubin or bile salts in the urine, but much urobilin or urobilinogen (§ 383). The spleen is usually enlarged and there may be perisplenitis. (2) There is a delayed direct and an indirect van den Bergh reaction. (3) This form of jaundice is caused by increased blood destruction and is of extrahepatic origin. Its causes are: (A) increased fragility of the red blood corpuscles, as in congenital or acquired acholuric jaundice; (B) increased destructive agents: (1) animal poisons, such as snake-venom; (2) streptococcal infections; (3) pernicious anæmia; (4) specific hæmolysis as in transfusion with incompatible donors. The icteric index is much increased (§ 331). Physiologically, this form occurs in the jaundice of the newly-born.

Of all these causes *gall-stones* and *infective hepatitis are the most common.*

To *diagnose* the type of jaundice (see Table XXI): 1. EXAMINE THE FÆCES, which are slate or clay-coloured in complete obstruction, and of normal colour in hæmolytic jaundice. The presence of fat or parasites may assist in diagnosing the cause. But it must be remembered, as possible fallacies, that the fæces may become stained if mixed with urine; and that the bile-duct may be only partially obstructed, and enough bile may thus escape to colour the fæces.

2. EXAMINE THE URINE for bile pigments and salts (§ 383).

3. Inquire as to the HISTORY OF THE ATTACK. Jaundice coming on suddenly, especially in a middle-aged woman previously in good health, almost invariably indicates obstruction by gall-stones. The intensity of the jaundice varies from week to week as the stones pass. Jaundice coming on slowly, and ultimately becoming intense, is generally due to a

tumour pressing on the common bile duct, and is most often seen in association with cancer. Severe jaundice persisting some weeks is almost certainly obstructive. Occupation in a munition factory or previous intravenous treatment with arsenobenzol derivatives, renders easy a diagnosis of the cause. A history of previous temporary attacks points in adult life to gall-stones; in youth, to infective hepatitis.

4. EXAMINE THE HEPATIC REGION CAREFULLY. If the liver is enlarged, cancer is the most probable cause; interstitial hepatitis is less common. If the gall-bladder is enlarged, cancer is more probable than stone. If ascites be present, the diagnosis rests between cancer and cirrhosis.

5. Inquire as to PAIN AND CONSTITUTIONAL SYMPTOMS. Pain of a spasmodic and severe character accompanies jaundice due to gall-stones and cancer. It is more constant and gnawing in character in congestion of the liver and catarrh of the bile-ducts. The temperature is not often

TABLE XXI.—DIFFERENTIATION OF TYPES OF JAUNDICE.

	Obstructive	*Toxic or Infective*	*Hæmolytic*
Onset	Stormy.	Quiet.	Chronic.
Colour of skin	Yellow, orange or greenish.	Yellow, orange or greenish.	Light yellow—"lemon yellow."
Distribution of pigment	Seen in conjunctivæ before skin.	Seen in skin before conjunctivæ.	Conjunctivæ rarely affected.
Irritation of skin	May be severe.	May be present.	Not present.
Colour of stools	Pale.	Normal or pale.	Normal.
Urine	Bile pigments present.	Often no bile pigments: urobilin present.	No bile pigments: urobilin present.
Liver	Large or very large.	Large, normal or small.	A little large or normal.
Gall bladder	May be palpable.	Not palpable.	Not palpable.
Spleen	Rarely palpable.	Rarely palpable.	Often palpable.
Anæmia	May or may not be present.	Usually not marked.	Severe.
Van den Bergh Reaction	Biphasic.	Biphasic or indirect.	Delayed direct or indirect.
Sedimentation rate	Usually normal.	Normal or prolonged.	Much increased.

elevated, but it may be so in infective hepatitis, jaundice due to poisons in the blood, pyæmic hepatitis, tuberculous affections, and local pus formation, such as liver abscess. Cerebral symptoms are rarely present, except when a fatal termination is at hand, unless the jaundice occurs in the course of pneumonia, fevers, or in acute yellow atrophy of the liver.

6. EXAMINE THE BLOOD with the van den Bergh test and estimate the icteric index (§ 331).

The *Prognosis* and *Treatment* of jaundice depend on its causal diseases (*q.v.*). The disappearance of bile pigment from the urine indicates that the attack is coming to an end, though it may be some weeks before the skin clears. Plenty of milk, preferably skimmed of excess cream and diluted or citrated, is the staple diet (§ 297, IV); extra amino-acids such as in casein digest or as methionine are advocated but are often nauseous. Fluids must be taken freely, and glucose and insulin given when the liver cells are damaged. The flatulent dyspepsia and many of the concurrent symptoms may be relieved by the administration of extract of

ox-gall (gr. 5 to 15) with meals, together with alkalies and carminatives after meals (formula 66). Calomel is a suitable purgative, followed by salts in the morning. Ammonium chloride, gr. 5 to 15 well-diluted, three times daily is used for the associated ascites, or, better still, injections of mersalyl, 1–2 c.c. The itching of jaundice is often a most troublesome symptom, but it can generally be relieved by pilocarpine or atropine, by calcium salts, or better, by ergotamine tartrate 1 mgm. t.i.d., or by sodium thiosulphate in doses of 10 gr. in saline intravenously; local treatment with alkaline lotions or bran baths, or bathing in potassium permanganate 40–60 grs. to 30 gallons of water, is beneficial. Phenobarbitone helps. Vitamin K and blood transfusion are given for bleeding. Great care is needed to secure efficient sterilisation of needles and syringes after use with jaundiced patients (§ 918).

By the time jaundice appears in a MUNITION WORKER the condition is serious. Symptoms of acute toxæmia may develop, with delirium, coma and death. Prophylaxis consists in strict cleanliness of hands and food, abundance of milk and glucose, and intermission of work in the trinitrotoluol department.

§ 327. **Icterus Neonatorum** is a mild transitory form of jaundice which affects a very large number (estimated by various observers at from 70 to 90 per cent.) of new-born infants. It appears usually on the second or third day of life, is not generally very intense, and rarely lasts longer than one or two weeks. The fæces are normal in colour, and apart from the jaundice the infant presents no other symptoms. The condition is almost certainly due to increased hæmolysis of the red cells. No *treatment* is required.

Some cases of jaundice in the new-born have a much graver prognosis. (1) Icterus Gravis Neonatorum is a severe form which affects several members of a family, and if untreated is fatal in 50–75 per cent. of cases: usually the first and second members of a family are exempt. It is recognised by (i.) being present at birth or within the first 24 hours after birth: (ii.) the accompanying severe hypochromic anæmia, in which nucleated red cells are excessive and persist for 3–4 weeks (erythroblastæmia): (iii.) purpura may develop: (iv.) there is enlargement of the liver and spleen. In cases which recover, damage to the brain may later cause spasticity, athetosis or mental defects (Kernicterus). Recent work shows this condition to be due to the newly-born child having an Rh factor present in its blood, the mother being Rh negative, but forming hæmolysins which destroy the infant's red cells (see §§ 537, 551, V). *Treatment* consists in giving repeated blood transfusions and injections of vitamin K. (2) Congenital syphilis, acting by stricture of the bile-duct or otherwise; or (3) congenital absence of the ducts. Both are usually fatal in a few months: in a small number of cases of congenital atresia, anastomosis of the common bile duct to the stomach or duodenum has been successfully performed. It should be remembered that jaundice in the new-born may be due to (4) sepsis. In stenosis of the ducts the stools are colourless; with sepsis there will be other symptoms.

§ 328. **Acholuric Jaundice.**—There may be no *symptoms*; it is a notable point in connection with the disease that the patients are often able to go about their work as if they were not the subjects of any abnormality. Symptoms when present are jaundice, weakness, a degree of anæmia, and splenomegaly, which may be extreme. These are liable to exacerbations in which the jaundice grows deeper, the anæmia and weakness more profound, and the general malaise may be associated with fever and perhaps vomiting. These attacks seem to be especially determined by an acute infection or by cold. Hæmorrhages from the gums or stomach or into the retina, gall-stones and intractable ulceration of the legs are rarer symptoms. The blood changes are characteristic: the red cells, usually 3–4 millions, are small in diameter

but more globular (spherocytes) and are abnormally fragile—a point which clinches the diagnosis (§ 533). A constant high reticulocyte count (10–50%) is also characteristic. The blood also contains an excess of bilirubin, whereas the urine contains only urobilin. The van den Bergh test (§ 331) shows a strong indirect reaction. The colour of the fæces is normal.

The *Etiology* is not known. The disease may be congenital or acquired. The former occurs in families, and may be transmitted by affected members of either sex. The acquired form is more severe, and spherocytosis and increased fragility are less constant.

The *Prognosis* of the congenital form is good as regards life, though cure is not to be expected. Death may occur during the hæmolytic crises or from biliary tract complications. In the acquired form the prognosis is much worse.

Treatment.—It is important to avoid cold and exposure. In the familial form splenectomy has now been proved successful and alone holds out a prospect of permanent cure. The chief indications for it in the case of a patient hitherto at work are (*a*) increasing and disabling anæmia or debility, or (*b*) frequent or excessive pain. Increased fragility and spherocytosis may persist after the operation.

PART B. PHYSICAL EXAMINATION

The liver lies chiefly in the right hypochondrium; the left lobe extends across the epigastrium above the stomach (Figs. 67, 84).

The gall-bladder is dealt with on p. 435. See Figs. 87 and 88.

The routine methods of examination of the liver consist of INSPECTION, PALPATION, and PERCUSSION. Examination of the urine and fæces and hepatic EFFICIENCY TESTS are necessary in many cases. X-ray examination may assist in the diagnosis of obscure cases—*e.g.*, hydatid and abscess.

§ 329. Inspection locally teaches us little, as a rule, unless the symmetry of the abdomen, as observed from the foot of the bed, be altered. However, the presence or the absence of *jaundice* should always be noted. If slight, it may be seen only in the conjunctivæ and urine and on observing by daylight. Deficient expansion of the lower chest is noticed with inflammatory disease of the liver. The lower edge of an enlarged liver may be seen moving with respiration. Note also if there are multiple small *telangiectases* on the skin generally, or dilated venules and capillaries on the face or enlargement of the veins of the abdominal wall, such as occur with cirrhosis and portal obstruction.

Palpation.—All the directions given in § 240 for the palpation of the abdomen must be followed when palpating the liver. The knees should be drawn up and the shoulders supported. Standing on the right side of the patient, place the palmar surface of the hands, previously warmed, on the right side of the abdomen, immediately above the iliac crest, pressing firmly yet gently inwards. The pads of the fingers should be inclined slightly upwards and inwards towards the median line, and should be pressed firmly down, working little by little upwards towards the costal margin. In this way the pads of the fingers, always held perfectly flat, will come in contact with the margin of the organ if it be enlarged. But if it is not enlarged, the liver can only be felt below the xiphisternum, for

laterally it lies altogether beneath the costal margin in the adult. In young children the liver is proportionately larger, and the lower edge normally protrudes beneath the costal margin. If the liver is enlarged, try to feel its surface and consistency by gently dipping the fingers down. Notice if its surface is irregular, smooth (as in fatty liver), or simply rough ("hobnail"), and if it is tender. Umbilicated nodules may be felt in cancer of the liver. When there is fluid in the peritoneal cavity, the method of "dipping" the fingers (suddenly) is also useful; anything but gentle use of the finger tips only excites contraction of the abdominal muscles, and so frustrates our object. Expansile pulsation of the whole liver is felt in cardiac disease with tricuspid regurgitation. The rectum should be examined in all cases of suspected liver disease. For examination of the *gall-bladder* see p. 435.

§ **330. Percussion** should be light so as to elicit only the superficial or absolute dulness of the organ. In percussing the upper margin, begin where there is a good lung note above, and percuss down from rib to rib in the nipple, mid-axillary, and scapular lines. Then repeat the process from space to space. In defining the lower edge, still lighter percussion

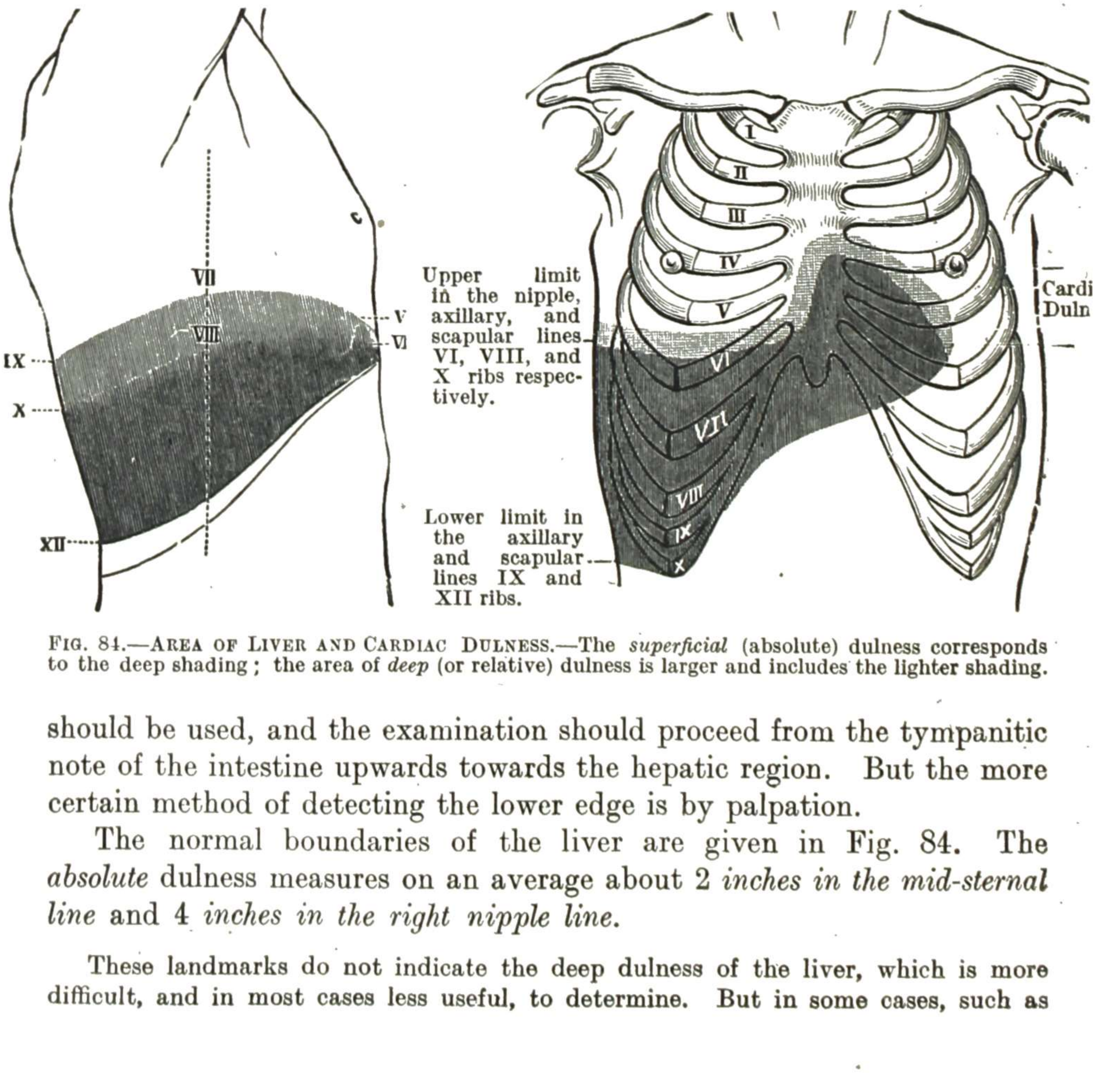

FIG. 84.—AREA OF LIVER AND CARDIAC DULNESS.—The *superficial* (absolute) dulness corresponds to the deep shading; the area of *deep* (or relative) dulness is larger and includes the lighter shading.

should be used, and the examination should proceed from the tympanitic note of the intestine upwards towards the hepatic region. But the more certain method of detecting the lower edge is by palpation.

The normal boundaries of the liver are given in Fig. 84. The *absolute* dulness measures on an average about 2 *inches in the mid-sternal line* and 4 *inches in the right nipple line.*

These landmarks do not indicate the deep dulness of the liver, which is more difficult, and in most cases less useful, to determine. But in some cases, such as

abscess or hydatid, it is desirable to make out the deep (or *relative*) dulness of the liver by heavy percussion. X-ray for diagnosis is not always available.

FALLACIES.—The physician should never feel satisfied with mapping out the liver once only, because the organ may be temporarily affected by many varying conditions, and the *percussion* boundaries by no means always give us a true index. Thus the lower edge may be masked by the dulness of the stomach after a full meal, by an accumulation of fæces in the colon, by a thickened omentum, by great rigidity of the muscles or œdema of the abdominal walls.

