A handbook of the diseases of the liver, biliary passages, and portal vein / by Henry R. Ruckley.

RUCKLEY ON THE LIVER

A HANDBOOK

OF THE

DISEASES OF THE LIVER, BILIARY PASSAGES,

AND

PORTAL VEIN,

BY

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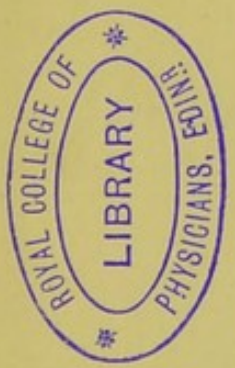

Discere si quæris, doceas, sic ipse doceris:
Nam studio tali tibi proficis atque sodali.

HIGH WYCOMBE:

F. WESTFIELD, 10, HIGH STREET.

LONDON:

W. KENT & CO., 23, PATERNOSTER ROW.

TO

EDWARD DILLON MAPOTHER, M.D.,

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FELLOW AND EX-PRESIDENT OF THE ROYAL COLLEGE OF SURGEONS,

IRELAND;

THIS WORK IS DEDICATED BY HIS FORMER PUPIL

IN ADMIRATION OF HIS TALENTS AS A

TEACHER, A SCIENTIFIC SURGEON, AND AN AUTHOR.

PREFACE.

IN these days when new medical books and new editions are constantly being published until the bookseller's shelves groan under the weight thereof, it may very reasonably be asked—"Why publish another?" My answer is that this little book was suggested by my own necessity. After a careful perusal of almost all the modern works treating on Diseases of the Liver, I felt that there was still wanting a small handy volume which would supply concisely the kind of information required by that large class of men of which I am an individual,—namely, students and general practitioners.

Far from aiming at the production of a work which would supersede the classical writings of Budd, Frerichs, Murchison, Von Schueppel, &c., I set myself the more humble task to introduce a very difficult subject to the student, and thus prepare his mind for the digestion of such great works as those of the authors just named, which I earnestly commend to his diligent study; for although it is quite true that narrow specialisms in the practice of medicine are to be deprecated, yet there can be no doubt of the great benefits conferred upon medical science by the work of specialists; and the man who would succeed in his vocation of relieving the sick, it behoves him to study all diseases from the specialist's point of view, and not be content with the smattering of knowledge to be gained from mere handbooks.

In the little volume which I now present to the student of medicine I have endeavored to be as concise as the clear statement of facts will allow, to avoid ambiguities of technicality and nomenclature as much as possible, and above all things to eschew dogmatism, which ought to have no place in medical literature.

Pathological anatomy has received a fair share of attention; not that I deem pathological changes to be the essence of disease, but rather the visible effects of some disturbance of the Vital Power. For example: in a forest we may observe the effects of the storm which has passed over it, in the fallen trees and shattered timber, but we can nowhere find the storm. The latter, like disease, is no real entity but a mere relative signification. Nevertheless, pathological anatomy is a useful and important study, serving as it does to illustrate the relation between the structural changes wrought in the body by disease and the phenomena attending these changes which we call the signs and symptoms of disease.

It is not possible to draw a line of demarcation which would denote in the ever-varying condition of the organism where health ends and disease begins; yet health may be defined to be that condition of the body in which the Vital Power continues to work for the preservation of the integrity of the organism, and succeeds.

Taking the above as a definition of health it follows that disease is that condition of the body in which the Vital Power is perverted (either in excess or defect), annulled in some locality, or modified in some, as yet obscure manner, the tendency of which is ultimately to destroy the organism, or a part thereof.

Between health and disease there is an intermediate condition, namely, convalescence, which may be defined as that condition of the body in which the Vital Power, having previously suffered perturbation, is in the act of settling down to its normal condition.

HIGH WYCOMBE,
1883.

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INTRODUCTION.

A Brief Summary of the Anatomy and Physiology of the Liver.

THE liver is the largest gland in the body, weighing in the healthy adult subject from 50 to 60 ounces avoirdupois, and is more constantly present under some form or other, throughout the Animal Kingdom, than any other gland.

In the human subject it is situated in the upper part of the abdominal cavity, called the *right hypochondriac* region, and extending across the *epigastric*, into the *left hypochondriac* region.

In the living subject the liver is protected, on the right by the six or seven lower ribs; and in front by the cartilages of the same, and by the ensiform cartilage—the diaphragm, of course being interposed. Being suspended by ligaments to the diaphragm above, and supported below, in common with the rest of the viscera, by the abdominal muscles, the situation of the liver is modified by the position of the body and by the movements of respiration. In the upright or sitting posture, the liver reaches slightly below the margin of the thorax; but in the recumbent position, the gland ascends an inch or an inch-and-a-half higher up, and is entirely covered by the ribs, except a small portion opposite the sub-sternal notch. Again, during a deep inspiration, the liver descends below the ribs, and in expiration retires behind them. The fundus of the gall-bladder touches the abdominal wall immediately beneath the margin of the thorax, opposite the tip of the tenth costal cartilage.

By means of the diaphragm, the liver is separated from the concave base of the right lung, the thin margin of which descends so as to intervene between the surface of the body and the solid mass of the liver.

The healthy human liver is a solid organ of a dull reddish-brown colour, with frequently a dark purplish tinge along the margin. In transverse diameter it measures about twelve inches, and between six and seven inches from its posterior to its anterior border, and about three inches from above downwards at its thickest part, which is towards the right and posterior part of the gland.

The upper surface of the liver is smooth and convex—covered by peritoneum, and accurately adapted to the vault of the diaphragm above, and is covered to a small extent in front by the abdominal parieties.

The under surface is unevenly concave, being superficially divided by fossæ or fissures into five lobes, which are named the right, left, spigelian, caudate or tailed lobe, and the quadrate or square lobe. The right lobe is of a long square form ; it is much larger and thicker than the left, which is nearly triangular in outline, and constitutes only one-fifth or one-sixth of the entire gland. The other three lobes are small, and might be said to form part of the right lobe, on the under surface of which they are situated.

The circumference or border of the liver, at which the two surfaces meet, is thick and rounded behind and to the right, but becomes gradually thinner towards the left and in front, which constitutes the anterior free margin of the liver, which is also the most moveable part of the gland.

Relations.—The left lobe of the liver is supported on the pyloric extremity and anterior surface of the *stomach*, on which it moves freely. When the stomach is quite empty, the cardiac end of that viscus may be overlapped by the liver. To the right of the longitudinal fissure, the liver rests and moves freely upon the first portion of the *duodenum*, and upon the hepatic flexure of the *colon*. Further back it is in contact with the anterior part of

the *right kidney*, and supra-renal capsule, by which it is indented. The posterior border of the liver is the most fixed part of the organ, being firmly attached by areolar tissue to the diaphragm, the peritoneum being here reflected away from the liver on to the diaphragm so as to form the coronary ligament. This border of the liver is curved opposite the projection of the vertebral column, and has a deep groove for the passage of the ascending cava, called the fossa of the vena cava.

Of the five fossæ or fissures of the liver, the (1) transverse or portal fissure is the most important, because it is here that the great vessels and nerves enter, and the hepatic duct passes out. It is situated across the middle of the right lobe, somewhat nearer its posterior than its anterior border, occupying its inner half only. The vessels entering at this fissure are the *vena portæ* which conveys the blood from which the bile is secreted, and the *hepatic artery* by which aërated blood is supplied for the nutrition of the capsule, the coats of the ducts and blood vessels, as well as for other parts of the organ. The *bile duct* also emerges from the liver in this fissure, to the right of the portal vein and hepatic artery. These three vessels are accompanied by numerous lymphatic vessels and nerves, and surrounded by an areolar tissue named the capsule of Glisson, which here dips down into the substance of the liver and accompanies the vessels for some distance. (2. 3.) The *longitudinal fissure* which separates the liver into the right and left lobes, is divided by its junction with the transverse fissure into two parts; the anterior named the *umbilical* fissure contains the remnant of the umbilical vein of the fœtus which constitutes the round ligament of the liver. The posterior part is named the *fossa ductus venosi* as it lodges the ductus venosus in the fœtus, in adult life degenerated into a slender cord or ligament. (4) The fossa of the vena cava has been already described. (5) The last remaining fossa (fossa cystis felleæ) is that in which the gall-bladder is lodged.

The so-called ligaments of the liver (five in number) are, with one exception (the round ligament) merely duplicatures of the peritoneum.

By far the most important attachment of the liver to the surrounding parts is the *gastro-hepatic omentum*, between the layers of which the hepatic artery, nerves and portal vein ascend to the liver, and the common bile-duct descends to open into the duodenum.

The Hepatic artery, a branch of the cœliac axis, is a small vessel in comparison to the size of the organ to which it is distributed. The nerves of the liver are derived partly from the cœliac plexus, and partly from the pneumogastric nerves, especially from the left pneumogastric, which will account for pain in the shoulders, particularly in the left shoulder in connection with hepatic disease. The nerves enter the liver at its transverse fissure, supported by the hepatic artery and its branches.

The Vena Portæ conveys by far the greater part of the blood which passes through the liver—and this is its chief peculiarity as a gland. It is formed by the union of nearly all the veins of the chyle-making viscera, viz., those from the stomach, the pancreas, spleen, omentum, and mesentery, and on entering the liver it divides up again into branches like an artery. It is from the blood so collected and conveyed that the bile is secreted in the liver.

The portal system may be likened to a tree; its roots ramifying throughout the chylopoietic viscera, and its branches spread out in the tissues of the liver, the trunk being represented by the Vena Portæ itself.

The Excretory Apparatus of the liver consists of the hepatic duct, the cystic duct, the gall-bladder, and the common bile-duct.

The hepatic duct descends to the right within the gastro-hepatic omentum, in front of the portal vein, and to the right of the hepatic artery. Its diameter is about two lines, and its length nearly two inches. At its lower end it meets with the cystic duct, coming down from the gall-bladder, and the two ducts uniting at an acute angle form the common bile-duct.

The cystic duct is about an inch in length. It runs downwards and to the left, thus forming an angle with the direction of the

gall-bladder, and unites with the hepatic duct to form the common bile-duct.

The gall-bladder is a pear-shaped membranous sac about three or four inches long, rather more than an inch across at its widest part, and capable of containing about eight or ten fluid drachms. It is lodged in a fossa on the under surface of the right lobe of the liver, so that its large end or fundus, which projects beyond the anterior border of the liver, is directed downwards, forwards, and to the right, while its body and narrow end or neck are inclined in the opposite direction. The upper surface of the gall-bladder is attached to the liver by areolar tissue and vessels, its under surface is free and covered by peritoneum, which is here reflected from the liver, so as to include and support the gall-bladder. The fundus of the gall-bladder which is free, projecting, and always covered by peritoneum, touches the abdominal parieties immediately beneath the margin of the thorax, opposite the tip of the tenth costal cartilage. Below it rests on the commencement of the transverse colon, and further back it is in contact with the duodenum, and sometimes with the pyloric extremity of the stomach.

These relations of the gall-bladder with the surrounding viscera are very important to be remembered in connection with the discharge of gall-stones.

The neck of the gall-bladder forms two curves upon itself like the letter S, and then having become much contracted, and changed in its general direction altogether, bends downwards and ends in the cystic duct.

The common bile-duct is the largest of the three ducts, and is formed by the union of the two other ducts. It is from two to three lines wide, and nearly three inches in length. It descends backwards, and having reached the descending portion of the duodenum, it continues downwards on the inner and posterior aspect of that division of the intestine, covered by, or included in the head of the pancreas, and for a short distance, in contact with the right side of the pancreatic duct. Together with that duct, it then perforates the muscular wall of the intestine, and after

running obliquely for three-quarters-of-an-inch between its several coats, and forming an elevation beneath the mucous membrane, it becomes somewhat constricted, and opens by a common orifice with the pancreatic duct on the inner surface of the duodenum, at the junction of its second and third portions, rather more than three inches below the pylorus.

Besides peritoneal investment, the gall-bladder has two distinct tunics, an areolar and a mucous. The areolar coat is strong and consists of dense shining white fibres, which interlace in all directions. In recently killed quadrupeds, the gall-bladder contracts on the application of a stimulus; and in the larger species, as well as in man, muscular fibres of the plane variety have been found in its coats. The mucous coat is elevated upon its inner surface into innumerable ridges, which, uniting together into meshes, give the inner surface of the gall-bladder a peculiar honey-comb appearance. The whole of the mucous membrane is covered by columnar epithelium, and it secretes an abundance of viscid mucus.

The gall-bladder is a reservoir for such bile as is not immediately required in digestion. The bile contained within it becomes darker and inspicated, receiving some addition of mucus, and becoming more ropy and viscid, but beyond this it undergoes no further change.

The structure of all the bile-ducts is alike. Their areolar coat is strong and distensible. The mucous membrane is provided with numerous glands.

Development.—The liver is formed in immediate connection with the intestinal tube by the thickening of the cells of the wall of that canal at the spot at which the hepatic duct is subsequently to discharge itself. This thickening increases so as to form a projection on the exterior of the canal; and soon afterwards the lining membrane of the intestine dips down into it, so that a kind of diverticulum is formed, surrounded by a mass of cells. When these cells have considerably increased in number, the exterior ones become metamorphosed into fibrous tissue to form the cap-

sule, aud those in the interior break down into ducts, which are developed in continuity with the cæcum derived from the intestine, and are lined by muscular and fibrous tissue developed from the primitive cellular blastema; while those which occupy the interevning space, and which form the bulk of the gland give origin to the proper secreting cells, which are now to come into active operation. As this is going on, the hepatic mass is gradually removed to a distance from the wall of the alimentary canal; and the cæcum is narrowed and lengthened, so as to become a mere connecting pedicle, forming in fact the main trunk of the hepatic duct.

In the human embryo, the formation of the liver begins about the third week, and is of very large size when compared with that of the body; and between the third and fifth weeks it is one-half the weight of the entire embryo. Up to the period of birth, the bulk of the liver relatively to that ot the entire body, is much greater than in the adult; the proportion being as 1 to 18 or 20 in the newly-born child, while it is about 1 to 36 in the adult.

The liver is engaged during fœtal life in the depuration of blood —most of the arterial blood coming from the placenta by the umbilical vein being conveyed to the liver by the vena portæ— but at the same time it is serving as a blood-making organ. The first Red corpuscles of the blood are formed in primordial cells of the germinal structure; and it is in the "vascular layer of the blastodermic vesicle, and in the mass of cells which constitutes the rudiment of the heart that this metamorphosis seems first to take place." The situation of the heart, and the course of the principal trunks of the "vascular area" are early marked out by the peculiar disposition of the aggregations of cells from which these organs are to be developed; and whilst the *outer* portions of these aggregations are transformed into the *walls* of the respective cavities, the *inner* portions seem partly to deliquesce, and partly to remain as isolated cells floating in the liquid thus produced. These isolated cells are the first blood corpuscles.

When the liver begins to be formed, this mode of formation ceases, and there takes place in the vessels of the liver a new

production of colourless nucleated cells, which are formed around free nuclei, and which undergo a gradual change (by the development of colouring matter in their interior) into red nucleated cells resembling those of the first blood. This new formation of blood corpuscles in the liver continues to take place during the whole of fœtal life.

Structure of the Liver.—When a torn surface of the liver is closely examined with the naked eye, it is seen to be made up of a multitude of small granular bodies, about the size of millet seeds, of an irregular form, and presenting a number of projecting processes upon their surfaces. These granular bodies are called lobules. The exterior of each lobule is said by some persons to be covered by a process of the capsule of Glisson, which dips down into the substance of the liver. This certainly is the normal condition in the pig's liver, in which the capsule of Glisson was first discovered, but not so in the human liver. Each of these lobules consists of a plexus of vessels derived from the branches of the portal vein, which after ramifying in its capsule, forming the interlobular plexus, enter at all points, and form a plexus within the lobule composed of radiating and transverse connecting vessels; this is called the intralobular plexus. These vessels having become reduced by division and sub-division to a capillary size, form a network from which arise the commencing radicles of the intralobular hepatic vein in the centre of the lobule. The interstices between the blood-vessels in each lobule are occupied by the commencement of the biliary ducts, and they also contain a peculiar substance composed of numerous microscopic nucleated cells. These cells are concerned in the secretion of the bile, and are named the *hepatic cells*.

The intralobular hepatic veins open into the sides of the subjacent subloblular veins; these uniting form larger and larger veins which converge towards the posterior border of the liver, and terminate in the vena cava, at the bottom of the fossa vena cavæ.

The blood from the hepatic artery after having fulfilled its

function of nutrition is conveyed into the portal vein and thence returns to the heart by the hepatic veins and inferior vena cava.

The biliary ducts emerge from the surface of the lobules and form an interlobular plexus of ducts ; these by uniting form larger and larger ducts which converge towards the transverse fissure of liver, and there end in the hepatic duct.

PHYSIOLOGY.—The hepatic cells mentioned above are nucleated cells, usually of a flattened spheroidal form, and from one 500 to one 2000 of-an-inch in diameter. The cell contents consist of yellow amorphous biliary matter with several oil-globules of various sizes. The materials of the biliary secretion are formed by these cells which line the interlobular extremities of the bile-ducts, either by exudation of the cell-contents, or by the delequescence of the cells themselves.

The Liver and the Kidneys furnish typical examples, the former of a *secreting* organ, and the latter of functions which are purely *excretory*. The liver by its cells elaborates from the portal blood substances which did not previously exist in it as such, and which are highly necessary in the economy of nutrition.

The Kidneys, on the other hand, merely strain from the blood effete matter which has been produced in the system by the metamorphosis of the animal tissues. The chief substance thus removed is urea, which it has been clearly proved is produced in the liver, passed into the blood, and excreted by the kidneys. Thus it will be perceived that the liver and kidneys are complemental of each other.

The process of *secretion* generally, is performed through the instrumentality of cell-growth. During their growth and development, the hepatic cells which line the interlobular extremities of the hepatic ducts, by a vital chemical process split up the albumenoid constituents of the portal blood into the elements of the bile on the one hand, which they pour into the hepatic ducts, by their subsequent decay or delequescence. On the other hand they form glycogen ($C_6 H_{10} O_5$) which is converted into sugar by a ferment in the blood, and nitrogenous products such as leucin

($C_6 H_{13} NO_2$) and tyrosin ($C_9 H_{11} NO_3$) which are ultimately resolved into urea ($CH_4 N_2 O$). These substances are returned to the blood, the sugar to be burnt in the system for the maintenance of animal heat, and the urea to be removed from the system by the kidneys as hurtful to the economy.

The most important function which the liver performs is obviously the secretion of *bile*, and by this means a considerable quantity of hydro-carbonaceous material is at least temporarily eliminated from the blood, to be used in the process of digestion and subsequently almost entirely re-absorbed into the system; the fæces normally containing very little, if any, bile in their composition, although their colour is in great part derived from bile, as is shown by their paleness when that secretion is not duly poured into the bowels.

The liver also exerts an assimilative or elaborating action on the freshly absorbed albuminous and saccharine constituents of the food, whereby they become more fitted for the nutrition of the body; and in the course of these assimilative changes, the activity of which is indicated by the high temperature of the organ, and as a consequence of them, a substance analogous to sugar (glycogen) and urea are formed.

The liver also has the power of disintegrating the albumen and fibrin of the blood. For while portal blood contains much fibrin, blood from the hepatic vein contains little or none. Brown-Séquard has calculated that no less a quantity than about 86½ ozs. of fibrin is daily lost to the blood in its passage through the digestive organs and liver. If this be so, we can readily understand that when anything occurs to interfere with this fibrin-destroying function, there should be a rapid increase of fibrin in the blood, as we know to occur in acute rheumatism and in other diseased states.

The white corpuscles of the blood take their origin in the liver, and the red corpuscles are destroyed there after they have fulfilled their term of existence. It is partly to this disintegration of the red corpuscles that the urine owes its colouring matter.

As previously mentioned, the oxydising and disintegrating pro-

cesses which are performed by the liver are attended by a production of heat. For while the average temperature of the body generally in health is between 98° and 99° Fahr., that of the liver is between 104° and 106° Fahr.

THE BILE.—Fresh bile as it flows from the liver is a neutral or feebly-alkaline fluid, of a light golden yellow, brown, or green colour, and a very bitter taste, followed by a sweetish after taste. Its specific gravity is about 1010. The proportion of solid matter which it contains is usually from 2·69 to 4·01 per cent., and nearly the whole of this consists of substances peculiar to bile.

Bile from the gall-bladder is brown, and its specific gravity is 1030 to 1040, it having undergone inspisation in that viscus, its relative solids are increased to from 8 to 17 per cent. It also become viscid from the admixture of mucus.

The following analysis of bile has been made by Frerichs :—

In 1000 parts.	I Man æt 18. Killed by a fall.	II Man æt 22. Killed by injury.
Water	860·0	859·2
Solid residue	140·0	140·8
Biliary Acids in combination with alkalies	72·2	91·4
Fat	3·2	9·2
Cholestearine	1·6	2.6
Mucus and colouring matter... ...	26·6	29.8
Salts...	6·5	7·7

In the bile the following substances have been found : (*a*) Two acids, viz., Glycocholic and Taurocholic ; these are united with soda as a base ; (*b*) Fatty acids ; (*c*) Cholestearine, a non-saponi-

fiable crystalline fatty substance; (*d*) Colouring matter, Bilirubin and Biliverdin, which are now known to be produced by the destruction of the red corpuscles of the blood in the liver; (*e*) Cholin, an enegertic base; (*f*) Lecithin, a body containing phosphorus; (*g*) Salts, principally of soda and lime.

Glycocholic and Taurocholic acids consist principally of carbon and hydrogen, and both contain nitrogen, and the latter is very rich in sulphur. Of these two acids, Glycocholic is always present in human bile, but Taurocholic acid is occasionally absent from it.

Cholestearin (C_{26} H_{44} O_2) is a fatty substance which crystallises in colourless rhombic plates, one corner of which is often indented. Gall-stones consist almost entirely of Cholestearin. This substance is not peculiar to bile, but is found in other fluids and parts of the body, particularly in the nervous system, and is probably elaborated by the liver for the especial nutrition of that system.

Cholestearin is insoluble in water, but soluble in boiling alcohol, in ether, chloroform and benzol. It is held in solution in the bile by the action of the alkaline salts of the bile acids, which, like soaps, are able to dissolve Cholestearin.

The purpose which the bile serves in the process of digestion is of a most important character, although our knowledge does not allow us to state with precision what this purpose is, because the pancreatic duct lies so near the common bile duct that any operation performed on the one is liable to interfere with the other. Bile exerts a slight action in converting starch into sugar. It speedily converts cane sugar into lactic acid. It emulcifies fatty matters, and aids in their absorption through the coats of the bowels. It checks destructive chemical changes in the chyme, thus acting as a disinfectant on the contents of the bowels, as is well-known, the fæces have a very offensive odour when bile is absent from the alimentary canal. Bile also has the power of checking the process of fermentation. Therefore, when there is a deficient secretion of bile in proportion to the food ingested, or the character of the biliary secretion itself has undergone any serious perversion, much more than the normal amount of putrefactive

fermentation takes place in the bowels, as is evinced by the occurrence of flatulence and frequently by diarrhœa. Furthermore the bile has a neutralizing effect on the acid gastric juice in the chyme, which, if not carried out through deficieny of bile, the continuance of acidity in the contents of the bowels would induce a state of irritation in their mucous membrane, and a perversion of their secretions. The bile also exerts a stimulating effect on the muscular coat of the intestines, increases their peristaltic action, and thus acts as "nature's purgative." That the bile has this effect is apparent from the great tendency to constipation which exists in cases of jaundice, and to diarrhœa when the biliary secretion is excessive.

The quantity of bile poured daily into the intestines has been variously estimated between 2½lbs and 3½lbs. The rate of secretion is greatly influenced by the presence of food in the bowels, being at its highest from one to nine hours after a full meal has been taken; which is to be expected, considering the bile is formed from blood charged with newly absorbed and imperfectly assimilated matter, and that the absorption of the nutritious elements of the food would be imperfectly carried out in the absence of bile from the intestinal contents.

That the bloodvessels of the intestines largely participate in the introduction of soluble alimentary, and every other description of matter into the system has been clearly proved by various observations upon the constitution of the blood of the mesenteric veins; these having shown that after the digestion of abuminous and farinaceous or saccharine substances, albuminose, dextrine, grape-sugar, and lactic acid, are detectible in that fluid, whose usual constitution is greatly altered by the presence of these substances, as well as by the augmented proportion of water which it contains. And so large quantity of fat is absorbed into the blood-vessels, that the superficial capillary network sometimes presents an opalescent whiteness in consequence.

But the nutrative materials which are absorbed by the intestinal veins are not allowed to pass into the general circulation until they have been subjected to an important assimilating pro-

cess which it is one great office of the liver to perform, whereby they are rendered more fit for the purposes they are destined to serve in the economy.

The secretion of bile diminishes when food is withheld for some time, but the depurating character of the secretion requires that it shall constantly be going on to a certain degree. The bile secreted during fasting is prevented by the sphincter of the common bile-duct from passing into the intestines. It therefore flows into the gall-bladder, there to await the next meal. Of the large quantity of bile secreted daily, only a very small quantity is discharged from the bowels. According to Biscoff, this amounts to about 46 grains of the altered biliary acids, whilst Voit estimated the quantity daily secreted at 170 grains. There is therefore left 124 grains to be accounted for. This is all absorbed by the osmotic process which is constantly taking place in the bowels, and is ultimately transformed into products which are eliminated by the lungs and kidneys. The liver also appears to have the power to remove from the blood, and store up in its own substance certain foreign matters of an injurious kind—such as copper and arsenic—which have found their way into the tributaries of the portal vein. Next to the lungs, the liver seems to have a tendency to arrest putrid inflammatory products or pus, or pus-forming materials, which is the *materies morbi* of blood poisoning, which give rise to inflammation and abscess in its substance.

The Glycogenic Function of the Liver.—It is to the elaborate researches of M. Claude Bernard that we owe the discovery of what he named the Glycogenic (or sugar-forming) function of the liver. Previously it was generally believed that the sugar-forming power was exclusively confined to the vegetable kingdom.

In 1848, M. Bernard observed that in animals fed exclusively on meat, the blood of the vena portæ—or that going to the liver—and of the system generally, appeared destitute of sugar, while the blood of the hepatic vein, ascending cava and right side of heart—or that coming from the liver contained a large quantity of that substance. He immediately therefore concluded that a new function was to be attributed to the liver, namely, the forma-

tion of sugar ; and that the sugar thus produced was carried into the circulation, and then by undergoing combustion, ministered to the maintenance of animal heat.

Other physiologists, however, considered that the sugar so found was derived from the food, and merely deposited in the tissues of the liver, which, they said, had a peculiar attraction for it. But this opinion could not be sustained, as it was shown that animals fed exclusively for months on butcher's meat, which contains not a particle of sugar or starch, presented sugar in their livers. In such cases it was obvious that the sugar could not be derived directly from the food, but must have been the result of metamorphoses taking place in the liver itself.

M. Bernard further discovered that if the vessels of the liver were thoroughly cleared of sugar by the injection of water, after the lapse of a few hours, more sugar could be obtained by re-injection, thus showing that the production of sugar must be external to the vessels, in the very substance of the liver itself, and not dependant on the vital action. This circumstance led him to inquire whether there might not be some substance formed by, and pre-existent in the hepatic tissue, from the metamorphosis of which the sugar proceeded, and he, coincidently with Hensen and Pavy, was soon successful in obtaining a peculiar substance possessing properties intermediate between those of starch and dextrine, and capable, under the action of ferments, of being readily converted into sugar, and ultimately undergoing alcoholic or lactic fermentation. This substance, which he termed Glycogen, is almost identical with starch, and has been called Zo-amyline, or animal starch. It is probably produced from the albuminous, amylaceous, and saccharine materials absorbed from the food.

It thus appears that the liver combines in itself the functions of a ductless gland, and those of a ramified tubular gland. The secretion which it pours into its ducts, and thence into the bowels, is of a compound character, that is to say, excrementitious and recrementitious. There is also formed by its cells during their growth, a substance closely resembling starch called Glycogen. This substance appears to be stored up in the tissues of the liver,

and in health, slowly absorbed by the hepatic veins, where, coming in contact with a natural ferment in the blood it is converted into sugar, and so carried away to be combusted, partly in the lungs, and partly in the system at large, for the maintenance of animal heat, and the nutrition of the system. And furthermore, it must be noted that the blood on emerging from the liver is much dencer, and contains a far larger proportion of solid constituents (although less fibrin), and is also far richer in white blood corpuscles than the portal blood. Thus it will be seen that the llver exercises a very important function in sangification in common with the duct-less glands.

The fact that sugar is found abundantly *post-mortem* in the blood of the liver must not lead to the inference that it is present in such quantity during life. In fact Pavy found no more sugar in the blood drawn from the right auricle than in the rest of the system; and he is of the opinion that during life very little of the Glycogen is taken up by the blood on passing through the liver; but that after death, and frequently from various disturbing causes during life, a conversion of Glycogen into sugar takes place in the substance of the liver itself, and quickly appears in the blood, often to so great an extent as to produce Diabetes.

Physical Examination of the Liver.

THE student being now perfectly familiar with the normal position and relations of the liver in an average healthy adult person, it remains to point out the method of examining that organ when supposed to be the seat of disease, and also to describe the variations in size and position which it may undergo, both in the healthy and diseased condition.

Suppose a patient before you with the abdomen exposed: you carefully survey the different regions with your eyes; this is *inspection.* By this means you note any variations in the size and shape of the abdomen, and superjacent part of the right half of the chest. You next pass your hand over the abdomen, making firm but gentle pressure; this is *palpation.* By this means you are enabled to estimate the resistance offered to the hand in the several regions, and to elicit tenderness if present. You then place a finger of one hand on the abdomen and strike a sharp blow on it with two fingers of the other hand; this is *percussion.* By this means a sound is produced which will enable you to form an idea whether a solid organ or tumor, or a hollow organ (intestine) is under your finger. If the former, a *dull* sound is produced; if the latter, the sound produced is said to be *tympanitic.*

There are two variations of palpation; in the one, the left hand or ends of the fingers are placed on one region of the abdomen, and with the tips of the fingers of the right hand a gentle blow or tap is struck in another region; by this means you elicit *fluctuation* if fluid be present in the abdomen. In the other, three fingers of the left hand are placed on the abdomen and the middle finger is struck as before. By this means you elicit what is known as "*hydatid vibration.*"

The *area of hepatic dulness* is usually estimated in (1) the *linea papillaris*, or a line falling perpendicularly from the right nipple. Here the dulness usually extends from the fifth intercostal space, down to the costal margin. (2) The *linea axillaris*, or a line falling perpendicularly from the centre of the right axilla. Here it extends from the seventh intercostal space down to the tenth. (3) The *linea scapularis*, or a line falling perpendicularly from the angle of the right scapula. In this situation the hepatic dulness extends from the ninth intercostal space down to the last rib; but here its lower margin is difficult to define from the dulness of the right kidney, It will thus be perceived that the superior margin of hepatic dulness is slightly arched; not horizontal.

The lower margin of the healthy liver can, under ordinary circumstances, only be discovered by palpation in the epigastrium; but when displaced or enlarged a considerable portion of it can be so demonstrated. In determining the lower margin of hepatic dulness, the better plan is by percussing on the abdomen upwards, pressing firmly with the finger which is to be gently struck. The sound produced, which at first is tympanitic, becomes dull when the lower margin of the liver is reached.

In morbid enlargement of the liver the area of dulness is increased in a direction either upwards or downwards. The dulness extends in an upward direction when the contents of the abdomen (tympanites, ascites, tumors) do not permit the downward enlargement, or when there exists a firm adhesion to the abdominal walls, or when the enlargement is produced by tumors which bulge above the the convex surface of the organ. Any diminution in the size of the liver always makes itself perceptible at the lower margin of dulness.

Previous to making an examination of the liver it is absolutely necessary that the stomach be empty and the bowels freely moved by medicine or an enema. The hands of the examiner must be warm, and pressure must not be applied too suddenly, because some persons suffer from extreme irritability of the abdominal muscles, particularly in the region of the liver. The patient must lie down with the head resting on a firm support, and the legs

drawn up. By directing the patient to take a full inspiration and then hold his breath, the lower margin of the liver is caused to descend rather lower down from behind the costal walls.

Under perfectly normal conditions nothing can, as a rule, be detected with respect to the liver from the sense of touch, inasmuch as this organ offers no greater resistance than that of the abdominal walls. There are, however, certain deviations from the normal conditions of the liver, which may be revealed by a physical examination, and lead to serious errors in diagnosis if accepted as evidence of disease. Thus *enlargement* of the liver *may be simulated* by—

(1.)—*Congenital aberrations in form.*—The liver is not subject to frequent or great deviations from its ordinary characters. In very rare cases it may, however, be more square than usual, and thus present a large surface to the abdominal wall. The left lobe of the liver is very variable in size and form, under perfectly normal conditions, and a sort of accessory liver is occasionally found appended to the left extremity of the gland by a fold of peritoneum containing blood-vessels. It is necessary, therefore, to be cautious in inferring liver disease from alterations in this situation.

(2.)—In *early life* the liver is relatively larger than in the adult, and consequeutly both its upper and lower margins have a more extended area than later in life.

(3.)—*Extreme deformity of the thorax*, the result of rachitis, by reducing the capacity of the chest, and depressing the diaphragm may cause the liver to be depressed below the costal margin, and lead to the appearance of hepatic enlargement. An inspection of the chest and spine will prevent any error from this cause.

(4.)—The *habit* of wearing stays, belts, and clothes, tightly fastened round the waist may cause displacement downwards, and deformity of the liver to such a degree as to lead to the appearance of extensive enlargement of the gland, or tumors of the abdomen.

The usual effect of tight lacing on the form of the liver is to produce a deep transverse furrow from side to side across the

under surface of the right lobe. This furrow is deepest at the right border, where it may even divide the mass of the liver more or less completely into two parts; the natural arched form of the liver conducing to this deformity. This furrow may extend across to the left lobe and be so deep as to completely separate the anterior from the posterior part, fibrous remains only connecting the two. Thus the appearance of a moveable tumor in the region of the liver may be produced, leading to serious errors in diagnosis, and if tympanitic coils of intestine have arranged themselves above the fibrous connection, the case is further obscured. The liver may be displaced downwards by this habit to such an extent that its lower margin reaches to the ilium, and the organ appears to fill up the whole of the right side and front of the abdomen.

In order to clear up the diagnosis in these cases attention must be directed as to the habits of the patient with regard to lacing or fastening the clothes, and also to the walls of the chest and abdomen for signs of compression in those parts. The mobility of the "tight lace" lobe, which at times is considerable, and its displacement following the acts of respiration, as well as the tight-lace depression, the existence of which can almost always be established, afford as a rule, sufficient data for determining the true state of affairs. The solution will, moreover, be simplified by bearing in mind the fact that the changes in question are rarely encountered except in adult females.

(5.)—*Pathological alterations in the adjacent organs* frequently cause depression of the liver and lead to the appearance of enlargement. Amongst these may be mentioned—

(a.)—Pulmonary emphysema, pheumothorax, extensive pericardial exudations, and mediastinal tumors.—In the case of any of these there is no difficulty in determining the superior line of hepatic dulness which is simultaneously depressed with the inferior line, so that the *area* of hepatic dulness *is not increased.*

(b.)—Extensive pleuritic effusions of the right side. Here the superior line of hepatic dulness cannot be detected because the percussion sound is quite as dull over the affected half of the chest as it is over the adjacent liver. It often happens that in-

spection alone will suffice to prevent our falling into the error of referring in its entire height, this dulness to the enlarged liver; for an obliteration of the intercostal spaces such as is produced by pleuritic exudations does not occur as a result of diffused enlargement of the liver. Moreover, enlargement of the liver may lead to rotation of the ribs, a thing which pleuritic exudations can never do.

(c.)—Tumors of the abdomen.—It occasionally happens that an appearance of an increase in the bulk of the liver is produced by tumors of the abdominal organs lying in such intimate proximity that the dulness proceeding from them is directly continuous with that of the liver. This occurs in tumors of the stomach, retroperitoneal lymphatic glands, pancreas, transverse colon, omentum, and right kidney. In some of these cases, however, the free edge of the liver, as well as its respiratory movements can be made out by the hand, and sometimes even perceived by the eyes. In other cases the tumor is in such intimate contact with the liver that the boundary between the two cannot be defined by the most careful palpation. Here, when the tumor is so immovable as to prevent the liver from descending with the act of inspiration, the absence of the respiratory displacement will usually afford a sure indication of the presence of a tumor other than hepatic, rather than a portion of diseased liver projecting low downwards; for even when such a morbid liver extends down to the flanks, the downward movement of its inferior border accompanying deep inspiration is usually perceptible.