Apparent *diminution* of the liver may arise from (i.) distension of the stomach or intestines with gas; (ii.) by contractions of Glisson's capsule, especially on the under surface, giving rise to puckering or distortion of shape anteriorly; or (iii.) emphysema of the lungs, which obscures the *upper* border very much. Great diminution or absolute loss of the liver dulness, owing to gas in the peritoneal cavity, is a diagnostic feature of perforation of the stomach or intestine.

Apparent *enlargement*, when attention is paid solely to the lower edge of the organ, may be due to a *displacement* of the liver downwards by (i.) pleural effusion, empyema, or pneumothorax; (ii.) intrathoracic tumours; or (iii.) enlargement of the heart or hydro-pericardium. These and other fallacies may arise from paying attention solely to the *lower edge* of the organ; and, finally, the liver may in rare cases be dropped or "*floating*." "Riedel's lobe" is mentioned under Abdominal Tumours. Tumour or enlargement of the *gall-bladder* may be percussed as a dulness extending down from the liver towards the umbilicus.

Fluid in the Peritoneum (Ascites) is a frequent accompaniment of some hepatic disorders, and its presence or its absence must always be carefully noted. The methods of investigating Ascites have already been given (§ 259 and § 260), in which PORTAL OBSTRUCTION is dealt with in full.

§ 331. Liver Function Tests. As stated in the introduction to this chapter, the liver has numerous functions and these may vary independently. There is a wide margin of reserve, so that 70% of the liver may be put out of action before signs of insufficiency occur. Some of the functions may be controlled quantitatively and thus the so-called liver function tests are made.

(1) Sugar Tests: According to the patient's weight, 30–50 G. of lævulose are given in water, and the blood sugar examined before the test, and at half, one and two hours after it. With a normal liver the blood sugar curve should show no rise above the fasting level greater than 30 mg. per 100 c.c.; any higher rise in blood sugar indicates liver insufficiency. The appearance of sugar in the urine is not so useful; lævulosuria and galactosuria, after taking these sugars, occur in 70–80 per cent. of cases of diffuse liver disease, but also in 10 per cent. of the normal. Diabetes mellitus invalidates the test.

(2) By means of the *van den Bergh test* small amounts of bile pigment (bilirubin) in the blood can be detected, even before any clinical sign of jaundice is evident.

For the VAN DEN BERGH TEST two solutions are required. (1) Concentrated HCl 15 c.c., sulphanilic acid 1 c.c., distilled water 1000 c.c. (2) Sod. nitrite 0·5 G., distilled water 100 c.c. Mix 25 c.c. of (1) with 0·75 c.c. of (2). For the *direct reaction*, 1 c.c. of blood serum is mixed with 1 c.c. reagent. A blue violet colour begins at once and attains its maximum in 10 to 30 seconds. The colour change may be delayed for 1–15 minutes or even longer. It may be reddish at first, slowly or rapidly changing to blue (*biphasic*).

The *indirect reaction* is also tested with serum. Add 2½ c.c. of 96 per cent. alcohol and 1 c.c. saturated ammonium sulphate and centrifugalise. A violet red hue results, rapidly attaining its maximum.

For the *quantitative estimation* of bilirubin, the colour reaction is compared in a colorimeter with a standard solution of cobalt sulphate in water or of methyl red in acetic acid : in normal persons the value is not above 0·5 mgms. (1 unit). It is of value in detecting latent jaundice, and in assessing the progress of a case of manifest jaundice. In the interpretation of the results, it is now recognised that neither the direct van den Bergh reaction nor its modifications enables the distinction to be made between hepatogenous jaundice and that due to obstruction. Even in hæmolytic jaundice the test may be unreliable owing to the presence of intercurrent damage to the liver cells.

(3) the *icteric index* is a measure of the yellow colour in the serum, which is mainly due to bilirubin. A solution of potassium chromate is used for comparison. The normal is between 4 and 6. Clinical jaundice gives values above 16, latent jaundice between 6 and 16. This test is not so reliable as the van den Bergh reaction.

(4) The excretory function of the liver may be tested by giving intravenous bilirubin, or rose Bengal, and noting the time of disappearance from the blood. Bromsulphthalein in doses of 2–5 mgm. per kilo of body weight is a reliable test.

(5) *The Quick (Hippuric Acid) Test :* The body contains no store of glycine, which is formed entirely by the liver. This is conjugated with benzoic acid to form hippuric acid. *Method.*—Sodium benzoate (5·9 G. in 30 c.c. water) is given by mouth as the bladder is emptied. The urine is collected at the end of four hours and the hippuric acid precipitated from it by conc. HCl, dried and weighed : normally 3·0 G. (as sodium benzoate) is excreted, the results being expressed as a percentage of this figure. An intravenous method may be used in cases of vomiting, 20 c.c. of 10 per cent. solution (2 G.) of sodium benzoate being injected intravenously.

(6) Changes in the *plasma proteins* are noted. In liver disease the albumin is diminished and the globulin increased, so that a diminution of the albumin-globulin ratio occurs : this is in proportion to the severity of the liver damage.

(7) *Serum alkaline phosphatase* is increased in obstructive jaundice ; flocculation tests in the serum are increased in non-obstructive jaundice.

(8) In all cases THE URINE SHOULD BE EXAMINED for bile, and sometimes for urea, leucin and tyrosin. The ammonia-urea ratio is an important test. One of the main functions of the liver is to convert ammonium salts into urea. If the liver function is inadequate, the formation of urea is decreased, and the resulting excess of ammonium salts and diminution of urea in the blood is reflected in the urine. The normal ratio of ammonium salts to urea is determined in a twenty-four hours' specimen of urine, and is 4 per cent. In liver insufficiency, this may rise to 30–40 per cent. This estimation is rapidly performed and is a useful index by which to gauge from day to day the effect of treatment.

X-RAY EXAMINATION may reveal abscess, tumour, irregularity, enlargement or diminution of the liver. Gall-stones are dealt with in § 353.

PART C. DISEASES OF THE LIVER

Routine Procedure.—FIRST : Ascertain *what is the patient's Leading Symptom.* The symptoms of disorder of the liver we discussed in Part A. —*e.g.*, gastric disturbance, pain (or a feeling of weight or discomfort in the hepatic region), and jaundice. If there be severe and paroxysmal pain, turn first to biliary colic (§ 353).

SECONDLY : Learn the *History* of the patient's illness, eliciting the

facts in chronological order, and in this way ascertain also whether the disease be *acute* or *chronic*.

THIRDLY: THE EXAMINATION OF THE LIVER must next be made. The routine method is given in §§ 329 and 330.

Ascertain: 1. Whether the liver is *enlarged*, locally or generally, or *diminished* (by abdominal palpation and percussion in the nipple line), and whether there is any *pain*, *tenderness*, or other abnormality; 2. Whether there is any *fluid* in the peritoneum; 3. If there is any *jaundice*. 4. Examine *the urine* for bile pigments, urates, etc. 5. In certain cases the *liver function tests* and *X-ray examination* must be carried out.

Classification.—For clinical purposes, diseases of the liver may be conveniently divided into ACUTE and CHRONIC Disorders.

If the illness is one of long standing, and has come on insidiously, the reader should turn to **Chronic Diseases of the Liver** (§ 340).

ACUTE DISEASES OF THE LIVER

If the illness has come on more or less suddenly, and is attended by considerable malaise or other constitutional symptoms, it is one of the **acute diseases of the liver,** probably: I. ACUTE OR SUBACUTE HEPATITIS: I*a*. INFECTIVE HEPATITIS; I*b*. ACUTE OR SUBACUTE YELLOW ATROPHY; I*c*. WEIL'S DISEASE. The less common acute diseases are: II. PERIHEPATITIS; III. ABSCESS; IV. ACTINOMYCOSIS; V. DISTOMIASIS.

I. *The patient, after a short spell of* MALAISE, VOMITING *and* DIARRHŒA, *becomes* JAUNDICED. *The disease is* ACUTE *or* SUBACUTE HEPATITIS, *and is due to interference with the function of the liver cells, due to bacterial, chemical or protozoal toxins. It may be mild* (CATARRHAL JAUNDICE), *or severe* (ACUTE YELLOW ATROPHY), *or due to* WEIL'S DISEASE.

§ **332.** I*a*. **Acute Infective Hepatitis**, previously known as *Catarrhal Jaundice*, occurs in sporadic or epidemic fashion, particularly in the autumn. It is milder in children than in adults, and one attack usually confers immunity.

Symptoms usually begin with a *pre-icteric stage*: (1) Anorexia is constant, and all solid food is refused for 2–3 days. (2) Nausea usually accompanies but vomiting is unusual. (3) Frontal headache, malaise and disinclination for any work are usually present. (4) A feeling of uneasiness or weight in the epigastrium is sometimes accompanied by actual pain. (5) Constipation is much more common than diarrhœa. (6) There is fever, sometimes only to 99°, more usually to 101° and occasionally higher for 3–4 days: this gradually settles to normal as the *icteric phase* is reached. (7) Jaundice occurs between 1 and 8 days from the start of the illness. It is accompanied by pale stools, bile-stained urine and often by pruritus. The depth of the jaundice is very variable, and so is its duration, which may be as short as a week, or may persist for even 2 months. (8) At the onset of jaundice the temperature usually settles to normal and the appetite simultaneously returns. (9) The liver is often enlarged and firm,

1–2 fingers' breadths below the right costal margin, and the spleen is usually palpable. (10) Occasionally skin rashes of macular, urticarial or even purpuric type occur. (11) The van den Bergh test gives at first a biphasic and later an indirect positive result. (12) Leucocytosis never occurs and a polymorph leucopenia is usual.

During the course of an epidemic, cases may reveal only the symptoms of the pre-icteric stage, and jaundice may never develop: even the serum bilirubin is not necessarily raised in such cases.

Etiology. Three related varieties are recognised. (1) *Primary infective hepatitis*, in which an agent (probably a virus), is transmitted from an infected person by droplet infection from the nose, and from the fæces and urine. The incubation period is usually 17–35 days, and more than one member of a family may be involved. (2) *Homologous serum jaundice* occurs when human serum containing an icterogenic agent is used for blood transfusion, or to convey immunity against measles, mumps or yellow fever. Convalescent serum from cases of measles and mumps, and pooled human serum used in the preparation of yellow fever vaccine, have produced liver damage, without or with jaundice, in a large number of subjects, after an incubation period of 56–239 days (average 101 days). This type of hepatitis has been transmitted from the serum and from the nose of persons in the pre-icteric and early icteric stages, to volunteers who were inoculated subcutaneously or intranasally. (3) *Post-arsphenamine jaundice* rarely occurs in the first two weeks after the initial dose, and this is probably chemical in origin. Much more often an icterogenic agent is transmitted by a syringe contaminated by blood from a previous patient, and produces hepatitis 12–19 weeks later: this agent is not readily destroyed when syringes are "sterilised" (§ 918), and can be transmitted to a third person by the subcutaneous inoculation of infected serum. Jaundice subsequent to the injection of gold salts and other chemicals may be of similar origin.

Whereas an attack of infective hepatitis provides almost complete immunity against a second attack, post-vaccinal yellow fever jaundice does not confer immunity against a subsequent attack of infective hepatitis, suggesting that infective hepatitis and homologous serum jaundice are due to related but separate agents: it is probable that the causes of homologous serum jaundice and post-arsphenamine jaundice are similar to one another, even if they are not identical.

Diagnosis. In *infective hepatitis*, pain is never severe and is often absent, bile is absent from the stools only for a short time, and they then contain bile but are pale, the spleen is often palpable and the gall-bladder is never enlarged: leucopenia is the rule. In *gall-stones* the onset is usually with severe biliary colic, jaundice is often deep and the stools persistently clay-coloured: the spleen is not palpable. *Cancer* occurs in middle-aged or elderly persons; jaundice is often insidious in onset and lasts for many months. Jaundice following acute infections and other abdominal inflammation may be due to *abscess of the liver*. *Post-arsphenamine jaundice*, and those varieties following *blood transfusions* and *human serum inoculations*, give a history of the cause.

Prognosis. Most cases clear up completely, but there is a danger of relapse: the malady usually terminates within 6–8 weeks. Rarely cirrhosis of the liver may ensue.

Treatment. The patient should be kept warm in bed for at least 2 weeks. Until appetite returns, the diet should consist mainly of milk and carbohydrates, avoiding irritating substances and fats. A large

amount of fluid should be drunk, and a rectal drip of saline may be given with 5 per cent. glucose. Insulin, 5 units, may be injected twice a day, and glucose taken by mouth. Methionine and casein digests do not benefit cases of infective hepatitis, but may modify attacks of post-arsphenamine jaundice. A brisk mercurial purge, followed by a saline twice a week, relieves the congestion of the intestines and the liver. To prevent relapse, patients should not be allowed to return to work until the indirect van den Bergh in the blood is under 1 unit.

§ **333.** I*b*. **Acute or Subacute Yellow Atrophy** (Severe Acute Hepatitis, Icterus Gravis) is a rare disease characterised by extensive necrosis of the liver cells, jaundice and cerebral symptoms, occurring especially in workers with trinitrotoluol, and usually ending fatally. Some cases are due to a severe form of infective hepatitis and presumably to a virus. A very severe form is associated with toxæmia of pregnancy.

Symptoms.—(i.) The premonitory symptoms may be slight, resembling infective hepatitis, and are associated with temporary enlargement of the liver. There is increasing tenderness over the liver. (ii.) In a few days or weeks severe symptoms set in, with deepening jaundice, headache, and delirium, and the patient passes into the typhoid state (cholæmia). (iii.) Hæmorrhages occur from the stomach, bowel, and kidney, and there may be petechiæ under the skin. (iv.) Fever is usually absent during the course of the illness, but at the end it may be high. (v.) With the onset of the severe symptoms the liver dulness begins to diminish rapidly. The spleen is usually enlarged. (vi.) The urine is characteristically altered, containing bile, albumen and blood, and showing diminished uric acid and urea, with increase of the ammonia coefficient and sometimes acetone. Leucin and tyrosin are sometimes found crystallising out on evaporating a few drops of urine (Fig. 99). (vii.) In the most severe cases, as in the toxæmias of pregnancy, early collapse, with tachycardia, prostration and death may take place before jaundice is apparent.

Diagnosis.—Acute Yellow Atrophy is not likely to be mistaken for any other liver disease after the acute symptoms set in. In phosphorus poisoning the liver is enlarged, and signs of irritant poisoning precede the onset of the jaundice.

Etiology.—Predisposing Causes.—(i.) Acute Yellow Atrophy is most common under middle age, though rare in children; and (ii.) in women, especially during pregnancy, often accompanying eclampsia. (iii.) Workers in trinitrotoluol and carbon tetrachloride. (iv.) Dissipation and excesses predispose. *Exciting Causes.*—(1) It may complicate fevers, such as typhoid fever, streptococcal infections, and influenza. It is found in (2) delayed chloroform poisoning and poisoning with phosphorus or cinchophen (atophan); and (3) in some cases of secondary syphilis. It is more frequent since the introduction of intensive treatment with arsenical preparations (see post-arsphenamine jaundice, § 332). (4) It may follow the passage of a gall-stone. (5) In a large number of cases no cause can be found, but a virus infection is suspected.

Prognosis.—The disease is often fatal. After the severe symptoms set in the patient may die in a comatose condition within a week. Pregnant women usually abort. Recovery may take place, followed by cirrhosis of the liver (interstitial hepatitis).

The *Treatment* is very unsatisfactory. During the preliminary stage the disease is treated as under infective hepatitis (§ 332). Warm baths, diaphoretics, rest, milk food, large doses of bicarbonate of soda and glucose, and diuretics may be tried. In all cases of syphilis under treatment with arsenic preparations intravenously, the urine and, if necessary, the blood should be watched carefully for the presence of bile pigments, and the treatment intermitted if they are found. Intravenous injections of sodium thiosulphate and the use of B.A.L. (p. 718) may be successful in cases due to arsenic. Insulin subcutaneously, combined with glucose by the mouth or intravenously, may help recovery.

§ **334.** I*c*. **Weil's Disease** (Synonyms: Spirochætal Jaundice, Spirochætosis Icterohæmorrhagica, Leptospirosis) has a sudden onset associated with fever, toxæmic

symptoms, and, in severer cases, with jaundice and renal involvement. The incubation period varies from 6 to 12 days. The onset is with rigor, headache and frequently vomiting, followed by backache, joint pains, tenderness of the muscles and severe prostration. Sore throat is common. The blood pressure is low, the tongue furred, the face flushed and the conjunctivæ injected. The temperature generally oscillates between 102° and 104° F. and then begins to fall by lysis: it is generally 7 to 14 days before the temperature is normal. Jaundice appears from the 4th to the 6th day in about 50 per cent. of cases and may be intense.

The stools are light or even clay coloured. The urine is scanty, contains bile pigment and bile salts, albumin and sometimes red blood corpuscles. The van den Bergh reaction often gives a direct biphasic reaction and bilirubinæmia is increased. The blood urea is raised to 60–300 mgm. per 100 c.c. A leucocytosis is the rule, varying from 12,000 to 30,000 per c.mm., with neutrophil polymorphonuclears increased to 80 per cent. Skin petechiæ, epistaxis, melæna and hæmorrhage from other mucous membranes are not infrequent, and herpes is common.

The outstanding feature of the physical examination is the extreme tenderness of the muscles, especially those of the legs, neck and abdomen. Abdominal rigidity may suggest an acute abdomen. The liver is generally enlarged and tender and splenomegaly may be present. Later, nocturnal delirium, a typhoid state, muscular twitchings and convulsions may develop and the patient die with anuria and uræmia. In other instances cholæmia, associated with increasing jaundice, hiccough, Cheyne-Stokes' respiration and coma, terminates the picture. Meningeal symptoms predominate in some cases; the cerebrospinal fluid then contains polymorph leucocytes, lymphocytes, increased quantities of globulin and sometimes also leptospiræ. In about 50 per cent. of cases jaundice does not appear and kidney involvement is slight; the fever may only last 2 to 4 days, and unless the occupation of the patient suggests the need of laboratory investigations the condition will be missed.

Diagnosis.—Features of importance include an occupational relationship to rats or submersion in infected water, profound prostration, extreme tenderness of the muscles, jaundice appearing about the 5th day, hæmorrhages, albuminuria and leucocytosis. At the onset, *meningitis*, later, *infective hepatitis*, may be suspected: the latter does not cause leucocytosis. In the tropics *yellow fever* and *relapsing fever* complicated by jaundice may prove confusing. During the first week leptospiræ can be demonstrated by blood cultures or by the intraperitoneal inoculation of blood into guinea-pigs. The agglutination test becomes positive in the second week and from the third week onwards leptospiræ can be isolated from the urine.

Etiology.—Rats are carriers of the disease, the causative organism—*Leptospira icterohæmorrhagiæ*—being passed in the urine and so infecting water and fungal slime. Human beings are infected during bathing or immersion accidents or through abrasions of the skin; hence canal workers, bargemen, rat catchers, coal miners, sewer workers, fish curers and farm workers are liable.

Treatment.—Anti-leptospiral serum (60 c.c. or more) prepared from immunised horses or 30 c.c. of serum obtained from convalescents should be administered intravenously as early as possible. Intravenous dextrose solution (5 per cent.) combined with insulin is of special value. Penicillin, 40,000 units three-hourly for 4 days, should be given as early as possible—leptospiræ are very sensitive to this drug.

Preventive treatment consists of rat destruction, avoidance of bathing in infected water and the protection of skin abrasions in workers whose occupation brings them in contact with rat-infected slime.

The less common **Acute Disorders** of the Liver remain to be considered, viz., PERIHEPATITIS and ABSCESS OF THE LIVER.

II. *The patient complains of* PAIN AND TENDERNESS *in the hepatic region, aggravated by movement. There is* NO JAUNDICE, *and other hepatic symptoms are absent.* The malady is probably PERIHEPATITIS.

§ 335. **Perihepatitis** is inflammation of the capsule of the liver, which becomes opaque and thickened (sugar-loaf liver), and by its contraction may lead to considerable distortion of the shape of the liver.

Symptoms.—(i.) Acute attacks usually set in suddenly, with pain in the hepatic region, radiating to the shoulder, and there is tenderness, increased on movement, pressure, or cough. (ii.) Fever is absent as a rule, and the patient may appear to be in his usual health. (iii.) Friction may be felt or heard. (iv.) Unless some other disease is present, there is no jaundice. Recurrent attacks lead to thickening of the capsule, recurring ascites, necessitating repeated tapping, and occasionally jaundice. The puckered liver, with its thickened, rounded, distorted edge, can sometimes be made out. The history of a *Cause*, especially *syphilis*, is usually obtainable. It is sometimes part of an inflammation of the liver itself, or is associated with an abscess, tumour, or cirrhosis. Sometimes the inflammation extends from adjacent organs, as in pericarditis, pleurisy, or gastric ulcer, or it may be part of a general peritonitis.

Diagnosis.—The characteristic pain and the absence of jaundice differentiate it from many other liver diseases. Other signs of syphilis aid diagnosis. Cases of gall-stones or gumma of the liver may at times be mistaken for perihepatitis.

Prognosis.—Simple cases tend to recover. In those which have lasted for a long time a certain amount of cirrhosis of the liver ensues. Portal obstruction may ultimately result from puckering at the fissure, and considerable distortion of the liver may result in the same way.

Treatment.—The diet must be spare, and the patient must be kept warm. Salines are given, with blue pill and rhubarb. Externally, hot fomentations and poultices give relief, and if the pain is severe, leeches are recommended. The cause when known must be treated—*e.g.*, syphilis (§ 552).

III. *There is* ENLARGEMENT *of the liver, accompanied by* PAIN *and tenderness, and the boundaries of the area of dulness are* IRREGULAR; *there are* SHIVERINGS, SWEATING, *and* INTERMITTENT PYREXIA. The disease is ABSCESS OF THE LIVER.

§ 336. **Abscess of the Liver.**—Solitary or multiple collections of pus may occur in the liver, due to septic infection, to suppuration of the bile channels (cholangitis), or portal vein (portal pyæmia), or more rarely to suppuration of pre-existing morbid conditions, such as hydatids or gummata. "Tropical" abscess occurs after amœbic infection of the colon, a common cause in the tropics; it is usually solitary, whilst pyæmic abscesses are generally multiple.

Symptoms.—(i.) The onset is usually *acute*, except in the tropical form, with pain and tenderness of the liver, accompanied perhaps by a dry cough, shallow respiration and digestive disturbance. The pain is affected by respiration, and may be worst when the patient lies on the left side. (ii.) The liver is enlarged, and the enlargement may extend downwards, or more often upwards, even to the nipple. There may be fluctuation. (iii.) Jaundice is rarely present. (iv.) Constitutional symptoms are severe. There is usually high fever, continuous at first, then with increasing oscillations. Rigors and sweats are common. Later, the patient falls into the typhoid state, with emaciation, vomiting, diarrhœa, and delirium.

Besides the acute type just described, there is a variety with an *insidious* onset. As amœbic hepatitis gradually develops into frank abscess formation, there is general failure of health, and periods of continuous, remitting or intermitting fever, sometimes followed by intervals of apyrexia. Cough and dull aching over the liver and in the right shoulder may be present from the beginning. *Amœbic abscess of the liver* generally affects the right half of the liver and is usually associated with physical signs at the base of the right lung; there may be a history of dysentery, while cysts of *Entamœba histolytica* may or may not be found in the fæces. Compensatory hypertrophy of the left half of the liver commonly occurs in destructive lesions implicating the right side.

Diagnosis.—(i.) The pain and pyrexia distinguish abscess from *hydatid* (when not in a suppurating condition). (ii.) A distended and *inflamed gall-bladder* may be palpable. Suppurative pylephlebitis, septic cholangitis, hepatic carcinoma and a breaking down gumma may simulate abscess. (iii.) Abscess is often mistaken for severe *malaria*. But malaria is amenable to quinine, the elevations of temperature are periodic, and each paroxysm has three stages. (iv.) Hepatic abscess may be diagnosed from other swellings of the liver by exploratory aspiration, revealing the chocolate "anchovy sauce" thick, tenacious pus. (v.) Physical signs suggestive of collapse or consolidation at the base of the right lung so frequently accompany liver abscess that their presence is an important aid to diagnosis. (vi.) X-ray shows upward enlargement of the liver (when the right lobe is involved), limited movement of the diaphragm, and sometimes local bulging due to a pointing abscess.

The insidious cases of liver abscess are always difficult to diagnose. No history of dysentery may be obtained and several examinations of the stools may be negative. In amœbic hepatitis there is often more fever than the total white count would suggest. With amœbic abscess the counts mostly range from 12,000 to 16,000 per c.mm. the polymorphonuclears 65 to 75 per cent. Where secondary streptococcal or staphylococcal infection supervenes, the leucocytes rise to 20,000–30,000 per c.mm. and the neutrophil polymorphonuclears equal 80–90 per cent. of the total cells; these counts also hold for pyæmic abscesses. Always suspect hepatic amœbiasis in a patient with obscure pyrexia coming from a tropical country.

Etiology.—Hepatic abscess, single or multiple, may arise from—(i.) Suppuration in a pre-existing focus of disease—*e.g.*, hydatid, gumma, tuberculous abscess, actinomycosis, or malignant growth; (ii.) ulceration of the biliary passages such as occurs in cholecystitis; (iii.) ulceration of the alimentary canal. The abscesses are usually multiple, except in amœbic dysentery, where often one large abscess, consisting of necrotic liver tissue which is sterile except for the presence of the amœbæ, dominates the picture; such an abscess may become secondarily infected with streptococci, etc. (iv.) Inflammation and pus-formation in the abdomen, especially in cases of old-standing suppuration of the pelvic organs and in appendicitis. (v.) Occasionally operations on the rectum or in any septic area produce an abscess in the liver, consequent on the conveyance of a septic embolus by the portal vein. (vi.) Portal pyæmia. (vii.) Trauma in a few cases.

Prognosis.—(1) The case mortality is high, except in tropical liver abscess. Death usually takes place in three weeks in cases with multiple abscesses. The pyrexia increases, and the patient dies in the typhoid state. The abscess may burst into the peritoneum, pericardium, or alimentary canal, with a fatal issue, or it may open externally and gradually heal by free discharge. Frequently the abscess, especially a "tropical" abscess, bursts into the right lung or the pleura. The patient develops a severe cough, with signs of consolidation of the right pulmonary base, and the abscess contents are brought up as a red-coloured sputum. Recovery may result, or the continued discharge may lead to death from exhaustion or lardaceous disease.

Treatment.—For multiple pyogenic abscesses, penicillin 50,000 units three-hourly subcutaneously and sulphadiazine 1 G. four-hourly by mouth should be tried. Where amœbic hepatitis (§ 517) or abscess is suspected give injections of emetine hydrochloride gr. i. daily for a period not exceeding 10 days. Absolute rest in bed is necessary. If the condition does not clear up, exploratory puncture of the liver should be made under local anæsthesia; if pus is discovered it should be aspirated and aspiration should be repeated several times if needed. Incision and drainage is performed only when secondary bacterial infection is present; penicillin and sulphadiazine should be given as well as emetine. If the stools still contain cysts, the treatment outlined for amœbiasis should be instituted (§ 308 (2)).

§ 337. IV. **Actinomycosis of the Liver** is a condition which may be mistaken for abscess of the liver. It is due to absorption of the ray fungus from the intestines, and starts as one or more foci in the liver substance, which slowly enlarge and may undergo suppuration.

The *Symptoms* consist of vague uneasiness referable to the liver, with gradually increasing enlargement—at first uniform, later unequal, the organ becoming prominent in one place. Exploration with a needle may yield no results; but if the tumour is laid open, the characteristic greenish fluid with yellow specks containing the ray fungus clinches the diagnosis. Actinomycosis appears to respond favourably to large doses of penicillin (Table XXX); otherwise maximal doses of potassium iodide (gr. 40–60 daily) should be given.

§ **338.** V. **Distomiasis of the Liver** is commonly found in the Far East, due to *Clonorchis sinensis*, while more rarely *Fasciola hepatica*—the sheep fluke—may affect man. In these diseases the bile ducts are invaded with flukes, leading to thickening and dilatation of the ducts and cirrhosis of the liver. Mild infections may be symptomless, but the more severe cases present anorexia, epigastric pain, hepatomegaly, diarrhœa, wasting, œdema, ascites and jaundice. Secondary bacterial infection may produce fatal cholangitis or liver abscess. The *Diagnosis* is made by finding the operculated eggs in the fæces, associated with eosinophilia.

Treatment.—Carbon tetrachloride 3 c.c. in a gelatine capsule is sometimes effective; and favourable reports have been recorded as to the value of emetine injections (1 gr. daily for 10 days). A course of antimony sodium tartrate or organic compounds of antimony may be given intravenously.

§ **339. Subphrenic Abscess.**—The *Symptoms* resemble those of tropical liver abscess. When occurring above the right lobe, the liver dulness is continued up in the axilla, perhaps as far as the level of the nipple, and is convex, or dome-shaped, upwards. The base of the right lung shows signs of congestion, and there are evidences of pleurisy at one or both bases.

Etiology.—The most common causes are appendicitis and ruptured peptic ulcer. Other causes are extension of hepatic abscess, empyema perforating the diaphragm, extension of pelvic abscess, and local tuberculous or (rarely) cancerous processes.

Diagnosis.—In a case of suspected abscess exploratory puncture may be performed, sometimes under general anæsthesia. The needle should not penetrate beyond 3½ inches, so as to avoid puncturing the portal vein. In a right-sided *empyema* of the chest the upper border of the dulness, when continuous with that of the liver, is concave, being higher towards the spine. In *hepatic abscess* the liver is tender and enlarged below the costal margin, but it is often impossible to distinguish subphrenic from hepatic abscess. A variety containing air so greatly resembles pneumothorax that it is called *pyopneumothorax subphrenicus.*

The *Prognosis* is fair if surgical treatment is carried out thoroughly and in time.

CHRONIC DISEASES OF THE LIVER

§ **340. Routine Procedure.**—It will be remembered (§ 329) in the physical examination of a patient suspected to be suffering from hepatic disease that the *first* and most important question to investigate is whether there is *any alteration in size*, especially enlargement *of the liver* (by palpation and percussion). (2) For reasons which will be apparent below, the question next in order of importance is whether there is any *pain or tenderness* in the organ. And then (3) is there any *jaundice?* (4) Is there any *ascites?* (5) In every case of suspected liver disease the spleen (§ 356), the stools, and the urine should be carefully examined.

The numerous *fallacies* in the alteration of the size of the liver dulness must be carefully studied (§ 330).

Classification.—Chronic diseases of the liver are divided into those in which the **size of the liver is unchanged** and those in which it is altered,

either **diminished or enlarged**; the latter again being divided according to the presence or absence of pain over the liver.

A. The organ is **Normal in size** in :—
Functional derangement of the liver .. .. .. § 341
B. The organ is **Diminished in size** in :—
Portal cirrhosis or Chronic hepatitis .. .. .. § 342
C. The organ is **Increased in size** :—

(*a*) WITHOUT PAIN OR TENDERNESS :
I. Hypertrophic cirrhosis (bacterial and toxic); I*a*. Biliary cirrhosis; I*b*. Cardiac valvular disease; I*c*. Chronic syphilitic disease; I*d*. Cirrhosis of biliary obstruction; I*e*. Tropical cirrhosis .. .. § 343
II. Fatty Liver .. .. § 344
III. Von Gierke's disease .. .. § 345
IV. Lardaceous liver .. .. § 346
V. Hydatid of liver and other rare conditions .. .. § 347

(*b*) WITH PAIN OR TENDERNESS :
I. Chronic congestion .. .. § 348
II. Cancer of liver .. .. § 349
III. Abscess of liver, tumours and other rare conditions occurring sometimes in acute form .. .. §§ 336, 350

A. *The liver is* **normal in size.** *The patient complains of* LETHARGY, *vague digestive disturbances, sleepiness after meals, furred indented tongue,* CONSTIPATION, *headaches, and there is a frequent deposit of* URATES IN THE URINE *on cooling.* There is probably FUNCTIONAL DERANGEMENT OF THE LIVER.

§ 341. Functional Derangement of the Liver.[1]—The liver is the largest gland in the body and has many functions, which may become deranged together or separately. Usually one or two functions are more severely affected. See liver tests (§ 331).

The common complaint, "My liver is sluggish," is often equivalent to saying that the bowels do not act properly, but in some cases other parts of the digestive system may be at fault. The causes of this complaint may be temporary or continuous. Help may be obtained by consulting the following classification.

I. TEMPORARY :
Acute dyspepsia, "bilious attack" (§ 281).
Migraine (§ 696).
Onset of colds, febricula.
Excess of food, alcohol, or exertion (§ 282).

Errors of diet, especially rich, sweet, greasy foods, and alcoholic beverages, *i.e.*, indigestible and excessive food rather than food with

[1] The introductory remarks at the head of this chapter may be referred to in this connection.

purin bodies. Alcohol *combined with sugar* (*e.g.*, port and other fruity wines) is especially injurious; or taken in the form of undiluted spirit, *e.g.* cocktails, particularly on an empty stomach, is more harmful than dilute alcohol at meal-times.

II. CONTINUOUS:

Constipation (§ 317).	Gastritis (§ 284).
Cholecystitis (§ 354).	Colitis (§§ 307, 310).
Chronic appendicitis (§ 249).	Acidosis (§ 384).