(d.)—Chronic constipation with accumulation of fæces in the transverse colon constitutes a condition which is most difficult to distinguish from enlargement of the liver, because in addition to an extended area of dulness, jaundice may be a notable symptom, and the patient may believe the bowels to be free, or may even complain of diarrhœa. Recently I had under treatment in the High Wycombe Cottage Hospital, a woman who presented the following train of symptoms,—namely, nausea and vomiting, rigors, jaundice, slow pulse, tenderness in the

right hypochondrium, with increased area of dulness in that region; urine tinged with bile. Purgatives and enemata were frequently administered with more or less effect. No amendment, however, taking place in the condition of the patient, I introduced my finger into the rectum, and there discovered an indurated mass of fæces, which had to be removed by the handle of a spoon before the bowels could be emptied by means of repeated enemata of warm water. Then the case was quite clear. An accumulation of fæces had taken place in the colon and rectum forming a tumor which compressed the common bile-duct and so produced jaundice, and apparently increased the area of hepatic dulness. Purgatives when administered, had forced down the less solid contents of the upper portion of the bowels through the distended and paralyzed colon, leading to the belief that the way was clear throughout.

Whenever there is a suspicion of an impacted colon, a digital examination of the rectum will relieve the case of any difficulty on that score.

A *diminution of the size of the liver* may be simulated by tympanitic distension of the bowels and stomach; the organ appearing smaller than it really is, in consequence of a portion of its anterior surface being covered by a fold of intestine, or because it has been forced upwards and backwards by the inflated bowels; indeed this backward displacement of the liver may take place to such an extent as to obliterate the anterior area of hepatic dulness; in consequence of which, the pulmonary resonance on the right side joins directly with the tympanitic resonance of the intestines. The most frequent cause of such a dislocation of the hepatic dulness is intestinal inflation, and a diminution in the capacity of the abdomen, resulting from the presence of fluid in the peritoneal cavity, or from tumors, especially those which project upwards from the pelvis. The recognition of the cause of the diminished hepatic dulness in such cases is usually not difficult.

Finally, in rare cases it occurs that the viscera of the chest and abdomen are transposed, the mass of the liver being placed in the left hypochondrium, and the apex of the heart to the right of the

sternum. This condition of the viscera might cause embarrassment by finding a diminished area of dulness in the right hypochondrium, which, however, may be avoided by noticing the position of the impulse of the otherwise healthy heart, coupled with the absence of any dislocating influence, such as left pleuritic exudation or pneumo-thorax.

Floating Liver.

In connection with the foregoing subject may be mentioned a peculiar condition of the liver, observed a few times, especially by continental physicians, namely, an abnormal downward displacement, and preternatural mobility of the organ, caused by a remarkable relaxation of its ligaments.

About twelve cases of this affection have been observed, and nearly all of them in women who had borne children. Chvostek *(Weiner Med. Blatt)* accidentally observed the affection in a man who was not previously aware of anything wrong in his abdomen.

The amount of downward displacement in these cases varies considerably. In Winkler's patient the superior margin of hepatic dulness in the right papillary line lay beneath the sixth rib, and the inferior margin, six centimeters below the costal border. In other cases the liver was entirely displaced from the right hypochondrium, its superior edge being found at a greater or less distance below the ribs, In Meissner's case the liver reached down to within two fingers breadth of the symphisis pubis.

Etiology.—The cause or causes of this condition are somewhat obscure, but an abnormal congenital elongation of the suspensory ligaments of the liver was probably a prime etiological factor in almost all the cases reported. With such a condition of the ligaments it is easy to conceive that the intestines might be pushed between the diaphragm and the liver by an ascending gravid uterus and thus the relaxation of the ligaments becoming aggravated, the liver by its own weight would tend to increase the downward displacement. Moreover, the relaxed and pendulous condition of the abdominal walls observed in most of the cases would still further favour prolapse of the organ.

Floating liver is very rarely met with in men, and when observed is probably congenital.

The symptoms manifested by this affection are an abdominal tumor, causing more or less uneasiness by its weight and mere presence, which by palpation is perceived by its volume and general configuration to correspond with the liver displaced from its natural position, while the hepatic dulness is absent from its normal situation, the tympanitic bowel sound being succeeded immediately by the pulmonary resonance in percussing from below upwards on the right side. The tumor is recognised as being the liver by it being possible to restore it to its normal position in the right hypochondrium, when the phenomena of hepatic dulness are also restored, In addition to these special symptoms, the patients generally complain of pain, weight, or fulness in the abdomen, and if the common bile-duct become twisted, jaundice will supervene. As a rule, however, more suffering is caused by apprehension than by disturbance of the functions of the body.

TREATMENT—Little can be done in the way of treatment beyond relieving thc patient's mind from any apprehension as to the gravity of the affection. A broad, firm, abdominal belt will afford support to the displaced gland, and probably also relieve pain.

On Jaundice.

By the word jaundice is meant a yellow discoloration of the skin and conjunctivæ produced by the presence of bile pigment in the circulating fluid. The word is a corruption of the French *jaunisse* from *jaune*, which signifies *yellow*. *Icterus*, by which the Greeks designated this morbid condition is derived from Ικτις a weasel or marten, an animal kept by them in their homes for the same purpose as we do cats, the eyes of which are yellow.

It must be borne in mind that jaundice is not itself a disease but a symptom produced by a great variety of pathological conditions operating in the economy of the body. The only variety of jaundice which has any claim to be considered as a disease sole is that which is produced by mental emotion, and is devoid of any appreciable structural change within the body. The failure of the medical attendant to recognise this fact has been fraught with the most lamentable results to his patients and grievous disappointment to himself. It is a common thing for a practitioner to recognise that his patient is suffering from "jaundice"—a diagnosis which the nurse or friends have probably already made —and then to proceed to administer "calomel," without even attempting to discover the nature of the disease which produced the jaundice, and therefore oblivious to the fact that his remedy may have no ameliorating effect, or may even do positive harm. It is seldom easy, and sometimes impossible to state with certainty during life the nature of the morbid condition which produces the discoloration of the skin, but it must always be our anxious consideration to solve that problem when presented to us; and let us remember that if we cannot do any good with our remedies, it is our plain duty to do no harm.

To assist the reader in arriving at a just diagnosis of the *cause* of jaundice, on which alone, the prognosis and treatment should be founded, is the object which shall be kept in view throughout the following pages.

Jaundice is caused by the yellow pigment of the bile circulating in the blood, and is first apparent to an observer in the conjunctivæ, and afterwards in the skin. The colour at first may be no more than a sallow tinge, not easily discovered in persons of dark complexion. The yellow state of the conjunctivæ produced by the presence of fat, which is habitual in some persons, must not be mistaken for jaundice. The state of the urine will clear up any doubt thus produced. When jaundice is produced by obstruction to the common bile-duct, as by a gall-stone impacted therein, the colour, at first light, gradually deepens as long as the obstruction lasts. When caused by a poison acting upon the system, such as yellow fever, phosphorus, &c., the discoloration may not be very great at any time, and yet these are always the most serious cases. The color which is usually spread over the whole body, varies in different persons, being darker in the old and wrinkled, than in fair young persons with plenty of fat. The conjunctivæ have a peculiar pearly appearance which is very striking.

It is important to remember that the colour may vary in the same patient from day to day according to the amount of bile secreted, food taken, rapidity with which the pigment is removed from the blood by the kidneys, or destroyed in the system, independently of any amelioration of the cause which produced the jaundice.

It is not uncommon to see, in slight cases of jaundice, the upper part of the body yellow, and the belly and legs free from discoloration. This condition is occasionally observed in Yellow Atrophy.

The lips retain the redness of health, but a yellow colour may sometimes be made apparent in them by pressure. The roof of the mouth where the bone is thinly covered usually presents a yellow tint.

The urine early in jaundice—generally before the skin—acquires a saffron yellow, greenish-brown, or almost black hue, according to the amount of bile pigment which it contains. It is by this secretion the greater part of the bile pigment is eliminated from the system in jaundice. The urine is commonly clear, and has an acid reaction, but may be turbid from the presence of urates. The quantity of urine passed in the 24 hours in jaundice is subject to great variations. The amount depending in a great measure on the quantity of urea formed, or in other words on the functional activity of the liver.

The chief pigment of the bile present in the urine in jaundice is Bilirubin, the best way of testing for which is as follows :—pour ordinary nitric acid into a test tube to the depth of an inch. On the surface of this acid, the urine to be tested is gently poured by means of a pipett down the side of the tube, so that the two fluids may touch, but not mix. A red line forms at the place of contact in every urine. If bile pigment be present in the urine, a zone above becomes *green*, then blue, violet, and lastly red, the uppermost ring being green. The green colour is that most characteristic of bile pigment, and is the one most frequently seen. This test is known as Gmelin's test. The tests for bile-acids are too tedious for clinical purposes.

Some of the bile pigment is eliminated from the system by the cutaneous glands, often in sufficient quantity to stain the linen yellow.

The milk of suckling women is not usually stained in jaundice, but such an occurrence has been observed by various writers, such as Drs. Bright and Hervieux; the latter stating that in an epidemic of jaundice in the Maternité at Paris, in all the woman who were suckling, the milk was coloured yellow.

It is very rarely found that the saliva or the tears are discoloured, nor either is the natural mucus from the air passages. But in a state of disease, such as pneumonia co-existing with jaundice, an albuminous exudation takes place from the blood—the pneumonic sputa—which may contain bile pigment. In the

case mentioned by Dr. Budd, in which the saliva was yellow, mercurial salivation was present to account for that unusual condition.

The fluids poured out into the intestinal tract are likewise devoid of colour in jaundice; otherwise the fæces themselves would be coloured. This is a rather curious fact, because the fluid exudation in the peritoneum, and all the other serious cavities are among the first to show the presence of bile.

The yellow colour often remains in the skin for some time after the cause of the jaundice has been removed, and after bile has ceased to appear in the urine. This is particularly the case in old persons, and is to be especially noted, in order to avoid the use of active remedies when no longer necessary.

The elimination of bile pigment from the system is promoted by warm baths, diaphoretics and diuretics.

A bitter taste in the mouth is not infrequently complained of by persons suffering from jaundice. Sometimes this is due to eructations from the stomach, but more frequently to the presence of bile-acids in the blood—taurocholic acid being intensely bitter.

Symptoms of disordered digestion resulting from the absence or deficiency of bile in the intestines are frequently observed in the jaundiced. There is considerable difference of opinion amongst writers on the liver, as to the existence of flatulence in connection with jaundice. Dr. Budd is silent on the point. Murchison and Frerichs state the patients suffer from flatulence. Hippocrates states that the jaundiced are not flatulent. Dr. Legg states his experience of chronic jaundice is, that it is not at all a prominent symptom. Recently I had under my care a patient suffering from acute jaundice in whom flatulence was a very distressing complication, which only subsided when the flow of bile into the intestines was re-established. Remembering the antiseptic properties of bile, on theoretical grounds we should expect flatulence to exist when bile is quite absent from the bowels, and the patient taking a large quantity of food of a kind prone to undergo the putrefactive fermentation. But when even a small quantity of

bile flows into the bowels, and food of a kind less prone to putrefaction is partaken of in small quantities, flatulence may be very slight or entirely absent.

Nearly all authorities are agreed as to the highly offensive character of the alvine evacuations passed by the jaundiced. Owing to the absence of bile pigment the motions are pale, or of a drab or clay colour. The bowels are very likely to be confined from the absence of the natural stimulating effect which the bile exercises on them in health. But on the other hand, diarrhœa may be produced by the irritating effect of putrid fæces. If bile still enter the bowels, the motions may be but little altered and voided regularly. The motions may be of a dark colour without any bile entering the bowels. This condition of the stools is sometimes produced by the admixture of altered blood. There are also certain drugs which cause dark stools, notably iron and bismuth.

Persons suffering from jaundice evince a great dislike to fat, and if partaken of, fatty matter is quickly discharged by the bowels.

If obstruction of the bile-ducts be complete the glycogenic function of the liver is suspended, and irritation of the fourth ventricle fails to produce sugar in the urine. Theoretically, we should expect at the same time to find a diminution in the urea excreted by the kidneys, but no experiments appear to have been made with a view to establish this point.

In cases of jaundice from obstruction to the bile-ducts the nutrition of the body is usually diminished, as a consequence of mal-assimilation of food, and absorption of the body fat, but this does not always occur.

When jaundice has lasted a long time the blood becomes impoverished by a diminution of the red corpuscles and fibrin, and as a result of this there is an especial disposition to hæmorrhage from the mucous membranes, and under the skin. The immediate cause of death in protracted jaundice frequently being a copious hæmorrhage from the stomach and bowels. This tendency to hæmorrhage is particularly observed in Acute Yellow Atrophy of the liver, but Legg believes it to be preceded by a

diseased state of the blood-vessels, and it is to that rather than to the state of the blood the hæmorrhagic diathesis is due. The proportion in which hæmorrhage occurs in Yellow Atrophy is about 80 per cent.

Itching of the skin is a very troublesome symptom frequently observed in jaundice, and in hepatic affections without jaundice. It most commonly occurs when there is obstruction to the bile-duct, is sometimes intermittent in its character, and sometimes precedes the development of discoloration of the skin. Recently I had under treatment a women suffering from jaundice who was much troubled by an eruption of lichen and boils which occurred all over her body. In this case Wright's Liquor Carbonis Detergens afforded great relief to the itching and tingling. The late Dr. Graves of Dublin, was, I think, the first to draw attention to a train of morbid phenomena which he observed eight or nine times. These were; first, inflammation of the joints, then jaundice, followed in a few days by urticaria. Neither Drs. Budd, Murchison, nor Legg appear to have witnessed this sequence. Rheumatism followed by jaundice is by no means a rare occurrence.

Xanthelasma or Vitiligoidea, is a remarkable condition of the skin, now and then observed in the eyelids and other parts of the body after jaundice has lasted for some months. This disease presents itself in two forms, V. plana and V. tuberosa. The first is chiefly met with on the eyelids, cheeks, &c. The spots vary from a pin's point to a florin in size, and are of a pale yellow white, slightly raised above the surrounding skin. They cause no pain or inconvenience to the patient, who is often not aware of their presence. V. tuberosa is found on the ears, neck, trunk, elbows, and hands. The skin over the tubercles is smooth, tense, and shining, and when pricked, gives out nothing but blood. This variety sometimes troubles the patient by preventing him from handling tools. This disease consists in a fatty infiltration of the skin, and is sometimes seen in persons who have never suffered from jaundice. I have now under observation two females who present well-marked Xanthelasma in their eyelids. Neither of them has suffered from jaundice, but both are the subjects of fibroid tumor

of the uterus. Nor does the disease appear to be hereditary in either case A remarkable circumstance in connection with one of the patients is that the spots first appeared in her and her sister at the age of 38 years, the latter being ten years her senior. No other member of either family is known to have had Xanthelasma.

There is no drug at present known to have any influence in removing these spots, and operative interference is to be avoded, particularly about the eyelids.

As might be expected from the important part which the liver plays in the maintenance of animal heat, the temperature of the body is slightly lowered in jaundice, provided the jaundice be not caused by a febrile disease, such as pyæmia or abscess of the liver. Even in those cases where a febrile complication is present, the temperature never rises so high as it would were jaundice absent.

Jaundice is occasionally accompanied by a slow pulse when there is an absence of fever in the system. The pulse may fall to 40, and less frequently to 20 beats per minute, but this is not an invariable symptom of jaundice. It occurs most frequently in the catarrhal form, and is most probably due to the presence of the bile acids in the blood, producing a paralyzing effect on the ganglia of the heart. The reason that the slow pulse is so seldom observed in jaundice is due to the small amount of bile acids secreted by the liver when there exists any obstruction to the bile-ducts. Murchison has repeatedly known the pulse to sink to 40 or 30 in cases of hepatic derangement independent of jaundice.

A soft mitral regurgitant murmur is very frequently observed in those cases of jaundice which are accompanied by a slow pulse. These murmurs, if transient, are probably caused by the toxic effect of the bile-acids.

Yellow vision is very rarely found in connection with jaundice, but as Xanthopsy has been observed independent of jaundice, it is very probable that in the few cases where they were found associated the connection was merely fortuitous.

The symptoms due to the nervous system in mild cases of jaundice are fretfulness, a disposition to sleep, and an indisposi-

tion to mental exertion, and peevishness; but in the more severe cases, such as Yellow Atrophy, phosphorus poisoning, &c., delirium, stupor, coma, convulsions, muscular tremors, subsultus, floccitation, paralysis of the sphincters, a dry brown tongue, and other indications of the "typhoid state" are frequently seen. These grave symptoms may also occur in cases of long-standing obstruction of the common bile-duct, where all, or a greater portion of the secreting tissue of the liver has been destroyed.

The cause of these nervous symptoms is very obscure; no theory of their nature being yet generally accepted. That they are not due to the presence of bile in the blood is evident from the great length of time persons may suffer from intense jaundice without developing them; and on the other hand they are developed very speedily in some cases of Acute Yellow Atrophy of the liver. Nor in the brain and its membranes can we, as a rule, find, by the naked eye at least, any pathological condition to account for them. Dr. Legg is inclined to attribute them to cloudy swellings in the walls of the blood-vessels of the brain, such as were observed by Steiner and Wunderlich with the aid of the microscope; precisely similar changes being observed in the rest of the vessels of the body. But in saying this we are only putting the difficulty further back; for, it may be asked, "What causes these changes in the blood-vessels?" This question brings us to the consideration of the

Theories of Jaundice.

When the body of a person who has died while suffering from jaundice is examined, the ducts of the liver may be found in either of the following conditions, namely:—(1) they may be found obstructed by a gall-stone, tumor, cicatrix, inspissated mucus, &c.; (2) they may be found free and tinged with bile; or (3) they may be found free and untinged with bile.

In the first mentioned condition of the ducts, the cause of the jaundice is obvious enough. Bile has been secreted, entered the

bile-ducts and gall-bladder, and finding no outlet into the duodenum, has been absorbed into the blood, and carried by that fluid into all the tissues of the body, dying them yellow.

But it is in the second and third condition of the ducts that the greatest diversity of opinion maintains among pathologists as to the mode of production of jaundice.

Aretæus, Bianchi, Virchow, and Breschet, believed that jaundice could arise from changes taking place in the blood without any implication of the liver. This doctrine of hæmatogenous jaundice is founded upon the supposed identity of the blood pigment and the bile pigment; but experiments made with a view to prove the identity of bilirubin and hæmatodin have not been at all conclusive; and Preyer of Jena, found that the spectra of these two bodies are altogether different. If it be admitted that the colouring matter of the bile is formed by the liver from the colouring matter of the blood—which seems probable—it must also be admitted that for the production of jaundice it is necessary that hæmatodin must first be submitted to some modifying influence exercised by the hepatic cells, otherwise hæmatodin appears to have no power of producing the phenomena of jaundice. The opinion of the late Dr. Murchison is, that the constituents of the bile poured into the intestines are absorbed into the system, and in the normal state become transformed in the process of absorption into products which are eliminated by the lungs and kidneys, while at the same time they assist in the assimilation of the nutritive materials derived from the food. But in certain morbid states the absorbed bile does not undergo the normal metamorphoses; it circulates in the blood and stains the skin and other tissues.*

Now, while this theory of Dr. Murchison's seems adequate to account for the production of jaundice in those cases in which the ducts are free and bile is still poured into the intestines; it altogether fails to account for the production of jaundice when the ducts are free and unstained by bile—a condition frequently found in acute Yellow Atrophy.

* Murchison on Liver, 2nd edition 1877, p. 331.

In order to account for the production of jaundice when the ducts are found *post mortem*, free and unstained by bile, the theory that its phenomena are produced by *suppression* of the functions of the liver is that held by a great many authorities. This theory demands the belief that bile is a normal constituent of the blood, and that the liver merely acts as a strainer to eliminate it from the system. This doctrine was taught by Glisson, Morgagni, and Van Switen. Budd also held this opinion, for he states "that the biliary pigment exists in the blood, and that in consequence of defective action of the secreting cells, it is not eliminated as it should be in the liver."* This theory of jaundice would be tenable if it could be shown that the constituents of bile *do* exist normally in the blood; but this is exactly what no modern observer has been able to do. And it is to be noticed that in those diseases of the liver which lead to destruction of the lobular structure of the liver, the jaundice, at first deep, gradually subsides as the disease progresses, as in acute yellow atrophy; also in cases of cirrhosis of the liver the jaundice is seldom or never intense at any period of the disease. Furthermore, I may add, that if the portal blood had the power to produce yellow discolouration of the tissues of the body, we might reasonably expect to find the intestines, mesentery, and even the liver itself yellow as a normal condition, instead of exceptionally in disease.

The third theory of jaundice is that which attributes its production to the absorption into the blood, of bile which has been secreted by the liver. This, without doubt, is the best founded of all the theories of jaundice, as in the great majority of cases, some obstruction to the flow of bile into the duodenum can be found. The obstruction may be high up in the ducts in the form of colourless mucus which hinders the descent of the bile, or low down in the common bile-duct, produced by some mechanical impediment, such as a gall-stone, or a plug of inspissated mucus.

But, it may be said, in some cases of yellow atrophy no obstruction to the bile-ducts can be found after death, even with the aid of the microscope. In such cases I am of the opinion that the ob-

* Budd on Liver, 3rd edition, p. 468.

struction which caused the jaundice has passed away. It may have existed at an early period of the disease as a tumid condition of the ducts, or some of them, or as hyaline plugs blocking up their lumen. When we remember the transient character of catarrh of the bile-ducts, it is not surprising that after death from a disease which has destroyed all the glandular structure of the liver, no trace of its existence can be found. The bile having a tendency to flow in the direction of least pressure, which is normally in the bile-ducts, it accordingly takes that course; but when the bile-ducts are obstructed, the least pressure is towards the hepatic veins, and into them the bile consequently flows and thence is carried to all the tissues of the body.

Taking this theory of jaundice as the most rational one, the question remains, "What produces the dreadful nervous symptoms sometimes observed in connection with jaundice"? In reply to this question a great many theories have been advanced. The oldest of these is that they are produced by *cholæmia;* that the constituents of the bile, especially the bile-acids, which have passed into the blood, exercise this influence. In order to establish this theory, experiments have been made on animals by injecting filtered bile into their blood, but the results have been conflicting in the hands of different investigators. But apart from these experiments, in a clinical point of view, it does not accord with experience that the nervous symptoms are in a direct ratio to the intensity of the jaundice; on the contrary they occasionally make their appearance sooner than the icterus, and may be present in characteristic form, even when the latter symptom is altogether absent. Furthermore, in cases of obstruction of the common bile-duct, where the jaundice is most intense, the nervous symptoms are not set up until after prolonged stagnation of the secretion has produced disintegration of the hepatic cells, and finally, it seems impossible that in a disease such as acute yellow atrophy of the liver, in which the formation of biliary acids gradually diminishes, the action of these substances on the brain should begin to manifest itself at the close of life, when the functional activity of the liver has ceased; while their influence upon the other portions of the nervous system—for instance, upon the

ganglia of the heart,—commences immediately after the appearance of the jaundice.

The fact that the kidneys participate in the glandular parenchymatous degeneration, and that the urea excreted in the later stages of acute yellow atrophy, has materially diminished in quantity or entirely disappeared, has led some writers to attribute the nervous symptoms to uræmia. But the maniacal paroxysms observed in acute atrophy differ widely in character from the epileptiform convulsions, with subsequent coma of uræmia. Moreover, as regards the diminution of urea in the urine, it is more probable that the formation of this substance in the system is diminished than that it is retained there as such.

Virchow thought the most probable cause of the brain symptoms to be the "retention within the blood of substances intended for the formation of secretions": but hitherto no such substance to which such an effect could be ascribed, has been discovered. Dr. Austin Flint believed he had discovered such a substance in cholestearine. But that this substance can exercise a toxic effect on the brain is by no means proven; on the contrary, it has been shown by experiments on dogs that no cerebral symptoms are induced in them by injecting into their blood as much as 2.75 grms of cholestearine dissolved in soap-water, within a period of sixteen days.

Murchison attributed these symptoms to the accumulation in the blood of *leucin* and *tyrosin*, which he says are formed in the system at the expense of urea, but there is no evidence that any such influence is exerted by them. On the contrary Panum and Billroth injected these substances in large quantities into the blood of animals without producing any disturbance of the brain functions.

Dr. Legg is inclined to attribute the nervous symptoms to changes taking place in the cerebral capillary blood vessels (cloudy swellings); but it is not easy to reconcile the explosive character of these symptoms with a process which must necessarily be gradual. It therefore seems to me we must fall back on the rather vague and unsatisfactory theory advanced by Budd, which

attributes their production to some poison which is either generated in the system or introduced into it from without. This poison is arrested in the liver where it works the destructive changes, and is then suddenly liberated to exert its action on the nervous system.—*Budd on Liver, p. 269.*

BILIOUSNESS.

IT is impossible to review the various important functions which it is the office of the liver to discharge in the processes of digestion and assimilation, without a perception of the grave consequences which must ensue in the vital economy when these functions are imperfectly discharged or entirely suppressed. The very fact of the bile itself being either excessive or deficient in quantity, or altered in constitution, is quite sufficient to give rise to grave symptoms of disease which require very judicious treatment. Indeed it may be said that most of our minor and many of our major disorders arise from some disturbing influence at work in the liver. It is upon the nervous system that the most marked effects are produced by functional derangements of the liver. The patient evinces great irritability of temper, with depression of spirits, which latter frequently leads to acts of suicide. Tom Hood truly said that "people often think they are pious when they are only bilious;" and the remorse felt after a debauch is often only too surely to be traced to an over-tasked liver, and the consequent derangement of the nervous system by the presence of the elements of the bile in the circulating fluid.

Some persons habitually evince a lethargic disposition which is doubtless due to constitutional defect in the functional activity of the liver, and others suffer from bilious diarrhœa from slight causes. These cases require very minute attention as to their diet and exercise, lest they should develop into the more grave consequences of organic disease in distant organs, such as the heart, kidneys, joints, &c.

When the Glycogenic function of the liver is disturbed sugar appears in the urine, and that intractible disease Diabetes is set

up. There is not the slightest doubt that the *materies morbi* of Gout—namely, uric acid, in combination with soda,—becomes elaborated in consequence of some defective action of the liver, and that hereditary influences have a marked tendency in predisposing to this as also to other forms of functional derangement of the liver. Of uric acid a mere trace only can be found in the blood under perfectly normal circumstances; but in the Gouty state it is enormously increased in quantity, and leads to very serious structural changes in the heart, lungs, blood-vessels, and membranes of the brain. The kidneys, also, in consequence of being called upon to remove from the system this highly irritating substance, in abnormal quantities, ultimately evince a high grade of structural disease.

Persons who are in the habit of passing large quantities of uric acid in their urine are very liable to have small concretions of that substance form in their kidneys or bladders. These concretions have a tendency to increase in size, in consequence of fresh deposits taking place upon their surfaces, and if not happily voided from the system with the urine they lead to the well-known symptoms of vesical or renal calculi, with all the attendant dangers and complications.

The term Biliousness is frequently applied in a vague manner to a train of symptoms of which gastro-duodenal catarrh is the prominent pathological condition. But that the liver is primarily or secondarily affected in this disease appears highly probable from the fact that a most intimate sympathy exists between the stomach and duodenum upon the one hand, and the liver upon the other, not only from their nervous supply being derived in common from the solar plexus and pneumogastric, but also from the tendency of disease of the mucous membrane of the stomach to spread by continuity of surface into the duodenum, and thence by the common bile-duct into the liver. It must also be remembered that a portion of the blood which circulates in the liver comes directly from the coats of the stomach. It thus appears that there is an intimate connection between the two organs by the three great means by which living union is rendered effectual, namely, nerves, blood-vessels, and surface.

Symptoms.—The prominent symptoms of the disorder known by the names Biliousness, Bilious Attack, Sick Headache, Bilious Headache, &c., are, furred tongue, oppression at the epigastrium, constipation, loss of appetite, vomiting, the vomited matter consisting at first of the contents of the stomach, and subsequently of bile, which latter appears to be thrown into the stomach from the duodenum, by the violent action of the abdominal muscles during the acts of vomiting. The presence of this substance in the stomach, by irritating its mucous coat, greatly aggravates the vomiting. There is also present giddiness and headache, which latter is frequently very severe, the sufferer being unable to keep his head upon the pillow, although every attempt to rise brings on violent retching. Throbbing of the vessels of the head and neck add greatly to the discomfort of the patient.

The condition depicted above, however, seldom continues for more than 12 or 24 hours; the transition to health taking place gradually, but sometimes suddenly as if by magic; an intense desire for food being the first herald of return to the normal condition. After the appetite has been satisfied the patient feels no further discomfort.

Etiology.—The causes of this complaint are some unusual excitement, or errors in diet. Any excess either in food or wine will produce this form of Biliousness, and it most frequently succeeds to partaking of immature wine or spirits—particularly the latter, which when new, contain a large quantity of fusil oil, which can only be effectually removed by the subtle chemistry of time.

Another variety of this affection, however, is frequently met with which may be termed Constitutional Biliousness. In the persons who suffer from this complaint the liver, from natural conformation, seems only just capable of fulfilling its functions under the most favourable circumstances. These persons are born with a tendency to bilious derangements which they very frequently inherit, and when any disturbing influence comes to bear, such as gross living, indolent habits, constipation, or residence in a hot climate, the liver, efficient under ordinary circumstances, is inadequate to the extra strain put upon it; then

Biliousness, with its train of discomforts supervenes. The same remarks apply to persons who evince a tendency to Gout. Such persons are frequently addicted to sedentary habits and unwholesome food, which produce derangement of the chylopoietic viscera and liver.

Symptoms.—The tongue in such persons as are the subjects of Constitutional Biliousness is habitually coated, and the breath offensive. Flatulence, nausea, and depression of spirits are complained of, and the bowels are irregular in their action; the motions being clay-coloured in consequence of bile being absent from their constitution. The urine is scanty and high coloured, in consequence of the presence of bile pigment and urates in that secretion. Vomiting is seldom a marked feature of this variety of the affection.

Treatment.—The first mentioned variety of Biliousness seldom requires anything further in the way of treatment than perfect quiet and abstinence from food for a few hours. A bottle of soda-water with about ten grains of Bicarbonate of Soda added to it will be found useful in checking the vomiting. Five grains of the compound Colocynth pill with an equal quantity of Compound Rhubarb pill taken at bed time, will complete the cure.

Constitutional Biliousness is a far more serious disorder and can only be effectually combated by constant attention to the diet and exercise. Persons subject to Biliousness should be careful to avoid excess in both eating and drinking, and should more especially shun those articles of food and drink which from experience they find are apt to disagree with them. New wine, beer, and spirits should be particularly avoided, as well as boiled meat, stews, soups, greasy or rich puddings, much butter or fats, and most articles of pastry, as they are very indigestible, and by overtasking the stomach, very apt to derange it. A sufficient quantity of fresh green vegetables, cooked, should be taken at two meals in the day, but salads, especially cucumbers, should be partaken of in very moderate quantities. Dried fruit, nuts and almonds, and the like are objectionable for persons of a bilious

tendency. For such persons a mutton chop, slightly underdone, is an excellent article for breakfast or luncheon, and good sound beef or mutton, either broiled or roasted, so that the gravy be retained, is better for dinner than many dishes apparently more delicate. These with fresh game or venison if not too high, form a good variety from which to choose a bill of fare.

The quantity of animal food per diem, except for the laborious, should be limited to from 6 oz. to 12 oz., and warm slops of all kinds, except moderately strong tea and coffee, should be taken as seldom as possible, and in general avoided altogether. Even cocoa and chocolate prove injurious to the delicate and bilious.

Out-door exercise, especially horse exercise, if it can be indulged in, and plenty of fresh air, adapting the clothing to the vicissitudes of our variable climate, are powerful factors in promoting the health of the Bilious. Above all, heavy and late suppers should be abandoned, as well as late hours, and no food should be taken after tea beyond a dry hard biscuit or dry crust.

In general, attacks of the bile may be prevented by the exercise of moderate judgment and temperance in living, but when the medical attendant is called to a patient who is suffering from a Bilious attack, he is expected to give speedy relief, and this can generally be done by directing our treatment to one or more of the most prominent symptoms. Thus, if there be evidence of an overloaded stomach an emetic dose (gr. x to gr. xx) of Ipecacuanha will be speedily followed by relief. If flatulence be present it can be removed by Charcoal or Bismuth. Constipation in these cases is best relieved by small doses of blue pill (gr. ½) with gr. 3 of Compound Rhubarb pill taken at night, and followed by a dose of one of the purgative mineral waters, preferably Hunyadi Janos, in the morning fasting. Podophyllin I have found to be very uncertain in its action. The dose which produces a mild effect in one person brings on violent griping in another, and has not the slightest effect upon a third. Nevertheless it will be found highly beneficial in a great many cases. When headache is much complained of it can generally be quickly relieved either by a

piece of muslin soaked in Tincture of Belladonna, and placed across the forehead, or by dry cupping, or a sinapism to the nape of the neck.

With regard to the use of stimulants by the Bilious, there is no doubt that in a great many cases they might be advantageously forbidden altogether. But there is another class of persons of delicate fibre and digestion who cannot, or who believe they cannot withstand the wear and tear of life without alcohol in some form. To such persons we must give the most explicit directions as to its use. Old Scotch or Irish Whiskey in moderate quantities well diluted (1 oz. in 3 oz. of water), and taken with food, I believe to be least hurtful. Marsala can do very little harm when taken in moderation, and good dry Champagne is not likely to be used habitually to excess on account of its high price. Any of these may be allowed to persons who are addicted to their use; but it is well to enforce upon such persons that none of them should be partaken of before dinner time, providing that meal is consumed after 2 o'clock p.m.

DISEASES OF THE BILIARY PASSAGES.

Catarrh of the Bile-Ducts.

CATARRHAL JAUNDICE.

THE mucous membrane which lines the bile-ducts is extremely prone to catarrhal inflammation, but very frequently when it occurs its existence is not made manifest by any prominent symptom, such as jaundice, and even in the dead body, when death has been preceded by symptoms which might well be attributed to catarrh of the bile-ducts, it is by no means always possible to demonstrate satisfactorily the traces of the disease; only a viscid, whitish fluid, chiefly made up of cylindrical epithelial cells being found in the ducts.

Catarrhal inflammation of the bile-ducts is a very common disease; by far the greatest number of cases of jaundice met with in practice being produced by that state of the ducts. It is, however, a very mild disorder which usually runs a quick course towards recovery.

In this disease the mucous membrane of the bile-ducts becomes swollen, and its natural secretion increased in quantity and more viscid in quality, thus tending to block up the ducts, and prevent the bile from flowing onward into the bowels. When the bile is thus pent up it becomes absorbed, and if the common bile-duct is obstructed, or a sufficiently large area of the liver is involved in the disease, jaundice supervenes. In this manner it is very probable the jaundice observed in connection with both acute yellow

atrophy of the liver and phosphorus poisoning is brought about, except that in these cases it is the capillary bile-ducts which are affected.

Catarrhal inflammation is much more common in the larger bile-ducts than in the smaller, because the former are more liable to be irritated by the passage of gall-stones down them, and to the extension of duodenal inflammation up them, than is the case with the latter.

The common bile-duct is occasionally found obstructed at its duodenal orifice by a firm plug of inspissated mucus. Such a substance passing down the duct might give rise to symptoms of gall-stone colic and cause embarrassment by no stone being found in the motions during life, or in the bowels after death.

It occasionally happens that the ducts in a portion of the liver remain permanently closed after catarrhal inflammation has subsided in the rest of the gland. Dilated bile-ducts, loaded with bile-pigment are then found in the rear of the obstruction.

Etiology.—Catarrhal inflammation of the biliary passages very commonly originates in the extension by continuity of surface of gastro-duodenal catarrh into the common bile-duct. Hence, all the agencies which induce gastro-duodenal catarrh may also excite catarrh of the bile-ducts. These agencies include such dietetic errors as overloading the stomach with indigestible food or spirituous liquors, bad smells, or other insanitary conditions. The disease may also exist as an epidemic, cold, wet, and windy weather predisposing to it; some of the cases even running on to a fatal termination in acute yellow atrophy of the liver; the majority, however, recovering. Stasis of blood in the veinous system of the liver, consequent upon mitral regurgitation or emphysema of the lungs may also produce catarrh of the bile-ducts. It may also be produced, especially in the cystic and common bile-ducts, by the passage of unhealthy bile or gall-stones down them. During its sojourn in the gall-bladder, the bile becomes concentrated, and if it be unhealthy its irritating effects on the ducts is

thus greatly increased; moreover, in the gall-bladder concretions are sometimes formed which are very liable to fret the cystic and common bile-ducts during their passage into the duodenum.

Certain poisons, especially phosphorus, mercury, and copper, amongst minerals, and the poison of venomous snakes, the syphilitic virus, cholera, and typhus, amongst animal poisons, frequently produce this disease.