B. *The liver is* **diminished in size**; *if the surface can be felt it is* HARD AND UNEVEN (*hobnail*); ASCITES *is probably present, but no very distinct jaundice; the patient is subject to* HÆMORRHOIDS, *and* HÆMORRHAGES *from the stomach and bowel.* The disease is PORTAL CIRRHOSIS (CHRONIC INTERSTITIAL HEPATITIS).

§ 342. Portal Cirrhosis of the Liver (Alcoholic Cirrhosis or Interstitial Fibrosis of the Liver) consists of a chronic hepatitis, with an increase of the interstitial fibrous tissue, leading to portal obstruction, and shrinkage of the organ. In the earlier stages a degree of fatty degeneration may give rise to considerable enlargement (§ 344), but later, with the formation of fibrous tissue, atrophy ensues. It is most common in men between 35 and 60 years old.

Symptoms.—(1) In the early stage of the disease the organ may be enlarged, though rarely much so; but in the second and third stages the liver dulness is diminished. The liver is small and hard, and the surface is often nodulated, hence it is known as the "hobnail," or "gin-drinkers'" liver. There is a feeling of uneasiness and weight in the hepatic region. (2) The onset is slow and insidious, extending sometimes over years. Gastric symptoms, such as capricious appetite, *morning sickness*, and other symptoms of alcoholic dyspepsia, are often complained of for a considerable time, together with nervous disturbances, such as drowsiness, muscle pains, especially in the neck, and headache after meals. These symptoms of chronic gastritis are followed by debility and emaciation. The patient's aspect is very characteristic, with dilated venules and capillaries in the cheeks. (3) Jaundice appears in the later stages of the malady in about one out of three cases. (4) Symptoms of portal obstruction occur (§ 260), and hæmatemesis is sometimes the first obvious symptom; the spleen becomes slightly enlarged, and ascites (which is present in 80 per cent. of the cases) may be very considerable in amount: when the ascites recurs after paracentesis, chronic peritonitis has probably supervened. (5) There is an increased tendency to hæmorrhage. (6) In the concluding stages of this disease, when the secreting tissue of the liver is destroyed, the patient falls into a comatose state, with muttering delirium (cholæmia), which resembles uræmia and the typhoid state, except that there is considerable pyrexia in the latter. This precise clinical resemblance is quite in keeping with the fact that the liver takes

part in the elaboration of urea, so that when its cells are destroyed the blood becomes charged with a number of nitrogenous products, which cannot be eliminated.

Etiology.—(1) Cirrhosis of the liver is due to chronic intoxication by poisons which are usually of exogenous origin. It affects men more often than women. (2) Alcohol undoubtedly predisposes to atrophic cirrhosis, especially when taken frequently in small quantities, or when taken *neat on an empty stomach*, the patient perhaps never becoming intoxicated. (3) Cirrhosis can occur in those who have never consumed alcohol: dietetic factors may play a part, and in Southern India toxic products from tapioca have been suspected. Syphilis, malaria, bilharzia and many bacterial infections may predispose. (4) In poisoning by T.N.T., carbon tetrachloride and tetrachlorethane, the process is subacute or even acute. (5) Splenic anæmia (Banti's disease, § 544).

Diagnosis.—*Cancer* of the liver is only difficult to diagnose from cirrhosis in the early stages; but usually it runs a more rapid course, and is accompanied by more pain, and more intense jaundice. The spleen is not usually enlarged in cancer. In *passive congestion* of the liver with ascites there are evidences of a cause, such as heart or lung disease. In the absence of ascites early cirrhosis may be mistaken for the other causes of liver enlargement. The enlargement of the spleen in atrophic cirrhosis may lead to the primary condition being overlooked. The liver is reduced in size in *starvation*. *Chronic peritonitis* with effusion may not be recognised as such until the organs can be palpated after paracentesis.

Prognosis.—The disease has a slower and more insidious onset than hypertrophic cirrhosis (below), and is in most cases a more serious condition. If the patient is seen before signs of portal obstruction supervene much can be done; if later, the prognosis is grave. The outlook is more favourable in patients who are young and where the general health is good. *Untoward Symptoms.*—Although restoration to comparative health has occurred after the development of ascites, with the onset and recurrence of rapid ascites the end is in view, the patient rarely living more than a few months. Pleurisy, renal disease, or tuberculous peritonitis are occasional complications.

Treatment in the early stages is practically the same as that employed for chronic congestion of the liver, and chronic gastritis (§§ 348 and 284). The habits of the patient must be corrected, and the diet reduced to the simplest elements; milk should be the staple food in advanced cases. A high-protein and a low-fat diet is best (§ 297, IV.): methionine and choline chloride (2 G. āā daily) have been advocated. Glucose and insulin aid. Alcohol must be avoided, and regular exercise taken. Treatment with liver extract, by mouth or by injection, has been tried with some success. A course of salines should be taken in the early morning, and rhubarb or mercurial pills at night. For hæmorrhages, vitamin K injections, calcium salts and hæmostatics such as coagulen Ciba are used. If portal obstruction and ascites have set in, see § 260. Patients sometimes

recover after repeated tappings, which give time for the establishment of the collateral circulation. Surgical measures have been devised to establish a collateral circulation, *e.g.*, "omentopexy," or stitching the omentum to the anterior abdominal wall. Relief of portal hypertension by anastomosing the portal vein to a neighbouring systemic vein is on trial.

C. We now turn to those chronic liver diseases in which **the size of the liver is increased.** These may be divided into two groups—those WITHOUT PAIN AND TENDERNESS are described immediately below. If the enlargement is attended WITH PAIN AND TENDERNESS, turn to § 348.

There are five diseases with **enlargement** of the liver **without pain and tenderness :** I. HYPERTROPHIC CIRRHOSIS ; II. FATTY LIVER ; III. VON GIERKE'S DISEASE ; IV. LARDACEOUS LIVER ; and V. HYDATID and other rare diseases. In INFECTIVE HEPATITIS (§ 332), CHRONIC CHOLELITHIASIS, and some other disorders, the liver is somewhat enlarged, but this is not their main feature.

Other rare causes of PAINLESS ENLARGEMENT of the liver are chronic blood diseases, noticeably LEUKÆMIA and SPLENIC ANÆMIA, ACHOLURIC JAUNDICE (§ 328), KALA-AZAR and MALARIA (§ 343, I*e*). TUMOURS (§ 350) may be unaccompanied by pain in the early stages.

I. *The* **liver is enlarged** *and* **painless** ; *its surface is hard,* JAUNDICE IS PRESENT, *but little or no ascites, and there is a long history of failing health.* The disease is probably HYPERTROPHIC CIRRHOSIS.

§ 343. Hypertrophic Cirrhosis of the Liver is a term employed in a generic or clinical sense to indicate a progressive enlargement of the liver due to an increase in the connective tissue of the organ with a tendency to jaundice. The condition may occur under at least four different aspects, due respectively to Syphilis, Gall-stones, Chronic Heart disease, and Kala-azar. A rare variety of hypertrophic cirrhosis accompanied by pigmentation of the skin has been described under the name of hæmochromatosis. Glycosuria appears later ; hence the name "BRONZED DIABETES." The pigmentation differs from that of Addison's disease in that it avoids the oral mucous membrane and appears on parts exposed to light rather than to pressure and friction. The pigment contains iron (§ 561).

I*a*. BILIARY CIRRHOSIS (Chronic Infective Cholangitis) is a condition occurring principally in young adults.

Symptoms. (1) There is a history of two or more attacks of acute hepatitis in preceding months. (2) The liver is uniformly, and often considerably enlarged, hard and sometimes rough. (3) The spleen is usually enlarged. (4) Recurring attacks of jaundice occur, with pyrexia even to 103° F., during which the urine contains bile and the stools are pale or clay-coloured. During these subacute exacerbations, the liver and spleen enlarge further, and the liver may become tender, with a feeling of a dull weight in the hepatic region. (5) In spite of the intense jaundice there are few or no signs of portal obstruction, and ascites is rarely, if ever, present. (6) Hæmorrhages, purpura and telangiectases may occur.

Etiology. The condition appears to be due to a subacute or chronic inflammation around the bile ducts, leading to partial obstruction. It is probably infective in origin ; a similar condition may arise following chronic biliary obstruction, *e.g.*, with gall-stones in the common bile-duct.

Diagnosis.—From *portal cirrhosis* it is known by the absence of signs of portal obstruction (§ 260). *Fatty* and *amyloid* livers are not accompanied by jaundice. *Cancer* has a more rapid and painful course.

Prognosis.—Sometimes patients die within twelve months, with an acute onset of the typhoid state, but most live for a number of years, with signs of progressive liver damage. In children the general health may appear unaffected for a long period.

Treatment is as for Hepatitis (§ 332). Mercurial inunction of the abdominal wall, or calomel, gr. $\frac{1}{10}$ to $\frac{1}{4}$ t.i.d. for three days, with intervals of three days, continued for some months has good results. Glucose and insulin aid restoration of liver function. A prolonged course of penicillin should be tried. Drainage of the gall-bladder has cured some cases.

I*b*. Right-sided Heart Failure results, as we have seen, in very considerable congestion of the liver. Long-continued passive engorgement of the liver gives rise to changes known as the "nutmeg liver," accompanied by more or less enlargement of the organ; and this may be attended by a considerable degree of fibrosis. The diagnosis depends on the presence of cardiac valvular disease and other features (see Passive Congestion, § 348).

I*c*. Chronic Syphilitic Disease of the liver generally takes the form of a diffuse hypertrophic fibrosis; or it may be met with in the form of *gummata*. Hepatic fibrosis may result from both hereditary and acquired syphilis, though the gummatous form is commoner in the latter. In the inherited variety two forms of fibrosis occur. In one there is fine diffuse fibrosis between the individual cells (pericellular cirrhosis), and this variety is usually accompanied by an enlarged spleen. The liver is smooth and firm. In the other, coarse fibrosis with perihepatitis occurs, as in the acquired disease.

The *Symptoms* are variable. The liver is moderately enlarged; there is not much tendency to jaundice and portal obstruction excepting in the final stages. There may be actual pain, especially when the capsule of the liver is involved; but as a rule there are only indefinite sensations of illness, accompanied in the gummatous cases by a low form of intermittent pyrexia. In the gummatous form nodular projections may possibly be made out on the surface of the organ. The presence of such projections, accompanied by intermitting fever and a history of syphilis in a young or middle-aged adult, makes the diagnosis practically certain. In the absence of a syphilitic history the occurrence of pain and local tenderness at intervals points to syphilitic rather than to alcoholic cirrhosis, because *perihepatitis and the involvement of the capsule* are prominent features of syphilitic cirrhosis. In the diagnosis from cancer we have mainly to rely on the Wassermann reaction, the response to therapy and the (usual) absence of jaundice and ascites in syphilitic disease.

The *Prognosis*, as a rule, is good, if the nature of the disease be discovered and it be treated adequately with antisyphilitic remedies.

I*d*. Cirrhosis of Biliary Obstruction.—Hypertrophic cirrhosis has been produced experimentally in one half of the liver by ligature of one hepatic duct, and it is met with clinically in association with gall-stones, tumours or glands pressing on the bile-ducts. There is a history of repeated attacks of biliary colic, enlargement of the liver, with jaundice of some years' duration. The acholic stools aid the diagnosis of this form of hypertrophic cirrhosis.

I*e*. Tropical Cirrhosis.—Many parasitic infections involve the liver, but only a few produce actual cirrhosis. Malaria may induce hepatitis and biliary pigment stones, but it is doubtful if a true malarial cirrhosis ever occurs; kala-azar parasites, however, may produce it. Biliary cirrhosis is found in clonorchiasis and bilharzial peri-portal cirrhosis in *S. mansoni* and *S. japonicum*. Though hepatomegaly with occasional jaundice occurs, ascites is rare. And see Abscess of liver (§ 336).

II. *The* **enlargement of the liver** *is* painless *and uniform; the surface is smooth and soft; there is* no jaundice or ascites, *and the* spleen is not enlarged; *there is a history of alcoholism, phthisis, or other toxæmia.* The disease is probably Fatty Liver.

§ **344. Fatty Liver** is a condition in which fat is deposited in the hepatic cells, commencing in the periphery of the lobules. It is nearly always associated with some other disease.

Symptoms.—(1) The liver is enlarged uniformly and is quite smooth. (2) Pain, jaundice, and portal obstruction are absent. (3) The accompanying symptoms are due to the cause of the fatty liver, and may consist, therefore, of debility, anæmia, etc. (4) The history of a *Cause* is important—viz., (i.) Chronic wasting disease, such as phthisis. (ii.) Fatty liver appears in association with fatty heart (*q.v.*) and general obesity. (iii.) It often accompanies chronic alcoholism; and a mixed degeneration with fat and fibrosis is not uncommon.

The *Diagnosis* from the painful enlargements of the liver is not difficult. In *lardaceous* liver there are also signs of lardaceous spleen or kidney. The *Prognosis* and *Treatment* depend upon the cause.

§ **345.** III. **Von Gierke's Disease** is a rare cause of enlarged liver, usually seen in the young, due to excessive glycogen accumulation. Adrenalin does not produce the usual rise in blood sugar.

IV. *The* **enlargement of the liver** *is* UNIFORM *and* PAINLESS; *the surface is smooth and hard; there is* NO JAUNDICE, NO ASCITES; *the* SPLEEN IS ENLARGED; *there is a history of prolonged purulent discharge, phthisis, or constitutional syphilis.* The disease is LARDACEOUS DEGENERATION.

§ **346. Lardaceous (Amyloid or Waxy) Liver** is a condition in which the liver tissue is replaced by lardaceous material, which starts in the capillaries and smaller arteries of the organ, leading sometimes to an immense enlargement.

Symptoms.—(1) The liver is enlarged uniformly and smoothly, and feels firm; (2) pain, jaundice, and portal obstruction are absent; (3) the constitutional symptoms are due to the causal condition, and to amyloid disease of other organs.

Etiology.—(i.) Long suppuration and purulent discharge, as from chronic osteomyelitis; (ii.) constitutional syphilis; and (iii.) tuberculous disease of the lungs or elsewhere. Amyloid liver has become much rarer since chronic suppuration has been obviated by improved surgical methods.

Diagnosis.—The presence or history of a cause renders the diagnosis of amyloid disease comparatively easy.

The *Prognosis* depends upon the amount of amyloid disease elsewhere. Diarrhœa, indicating amyloid changes in the intestines, abundant pale urine, with albuminuria, indicating amyloid disease of the kidneys, are untoward signs. If the cause is remediable, as by surgical treatment, the liver may decrease in size.

Treatment.—The indications are (i.) to remove the cause, and (ii.) to keep up the strength. The former is attained by anti-syphilitic treatment in the case of syphilis, and by surgical treatment in the case of long-standing discharges. Tonics, such as iron and quinine with cod-liver oil, are useful.

V. *The* **enlargement of the liver** *is* PAINLESS, *but* NOT UNIFORM, *and the upper margin of the liver dulness is perhaps* ARCHED; *there is no jaundice or ascites and the spleen is not enlarged; a thrill may be felt on percussion.* The disease is HYDATID CYST.

§ **347. Hydatid of the Liver** depends on the presence in the liver of the parasite, *Echinococcus granulosus*, rare in this country, though common in Australia, New Zealand, the Argentine, Greece, and Iceland, where dogs live in close association with man.

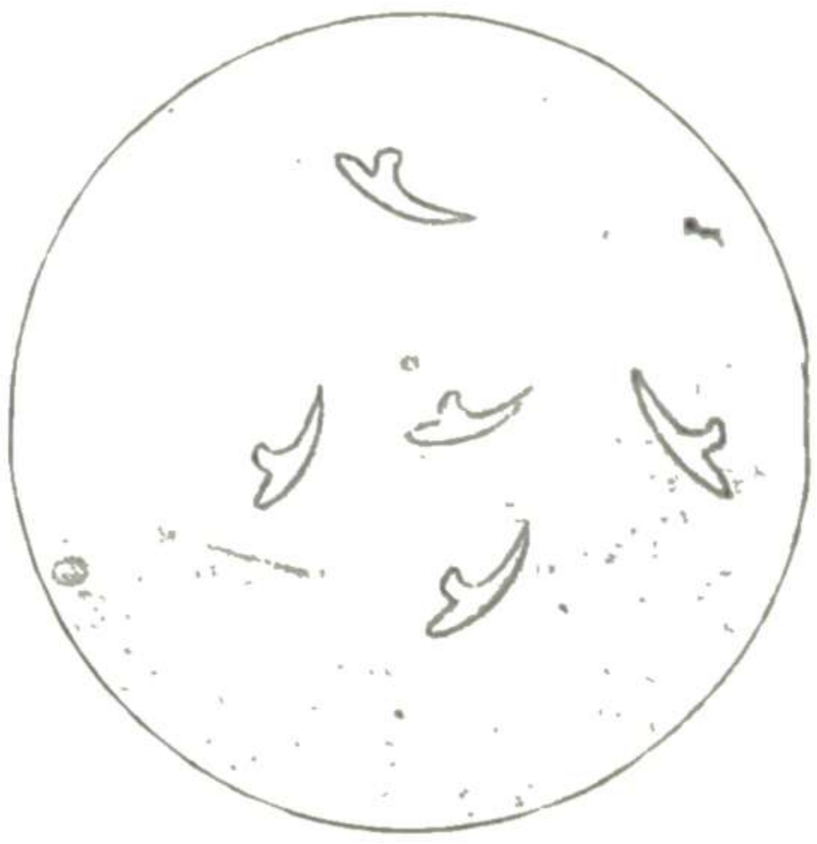

FIG. 85.—Hooklets, from a HYDATID CYST in man, magnified about 150 times. These form the crown of hooklets around the anterior end of the scolex, and are absolutely distinctive of hydatid fluid.