Young persons who have previously enjoyed good health are most frequently the subjects of catarrh of the bile-ducts; but persons advanced in years, who are subject to gout not infrequently suffer from it.

Symptoms.—The symptoms of catarrhal inflammation of the bile-ducts are those of gastro-duodenal catarrh with jaundice superadded. There is some degree of fever, with slight, if any, pain in the region of the liver. If many of the ducts in the liver, or the common bile-duct become closed by tumefaction, or viscid secretion, there will be jaundice accompanied by slight enlargement of the liver, which latter, if accessible to palpation, is found to be smooth. Gastric symptoms are usually present in a marked degree. The urine is generally discoloured by the presence in it of bile pigment; and if the common bile-duct be completely occluded, the motions will be found to be clay-coloured, and the gall-bladder slightly distended.

Diagnosis.—The diagnosis of catarrhal jaundice must be based upon the age of the patient, and the symptoms of gastric catarrh which existed prior to the jaundice or accompanied its advent, and which can usually be traced to some indiscretion in diet or exposure to cold, &c. Diseases of the heart and lungs must be carefully excluded. Catarrhal jaundice can easily be recognized by the following characteristics: the liver is generally found not greatly enlarged, with a distended gall-bladder, and complete absence of pain.

Prognosis.—The prognosis is generally favourable in catarrhal jaundice, recovery in from ten to thirty days being the almost invariable rule. But until that happy termination takes place, great caution in pronouncing a favourable prognosis is to be observed,

because it is in these apparently simple cases of jaundice that grave symptoms of acute yellow atrophy now and then develop themselves.

TREATMENT.—The treatment need not be very active. A mild mercurial or podophyllin purgative to begin with, followed by alkalies and bismuth before meals will fulfil two of the indications for treatment, namely to get rid of offending matters out of the bowels, and to allay the irritations in the mucous membrane of the stomach. The diet must be carefully regulated, and should only consist of soups and light vegetable food as long as gastric symptoms are present. Alcoholic drinks must be forbidden, and acid drinks, such as lemonade, allowed. When there is a history of gout, great benefit will be derived from the addition of Colchicum and Iodide of Potassium to the remedies already mentioned. In the syphilitic variety of the disease a mild mercurial course is the appropriate treatment, except in those cases in which the jaundice has arisen during the administration of that drug, or as a result of its abuse, under which latter circumstances the Iodide of Potassium and Chloride of Ammonium must be resorted to.

When the gastric symptoms have subsided a more liberal bill of fare may be allowed. White fish, chicken, and in a short time mutton and beef may be ordered; and the the alkaline treatment may be changed to a course of nitro-muriatic acid in Infusion of Taraxicum.

DISEASES OF THE BILIARY PASSAGES.

Exudative Inflammation of the Biliary Passages.

CHOLANGITIS AND CHOLECYSTITIS.

BESIDES catarrhal inflammation, the bile-ducts are liable to suffer from (1) Purulent Inflammation, where the matter secreted is pus; (2) Diphtheritic or Croupal, where the matter effused forms a solid albuminous layer on the diseased surface; and (3) Ulcerative Inflammation, where necrosis and abrasion of the upper layer of the mucous membrane is produced. These diseases of the biliary passages, which are far more serious in their results than catarrhal inflammation, and give rise to quite a different train of symptoms, have been but slightly investigated, partly owing to their rarity, and partly owing to the ambiguity of the symptoms they give rise to.

The symptoms produced by exudative inflammation of the biliary passages are of a very vague character and may be entirely masked by those of other diseases during the course of which they usually occur. Simple catarrh of the bile-ducts may run on to the more grave forms of inflammation without our being aware of the process which is taking place.

Etiology.—The vast majority of all the cases of purulent inflammation and ulceration of the bile-ducts are produced by the presence of gall-stones or other foreign bodies within them, irri-

tating the mucous membrane, the disease being usually confined to the immediate neighborhood of the irritant. Sometimes, however, the disease spreads from the part primarily affected, and implicates the whole system of bile-ducts. The purulent discharge from one of these ulcers produced by the irritation of a gall-stone may contaminate the portal blood, and thus lead to multiple pyæmic abscesses in the liver.

Purulent inflammation and ulceration of the gall-bladder and bile-ducts may also occur during the course of typhoid fever, yellow fever, dysentery, &c., occasionally they are produced by external pressure of a tumor or cancer; and the gall-bladder may be so affected by the irritating cancerous discharge mixing with the bile.

When these diseases have lasted for some time, gangrene of the walls of the gall-bladder may take place, which if not preceded by adhesive peritonitis over the affected part, rupture or ulcerative perforation of the gall-bladder or larger bile-ducts may occur, followed by extravasation of the bile into the peritoneum, leading to fatal peritonitis.

The frequency with which ulceration is set up by concretions in the gall-bladder in typhoid fever and allied diseases is doubtless due to the low vitality of the system which is known to exist in those cases.

These diseases of the gall-bladder, as might be expected from their causation by gall-stones, are almost exclusively confined to mature age, and are more frequently observed in the female than in the male sex.

Pathology.—As the result of gall-stones, the gall-bladder is sometimes found after death greatly increased in size, the contents consisting of bile, mucous, or pus, in which varying numbers of gall-stones are floating. Its walls may be in a condition of inflammatory swelling and thickening; the mucous membrane may be found merely reddened or it may present here and there whitish-gray croup-like false membranes, or the inflammatory process may have progressed to ulceration. At times only a single ulcer may

be present, at others, several, the mucous membrane being entirely broken up and destroyed. The ulcers are generally found in the position usually occupied by the gall-stones—namely, in the fundus of the gall-bladder. The walls of the gall-bladder in the situation of the ulcers have a great tendency to gangrene, so that escape of its contents into the peritoneum followed by rapidly fatal peritonitis, or if adhesive inflammation have preceded perforation, to extensive cavities filled with bile and pus, which may burrow in various directions, and ultimately open either in the abdominal walls, or even through the diaphragm into the mediastinum, pleura, or bronchi.

The bile-ducts, both within and without the liver, as a result of purulent inflammation, may be found dilated and thickened, and either filled with pure pus, or a mixture of pus and bile. The mucous membrane of the larger ducts may be swolen, flaccid, excoriated, or even superficially ulcerated. Ulcerative perforation of the common bile-duct is of comparatively frequent occurrence.

The finer bile-ducts within the liver may be found distended with pus, or their walls may be broken down in numerous places, leading to suppuration of the hepatic tissue. In these cases, which are almost always due to the pressure of calculi, the liver is infiltrated with a large number of abscess-like cavities, which rarely exceed the size of a pea, but may form large cavities by the coalescence of several smaller ones. These cavities contain a thick, yellowish green mass, consisting of bile and inspissated pus, with which small soft biliary calculi are often mixed. They are termed *biliary abscesses*, on account of their contents, in contradistinction to the ordinary hepatic abscesses, The small abscesses are originally surrounded by softened hepatic tissue, but at a later period they appear to be more sharply defined, and surrounded by a fibrous capsule. The hepatic tissue which remains unaffected by ulceration in these cases, is of a deep bilious or olive-green colour, and is found in a state of parenchymatous or even granular degeneration.

Milder phlegmonous inflammations also occur in the bile-ducts within and without the liver. They lead to a connective tissue

thickening of the walls of the canals, which are also usually found filled with calculi, and diffusely dilated, or provided with irregular pouches corresponding to the individual concretions. The increase of the connective tissue and the pressure of the distended bile-ducts may lead to circumscribed atrophy, or wide-spread cirrhotic contraction of the liver.

In the extremely rare cases of *croupous* inflammation of the bile-ducts, succulent whitish, tubular membranes of fibrin are observed in them, exactly similar to those found in the bronchi in plastic bronchitis. These extend with uniform thickness over the mucous membrane of the ducts, from which they are readily detached, leaving the mucous membrane sometimes unchanged and sometimes reddened and flaccid.

Symptoms.—Inflammation of the bile-ducts may run its course without producing any symptoms, and it is not until a *post-mortem* examination has been made that the changes which have taken place are revealed. This is especially the case if the disease have been developed during the course of typhoid or other fever. Definite symptoms only make their appearance, as a rule, when the bile-ducts are obstructed, and stasis of the bile has taken place, or when the inflammatory process has spread to the adjacent organs; but even under such circumstances the process may be latent.

Inflammation of the gall-bladder is characterised by a dull pain which is at first confined to its immediate vicinity, but may spread over the entire right hypochondrium. The gall-bladder may, under certain circumstances, be felt projecting from the liver as an elastic or fluctuating, somewhat tender pear-shaped tumor.

Among the general symptoms may be mentioned *fever*. Usually the temperature remains within moderate limits. Slight chilly sensations, or even well-marked rigors are observed, and may be repeated at regular or irregular intervals for a long time. In many cases inflammation of the gall-bladder only discloses itself

when local peritonitis, or the alarming symptoms of perforation of the peritoneum with extravasation of bile develop themselves.

Exudative inflammation of the hepatic and common bile-duct cannot be distinguished from catarrhal inflammation of those parts unless complete obstruction take place. In that case the jaundice will be more persistent, the liver will be enlarged and tender, and bile will be entirely absent from the motions.

The fever and chills may continue for months with occasional remissions. Emaciation gradually progresses, the strength concurrently diminishing, and death finally occurs from exhaustion, or in rare cases convalescence sets in.

Diagnosis.—The diagnosis in these diseases is beset with the greatest difficulties. In general we may suspect inflammation of the biliary passages has set in when pain and swelling are observed in the region of the liver, accompanied by repeated irregular chills, rise of temperature and other symptoms of febrile movement, in a patient who is jaundiced, and whom we know, or have good reason to believe to be suffering from gall-stones. If these symptoms occur with free intervals of longer or shorter duration, but with progressive emaciation and failure of strength, we must suspect chronic inflammation of the bile-ducts. However, as previously mentioned, the whole process of inflammation and ulceration may progress for a long time without producing any appreciable symptoms whatever, until perforation, leading to extravasation of bile produces symptoms of peritonitis from which the sufferer speedily sinks.

When the gall-bladder is sufficiently dilated as to be discovered by palpation as a tender, elastic or fluctuating, pear-shaped tumor, inflammation in that viscus may be diagnosed with probable certainty. As a rule, however, the only symptoms present are those of local peritonitis, in the region of the gall-bladder; the inflammation and ulceration in the gall-bladder not giving rise to any definite symptoms.

Prognosis.—Purulent and ulcerative inflammation of the biliary passages are very grave diseases which carry in their train a num-

ber of serious consequences, amongst which may be mentioned rupture of the gall-bladder and bile-ducts with extravasation of bile, hepatic abscesses, hæmorrhages, purulent pylephlebitis, cirrhotic contraction and parenchymatous degeneration of the liver, &c. The prognosis, therefore, must be, at least in those cases in which the symptoms are sufficiently intense to admit of diagnosis, very unfavourable. Recovery, however, very often occurs in the milder cases; this happy event usually taking place after the removal of the cause of the disease, such as gall-stones. That even deep and extensive ulceration may recover spontaneously has been abundantly proved by the cicatrices which are frequently found *post-mortem* in the mucous membrane of the gall-bladder.

The duration of the disease may extend from a few days, as in those cases which occur in connection with typhoid diseases, to several months or years, as is the rule when it is due to the irritation of gall-stones.

Treatment.—In the severe forms of the disease, the local abstraction of blood by means of a few leeches or cups applied over the gall-bladder, or by leeches applied in the neighborhood of the anus may relieve pain and subdue the inflammatory process. Warm poultices, or an ice bag, according to the inclination of the patient, applied over the region of the liver will have a further soothing effect, or if pain and tenderness be very great, extract of opium and extract of belladonna in equal proportions may be smeared on the same region previously to applying the poultice. The bowels must be kept open by means of mild saline purgatives, and the diet restricted to vegetables. The patient must be kept extremely quiet on his back, and all pressure in the region of the liver, movements of the body, straining at stool, &c., must be carefully avoided. The internal use of opiates may be indicated, and may be administered in full doses.

In the milder and more chronic forms of the disease, where the local symptoms are less intense, the repeated application of mustard or cantharides blisters are indicated. The alkaline mineral waters of Carlsbad, Ems, and similar waters should be system-

atically administered, and treatment directed to the symptoms generally as they arise. Quinine is the remedy indicated, should rigors and other febrile symptoms set in; at the same time the diet must be strengthening and non-irritating.

DISEASES OF THE BILIARY PASSAGES.

Dilatation of the Gall-Bladder.

DILATATION of the Gall-bladder may be produced by several very different causes. In its simplest form, dilatation of the gall-bladder consists in an accumulation of watery fluid free from bile within its walls, which are thin and distended; the cystitc duct, or the neck of the gall-bladder is permanently closed so that ingress and egress of bile no longer take place. This condition is properly termed dropsy of the gall-bladder (hydrops cystidis felleæ).

Another form of simple dilatation of the gall-bladder is caused by an accumulation of bile within it. A few cases have been recorded where a gall-stone has been so fixed in the neck of the gall-bladder or cystic duct, as to act as a valve, permitting the bile to enter the gall-bladder, but preventing its exit. By this means the bile has accumulated in the gall-bladder, and produced a simple dilatation very difficult to diagnose, because jaundice may be absent, and the colour of the motions natural; but the rarity of such an occurrence renders embarrassment from this cause extremely infrequent.

Simple dilatation of the gall-bladder may arise without closure of the ducts, in consequence of paralysis of its muscular coat, which prevents it from contracting to expel its contents.

Empyema of the gall-bladder is produced by the accumulation within it of the purulent products of inflammation, consisting of either pure pus or pus mixed with bile.

ACCUMULATIONS OF GALL-STONES within the gall-bladder sometimes take place to such a degree as to present a distinct tumor appreciable by palpation through the abdominal parieties.

CANCEROUS DEPOSITS in the walls of the gall-bladder may produce a hard, nodulated tumor perceptible through the abdominal walls.

ETIOLOGY.—Simple dilatation of the gall-bladder is, as a rule, produced by occlusion of the common bile-duct. The occlusion may be either temporary, when caused by catarrhal inflammation; or permanent, when caused by an impacted gall-stone or obliteration of the duct as a result of adhesive inflammation. Under these circumstances the gall-bladder may become tremendously distended, so as to contain many pints of bile. But it is not often that the dimensions of the tumor are so great, and if the obstruction be temporary, these never surpass very moderate limits.

In order that dropsy of the gall-bladder may occur, it is first necessary that the cystic duct be permanently closed. As soon as the flow of bile to and from the gall-bladder has ceased, the bile contained within the gall-bladder slowly undergoes a change; the biliary constituents and mucus are gradually absorbed, and chronic inflammation setting in, serum transudes from the blood vessels of the walls of the gall-bladder which may thus be distended to a very great degree.

Empyema of the gall-bladder is brought about in a manner somewhat similar to that of simple dropsy, except that the inflammation set up is of a purulent character.

PATHOLOGY.—Nothing very remarkable is observed *post-mortem* in these cases beyond an increase in size of the gall-bladder, which is more or less firmly adherent to the liver. The walls are correspondingly attenuated, and the mucous membrane smoother. The size to which this viscus may dilate is subject to great variations, from half a pint to many quarts of fluid having been removed from it. Babington relates a case in which the gall-bladder contained three wash-hand basinfuls of bile.

The constitution of the fluid contents of the gall-bladder approaches nearer to that of bile the shorter the time the disease has been developing. When a long period has elapsed the fluid will be found thin and almost colourless, or of a pale green tint.

In empyema, the gall-bladder is found as a tensly-filled pyriform sac, varying in size up to that of a man's head. The sac is sometimes more or less firmly attached by inflammatory adhesions to the surrounding parts. The walls of the inflamed gall-bladder are almost always friable and easily torn. Ulcers are usually present on the mucous membrane. The contents consist of pus, or pus mixed with bile. Frequently numerous gall-stones are also present.

Symptoms.—The most important symptom of enlargement of the gall-bladder is the presence of a pyriform tumor extending from the region of the gall-bladder downwards. This tumor can often be detected by inspection, as a flattened prominence of the abdomen. The extent and shape of the tumor can be determined by percussion, and it will be noticed that it becomes smaller as it approaches the border of the liver. If the tumor containfl uid it will be found to be elastic and more or less distinctly fluctuating, and the lower portion of it can often be moved somewhat from side to side.

Diagnosis—The principal diseases which must be differentiated from tumor of the gall-bladder are (1) Echinococcus sacs, (2) Abscesses of the liver, (3) soft cancers which proliferate at the lower border of the liver. (4) When the tumor is exceedingly large, free *ascites*, *ovariam cyst*, or a huge *hydronephrosis*, might at first be suspected rather than dilatation of the gall-bladder, and as a matter of fact, these tumors have been mistaken for each other by some of our most able modern surgeons.

Echinococcus sacs have usually a more hemispherical shape, as they project from the liver by a broad base, while the enlarged gall-bladder presents an elongated pyriform shape.

In the case of an hepatic abscess we have to deal with a tumor which is at first firm, then gradually becomes softer: it is also

accompanied with great constitutional disturbance such as fever, rigors, pain in the region of the liver and right shoulder; the swelling produced by it is much broader and more diffuse than that produced by tumor of the gall-bladder. In dilatation of the gall-bladder, the tumor always presents the same resistance and elasticity, can be more sharply defined from the beginning, has a more slender form, and constitutional disturbance is altogether absent or very slight.

Soft cancers which proliferate at the lower border of the liver may be diagnosed by their more rounded and nodular form and the usually marked cancerous cachexia.

If an accumulation of gall-stones cause the enlargement, the tumor is hard and nodulated, usually moveable, and painless on pressure. Occasionally a crackling sensation is experienced on manipulating the tumor, or the patient complains of a sensation of a weight rolling from side to side when he turns in bed. There may be jaundice or there may be a previous history of biliary colic. The size of the tumor does not vary, or its growth is slow and imperceptible. Should ulceration of the gall-bladder occur as a result of the accumulation, the symptoms will be very materially changed. Local peritonitis setting in, very severe pain is developed over the site of the tumor, the latter becomes adherent and immovable, and its dimensions increase rapidly.

Cancer of the gall-bladder presents a hard somewhat nodulated tumor about the size of an orange, more or less in the region of the gall-bladder. It is usually adherent and immovable, very tender on pressure, and usually the seat of severe lancinating pains. The growth is usually rapid. Jaundice and vomiting are common symptoms, the former owing to the extension of the cancer to the common bile-duct, and the latter to the pressure of the tumor on the pylorus. Fistulous communications with the intestinal canal, and particularly with the colon, are not uncommon, and consequently the passage of a large gall-stone, with or without hæmorrhage per anum, occurring in connection with a tumor like that now described, would corroborate rather than

refute the diagnosis of cancer. In addition to the other specific symptoms there is also rapid emaciation with the well-known phenomena of the cancerous cachexia.

Without making an exploratory puncture, it is almost impossible to be certain whether a fluctuating tumor of the gall-bladder contains bile, mucus, water, or pus.

An accumulation of bile in the gall-bladder may be surmised from the following characters; viz., jaundice is present which gradually becomes intense, bile is absent from the motions, the liver is enlarged and tender, the tumor is elastic or fluctuating, pear-shaped, and somewhat tender, and projects from the edge of the liver in the situation of the gall-bladder.

Empyema of the gall-bladder, caused by closure of the cystic duct from the irritation of gall-stones may be recognised by the pain and tenderness being greater than in simple distension from bile. To these are added febrile disturbance, rigors, pyrexia, and night sweats. There are in fact all the characters of hepatic abscess, from which even its shape and situation may not suffice to differentiate it. The previous history will, however, assist in throwing light on the case. It is only the tropical abscess of the liver which is simulated by empyema of the gall-blader, and that variety of abscess is of very rare occurrence in persons who have dwelt exclusively in a temperate climate. Furthermore, there is an absence of jaundice, the motions contain bile, there is no general enlargement nor tenderness of the liver, and there may be a previous history of gall-stones.

Course &c.—Dilatation of the gall-bladder, due to occlusion of the common bile-duct and empyema, generally devlop more quickly than simple dropsy due to closure of the cystic duct, which latter runs a very chronic course. Enlargements of the gall-bladder, the result of the inflammatory processes very often slowly disappear under treatment; occasionally they open and discharge externally or into the bowels, but frequently they rupture and pour their contents into the peritoneum, and this untoward event is not confined to the varieties under consideration, but has

been noticed in simple dilatation from accumulation of bile. G. Frank (quoted by Von Schueppel) observed a pregnant woman in whom the act of parturition was the exciting cause of rupture of the distended gall-bladder, its fundus having contracted adhesions to the fundus of the uterus.

Besides the danger of rupture, the continuance of ulceration and suppuration may lead to hectic fever with rapid emaciation, night sweats &c., which ultimately destroy life by exhaustion.

TREATMENT.—The treatment of simple dilatation of the gall-bladder from closure of the common bile-duct does not differ from that of jaundice due to gall-stones and catarrhal icterus, (which see), and the treatment recommended for inflammation of the biliary passages will be appropriate for inflammatory conditions of the gall-bladder.

If closure of the neck of the gall-bladder have occurred, and distension of that organ have attained such dimensions as to threaten rupture, or if the organ be filled with pus, and symptoms of hectic fever with emaciation develop, then the evacuation of the gall-bladder by puncture is indicated. The operation presents no danger if the gall-bladder be sufficiently adherent to the abdominal walls. If this be not the case, we must artificially produce adhesive inflammation by the application of Vienna paste, or by making an incision into the abdominal walls down to within about one or two lines of the peritoneum, as originally suggested by Graves for opening abscesses of the liver. This incision must be plugged with lint, and in a few days, adhesion having taken place, a suitable sized trocar is to be plunged into the gall-bladder. Generally a single puncture suffices, but if the gall-bladder refil, the operation must be repeated.

DISEASES OF THE BILIARY PASSAGES.

Occlusion and Dilatation of the Biliary Passages.

THE most prominent symptom produced by occlusion of the common bile-duct or the hepatic duct, namely, jaundice, has already been treated of: it now remains to examine into the effects produced, not only on the bile-ducts and liver, but also on the system at large, by the persistence of the obstruction for a lengthened period; or in other words, the symptoms, pathology, and treatment of *chronic jaundice.*

Constriction or occlusion may occur in any part of the excretory apparatus of the liver, and may either be the result of disease of the ducts themselves, more especially inflammation, or may depend upon the condition of the surrounding parts, such as tumors pressing upon the ducts, or connective tissue deposits, &c., &c.

The various causes which give rise to chronic closure of the biliary ducts may be divided into three classes. (1) *Foreign bodies* which lie in the lumen of the common bile-duct or hepatic duct and obstruct it, such as calculi or worms. (2) *Inflammatory processes* in or about the ducts may lead to their obstruction by the contraction of cicatrices of ulcers producing stenosis of the duct without obliteration of its lumen, or complete occlusion by adhesion of its walls; or cords of connective tissue in the hepato-duodenal ligament may lead to stenosis or occlusion by simple compression. (3) *Tumors*, such as cancerous growths situated in

the liver or surrounding organs may compress the bile-duct from without, or by growing into its wall may narrow or fill the lumen of the canal.

Every constriction, and still more, every occlusion of the bile-ducts entails retention of the secretion and a dilatation of the ducts, which according to the site of the obstruction, is at one time limited to a few branches of the duct within the liver, or, when the hepatic duct itself is obstructed, extends over all its branches, or, in the case of obstruction of the common bile-duct, which is of much more frequent occurrence, the large ducts, together with the gall-bladder, are involved in the dilatation.

Pathology.—The pathological consequences of closure of the biliary ducts are best exemplified in those cases in which the obstruction is situated in the duodenal orifice of the common bile-duct, for instance, when complete atresia has been caused by inflammatory adhesion of the walls of the duct. Under such circumstances the bile-ducts outside the liver become dilated, sometimes to an enormous extent, and also increased in length. In its higher grade the common bile-duct looks like a well-filled loop of small intestine. The hepatic duct is dilated to the same extent, and each of its two chief branches will readily admit one or two fingers, and the other branches within the liver are also relatively dilated. The dilated bile-ducts present either a simple cylindrical shape, or they are provided with numerous shallow and ampulla-shaped recesses. If the cystic duct be patent, both it and the gall-bladder may be greatly increased in size. The walls of the larger bile-ducts and gall-bladder appear in comparison with the marked dilatation, relatively not greatly thickened. The mucous membrane of the ducts is smooth, and the openings of their mucous glands are enlarged.

The fluid contained in the dilated bile-ducts varies both in quantity and nature according to the degree to which the dilatation has advanced and also the time during which it has continued. In the very high grades of dilatation of the ducts and gall-bladder the quantity of fluid contained in the former may amount to about the same as in the latter.

In the earlier stage of the disease the fluid which accumulates in the ducts is of course pure bile. After a time the quantity of mucus contained in it becomes greatly increased, and after the disease has lasted for several weeks or months the fluid has lost its bilious character and is of a clear, pale glass-green colour, and is more or less mucous, the consistence of which resembles thin synovia.

The sequence of events which accrue from long-continued cessation of the flow of the bile appears to be as follows. When from any cause, occlusion of the common bile-duct has occurred, at first the bile is secreted as before, but its onward flow into the duodenum being stopped, it accumulates in the hepatic ducts, and also in the gall-bladder if the cystic-duct be patent. As a result of increased pressure the ducts of the liver are dilated, and progressive enlargement of the liver is induced which may continue for a long time. Under the circumstances already mentioned, the gall-bladder participates in the dilatation, but if the hepatic duct be occluded dilatation is confined to the ducts within the liver. Concurrently with retention of bile within the liver, absorption of that fluid takes place, and as a first result, the hepatic tissue itself becomes deeply stained of an icteric hue, which soon spreads to all the tissues of the body producing the well known phenomena of jaundice. The formation of bile in the hepatic cells and the absorption of that which is retained in the ducts takes place *pari passu* for a long time. But at a later period the pressure exerted by the dilated bile-ducts results in atrophy of the glandular tissue of the liver, secretion is impaired, and absorption getting the ascendency, the liver is reduced in size, and assumes a flabby shrivelled appearance. In many cases the hepatic cells ultimately become destroyed, while the entire organ is softened, and death supervenes under symptoms of blood-poisoning, or ulcerative processes are developed on the mucous membrane of the ducts, which ultimately implicate the surrounding parenchyma, and entail all the consequences that result from such ulceration.

Symptoms.—The most prominent symptom of occlusion of the hepatic and common bile-ducts is *jaundice* which has already been

fully discussed. If the occlusion be very suddenly developed the jaundice rapidly assumes a deep tint, bronze, brown, or dark olive green. The jaundice is not only permanent but is maintained at the same height as long as the obstruction lasts or the patient's life is continued; any variations in its intensity usually remain within very narrow limits. If a marked diminution in the intensity of the jaundice takes place despite the continuance of occlusion of the duct and complete decoloration of the motions, this must be regarded as an unfavourable sign, because it would show that the formation of bile has decreased in consequence of progressive parenchymatous degeneration of the liver.

Chronic jaundice is usually accompanied by severe pruritus of the skin which causes great discomfort to the patient. This disagreeable symptom may precede, concur with, or follow soon after the discoloration, and is usually most severe on the palms of the hands, the soles of the feet, and between the toes; but is frequently diffused over the entire body. It increases at night, in bed, or in a warm room, the patient unavailingly scratches the skin, and sleep is inhibited. The itching is probably a neurosis caused by the presence of bile acids in the blood, but in rare cases a prurigo-like papillary eruption, an urticaria or lichen, and even boils may be seen on the skin.

The functions of the intestines are greatly perturbed in consequence of the absence of bile from their canal. Constipation is the common result of the withdrawal of nature's stimulus. The motions from the bowels are pale coloured like moist pipe-clay, very fœtid, and of the consistence of thick porridge. Hæmorrhages from the bowels and stomach are frequent during the later stage of the disease, and may impart a deceptive colour to the motions.

The digestive functions are also deranged. The tongue is coated with a thick, greasy, brownish gray fur, a bitter taste in the mouth is complained of. Nausea with loss of appetite with a tendency to vomiting in the morning and an antipathy to fatty articles of food are usually present, and if fat be partaken of stearrhœa supervenes: this is especially the case if the pancreatic

duct be involved in the disease which obstructs the common bile-duct, as, for instance, cancer of the head of the pancreas.

Loss of appetite does not follow as a matter of course from stoppage of the flow of bile into the intestines, on the contrary, dogs in whom biliary fistulæ have been established, develop ravenous appetites, and have been known to live in this condition for years.

Headache and a general feeling of *malaise* are present; moreover the sufferers are morose, at times irritable, apathetic, or hypochondriacal. They are incapable of prolonged work, their energies are rapidly exhausted, and general emaciation of the body takes place as a result of defective nutrition.

The local symptoms due to closure of the bile-duct are enlargement of the liver during the first period of the disease. The liver emerges from behind the costal wall and may be demonstrated by percussion reaching two or three inches below the costal margin. The patient experiences a feeling of fullness in the right hypochondriac and epigastric regions, and pain, due to stretching of the capsule of the liver and distension of the gall-bladder is present in most cases. The pain is greatest in those cases where there is peri-hepatitis, or cancer of the liver, or where the bile-duct is compressed by a tumor which at the same time compresses and stretches the hepatic plexus of nerves. The liver is readily accessible to palpation, and is felt as a smooth, moderately firm, and uniformly resisting body. If the common bile-duct be occluded and the cystic duct permeable, the gall-bladder will also participate in the dilatation from the retention of bile, and may be discovered as a hemispherical, or rounded, elongated swelling, which may be sharply defined by careful percussion. By palpation, also, the gall-bladder is usually readily perceptible as a smooth, tense body, and is often visible as a prominent tumor. In some cases the gall-bladder reaches such dimensions that it lies upon the ilium and simulates an independent cystic tumor, which might be mistaken for a cystic tumor of the ovary.

The enlargement of the liver usually progresses for several months, a halt then occurs, and after this has lasted for some

time, a progressive diminution of the organ takes place, while the gall-bladder remains at its former size, or even becomes more perceptible than previously. After the liver has shrunk down to its normal dimensions it may still further diminish in size so as to constitute a true atrophy of the organ.

When occlusion of the bile-duct has lasted for a long time and parenchymatous degeneration has taken place in the liver severe nervous symptoms sometimes develop, such as somnolency merging into coma, delirium, carphology, &c., which forebode the near approach of death. Various opinions have been advanced in explanation of the production of these symptoms which the reader will find discussed in the article on Jaundice.

Diagnosis.—The mere fact of jaundice being present in any given case is sufficient evidence of the existence of some obstruction of the bile-ducts; but when we come to decide the nature and locality of the obstruction we are face to face with a very difficult problem, on the solution of which the prognosis and treatment should alone depend. The difficulty is greatest when we attempt to differentiate between the various diseases which are known to produce permanent occlusion of the hepatic and common bile-ducts, and it may be stated *in limine*, that in some cases it is not possible to say with any degree of certainty, what morbid condition is at work: but this fact ought not to deter the practitioner from *attempting* to solve the difficulty.

The lower grades of jaundice which are exemplified in Catarrhal and Hyperæmic icterus, are only liable to be confounded with occlusion of the bile-duct during the first few days, before the jaundice has arrived at its full intensity. In the earlier stages, Catarrhal icterus is recognised by the pre-existence, usually, of exposure to cold, or of gastric symptoms, the slight discoloration of the skin, and finally, the short period during which the disease lasts. Hyperæmic icterus may be excluded from the diagnosis by noting the absence of any indication of disease in the chest walls, lungs and heart. In young children, simple icterus neonatorum rarely lasts beyond two weeks after birth; and a severe icterus which lasts beyond that period may be attributed to con-

genital impermiability of the large bile-ducts, as there is no disease of the liver occurring at this period of life which is attended with chronic jaundice and complete discoloration of the fæces, not even syphilitic disease of the liver.

When, in the adult, occlusion of the bile-ducts has continued for a long time, the question arises: What is the morbid condition at work which produces the jaundice?

The only disease of the liver characterised by chronic jaundice unaccompanied by occlusion of the large excretory ducts which is liable to be confounded with occlusion of the ducts is the so-called hypertrophic variety of cirrhosis; but there are a great many diseases, not only of the liver itself but also of the surrounding organs, which may produce occlusion of the bile-ducts and chronic jaundice, which it will be necessary, as far as possible, to differentiate.

The hypertrophic variety of cirrhosis may be recognised by the intermittent character of the jaundice, the stools being sometimes bilious and sometimes clay-coloured, the complexion undergoing simultaneous variations. The liver is more highly enlarged than is the case in occlusion of the bile-ducts, and usually extends to a hand's breadth below the ribs; besides, the gall-bladder is not distended.

We now come to the differential diagnosis of those conditions which may produce occlusion of the common excretory duct.

A large gall-stone remaining impacted in the common bile-duct and permanently occluding it, may be accepted as the cause of chronic jaundice when the history shows that the patient has formerly suffered from paroxysmal pain in the epigastrium followed by jaundice of short duration, or if well marked symptoms of hepatic colic have preceded the present attack of jaundice which is also accompanied by the symptoms of dilatation of the bile-ducts recounted above. The diagnosis will be assisted by attending to the etiological relations of gall-stones. Febrile symptoms of an irregular character, caused by inflammatory processes about the bile-ducts rather favour gall-stones, because they frequently irritate the ducts.

Closure of the duct by cicatritial contraction inside the duct itself can only be surmised if the clinical history seems to indicate an attack of gall-stone colic, the stone having injured the mucous membrane of the duct during its passage into the intestine.

Occlusion of the ducts by fibrous bands which have been developed in the course of perihepatitis is recognised with very great difficulty in the living subject by the symptoms of that disease following some one of its known causes, such as perforating ulcer of the stomach, general peritonitis, constitutional syphilis, &c.

Cancerous growths which start from the liver or the walls of the bile-ducts and obstruct them, may usually be recognised by palpation, or by the fact of cancer being present in other parts of the body, and by the cancerous cachexia which finally develops.

Cancer of the duodenum and of the head of the pancreas may be recognised by the following points :—The patient has complained for several weeks before the development of jaundice of a severe pain in the region of the duodenum, which continues after it is developed, and is increased a few hours after meals, at which time it is accompanied by nausea and even vomiting. Hæmatemisis and melæna are usually present when ulcerative degeneration of duodenal cancer has set in. In only a few cases can a hard, tense tumor be discovered through the abdominal walls. Jaundice continually increases in severity until death, which usually takes place in about four or five months after the development of the jaundice. This last is an important diagnostic sign, because intense jaundice produced by cancer seldom continues for a longer period than six months.

Prognosis.—This depends entirely upon the nature of the cause producing the occlusion, and how the system tolerates the presence of bile in the blood. If an impacted gall-stone be the offending body the condition may continue for years (4 years, Budd, 6 years, Murchson) and the patient ultimately recover, through a fistulous communication being established whereby the bile finds its way into the intestines, or less favourably, externally

through the abdominal walls. On the other hand, hæmorrhages, exhaustion, or severe nervous symptoms may set in early and destroy life.

If cancerous growths have produced the occlusion, the prognosis is absolutely fatal; death usually taking place in these cases within six months of the development of jaundice.

During the first month of the disease the practitioner should be extremely cautious in giving an opinion as to the possible duration and termination of chronic jaundice, especially if the cause of the occlusion have not been positively settled.

TREATMENT.—This must be directed to the relief of the most annoying symptoms, to aid the removal of the biliary constituents from the blood, and to the maintenance of the nutrition and strength of the patient.

Regulation of the diet is of primary importance. The food should consist of milk, eggs, lean meat; all articles which are difficult of digestion must be excluded, as also alcoholic and malt liquors, fatty or saccharine matters, &c.

The bowels must be regulated. In most cases there is a tendency to constipation which must be relieved by the administration of saline purgatives, such as sulphate of magnesia, tartrate of potash, tartrate of soda and potash, &c. No good can be expected from drugs which stimulate the action of the liver, and mercury administered for any length of time would only tend to promote marasmus. No objection can, however, be urged against its occasional administration as a purgative.

Flatulence and other dyspeptic symptoms may be relieved by the administration of charcoal, which will also have a tendency to correct the fœtor of the motions. For the same purpose purified ox bile may be administered in doses of from gr. ij. to gr. x. in pills or gelatine capsules about two hours after meals. Creosote, carbolic acid, or better still sulpho-carbolate of soda in gr. x. or gr. xv. doses either before or after meals are very powerful in arresting putrifactive fermentation in the bowels. Cholate of soda in gr. x. doses in peppermint water, the alkaline carbonates,

and mineral waters, in combination with taraxacum or gentian, are all beneficial, not only to the flatulence but also to the digestive disturbances of all kinds.

The functions of the kidneys must be attended to, as they are the principal organs by which the blood is depurated of the constituents of the bile. Copious draughts of water produce a more rapid change in the fluids of the body and serve to promote the removal of these substances from the system. The occasional use of mild diuretics will also be advisable if there be no kidney disease.

The functions of the skin also demand attention. The patient must be warmly clad, and avoid sudden chills : diaphoretics and warm baths occasionally, will tend still further to promote the removal of bile pigment from the blood.

The annoying itchiness of the skin may sometimes be relieved by warm baths, fatty and irritating inunctions, and the internal administration of bicarbonate of soda or potash, acetic acid baths (½ pint of acid to 3 gallons of water), or a lotion of chloroform (1 part) and glycerine (5 parts). Olive oil, calomel ointment, corrosive sublimate washes &c., are also sometimes useful. I have seen great relief afforded by Wright's Liquor Carbonis Detergens. If these means fail, as they frequently do, recourse must be had to opium to procure sleep.

The patient's strength must be maintained by tonics, such as quinine, mineral acids with nux vomica or bark, and finally it may be necessary to allow small quantities of alcoholic stimulants.

DISEASES OF THE BILIARY PASSAGES.

GALL-STONES.

CHOLELITHIASIS.

THE Cholestearine of the bile is liable to be precipitated and to crystallize in the gall-bladder and bile-ducts during life-time, thus forming what are known as gall-stones or Choleliths.

These concretions vary in size from a small seed to a common fowl's egg. They are found solitary, but more frequently aggragated, as a rule, about five or ten at a time. The solitary calculi are usually the larger.

Solitary calculi when found in the gall-bladder, are globular, oval, or pyriform; but it is much more common to find them associated, in which case they have usually numerous polished facetts, the result of mutual pressure and attrition. They are occasionally found in almost incredible numbers, as many as 7,802 were counted by Otto in a single gall-bladder.

Gall-stones usually form round a nucleus, which may be biliary gravel, a tube cast, inspissated mucus, or some foreign body, such as a dried-up ascaris, or a fragment of a Faciola hepatica. On the nucleus, which is usually black, consisting of cholepyrrhin and lime, cholestearine is deposited, first in radiating crystals and subsequently in concentric laminæ. This laminated appearance of the crust is doubtless caused by pressure exerted on them by the gall-bladder. They vary in colour according to the amount of

bile pigment they contain, from pure white like balls of camphor (when they consist of almost pure cholestearine), to a deep black, but are most frequently of a reddish brown. Their weight is inconsiderable; when dry, they float readily in water. They have a soapy feel, and when placed in the flame of a candle, readily melt and burn with a bright flame.

When formed in the branches of the hepatic duct, they are small, rough, or tuberculated, and of a dark olive colour, approaching to black—so that they have been compared to peppercorns. They do not, however, often form in this situation except as a result of obstruction of the common bile-duct, or local inflammation and adhesions of its radicles. A cyst is thus formed in which the bile becomes inspissated and the colouring matter thrown down forming dark-coloured granules. These granules may become cemented by mucus secreted by the coats of the duct, so as to form a small calculus. Thus formed, concretions in the liver may give rise to cystic dilitation and ulceration of the ducts, and multiple abscesses in the liver.

Gall-stones of the hepatic variety are now and then found in the gall-bladder. They are heavier than the stones usually found in this situation, and do not burn so readily, and when burnt sometimes leave a considerable quantity of ash, consisting of carbonate and phosphate of lime. They are composed chiefly of biliary colouring matter, mucus, and lime.

Etiology.—In every case the presence of gall-stones is evidence of an unnatural state of the bile at the time of their formation.

There are certain circumstances of life which conduce to the formation of gall-stones. These are principally age, sex, and habits.

1. Age.—Gall-stones are seldom met with in young persons; the tendency to them increasing with age. After 25 or 30 they are of very common occurrence. They are, however, occasionally observed in early life; and have been known to produce fatal results by obstructing the common bile-duct even in infants.

2. Sex.—Females are more prone to suffer from gall-stones than males, in about the proportion of 3 to 2. Probably the preponderance of the disease in the female sex arises from the less active lives they lead conducing to biliary derangement.

3. Habits.—Excess in eating and drinking seems to predispose to the formation of gall-stones, as it also does to stoutness, and it is well-known that corpulent people are particularly liable to suffer from this disease. The habit of taking but one meal a day, by allowing the bile to remain a long time in the gall-bladder, where it becomes inspissated, has a like tendency.

Sedentary occupations, and confinement in-doors are conditions of life which dispose to gall-stones. They are especially frequent among literary men, prisoners, and people long bed-ridden. On the contrary, men who lead an active life in the open air seldom suffer from them.

Fat persons, past the middle age of life, who lead indolent lives and partake of rich food are very subject to gall-stones.

There is sometimes evidence of an hereditary tendency to gall-stones, and this is most marked in those families which suffer from the gouty and rheumatic diatheses.

Gall-stones are very frequently found in connection with cancer of the liver. This connection probably arises from pressure on the ducts by the tumor causing stasis of the bile.

Symptoms.—Any number of gall-stones may be present in the gall-bladder without producing any inconvenience whatever; but should one or more of them be washed down in the current of the bile into the cystic or common bile-duct, well-marked symptoms of hepatic colic are set up, which are particularly severe should the calculus be of large size, or rough and angular in form.

When a gall-stone has entered the cystic duct, the patient is attacked with a feeling of pain in the epigastrium, nausea, with much flatulence, an unusual nervous excitability, yawning or shivering, with a sense of constriction round the lower part of the chest. This is usually experienced two or three hours after the

principal meal, or after some severe muscular exertion, or shaking of the body, as by horse or carriage exercise. The pain usually moderate at first, gradually increases in severity, so as to cause the patient to bend double, with his chin resting on his knees, but also constantly changing his position, hoping in some way to obtain relief. There is great tenderness in the epigastric region, which radiates to both hypochondria, and to the right or both shoulders, especially the lower angle of the right scapula, or towards the neck, or even the right arm, but never descends downwards into the abdomen.—(Murchison p. 337.)

Vomiting rapidly comes on, of half-digested food. The bowels are confined. Rigors may occur; but more commonly only a sensation of coldness. The pulse is retarded. The act of vomiting is usually followed by temporary relief of pain. Very often it is accompanied by distressing hiccough.

In persons of a nervous constitution the pain sometimes causes fainting, delirium, or even epileptiform convulsions.

The pain comes on in paroxysms, which if frequent or protracted, induce great lassitude and exhaustion, the face being pale, the pulse slow, and the whole body covered with a cold sweat; occasionally there is profound collapse, which in rare cases has been fatal.

At the outset the pain may be relieved by pressure, but after a time the region of the gall-bladder becomes very sensitive to pressure; which condition continues for some time after the paroxysm has subsided.

Should the stone recede into the gall-bladder, all the symptoms cease: if it remain impacted in the cystic duct, dropsy of the gall-bladder may supervene, and perhaps ulceration or gangrene of the duct. But should it pass onwards into the common bile-duct there will be a sense of partial relief, owing to the larger calibre of that duct; but on arriving at the duodenal orifice, all the symptoms may return; and if in this situation, or in the common bile-duct, the stone become impacted, jaundice must supervene, since there is no outlet for the bile. It is, however, possible that bile may pass into the bowels even under these circumstances, if the

stone be of an angular shape so as not to completely occlude the lumen of the duct. If the stone pass onwards into the duodenum all symptoms of colic cease.

If the obstruction be permanent and complete, the jaundice will gradually increase in intensity, the liver progressively enlarge, the gall-bladder become much distended, and death will ultimately occur, unless the stone be forced into the bowel, or adhesive inflammation taking place between the duct and some part of the intestinal canal, or abdominal walls, the stone may be voided by ulceration and perforation, and thus lead to recovery.

This happy result, however, does not always take place, for gall-stones may ulcerate through the gall-bladder or bile-duct without the supervention of adhesive inflammation, and so pass into the peritoneum together with a quantity of bile, inducing fatal peritonitis. They may also, by producing ulceration or sloughing contaminate the portal blood, lead to multiple pyæmic abscesses in the liver.

Even after a gall-stone has passed into the duodenum, the patient cannot be said to be out of danger. It is known that even large gall-stones can be evacuated per anum without giving rise to any trouble or annoyance. In other cases their passage down the intestines is accompanied by severe colics, irregular evacuations meteorism, aud vomiting. In a third set of cases, the gall-stone is arrested at some point in the intestine, and there blocks up its lumen. This occlusion may be permanent and cause death. It is often overcome, sometimes after the most severe symptoms of internal strangulation have appeared, accompanied by stercoraceous vomiting lasting for several days. The passage is sometimes freed suddenly with immediate remission of the severe symptoms ; this happens when the gall-stone obstructs the lower part of the ileum, and passes suddenly into the cæcum, or when it is evacuated after having lodged in the rectum. It is not always upon the size of the stone that the one or the other of these occurrences depends. The shape also is of importance. Thus a long cylindrical stone with a small transverse diameter may pass without causing any trouble, while a much smaller one, if it be

spherical and have a greater diameter, may occlude the intestine. The size of the intestine also, which varies within physiological limits, as well as from accidental causes, has some influence.

It is scarcely possible for a calculus which has passed down through the common bile-duct to cause obstruction of the bowels, this untoward circumstance being usually brought about by a large stone which has ulcerated its way into the duodenum. Abercrombie, however, found a fatal case of lodgment of a gall-stone in the small intestine which had evidently passed down the duct. The common bile-duct in this case was so enlarged that it would admit a finger. The stone measured 4 inches in longitudinal, and 3½ in transverse circumference.

A gall-stone may lodge at any point in the small intestine, but most frequently it does so in the lowest part of the ileum, one or two inches above the ileo-cæcal valve. The cause lies in the diminution which the calibre of the intestine undergoes as it approaches the cæcum, and partly also in the fixation of the lower part of the ileum by a short mesentery.

Sometimes it happens that after a gall-stone has made its way into the lowest part of the ileum, it remains there for a long time obstructing it only incompletely, and gives rise to a set of symptoms composed of those of constriction of the intestine, and repeated temporary occlusions. (Meteorism, colics, visibleness of the hypertrophied folds of the intestine: attacks which resemble hepatic colic, except that they are associated with much meteorism.) Circumscribed peritonitis results from the irritation excited by the gall-stone in the intestinal walls, and produces annular narrowing of the ileum at the corresponding point, which finally reaches such a degree that the engaged gall-stone becomes fixed and causes death with the symptoms of permanent occlusion. Sometimes the stone by remaining in the canal for a long time, pushes out its wall on one side in the form of a false diverticle. Such diverticles have been found in the duodenum as well as in the ileum. Finally, the rarest termination is that of evacuation of a gall-stone through the abdominal wall with formation of an intestinal fistula.

A small stone remaining for a long time in the intestinal canal or in a diverticle, may grow by the superposition of fresh layers by the passing contents of the bowel. In this way are formed stones having a cholestearine nucleus in the centre, and a shell composed of phosphate of ammonia and magnesia and the phosphate of lime, in which, however, may be mingled decomposed bilirubin and a small amount of cholestearne.

A gall-stone that has been lying in a diverticle, may as a result of vigorous peristaltic action, or because it grows more and more into the lumen of the canal, make its way out of it into the intestine, and either occlude the latter or be evacuated *per anum*.

Fatal obstruction by gall stones has been observed only in the small intestines. Still, most severe, though temporary, symptoms of impermiability of the colon have been repeatedly caused by the lodging of one or more stones in the sigmoid flexure or in the rectum. When there is stenosis of the ileum or colon, occlusion can be caused by smaller stones which would otherwise pass freely along the canal.

The symptoms of intestinal obstruction caused by gall-stones vary according to the locality of the same. If this be in the duodenum, vomiting begins at once, and furnishes, until death abundant quantities of bile. Collapse is rapid, the abdomen retracted, the urine scanty or lacking. Death may ensue in from six hours to two days. When the obstruction is situated in the lower part of the ileum, notable meteorism, limited at the beginning rather to the hypo and mesogastrium, occurs, and the vomiting becomes feculent. The secretion of urine is not always diminished. The duration of the disease in those cases varies from four to seven days.

Lodgment of a gall-stone sometimes follows immediately after a severe attack of hepatic colic, in other cases, a long time, three years, for example, in Murchison's case, after such an attack, or after the symptoms of peritonitis in the region of the liver; which latter led to adhesions between the gall-bladder and duodenum, and thus prepared the way for the breaking through of the stone, a process which, however, may remain entirely latent.

The diagnosis of obstruction by gall-stones can, under favourable circumstances, be made with probable accuracy, by its occurring in females of advanced age, with a previous history of hypochondriac pain.

It is a common occurrence for a gall-stone, when impacted in the common bile-duct, to ulcerate its way into the duodenum; adhesive inflammation first having taken place between the duct and that portion of alimentary canal. Fistulous openings, the result of the passage of gall-stones, have also been observed between the gall-bladder and stomach, and, rarely, between the gall-bladder and colon. They have also made an exit for themselves through the abdominal walls; into the pleura, liver, portal vein, urinary passages and vagina.

Diagnosis.—The symptoms which accompany the passage of gall-stones are not always so violent and well-marked as above described. Indeed they have been frequently discharged from the bowels without producing jaundice, and it is easy to imagine that after the ducts have been dilated by the passage of a large stone, numerous smaller stones may pass into the bowels without causing any great inconvenience, and Sir Thomas Watson says there are persons who get rid of scores of stones in this way during their lives. It also frequently occurs that patients who have suffered from repeated attacks of what have been considered as gastralgia or cramp in the stomach, subsequently manifest all the symptoms of hepatic colic, brought about by the escape of a larger stone down the duct. These mild cases may usually be diagnosed by observing that the attack of pain is followed by even the slightest tint of yellow in the conjunctivæ, or by the presence of bile in the urine. If the patient be a female, fat, and advanced in life the diagnosis is still further strengthened.

Intestinal colic sometimes simulates hepatic colic, but may be distinguished from it in the situation of the pain being referred to the naval rather than the epigastrium and right scapula; the absence of jaundice, or bile pigment in the urine, or tenderness over the gall-bladder. The circumstances under which the attack

occurs must be taken into account, such as in connection with an obvious error in diet, or if Burton's blue line be present on the gums, or if there be a history of lead colic or palsy. Renal colic may be distinguished from hepatic by the pain being referred chiefly to one kidney, and from thence shooting down into the groin of the affected side, and into the testicle, which is retracted; the absence of jaundice; tenderness over the kidney and not over the gall-bladder; hæmaturia, with irritability of the bladder. But the most decisive proof of the colic being due to the passage of gall-stones lies in the finding of them in the fæces. They ought always to be searched for after the occurrence of paroxysmal pain in the epigastric region, as it is satisfactory both to the patient and the medical attendant to have proof of what caused the attack, and also their appearance may furnish valuable data on which to ground a prognosis. If one large smooth globular calculus have been passed, it is very possible the patient may not experience any further trouble. But if there be found, one or more stones with several facetts, the probability is there are more yet to pass.

The failure to find a stone after well-marked symptoms of hepatic colic have manifested themselves must not be taken as proof that those symptoms were not caused by the irritation of a gall-stone; because the stone may have been disintegrated in the bowels, or it may have slipped back into the gall-bladder instead of into the duodenum. Gall-stone colic may also be produced by the passage of biliary gravel, or inspissated gritty bile.

The only reliable method of searching for gall-stones in the dejecta is to wash them on a fine hair sieve or piece of muslin. This process ought to be repeated every day for four or five days, or until the stones are found.

Having formed a diagnosis of gall-stone colic, it will be perceived from what is said above that the prognosis must be very guarded.

Treatment.—When a patient is suffering from symptoms of the passage of gall-stones, the indications for treatment are four-

fold. 1st, to relieve the pain; 2nd, to promote the passage of the concretion; 3rd, to dissolve any stones that may be remaining in the gall-bladder, and 4th, to prevent the formation of fresh stones.

The two first indicatio ns are best fulfilled by the inhalation of chloroform or ether. When the patient is fully under the influence of either of these drugs, the spasm of the bile-duct is relaxed, and thus one great obstacle to the passage of the stone is removed. The patient is also oblivious to all pain and suffering, therefore that restlessness which might tend to produce rupture of the gall-bladder is obviated.

But should those drugs be contraindicated, or the symptoms not be sufficiently violent to warrant their administration, great relief may be afforded by a warm bath, or hot fomentations, or a large linseed poultice applied to the abdomen after smearing it over with the extract of belladonna and the extract of opium in equal proportions. Effervescing draughts containing hydrocyanic acid may be administered to relieve vomiting, and ice may be sucked with the same object.

Opium is a sovereign remedy in these conditions and must be employed very boldly. A grain of solid opium or a quarter of a grain of morphia may be given every two hours until the pain subsides. Hypodermic injections are still better because they act more speedily and obviate the risk of the drug being rejected by vomiting.

A quarter of a grain of morphia with $\frac{1}{60}$ grain of aconitine may be injected under the skin of the arm and repeated from time to time according to the requirements of the case. In protracted vomiting, or if the patient be at a distance from his medical attendant, morphia may be administered in the form of a suppository placed in the rectum.

Although opium is well tolerated in these cases, it is well not to continue its administration for a longer period than is absolutely necessary for the relief of pain, because where a large quantity has been administered, after the stone has passed, and the nervous system has regained its normal condition, fatal nacotism may suddenly and unexpectedly supervene.

Large draughts of hot water containing one or two drachms of bicarbonate of soda sometimes give immediate relief; the alkali counteracting the depressing symptoms produced by the acidity of the stomach, while the hot water acts like a fomentation to the seat of pain. The first portions of water are commonly rejected almost immediately; but others may be repeatedly taken, and after sometime it will usually be found that the pain will become less and the water be retained. A few drops of laudanum added to the alkaline solution may sometimes be advantageous. Absorption of the water by the portal system has also a tendency, by increasing the flow of bile, to promote the passage of the calculus.

After the paroxysms of pain are over the administration of saline or mercurial purgatives are usually necessary to obviate the constipating effect produced by opium, and to relieve hyperæmia of the liver. During the attack they are of doubtful benefit in expediting the expulsion of the stone, and may add to the exhaustion of the patient which it should be our object to prevent.

The third and fourth indications for treatment are the solution of any concretions which may still remain in the biliary passages, and preventing the formation of new calculi. It is very probable that no drug administered by the mouth has any power in dissolving gall-stones in the gall-bladder. Out of the body, chloroform and ether will dissolve cholestearine, which is the main constituent of these concretions, but neither can reach the gall-bladder in a sufficiently concentrated form to accomplish this object during life. Still there are certain remedies which enjoy a great reputation for the favourable influence they exert in the removal of gall-stones. Among these is Durande's remedy. It consists of a mixture of three parts of sulphuric ether and two parts of oil of turpentine. Of this 20 or 30 drops may be taken two or three times daily, but the benefit derived from it is extremely problematical.

The only thing we can do in this direction will also fulfil the fourth indication, that is, promote the patient's health in every possible way. Exercise of a gentle character, in the open air must be enjoined. If we have reason to suspect the existence of

more calculi in the gall-bladder, the patient must be warned of the danger of making any sudden violent exertion. Horse exercise, or riding in a carriage over a hard, rough road must be avoided. The digestion must be attended to by excluding everything from the dietary calculated to promote acid dyspepsia and gout. In the way of drugs, the neutral salts of potash, soda, and magnesia, and the chloride of ammonium may be administered with great advantage; or the patient may be sent away to drink the waters of Carlsbad, Vichy, Ems, Kissengen, Homburg, or other alkaline waters. Although these waters may not exercise any power in dissolving gall-stones, other than the mere swallowing of a large quantity of water tends to dilute the bile, and thus lessens the tendency to crystalization in it, it is certain that they also lessen the tendency to acid dyspepsia and gout, reduce congestion of the liver, while the change of scene and society will have a beneficial effect in promoting the general health of the patient.

In addition to those means, we may endeavour to improve the quality of the bile by the administration of small doses of blue pill. It may be given most safely to persons of full habit who have lived freely, and in whom there is no reason to suspect organic disease.

Finally, the patient must be much in the open air, and sleep in an airy bed-room, rise early and take plenty of exercise, abstain as much as possible from fat meats and malt liquors, drink diluents rather freely, and not make the intervals between the meals too long.

After a gall-stone has passed from the duct into the bowel, should symptoms of intestinal obstruction arise, such as obstinate constipation and vomiting; warm baths and hot fomentations must be resorted to, and opium and belladonna in full and repeated doses administered, and copious enemata of warm water and oil thrown up the rectum. Gentle pressure and manipulation of the abdomen may in rare cases dislodge the stone. In these cases the rectum should always be examined by the finger introduced within the anus, because it is possible the stone may be impacted in that situation. But obstruction continuing after these measures

have had a fair trial, no time should be lost in resorting to abdominal section for its relief, because the patient is almost certain to die if left alone. Besides, the results of modern ovariotomy prove that the intestines will bear with impunity, exposure to the air and a considerable amount of manipulation.

If after the stone has passed into the bowels, symptoms of an inflamed gall-bladder supervene, such as fever, tenderness at the lower margin of the right ribs, with a distinct tumor in that situation; rest, opium in full and repeated doses, and a few leeches applied over the gall-bladder, are the remedies to be depended upon.

The external fistulæ left after the exit of gall-stones do not demand any particular treatment other than cleanliness. If there are more stones to come away it would be foolishness to try to close them. If but one was present, which has come safely away, the fistula will most probably heal without any interference.

Internal biliary fistulæ are not amenable to either medical or surgical treatment.

DISEASES OF THE BILIARY PASSAGES.

Entozoa of the Biliary Passages.

FOUR species of Entozoa have been found in the biliary passages of man, namely,

1.—Fasciola Hepatica (Lin.) } Liver flukes.
2.—Distoma Lanceolatum }
3.—Ascarus Lumbricoides.
4.—Echinococcus.

Of these the flukes are the only ones whose regular and permanent habitat is in this situation.

1. Liver Flukes.—Fasciola hepatica, the large liver fluke, is a trematode helminth of an oval form, varying in length from eight to fourteen lines, and from four to eight lines transversely at its broadest part, body very flat, frequently curved towards its ventral surface during life; has two suckers, ventral and oral; sexual organs placed in middle line between the suckers; intestines branched; colour of body, pale brownish-yellow, with a slight rose tint.

Distoma lanceolatum is a small trematode helminth; about a third of an inch in length, and about one line and a half in breadth; lanceolate form; surface smooth; two suckers, ventral and oral; reproductive organs in central line, in front of ventral sucker; intestines bifurcated; ova visible through transparent body, gradually pass from a dark brownish colour in front to a pale yellow colour behind.

Liver flukes, although so frequently found infesting the bile-ducts of sheep and oxen, and often in vast nnmbers, are very rarely met with in the human subject. In the sheep, the incursion of liver flukes produces symptoms of marasmus and anæmia from which whole flocks have been carried off during a single epidemic. In Davaine's elaborate work on Entozoa a record is given of fifteen cases in which liver flukes gained access to the human body. The rarity of its occurrence, however, is sufficiently explained by the circumstance, that man in a civilized condition, can seldom have occasion, either accidentally or otherwise, to swallow the intermediate moluscan host in which the higher larvæ of this parasite probably dwell. Even in the few instances which are reported of liver flukes being found in man, in but a small minority of them have the parasites existed in sufficiently large numbers to produce morbid symptoms during life.

The fasciola hepatica infests by preference the gall-bladder and larger bile-ducts, while the distoma lanceolatum infests the smaller ducts. Both species may be found in the same host.

These parasites in the sheep cause dilatation and catarrh of the biliary passages, with atrophy of the hepatic parenchyma and profound marasmus and anæmia, but only in rare cases jaundice.

The diagnosis of liver flukes in the human subject can only be arrived at in the event of any of the parasites being ejected by vomiting or in the stools.

2. Ascarus Lumbricoides.—Round worms, whose usual habitat is the small intestines, are occasionally found in the biliary passages, whither they have migrated by reason of their well-known proclivity for forcing themselves through narrow openings. As a rule, only a single round worm is found, it may be, in the common bile-duct or coiled up in the gall-bladder, or in the hepatic duct. A few instances are, however, on record in which large numbers of these worms had been found in these situations, having given rise to obstruction to the flow of bile, dilatation, or catarrhal, exudative or ulcerative inflammation of the ducts, or even to abscesses in the liver. In very rare cases they die and shrivel up in the gall-bladder and then form the nuclei of gall-stones.

The chief symptom produced by round worms in the biliary passages is jaundice, but this condition can only be assumed to arise from this cause if there be a history of lumbrici having been passed per anum or ejected by vomiting.

3. ECHINOCOCUS.—Hydatid vesicles are very rarely found in the biliary passages, and then only in consequence of an accidental communication having been formed between the mother cyst and the lumen of the bile-ducts. When such a communication has been established, the vesicles can pass out of the cyst into the lumen of these canals, dilate and occlude them, and give rise to inflammation and ulceration of their mucous membrane; or the vesicles may gradually pass down the ducts and finally reach the intestine and be voided per anum. The passage of hydatid vesicles through the bile-ducts produces symptoms of hepatic colic which in some cases can only be distinguished from gall-stone colic by detecting hydatid vesicles in the alvine evacuations. It very rarely happens that complete discharge and cure of the hydatid cyst takes place in this manner; usually the common bile-duct becomes permanently occluded by the vesicles and fatal jaundice developes, or suppuration of the sac, or inflammation and ulceration of the ducts supervene and cause death by pyæmia.

Since the above was written a discovery fraught with interest to the naturalist, and of the greatest importance to agriculturalists, has been made by Mr. A. P. Thomas, M.A., of Oxford, who after the most careful and elaborate researches ascertained that the intermediary host of the common liver fluke is a small amphibious snail called *Limnæus trunculatus*, which is very abundant in meadows subject to periodic floodings.

DISEASES OF THE PORTAL VEIN.

Thrombosis and Occlusion.

PYLETHROMBOSIS.

THE majority of blood coagula (thrombi) found in the portal vein occur independently of any inflammation of the venous wall, and the wall of the vessel frequently becomes inflamed secondarily; but inflammation of the vein constitutes the starting point of the morbid process in a comparatively small number of cases.

Coagula of blood are developed in the portal vein (pylethrombosis, from πύλη—porta—*a gate*, and Θρομβος—a *clot of blood*) as in other parts of the venous system, (1) in consequence of weakened force of the circulation, produced by diminished action of the heart, or by marasmus—*marasmatic thrombosis*. This form of thrombosis is of very rare occurrence in the portal vein, as compared with other veins, and usually occurs only a few days before death as a result of thrombi in the pulmonary artery which completely arrest the venous circulation, or it may develop during the long-continued final agony which precedes death.

(2.) The most frequent cause of pylethrombosis is *compression* exerted on the numerous capillaries of the portal vein within the liver, by those diseases which induce destruction of numerous capillaries, or constriction of the branches of the portal vein, or compression of the trunk of the vessel below the liver by contractile tissue and by tumors of various sorts; (*compression thrombosis*).

The diseases which produce compression thrombosis are of a chronic character, and therefore the latter is developed more slowly. The thrombus has time to undergo various changes, in which the walls of the vein participate, while the sequelæ of occlusion of the portal vein are so prominently developed, as to enable us to diagnose the morbid process during life.

The most common causes of this chronic form of pylethrombosis are simple and granular induration, cirrhosis of the liver and likewise chronic atrophy of the liver, which in their later stages lead to obstruction of the portal vein. Below the liver, pylethrombosis may be produced by contractile connective tissue the result of chronic peritonitis, perforating ulcers of the duodenum, and much more frequently by new growths which start from the neighboring organs, especially cancer of the stomach, pancreas, and duodenum, and enlarged glands in the hepatico-duodenal ligament, which compress the portal vein.

Symptoms of Obstruction of the Portal Vein.—The process by which obstruction of the portal vein is brought about does not make itself manifest by any local symptoms, and it is only in exceptional cases that the thrombus produces such irritation of the walls of the vein as to develop pain.

After a long continuance of the symptoms of those diseases which lead to thrombosis of the portal vein, the symptoms which are the result of stasis of blood in the radicles of the portal vein suddenly make their appearance; these are, ascites which develops with great rapidity, the superficial veins of the abdominal parieties enlarge and extend in the form of thick cords from the abdomen, over the lower part of the thorax towards the axillæ; the spleen rapidly enlarges, diarrhæa of a watery or bloody character, not infrequently accompanied by vomiting, sets in; the urine is scanty and dense, the strength rapidly declines, and the patient presents a pale cachectic appearance; the feet become œdematous (later than the ascites), and continue so until death.

Analysis of the Symptoms.—These symptoms are not always developed with equal severity; they are more or less strongly

marked according as the obstruction of the portal vein is complete or incomplete, or takes place suddenly or slowly, and according as the obstruction is compensated for or not by the collateral channels of circulation.

Ascites is a very constant symptom of portal obstruction, and is only absent in those exceptional cases in which relief has been afforded to the stasis by copious hæmorrhages from the stomach and intestines, or by the prompt and efficient development of the collateral circulation. The same remarks apply with reference to enlargement of the spleen.

Enlargement of the superficial abdominal veins is not a constant symptom but is observed in about one-half of the cases, and in these the inferior vena cava is probably occluded as well as the vena portæ.

Diarrhœa is the most constant symptom referrible to the intestinal functions, and is analagous to the ascites in its mode of development; both are the result of increased transudation of serum from the stagnant blood of the capillaries.

Diarrhœa is exceptionally absent when an efficient collateral circulation is established. In one-third of the cases the evacuations are either mixed with blood, or consist of pure blood.

Hæmatemisis is of rare occurrence. Occasionally it may be the first and almost the only symptom of pylethrombosis, in consequence of a profuse hæmorrhage suddenly cutting short the patient's life.

The liver is almost always diminished in size, the exceptions being those cases in which cancer of the liver has preceded the portal obstruction.

Jaundice is a rare symptom in these cases.

Diagnosis.—The most reliable indication of obstruction of the portal vein is the extraordinarily rapid return of the ascites after the fluid has been evacuated by tapping; this does not take place with the same rapidity when the dropsy results from any other cause.

PROGRESS AND PROGNOSIS.—The termination of pylethrombosis is, perhaps without exception, fatal. In the majority of cases the duration is short; from a few days to several weeks. But cases are known in which life has been prolonged for months, and some even in which the compensatory collateral circulation was so complete that years have passed before a fatal termination took place. The quantity of fluid poured out in these chronic cases is sometimes enormous. I myself drew off eighty-one (measured) pints of fluid by a single puncture from a woman who had well-marked symptoms of portal obstruction, the disease having lasted in her case for a great many years. I had not an opportunity of verifying the diagnosis by *post-mortem* examination in consequence of my leaving the neighbourhood in a few months after operating on her. Such cases are very rare.

From what has preceded it will be gathered that the prognosis is very unfavourable.

TREATMENT.—The object of our treatment in these cases must be to relieve the symptoms which are due to the stasis of blood as much as possible. Diarrhœa and hæmorrhages may be moderated by astringents such as tannic acid, rhatany, sesquichloride of iron, subcutaneous injections of ergotine, &c. The ascites can only be temporarily relieved by puncture, which, however, ought to be postponed as long as possible, and not be repeated until dyspnæa becomes dangerous, because the removal of the fluid promotes wasting of the body. The use of drastic purgatives for the removal of the ascites would only promote the exhaustion. It is useless to have recourse to diuretics, because it is impossible to excite the functions of the kidneys, owing to the diminished pressure in the arterial system. Every thing calculated to irritate the gastric and intestinal mucous membrane must be avoided. Finally, the administration of concentrated and easily digested nutriment, good strong wine, and preparations of iron and quinine offer the best hopes of benefiting the patient.

DISEASES OF THE PORTAL VEIN.

Purulent Inflammation of the Portal Vein.

PYLEPHLEBITIS SUPPURATIVA.

PURULENT inflammation of the portal vein occurs at every period of life from birth to old age. It is almost invariably a consecutive lesion resulting from suppurative processes in the various abdominal organs. Cases are rare in which the disease is produced by an external wound.

The pathological changes in suppurative pylephlebitis pursue the following course. Ulcerative inflammation is set up in some portion of the alimentary canal, most frequently in the vermiform appendix and cæcum by the retention of hard fæcal matter, or of foreign bodies, such as a splinter of bone, &c.; the inflammatory products are carried into the circulation by the ilio-cæcal vein and phlebitis is thus developed, which remains circumscribed or is propagated to the trunk of the portal vein, producing a condition of purulent infiltration of its walls; coagulation of blood takes place in the vein; the thrombus thus produced undergoes purulent softening, the lumen of the vessel appears filled with pus or an ichorous fluid; the sheath of the vessel and its middle coat present a condition of increased vascularity or of infiltration with serous, fibrinous, or purulent matter; the inner coat presents a discolored, torn appearance, and is covered with fibrinous layers or fluid pus. These alterations in the vein are most distinctly developed in the locality where the disease commenced, but spread

from thence into the hepatic branches of the portal vein, the channels of which become blocked up by extension of the thrombosis; puriform softening of the thrombus takes place throughout the whole extent of the vessel, and ultimately, purulent infiltration and ulcerative destruction of the walls of the portal vessels as far as their finest ramifications. Sometimes particles of thrombi are broken off and carried by the current of blood into the liver where they cause metastatic deposits; these embolic deposits are found in various stages of development, from reddish-brown infarctions to cavities filled with yellow pus.

Suppurative pylephlebitis almost always exists as a secondary lesion resulting from suppurative or ulcerative processes in the organs in which the roots of the portal vein originate.

Traumatic or primary pylephlebitis as the result of a direct lesion to the walls of the portal vein itself has never been known to occur; but in one of the following cases a wound of the superior mesenteric vein, and in the other a wound in the immediate neighbourhood of the same vessel produced suppurative phlebitis of that vein which extended to the vena portæ.

The first case was reported by M. Lambron in the *Archives Générales de Médicine*, in 1842. In this a fish-bone 1.18 inch long, and as thick as a large pin, had perforated the stomach close to the pylorus, passed through the head of the pancreas, penetrated into the trunk of the superior mesenteric vein, and, by entering into the channel of this vessel, purulent inflammation was set up which extended to the portal vein and its branches.

Another somewhat similar case was reported in 1866, by Von Jan, of Erlangen. In this case a piece of stout wire was found to have penetrated the posterior wall of the duodenum near the pylorus. One end of the wire lay in the cavity of the duodenum, and the other end, which was sharp, had passed through the root of the mesentery close to the superior mesenteric vein, which was found as a firm cord filled with a loose thrombus. This condition had evidently given rise to traumatic suppurative pylephlebitis.

A not infrequent cause of traumatic suppurative pylephlebitis is the extension of inflammation from the ligatured umbilical vein

to the trunk and right branch of the portal vein in newly-born children. But in some of these cases the traumatic origin of the disease is not so probable as its causation in septic infection.

Symptoms.—The symptoms of the morbid processes which induce suppurative pylephlebitis, such as perityphlitis, ulceration of the stomach or intestines, inflammation of the spleen, chronic peritonitis, gall-stones, &c., usually precede the symptoms of suppurative pylephlebitis. After these have lasted for a longer or shorter period the supervention of inflammation in the portal vein is announced by pain, which is increased on deep pressure with the tips of the fingers in the epigastrium over the trunk of the portal vein, in the right hypochondrium when the branches are diseased, or in the cæcum left hypochondrium or umbilical region when the roots are first affected. Soon afterwards the patient has a rigor, followed by heat and profuse sweating; this series of symptoms occurs at irregular intervals, the temperature remaining abnormally high in the intermissions. The liver and spleen become enlarged and tender upon pressure. Jaundice is present in about three-fourths of the cases. Diarrhœa of a bilious character is usually present, but constipation has been observed in a few cases. Subsequently the symptoms of diffuse peritonitis,—painful distension of the abdomen, vomiting &c., usually supervene. The patient rapidly looses flesh and strength, the fever assumes a hectic character, and ultimately delirium or somnolence is developed and terminates in death. In rare cases death is preceded by the symptoms of metastatic deposits of pus in the lungs, the joints &c., as a rule, however, the pyæmia does not extend beyond the territory of the portal vein.

Diagnosis.—When all the symptoms of suppurative pylephlebitis which have been enumerated are completely developed and carefully noted as they arise, the difficulty of diagnosis will not be very great. On the other hand, if the essential symptoms are absent or not well marked, or if the patient does not come under observation until the disease has arrived at an advanced stage, the difficulties of diagnosis will be greatly increased, and at the most

all we can do is to ascribe the symptons present as probably due to some disease of the portal vein. There are no definite symptoms by which we can recognize the disease under all circumstances; the concurrence of a definite series of symptoms, and the manner in which they are developed, constitute our only guide. The following are the most important factors upon which the diagnosis should be based. (1.)—The presence of a disease which we know may act as an excitant of pylephlebitis, especially perityphlitis, purulent or ulcerative disease in the stomach, intestines, spleen, biliary passages, or the umbilicus of new-born children.

(2.)—Pain in the epigastrium above the umbilicus, or in the right hypochondrium, or in any of the other localities from which pylephlebitis has been known to start.

(3.)—Attacks of rigors, recurring at irregular intervals, and followed by profuse sweats.