Symptoms.—(i.) There is a slowly increasing enlargement of the liver, which is

smooth, globular, and elastic, sometimes fluctuating. The right chest may be bulged outwards, with dulness in the axilla. When the fingers of the left hand are laid on the tumour and tapped with those of the right hand, the "hydatid fremitus," or "thrill," is felt in some cases. (ii.) Pain is absent unless the tumour is very near the surface, when pain may be present, because *the capsule* is involved. (iii.) No constitutional symptoms appear unless the tumour presses upon the surrounding structures, or becomes inflamed and suppurates. (iv.) Rupture into the peritoneal cavity may be followed by anaphylactic shock and urticaria, and later by the growth of secondary cysts. Jaundice may occasionally be caused by cysts obstructing the bile-ducts.

Etiology.—The parasite enters the alimentary canal of man by means of food or water contaminated by fæces containing the ova of the *Echinococcus granulosus* (*Tænia echinococcus*), a tapeworm which infests the dog. The embryo is carried to the liver, where it encysts and grows, the liver tissues forming a fibrous capsule known as the adventitia. The cyst so developed has a lining membrane composed of an endogenous germinal-layer and an exogenous hyaline layer, and contains a clear fluid. The endogenous layer buds the tiny brood-capsules in which scolices or embryonic heads develop, each with a crown of hooklets. Daughter cysts and grand-daughter cysts may also be formed.

Diagnosis.—*Abscess* of the liver produces pain and fever, and on aspiration yields purulent material like anchovy sauce. *Pleural effusion* on the right side, leading to dulness in the axilla, may resemble hydatid. In such cases a *bulging* outwards of the lower ribs over the liver points to the presence of hydatid. A *renal cyst* has resonance in front, due to the colon. A history of residence in Australia, the Argentine, etc., should lead one to suspect hydatid in cases of slowly increasing enlargement of the liver, *with few other symptoms.* The symptoms of suppurating hydatid cyst of the liver are very like those of inflammation of the gall-bladder. X-rays are of value in diagnosis. Hydatid cyst fluid is pathognomonic, although exploratory puncture entails serious risk, as it may set scolices free, which subsequently form secondary cysts. The fluid is clear, opalescent, of low specific gravity, and contains a large excess of chlorides, no albumen (unless inflammation has taken place), and most characteristic of all—echinococcus hooklets (see Fig. 85 and Table LX). The blood sometimes shows eosinophilia, and the serum may give the complement fixation reaction with a suitable antigen in 70 per cent. of cases. Infested patients react to an intradermal injection of hydatid fluid (Casoni); it is a group reaction for infection with tapeworm, for in cysticercosis similar positive reactions are occasionally recorded.

Prognosis.—The patient may live for several years with no other symptoms than a slow increase in the size of the liver. The prognosis must be guarded even if the cyst is safely removed; other cysts may be present which will develop later. A cyst may remain quiescent for twelve years or more without losing its potentiality for mischief. The cyst may suppurate, giving rise to the symptoms of liver abscess, or pyæmia may be set up. When a cyst leaks into the surrounding tissues, anaphylactic symptoms may occur—collapse, vomiting, and urticaria associated with eosinophilia. Sometimes death occurs by the sudden rupture of the cyst into the pleura or peritoneum.

Treatment.—Hydatid cysts most often involve the inferior aspect of the right half of the liver and are generally accessible through an anterior abdominal incision. A transpleural route may be necessary for cysts impinging on the diaphragm. After opening the abdomen, packing off and locating the cyst, aspirate the fluid and inject 6 to 10 c.c. of pure commercial formalin. Subsequently the adventitia is incised and daughter cysts removed. The cyst cavity is filled with saline and the adventitia sutured together, where possible. Drainage is better avoided.

There are three diseases in which **enlargement of the liver** is attended **with pain and tenderness :** I. CHRONIC PASSIVE CONGESTION, II. CANCER

OF THE LIVER, and III. ABSCESS OF THE LIVER. In CHRONIC CHOLELITHIASIS and several ACUTE DISORDERS the liver may be slightly enlarged and tender.

I. *The* **enlargement** *is moderate, smooth, and uniform,* PAINFUL, *and* TENDER ; *some jaundice and ascites may be present, the* SPLEEN IS SLIGHTLY ENLARGED, *and there are signs of congestion of the abdominal viscera.* The disease is probably CHRONIC CONGESTION OF THE LIVER.

§ **348. Chronic Passive Congestion** of the liver is a condition in which the enlargement is due to venous obstruction.

Symptoms.—(i.) The liver is tender, and a sensation of weight and fulness is complained of in the hepatic region. Expansile pulsation synchronous with the heart may be conveyed to the palpating hand in the early stages, but as the organ becomes firmer this is lost. (ii.) Signs of general venous obstruction appear. (iii.) Ascites develops, and the spleen is slightly enlarged. Some degree of jaundice may occur. (iv.) Gastro-intestinal disturbances are common.

Etiology.—Passive congestion is the result of any backward pressure due to obstruction or failure of the circulation. In most cases this is caused by heart or lung disease, and especially mitral stenosis.

The *Diagnosis* is often aided by the recognition of the heart disease on which it depends. In some cases of *ascites* with anasarca of the legs, we may find both *hepatic enlargement* and *albuminuria*, and a difficulty may arise as to which was the primary cause of the condition—heart, liver, or renal disease. The difficulty is increased if extensive bronchitis prevents accurate auscultation of the heart. In such cases, *the liver* may be excluded as the primary cause, if the dropsy in the legs clearly preceded the dropsy in the abdomen. The presence of hepatic enlargement is then a sign of great value as helping to exclude *renal* mischief, because enlargement of the liver is not a usual sequence of kidney disease, although it is a fairly constant result of *cardiac* valvular disease. In *paroxysmal tachycardia* the enlarged liver quickly decreases in size when the heart resumes its normal rate.

Prognosis.—The prognosis depends on the cause of the congestion ; the state of the heart is generally the measure upon which the patient's chance of a longer or shorter life depends. In mitral stenosis an enlarged liver with ascites is less grave than in mitral regurgitation, because it normally occurs at an earlier stage in stenosis. It is most serious in aortic disease, and especially regurgitation, as it indicates mitral and tricuspid insufficiency.

The *Treatment* is that of the cause, and our attention must be directed to the heart and lungs. Purgatives and light foods are necessary in order to relieve the strain on the portal system. Leeches over the liver or venesection may be indicated.

II. *The* **enlargement of the liver** *is* IRREGULAR : *the* PAIN *and tenderness may be great ;* JAUNDICE *and* ASCITES *are present ; the spleen is not enlarged ;*

the patient is advanced in years, feeble and emaciated. The disease is CANCER OF THE LIVER.

§ **349. Cancer** of the liver is rarely primary, and is usually secondary to disease elsewhere. It occurs after middle life and is rare before thirty-five.

Symptoms.—(i.) Pain is an almost constant feature of cancer of the liver; it is continuous, with exacerbations, and is independent of food or posture. A certain amount of tenderness develops. (ii.) The enlargement of the liver is irregular and may become an enormous size, and often umbilicated nodules may be made out. These are of a hard consistence, and increase rapidly. There is also less commonly diffuse cancer, in which there are no nodules, and in which the liver is only slightly and uniformly enlarged. (iii.) Jaundice is usually present, *sooner or later*, and is intense and progressive; an intense jaundice persisting over five to seven weeks in an old person should indeed always lead one to suspect cancer. Ascites generally occurs either from involvement of the glands in the fissure, or of the peritoneum. The spleen is not enlarged. (iv.) The general health of the patient is bad, and emaciation and cachexia may be present before any local signs are discovered. Cancer may be present in another part of the body. Fever may occur at intervals, and a polymorph leucocytosis (sometimes marked) is usual. Rectal examination may reveal malignant glands in the pelvis.

Etiology.—Cancer is liable to spread to the liver (*a*) via the portal blood stream or the abdominal lymphatics, from primary disease in the stomach, colon or other abdominal organs. (*b*) It may invade the liver via the systemic blood vessels from a primary site in the breasts, lungs, testicles, etc.

Diagnosis.—Jaundice is rarely entirely absent in cases of cancer of the liver: this and the cachexia alone may justify a diagnosis. The diagnosis from *cirrhosis* may be difficult when nodular enlargement cannot be made out, and when considerable ascites is present. In cirrhosis there is little or no pain and tenderness, the history of the illness is of longer duration, the spleen may be enlarged, and the jaundice is not so intense. The *inflammatory thickening* under the liver after a long history of gall-stones may resemble cancer, and can be distinguished only when time shows little or no increase in the enlargement. In doubtful cases, the abdomen should be thoroughly examined after removal of the ascitic fluid. *Syphilitic* liver has not so much pain and tenderness, is of slower growth, and rarely produces ascites.

Prognosis.—Cancer of the liver is usually fatal within six to twelve months, death taking place from exhaustion. Untoward symptoms are rapid growth, ascites, or respiratory difficulties due to extension of the disease to the lungs and pleura.

Treatment can be palliative only. Treatment of ascites makes the patient more comfortable. Morphia or opium is administered for the pain, and attention must be given to the relief of the symptoms of gastric

distress, and to aid nutrition. With rest and care there may be periods during which the disease makes no progress, and which hold out to the patient false hopes of his ultimate recovery.

III. **Abscess of the Liver** also produces considerable hepatic enlargement, which is PAINFUL and TENDER. It has already been described among the Acute Diseases, § 336; but sometimes it runs a very chronic course.

§ 350. Tumours of the Liver other than CANCER (§ 349), HYDATID (§ 347), and GUMMA (§ 343, Ic.), are more rare. Their presence is manifested by *enlargement of the organ*, which may be regular or irregular, accompanied in some cases by constitutional symptoms. When, as in some cases of ACTINOMYCOSIS and FASCIOLA HEPATICA (*Distoma hepaticum*) (§§ 337, 338), they assume an inflammatory form, pyrexia is present. SARCOMA OF THE LIVER is occasionally met with—*e.g.* Lympho-sarcoma—but it is most often secondary to deposits elsewhere, and the liver condition is only a subordinate part of the case. The patient may be younger than in the other forms of malignant disease. Chondro-sarcoma, Melano-sarcoma, Tubercle, Angioma, Lymphadenoma, and Fibroma occur very rarely. Riedel's lobe is often mistaken for tumour (§ 263).

THE GALL-BLADDER

PART A. SYMPTOMATOLOGY

§ **351.** The cardinal symptoms commonly associated with gall-bladder disease are **pain** or **discomfort in the upper abdomen** and back, **flatulence, nausea** or **vomiting.** Occasionally **constitutional symptoms** are also present. Pain or discomfort is usually epigastric, often worse on the right side, and may be related to meals. It varies from a dull ache to acute paroxysms of colicky pain, as when a calculus becomes impacted in the neck of the gall-bladder or in the cystic duct. The pain is often referred to the lower right ribs, the angle of the right scapula or between the scapulæ. Flatulence in the abdomen may be severe; it produces a sense of fullness, so that the patient loosens the clothing. Nausea is rarely present before a meal, but after a few mouthfuls of food the patient may feel so distended and nauseated that he cannot eat more. Vomiting may be occasional, or in attacks, associated with the other symptoms. With colic it is usually severe. A characteristic feature of gall-bladder disease is the aggravation of the symptoms by food containing eggs, cream and animal fats, so that the patients avoid these foods. Pyrexia and other constitutional manifestations accompany catarrhal or suppurative processes in the gall-bladder. **Jaundice** is present when there is obstruction of the hepatic or common bile ducts.

PART B. PHYSICAL EXAMINATION

When examining the gall-bladder one must ensure that the abdominal wall is entirely relaxed. With the patient in the supine position and the knees drawn up, palpate gently with the fingers laid flat on the abdominal wall, the patient breathing gently all the time. Occasionally more satisfactory results are obtained by making the patient sit and lean

forward with the knees flexed, completely relaxed, whilst one palpates with the tips of the fingers under the right costal margin. The gall-bladder may be best felt when a deep breath is taken; when enlarged it is felt as a tender globular swelling coming forward at the tip of the ninth right costal cartilage. It usually remains just under the surface of the anterior abdominal wall, moves freely downwards with respiration, but cannot be moved laterally; when very large it is dull to percussion and may extend even to the right iliac fossa. With cancer of this organ, the surface becomes hard and nodular (and see § 263. I). Even when the gall-bladder is not large enough to be felt, with inflammation the upper right rectus muscle shows rigidity. If the lower hepatic margin in the right hypochondrium be divided into outer, inner and middle segments, when the patient takes a deep breath he flinches and his face expresses pain on deep palpation of the middle segment but not with palpation of the other segments. In diseases of the gall-bladder it is essential to examine the back, as referred areas of tenderness may be met (*a*) over the 11th and 12th right ribs, (*b*) over the 5th–8th dorsal spines or (*c*) over the para-vertebral muscles between the scapulæ, especially over the right side. A friction rub is occasionally audible over the gall-bladder.

There are two forms of special investigation: (*a*) X-ray examination reveals gall-stones if opaque material such as calcium is present. Cholecystography is performed after giving iodophthalein B.P. by mouth or intravenously (Graham-Cole test). When it is taken by mouth, radiograms 12 hours later reveal the degree of filling; if a fatty meal is then given, a further plate reveals the degree of emptying. Filling may be promoted by giving two 1 oz. doses of glucose the day before. If the gall-bladder does not fill, the glucose is immediately repeated, a fat-free diet given, and more iodophthalein 24 hours after the first dose.

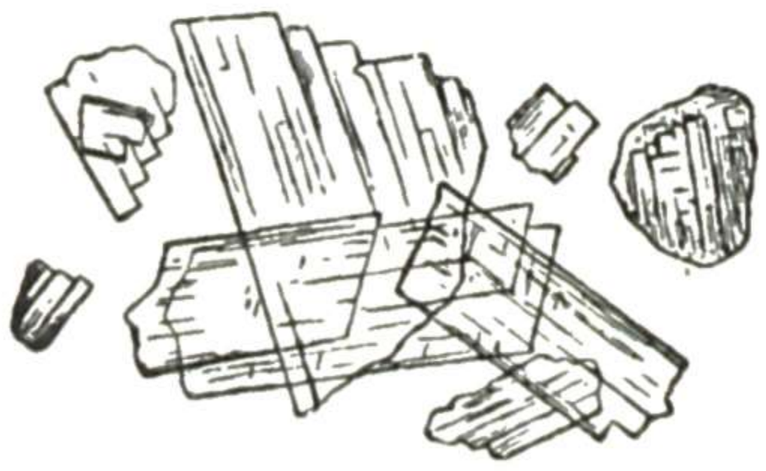

FIG. 86. — Cholesterol Crystals. Microscopic appearance presented by fragments of gall-stones in the fæces or from the duodenum.

Normal filling and emptying are occasionally compatible with a diseased gall-bladder, but as a rule improper filling and emptying, especially after the intravenous method, indicates a pathological condition. Non-opaque stones may be visualised only when they are surrounded by opaque substance (Fig. 87). (*b*) By introducing a long rubber tube into the duodenum, especially after a period of starvation, a sample of resting duodenal contents may be obtained; if then 30 c.c. of concentrated magnesium sulphate or 20 c.c. of hot olive oil or of peptone (10 per cent.) are introduced through the tube, a profuse flow of bile from the gall-bladder may be obtained within a few minutes and a sample aspirated; this is examined for micro-organisms (especially *B. coli* and those of the typhoid group), cholesterol crystals (which may be deformed when gall-stones are present) (Fig. 86), for lipoid globules (from a "strawberry gall-bladder"), and for cells. After magnesium sulphate desquamated cells from the duodenal wall may be present and must be differentiated from pus cells. If achlorhydria is present, the results must be viewed with caution.