(4.)—Painful enlargement of the liver and spleen, accompanied by jaundice, bilious diarrhœa, and rapid emaciation: and finally

(5.)—Typhoid symptoms of blood poisoning and the symptoms of general peritonitis.

The following are the diseases which may be confounded with suppurative pylephlebitis.

(a.)—Hepatic Abscesses, in common with pylephlebitis present enlargement and tenderness of the liver, rigors and fever, but the causes of both are entirely different; in hepatic abscess, pain is limited to the liver, pyæmic infection rarely ensues, diarrhœa, rapid emaciation, and extreme prostration are wanting.

(b)—The same remarks which have been made with regard to hepatic abscesses, apply with equal force to *Occlusion of the Bile-ducts* by means of calculi.

(c)—Intermittent Fever may for a time be mistaken for pylephlebitis, on account of the rigors followed by fever and sweating, enlargement of the spleen, and occasionally enlargement of the liver. But the irregular intervals between the rigors, jaun-

dice if it be present, the incompleteness of the remissions, rapid loss of strength, and lastly the inutility of quinine in controling the phenomena of the disease prevent a long continuance of the confusion.

I recently had under observation a case of suppurative pylephlebitis which when first seen could with difficulty be diagnosed from intermittent fever in consequence of the rigors, chills and sweats occurring every other day with the regularity of ague : but the presence of jaundice, the incompleteness of the remissions between the rigors &c. which latter soon became more frequent and irregular in their advent, notwithstanding the administration of quinine, rapid emaciation and loss of strength served to point to the true nature of the disease.

Prognosis.—A diagnosis having been formed of suppurative pylephlebitis, the prognosis is absolutely unfavourable. The disease always terminates in death. The septic infection of the blood and the extreme exhaustion as the result of fever &c., must be regarded as the chief cause of the fatal termination. Life may be prolonged for about six weeks, or death may take place in a few days after the disease sets in.

Treatment.—In the present state of therapeutics we are completely powerless in controling the progress of suppurative pylephlebitis. The most we can do is relieve the troublesome symptoms. Local or general blood-letting will only exhaust the patient's strength. As much may be said with regard to mercury in any form. Ice may be sucked to relieve the dry, parched tongue, the strength may be sustained by mild nutritious diet, such as beef-tea, wine, &c. There is no objection to the administration of quinine in the hope of controling the fever : diarrhœa and pain may be relieved by opium. Beyond this we cannot go.

HYPERÆMIA OF THE LIVER.

THE portal system presents some points of peculiarity which render it very liable to suffer from variations in blood-pressure, both within and without the liver.

Interposed between the veinous trunks of the abdominal viscera and the right side of the heart for the purpose of submitting the newly absorbed materials of the food to certain assimmilative processes within the liver, the portal system begins and ends in capillaries without the interposition of an impelling organ. The *vis a tergo* of the heart, which is quite sufficient to propel the blood through one set of capillaries into the veins, is almost expended in overcoming the resistence of a second set of capillaries in the parenchyma of the liver. In order that the blood may return to the heart some new force must therefore be brought to bear upon it. This force is found in the *inspiratory movement.* During the act of inspiration a vacuum is created in the thorax which causes the blood in the open hepatic veins to be drawn into the inferior cava within the chest, in order to fill up the vacuum.

There are also other circumstances which influence the circulation of blood in the liver. Thus, during digestion, the active flow of blood in the stomach and intestines, and the large quantities of fluid absorbed, cause the blood in the portal vein to flow more actively than at other times. It seems probable also that the *vis a tergo* in the blood of the hepatic artery is not destitute of influence in assisting the onward flow of the portal blood.

Notwithstanding these aids to the circulation, the flow of blood in the portal system is very languid, and stasis is very liable to occur in it through any disturbing influence. The absence of

valves in all the veins, both roots and branches, of the portal system, not only act conservatively by allowing an unusually free passage of blood from one part to another, during the very varying conditions to which it is subjected, but also favour morbid hyperæmia in that system.

The amount of blood in the liver, in a state of health, varies greatly at different times. Thus, it is greatest after a full meal, and is reduced in quantity during fasting. But in morbid hyperæmia of the liver the alternations of health do not take place; the amount of blood in the organ is permanently increased, which causes it to swell and become sensitive to pressure.

Hyperæmia or Congestion of the liver is divided into two forms, viz., Passive or obstructive hyperæmia, in which there exists some impediment to the efflux of blood; and Active hyperæmia, that state of the liver in which the afflux is abnormally increased. Many cases, however, are met with in practice in which both these conditions are simultaneously in operation.

Etiology.—Any circumstance which interferes with the outflow of blood from the hepatic veins into the vena cava, has a tendency to produce stasis of blood in the liver, but the most frequent causes of hepatic hyperæmia are mitral stenosis and mitral insufficiency. Incompetence of the tricuspid valve, when it occurs, is still more certain to produce this disease. There are also certain diseases of the lungs and chest wall which, by impeding the circulation of blood through the pulmonary artery, induce functional incompetence of the tricuspid valve, and consequent increased lateral pressure in the inferior vena cava, which in turn is propagated in the hepatic veins, and from thence to the portal system. These diseases are,—pulmonary emphysema, pneumonia, congenital atelectasis, extensive atrophy of the lungs, deformity of the chest walls and angular curvature of the spine, causing long-continued compression of a large portion of the lungs, pleuritic exudations, large intrathoracic tumors &c.

The habit of eating too much or too frequently, particularly when combined with a sedentary mode of life, is very liable to produce

hyperæmia of the liver, because the afflux of blood which normally takes place after a full meal has scarcely had time to subside before the organ is again replete with blood charged with newly absorbed material. The hyperæmia thus becomes permanent, and sedentary habits, which are so inimical to free respiration, further promote the morbid condition by abolishing the suction force of deep inspiration, which is such an important agent in drawing away the blood from the hepatic veins.

There are *certain articles of food* which, by reason of their acrid properties, are said to produce hyperæmia of the liver, when taken in quantity at least. These are, irritative spices, alcohol, and coffee. After partaking freely of any of these articles, a feeling of tightness, fulness and pain in the right hypochondrium, and a distinct enlargement of the gland, manifest their action on the liver, which, however, ceases after a short time; and it is only from the frequent recurrence of the irritation that any serious disease follows.

MALARIAL INFLUENCES, both in the tropical and temperate zones, frequently lead to hyperæmia of the liver, and tropical dysentery is frequently associated in its early stages with it. Tropical abscess of the liver is always preceded by hyperæmia.

SUPPRESSION OF HABITUAL HÆMORRHAGES.—At the "change of life" when the menses begin to recur at irregular intervals, hyperæmia of the liver is very liable to precede each menstrual epoch, and to again subside when the uterine flow has taken place. In this way hepatic tumefaction may return repeatedly until the final cessation of the menses. The same condition is also liable to occur in younger females in consequence of the sudden suppression of the menses, as from cold &c. The sudden cessation of hæmorrhoidal discharges of blood, as a result of successful operations for the cure of piles, after the system has become habituated to the periodical loss, is frequently followed by hyperæmia of the liver, in consequence of the disturbance wrought in the blood pressure in the portal system, by the interruption of the discharge from the rectum.

PATHOLOGY.—The more transient forms of hyperæmia, viz., those produced by sedentary habits, suppressed discharges, irritating food &c., reveal little or nothing on *post-mortem* examination. But when the hyperæmia has been due to obstruction to the outflow of blood in the hepatic veins, the liver is found increased in volume and firmness. On section, the organ presents a mottled appearance exactly similar to that of the cut surface of a nutmeg; hence the condition of the liver observed in cases of fatal mitral obstruction is called "nutmeg liver." This nutmeg-like appearance, which is also visible through the capsule, and which varies in character according to the degree of the congestion, is produced in the following manner. The obstacle to the efflux of blood in the hepatic veins causes those vessels to become distended as far as their ultimate capillaries in the centre of the lobules, imparting to that portion of the lobules a more or less dark appearance. The periphery of the lobules, which is occupied by the ramifications of the portal capillaries, presents a pale brown colour which contrasts strongly with the darker central parts. The paleness of the periphery of the lobules is due to anæmia of the portal capillaries and the presence of fat in the hepatic cells.

When hyperæmia has been very prolonged and intense, a peculiar atrophy of the liver is gradually developed. The gland begins to diminish in size, the intralobular roots of the hepatic veins become dilated under the powerful pressure of the obstructed blood, and thus lead to the disappearance of the hepatic cells enclosed in their meshes, whilst the cells in the periphery of the lobules remain unaffected. Under these circumstances the liver is ound to be small, its surface uneven from the shrinking of parts, its capsule rather opaque, and on section the nutmeg appearance is found to have almost entirely gone, nothing being left but dilated blood-vessels.

SYMPTOMS.—Inasmuch as the obstruction to the flow of blood in the hepatic veins necessarily interferes with the circulation o blood throughout the entire capillary system of the liver, and through this, re-acts upon the primary roots of the portal vein, it

is to be expected that symptoms due to gastric and intestinal disturbance occupy a prominent position in obstructive hyperæmia of the liver. The same train of symptoms is also frequently associated with the hepatic hyperæmia of gourmands and topers, in consequence of persistent irritation of the mucous membrane of the stomach and bowels.

The symptoms due to disturbance of the digestive system are, nausea with loss of appetite, headache, and disinclination for exertion, furred tongue, a bitter taste in the mouth, frequent flushings in the face, vomiting, flatulence, and constipation. Occasionally a transient diarrhœa makes its appearance. The vomiting in connection with hepatic affections usually takes place in the morning. The urine is generally scanty and highly coloured, and almost always contains small quantities of albumen, and sometimes also of bile pigment.

The symptoms due to engorgement of the liver are, a sense of fulness and tension in the right hypochondrium, which is aggravated by strong movements of respiration and by external pressure, and pain may be experienced in the right shoulder, in consequence of the connection of the subclavius nerve with the phrenic.

The liver is generally found to be uniformly enlarged so that its anterior margin may project from an inch to a hand's breadth or more below the margin of the ribs in the right papillary line. If the portion of liver so projecting can be made out by palpation, it is found to be smooth and elastic, not nodulated. The hyperæmic tumor of the liver is characterized by the fact that in can increase and subsequently diminish in size more rapidly than is the case in enlargement of the gland induced by any other cause. Thus in affections of the heart and lungs there may frequently be seen, during a violent attack of dyspnœa, a remarkable increase in the volume of the liver, and on the other hand after a copious loss of blood, through venesection or hæmorrhoidal discharge of blood, a perceptible diminution in the size of the organ may take place.

After hyperæmia of the liver has lasted for a few days, sallowness of the complexion is observed, which after a time may pass

into true jaundice. The jaundice, however, is rarely intense, bile being seldom entirely absent from the motions. In these cases jaundice is produced by the compression exerted on the smaller bile-ducts, by the distended blood-vessels. The hepatic cells remain active, but the bile secreted by them cannot find an exit, and is therefore resorbed into the blood.

Ascites very frequently accompanies the severe varieties of obstructive hyperæmia, when the obstruction to the circulation has given rise to atrophy of the liver.

Treatment.—The treatment must be directed to the causative agencies which produce the hepatic affection, If congestion of the liver arise from the immoderate consumption of food or alcohol, associated with indolent habits, a suitable change in the diet, together with active exercise in the open air are indicated. The diet should be reduced to the quantity appropriate to the individual case, taking care to avoid such articles as are difficult of digestion or prone to produce flatulence; and all fermented drinks, strong coffee, and foods highly seasoned or rich in fat must be interdicted. The diet must consist of lean meat, eggs, white bread, tender vegetables, and acid fruits, taken in moderation, while water, or simple acid beverages may be partaken of freely. Regular exercise in the open air, such as walking, riding, &c., ought to be taken daily, and the patient should be directed to frequently draw voluntarily a long inspiration, in order to accelerate the hepatic circulation.

When the hepatic hyperæmia is dependent upon suppression of the menses, we must endeavour to re-establish the uterine functions by warm baths, the uterine douche, or leeches to the vaginal portion of the uterus, and such emenegogues as may be appropriate to the case under observation. If suppressed hæmorrhoidal discharges of blood have caused the hypermæmia, leeches appled around the anus, may afford relief.

The treatment of obstructive hyperæmia can, in most cases, only be palliative, and must, in the earlier stages, be directed to the relief of the primary disease. If this be capillary bronchitis,

the appropriate treatment for that disease will relieve the hyperæmia of the liver. If valvular disease of the heart or emphysema constitute the primary disease, improvement most frequently follows the timely exhibition of digitalis.

With a view to reduce the quantity of blood in the liver, recourse must be had to purgatives, especially such as produce copious watery evacuations from the bowels, and thus cause a drain from the portal system of veins. If the patient be robust we may safely administer any of the following salts or purgative waters, viz., sulphate of soda or magnesia, tartrate of soda and potash, tartrate of potash ; or such mineral waters as Hunyadi Janos, Frederickshalle, or Pülna water. The salts must be dissolved in warm water, and the mineral waters, at least as met with in this country, must be diluted with warm water, and taken in the morning on an empty stomach, in order to avoid washing away any partially digested food remaining in the alimentary canal.

In the case of a debilitated patient suffering from obstructive hyperæmia of the liver, the milder vegetable purgatives are to be preferred as they admit of long-continued use, but even under such circumstances salines and purgative mineral waters are by no means contraindicated if used with judgment. A mild course of blue pill or podophyllin at bed time will often materially assist the saline course by promoting the secretion of bile.

If there be tenderness in the region of the liver, local depletion in the form of a few leeches or cupping will relieve it ; or what is better still, a few leeches may be applied around the anus, followed by linseed poultices over the liver.

After purgation, if any symptoms of gastric catarrh be present, an alkaline course must be prescribed, and here the alkaline mineral waters of Vals, Vichy, and Ems, or the artificial effervescing Vichy salts, may often be advantageously substituted for the alkaline preparations of the pharmacopœia.

Chloride of ammonium in doses of gr. xx twice or three times a day is of great utility in the treatment of hyperæmia of the liver. It increases the secretions of both the skin and the kidneys, re-

lieves portal hyperæmia, and hepatic pain, and is said to promote the absorption of hepatic abscesses. It may be administered in combination with either acids or alkaline remedies.

In the final stage of severe and protracted hyperæmia of the liver, resulting from valvular disease of the heart, when atrophy of the liver has occurred, ascites may present itself, and give rise to such urgent dyspnœa as to threaten death by suffocation. In such a case life may be prolonged by the operation of paracentesis.

HÆMORRHAGE.

HÆMORRHAGIC deposits are occasionally found in the tissues of the liver. These are most frequently due to disease of the blood itself, as in purpura and scurvy, in which there is a tendency to hæmorrhage from all the parts. They may also arise from excess of pressure in the blood-vessels in cases of obstructive hyperæmia, and from the degenerative process going on in the centre of cancerous deposits. Traumatic causes, blows, concussion, &c., may produce them.

The existence of hepatic hæmorrhage may be inferred when, after violence has been inflicted over it, there arises a sudden increase in the volume of the liver, attended with pain ; symptoms of anæmia increasing in a degree proportionate with the enlargement of the tumor.

The treatment consists in applying cold to the part, and administering opium to relieve pain.

WOUNDS OF THE LIVER.

SOLUTIONS of continuity in the substance of the liver, the result of external violence, although fraught with great danger to life, are by no means invariably fatal. The dangers to life arise, in the first instance, from shock to the system and from hæmorrhage; if the patient survive he is still in danger from the supervention of traumatic peritonitis, septicæmia, and pulmonary embolism. The gravity of wounds of the liver depends in a great measure upon the fact whether the portal vein, or the vena cava be divided or not, and also upon the extent of the bleeding surface, because a large number of small vessels will pour out as much blood as one or two large vessels.

Injuries of the liver may be produced in various ways:—1st, by gunshot wounds traversing the right hypochondrium; 2nd, by sword thrusts. or punctures of other sharp instruments, such as a stick, or a fractured rib; 3rd, by severe blows or falls, or the passage of a heavy body over the abdomen or thorax; and 4th, from violent muscular exertion.

Thierfelder cites a case in which a bullet penetrated transversely both hypochondria; bile was discharged for a long while from the opening on the right side, and complete recovery ensued without the supervention of any inflammatory symptoms in the liver.

When a bullet becomes lodged in the substance of the liver a cyst may form about it and so render its presence innocuous.

Punctured wounds of the liver are almost invariably followed by hæmorrhage into the right pleura, because the diaphragm is usually also punctured; and if a spicula of bone, or other foreign body become lodged in the substance of the gland, abscess is almost certain to supervene.

The liver is very frequently ruptured by a severe blow or fall upon the abdomen, a fall from a height upon the feet, or by a heavy weight passing over the abdomen or thorax. The portion of the gland most liable to rupture is the convex surface and anterior margin. The rupture seldom extends deeply through the substance of the gland, the injury usually being limited to fissures or cracks of about one or two inches in depth, which generally take a direction from before backwards, with a slight obliquity. It rarely happens that the liver is intersected transversely. The right lobe, on account of its size, is more frequently affected than the left. The lacerated edges of the wound do not separate much, and the surfaces present a granular appearance. But little blood is met with in the laceration; it is commonly found effused in the lower part of the cavity of the peritoneum, or in the hollow of the pelvis.

If the wound be slight and uncomplicated, and no great hæmorrhage occur, recovery may take place; but if it extend far back and involve the vena cava or the portalve in, the hæmorrhage is sufficient to cause the instant destruction of life. Under other circumstances a person may survive some hours, as the blood may escape only slowly, or it may be suddenly effused in fatal quantity after some hours or days, as a result of violent exertion or fresh violence applied to the abdomen.

Even should the patient escape the primary dangers of shock and hæmorrage his life is still in jeopardy from the supervention of diffuse peritonitis, septicæmia, and, if the rupture be situated far back in the neighbourhood of the hepatic veins, thrombosis of those veins may occur, and lead to embolism of some of the branches of the pulmonary artery.

Rupture of the liver has been known to occur from violent muscular exertion, such as endeavouring to avoid a fall from a horse, or as the result of violent vomiting; but considering that these severe injuries to the liver may be produced by a blow, &c., without any sign whatever of external violence being found, it is very important in a medico-legal point of view that we should

exhaust every other possible means of their production before attributing such injuries to this very rare cause.

Symptoms.—Cases of rupture of the liver are occasionally met with in which no symptoms whatever are observed, indicative of the nature of the injury, which latter is only disclosed by *post-mortem* examination; or, should recovery take place, such cases would be classed as traumatic peritonitis. Usually, however, severe pain over the hypochondriac region is complained of soon after the receipt of injury; followed by dulness on percussion in the flanks, as a result of extravasated blood, and great collapse. If the patient survive, diffuse peritonitis may be developed, with bilious vomiting, clay-coloured stools and jaundice. If the laceration be extensive, death soon takes place from shock and hæmorrhage; coldness and general pallor of the whole body, with failing pulse, sighing respiration, and restlessness preceding the fatal issue.

Treatment.—In common with those of all the abdominal viscera, wounds of the liver require to be treated with extreme caution, because there is always an element of doubt as to their extent and nature. Absolute rest in the recumbent posture must be secured and maintained. If pain be present, fomentations and poultices must be resorted to; and perhaps a few leeches also. Opium is the sheet anchor in all these cases. A full dose of it should be administered at first, either hypodermically or by the mouth, and its effects maintained by small and repeated doses. This drug allays pain, assists materially in maintaining rest to the injured peritoneum and viscera, and tends to arrest inflammatory action. On no account ought a purgative to be administered, as it would only tend to upset nature's process in repairing the injury. The state of the blader should be attended to, as retention of urine from disturbed nerve influence is frequently observed in these cases.

PERIHEPATITIS.

BY PERIHEPATITIS is signified inflammation of the serous capsule which surrounds the liver, and also of Glisson's capsule in the transverse fissure. The latter variety has already been more appropriately considered in connection with Diseases of the Portal Vein and Biliary Passages.

ETIOLOGY.—Perihepatitis is frequently met with as a part of general peritonitis, under which circumstance its results to the parechymatous tissue of the liver are not important.

More frequently it is induced by diseases of the substance of the liver itself. It is constantly associated with cirrhosis, under which circumstance the organ is frequently found surrounded by a capsule of adventitious tissue which glues it to the surrounding organs. This false membrane is one of the surest *post-mortem* evidences of the habit of spirit drinking. The disease is also developed to a limited extent at the point where abscesses and syphilitic gummata push forward, but is far less frequently observed in connection with cancerous and hydatid tumours, which often attain a remarkable size without producing any inflammation or thickening of the capsule. The portion of the liver which is habitually compressed by tight lacing always exhibits signs of chronic inflammation in the capsule.

PATHOLOGY.—When the serous investment of the liver participates in general peritonitis, the anatomical changes which it undergoes do not differ essentially from those exhibited by the peritoneum. If the inflammation arise from local causes it usually results in the formation of a coat of adventitious tissue which may lead to adhesions between the liver and neighbouring organs, such as the stomach, anterior abdominal wall, or colon;

on the other hand it may merely produce local or diffused thickening of the capsule. The parenchyma of the liver usually undergoes no perceptible alteration in consequence of inflammatory processes in its capsule; at the most, under the pressure of moderate exudations a superficial anæmia may be produced. But when a very thick layer of adventitious tissue surrounds the liver, it exercises an immense compression upon the gland by reason of its contraction; rounding its edges in a remarkable manner, and flattening the gall-bladder, so that the entire organ may approximate the figure of a globe. When such a membrane is found upon the liver a similar one is usually found upon the spleen.

SYMPTOMS.—The chief symptom by which perihepatitis can be recognised is *pain* in the hepatic region, which, if the inflammation be sufficiently extensive, presents the following characteristics; it is aggravated by pressure, by lying upon the right side, by motions of the body, and above all by the movements of respiration. The lancinating character of the pain will serve to distinguish it from pain arising from affections of the parenchyma which is usually of a dull and pressing character.

In addition to the pain there is usually some fever present, and perhaps slight jaundice, and in exceptional cases a friction sound.

When perihepatitis has given rise to extensive firm adhesions between the liver and the anterior abdominal walls, then the downward displacement of its anterior border which accompanies deep inspiration ceases. In those cases in which the organ is subjected to great compression by the retraction of a thick coat of adventitious tissue, the same symptoms appear as in the second stage of cirrhosis, and the affection can scarcely be distinguished during life from the latter disease.

The TREATMENT of acute perihepatitis consists in rest in the horizontal posture, local abstraction of blood, warm poultices, calomel, and neutral salts.

HEPATITIS.

Suppurative Hepatitis.

ABSCESSES in the liver may be divided into (1) *Traumatic*, (2) *Secondary*, *Metastatic* or *Pyæmic*, and (3) *Idiopathic* or *Tropical* abscess.

Traumatic Abscess is occasionally met with as the result of a blow over the right side; a penetrating wound of the liver, as by a spicula of bone from a fractured rib; or a severe concussion produced by a fall from a great height without striking the region of the liver. This variety of abscess, however, is of very rare occurrence, as the liver appears to be more tolerant of mechanical injuries than might be expected of such a highly vascular organ.

Pyæmic Abscesses.—Purulent deposits in the liver, the result of general pyæmia, are of very common occurrence in this country. They usually occur as numerous small collections of pus scattered about the organ close to its surface. The chief cause of multiple hepatic abscesses is suppurative inflammation somewhere in the domain of the portal vein, such as in the biliary passages, stomach, or bowels. These abscesses are essentially embolic in character, and are said to be formed in the following manner. A portion of clot is detached from a vein in the neighbourhood of the suppurating point and is carried into a branch of the portal vein where it lodges, and by reason of its inflammatory nature sets up inflammation in the hepatic tissue around it.

Multiple abscesses in the liver being a part of general pyæmia are very seldom recognised during the life of the patient, because their presence is obscured by the gravity of the general mischief going on in the system, such as abscesses in the lungs and joints, together with the exhausting rigors, pyrexia, &c. Moreover the general disease is usually very rapidly fatal.

IDIOPATHIC or TROPICAL ABSCESSES are generally solitary and large, and run a chronic course.

An immense amount of controversy has arisen as to the origin of these abscesses in the liver into which it is not my purpose to enter. Neither shall I pretend to hold the balance between the contending factions. A brief statement of the views held by persons who have had considerable opportunity of forming an opinion on the subject will, however, be desirable.

More than thirty years ago Budd noticed that the majority of hepatic abscesses he met with were in persons who had been in the tropics, and in whom, on *post-mortem* examination the colon presented old ulcers, the result of tropical dysentery; from which fact he was led to believe that a morbid material, taken up from the ulcers by the portal vein and deposited in the liver had set up suppurative inflammation in that organ. In short, Budd attributed tropical abscess to a *local* pyæmia.

Admitting the facts of Budd's observations as far as his experience went, many medical men who have had large experience of hepatic abscesses in the tropics, differ very materially from him in his conclusions; and in the present day it cannot be said with any degree of certainty whether the colic ulceration and the hepatic abscess stand to each other in the relation of cause and effect, or *vice versa*, or whether the connection between the two diseases is merely fortuitous. This uncertainty arises from the facts, first, that the ulceration in the colon is sometimes evidently of recent origin, while the hepatic abscess is chronic. Secondly, while on the one hand, intestinal ulceration is extremely common in this country, on the other hand abscesses in the liver are very rarely met with. Thirdly, in tropical countries, abscesses of th

liver frequently occur quite independently of any symptoms of dysentery. Fourthly, hepatic abscess may precede dysentery. Fifthly, patients have died of hepatic abscess in the tropics, and the most careful search failed to reveal any trace of old or recent ulceration in the stomach or intestines.

On the whole, I think there can be no reasonable doubt but that there is a specific difference between the multiple hepatic abscesses met with in cool climates, and the large solitary abscesses of tropical climates, not only on account of their size and number, but also on account of the chronic character and amenability to treatment evinced by the latter.

According to Hirsch, tropical abscess of the liver is endemic in Upper Egypt and Algiers, in which countries, the disease constitutes as much as 5% of all illnesses, and at times even a much larger proportion. In the corresponding zones of America it is of rare occurrence. In Europe the disease is relatively frequent, in Andalusia, Malta, Sicily, and in the Ionian Islands. Within the tropics there are certain regions which enjoy an almost entire immunity from this affection; such as the Island of Singapore, South coast of China, the Sandwich Islands, and the Continent of Australia.

This disease occurs most frequently at that period of the year which is characterised by high diurnal and low nocturnal temperature. Europeans resident in the tropics are far more liable to abscess in the liver than the natives, and this appears to arise from the fact that such persons carry with them the habit of indulging immoderately in animal diet and alcoholic stimulants, instead of copying the frugal habits of the natives who subsist almost entirely upon vegetables. These dietetic errors, combined with indolent habits, tend to produce hyperæmia of the liver, which is very probably the prelude to tropical abscess.

Tropical abscess is much more frequently observed in males than in females, and very rarely occurs during the first ten years of life. Unfavourable hygienic conditions and bodily hardships tend to predispose to the disease, and a sudden chill has a decided effect in producing suppuration in the liver.

Dysentery is frequently found associated with liver abscess in the tropics, but the connection between the two diseases, as already stated, is not quite clear.

Anatomical Changes.—Suppurative inflammation in the liver is always limited to isolated patches, varying in number according to the mode of origin of the disease. If this have been from purulent infection a large number of small abscesses are usually observed scattered throughout the organ, and especially prominent in many cases upon the surface, where they form yellow flattened projections. They generally vary in size from a hazel nut to a walnut, but by the coalescence of two or more smaller ones, large abscesses may result. On the other hand, traumatic, and especially tropical abscesses are usually found solitary and of a large size. Occasionally, however, small secondary abscesses are found in connection with large tropical abscesses as the result of local pyæmic infection. The size to which these solitary abscesses sometimes attain is truly astonishing. Most frequently they vary in size from that of a man's fist to a child's head, but they have been known to involve the entire organ, and Murchison mentions a case under his care in which 160 ounces of pus were drawn off by tapping during life.

The difference between pyæmic and tropical abscesses in size and number is due mainly to the longer time the patient usually survives with the latter disease in comparison to that in the former.

The most frequent site of tropical abscesses is in the right lobe of the liver and near to its surface.

The mode of formation of pyæmic abscesses which have been most studied, appears to be as follows :—Isolated lobules are first observed as yellowish dots, their margins still remaining firm ; these gradually unite, forming small collections of pus, which increase rapidly, and, becoming incorporated with others in their vicinity, at length give rise to extensive abscesses. During this process the hepatic cells are gradually destroyed and their products become mixed up with those of the purulent softening of

the inflamed parts. Recent abscesses consist of pale yellow viscid pus, bounded by softened hepatic tissue, which hangs in shreds into the interior. Enlargement of these abscesses takes place by means of progressive disintegration of their walls, and by this means it frequently happens that adjacent abscesses coalesce, thus forming larger abscesses with uneven walls, and traversed by cord-like processes or bridges of hepatic tissue.

When the abscess reaches the surface of the liver it usually gives rise to adhesive inflammation which glues together the adjacent surfaces of the peritoneum, and when the suppuration then extends beyond the boundary of the liver the abscess may discharge itself in various directions. The most frequent course for the pus to take in these cases is into the stomach, the duodenum, or the colon. It rarely happens that the pus finds its way into the bowels through the bile-ducts or gall-bladder. The most favourable course the pus can pursue is directly outwards through the abdominal wall, but frequently it is deflected from this course by coming in contact with the faciæ and tendons, and may thus be caused to burrow for a long distance, and finally seek an outlet in the axillary, sacral, or inguinal region. By perforating the diaphragm, the pus may sometimes be discharged into the right pleura, bronchi, or pericardium. In the latter case, death speedily follows. In very rare cases the abscess opens into the right kidney, and only exceptionally into the inferior vena cava and the portal vein.

If, however, adhesive inflammation of the capsule of the liver to an adjacent part have not preceded bursting of the abscess, the pus is discharged into the peritoneum where it rapidly sets up fatal peritonitis.

An abscess which has discharged its contents, either externally through the abdominal wall, or into one of the hollow viscera, may heal in the following manner: suppuration ceases and the walls of the cavity become approximated, and ultimately are united by newly-formed connective tissue; from the contraction of this tissue there is produced in the parenchyma of the liver a radiating cicatrix composed of fibrous bands; and a depression

remains on the surface of the liver. In very exceptional cases abscess of the liver may heal, or at least remain dormant for a long time, by the fluid contents becoming absorbed and the walls approximating and ultimately becoming united into a firm cicatrix which usually encloses a cheesy or calcareous mass. But even in these apparently favourable cases it is nearly certain that after having lain dormant for a long while, a recrudescence of the disease may occur at any time.

Besides these modes of termination of the disease, when the suppuration advances, and the abscess is not circumscribed, death usually ensues under typhoid symptoms, or those of hectic fever, leading to exhaustion. When the abscess makes its way outwards, or into any of the neighboring organs or cavities, the symptoms vary extremely according to the direction taken by the pus. When the abscess penetrates into the cavity of the right pleura it gives rise to the ordinary train of symptoms of pleurisy. When it finds its way into the adherent right lung, suppurative pneumonia ensues, and when a communication is established between the abscess and the bronchi, pus of a bloody, putrid character is usually expectorated.

Symptoms.—A large number of cases of hepatic abscess are reported in which the disease gave rise to no symptoms whatever whereby the nature of the affection could be diagnosed during the life of the patients; nor even to any local changes or functional derangements which might have pointed to the liver as being the seat of disease. All that can be done under this heading is to describe the symptoms present in a well-marked case of hepatic abscess. Such are those in which the abscess is of the traumatic variety.

Here we probably have the history of a blow inflicted over the region of the liver. There is pain and tenderness, or a feeling of weight or uneasiness in this region. The pain may extend into the right shoulder, and the ribs are observed to be deficient in respiratory movements over the liver. The area of hepatic dulness is found to be uniformly augmented. Constitutional symptoms

are soon developed. These are, hot and dry skin, thirst, frequent and full pulse, loss of appetite, tongue coated with yellow fur, bilious vomiting, bowels sometimes confined, and at other times relaxed, with bilious evacuations.

As soon as suppuration commences, the gastric disturbance usually becomes aggravated, the fever increases in intensity, and rigors occur at regular or irregular intervals followed by heats and profuse and exhausting sweats. When the abscess is completely developed, the general enlargement of the liver usually subsides, and if the abscess be favourably situated for palpation, a circumscribed fluctuating tumor may be felt. In many cases, however, the abscess is either surrounded by hepatic parenchyma, or is so deep-seated as to render its detection by this means quite impossible.

The foregoing description applies only to a typical case of abscess in the liver, but such cases are very rarely met with in practice. Some or all of the above-mentioned symptoms may be, and frequently are, absent in a majority of cases. It will therefore be necessary to make an

Analysis of the Symptoms.

(a) Pyrexia may be absent during the entire course of chronic abscess of the liver, and also during the first stage of acute cases. As a rule, however, it is present at the very outset of the latter. The most usual form of fever is the regular intermittent, in which the attack, ushered in by a chill, followed by a hot stage, ending in a profuse perspiration, occurs daily about the same time. During the paroxysm of fever the temperature generally rises to 104° Fahr. In subacute cases this form of fever may persist for weeks. In some cases, however, rigors followed by profuse perspirations occur at very irregular intervals, it may be twice in the

same day, or every third day. Murchison says rigors and night sweats are less prominent symptoms in the tropical than in the pyæmic abscess. The pulse usually stands in the ordinary relation to the temperature during the earlier stages of the disease, but as death approaches it becomes continuously accelerated and progressively weaker.

(b) PAIN, TENDERNESS, AND TENSION in the hepatic region are very often absent. When present, pain is slight and dull when it depends upon alterations in the hepatic parenchyma, and is wont to abate after the development of the abscess. As the abscess approaches the surface, the capsule of the liver becoming distended and probably also inflamed, the pain becomes more acute like that of pleurisy, and tenderness also is superadded. When pain in the side is the result of perihepatitis a friction sound produced by the acts of respiration can sometimes be heard with the stethoscope placed over the liver. In some cases, however, the pain is so much increased by a deep inspiration that the patient cannot be persuaded to take it. Pain in the right shoulder is very commonly complained of when the abscess is situated in the convex surface of the right lobe of the liver.

(c) DERANGEMENT OF THE DIGESTIVE ORGANS.—Indications of gastric derangement, such as a yellow or grey-coated tongue, loss of appetite vomiting, are somewhat common in connection with hepatitis, especially after suppuration has occurred. With regard to vomiting in these cases, authorities differ widely in opinion as to its cause. Budd attributes it solely to sympathy between the stomach and liver, and Frerichs to compression on the stomach by a projecting abscess. Constipation or diarrhœa is usually present in idiopathic hepatitis. Dysentery may precede or follow liver abscess, or may be absent during the entire course of the disease.

(d) JAUNDICE is a very rare symptom in hepatic abscess. It occasionally occurs in a very slight form during the primary stage of hyperæmia, but may be more severe and persistent at a later stage owing to compression of the ducts by purulent collections. It is present in about 6% of the cases.

(e) ASCITES AND ŒDEMA occur rarely, and then merely as accidental accompaniments of the disease, brought on by compression of the portal vein and inferior cava.

(f) THE RESPIRATORY ORGANS frequently manifest various disturbances in connection with hepatic abscess. If the liver be considerably enlarged it hinders free motion of the diaphragm, and dyspnœa is produced by even slight physical exertion. Persistent difficulty of breathing comes on when a large abscess on the upper surface of the liver bulges up into the thorax. The compression of the lung in this manner may be so considerable as to render indistinct the vesicular breathing, and cause bronchial breathing alone to be heard in the compressed lung. A short, dry, sympathetic cough is also frequently noticed,—the hepatic cough of some authors.

(g) FLUCTUATION can usually be detected in the abscess when it comes towards the surface, and the area of fluctuation is usually surrounded by a margin of hardened tissues.

DIAGNOSIS.—A fluctuating tumor in the region of the liver, accompanied at one stage or another of its development by rigors and fever in a person living in, or who has lived in, the tropics, is very probably an hepatic abscess. But as matters are not always quite so clear, it will be necessary to take a brief survey of the diseases likely to be confounded with liver abscess.

(1) HYDATID TUMORS may be recognised by their slower growth, absence of pain, and constitutional disturbance. In the case of one of these tumors having suppurated, the treatment is the same as that for abscess.

(2) A DISTENDED GALL-BLADDER may generally be distinguished by its pear-shaped or semi-globular form, an abscess generally having the shape of a low, rounded prominence, with a broader base. The doughy feeling which is usually observed in hepatic abscesses adherent to the abdominal wall is absent from the distended gall-bladder.

PROGNOSIS.—This is generally unfavourable. About 17% of persons suffering from tropical abscess recover; but with pyæmic abscess recovery rarely, if ever, occurs. Generally speaking, the chances of recovery are impaired by the co-existence of dysentery, obstinate intermittent fever, and symptoms of general peritonitis. When the abscess bursts into the pericardium or peritoneum, the result is always fatal. The prognosis is slightly more favourable when the abscess discharges into the pleura. It is still more favourable when the pus is discharged through the bronchi, stomach, or colon. The opening of the abscess through the abdominal or thoracic wall, when left to itself, and not expedited by operative interference, leads but seldom to recovery, on account of the exhaustion produced by the burrowing of the pus.

TREATMENT.—This resolves itself into two classes, namely, that to be adopted before suppuration has occurred; and secondly, that which is appropriate after that process has taken place.

The treatment before suppuration must consist in a moderate abstraction of blood, either locally by means of cupping-glasses or leeches applied over the liver, or of leeches in the immediate vicinity of the anus. Advantage will also be derived from cold compresses or an ice-bag over the hepatic region. In some cases warm applications will be more grateful to the patient's feelings.

With regard to purgatives, calomel appears to be the favourite drug with many persons who have had great experience in the treatment of this disease. It must, however, only be administered occasionally as a purgative, and on no account continued so long as to produce salivation. Its use is contraindicated when irritability of the stomach, or dysentery is present. Chloride of ammonium in doses of twenty grains twice or three times a day diminishes portal congestion and relieves hepatic pain. Ipecacuanha appears to be a particularly appropriate remedy in cases of hepatitis complicated with dysentery. It must be given in large doses, from thirty to ninety grains every six or twelve hours, according to the severity of the case.

Pain and sleeplessness must be combated by opium internally, or morphia subcutaneously. The diet should be mild and unstimulating.

After suppuration has taken place the treatment must be somewhat varied. The antiphlogistic measures suitable for the initial stage of the disease must now be suspended, and tonic medicines, and a supporting regimen adopted in its stead. Any local inflammatory symptoms must now be treated with warm emolient poultices or vesicants. The supervention of exhaustion must be prevented as far as it is possible by the administration of mineral acids and quinine, together with a generous diet, to which wine or spirits—inadmissable at the early stage of the disease—may now be added. Purgatives, if now required, must consist of the vegetable series, such as rhubarb, aloes, senna, and the like. In most cases it will be found necessary to administer opium in some form or other for the relief of pain, diarrhœa, sleeplessness, or cough. At this time all sudden motions of the body must be avoided on account of the risk of rupturing the abscess, because the pus might possibly take the unfavourable course into the peritoneum.

When an abscess has developed in the liver, the question as to the propriety of evacuating the pus by operative measures will present itself to the mind of the medical attendant. Under what circumstances then is he justified in doing so?

The most favourable circumstances for operative interference are: in a young robust patient, with an abscess recent and small, pointing in the abdominal wall, with fluctuation distinct, and adhesion known to have taken place between the visceral and parietal peritoneum over the abscess. In such a case as the one just depicted, with proper antiseptic precautions, a free incision down to the pus may be made with well-founded hope of a favourable result.

But unfortunately matters are seldom, if ever, so straightforward as in this hypothetical case. In the first place we may not be certain of the presence of pus in the liver although the symptoms may be such as to lead us to suspect it. In that case we must

assure ourselves of its presence by plunging the aspirator needle into the most prominent point of the bulging tumor and drawing out a portion of the pus.

Secondly, being satisfied on the first point, we may not be certain that adhesion has taken place between the layers of peritoneum over the abscess. This is diagnosed by the occurrence of acute local pain and friction sounds which subside and leave the liver immovable during the acts of respiration. If there be any doubt as to the existence of the necessary adhesions the better way to proceed is in the manner first recommended by Graves of Dublin. Make an incision about four inches long over the centre of the tumor in the right hypochondrium down to within one or two lines of the peritoneum, and plug the incision with lint and thus keep it open for a few days until adhesion has taken place. After this the abscess may discharge itself, or if not, the incision may be carried down to the pus, or a trocar and canula may be plunged into the abscess, and the canula left there; or better still. a drainage tube may be inserted in its stead.

All operative proceedings about the liver must be undertaken with strictly antiseptic precautions. The use of the aspirator is better confined to diagnostic purposes, because when used to evacuate the abscess, pus is almost certain to re-accumulate. The trocar, however, may be useful for small abscesses, but the weight of authority in the present day is decidedly in favour of free opening and drainage, together with antiseptic precautions, when large abscesses have to be dealt with.

INTERSTITIAL HEPATITIS.

CIRRHOSIS.

THE disease of the liver called Cirrhosis by Laënnec (from κιῤῥός—yellow), consists in a hyperplasia of the interlobular connective tissue which subsequently indurates and contracts, in consequence of which the lobules are pressed together into a number of nodules or so-called granulations. This will account for the diminution in size of the organ always observed in the advanced stages of the disease, the impairment of its secretory functions, and the impediment to the circulation within it. From the prevalency of the disease amongst immoderate drinkers, it has been called by English practitioners "gin-drinker's liver;" from the surface of the liver being irregular and covered with projections it has been called "hobnailed liver;" and finally, on account of the granular appearance of the cut surface, "granular liver."

ETIOLOGY.—The most common cause of cirrhosis is the habit of taking spirits or strong wine in an undiluted form and on an empty stomach. When alcohol is introduced into the stomach it is almost immediately absorbed by the gastric veins and carried to the liver where it is presented in a much less diluted form than to any other organ. Here it gives rise to irritation which would soon subside if it were allowed to do so; but more fuel being constantly added to the fire, the irritation is kept up, and thus a chronic form of interstitial hepatitis supervenes with subsequent induration of the parenchyma.

A large number of cases of cirrhosis, however, are observed which cannot be attributed to the abuse of ardent spirits nor any other known cause, with the exception of a few cases which appear to have a syphilitic origin. In the syphilitic variety, it is true, the process is not always developed with the same regularity throughout the entire organ as is usually observed where it is induced by the action of alcohol; yet undoubtedly the syphilitic affection of the liver occasionally makes its appearance as cirrhosis.

Cirrhosis of the liver is more common at an advanced age than in early life, for the simple reason that the vicious habits which produce the disease have then had time to work their baneful influence. Previous to thirty years of age this disease is of rare occurrence. As regards sex, the disease appears to be pretty evenly distributed between males and females.

Morbid Appearances.—The cirrhotic liver, in the majority of cases, is found on *post-mortem* examination, reduced in size, and attached by adventitious tissue to the neighboring organs, such as the diaphragm, the colon, the stomach, &c. The reduction in size depends upon the time during which the disease has lasted. In the slighter forms of granular induration, the liver is somewhat enlarged, or of normal size. But in the advanced stages of the disease the liver may be reduced to one half of its normal size. The diminution in size is particularly noticeable in the left lobe which may be shrivelled up into a small membranous appendage of the right. In the right lobe, the sharp margin is usually transformed into a thin flabby rim of connective tissue. The surface of the liver is rendered uneven by numerous nodules, and knobs which show a yellow colour through the capsule. The serous envelope is almost always thickened and coriaceous, and of a grayish-white colour, especially in the depressions between the granulations. These so-called granulations vary in size from that of a millet-seed to that of a pea. On cutting into the cirrhotic liver its structure is found to be very firm, amounting almost to a cartilaginous hardness and a grating sound is emitted as the knife passes through it. The cut surface presents a

network of broad or narrow whitish-gray lines composed of indurated connective tissue with irregular-shaped granules usually of a deep yellow colour in their interstices. It was this appearance of the cut surface which induced Laënnec to apply the name cirrhosis to the disease, under the erroneous impression that the yellow granulations were new formations, by the development of which the normal structure of the liver is destroyed. This, however, is not the case; on the contrary, the yellow granulations consist of the remains of the hepatic lobules, brought into greater prominence, as the result of morbid changes in the highly vascular connective tissue which pervades the entire organ and supports the blood-vessels and secreting structure of the liver, known by the name of Glisson's capsule.

When death takes place at an earlier stage of the disease, the liver, as has been already mentioned, is almost always enlarged, sometimes even to a considerable extent. The form of the organ does not vary much from the normal. The surface is covered with a smooth, or somewhat opaque and thickened capsule, and on close inspection, granulations, varying in size from a pin's head to a pea, may be discovered. On section, granulations, similar to those seen on the outer surface are observed, of a more or less intensely yellow colour, separated from each other by strips of pale rose-red or gray-red areolar tissue. The latter is considerably thickened, and forms net-like streaks which penetrate deeper between the lobules than the normal interlobular structure.

The transition from the condition of the liver just described to that first depicted is brought about by the subsequent hardening and contraction of the interlobular connective tissue which throws the surface of the organ into irregular projections.

Microscopic examination shows that the abnormally proliferated interlobular connective tissue is crowded more or less with round and spindle-shaped cells. This infiltration is usually most abundant in the vicinity of the finest ramifications of the portal vein. A portion of the cells of this infiltration is developed into a fibrous texture, very rich in fixed corpuscles, which by shriveling come gradually to resemble firm cicatritial tissue. Another portion of

the cells is in all probability to be regarded as the first instalment of newly developing vessels branching out from the capillaries of the interlobular arterioles; for the compression exerted by the new growth tells more upon the feebler current in the portal vein, therefore the hepatic artery enlarges to compensate for it.

The impediment to the portal circulation may lead, in severe cases, to stagnation of blood with thrombosis in the portal vein. If life be prolonged after this, bile is secreted from the blood of the hepatic artery, but fatal evils depending on stasis in the portal system, namely, ascites, marasmus, hæmorrhages, &c., are developed, which ultimately destroy life in various ways.

Symptoms.—Cirrhosis of the liver is usually developed insidiously. It is only in those exceptional cases where the disease commences with acute inflammation in the capsule that the early stage comes under the observation of the physician.

The group of symptoms known as biliousness—nausea, furred tongue, bitter taste in the mouth, flatulence, loss of appetite, pain after solid food, irregular bowels—usually accompany the setting in of the morbid processes which lead to cirrhosis in persons addicted to intemperance. Later on, a dull pain, with slight tenderness in the right hypochondrium is complained of, and on examination the liver is probably found to be enlarged. At the same time a slight degree of fever may be noticed, together with faint jaundice, amounting, perhaps to no more than sallowness of the skin. This stage of the disease corresponds to the hyperplasia of the connective tissue.

After a period varying in length these symptoms abate, and as the new growth hardens and contracts a new train of symptoms manifest themselves. The earlier symptoms may have been entirely absent, or so slight as to have passed unnoticed, but when contraction takes place the symptoms produced obtrude themselves with unerring certainty. These symptoms are due principally to obstructed portal circulation.

The area of hepatic dulness has now diminished, particularly in the locality usually occupied by the left lobe of the liver. The reduction in volume of the cirrhotic liver usually proceeds

slowly, and the area of dulness in the papillary line may ultimately be reduced to a narrow strip which extends over from two to three ribs. The nodulated surface of the liver may sometimes be detected by palpation, especially after paracentesis has been performed, and may be distinguished from similar projections in the abdominal walls by their upward and downward motion accompanying the acts of respiration. More frequently, however, by the time the irregularity of the surface is sufficiently developed for this purpose, the lower margin of the liver has receded behind the costal wall, or is concealed by ascitic fluid. Pain and tenderness, present as a rule in the hepatic region as long as the liver is enlarged, have now quite subsided, and that region is quite free from tension. It is only in consequence of transitory attacks of perihepatitis that sensitiveness in the hepatic region develops during the contraction stage of cirrhosis.

In consequence of the obliteration of numerous capillary branches of the portal vein by the shrinking of the connective tissue, and of the insufficiency of the new channels of communication which are established between the portal vein and the vena cava, through the medium of the hepatic veins, for the free circulation of the blood, there results an increased lateral pressure in the portal system which gives rise to a series of anatomical changes and functional derangements which will now claim our attention. This increased blood pressure is propagated in those veins outside the liver which form communication between the portal system and the right side of the heart. Those veins are dilated and enlarged, and thus serve as outlets for the obstructed portal blood. The channel most suitable for establishing the collateral circulation in these cases is a vein inside the round ligament of the liver, and running almost its entire length, which receives several sub-peritoneal twigs from the abdominal wall, and discharges into the *sinus venæ portæ*. This channel for the collateral circulation was first described by Sappey, and the vein itself has recently been demonstrated by Baumgarten to form the remaining portion of the inperfectly obliterated umbilical vein, which in most persons continues pervious during entire life. It is sometimes found sufficiently large to admit a thin or thick steel

sound, and in cirrhosis it may become dilated to the size of a goose-quill or even the little finger. When the distended umbilical vein is the channel for the collateral circulation, the current of blood takes a course the reverse of the normal, and flows from the portal vein to the abdominal walls, the veins of which are thereby greatly dilated, and project as highly tortuous, bluish ridges beneath the skin. These then form a plexus that surrounds the umbilicus in the form of a wreath, named the caput medusæ, from whence varicose veins extend on both sides of it, upwards as far as the epigastrium and over the anterior surface of the thorax where they join the axillary veins, and downwards towards the inguinal region.

Another channel for the collateral circulation is to be found in the newly formed adhesions of the liver to the diaphragm and abdominal wall. In this way an anastomosis is formed between the perihepatic ramifications of the portal vein and the veins of the diaphragm.

Although it is a widely accepted theory that the portal system is principally relieved in these cases through the anastomosis between the inferior mesenteric vein and the hypogastric vein, through the inferior hæmorrhoidal, yet there are few direct proofs in support of it, and Frerichs, Sappey, and Thierfelder have not found hæmorrhoidal varices by any means frequent in cirrhosis.

A varicose dilatation of the æsophageal veins, by means of which the blood is discharged from the coronary vein of the stomach into the vena azygos was first observed by Fauvel, and has frequently been seen in recent years.

It is only in extremely rare instances that the collateral circulation is sufficiently established in cirrhosis to completely compensate for the obstruction in the portal system; consequently stasis of blood in this system is developed, which manifests itself in various ways.

The spleen enlarges, causing an increase in the area of splenic dulness. This, however, is not constant, but occurs in about two-thirds of the cases, and may be completely masked by meteorismus or by fluid in the peritoneum.

Ascites almost constantly accompanies cirrhosis of the liver. It is only absent where death supervenes before extensive shrinking of the newly-formed tissue has taken place, or in those very rare cases in which an efficient collateral circulation has been established. It results from the transudation of serum from the engorged portal system. The effusion usually commences at an early stage of the disease, and may even precede all other symptoms of obstruction : once present, as a rule it does not again disappear, and when removed by paracentesis, the fluid rapidly reappears. Ascites usually precedes œdema of the feet, in fact, the pressure exerted by fluid in the peritoneum upon the inferior cava and iliac veins impedes the return of the blood from the lower extremities and interferes with the functions of the kidneys. In this way œdema of the feet and scanty (sometimes albuminous) urine are produced which rapidly disappear on the removal of the ascitic fluid.

The fluid effused into the peritoneal cavity is usually clear, yellow in color, has a specific gravity of from 1012 to 1016, and contains about one per cent. of albumen.

The stomach and intestines very commonly manifest symptoms of derangement. When these symptoms occur early in the disease they usually are the result of the pernicious effects of alcohol on the mucous membrane, but later on, when the portal circulation is embarrassed, they usually depend upon nutritive and functional derangements developed in the walls of the digestive canal under the influence of prolonged stasis of the blood. Catarrh of the stomach and bowels are very commonly observed, the appetite is bad, the bowels confined, and nausea is complained of. Profuse hæmorrhages from the stomach and bowels frequently take place, and sometimes prove fatal. They usually occur when the ascites has attained such a degree that further escape of serum into the peritoneum is prevented by tension of the abdominal walls. Hæmatemisis, however, may occur previous to ascites, and thus constitute the first indication to the morbid process going on in the liver. In some cases these hæmorrhages are followed by great relief of all the other symptoms.

Another effect of the lethargic circulation in the portal system is to diminish, and subsequently entirely suspend, intestinal absorption of nutritive material. The blood-forming and assimilating functions of the liver are greatly impaired, and a quantity of the albuminous constituents of the blood are lost to the system in the ascitic transudation. It is not surprising therefore that progressive emaciation and loss of strength are inseparable from cirrhosis.

Jaundice of an intense character is decidedly rare in cirrhotic contraction of the liver. It may be present at an early stage of the disease as a result of gastro-intestinal catarrh, in which case it seldom surpasses a light yellowish discoloration of the skin and conjunctivæ. As the disease advances jaundice is very seldom observed. We are therefore forced to conclude that the capillary bile-ducts are not rendered impervious by the shrinking of the newly-formed connective tissue until after the morbid process has obliterated the interlobular veins which convey the materials necessary for the elaboration of the bile in the hepatic cells.

The urine is almost invariably scanty, high colored, and acid in reaction, and deposits large quantities of pink, dark red, or brownish urates. After paracentesis of the abdomen the urine almost always increases in quantity, and if previously albuminous, the albumen disappears from it if the kidneys are healthy.

In consequence of the progressive atrophy of the secreting parenchyma, the quantity of bile secreted is synchronously diminished. The stools gradually become more and more pale in color although jaundice may be absent. We should expect to find the quantity of urea excreted by the kidneys also diminished in quantity, but this point is not quite settled yet.

Diagnosis.—At the bedside it is not always possible to diagnose with certainty cirrhosis of the liver. The indications which must guide us are the following:—adult age of patient; a previous history of intemperate habits together with persistent derangement of the digestive organs with no obvious organic disease of the stomach; the presence of ascites with enlarged veins on the abdomen; enlargement of the spleen; diminished area of hepatic

dulness; increasing paleness of the fæces; wasting of the body fat. These indications when they occur simultaneously are sufficient as a basis on which to found both our prognosis and treatment, but are not sufficient to warrant a certain diagnosis of cirrhotic contraction, the only reliable indication of which is being able to feel the nodules on a contracted liver; this can only be effected after paracentesis, the relaxed abdominal walls then permitting the hand to be thrust in deeply under the costal margin.

PROGNOSIS.—In advanced cirrhosis with ascites the prognosis is highly unfavorable; death, as a rule, is inevitable. A few cases, however, are reported in which the disease appeared to have remained stationary after paracentesis had been performed, in some of them repeatedly, but there appear to be good grounds for doubting the accuracy of the diagnosis in almost every case of the kind with which I am acquainted.

TREATMENT.—The treatment of cirrhosis entirely depends upon the stage the disease is in at the time of observation. With a person whom we have reason to believe, from his clinical history, habits, &c., to be suffering from incipient cirrhosis, prophylactic treatment will not only be safe but also judicious. Alcoholic stimulants, irritating condiments, strong coffee, and such like articles must be interdicted. The diet must be plain and nutritious, consisting of such things as mutton, milk, farinaceous substances, vegetables and ripe fruit. The drink must be restricted to water, weak tea, or some bitter infusion in combination with ammonia and ginger, which will allay the patient's craving for stimulants. The digestive functions which have suffered impairment through intemperance, will be restored by the administration of alkalies and bitter infusions. The bowels must be regulated by the use of salines and mineral waters, such as sulphate of magnesia, soda or potash, bitartrate of potash, Carlsbad or Cheltenham salts, Friedrichshall or Püllna water, or perhaps better still, Hunyadi Janos water, as it has less tendency to gripe or produce nausea than Püllna water. In the meantime, plenty of open air exercise will be indispensible.

Unfortunately, we rarely have an opportunity of treating cirrhosis in its earliest stage, when the disease is amenable to treatment, and still more rarely can we prevail upon the subjects of it to follow our advice. It is almost invariably in the last stage of the affection that medical advice is sought, and then it is too late to be of any service, because no treatment can restore the portion of the liver already destroyed, or remove the obstruction to the portal circulation within that organ. At this late period all we can hope to do is, relieve the most urgent of the symptoms.

As the patient's constitution is usually in a shattered condition at this period, all violent measures and debilitating remedies must be strictly avoided. Any exacerbation of perihepatitis must be treated by rest, warm cataplasms, and fomentations; local abstraction of blood being, if possible, avoided. The patient's strength must be maintained by appropriate means at a fair standard as long as it is possible to do so. In order to accomplish this end, the treatment recommended for the incipient stage of the disease will be suitable in this stage also, with the addition of some preparation of iron and a little wine or beer, if the strength be very much reduced.

Ascites, generally sooner or later, demands treatment; and our most anxious consideration will be engaged in order to discover the most appropriate means of mitigating it. One class of remedies, however, which have a reputation for dispersing dropsical effusions may be summarily dismissed from our purview, namely, *hydragogue cathartics*. They only do harm by weakening the patient already debilitated, and by irritating the stomach and bowels which it ought to be our object to soothe. Another class of remedies frequently employed for the removal of ascites is *diuretics;* but it is only in exceptional instances that these drugs prove efficacious, and then it is in all probability, in consequence of the ascites having been promoted by some concurrent derangement of the kidneys. The resin of copaiba in ten or fifteen grain doses, three times a day has frequently been found to remove ascites by its action on the kidneys. But these remedies more often fail to increase the flow of urine, and even when they

succeed in that, the ascites may not be diminished thereby; whereas, after paracentesis the flow of urine may increase spontaneously.

With regard to paracentesis, opinions are divided, not only as to the time at which it should be performed, but also as to its effect in hastening or delaying the fatal termination. The venerable opinion entertained by Budd, Frerichs, Thierfelder, and many others, is that the operation should be delayed as long as possible, and not undertaken or repeated until the pressure of the ascitic fluid so embarrasses the respiration as to threaten death by suffocation; because, say they, by withdrawing the fluid, which is certain to re-accumulate, you also withdraw a quantity of albumen from the blood, and thus cause a drain upon the patient's system which his constitution, already enfeebled by dissipation and disease, is unable to withstand, therefore you only hasten the fatal termination.

But a new school has arisen of late years of which Murchison, Dr. F. T. Roberts, and others are disciples, which preaches the doctrine of early and repeated tappings in cirrhosis. I shall here quote Murchison as an exponent of this modern school of practice. He says (Clin. Lect. on Dis. of Liver 2nd. Edit. p. 288), "the advantages of early tapping are, that by removal of pressure the establishment of a collateral circulation through the more healthy portions of the liver itself, as well as through the veins of the abdominal parieties, is promoted. Secondly, the functions of important parts which had been impaired or arrested by the pressure are restored. Not only are the lungs relieved, but by the removal of pressure from the portal and renal veins, assimilation and the secretion of urine are increased. I have known hæmorrhage from the bowels arrested by paracentesis, and it is a common observation that patients with much ascites, who, notwithstanding the most powerful diuretics, have been passing only a small quantity of urine containing much albumen, will, after paracentesis, and independently of drugs, void large quantities of urine free from albumen. And thirdly, diuretics and other remedies, which, when the abdomen is full of fluid have produced no effect, pro-

bably from not being absorbed, will often after paracentesis act powerfully. and thus retard or prevent the reaccumulation of fluid in the peritoneum. As soon, therefore, as the abdomen becomes moderately distended with fluid, and the remedies which I have mentioned to you fail to produce any effect, I would recommend you to lose no time in having recourse to paracentesis. Even should the fluid reaccumulate you need not despair."

In support of this teaching, Murchison quotes a case in which tapping was performed four times, and after the fourth tapping there was no reaccumulation of fluid. It is unfortunate that in the account of the morbid appearances observed at the *post-mortem* examination in this case there is nothing more definite stated as to the condition of the liver than that it was found to be "very small, and weighed only 35 oz.," (no uncommon weight for a healthy liver), and further that it was "in an advanced state of cirrhosis;" statements, which, to say the least of them are irritatingly vague. But as it is also stated that the "omentum was firmly adherent to the abdominal wall," it may fairly be asked—What part did chronic peritonitis play as a factor in producing the ascites? And as the patient suffered from leukæmia, may not the latter disease have had some influence in the same direction?

Another case of reputed recovery from cirrhosis after tapping was reported by Professor Leudet of Rouen:—but here the primary disease which was relieved by tapping, appears to have been chronic peritonitis, as evinced by "old false membranes found in the abdomen;" the patient subsequently dying with thrombosis of the portal vein. (Vide Ziemssen's Cyclop. vol. ix, p. 205, et sq.)

It would be very easy to point to several cases of reputed recovery from cirrhosis after repeated tappings, but as none of them, as far as I am aware, have been verified by *post-mortem* examination, they are quite useless as guides to the relative merits of the mode of treatment adopted. We are, therefore, justified in suspending our judgment in this question until time shall reveal a more conclusive refutation of the older doctrine than we are at present possessed of.

In the meantime I would advise (and practice) deferring paracentesis in genuine cirrhosis until the symptoms of impeded respiration are sufficiently urgent to warrant it; because in every case of this character in which I have seen the operation performed, reaccumulation of the fluid has occurred with unerring certainty, and each time the operation was repeated, exhaustion was more pronounced, until at last, it was evident that the patient would succumb under another attempt to remove the fluid.

If, however, the practitioner prefer to adopt the modern method of treatment nothing will be lost by delaying paracentesis until urgent symptoms have manifested themselves, as two cases of reputed recovery from cirrhosis in consequence of early and repeated tappings are reported in the *Lancet*, (1st, Nov. 19, 1881, p. 868, 2nd, ibid. May 6th, 1882, p. 730), in neither of which does the operation appear to have been one of choice, but of necessity. In the first-mentioned case we read, "the dropsical symptoms having *increased to a marked degree* paracentesis abdominis was performed;" and in the second case we read "*the breathing becoming daily more difficult*, that the question of tapping could not be much further delayed." And yet in both these cases recovery is reported as having followed the operation.

SYPHILITIC HEPATITIS.

CONSTITUTIONAL Syphilis manifests itself in the liver in three different forms :—1, as interstitial hepatitis and perihepatitis; 2, as hepatic gummata; and 3, as amyloid degeneration.

(1.) INTERSTITIAL HEPATITIS.—Cirrhosis of the liver when found in connection with constitutional syphilis, has, by some authorities, been attributed to the syphilitic process. However this may be, it must be very difficult to distinguish between the syphilitic variety of the disease and that form of it which is produced by spirit drinking, especially when, as is commonly the case, the two etiological factors are at work in the same persons. Drs. Wilks and Moxon, and Professor Thierfelder express their inability to differentiate between the two forms of the disease.

(2.) HEPATIC GUMMATA are often observed in childhood, but more frequently at a maturer age, and represent the purest form of syphilitic disease of the liver. They are found in the liver otherwise healthy, or in connection with other morbid processes, such as fatty or amyloid degeneration, cirrhosis, &c. Most frequently the healthy portion of the liver which is unaffected by the syphilitic deposit has undergone a compensatory hypertrophy.

These nodules vary greatly in number, from one to fifty or more, and may be found in any part of the gland, deep-seated or superficial, but they appear to have a predilection for the vicinity of the suspensory ligament, and that of the transverse fissure and along the course of the large branches of the portal vein. In size

they vary from that of a pea to that of a good sized plum, or even larger.

The outline of the liver affected with syphilitic gummata is more or less altered Scar-like depressions mark the surface, and these may be so numerous and deep as to divide the liver up into a cluster of irregular masses resembling, somewhat, a bunch of grapes ; or, the disease being confined to a single lobe, or part of a lobe, the portion affected may be entirely destroyed, the rest of the gland undergoing compensatory hypertrophy.

It is comparatively seldom that recently-formed syphilitic tumors of the liver come under observation. They appear as sharply localized, deeply reddened swollen patches, the portal venules of which have a gray lymph around them. Upon the margin of the new growths the progressive proliferation in the connective tissue may be traced along different broad streaks in the form of fine cellular infiltration.

In the later stage of the disease, the one usually met with, the nodules are pale yellowish, cheese-like, of irregular outline, surrounded by a fibrous zone, the outer edge of which fades off into the surrounding tissue, the lobules dwindling gradually in its grasp. The fibrous zone is never very broad, the deposit being strictly localised. The cheesy centre varies in consistence from a gristle-like toughness to a pulpy softness. Cretaceous deposits in the centre of the nodules have been observed by Wegner and Wronka in the fœtus at nine months.

Symptoms.—The effects produced upon the system by gummy syphilitic hepatitis are not very striking, because while one portion of the gland is being reduced by the disease, other parts become hypertrophied to compensate for the loss of secreting tissue. The diagnosis must be founded upon the occurrence of severe pain and tenderness in the hepatic region in connection with a history or other manifestations of constitutional syphilis. If the liver is accessible to palpation its surface will be found to be uneven or nodulated from the cicatrix-like depressions which form upon it, or from the projection of enlarged and softened gummata. The syphilitic cachexia often forms a marked feature in these cases.

The TREATMENT consists in rest, good diet, saline purgatives, and iodide of potassium. When anæmia is present in a marked degree, iodide of iron is the better remedy.

The consideration of AMYLOID DEGENERATION is reserved for another chapter.

HYPERTROPHIC CIRRHOSIS.

General Sclerosis of the Liver.

UNDER the above name has been described by several French writers a form of interstitial hepatitis, the distinctive pathological peculiarity of which is the circumstance that the reduction in the size of the liver which usually takes place owing to the shrinking of the newly-formed connective tissue which characterises the later stages of ordinary cirrhosis, does not supervene, even when the disease has lasted for a long time, but enlargement of the organ persists even until the fatal termination of the disease.

Etiology.—In common with ordinary cirrhosis, the abuse of alcoholic drinks is the cause of this disease. The reason why the proliferated connective tissue does not contract, as in ordinary cirrhosis, remains to be investigated.

Symptoms.—The earliest symptoms of hypertrophic cirrhosis are, pain and swelling in the hepatic region, recurring at intervals, and accompanied by jaundice, although bile is seldom, if ever, entirely absent from the motions. The liver, the lower margin of which is sharp, is found projecting a variable distance below the costal margin, it may be two or three inches, or on a level with the umbilicus, or even below it in the right papillary line. The spleen is invariably enlarged, sometimes greatly so; but the left lobe of the liver may extend across the abdomen as far as that organ, and thus cause the splenic dulness to become merged in

the hepatic. As a consequence of the enlargement of the liver and spleen, the upper abdomen projects forwards in both hypochondria. Symptoms of gastro-intestinal catarrh are inseparable from this disease, and usually set in early, and the nutrition of the body suffers sooner or later. Epistaxis frequently occurs early in the disease; hæmatemisis, on the other hand, is rarely observed. Ascites usually sets in as a sequence to anasarca, brought about by cachexia, and death is frequently ushered in by severe cerebral symptoms.

Pathological Anatomy.—The liver is found enlarged and increased in weight, its surface smooth or somewhat uneven from the projection of flattened irregular prominences of the parenchyma. The color of the organ throughout may be yellow or greenish-gray, intermingled with grayish-white spots or stripes. These stripes, which divide with difficulty, are in fact, new-formed connective tissue, in which no parenchymatous element can be detected.

The early appearance of jaundice in this variety of interstitial hepatitis is owing to the capillary bile-ducts being involved from the commencement of the disease in a state of catarrhal inflammation which persists, with slight fluctuations, throughout the entire course of the disease. From the walls of the ducts so affected the interstitial proliferation takes its start, instead of from the vicinity of the interlobular veins, as is the case in ordinary cirrhosis.

Duration.—The disease, in order to properly come under this head, could scarcely be of a shorter duration than one or two years. A case reported by Frerichs (Observation No. xix, vol. 2, p. 86) had a duration of six years; another case observed by Murchison lasted two or three years. (Diseases of Liver, 3rd edit., p. 249, et seq.)

Diagnosis.—From *ordinary cirrhosis* this disease can only be diagnosed by the fact of the enlargement of the liver persisting over a period of years, and by the presence of jaundice.

From *hydatid tumor* of the liver it can be diagnosed by noting the absence of fluctuation, and the existence of splenic enlargement and cachexia, neither of which mark the progress of hydatid tumor.

From *cancerous* enlargement of the liver this affection can be diagnosed by the absence of irregularities of the surface, and the presence of enlargement of the spleen, which latter is very rarely observed in connection with cancer of the liver.

From *amyloid degeneration*, hypertrophic cirrhosis is distinguished by the sharpness of the lower margin of the liver and the dissimilarity of the causes.

TREATMENT.—The treatment must be entirely symptomatic.

ACUTE YELLOW ATROPHY.

JAUNDICE associated with severe brain symptoms, often terminating fatally, was recognised even in the time of Hippocrates, but serious attention does not appear to have been directed to the pathology of the disease until the year 1834, when Dr. William Griffin of Limerick, published in the *Dublin Journal of Medical Science*, an account of four cases which he witnessed in members of one family. Since that time numerous cases have been reported in the various medical journals. It is to Busk, however, we are indebted for a distinct advance in the pathology of the disease, by demonstrating to Budd in 1844, the complete destruction of the hepatic cells in the softened portion of the atrophied liver. Further advance in our knowledge was made by Buhl in directing attention to the participation of the heart and kidneys in the degenerative process.

The train of morbid phenomena to which the name Acute Yellow Atrophy of the liver has been given is produced by some specific poison circulating in the blood and contaminating the whole system. What that poison is our knowledge does not suffice us to say. The fact that the same train of symptoms are occasionally observed as sequelæ to the specific fevers—typhus, enteric, yellow fever, scarlet fever, &c.,—renders it highly probable that a certain affinity exists between the poisons which produce all these diseases: a variation in intensity of the poison, and in the circumstances surrounding the patient, only being necessary to determine the variety of disease produced.

Poisoning by phosphorus produces all the symptoms, both clinical and pathological, of acute yellow atrophy of the liver. Indeed some authorities state that all cases of acute yellow atrophy are necessarily cases of phosphorus-poisoning; and Wagner and others believe that many of the cases recorded are of that character.

The jaundice which accompanies the general disease is such a prominent symptom as to lead to its consideration in all works treating on liver diseases.

Broadly stated, the disease is characterized by jaundice—rarely very intense--nervous symptoms setting in early, speedily followed by coma, convulsions and death. After death the liver being found friable, or rotten, or shrunken, and all the glands and muscles of the body are found to be in a state of parenchymatous degeneration : the nervous symptoms having been caused by degeneration of the vessels of the brain, interfering with the blood supply to that organ.

Acute Yellow Atrophy of the liver is one of the rarest diseases known to man, (Legg on the Bile, &c., p. 424,) yet Dr. Hanlon of Portarlington, attended three cases of it in members of one family, the notes of which he sent to the late Dr. Graves, of Dublin, (Clinical Lectures, 1864, p. 634.) and Dr. William Griffin, of Limerick, also attended four members of one family while suffering from it, two of whom recovered.

Etiology.—The causes of this disease are very obscure, and still await thorough investigation. Amongst the etiological factors, age appears to have some influence in predisposing to it ; most cases occurring in persons between the ages of 15 and 35. Sex also has a like influence. Out of 100 cases collected by Dr. Legg 69 were females. Pregnancy is a most important predisposing cause. Of the 69 females just mentioned 25 were pregnant, and three others recently delivered. The disease most commonly occurs from the third to the sixth month of pregnancy. Other predisposing causes are said to be indiscretion in diet, drunkenness, venereal excess, constitutional syphilis, mental

anxiety, fear, grief, malaria, the poison of typhus and allied diseases, and lastly phosphorus.

SYMPTOMS.—The premonitory symptoms of Acute Yellow Atrophy are most commonly those of gastro-intestinal catarrh, viz., furred tongue, nausea, and loss of appetite. occasional vomiting, and irregular bowels,—diarrhœa or constipation,—and slight pyrexia. These, it will be perceived, differ in no way from those which precede simple catarrhal jaundice. At other times the patient complains only of rheumatic pains, or of an uneasy sensation which he is unable to define. But in a great many cases there are no prodroma whatever; the patient not being aware of anything the matter with him until he percieves he is yellow.

When a patient, the subject of Acute Yellow Atrophy, first comes under the observation of the practitioner he is usually suffering from *jaundice*. This may be slight, and never become intense, but it is always present sooner or later and forms a prominent symptom of the disease. In some cases it is confined to the upper portion of the body. Neither the pulse nor the body temperature evince any marked change; they may be either normal or sub-normal. The urine is colored with bile pigment, and the fæces are devoid of bile, gray or white; or they may show a slight yellow color or be particolored.

The length of time during which the jaundice lasts before the nervous symptoms set in is liable to very great variation, being measured by hours in some cases and by weeks and even months in others. In pregnant women the disease runs a very rapid course.

As the disease advances, the area of hepatic dulness undergoes a marked diminution, while that of the spleen increases. In the course of a week or ten days, one third or even more than one half of the bulk of the liver may disappear.

Vomiting at one stage or another of the disease is present in about one half of the cases. If it occur early, the vomited matters may consist merely of food or bile, but towards the end

of the disease they are usually black from the presence of altered blood.

Tenderness on pressure in the region of the liver is commonly present, but pain is seldom complained of, the patient usually being comatose when this condition comes on.

It is not, however, until the nervous symptoms set in that the gravity of the disease is appreciated. The first of these to manifest itself is dilatation of the pupil; and when this symptom becomes apparent the medical attendant should be on the alert. Following quickly upon dilatation of the pupil are headache,—sometimes very violent,—rigors, extreme restlessness; and this condition is succeeded by low muttering delirium, tremors, subsultus, muscular rigidity and carphology, retention or incontinence of urine, involuntary passage of fæces, stupor, coma and convulsions. The pulse, previously slow, becomes accelerated on the accession of the nervous symptoms, and may be 140 or 150 per minute. The temperature is not liable to any very great variation in this disease unless an acute inflammation be present in some tissue of the body; otherwise it remains normal.