PART C. DISEASES OF THE GALL-BLADDER

Gall-bladder Disease may be: A. **Acute**—I. Acute Cholecystitis, II. Gall-stone Colic; or B. **Chronic**—III. Chronic Cholecystitis, IV. Cancer of the Gall-bladder.

I. *The patient complains of* PAIN *in the* GALL-BLADDER REGION; *the pain is* PAROXYSMAL, *or* DULL AND CONTINUOUS, *and* RADIATES TO THE RIGHT SHOULDER. *There is* TENDERNESS *over the* GALL-BLADDER, *vomiting and some fever. The disease is* ACUTE CHOLECYSTITIS.

§ 352. Acute Cholecystitis may be catarrhal, suppurative, or gangrenous, according to the severity of the infection.

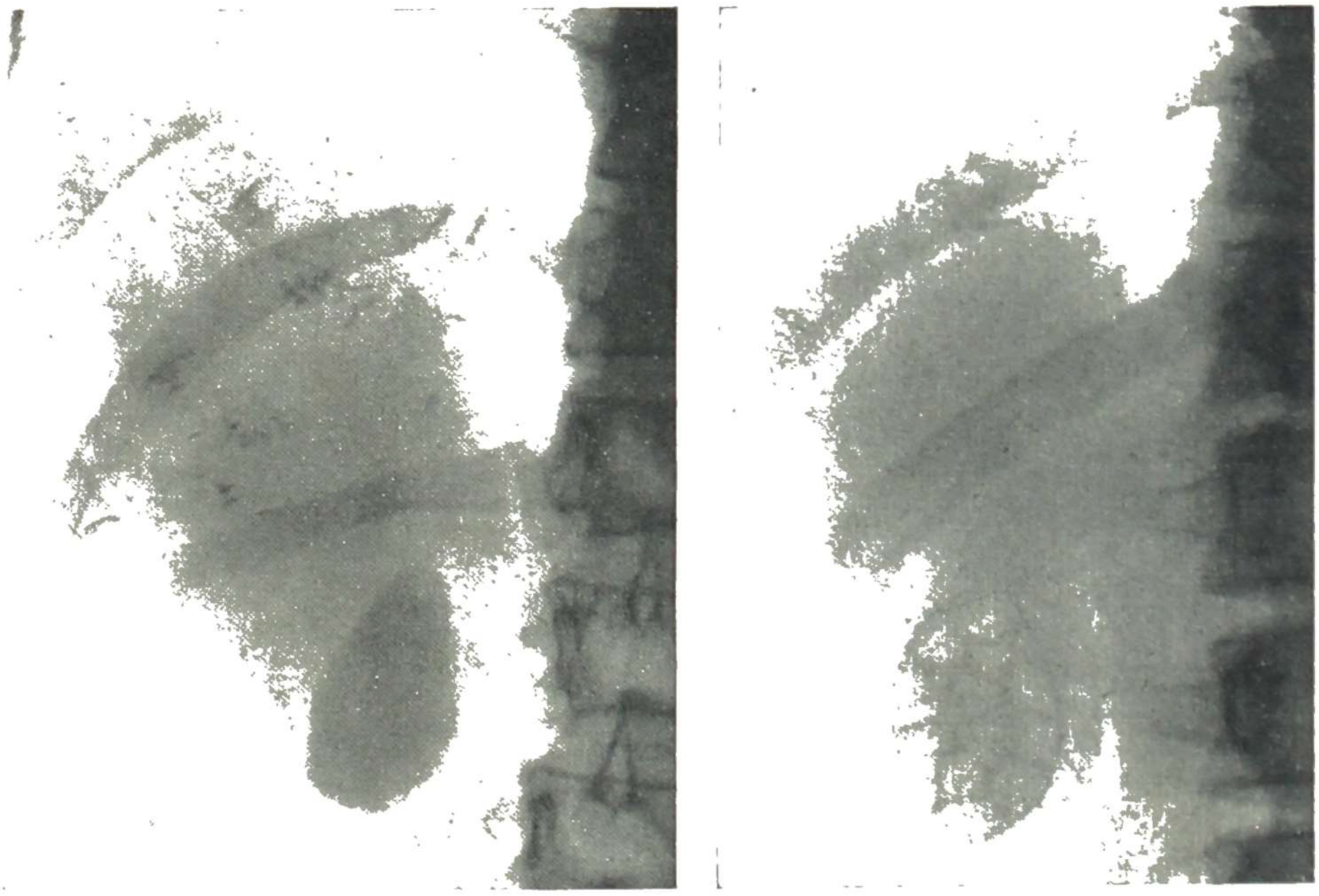

FIG. 87.—*Left:* a normally filled gall-bladder 13 hours after administration of the dye. *Right:* same type of gall-bladder, filled with typical facetted stones displacing the dye.

Symptoms.—(1) Local pain in the right hypochondrium and epigastrium. (2) Pain is spasmodic or dull and continuous, and radiates through to the back and right shoulder. (3) Tenderness in the right upper abdomen; this becomes localised below the tip of the ninth costal cartilage. (4) Rigidity of this area; if the muscles are relaxed, the enlarged gall-bladder may be felt. (5) Symptoms may be mild, like dyspepsia, or severe with vomiting, jaundice, rigors and much general disturbance.

Etiology.—Predisposing: (1) stagnation of bile; (2) calculi of solitary cholesterol type; (3) foreign bodies, worms and ova in the gall-bladder; and (4) previous attacks. The *exciting cause* is infection, which may come

from tonsils or teeth; or follow pneumonia, influenza or typhoid fever, gastric, duodenal or appendicular disease.

Diagnosis.—Absence of jaundice is not a point against cholecystitis. In *gall-stone colic* the pain is more severe, while local signs of tenderness, paralytic distension of intestines and palpable gall-bladder favour cholecystitis. Leucocytosis is rare with gall-stones, unless accompanied by cholecystitis. In perforated *duodenal ulcer* there may be a history of characteristic indigestion. In *acute pyelonephritis*, pus and *B. coli* are found in the urine. *Appendicitis* may cause difficulty; appendicitis and cholecystitis may co-exist. Right *diaphragmatic pleurisy* or *basal pneumonia*, *herpes zoster* and *intercostal neuralgia* must be excluded.

Prognosis.—The attack may subside or pass into chronic cholecystitis. If it proceeds to suppuration or gangrene, local or general peritonitis may supervene and life may be endangered.

Treatment.—The patient should be in bed on milk or light diet. Local applications of fomentations, a kaolin poultice, dry cupping or leeches may be used, or morphia may be required for the pain. Vomiting may be relieved by bismuth carbonate, hydrocyanic acid or an effervescing mixture. Salicylate of soda and hexamine with alkalies are useful as antiseptics. Penicillin injections have given good results. A drachm of magnesium sulphate taken in a dessertspoonful of water, fasting, in the morning, and followed after an hour by a pint of hot water or weak tea, and pure olive oil ℥120 between meals, act as a stimulus to gall-bladder evacuation. If fever and rigors persist, operation on the third or fourth day is indicated. In cholecystitis, even without gall-stones, drainage is not sufficient to cure, and the gall-bladder should be removed.

II. *The patient, usually an elderly female, is suddenly seized with* PAROXYSMS OF SEVERE PAIN *in the hepatic region, and in the course of twelve to twenty-four hours she may become* JAUNDICED, *the stools becoming clay-coloured.* The attack is one of BILIARY COLIC.

§ **353. Gall-stones** and **Biliary Colic.**—Gall-stones are concretions which form in some part of the biliary passages, most commonly in the gall-bladder. CHOLELITHIASIS is the condition in which gall-stones are developed. When gall-stones move along any of the ducts, they give rise to Biliary Colic.

GALL-STONES may be *metabolic*, consisting of deposits of cholesterol or bile-pigment, or *infective*, of mixed composition. They vary in size from a sand-grain to a golf-ball. When solitary, they are round or oval in contour. The facets or flattenings of their surface are caused by the pressure of one against the other; this indicates that there has been more than one stone in the gall-bladder or bile-ducts. The colour varies from yellow to dark brown; their chief physical characteristics are the smooth "soapy" surface, the ready way in which they crumble between the thumb and finger (though sometimes they are very hard), and their lightness as compared with renal calculi. They generally consist chiefly of cholesterol mixed with calcium and bile pigment, but are sometimes pure cholesterol, pure bilirubin, or pure calcium carbonate. Cholesterol is contained and held in solution by bile salts in normal

bile. When from various causes the liver is unable to produce the bile salts in sufficient quantity, there is a high cholesterol content in the blood and bile, with eventual deposition of cholesterol and formation of gall-stones. Normal individuals can eat food containing cholesterol, because more bile salts are produced by the liver and hold the cholesterol in solution. With other individuals this capacity is defective. The foods which increase the cholesterol content of the blood are: egg yolk, butter, cream, liver, kidney, pancreas, brain and meat fats.

Biliary Colic.—Symptoms may be absent when the stone is at rest, but when it begins to move (i.) the pain is agonising; it starts in the epigastrium and shoots into the right hypochondriac region towards the spine and up to the right shoulder, but never passes downwards. The paroxysm is usually so severe that the patient is in a state of partial collapse, with vomiting, hiccough, subnormal temperature, and a quick, weak pulse. Sometimes there is a rigor, and the temperature rises a few degrees. Between the paroxysms of acute pain there is a constant dull aching and tenderness over the hepatic region. The attack lasts from a few hours to a few days. (ii.) The liver may be enlarged and if a stone becomes impacted in the hepatic duct the enlargement may be considerable. (iii.) Jaundice usually appears twelve to twenty-four hours after the paroxysm, and lasts from a few days to a few weeks. It is most intense when the stone is impacted in the common duct, and may give rise to severe pruritus.

The *Symptoms* which arise vary somewhat with the *position of the gall-stone* (Fig. 88). Thus: (i.) If a stone is impacted in the *common duct* there are biliary colic, jaundice, and sometimes a distended gall-bladder, and if the impaction continues the liver becomes enlarged. (ii.) If a gall-stone is impacted in the neck of the gall-bladder (*i.e.*, in the *cystic duct*), *biliary colic without jaundice* is present. In time the gall-bladder may be distended with mucus, and form a definite abdominal tumour (mucocœle), but more often the chronic irritation of many calculi leads to chronic fibrosis of the gall-bladder which prevents its becoming enlarged. Considerable distension of the gall-bladder is not usually associated with the presence of many gall-stones, but more often with cancer of the pancreas or chronic pancreatitis.

(iii.) Stone impacted in the *hepatic duct* is rare. It causes biliary colic and jaundice, but the gall-bladder is not distended. (iv.) Stones occasionally form in the *radicles of the hepatic ducts*, and give rise to indefinite symptoms, sometimes without pain, and usually without jaundice. (v.) Sometimes small particles of cholesterol (biliary sand) in the *gall-bladder* give rise to recurring paroxysms of pain, unaccompanied by other symptoms, eluding diagnosis. (vi.) The stones may become encysted, but more often, without surgical intervention, abscess and fistula result.

Diagnosis of Biliary Colic.—It is distinguished from the two other forms of colic in Table XIV, § 246. The severity of the pain and its paroxysmal character usually distinguish it from other acute diseases of the liver. *Pseudo-biliary colic* is sometimes met in nervous women. The diagnosis

from *cancer* of the liver may be very difficult. Both occur at the same age, and both cause jaundice; further, cancer of the gall-bladder may follow after years of trouble from gall-stones. In cancer, jaundice steadily becomes more intense. It must be remembered that in some cases gall-stones are passed without colic, but with jaundice; consequently, *recurring attacks* of jaundice in an elderly woman should lead one to suspect gall-stones. A radiogram may show gall-stones, but a negative plate is not conclusive. Negative shadows may be defined in the opaque gall-bladder with the Graham-Cole test (Cholecystography, p. 436). In all suspected cases the stools should be carefully examined for stones. *The presence of ascites* points to cancer, which rarely exists long without peritoneal effusion.

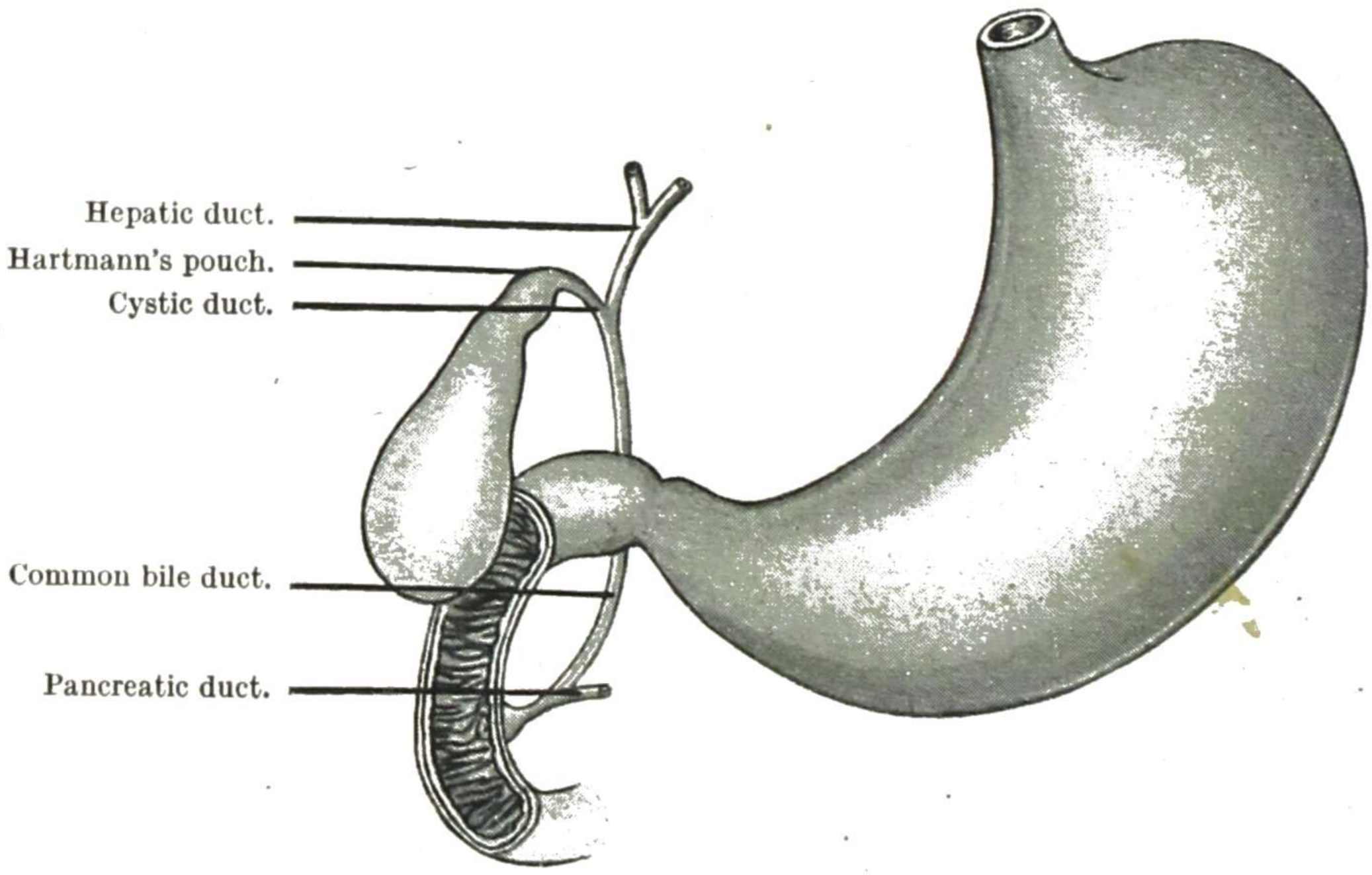

FIG. 88.—The STOMACH AND DUODENUM opened to show the ducts in connection with the Liver and Pancreas.

Gall-stones at rest in the Gall-bladder occur often in elderly women: they give rise to *Symptoms* the cause of which may be difficult to diagnose. They are the symptoms of cholecystitis (§§ 352, 354) which precedes or accompanies gall-stone formation. They consist of (1) flatulence, especially after fats, (2) pain referred to the right upper abdomen and shoulder, (3) subcostal ache, especially when chilled.

Etiology of Gall-stones.—(i.) They occur usually after the age of 50; (ii.) are much commoner in women, especially in multiparæ, and (iii.) in stout persons of sedentary habit whose diet is rich in fat and sugar. (iv.) There is often a history of gout, asthma or migraine. (v.) They may follow cholecystitis due to typhoid, coli or streptococcal infection, or any cause of stagnation of bile in the gall-bladder. (vi.) The colic is often

determined by a sudden strain, by motoring or by an overloaded stomach especially with rich fatty food.