The nervous symptoms are said by some authorities to be due to uræmia, but it seems more probable that they are due to the changes which take place in the coats of the cerebral vessels.

In the delirium the patient is exceedingly violent for a short time, after which he relapses into the drowsy state in which he was previously. By degrees the coma becomes more and more profound without any delirium. The patient can no longer be awaked, and death soon follows.

The urine in this disease varies in color with the intensity of the jaundice, and is rarely very dark. The specific gravity ranges from 1015 to 1030. Early in the disease the amount passed per diem is neither above nor below normal, but towards the last it becomes scanty or entirely suppressed, the urea excreted, as a matter of course undergoing a concurrent diminution, but in its place two remarkable products of disintegration make their appearance, namely, Leucin and Tyrosin. The existence of these

two substances in the urine, Murchison believed to be pathognomonic of acute yellow atrophy, but the failure to find them must not exclude that disease from the diagnosis.

Hæmorrhages are very common in yellow atrophy, particularly from the stomach, bowels, or nose. Blood is often vomited in large quantity. Petechiæ, purpura spots and vibices often appear on the skin. After death, ecchymoses are found in different parts of the body.

Pathology.—On examining the body of a person who has died from Acute yellow Atrophy, the liver is found to be considerably diminished in size, often to the extent of one half or one third of its normal volume, its place being filled up by the intestines, so that on the abdomen being first opened the liver does not come into view. On taking out the liver it is found to preserve its natural form, but its consistence is remarkably soft and flabby; the investing capsule is thrown loosely into folds in consequence of the shrinking of its contents, and when placed upon its edge the gland doubles down upon itself, unable to sustain its own weight. The color of the organ on the outside is generally of a dirty greenish-clayey color, but the cut surface is bright yellow like moistened rhubarb, the outline of the lobules has entirely disappeared, the acini indistinctly separated from each other by a gray peripheral zone. Upon microscopical examination of sections of the diseased organ it will be observed that the hepatic cells are completely destroyed and are only represented by granular relics dispersed in the lax and flocculent remains of the capillary network, little of the tissue structure remaining to be seen.

Another condition of the liver frequently observed is, where the *yellow* soft spots alternate *red* and relatively firm and tough substance. The generally received opinion is that the red atrophy is merely a subsequent stage or chronic form of the yellow atrophy, the red atrophy always predominating in those cases in which the disease has run a long course. In the red atrophic portion the vascular network of the lobules is thickened much and shrunken, at the expense of the space which should contain the hepatic cells. In these spaces, small nucleated cells like epithelium are found

scattered; these appear to be degenerated hepatic cells which have lost their proper character without perishing entirely; but for the most part the spaces contain only granular relics.

Leucin and Tyrosin are frequently found in considerable quantity in the atrophic liver, particularly after exposure to the air for some time.

Besides the changes in the liver, the heart and other muscles generally have their fibres in a state of fatty degeneration. The kidneys are also loaded with fat and deeply jaundiced, and crystals of Leucin and Tyrosin are present in them. According to some authorities the coats of the blood vessels of the brain and of the body generally have undergone fatty degeneration also.

PROGNOSIS.—If, during the course of a simple jaundice, the nervous symptoms described above set in, the prognosis immediately becomes most unfavorable. The chance of the patient recovering is exceeding slight. Recovery has, however, taken place in exceptional cases even after delirium and severe hæmorrhages have set in and lasted for two or three days or even longer; (Legg on the Bile &c., p. 542).

TREATMENT.—It is obvious that during the first stage of the disease the remedies for gastric catarrh and catarrhal jaundice are the ones indicated; but when the grave nervous symptoms manifest themselves all treatment is futile; there is no remedy known that will stop the progress of the disease once commenced. Drastic purgatives appear to be the most promising of good results. After these have operated, Frerichs recommends the use of mineral acids and quinine, and Budd recommends alkalies. It must be confessed that the treatment is wholly empirical, and after administering a strong purgative, our greatest reliance must be placed in the *vis medicatrix naturæ*.

CHRONIC ATROPHY OF THE LIVER.

Brown Atrophy.

A SLOW general atrophy of the liver is occasionally observed as a concomitant symptom of the marasmatic process resulting from old age or chronic diseases of the digestive organs which interfere with the general nutrition of the body, or in connection with long-standing intermittent or remittent fever.

Pathology.—The liver is uniformly reduced in volume, with, perhaps, its anterior margin wasted to simply connective tissue. The entire organ has been known to weigh only 22 ounces. It is rather firm in consistence, owing to the preponderance of the connective after the destruction of the secretory tissue; but there is no increase of fibrous tissue within or without the organ; the capsule is thin, and the substance of the liver is dark brown or bluish-red in color. Microscopic examination shows the hepatic cells are reduced in size and loaded with brown granules. But the most important anatomical lesion is the destruction of the ramifications of the portal vein, the branches of which terminate in blind sacs, so that the organ cannot be minutely injected from the portal vein.

Symptoms.—A progressive diminution in the area of hepatic dulness, observed from month to month, together with a concurrent decline in the quantity of bile in the motions, without jaun-

dice, occurring in a person suffering from cancer of the stomach or intestines, point tolerably conclusively to brown atrophy of the liver. That the atrophy does not arise from cirrhosis may be assumed from the fact that the left lobe of the liver is not chiefly reduced in size.

No special treatment is indicated in this disease beyond checking the marasmatic process by appropriate diet and strengthening medicines.

PHOSPHORUS POISONING.

THE symptoms produced by Phosphorus when introduced into the system in poisonous doses, are very closely allied to those of Acute Yellow Atrophy of the liver, and Wagner and others maintain that all the cases of that disease reported are in fact cases of phosphorus poisoning, which after all may turn out to be true.

Poisoning by phosphorus has hitherto been very uncommon in this country, but is likely to become less rare in consequence of the prevailing fashion of prescribing and advertising phosphorus, not only in pills but also in so-called temperance drinks.

Phosphorus is a very common means of self destruction on the continent amongst the lower orders of life, but its odor and taste prevent it from being criminally employed as a poison, and render it easy of detection in articles of food.

Women far more commonly poison themselves with phosphorus than men, and it is during youth and adolescence that they do so. This fact is worthy of notice because the same circumstances of sex and age prevail in Acute Yellow Atrophy.

The dose of phosphorus sufficient to destroy life is from one to three grains. The method employed in Germany of taking the poison is to make an infusion in coffee of the heads of from 100 to 1000 lucifer matches, and to take the infusion by the mouth. Phosphorus paste, sold as rat poison is the usual form in which the substance is employed in this country. The red or allotropic phosphorus is not possessed of poisonous properties, and as some manufacturers of matches use that form of phosphorus, intending suicides have been disappointed in the result of taking it. This

is probably owing to its insolubility. Large solid pieces of common phosphorus may be given by the mouth, and passed by the anus without causing serious discomfort, owing to the same cause. It is when administered in a state of minute division or dissolved in oil that its full toxic effects are exerted.

Symptoms.—When a poisonous dose of Phosphorus has been taken, there may be no symptoms for several hours, or even two, three or four days. Sometimes, however, they come on sooner, even at the end of the first hour. In the first instance the patient experiences a disagreeable taste resembling that of garlic, which is peculiar to this poison, and an alliaceous or garlic odor may be perceived in the breath. There are burning pains in the epigastrium, sometimes also in the throat; thirst, nausea, and vomiting; the vomited matters, consisting of the contents of the stomach, have a garlic odor, and white vapors may be seen to proceed from them, which in the dark may be luminous. The breath may be phosphorescent from the retention of particles of the poison in the mouth or on the pharynx. At the end of from ten or twelve hours to two or three days the vomiting and pains in the stomach usually subside, but the medical attendant must not be deceived by this apparent improvement. It may possibly be that the whole of the poison has been rejected by vomiting, in which case recovery may take place, but a favorable prognosis cannot be founded on that contingency. At the end of three, four, or five days an exacerbation of the symptoms occurs, accompanied this time with jaundice, quickly followed by severe nervous symptoms. Pain and tenderness now are spread over the whole abdomen, and diarrhœa commonly occurs, the motions being either colorless or tinged with blood.

About the second day, and before the supervention of severe nervous symptoms, the liver, if carefully examined, will be found to be uniformly enlarged, and soon becomes tender. About the third day the sufferer becomes jaundiced, but this symptom may occur as early as fourteen hours after the ingestion of the poison, or may be delayed as late as the fourteenth or twenty-first day.

If the patient be going to recover, the jaundice and enlargement

of the liver gradually subside, and a general amelioration takes place in all the symptoms. Recovery may take place even after the occurrence of violent delirium.

After the jaundice has set in, nervous symptoms manifest themselves. These are somnolence, increasing to coma, broken by furious or maniacal delirium, often of an erotic character; carphology, dilated pupils; involuntary stools and urine are passed, and death soon closes the scene.

In some cases no nervous symptoms are manifested up to the moment of death, which may take place suddenly from collapse and cardiac paralysis, but more commonly the patient dies comatose from a gradual failure of the respiration and circulation.

There is usually a slight degree of fever in the system in phosphorus poisoning. The tongue is whitish or abnormally red; sometimes it is furred. The temperature ranges between 100·4° and 103° Fahr. until fatal collapse comes on when it falls to 97·7° or 95° Fahr. The pulse is somewhat increased in rapidity at the beginning, until jaundice comes on, when it falls to about 60 or 56. Later on it becomes feeble, small, thread-like in the fatal cases, but recovers its strength in those which recover. In some cases there is a complete absence of fever, or it comes on just previous to death. The glycogenic function of the liver is either greatly decreased or entirely suspended.

The urine, as usual in jaundice, contains bile pigment; it is also scanty and albuminous, and sometimes contains sugar. As death approaches the amount of urea falls very considerably, and in its place leucin and tyrosin appear. A very remarkable, and apparently constant constituent of the urine in phosphorus poisoning is scarco-lactic acid.

The blood is thin and seems to lose its power of coagulation. In women, fatal doses of phosphorus very commonly produce a bloody pseudo-menstrual discharge, and when pregnancy exists almost invariably induce abortion or miscarriage.

Dr. Legg says the bodies of persons poisoned by phosphorus show no signs of putrifaction but A. S. Taylor cites a case of

death from phosphorus in a girl, in which a tendency to rapid putrifaction was the only fact observed, an inspection of the body not being permitted.

The hæmorrhagic diathesis which is developed in phosphorus poisoning, is, in a great measure, due to fatty degeneration of the small arteries and capillaries; the altered state of the blood no doubt assisting.

PATHOLOGICAL ANATOMY.—The mucous membrane of the stomach and intestines is found thickened, opaque, whitish, grayish, or yellowish-white, the epithelial cells having undergone fatty degeneration. The liver is, as a rule, increased in size, friable, and light colored; sometimes it is mottled, and sometimes portions of it are deeply stained with bile. The cells are gorged with fat-globules, and in some cases hyperplasia of the interstitial tissue has taken place. In protracted cases the liver undergoes atrophy with destruction of its secreting cells, and it is in such cases that the characteristics of Acute Yellow Atrophy are seen. Except in these latter cases, the liver in phosphorus poisoning is characteristic of the well-marked "fatty liver." In the head, extravasations of blood are found in the meninges of the brain. The heart is found pale and flabby, mottled gray and yellow, having undergone complete fatty degeneration. The same change may be noticed in the voluntary muscles of the rest of the body. The spleen is often found enlarged to double its natural size, and its consistence increased; sometimes, however, it remains quite natural. The kidneys have undergone fatty degeneration. In short, acute parenchymatous degeneration of all the glands of the body is present.

The mode of production of jaundice in phosphorus poisoning, as in Acute Yellow Atrophy of the liver, is a moot-point amongst pathologists, but the most probable theory is that which attributes its production to catarrhal inflammation of the mucous membrane of the ducts. This may be limited to occlusion of the large ducts from the extension of the duodenal inflammation, or may only involve the finest bile-ducts high up in the liver. The lumen of

the ducts being thus obliterated, the bile pigment which is secreted by the still active hepatic cells is resorbed into the system and manifests itself in the urine, conjunctivæ, and skin.

It has been abundantly proved that phosphorus is absorbed into the system as such, by being dissolved in the various fatty matters contained in the alimentary canal, and by being rendered volatile by the heat of the body; the abundant vapors so produced passing readily through animal membranes. The ingestion of fatty matters always produces an aggravation of the symptoms in phosphorus poisoning, thererefore castor oil must not be administered as a purgative in these cases, as I have known it to be done. Milk also is highly objectionable.

Diagnosis.—If the patient be seen soon after taking the poison, phosphorus may be detected by the alliaceous odor of the breath or of the vomited matters, and by the luminosity of the latter in the dark. But if not seen until the first symptoms have passed away, and he be reticent as to the cause of his illness, the diagnosis will be beset with difficulties; there being no certain means of distinguishing during life, between phosphorus poisoning and acute yellow atrophy of the liver, or sub-acute poisoning by arsenic, antimony, alcohol, &c. After death, it is only in cases which have run a very rapid course that phosphorus can be found in the system: at the end of a few days it cannot be detected.

It is not always possible even after death to diagnose between phosphorus poisoning and acute yellow atropy, by the pathological lesions found in the body; but as a rule, in the former the liver has the appearance of a "fatty liver," uniformly yellow, the lobules well-marked and large, the cell-wall preserved, and filled with large oil drops. In yellow atrophy, on the contrary, the outlines of the lobules are lost, and the cells broken down, are found as granular relics in the lax and flocculent remains of the capillary network.

The pathological distinctions between phosphorus poisoning and yellow fever are very obscure.

Treatment.—When called to a case of phosphorus poisoning,

the indications for treatment are obviously, first, to remove as much as possible of the poison from the system; and, secondly to render inert such portions of it as may cling to the mucous membrane of the alimentary canal. The first indication is best fulfilled by the administration of emetics of sulphate of copper. This drug should always he chosen on account of the rapidity with which the metal unites with phosphorus, forming the phosphide of copper. It should be given in three grain doses in dilute solution, every five minutes until vomiting is induced. Oil of turpentine has been recommended by Andant as an antidote to phosphorus, but it appears to be only the crude French oil of turpentine which possesses antidotal properties. After emesis, the French oil of turpentine may be given freely in mucilage or an emulsion. If vomiting prove obstinate sulphate of copper with opium should be administered in such doses as the stomach will retain. Sulphate or citrate of magnesia should be used as a quickly acting purgative; and if pains be severe, hypodermic injections of morphia may give relief. Phosphorus being freely soluble in oils, all oily or fatty matters, milk, eggs, &c., should be sedulously withheld from the patient.

HYPERTROPHY OF THE LIVER.

BY hypertrophy is meant an increase in the size of the organ, dependent upon an increase in the size or the number of the hepatic cells without the addition of any adventitious matter.

The circumstances are various under which this increased growth of the secreting cells takes place. Thus, when the function of one portion of the gland—the left lobe, for instance—is destroyed by exudation processes, such as syphilitic hepatitis, or obliteration of one of the branches of the portal vein, the healthy portions of the gland augment in size and thus compensate for the spoilt portion.

In DIABETES MELLITUS a simple hypertrophy of the glandular parenchyma not infrequently occurs, where it is probably connected with an increased and accelerated formation of glycogen in the hepatic cells. The liver is uniformly enlarged, with the edges somewhat blunted.

In LEUKÆMIA the liver is not infrequently enlarged as well as the spleen and lymphatic glands. Its weight increases to four, six, or even ten or twelve pounds. Its consistence is either normal, or soft and flabby, but more frequently it is dense and firm. The organ is, as a rule, anæmic; more rarely hyperæmic. The lobules are usually large and prominent; the secreting cells are of large size; most of them contain several nuclei, and are filled with a quantity of fine granular matter.

In this form of hypertrophy, the functions of the liver are usually impaired; the secretion of bile is diminished, or almost entirely arrested.

Hypertrophy of the liver is said by some physicians to occur as a consequence of prolonged residence in tropical climates, but proof is wanting that the conditions observed by them were not due simply to hyperæmia or to fatty degeneration, rather than to an increase in the size or number of the hepatic cells.

CANCER OF THE LIVER.

CANCER is the most common serious organic disease of the liver met with in this country amongst temperate persons. It may occur independently of deposits in other parts of the body, in which case it is spoken of as *primary cancer;* or as a result of such deposits, the cancer cells having been conveyed by the blood from the original seat of the disease, to the liver, where they are arrested and set up morbid changes in its tissues: in this case it is spoken of as *secondary* or *metastatic cancer.*

The kinds of cancer that occur in the liver are chiefly CARCINOMA, MEDULLARY SARCOMA, occasionally EPITHELIAL CARCINOMA, and very rarely COLLOID.

Secondary cancer is much more common in the liver than primary; the proportion being about five to one.

Cancer of the liver usually takes the form of isolated tubercles or nodules imbedded in the hepatic parenchyma. Less frequently extensive portions of the hepatic tissue are infiltrated with cancerous matter, without any definite line of demarcation. The size of the nodules may vary from that of a millet-seed to that of an apple or even a child's head. Their form is usually rounded, it is only when they reach the surface of the liver that they appear flattened or umbilicated, the peritoneum over them being opaque and thickened from inflammation. There may be one or several of these nodules; sometimes they are distributed in large numbers throughout the hepatic tissue, on the outer surface as well as in the interior of the organ. A large part of the cancer-growth is found at and involving the surface of the liver. If you do not see cancer nodules on the surface of the liver, you are nearly certain

not to see them on section. The larger the size of the nodules the smaller is their number. They are usually numerous when the hepatic cancer is secondary to cancer in other organs, but primary cancer usually occurs as a solitary rounded tumor, which, however, may lead to secondary deposits in the surrounding hepatic tissue, or in the biliary passages.

If one of these cancerous nodules be cut across through its centre, it generally appears as a soft tumified mass, of a dirty white color, pressing above the plane of the incision, and bounded by hepatic tissue. Its surface presents a greater or less number of red dots and streaks, according to the degree of vascularity of the mass. On scraping the cut surface with a knife a creamy juice will be pressed out, which is always more abundant in proportion as the cancer is soft and medullary. After the juice has been squeezed out the meshes of the fibrous stroma of the cancerous tissue are distinctly visible.

A distinct line of demarcation in the form of a cyst very rarely exists between the cancerous growths and the hepatic tissue ; more frequently they mutually intermix in an imperceptible manner. Encysted cancerous tumors are always very soft and fluctuating, having much the feel of an abscess. When cut across and macerated, the pulpy matter is washed out, and a beautiful filamentous mass is left.

As the cancerous matter is deposited, the blood-vessels at the diseased places undergo important changes. In proportion to the hyperplasia of the interlobular tissue, the branches of the hepatic artery become more prominent, while those of the portal vein are to a corresponding degree diminished. Only isolated branches of the portal vein penetrate into the cancerous mass, but large branches of the hepatic artery penetrate its fibrous stroma. In the case of large cancerous tumors, or numerous smaller deposits, the trunk of the hepatic artery itself undergoes great augmentation in size.

As the cancerous matter continues to be deposited it may either form bold tumors that push aside the hepatic tissue, or it may invade the hepatic lobules which become swollen, softened, and paler

in color, and thus gradually assume the aspect of the mature cancer with which they soon coalesce. This latter form is described as *infiltration* cancer. An infiltration, however, in the sense of a pouring in of cancer among the healthy tissue is not to be seen here or anywhere, for the cancer is not added to the tissue, but composed of the transformed tissue itself.

The tumor continues to grow uninterruptedly at its periphery until the death of the patient. Meanwhile the older central portion always undergoes degenerative changes owing to the senescence of the cancer cells; hence this portion wastes, and becomes transformed into a dry compact, cheesy substance, of a pale grayish color: in this manner is brought about the umbilicated or cupped appearance always presented by the large cancer nodules that rise above the surface of the liver. Besides the natural senescence of the cancer cells, other influences are at work to produce the wasting and sinking changes in the middle of the cancer. The new growth rapidly breaks into the veins, especially the portal veins, producing cancerous thrombi. The blood supply is thus cut off, and as a consequence the mass is starved. The bile-ducts, also, are compressed and made impermeable by the new growth and the larger ducts are occasionally found to be filled with cancerous masses.

The liver affected with cancer is sometimes enormously enlarged, and has been known to attain more than seven times its normal weight, the increase in size taking place at the expense of the true liver tissue, of which only small patches may remain scattered throughout the mass. The flow of bile becomes obstructed in the normal portion of the liver as a consequence of compression of the bile-ducts, imparting to the gland a yellow, deepening into a greenish, hue.

CARCINOMA in the liver may be either of a medullary softness or of scirrhus hardness, according to the extent of development of the fibrous stroma, and the amount of cancer juice which it contains. It has the usual brain-like character and softness, and does not infiltrate the tissue, but forms large masses which thrust it aside. The softness caused by senescence of the cells in the

older central portion of these cancers is often extreme, so that they may appear like abscesses, and by this softening, blood-vessels may be opened, and hæmorrhage may occur into the interior of the cancer, giving rise to a remarkably rapid increase in its volume, and sometimes likewise to symptoms of anæmia. Smaller hæmorrhages of various ages accompanied with the usual changes in the effused blood, may give rise to different shades of brown and purple in the section. When the cancer bursts through the peritoneal envelope of the liver, hæmorrhages may ensue, which rapidly prove fatal.

Scirrhus of the liver is often of the infiltrating kind. The cells are few and soon degenerate, so that in the older central part, little more than interlaced fibrous tissue may be found, with fatty relics of cells; while in the outer parts of recent formation, the cells are more numerous and a reticulated appearance is evident to the naked eye. Very little cancer juice can be scraped from the surface of these tumors, and yet this kind is often very infectious. It is generally secondary to cancer of the breast.

Melano Sarcoma in the liver is almost always due to metastasis. The seat of the primary disease is most frequently in a mole on the skin, or in, or about the eye. It appears as a number of small nodules, or in the form of a widespread diffuse infiltration. The nodules vary in color, from black to brown or gray, yellow or white, they are irregular in form, and ill-defined in outline, and impart to the liver an appearance like that of granite. This form of cancer grows very rapidly.

Colloid cancer probably never occurs in the liver as a primary disease, but the gland is sometimes involved when the peritoneum is affected.

Etiology.—The etiological nature of cancer of the liver, like that of cancer of any other part of the body is entirely unknown. We are only acquainted with certain causative factors which accompany its development, and which it is customary to connect, although remotely, with the disease. The most important of these are—

(1) HERIDITY.—Professor Leichtenstern, with very great labor, collected statistics of 1127 cases of cancer, and of these 17% were hereditary. Such statistics are very fallaceous. In my experience patients are very reticent about their parents having suffered from cancer, besides they may not be aware of the fact. Prof. Paget found one fourth of his cases to be heriditary. Dr. Murchison, in the course of his practice knew of two sisters who died of cancer of the liver, in one instance within a fortnight, and in another within a few months of one another. In 1874 I attended a lady who died from cancer of the liver. In 1882 her daughter died of cancer of the stomach. Within the last four years I attended a father and son, the former of whom died of cancer of the rectum and the latter of cancer of the æsophagus. On the whole it appears to me that hereditary influence is very powerful in predisposing to cancer.

(2) AGE.—Cancer of the liver is much more common at an advanced period of life, especially in persons over 40 than in those under that age.

(3) GRIEF or ANXIETY.—Dr. Murchison states that in a large proportion of cases of cancer of the liver, the first symptoms of indisposition have been preceded by protracted grief or anxiety.

Cancer of the liver most frequently occurs as a secondary lesion, the primary seat of the disease being usually in those organs which empty their blood into the portal vein, viz., the stomach, the intestines, the peritoneum, and the pancreas.

SYMPTOMS.—When all the symptoms produced by hepatic cancer are fully developed the diagnosis is exceedingly simple. This, however, is not always the case, exceptionally, the disease runs a latent course, or is masked by some other disease (such as cancer of the stomach), which exclusively engrosses the attention. As a rule, however, a group of symptoms are developed in connection with cancer of the liver, which are sufficiently characteristic to enable us to have a clear insight of the changes taking place in that organ.

The earliest symptoms of cancer of the liver are; a person of middle or advanced age complains of loss of appetite, flatulence, constipation, distention and tenderness of the epigastrium, or right hypochondrium. After these symptoms have lasted for a variable period, or even before they are noticed at all, a swelling is discovered in the hepatic region, which is usually painful and tender upon pressure. The liver is found to be enlarged, sometimes reaching down to the umbilicus, or even to the brim of the pelvis; its surface and margins in exceptional cases are smooth, but in most cases an unevenness of its surface caused by cancerous nodules projecting above it, or even a palpable tumor can be distinguished through the abdominal walls. In some cases jaundice and ascites are present, or sometimes one and sometimes the other is observed. From time to time the symptoms undergo aggravation, the pains become more acute and extend towards the shoulder and hips; the abdominal walls become tense, vomiting, a short dry cough, and difficulty of breathing set in; the skin becomes hot and dry and the pulse accelerated. As the disease progresses the patient gradually looses flesh and strength, and generally becomes very anæmic; the spirits are depressed, and ultimately death from exhaustion supervenes.

Occasionally death takes place at an earlier period from some intercurrent disease, such as peritonitis, hæmorrhage, or dysentery, &c.

Analysis of the Symptoms.

(1) Enlargement of the Liver.—A nodulated, tender, enlargement of the liver is the most significant, and by far the most constant of all the symptoms of cancer of the organ. The enlargement may be moderate in extent, but frequently it attains to huge proportions; 15lbs., 20lbs., and 25lbs. have been reported as the weights of cancerous livers. The lower margin of the liver is not thick and blunt as in simple enlargement, but a more or less

wavy outline can usually be demonstrated by percussion, and perceived by palpation, and it is no uncommon circumstance for the cancerous nodules to be visible through the abdominal walls as uneven tumors which participate in the movements of respiration. In some cases a sensation of friction can be felt by placing the hand over the liver and directing the patient to take a full inspiration. With the aid of the stethoscope a friction sound can also be heard in these cases.

The degree of tenderness in the organ varies very greatly in different cases, according as the growth of the new formation is rapid or slow, and according to the presence or absence of inflammation in the surrounding peritoneum.

(2) JAUNDICE occurs in about one-half of all the cases of hepatic cancer, and when it comes on it continues permanently until the death of the patient. It results either from compression of the ducts by the cancerous mass, or from obstruction of their lumen from within.

(3) ASCITES is observed in more than one-half of all the cases of hepatic cancer. It is a consequence of stasis in the portal circulation brought about by compression or obstruction of the trunk or large branches of the portal vein by cancerous matter, or by the contraction of inflammatory products in the hepato-duodenal ligament, produced by the irritation of the cancerous growth.

(4) THE SPLEEN is very seldom enlarged in connection with cancer of the liver, a circumstance which constitutes an important distinction of the cancerous from the waxy and cirrhotic liver.

(5) DIGESTION.—Disturbance of the functions of the stomach and bowels in consequence of the abnormal distribution of the blood supply in the gastro-intestinal tract is of very frequent occurrence. Even at a very early period of the disease the patient complains of nausea, flatulence, loss of appetite, constipation, distension of the abdomen after meals. When the disease has lasted some time the stools are deficient in bile, and are clay-colored.

The urine is invariably scanty and high colored, and the presence of bile pigment in it can usually be demonstrated by Gmelin's test.

(6) Respiration.—The breathing is occasionally embarrassed as a consequence of the upward dislocation of the diaphragm by an enlarged liver, or by ascites. Pleurisy on the right side, or the diaphragm becoming involved in the cancerous degeneration of the liver, will have a like effect.

(7) Cachexia.—Before cancer of the liver has lasted long, the phenomena of the cancerous cachexia present themselves in a marked degree. The patient becomes anæmic, emaciated, and if jaundice be not present the color of the skin becomes pale and earth-colored like that of chlorotic persons, the feet become œdematous, and flesh and strength rapidly decline.

It would be wrong to suppose that the mere presence of cancer in the body is capable of producing these grave changes in nutrition; on the contrary, I have known women who have suffered from scirrhus of the breast for a great number of years whose complexions appeared the picture of health, and whose body-fat had not suffered the least diminution. But when cancer, by reason of its discharges or development in an important organ like the liver, interferes with the functions of digestion and assimilation, and thus undermines the vital powers, then the sufferer presents a pale waxy anæmic appearance, differing in no important point from the man worn out by exhausting discharges from a diseased bone or joint.

Diagnosis.—It is not difficult to recognise cancer of the liver from the foregoing symptoms when the liver can be felt; but in those rare cases where the liver is not enlarged, matters are different. Here, however, persistent tenderness upon pressure in the region of the liver, commencing ascites, and the absence of any other cause to account for the cachexia may lead to a suspicion of cancer of the liver.

The following are the diseases of the liver which are most frequently confounded with cancer:—

(1) AMYLOID DEGENERATION OF THE LIVER.—The smooth infiltration form of cancer may be mistaken for amyloid degeneration of the liver. In both there is a smooth, uniform, very hard enlargement of the liver; but in the amyloid enlargement the progress of the disease is slow, there is an absence of pain or of cachexia, and there is usually also enlargement of the spleen, with albuminuria, and a history of a definite exciting cause, such as caries or necrosis of bone, or a protracted discharge from a suppurating surface, or constitutional syphilis.

A mistake is far more often liable to be made in the case of cirrhosis and amyloid liver combined; which, like the cancerous, has a hard nodulated surface, and is also sometimes painful. In most cases of cirrhosis, however, the nodules are much smaller, and tenderness upon pressure is only periodically present as a result of inflammation. These circumstances together with the distinguishing signs of amyloid liver will suffice to establish the diagnosis.

(2) SYPHILITIC HEPATITIS with projecting gummata or lobulated by deep cicatrices may be mistaken for cancer. In both affections there may be a large nodulated tender liver, with jaundice, ascites, and severe pain; but the syphilitic disease may often be distinguished by the comparatively early age of the patient, the previous history, and the existence of other evidence of syphilis.

(3) MULTILOCULAR HYDATID TUMORS of the liver may present many of the clinical characters of cancer; but the constitution does not suffer to anything like the same degree; moreover, fluctuation is distinctly perceived in most cases, the tumors are rounded and globular, either completely painless or only occasionally the seat of pain, and their progress is more chronic than that of cancer.

(4) OCCLUSION AND DILATATION of the bile-ducts may be mistaken for cancer of the liver: for differential diagnosis see page 67.

DURATION AND PROGNOSIS.—The impossibility of determining accurately the date of the commencement of the cancer deposit

in the liver renders it impossible to estimate the duration of the disease. If we compute from the commencement of dyspeptic symptoms we are very liable to fall into great errors. After the development of undoubted symptoms, the probable duration of the patient's life may be estimated by the size and rapidity of growth of the tumor, and the degree to which the constitution has suffered from it. Medullary cancer may prove fatal at the end of from four to eight weeks: on the other hand, life may be prolonged over years with scirrhus. In every form of cancer, however, the prognosis is fatal.

TREATMENT.—There is no known remedy which can arrest the progress of, or eliminate from the system, internal cancer, unless Prof. Clay has discovered that remedy in Chian turpentine, which the profession appear to doubt. The treatment of cancer of the liver must therefore be entirely paliative. Dr. Budd wisely remarks—"Practitioners have, indeed, hoped to destroy cancerous tumors by some powerful alterative, or if not to destroy them, at least to retard their growth. Various powerful medicines—alkalies, mercury, arsenic, iodine—have been tried in turn with this view, and all—it is almost needless to remark—have signally failed. They aggravated suffering and hastened death, by adding their own noxious effects to those of the malady; but there is no evidence that they have, even in the slightest degree, retarded the growth or prevented the multiplication of the tumors."

The routine practice of administering "calomel" in every form of hepatic disease has done more injury in cancer than in any other disease of the liver.

The treatment must be directed to the relief of pain, the subdual of local inflammation, the correction of errors in digestion, and the maintenance of the patient's strength and nutrition.

The diet must consist of milk, eggs, meat, especially scraped raw beef mixed with cream, which is very digestible; good strong wine will also be necessary.

Any inflammation of the peritoneum must be kept in check by warm fomentations, poultices, &c.; the local abstraction of blood by leeches would only serve to weaken the patient.

The digestive disturbances will be corrected in a measure by regulating the diet and still further by the administration of quinine, iron, bitter extracts, dissolved in aromatic infusions and combined with ant-acids. Bismuth, hydrocyanic acid, lime water, nux vomica, or ice, will relieve vomiting. Flatulence may be relieved by charcoal, creosote, or carbolic acid, all of which have the power of correcting putrefactive fermentation. Constipation is best relieved by rhubarb, aloes, or simple enemata. If bile be absent from the bowels choleate of soda, as recommended in Occlusion of the Bile-ducts, must be resorted to. For the relief of sleeplessness, pain and pruritus of the skin, opium and chloral hydrate are the most appropriate remedies.

AMYLOID DEGENERATION OF THE LIVER.

AMYLOID DEGENERATION of the liver is merely a local expression of a general cachexia, brought about by certain chronic diseases which profoundly disturb the general nutrition of the body, and moreover, the spleen, kidneys, lymphatic glands, and digestive tract always participate in the degenerative process.

This disease has received a great many appellations. Those who were influenced by the physical characters of the diseased organ named it either *lardaceous liver* on account of the peculiar sensation communicated to the knife when cutting it, like cutting raw bacon; or *waxy liver* from its resemblance to wax. Graves and Budd who observed the frequency with which the disease occurs in connection with scorfulous diseases, especially of the bones, spoke of the disease as *scrofulous enlargement* of the liver. Virchow, to whom we are indebted for our knowledge of the nature of the adventitious matter deposited in the liver, introduced the term *amylaceous* degeneration for the condition present, and that name has been adopted by all recent writers on the subject.

In the liver, amyloid degeneration consist in the fact that a substance foreign to the healthy organism, namely the amyloid substance, is deposited in the parenchyma of the organ. This substance, no person has ever yet succeeded in isolating, because, in the first instance, it is not a free deposit which can be gathered out of the tissues by mechanical means, but is deposited in the

muscle cells of the small arteries, and in the walls of the capillaries; and secondly, no solvent of the substance has yet been found; in fact it is remarkably resistant to chemical influences, and especially also to decomposition. Almost the only thing that is known about the amyloid substance is its behavior in the presence of iodine and sulphuric acid, a watery solution of iodine causing it to assume a mahogany-red color, which on the addition of sulphuric acid, changes to a blue or violet. The same changes in color are produced in amyloid matter by using iodine or chloride of zinc or chloride of calcium. Iodide of methyl-aniline will color amyloid substance of a bright ruby red or violet red, while the tissues free from the substance are colored of a pale blue by the reagent.

The behavior of the substance when treated with iodine and sulphuric acid lead Virchow to apply to it the term *amyloid*, under the impression that it belonged to the class of vegetable carbohydrates, but against this opinion it may be stated that no one has ever yet been able to convert amyloid substance into sugar or alcohol, which can easily be effected in the case of vegetable starch and allied substances. Kekulé and Schmidt believed that amyloid substance was closely allied to the albumenoid principles, but it differs in various respects from this class of substances, especially in the fact of its insolubility in fluids containing pepsine.

How or where the amyloid substance originates in the system is equally veiled in mystery with the nature of the substance. As it has never been found in the blood, it seems probable that it must originate, through some perversion of nutrition, at the very place it is deposited, not improbably by transformation of the fibrin of the blood into amyloid matter. Why the substance selects the liver, spleen, kidneys, lymphatic glands, and digestive tract for its habitat, remains clouded in obscurity.

There is no analogy between the amyloid matter and scrofulous or tubercular products, because, the latter consist of new-formed cellular elements, whereas in the former there are no new cells, but a new morbid substance is deposited within the existing elements of the tissues.

ETIOLOGY.—Amyloid degeneration of the liver never takes place unless the nutrition of the body has been profoundly disturbed by some antecedent chronic ailment which has brought the system into a cachectic condition.

Among the diseases which may lead to the development of amyloid liver, are the following.

(1) CONSTITUTIONAL SYPHILIS.—This is doubtless a more fertile source of amyloid degeneration than all the other causes put together. Scrofula has very justly, according to their views, been credited with a foremost place in the etiology of amyloid degeneration by several authors, but it seems to me that there are very good grounds for believing scrofula to be neither more nor less than hereditary syphilis, therefore any argument to sustain the relative potency of scrofula and syphilis as etiological factors of this disease would be but playing at cross-purposes with words. Viewed in this light, syphilis will be found to be almost the one great cause of amyloid degeneration, not only when the bones are diseased, but even when the osseous system is quite uninvolved.

Although the syphilitic virus alone is capable of producing amyloid degeneration, this result follows with greater certainty when it has set up disease in the bones or soft parts, accompanied by protracted suppuration. Newly-born children suffering from congenital syphilis have been found by Gubler, Rokitansky, and others, with amyloid degeneration of the liver.