Course and Prognosis.—The prognosis as to recovery from an attack of biliary colic is excellent, but recurrence may be expected. A stone usually forms in the gall-bladder and becomes impacted for a time in the neck of the cystic duct, giving rise to biliary colic without jaundice. It may then pass down the common duct, and cause jaundice. This rarely lasts more than a few weeks, but cases have been reported where it lasted two years. Impaction with infection is followed by: (i.) *Ulceration* of the ducts, with pyrexia, or abscesses of the liver and bile-ducts (cholangitis), and consequent subacute pyæmia; (ii.) *perforation* into adjacent tissues, leading, for example, to fatal peritonitis; (iii.) inflammation and *abscess* (empyema) of the gall-bladder, which may open externally, perforate into the peritoneum, or rupture into the intestines; (iv.) formation of *fistula* between the gall-bladder and the colon or duodenum, through which stones can pass of such a size that they may cause intestinal obstruction. (v.) *Cancer* may supervene in later years.

Treatment.—During the attack treatment aims at relieving spasm and controlling pain. If mild, a tablet of trinitrin may give relief. If severe, belladonna is the drug of choice: a dose of 15 minims of the tincture may be repeated after 2 hours, or a full dose of atropine given. A hypodermic injection of morphine or pethidine may be necessary for the pain, but morphine tends to increase biliary spasm. Chloroform inhalations are used in severe cases. Hot water with grains 60 of bicarbonate of soda to the pint aids the flow of bile, and hot turpentine stupes relieve pain. Sometimes an attack of pain is warded off by giving a hot bath (100° F.).

Between the attacks the diet must be supervised. Foods containing cholesterol must be omitted, therefore forbid most animal fats, especially if cooked. A little butter is allowed, but no cream or yolk of egg, no kidney, liver, brain, sweetbread or the fat of meat, pork, goose and duck. (For specimen diet see § 297, IV.) To flush out the biliary passages, the liver can be made to secrete more bile by administering bile acids, potassium salts, salicylates, and oil of peppermint: particularly powerful is dehydrocholic acid (decholin): Carlsbad Sprudel salt is popular, as it is rich in potassium salts. When it is desirable to cause the gall-bladder to contract and empty itself (gall-bladder drainage), give magnesium sulphate in doses of gr. 30 to 60 in concentrated solution before breakfast, and olive oil between meals. Where there is hyperchlorhydria, a tablespoonful of olive oil with a small dose of tincture of belladonna is useful, given before meals. Hexamine and sodium salicylate are excreted in the bile; the latter increases the excretion of bile salts and cholesterol; the hexamine sterilises the infected bile, when given in large doses with citrate of potassium in order to prevent irritation of the urinary bladder. Felamine is a useful preparation, in 5-grain tablets twice or thrice daily. Surgery is indicated where there is suppuration, when the gall-bladder

remains distended, the common duct is blocked, or biliary colic frequently recurs. The old practice of giving large amounts of olive oil does not remove gall-stones; the resulting masses passed in the fæces are aggregations of fatty acid crystals, not the gall-stones.

B. Chronic Diseases of the Gall-bladder

III. *The patient, a young adult or middle-aged, complains of* FULLNESS *weight or oppression* IN THE EPIGASTRIUM *about half an hour after meals,* WORSE AFTER GREASY *or* ACID FOOD. *Relief is obtained by belching, and cessation almost at once by vomiting. There is* CHILLINESS *or* SHIVERING *in the evenings, and a shoulder ache or stabbing* PAIN *in the* RIGHT SIDE *with a deep breath. The disease is probably* Chronic Cholecystitis.

§ 354. Chronic Cholecystitis is one of the commonest of all abdominal diseases, and is often undiagnosed in the early stages when medical treatment is available. It may follow acute cholecystitis, it may precede or accompany gall-stones. Or, it may be chronic from the onset, brought on by sedentary habits which predispose to stagnation of bile and infection. The infection, usually borne by the blood-stream, is first seated in the wall of the gall-bladder. Cholesterol metabolism is interfered with, hypercholesterolæmia follows, and the mucosa of the gall-bladder becomes engorged with cholesterol ("strawberry gall-bladder"). Cholesterol stones form in the lumen followed by infection of the contents of the gall-bladder; at this stage are formed the mixed gall-stones of cholesterol, bile pigments and calcium.

Symptoms.—(1) Continual flatulent dyspepsia, fullness or oppression in the epigastrium, coming on soon after food; (2) worse after fruit, eggs, cooked fats, pork, pastry, pickles or heavy meals; (3) relieved by belching and ceasing almost at once after vomiting; (4) distension or tightness relieved by bending forwards, flexing the right thigh on the abdomen or loosening the clothing; (5) acidity or heartburn, sometimes a gush of saliva into the mouth; (6) chilliness or "gooseflesh," especially in the evenings. Attacks of "biliary fever," *i.e.*, shivering, nausea, vomiting, diarrhœa and faintness, with slight temperature, may occur at intervals for months or years, especially after exertion. (7) Aching in the right shoulder or stabbing pain with tenderness at the angle of the right scapula. Tenderness may occur in the areas supplied by the seventh to ninth thoracic segments, the areas which supply the sympathetic nerves to the gall-bladder and bile-ducts. (8) There may be congestion and œdema at the right base. (9) There is sometimes reflex gastric hyperchlorhydria, but the stomach juices are usually sub-acid. (10) Remote symptoms from the gall-bladder as a source of infection are chronic infective arthritis, fibrositis, phlebitis, anæmia or myocardial degeneration with palpitation, extrasystoles and breathlessness on exertion.

Etiology.—Chronic cholecystitis may occur at any age, but is frequent in the young. It follows (1) biliary stasis from sedentary habits, insufficient exercise or constipation; (2) infection, which takes place usually (*a*) by the blood-stream, but may spread (*b*) by direct extension from pre-existing hepatitis by way of the lymphatics, or (*c*) by ascent up the bile-ducts from the duodenum, and (3) disturbance in cholesterol metabolism.

Diagnosis.—Persistence of symptoms of flatulence ("wind") is characteristic of chronic cholecystitis. In cases with reflex superacidity of stomach contents, symptoms may resemble those of *duodenal ulcer*. The pain, coming long after meals or in the early morning, is relieved by food and alkalies; but with gall-bladder disease the pain is less regular and is made worse by fats. *Spastic gall-bladder* has much the same symptoms, but is relieved by belladonna (Newman). In *intercostal neuralgia*, the tenderness is in the abdominal parietes, not deep: and with a painful *slipping costal cartilage*, there is local tenderness of the costal margin. With X-ray, there may be (*a*) opacity in gall-bladder region; (*b*) irregularity and fixation of the hepatic flexure; (*c*) with cholecystography, non-filling or irregular filling points to a diseased gall-bladder; negative shadows of stones may be seen. A barium meal will demonstrate any lesion in the adjacent pylorus or duodenum. Duodenal intubation may show pus cells or bacteria (§ 351). The symptoms may resemble those of *psychoneurosis* and a careful enquiry into the environment and former history of the patient will help in diagnosis.

Course and Prognosis.—Gall-bladder disease must be thought of as a focus of infection in "toxæmic" states. If neglected, cholecystitis may lead to gall-stones, empyema or cancer of the gall-bladder.

Treatment.—Indications are (1) to prevent the stagnation of bile by exercise, plenty of fluids, magnesium sulphate, gr. 60 in water fl. oz. 2, first thing in the morning; (2) reduce bile cholesterol by a dietary of vegetables and carbohydrates, avoiding cream, egg-yolk, sweetbreads, brain, liver, kidneys and large meals; (3) treat infective foci by removal of diseased teeth or tonsils and attention to bowel, appendix, pelvic organs and nasal sinuses. Salicylate of sodium or hexamine combined with sodium bicarbonate and potassium citrate act as disinfectants of the biliary tract. Since the infection is intramural, drainage alone at operation will not effect a cure, and the gall-bladder should be removed. In older people palliative treatment should be recommended and a course of penicillin injections tried; in younger patients, operation.

IV. *The patient, a stout woman of sedentary habits, between fifty and sixty, who has suffered for years with "windy spasm" or mild colic, has a* CONSTANT OPPRESSION OR DISCOMFORT *in the* RIGHT HYPOCHONDRIUM, *loses weight and appetite, is* JAUNDICED *and has a palpable* TUMOUR IN THE GALL-BLADDER REGION. The disease is probably CANCER OF THE GALL-BLADDER.

§ **355. Cancer of the Gall-bladder** is uncommon. It is closely associated with cholelithiasis. Calculi are found in 70 to 90 per cent. of cases, and primary carcinoma of the gall-bladder occurs in 4 to 14 per cent. of all cases of cholelithiasis. It is much more common in women than in men (4 : 1).

Symptoms.—(1) The symptoms preceding the onset of carcinoma of the gall-bladder are those of the pre-existing cholelithiasis and cholecystitis. Biliary colic may occur, but usually there is only discomfort and heaviness in the right upper abdomen. (2) A tumour may be felt, at first round and smooth, but later nodular and hard. It moves with respiration. (3) Jaundice follows from pressure on the ducts by secondary glands or from catarrh. (4) Ascites occurs from pressure on the portal vein or secondary growths in the peritoneum.

Diagnosis.—The presence of a hard, nodular, progressively increasing tumour in the gall-bladder region of an elderly woman is suggestive. Gall-stones may cause inflammatory thickening round the gall-bladder, but enlargement of the gall-bladder is in favour of growth. Jaundice may come on suddenly with diarrhœa and vomiting, simulating infective hepatitis, but is progressive. Carcinoma of the stomach or hepatic flexure of the colon may cause confusion.

Treatment.—Extirpation by total removal offers the only hope of recovery. Medical treatment must be merely palliative.

THE SPLEEN

There is still some doubt as to the precise part played by the spleen, and symptoms may be altogether absent when it is diseased. Great diminution in size has been found *post-mortem* without any symptoms during life. The duties of the spleen are still uncertain; it does not appear to have an internal secretion as do the thyroid, suprarenal, and pituitary glands. One of its main functions is connected with the reticulo-endothelial system, of which it forms an important part. The reticulo-endothelial cells are widely distributed throughout the body. In certain diseases of the spleen, and after splenectomy, some of the functions of the spleen can be carried out by the other parts of the reticulo-endothelial system. In embryonic life the spleen is concerned with the formation of red and white blood corpuscles. In certain of the "blood diseases" in which it is enormously enlarged it resumes these functions. In the adult it is an important site of formation of lymphocytes and monocytes. It also deals with the removal from the circulation of old red cells, of pigments and parasites as in malaria. It enlarges during digestion, and owns muscle fibres which give it the power of rhythmical contraction, by which it can force its store of red blood cells into the circulation, *e.g.*, after hæmorrhage. In all probability the spleen is in some way necessary to the proper fulfilment of the digestive processes.

The spleen may show various congenital abnormalities. Of these the commonest is the presence of accessory spleens; less common are multiple spleens and a multilobular organ.

PART A. SYMPTOMATOLOGY

§ 356. In addition to the local pain and discomfort due to enlargement, the symptom which is found to be most constantly associated with disease of the spleen is *anæmia*, the various causes of which are discussed elsewhere (§ 535). From this arise symptoms which include *extreme pallor* of the skin, great *weakness*, and *alterations in the blood-cells*. The size of the spleen is not necessarily a measure of the severity of the symptoms. Thus in "ague cake," for example, great enlargement takes place without any symptom beyond the inconvenience due to the size of the organ. In other instances a large spleen may, by simple pressure or by the formation of adhesions, give rise to signs of disease in the neighbouring organs, especially the stomach. Pain and local tenderness accompany acute enlargements, and there may also be pyrexia and vomiting. The liver and spleen are often enlarged together; one may precede the other, or both may be results of a common cause.

PART B. PHYSICAL EXAMINATION

The only physical sign which can be relied upon as diagnostic of splenic disease is enlargement of the organ, and this is most readily made out by **Palpation.** When the spleen is enlarged, the anterior edge, being free, makes its way downwards and forwards towards the umbilicus. The *notch* in the anterior border is so characteristic that it forms a strong point in diagnosis of any splenic tumour. METHOD.—Stand on the right side of the patient, who should be lying on his back. Pass the left hand across the abdomen, and lay it posteriorly over the eleventh rib on the left side; then place the right hand flat upon the anterior surface of the abdomen, with the tips of the fingers just below the eleventh rib. By gently dipping down into the abdomen, and tilting the organ upwards with the left hand during inspiration, the splenic edge and its notch may be felt. It is more readily palpated when the patient draws a deep breath. Normally, the spleen cannot be detected by palpation, and even slight enlargements may not always be appreciable. An enlarged spleen always has a space between its posterior edge and the erector spinæ behind, into which the fingers can be dipped—at any rate, in spare subjects. *Fallacies.*—Without being enlarged, the spleen is readily palpable when it is displaced downwards, or is "floating." It is sometimes displaced downwards in cases of deformed chest (*e.g.*, rickets), large pleural effusions, and emphysema.

It is important to notice the CHARACTER of the enlarged spleen: a *soft* spleen may be due to some recent cause, *e.g.*, septicæmia or typhoid fever; a *firm* spleen to a disease of longer standing, *e.g.*, pernicious anæmia. A

hard spleen indicates fibrotic changes in the organ. Also VARIATIONS IN SIZE should be recorded in relation to the left costal margin or the umbilicus.

The **Percussion** of the spleen is attended with some difficulty. The organ is situated in the left hypochondrium, between the upper border of the ninth rib and the lower border of the eleventh ; and roughly between the mid-axillary and scapular lines (Fig. 41, § 108). It extends obliquely forwards and downwards nearly to the costal margin and lies wholly beneath the ribs : the upper third is overlapped by the lung. Percussion does not afford a very accurate means of investigation, but if it is desired to make use of this method of detecting splenic enlargement, it is best to percuss along the length of the 11th left rib and at the end of an expiration, because the spleen is then less covered by lung.

X-ray examination may be carried out.

Fallacies.—The dulness of *splenic enlargement* may be simulated by pleural effusion or consolidation of the left lung. The area of splenic dulness may be *diminished* by emphysema of the lungs, or distension of the stomach or colon by gas. The splenic dulness may be absent when there is a wandering spleen, or with congenital absence.

§ **357.** SPLENIC ENLARGEMENTS have seven chief characteristics : (1) The splenic *notches* are felt on its anterior border ; (2) the mass moves with respiration if not bound down by adhesions ; (3) it is dull to percussion because the resonant colon does not lie in front of splenic tumours, as it does in front of renal tumours. (4) It is palpable just under the anterior abdominal wall. (5) It is impossible to palpate above the organ, as it comes down beneath the left costal margin. (6) An enlarged spleen rarely crosses the midline above the umbilicus. (7) When an area of dulness is due to splenic enlargement, its outline *resembles in shape* that of the normal spleen. (8) It is distinguished from neoplasms of the peritoneum, stomach, intestines, etc., by its smooth and firm surface. Irregular enlargements of the spleen are rare, and can only be diagnosed after careful examination has excluded disease of other viscera.

Splenic enlargements or tumours may have to be diagnosed from the following conditions : (1) *Renal tumours*, and especially movable kidney, in which there is resonant intestine in front of the tumour, and absence of resonance in the flank ; (2) *enlargement of the left lobe of the liver*, in which the dulness is continuous with that of the right lobe, whereas splenic dulness rarely reaches to the middle line ; (3) *cancer of the cardiac end of the stomach*, in which the dulness is less absolute, and there is " coffee-ground " vomiting, etc., and the splenic notch is absent ; (4) *ovarian tumour*, which (i.) will have grown from below upwards, (ii.) the hand cannot be pushed between the tumour and the pelvic brim as it can in the case of a splenic tumour, and (iii.) can be felt on vaginal examination ; (5) *accumulation of fæces*, in which (i.) the tumour has an irregular outline, (ii.) doughy consistence, and (iii.) a course of purgatives and enemata will remove it ; (6) *post-peritoneal tumour*, in which (i.) there is no notch, and (ii.) no resonance behind it ; (7) *abdominal aneurysm*, when of sufficient

size to be mistaken for the spleen, is attended by pain in the back, and evident expansile pulsation; (8) *deep-seated abscess in the abdominal parietes* is tender, has a vague irregular outline, and is situated more superficially than a splenic tumour. In (9) *cancer of the splenic flexure* of the colon the mass varies from day to day and there will be intestinal symptoms; (10) *pancreatic* and *suprarenal* tumours and *perinephric abscess* may give rise to difficulty. (11) Rare causes of error are localised *tuberculous* masses and the thickened colon of *bilharziasis*.

PART C. DISEASES OF THE SPLEEN

§ **358.** The diseases of the spleen are all—if we except the relatively rare cases of wandering spleen and atrophy—comprised under the causes of **enlargement of the organ,** and its **diagnosis** therefore becomes a matter of considerable importance. Enlargement is detected by palpation aided by percussion as above mentioned. The mechanical effects of pressure, when the spleen is very much enlarged, are mainly dyspnœa and gastro-intestinal disturbance. These may be aggravated by attacks of perisplenitis, with acute pain locally, vomiting, pyrexia, and sometimes diarrhœa. Œdema of the base of the left lung is not uncommon.

The **Causes of Enlargement of the Spleen** are most readily differentiated according as they depend upon or are associated with the following:

I. Acute infections.
II. Chronic infections.
III. Portal obstruction or congestion.
IV. Blood diseases.
V. Parasitic and tropical diseases.
VI. Infancy and childhood.
VII. Irregularity of the surface of the spleen.

Method of Procedure.—As pointed out in Part A, advice is rarely sought for symptoms directly pointing to the spleen. Frequently the spleen is found to be enlarged when the patient is being examined for disease elsewhere. It should be remembered that in many diseases the detection of an enlarged spleen may be an important clue to the diagnosis.

Inquiry should be made as to the HISTORY. Thus residence abroad suggests malaria; prolonged suppuration, lardaceous disease; fever and rigors, the presence of some pyæmic cause.

The AGE of the patient is important (see VI. below); in childhood certain conditions are common which are rare in adults.

The TEMPERATURE aids the diagnosis of certain infections.

EXAMINATION OF OTHER ORGANS may render the diagnosis easy. The condition of the LIVER is of especial significance in several diseases. Thus a large liver, jaundice, and a normal spleen point to gall-stones or cancer, but if the spleen as well as the liver is large, these symptoms suggest cirrhosis or other obstruction. A very enlarged spleen with but slightly enlarged liver suggests some of the "blood diseases" which can be accurately differentiated only by an EXAMINATION OF THE BLOOD (§ 527).

I. **Acute Infections.**—Almost all acute infections are apt to be accompanied by slight enlargement of the spleen, and as far as the acute specific fevers are concerned this is usually of little clinical significance. The enlarged spleen often feels soft, is unaccompanied by local symptoms and is especially found with typhoid, abortus and typhus fevers. Sometimes, and particularly in TYPHOID fever, a splenic abscess may complicate the original condition. In such a case local symptoms of tenderness and pain will draw attention to the spleen. Again, these symptoms may arise in the course of some systemic infection, and be due to suppuration supervening in the area affected by an EMBOLISM or in some pre-existing cyst or tumour. Embolism due to cardiac disease causes (i.) acute sudden pain, and (ii.) local tenderness due to perisplenitis. Embolism due to pyæmia is usually known by the presence of the causal condition. In such diseases as leukæmia, in which the massive enlargement of the spleen is a prominent feature, the organ is liable to attacks of ACUTE CAPSULITIS, which may give rise to difficulty in diagnosis unless the possibility of their presence is borne in mind. A friction rub, due to localised peritonitis, may be audible during the acute attacks.

The *diagnosis* of the cause may be very difficult, but should be solved by patient investigation. Meanwhile, expectant treatment is to be adopted, and consists of rest in bed, attention to the bowels and hot applications to the spleen (for pain). If the attack does not subside and the local signs become worse, the advisability of surgical interference must be considered. Fortunately this is rarely needed, and the attacks tend to resolve in a few days, leaving adhesions which may lead to trouble later.

II. **Chronic Infections.**—(1) INFECTIVE or MALIGNANT ENDOCARDITIS (§ 50) may give rise to embolism, which causes acute symptoms, or to a more chronic enlargement not wholly due to congestion, and due to the accompanying septicæmia. The symptoms in the latter case may be exactly similar to those of splenic anæmia (§ 544), and may occur when there is no suspicion of cardiac trouble. The importance of this lies in the fact that it is possible to remove the spleen with advantage to the patient in splenic anæmia, but the operation should not be performed in endocarditis. ABSCESS of the spleen may also occur in the course of this disease.

(2) SYPHILIS may cause a uniform enlargement of the spleen in the early stages of the toxæmia. Later, both spleen and liver may enlarge, and the diagnosis be difficult. Ascites and anæmia may supervene.

(3) TUBERCULOSIS may occur as miliary tubercle, as an abscess, as a capsulitis, or even as multiple tuberculomata. In no case is it likely to be diagnosed apart from the existence of tuberculosis elsewhere: X-ray examination of the lungs may reveal chronic miliary tuberculosis, which may resolve in course of time after splenectomy. It is rarely primary in the spleen, and is then an exception to the rule; if diagnosed it may be operated upon. In some cases of splenic tuberculosis there is a

marked polycythæmia instead of the anæmia which usually accompanies tuberculosis.

(4) CHRONIC SEPTIC SPLENOMEGALY resembles Splenic Anæmia except in that it may present a leucocytosis. It is especially common in tropical climates after dysentery or other intestinal disorders. The prognosis is good if the causal sepsis can be eradicated. It does not as a rule lead to hæmatemesis. The importance of the condition rests in the fact that it is liable to be confused with Splenic Anæmia and a bad prognosis given in consequence.

(5) In the absence of fuller knowledge, BILIARY CIRRHOSIS (HANOT'S DISEASE) may come under this heading. The spleen may be enlarged before the liver in some cases. The diagnostic signs are considered in § 343.

(6) AMYLOID disease of the spleen is becoming very rare. It is known by: (i.) There is usually a history of syphilis, phthisis, or of chronic purulent discharge; (ii.) the liver shows signs of amyloid disease, and diarrhœa may be present, due to involvement of the intestines; (iii.) the spleen may be much larger than is usual with acute or chronic infections.

III. **Portal Obstruction or Congestion.**—Any cause of portal obstruction, of whatever degree, will naturally lead to congestion in the whole of the splanchnic area, and in this the spleen will share. Thus the spleen is slightly enlarged in (1) CARDIAC and CHRONIC LUNG DISEASE, with backward pressure in the venous system. The obstruction may be more absolute, as in (2) THROMBOSIS of the INFERIOR VENA CAVA. In this case the enlargement of the spleen may reach a greater degree than in congestive conditions of the liver, and where the thrombosis affects only the splenic vein the hypertrophy may be extreme, and the symptoms conform to those of splenic anæmia (of which, according to some authorities, it is the chief cause) (§ 544). (3) CIRRHOSIS of the LIVER (§ 342) is associated with splenic hypertrophy. (4) In SYPHILITIC fibrosis, however, the liver and spleen are usually simultaneously affected. (5) One cause of splenic congestion and hypertrophy must be mentioned, although of great rarity —viz., TORSION of the splenic pedicle. This may occur when the spleen is displaced by its increased weight (in splenomegaly), or when it has an unusually long pedicle, as in splenoptosis and wandering spleen. It is unlikely to be diagnosed except by operation.

§ **359.** IV. "**Blood Diseases,**" or diseases of myeloid and lymphatic tissue. They merit individual remark, but for full descriptions the reader is referred to other paragraphs. In almost all of these the acute attacks of capsulitis above mentioned are apt to occur.

(1) PERNICIOUS ANÆMIA is often and (2) SIMPLE HYPOCHROMIC ANÆMIA sometimes associated with slight enlargement of the spleen. This rarely reaches a large size: the presence of a large spleen would be a sign that such a diagnosis requires revision.

(3) In SPLENO-MEDULLARY LEUKÆMIA (§ 543) the spleen is characteristically enormous, but it is to be remembered that in LYMPHATIC LEUKÆMIA

and in (4) CHLOROMA it may be just as large, even reaching to the pelvis. In the latter diseases some degree of enlargement is almost invariable. These diseases are diagnosed largely by the blood examination.

(5) LYMPHADENOMA (§ 572) is known by: (i.) One or more groups of enlarged lymphatic glands are present; (ii.) the splenic enlargement is usually not excessive until a late stage of the disease.

(6) SPLENIC ANÆMIA (§ 544) could hardly be diagnosed without the enlargement of the spleen, which usually reaches very considerable proportions. As will be gathered from the remarks made above, this disease is no doubt destined to be subdivided into several groups when further knowledge is available. In the tropics it may be simulated by kala-azar and other diseases. There is a form of splenic anæmia which is found particularly in infants, and tends to occur in twins. In this the prognosis is better than in the adult form; the blood changes differentiate it.

(7) ACHOLURIC JAUNDICE is associated with some enlargement of the spleen in the majority of cases. It is known by: (i.) it is often a disease of family incidence, (ii.) the presence of jaundice, and (iii.) the characteristic blood changes (§ 328).

(8) ERYTHRÆMIA is diagnosed by (i.) polycythæmia, which may reach a very high degree, and (ii.) the cyanosis, weakness, and paræsthesiæ to which it gives rise (§ 31).

V. **Tropical Diseases.**—MALARIA and KALA-AZAR are the most common. In acute malaria the enlargement is not very great, but after many attacks it may be enormous. A history of attacks of malaria occurring in a person who has been abroad leads one to suspect the cause of the splenic enlargement; but the diagnosis is made certain by finding the parasite in the blood. Anæmia is common, and periodic fever occurring on alternate days or every third day is suggestive of malaria. In kala-azar the spleen is usually large, and is rendered the more prominent by the emaciation of the subject. The diagnosis rests on the discovery of the parasite by blood culture on rabbit blood agar medium incubated at room temperature, or in the material obtained by liver, sternal or spleen puncture, while the formol-gel reaction in the serum is characteristic. Only occasionally is it possible to demonstrate Leishman-Donovan bodies by the microscopical examination of blood smears. TRYPANOSOMIASIS and RELAPSING FEVER may also cause splenic enlargement, while splenomegaly is not uncommonly associated with INTESTINAL BILHARZIA in Japan (*S. japonica*) and Egypt (*S. mansoni*).

Rarer **parasitic** causes are HISTOPLASMOSIS and TOXOPLASMOSIS.

VI. In **Infancy** and **Childhood,** RICKETS (§ 596) is a common cause of slight enlargement of the spleen. It may depend on the catarrh of mucous membranes often associated with rickets, especially as in children the spleen enlarges much more readily than in adults, and for causes inadequate in an older person. Congenital SYPHILIS and TUBERCLE may

be present in children, and are recognised by signs of the disease elsewhere; in syphilis the liver also is enlarged. A special form of splenomegaly associated with ANÆMIA in infants (§ 551. IV) has been mentioned above; there is also a special form of KALA-AZAR in infants, encountered in the Mediterranean basin. In cyanosis from CONGENITAL HEART DISEASE there may be marked enlargement of the spleen. Congenital ERYTHRÆMIA is also described.

VII. **Irregularity** of the surface of the enlarged spleen. This group includes quite a different class of disease to that above mentioned. The most important cause of enlargement is sarcoma, for there is some hope of cure if the spleen be removed early enough. It is rare, and usually occurs in children or young adults. It can only be diagnosed by exclusion. Other new growths are even more rare, and include lymphangioma, fibroma, pulsating angioma (which may give rise to suspicions of aneurysm), secondary cancer, and cysts such as dermoids, and congenital polycystic disease.

HYDATID cyst in the spleen may be diagnosed by (i.) the presence of marked eosinophilia in a person who (ii.) has resided in an affected country, (iii.) the serum and intradermal reactions (§ 347), and (iv.) sometimes by the presence of cysts elsewhere; (v.) the cyst may present the characteristic thrill on palpation.

LYMPHADENOMA may give rise to irregular enlargement, and certain congenital malformations are irregular.

The *Treatment* and *Prognosis* of splenic enlargement depend, for the most part, on the primary condition. The treatment of lardaceous disease and of hydatid is given under Hepatic Disorders (§§ 346, 347). The treatment of "Ague Cake" consists of (i.) removal to a non-malarious district, and the administration of anti-malarial drugs; (ii.) unguentum hydrargyri iodidi dilutum, rubbed over the splenic area, is a remedy which may be of value; (iii.) violent movement must be forbidden, as the spleen may rupture. In chronic syphilitic splenomegaly arsphenamine therapy has been curative.

§ 360. **Wandering Spleen** (Floating, Dropped, or Dislocated Spleen, Splenoptosis) may be readily mistaken for enlargement of that organ when met with in the lesser degrees of displacement. But when the dislocation is, as generally happens, considerable, it is more often taken for a floating kidney. However, the presence of the notch, the fact that it can be made to recede upwards and that it comes down in front of the colon, aid the diagnosis. The condition is mostly met with in multiparæ with pendulous abdomen. It may be accompanied by nervous symptoms, though these are less constant than in dislocation of some of the other viscera. If troublesome, the condition may be relieved by removal of the organ, an operation which has been performed several times with good results.

Atrophy of the Spleen is, as a rule, unattended by symptoms. It is, as Bristowe said, a condition not infrequently met with. It may be congenital, but its commonest causes are: I. CIRRHOSIS of the spleen, due to an increase in the interstitial tissue, and usually secondary to cirrhosis of the liver, and II. CONTRACTION OF THE FIBROUS CAPSULE, usually of syphilitic origin. The syphilitic deposits in the capsule of the spleen sometimes take on a cartilaginous change, and form plates of cartilage. It is often only found at autopsy, being unattended by symptoms during life.

§ 361. The following are the indications for SPLENECTOMY: (i.) Rupture of the spleen, torsion or similar acute emergency. These constitute an absolute indication in all cases in which the patient is likely to survive the operation. (ii.) Tumours or

abscess of the spleen which cannot be dealt with by less radical measures. (iii.) Persistent attacks of peri-splenitis or other disabilities directly dependent on the size and weight of the organ. These are a sufficient indication in nearly all cases but do not allow of operation in leukæmia or erythræmia. (iv.) Cases in which splenectomy has been shown to exercise distinct benefit on the general disease present: *e.g.*, early cases of splenic anæmia, chronic infective splenomegaly and acholuric jaundice. Cases of pernicious anæmia are not benefited, nor does Hodgkin's disease fall within this group. (v.) Cases in which the circulation through the liver is embarrassed, as in cirrhosis, and in which diminution of the volume of blood passing through the portal vein may be of advantage. Banti's syndrome falls in this group, as well as a proportion of anomalous cases described as Hanot's cirrhosis.

Of the blood diseases only three—Splenic Anæmia or Chronic Infective Splenomegaly, Acholuric Jaundice, and Purpura Hæmorrhagica (§ 653) with a great diminution in the blood-platelets—demand treatment by splenectomy. Splenectomy is undertaken only when it is evident that disease is impairing, or likely to impair the general health and working ability of the patient. Then the operation should be done early, and particularly if there has been even a single attack of peri-splenitis. Peri-splenitis causes adhesions and one attack will certainly be followed by others. What is, in skilled hands, a fairly simple and safe operation will then become much more difficult and dangerous and the patient should not be allowed to drift on in ignorance of this fact. In no case should a patient be submitted to operation without adequate preparation, by transfusion if necessary. Pulmonary collapse at the left base is especially to be guarded against after operation—by deep breathing, by avoiding constriction of the chest with bandages, etc.

CHAPTER XIII

THE URINE

A PROPER understanding of the pathological processes in the kidneys is facilitated by recalling certain physiological facts. The unit of the kidney is the nephron, comprising the glomerulus, the absorbing tubules and the collecting tubule. Under the pressure of the blood in the glomerular capillaries, the glomeruli filter a fluid similar to blood plasma, minus its proteins. The greater part of this fluid is reabsorbed into the blood stream from the tubules ; a concentrated residue of urine is left, containing the waste products. To accomplish this, only a few nephrons are in action at a time for short periods. In diseased conditions there may be a generalised inflammation or degeneration of the nephrons (nephritis or nephrosis), or a patchy destruction of groups of them owing to narrowing or obliteration of their blood supply (arterio-sclerosis).[1] Such is the enormous reserve power of the kidneys that a large part may be destroyed and yet the remainder can carry out the necessary work ; but the reserve power is diminished. With further destruction each remaining nephron is called into continuous activity. To maintain the action of the glomeruli, the blood pressure rises. The blood is still cleared of waste products (*e.g.*, urea) to a normal extent, but, as the power of the absorbing mechanism of the tubules is diminished, a larger volume of the dilute urine passes down them. With still further damage to the nephrons, less fluid is secreted by the glomeruli, and so the blood now contains an excess of waste products (uræmia) and the volume of urine falls.

When the glomeruli are inflamed, albumen and even blood leak into the urine, as in acute nephritis. Some forms of renal disease are associated with general œdema and with intense albuminuria ; modern research suggests that the primary defects are not in the kidneys, but in the composition of the blood and in the semipermeability of the capillaries throughout the body (in which the renal capillaries share). The loss of such a large quantity of albumen from the blood may so lower the osmotic pressure of the blood plasma that fluid is attracted from the blood into the tissues. The cardinal features of renal disease are therefore best seen by examination of the urine, aided later by examination of the blood and the tissues. In practice it is not always possible to separate kidney diseases proper from disorders of other parts of the urinary tract, because changes

[1] Richard Bright in 1836 described the acute and chronic forms of inflammation of the kidneys, associated with albuminuria and hyperpiesia. Unfortunately the term chronic Bright's disease is still sometimes loosely and incorrectly applied to high arterial pressure and its associated symptoms, and so is better avoided.