(2) PROTRACTED SUPPURATION.—In so-called scrofulous individuals, prolonged suppuration and ulceration in the bones and joints in connection with caries of the vertebræ, and necrosis of the long bones, often gives rise to the development of amyloid degeneration, and the same result is likely to follow traumatic affections of the bones, and also simple ulceration of the soft parts, such as ulcer of the leg, provided they give rise to protracted suppuration.

(3) CHRONIC PHTHISIS, such as is known by the name "fibroid," is a frequent cause of amyloid degeneration, because

here we generally find wide-spread ulceration, not only in the lungs, but also in the intestines, the larynx, and the trachea.

(4) Sex.—Amyloid degeneration is much more common among males than among females. This difference is remarkable, because, as Frerichs says, the diseases which predispose to the hepatic affection, by no means exhibit any predilection for males.

(5) Age.—Amongst a total of 116 cases of amyloid degeneration collected by Frerichs and Wagner, the disease was developed between the ages of 20 and 50 years in no less than 92 cases.

Pathological Anatomy.—When amyloid degeneration of the liver has reached a very high grade and is unassociated with other morbid processes, the organ is found enlarged to a variable degree, sometimes almost filling the whole abdomen, and its weight is also proportionally increased. The enlargement is uniform in every direction, so that the form of the organ is not essentially altered, but the amyloid liver has a tendency to mould itself over adjacent organs which it touches, thus the left lobe may encapsulate the spleen, and the posterior part of the right lobe may have a very deep depression for the kidney. The serous covering undergoes no change, but remains smooth, transparent, tense, and free from morbid adhesions. The anterior free edge of the liver retains its sharpness, unless, as is usually the case, fat is also present, when it becomes blunt and rounded. Instead of feeling soft, the liver is of a peculiar doughy firmness, and appears very anæmic, being of a peculiar light grayish or yellowish gray color, sometimes with an admixture of red. On cutting into it a peculiar sensation is communicated to the knife, like cutting raw bacon, hence the name *lardaceous*. The cut surface is remarkable for the high degree of anæmia which it presents; only the larger vessels giving escape to a certain amount of thin blood or serum; otherwise the tissue seems dry, almost bloodless, of a very pale grayish yellow or dirty grayish brown color, and a characteristic dull, waxy lustre. The tissues cut firm, so that very thin sections may be taken off with ease, and if one of these be held to the light it will be found of an almost glassy transparency, which is

only interrupted by the opacity of the lobules, which latter are remarkably well defined in the amyloid liver. On applying an aqueous solution of iodine to the section, the translucent portion is observed to assume a reddish brown hue, which on the careful addition of sulphuric acid, gives place to a dirty violet, or more rarely, to a blue tint.

In the slighter grades of amyloid degeneration the size of the liver is little altered, and its physical characters differ somewhat from that depicted above; but in proportion to the degree of degeneration present, the organ will always present a more or less increased consistency to the touch, anæmia, paleness of color, waxy lustre of the cut surface, and translucency of the infiltrated tissue.

The very slightest grades of amyloid degeneration are not recognizable by the naked eye, but require the aid of the microscope, together with iodine and sulphuric acid, for their detection.

Symptoms.— Amyloid degeneration of the liver may generally be recognised at the bed-side by the following circumstances:— The liver is found to be uniformly enlarged, perhaps greatly enlarged, by the area of hepatic dulness being increased in the median, papillary, axillary, and scapular lines. The enlargement takes place both upwards into the thorax and downwards into the abdomen. On palpation, the portion of the liver which extends below the costal margin is found to be smooth, dense, firm, and resisting, and almost of wooden hardness. The lower margin is more rounded than normal, and is quite free from indentations, provided no other morbid processes have altered the surface of the organ. Pain and tenderness are usually absent from the enlarged liver, and even tolerably hard pressure upon it is borne with impunity. At most the patient is merely annoyed by a feeling of fulness and pressure in the right hypochondrium. In rare cases, acute pain comes on in consequence of perihepatitis, excited by the syphilitic virus. The growth of the liver is slow and imperceptible, and often extends over several years.

The constitutional symptoms of amyloid degeneration are those of a high grade of cachexia. The patient is emaciated, anæmic,

of a pale complexion, and prone to dropsical effusions. The symptoms naturally vary in individual cases, according to the nature of the primary disease which gave origin to the amyloid liver, as well as according to the extent to which other organs, such as the spleen, kidneys, and intestines participate in the degenerative process.

Jaundice very rarely occurs in connection with amyloid degeneration of the liver, and when it does occur it is in consequence of pressure exercised upon the bile-ducts by large lymphatic glands.

Contrary to what might be expected from the amyloid degeneration of the capillaries, and consequent narrowing of their lumen acting as an impediment to the circulation of blood through the liver, ascites is not a prominent symptom of this disease. In the higher grades of the disease, however, it is liable to be developed in consequence of profound anæmia, intercurrent peritonitis, or complicity of the kidneys in the amyloid degeneration.

Enlargement of the spleen is frequently associated with amyloid liver, and this enlargement generally, but not invariably, depends upon amyloid degeneration of its tissues. Occasionally the spleen is of normal dimensions or even shrunken, and under either of these circumstances it may also be the seat of amyloid deposit.

As the swelling of the hepatic tissue crowds aside the hepatic cells, thus leading to their atrophy, it is not surprising that a diminished production of bile should become manifest by pale gray or colorless stools, tympanites, and the escape of foul smelling wind from the bowels.

In many cases of amyloid liver the functions of the stomach and intestines become disturbed. The appetite ceases, and vomiting, with a clean tongue takes place from time to time; diarrhœa, with pale mucous stools, ensues, without any obvious cause, and persists obstinately for a long period, or returns from time to time, with short intermissions. The cause of the diarrhœa lies in anatomical lesions of the intestinal mucous membrane which is either the seat of amyloid degeneration or of extensive ulceration. Such lesions of the mucous membrane by impeding

digestion and absorption, as well as by the profuse secretion to which they give rise, naturally tend to aggravate the already existing anæmia and hasten the marasmatic process.

When, as frequently occurs, the kidneys are involved in the amyloid degeneration, the urine becomes albuminous, and towards the termination of life the quantity of urine secreted becomes diminished.

DIAGNOSIS.—When the disease is fully developed the diagnosis is generally not difficult if the physical characters of the amyloid liver, and the circumstances under which it occurs are borne in mind.

DURATION.—The disease always pursues a very tedious course, and may extend over many months. Once it has begun, the degenerative process almost always advances steadily to a fatal termination, which usually takes place from general exhaustion or some intercurrent disease, pneumonia, purulent peritonitis, œdema of the lungs, &c.

PROGNOSIS—Some excellent authorities (Graves, Budd, Frerichs, Murchison,) have expressed the opinion that recently infiltrated amyloid matter may be removed by appropriate treatment. But unfortunately, when the disease is still recent the diagnosis is very uncertain, and Frerichs admits that the removal of the hepatic enlargement does not always lead to recovery.

In most cases the prognosis is unfavorable, and the more certainly so the farther the disease has advanced. When the kidneys and the mucous membrane of the intestines participate in the degeneration, the case invariably terminates in death.

TREATMENT.—When amyloid degeneration of the liver is in a sufficiently advanced stage to be diagnosticated, all treatment is of little avail. But remembering the diseases which notoriously bring amyloid degeneration in their train, much may be done to prevent the rise of the latter by abreviating the course of the former. Thus, diseases of the bones, and ulcers in soft parts,

associated with profuse suppuration must be arrested by appropriate treatment or a surgical operation if necessary. Dr. S. Ringer speaks highly of the influence of the sulphides of calcium, potassium, &c., in arresting suppuration. He administers the former drug in doses varying from one-tenth of a grain to one grain, according to the age of the patient, several times a day, or even every two hours. The drug must be persistently administered for several weeks in refractory cases.

Constitutional syphilis in particular, must, if possible, be eradicated from the system by iodide of potassium, or iodide of iron. In the mean time the general nutrition of the body must receive great attention; for, by keeping that up to a proper standard cachexia is postponed, and anæmia is prevented, and the probability of amyloid degeneration supervening is greatly diminished.

When amyloid degeneration of the liver is actually present, an attempt may be made to restore the tissues. This will be accomplished with most probability by those means which tend to improve the general nutrition. The diet should be nourishing and easy of digestion; consisting largely of meat, with a moderate amount of alcoholic stimulants, If there be a history, or any indication of syphilis, iodide of potassium and the syrup of the iodide of iron afford the greatest chance of benefiting the patient. Murchison highly extols tincture of iodine of the British Pharmacopœia in doses of ten or fifteen minims three or four times a day.

The carbonate and the chloride of ammonia have been successfully employed by Budd in reducing enlargement of the liver, and W. Bigbie has had a like experience from the administration of the latter salt in doses of from fifteen to thirty grains three times a day.

Further treatment must be essentially symptomatic; diarrhœa must be combated with opium in combination with vegetable or mineral astringents. Persistent vomiting must be treated with ice, bismuth, hydrocyanic acid, and sinapisms to the epigastrium. Warm baths are indicated when albuminuria is present, in order

to stimulate the secretion of the skin. Dropsy must be met by diaphoretics and diuretics. Finally, if the patient be in a condition to undertake a journey, great advantage may be derived from drinking the sulphurous waters of Harrogate, Berèges, &c.

FATTY LIVER.

IN its normal condition, the liver only contains about two or three per cent. of fat, but that quantity may, under certain circumstances, be so greatly exceeded as to render the liver easy of combustion by merely holding a portion of it in the flame of a lamp for a sufficient length of time to drive out the water. Between these two conditions it is impossible to state accurately where health ends and disease begins. It will not only depend upon the quantity of fat present in any particular liver, but also still more upon the cause and accompanying circumstances of its accumulation there, whether we are to regard the fatty liver as a pathological condition or otherwise. Many fishes are remarkable for livers abounding in fat, and in some hybernating animals a quantity of fat accumulates in the liver during autumn which is gradually used up during the winter sleep. It frequently occurs also that persons who, in the bloom of health, have met their death by violence, present at the autopsy, remarkably fatty livers. It therefore must be conceded that the liver is a natural reservoir for fat taken as food, but not immediately required in the economy of the system.

Certain individuals, and even certain families evince a remarkable tendency to the formation of fat, which is at first deposited in the subcutaneous cellular and other tissues of the body, and subsequently in the secreting cells of the liver. Besides the hereditary influence at work in such persons the tendency to corpulence is still further favored by the diminished secretion of bile causing a decreased demand for fat in its formation, by indolent and sedentary habits, and by over-indulgence in food rich in

carbo-hydrates. Among the agencies of a more general nature which favor this condition of body are, middle age, female sex, and a temperate humid climate. Occurring under such circumstances as the foregoing, fatty liver may be considered a transient affection, which may, to a great extent, be removed by a change of habits, diet, &c.

But above and beyond the condition just stated, there is a fatty degeneration of the liver which is undoubtedly a pathological condition. This is witnessed in its most pronounced form in acute phosphorus poisoning.

In the human liver, fat accumulates in the interior of the parenchymal cells on the periphery of the lobules. With the aid of the microscope cells may usually be seen in various stages of fatty degeneration. Small flat globules in some, large ones in others, and finally, cells entirely filled with oil.

Fatty liver is very frequently observed in connection with certain diseases of the general system which do not directly implicate the liver. This is notably the case in phthisis, cancerous cachexia, caries and necrosis of bones, and other wasting diseases, and also in connection with the drunkard's dyscrasia. Occurring under these circumstances, the disease is known by the name of the "cachectic fatty liver."

Fatty liver occurs with great frequency in phthisical patients, and those suffering from wasting discharges, because the high temperature which accompanies hectic fever in such cases, renders the body fat more fluid than normal, and therefore more readily taken up by the blood-vessels. By these the fat is conveyed to the liver where it is arrested by the hepatic cells in virtue of their well-known affinity for that substance. The further detention of fat in the liver is favored by the paucity of oxygen in the portal blood, and by the enforced inactivity of the respiratory and muscular systems acting as impediments to its combustion in the system at large.

The higher grade of fatty liver met with in pulmonary tubercle than in other wasting diseases is to be accounted for by the de-

ficient supply of oxygen in the system for the combustion of fat consequent on the diminished functional activity of the lungs; whereas in the latter diseases the lungs usually remain unimpaired.

But that the mere diminished activity of the lungs present in phthisis is not adequate to account fully for the accumulation of fat in the liver in connection with that disease is shown by the fact that in such diseases as emphysema, valvular disease of the heart, stenosis of the large bronchi, &c., in which the respiration is equally impeded, fatty liver does not occur.

The fat deposited in the liver being to a great extent the expression of the amount of fat drawn from the system at large, it follows that the disease assumes a higher grade in those persons in whom the adipose tissue is more highly developed. For this reason fatty liver is more common in phthisical females than in males.

In habitual drinkers in whom the drunkard's dyscrasia is developed the blood becomes loaded with fat, and a milky turbid serum separates from it (lipæmia). From the blood the fat is deposited in the liver, and as a consequence fatty liver reaches a high grade; and concurrently with this process, hyperplasia of the connective tissue of the liver may take place, leading to cirrhotic fatty liver. When we take into account the small amount of solid food which such persons usually consume it seems probable that the accumulation of fat in their blood takes place, not in consequence of increased fat formation but in consequence of diminished fat combustion, resulting from the presence of alcohol in the system.

In acute phosphorus poisoning fatty liver is a marked feature, but under such circumstances the process is one of fatty transformation of the hepatic cells, rather than fatty accumulation from the general system.

From what has been stated it appears that fatty liver is met with under four diverse conditions. First, from individual proclivity a larger amount of fat is produced in the system than can be combusted; the surplus is deposited in the tissues of the body, and, when the serum of the blood becomes milky from its presence

(lipæmia), in the liver also. Second, a substance (alcohol) in the system checks fat metamorphosis; the fat circulating in the blood becomes arrested in the liver. Third, the fat in the cellular tissue becoming liquified in consequence of morbid processes going on in the system, is taken up by the blood-vessels and conveyed to the liver; and fourth, a poison (phosphorus) in the system transforms the albumen of the liver cells into fat,—true fatty degeneration.

PATHOLOGICAL ANATOMY.—In a well-marked case, the fatty liver is observed to be greatly enlarged; it may be to double its normal dimensions, and although the weight of the organ is also increased, its specific gravity is lowered, so that in extreme cases it may even float when placed in water. As the deposit of fat takes place uniformly throughout the entire organ, the external form of the liver is not altered to any great extent, except that its edges are blunted or more rounded than usual. The color of the gland is paler, and its substance softer, and more greasy than natural. These peculiarities of the fatty liver are best observed while the organ is yet warm, because on cooling the fatty deposit "sets," and thus offers more resistance to the knife. On cutting into the fatty liver a layer of grease clings to the knife, or to the hands which touch the cut surface. The cut surface presents variable appearances in the different grades of fatty degeneration. In the highest grades the lobular structure of the parenchyma is obliterated, and the cut surface presents a uniform pale yellow or grayish white, shining, fatty appearance. In the lower grades of fatty liver the lobular structure remains intact, and the lobules are observed of a liver-brown or red color, surrounded by a zone of pale yellow, or whitish-gray fatty infiltration. Sometimes contiguous lobules are connected by bands of capillary vessels, and then the cut surface presents the appearance known as the "nutmeg liver."

The above description only applies to typical cases of fatty liver. Lesser degrees are constantly met with. Thus, the liver may not be enlarged, or may even be smaller than normal. The latter is especially true in cases of cirrhotic fatty liver. A correct judg-

ment cannot always be formed with regard to the fatty contents of a liver merely from its gross physical characters. In cases of doubt a piece of the suspected liver may be placed in a tube with some hot ether and allowed to digest for a few moments. Then pour out the contents of the tube on to a plate, when, if fat be present it will solidify on cooling. But the best test is the microscope, for by this means the quantity of fatty matter present can be estimated with great exactitude. The oil globules are observed on the periphery of the lobules as a dark margin around each lobule. It is only in the very highest grades of fatty liver that the cells in the centre of the lobules contain fat.

Symptoms.—The symptoms of fatty liver are vague and ill-defined. Constitutional disturbance may be entirely absent, or if present, clearly traceable to antecedent pathological conditions leading to the affection, or to accidental complications. The diagnosis will be greatly assisted by remembering the conditions under which fatty liver occurs. Thus, when we find in a phthisical female, enlargement of the liver, unaccompanied by pain, jaundice, and ascites, we may safely conclude that she is the subject of fatty liver. The same conditions occurring in great eaters, inebriates, and those suffering from wasting discharges, point to a like conclusion.

When favorable conditions present themselves for a physical examination, such as a high grade of the disease, together with thin, relaxed abdominal walls, the fatty liver has the following clinical characters :—The area of hepatic dulness is greatly augmented, and its inferior margin may be on a line with the umbilicus. By palpation it is perceived that the liver is uniformly enlarged and painless, with a smooth, soft, doughy consistence, and an obtuse margin. There is neither jaundice nor ascites, and the superficial veins of the abdomen are not varicosed. The spleen, moreover, usually retains its normal dimensions.

The constitutional symptoms which accompany fatty liver are in general attributable to diseases which underlie the same. They are, loss of appetite, flatulence, irritability of the bowels,—now constipated, and on the least provocation profuse and exhausting

diarrhœa sets in, accompanied at times by hæmorrhages from the rectum. Debility and anæmia are also present in a marked degree, and the skin presents a bloodless, almost semi-transparent, and waxy appearance.

TREATMENT.—When fatty liver depends upon gross feeding and indolent habits, an opposite mode of life will diminish the amount of fat in the body generally and also in the liver. The patient must rise early and take plenty of open air exercise. The diet must be regulated so as to consist of lean meat, white fish, bread, green vegetables, with plenty of salt at meals; avoiding all substances rich in starch, sugar or fat. Alcoholic drinks, especially beer and sweet wines must be forbidden, the drink being restricted to water, tea without milk or sugar, and a small quantity of claret or hock. By these means the patient will not only get rid of much of his fat, but will also be better nourished and his strength increased.

If, however, the patient evince any manifestation of fatty heart, such as feeble cardiac impulse, faint cardiac sounds, weak pulse, attacks of vertigo or syncope, dyspnœa, &c., great caution will be necessary in applying the foregoing regimen. In such cases it must not be resorted to too suddenly, and its operation must be carefully watched in order to avoid all dangerous accidents on the part of the heart. The withdrawal of alcoholic stimulants in particular will need great caution when the heart is weak.

In order to correct any digestive derangements which may be present, recourse must be had to alkalies, either alone or in combination with bitter infusions, such as taraxacum or gentian. The waters of Carlsbad, Marienbad, Vichy, Kissengen, Homburg, &c., enjoy a reputation for reducing fatty liver; but when there is a marked tendency to diarrhœa it will be best to avoid all the waters just mentioned, and to employ in their place those of Eger or Ems. When anæmia is a marked feature in the case, preparations of iron, dialysed iron, or the chalybeate waters of Tunbridge, Spa, Schwalbach, &c., give excellent results.

If the bowels be torpid, aloes, rhubarb, or colocynth must be

employed; while on the other hand, a tendency to diarrhœa must be met by astringents.

When fatty liver occurs in the course of phthisis or other wasting disease, no direct treatment will be called for, but its presence must be regarded as contraindicating the use of cod-liver oil.

MELANÆMIC LIVER.

Pigment Liver.

THAT black substances are formed in the spleen and in the blood of the portal vein, which may give rise to disease was a tenet of the humoral school more than 2,000 years ago. Galen thought that this so-called atra-biliary matter accumulated in the spleen, as a product of the formation of bile, from which locality it gave rise to obstructions of the vessels, enlargement of the abdominal viscera, and dangerous nervous symptoms.

This theory held its own in explaining all abdominal affections generally, and those of the portal system in particular until the end of last century, when Reil showed how the doctrine of black bile was opposed to physiological observations. Notwithstanding the protestations of Reil, the majority of physicians continued to believe in the pathogenetic potency of black bile, and even in the early part of the present century the marsh fevers of the tropics were regarded as atra-biliary fevers.

Scarcely had scientific medicine removed this remnant of the humoral pathology, when facts came to light which rendered it necessary to fall back upon the old doctrine. Numerous cases were observed in which black matter produced by decomposition of the blood accumulated in the spleen, from whence it was carried into the portal vein. The black matter at one time obstructed the hepatic vessels, and at another passed through these, and, entering the general circulation, filled up the capillaries of the brain, and of other organs, giving rise to symptoms similar to those described by the ancients.

A graphic description of such a case is given by Dr. Bright in his "Medical Reports," published in 1831, although he was quite ignorant of the pathology of the affection.

To H. Meckel is due the honor of having, in 1837, discovered that the dark color of the organs depended upon an accumulation of pigment in the blood,—melanæmia.

The essential character of melanæmia is the presence of black pigment-matter in solid form in the blood. The pigment is found both free and enclosed in cellular bodies of various forms. These cellular bodies are the colorless blood corpuscles which take up the free pigment in the blood, and thus become pigment-carrying cells. When these corpuscles leave the blood-vessels, they carry the pigment with them into the tissues surrounding the vessels where it may remain deposited for a long time.

In the liver, the pigment lies principally in the interior of the blood-vessels, and partly outside of them, in the interstitial connective tissue, leaving the parenchymal cells of the organ quite free. The amount of pigment matter accumulated in the capillaries is in some cases so considerable as to cause serious impediment to the circulation of the blood in the liver.

The cause of melanæmia lies in repeated attacks of marsh fever. With every paroxysm of intermittent or remittent fever a large number of red blood-corpuscles are destroyed, setting free the coloring matter, which separates in solid form and circulates with the blood. The coarser particles of the pigment matter lodge in the liver, in the peripheral zone of the lobules, and the finer particles pass through the liver and are found in various parts of the body, especially in the lungs, brain and kidneys.

The place of origin of the pigment matter is not at present definitely settled, but there are good grounds for believing that the blood of the splenic vein and of the portal vein furnish the largest proportion of it. The blood of these vessels is very much richer in pigment than that of any other part of the vascular system.

Pathological Anatomy.—When the disease is of recent origin the melanæmic liver is of normal dimensions or enlarged

hyperæmic, softened and friable, and of a dusky brown color. In more chronic cases the organ is diminished in size, and of a steel-gray, blackish, or chocolate color. This coloration is either uniform, or the brownish lobules are seen surrounded by a black border, owing to the interlobular veins being filled with pigment; in general, however, the pigment matter is more uniformly distributed, extending from the circumference of the lobules halfway to their centre, or pervading their entire capillary structure, the peripheral hepatic cells remaining quite free from the melanæmic pigment, while those in the centre of the lobules have suffered atrophy from compression by the overloaded capillaries.

The symptoms due to involvement of the liver in melanæmia are not at all definite, and as treatment would naturally be directed to the cause of the disease, intermittent fever, it will be perceived that pigment liver is important in a pathological, rather than a clinical point of view.

ECHINOCOCCUS

OR

HYDATID TUMORS OF THE LIVER.

NATURAL HISTORY.—Tænia echinococcus is a small tape worm which infests the dog and wolf, its special haunts being in the duodenum and upper portion of the small intestine. It seldom exceeds a quarter of an inch in length, and develops four segments including that of the head, the final segment, equalling in length the three anterior ones, contains about 5,000 eggs. These eggs while still within the body of the parent develop into six-hooked embryos, the latter still retaining the egg covering. When this segment of the entozoon has arrived at a stage of maturity, it is now about to undergo passive migration, and having detached itself from the strobila is soon expelled from the bowel of the host. In its free condition the proglottis moves about for a time, but the growth of the embryos within its interior causes it sooner or later to burst, and thus the embryos become scattered in every direction. These embryos are introduced into the human body by the individual partaking of water or food into which the eggs have been carried by accident or otherwise; then having got rid of the egg-covering, with an especial predilection for the liver, the embryos bore their way into that organ, or are carried by the circulation into other organs. In these situations they sooner or later become transformed into simple vesicular bladder-like bodies, commonly called *hydatids*.

Thus far the larval development strictly conforms to that ordinarily observed in cestode worms, but from this period a series of

changes occur which are not only unique in themselves, but on many accounts are deserving of the serious attention of the physician and pathologist. Instead of displaying the ordinary cysticercal form, characterised by the head. neck, body, &c., the vesicle retains throughout its life changes a more or less globular or oval figure. In the earliest stages at which it has been observed, the juvenile hydatid is perfectly spherical, and is invariably surrounded by a capsule of condensed tissue derived from the organ in which it is situated.

In the human liver only a single hydatid is usually developed, but occasionally two or more are found in the same organ. In the sheep and other animals on the contrary, multiple hydatids are usually met with. The capsule which surrounds the entozoon, and which is usually called the *cyst* is abundantly supplied with arborescent branches of the hepatic artery and vena portæ. Within this cyst, but having no connection with it, is a gelatinous, transparent, gray bladder, composed of numerous concentric hyaline layers,—the so-called mother sac of the echinococcus, that is to say, the embryo which has increased to a remarkable extent. This sac displays a peculiar tremulous motion, and curls back the reverse way of its natural curve wherever there is a free cut margin. Within the sac is a clear, transparent, colorless fluid with numerous large and small vesicles floating loosely in it, or adherent to the wall of the mother sac. These so-called daughter vesicles vary in size from a millet-seed to a goose-egg; their number not infrequently amounting to several hundreds, or even thousands. The large daughter vesicles sometimes contain smaller vesicles of a third generation. and occasionally the latter, in their turn, contain others of a fourth generation. On closer examination, a number of delicate white particles may be observed upon the inner surface of the sac, and sometimes upon the secondary sacs also. These minute bodies, which are usually aggregated in groups, when examined under the miscroscope are seen to be enclosed in a delicate membrane. They are the scolices of the Tænia echinococcus in various stages of development. By a process of inversion the heads are withdrawn, as it were, into the cavity of the brood-capsules, and thus the latter display a

birds-nest-like appearance. Frequently, however, a considerable number of them are seen everted, displaying the echinococcus heads. At this stage of its existence the entozoon is from 1-20th to 1-6th of a line in length, has a head similar to that of the Tænia, furnished with a rostellum and four suckers. The rostellum supports a double crown of hooks, the number of which is about twenty-four. The head of the scolex is separated from the body by a grove, and at its posterior extremity presents an umbilical depression, which gives insertion to a cord by means of which the scolex is attached to the inner surface of the sac. The general aspect of the body is finely granulated, and in addition presents a number of highly refracting calcareous corpuscles, which more particularly abound at the parieties and inferior part of the scolex.

In order to complete the life-history of Tænia echinococcus, it may be stated that it is only through hydatid offal being devoured by dogs and wolves that the entozoon is enabled to enter upon its strobila conditon, and thus complete the cycle of its metamorphoses.

Other forms of hydatids are occasionally observed in the liver besides that just described. Thus, the scolices may grow from the inner surface of the mother sac, and there may be no daughter vesicles present. The daughter and grand-daughter vesicles are in fact scolices differentiated in order to form secondary brood capsules. Hydatids are also occasionally met with which contain no scolices at all; these are called acephalocysts, and are in fact abortive specimens.

Pathology.—Hydatids may be found in any part of the liver, in the right or left lobe, on its upper or under surface, buried deeply in the substance of the gland as well as projecting from its surface or margins.

The liver which is the seat of an hydatid growth may present various alterations in form and size. The tumor may increase to such an extent as to fill the greater portion of the abdominal cavity and also of the right side of the thorax. As the animal

grows, so the hepatic tissue becomes compressed and atrophied to form a wall around it. When the hydatid is situated near the surface of the liver and protrudes, the hepatic cyst is protruded also, thus appearing as if it were formed around the hydatid by some independent process of its own, but this is really not so. The remaining portion of the gland in most cases retains its normal appearance, or is compressed and thickened: occasionally a compensatory hypertrophy takes place in it.

When an hydatid tumor has lasted for a long time the external hepatic capsule often becomes ossified, under which circumstance the contained hydatids die because the obstruction can no longer be overcome. Then, instead of floating in the fluid of the parent all are found collapsed and dried up, and a quantity of fatty or putty-like matter is found amongst them, in which a quantity of bile pigment is not infrequently present; the intrusion of bile, not improbably, having caused the death and retrograde metamorphosis of the hydatid.

Termination.—In a large proportion of the cases of hydatid of the liver the animal is killed, and in consequence rendered harmless, by a communication becoming established between the mother sac and a small bile-duct; the bile acting as a poison to the animal. In the case of a large bile-duct becoming perforated the termination is almost always fatal from permanent occlusion of the common bile-duct or from pyæmia.

The most usual termination of an hydatid is to cease to grow, die, and shrivel up. If, however, the tumor continue to grow the tendency is for it to ultimately burst and discharge its contents. This may take place in any of the following directions, namely:—into the right pleura, the pericardium, lungs, bronchi, stomach, colon, peritoneum, or externally through the abdominal walls. Of these the perforation into the cavity of the chest is of most frequent occurrence. When the contents of the cyst are discharged into the pleura, acute pleurisy, which almost invariably terminates fatally, is the result. When adhesive inflammation between the tumor and the lower lobe of the right lung, with perforation of the diaphragm have preceded bursting of the hydatid,

the contents of the latter excavate a large cavity in the lung which may ultimately communicate with one of the bronchi, and thus lead to the discharge of the contents by expectoration. In this manner a cure may take place, but in most cases death from various causes ensues.

In exceptional cases the hydatid contents find their way into the pericardium, which event is quickly followed by pericarditis and death.

Rupture into the peritoneum is an extremely fatal occurrence, which frequently takes place from a blow, fall, or strain. Death usually ensues in a few hours, but may be delayed for some days.

Discharge of the hydatid by rupture into the stomach or intestines is a very favorable event, although not entirely free from danger, because secondary abscesses of the liver may form and destroy life. On the whole a relatively larger number of recoveries take place after discharge in this direction than in any other.

It is not a very common mode of termination for an hydatid to discharge through the abdominal parieties or the intercostal spaces, and even when it does occur life may be placed in jeopardy by the supervention of suppuration in the cyst or in the abdominal walls, or by hæmorrhage, peritonitis, &c.

Several cases are reported in which hydatids opened into the inferior vena cava, and mingled their contents with the blood. In such cases the vesicles are carried to the right side of the heart, from which they are propelled into the pulmonary artery and become impacted in its branches, leading to instant death by asphyxia.

Rupture into the biliary passages will be found treated of under the head of "Entozoa of the Biliary Passages." Page 85.

Symptoms.—It frequently happens that hydatids are found *post mortem* in the liver, which during life never betrayed their existence by any symptoms whatever. Indeed the tumor may attain considerable proportions without causing any pain or uneasiness which might draw the patient's attention to it. In most cases, however, local changes take place which indicate beyond a doubt

the existence of the hydatid. The first symptoms usually complained of are a feeling of weight and fulness in the side, some gastric disturbance and dyspnœa. On making a physical examination of the liver, that organ may be found to be greatly enlarged, but the enlargement is not uniform, it usually follows one direction in particular, so that the ordinary form of the gland is lost. If the tumor project upwards, the natural arched outline of the superior margin of hepatic dulness will be exaggerated; if it project downwards the inferior margin of dulness will be found lower down than normal at certain points where palpation reveals a distinct protuberance or tumor. The specific characters of hydatid tumors are as follows:—pain is usually absent from them, and they can be handled freely without eliciting tenderness; they are smooth, globular, elastic, and frequently fluctuating, not dense or doughy. Occasionally what is known as hydatid vibration can be felt in the tumor. This sign is elicited by placing three extended fingers firmly over the most prominent part of the tumor, and percussing the middle one, when a peculiar tremor is felt which is said by some authorities to be due to the secondary cysts in the interior striking the wall of the parent. This sign is not invariably present, and Frerichs failed to find it in more than one half of his cases.

Jaundice and ascites are rare accompaniments of hydatid tumor of the liver. The former, when present, is due to pressure on the common bile-duct; and the latter arises in consequence of compression of the portal vein or of the inferior vena cava.

Diagnosis.—When an hydatid tumor of the liver has attained such proportions that it can be readily seen and felt, there is seldom any difficulty in recognising its nature. The mere fact that a large globular, fluctuating tumor connected with the liver, has grown slowly, unattended by pain, fever, jaundice, ascites, or constitutional disturbance, is almost evidence enough that the tumor is an hydatid.

The following are the diseases most likely to be confounded with hydatid tumors of the liver.

(1) Abscess in the Liver.—This disease can usually be recognised by the previous history. The persons most likely to be the subjects of liver abscess are those who have resided in the tropics. Besides, the tumor is more rapidly developed, and is accompanied by pain and fever; cachexia also makes its appearance early in the disease.

(2) Cancer of the Liver is distinguished by its more rapid growth, irregular surface, tenderness and hardness, and by the absence of elasticity and fluctuation, and the presence of cancerous cachexia, and probably also of cancer in other parts of the body.

(3) Dropsy of the Gall-Bladder, or Hydrops Cystidis Felleæ may simulate a pendulous hydatid, but may usually be diagnosed by its being preceded by jaundice and attacks of colic. Besides the situation of an hydatid rarely corresponds to that of the gall-bladder, and hydatid vibration is absent.

(4) Aneurisms of the Aorta and hepatic artery may present smooth globular tumors, very like hydatids; but their outline is different, being more or less spindle-shaped; besides they are the seat of pulsation, and often of acute pain.

(5) Pleuritic Exudations.—Hydatids which mount up into the thorax are very liable to be confounded with pleuritic exudations, because in all these diseases we find dyspnœa, dulness on percussion, absence of respiratory murmur and of vocal fremitus, and ultimately bulging of the intercostal spaces. But the hydatid may be distinguished by carefully marking the upper line of dulness through its entire extent, which will be found to be arched upwards in this disease whereas in pleuritic exudations this line is horizontal and changes with the position of the patient.

(6) Hydronephrosis.—A renal cyst may be distinguished from an hydatid of the liver by the absence of respiratory displacement, the presence of the colon in front of it, and by its situation and direction of growth.

(7) Localized Ascites in the neighborhood of the liver may be recognized by the concurrence usually of general ascites and other

symptoms of hepatic disease. The diagnosis will be assisted by the withdrawal by means of an aspirator of a small quantity of the fluid, which, in the case of ascites will contain albumen, but in the case of an hydatid cyst will be found strongly impregnated with common salt, and the microscope will probably reveal echinococcus hooks in it.

PROGNOSIS.—As long as an hydatid of the liver continues to grow it is a standing menace to life, and, moreover, the means at our disposal for inducing retrograde metamorphosis in the cyst are uncertain in their effect, and not infrequently endanger life.

TREATMENT.—No drug taken by the mouth has the slightest influence on the vitality of an hydatid. The only way to get rid of the tumor is by the speedy evacuation of its contents. There are various ways of attaining this end, but it is only necessary to refer to two of them. 1st By puncture with a fine trocar; and 2nd by opening and emptying the sac. The dangers of any operative interference with the tumor are mainly these; first, an escape of the fluid into the peritoneum which would inevitably set up fatal peritonitis; and, secondly, suppuration of the cyst owing to the admission of air into it.

The escape of hydatid fluid into the peritoneum may be prevented, when puncture with a small trocar is resorted to, by removing only a portion of the fluid of a large cyst, and by making pressure over the site of puncture while in the act of withdrawing the trocar. The danger from admission of air must be obviated, no matter the mode of operating, by the use of the antiseptic spray.

Previous to evacuating the cyst by incision it is absolutely necessary that adhesion exists between the opposed surfaces of the peritoneum over the tumor. This is best insured by Simon's method. He introduces a small trocar and withdraws about a couple of ounces of the fluid which he utilizes for the purpose of confirming the diagnosis. Then three more trocars are introduced at a distance of about half an inch apart. These trocars are left in position until fluid escapes alongside the canulas. An amount

of fluid similar to that which was withdrawn at first is removed through the canulus daily, and at about the end of the fifth day, suppuration having set in, the tumor may be laid open by uniting the punctures lying farthest from each other by successive strokes of the knife.

ASCITES.

AN accumulation of fluid in the peritoneal cavity called *ascites*, is so common a complication of diseases in and about the liver that it is desirable to devote a few pages to its consideration. The name ascites is derived from the Greek ἀσκός a leathern bag, which the ancients used to contain fluids of various kinds. The disease is also called abdominal dropsy to distinguish it from general dropsy, (called also œdema or anasarca), in which the fluid is effused in the subcutaneous cellular tissue.

ETIOLOGY.—Effusion of fluid in the cavity of the peritoneum is produced either by arrest of absorption, preternatural secretion, absorption remaining normal, or by transudation of serum from the veins of the abdominal viscera, absorption being abolished.

In the normal condition a very large quantity of serous fluid is secreted by the visceral and parietal peritoneum which has the effect of a lubricant on the abdominal viscera, allowing them to glide freely upon each other, without any attendant attrition. This fluid secretion never exceeds a slight moisture upon the surface of the peritoneum, because absorption and secretion are exactly balanced. But in the diseased condition absorption may be defective, or even entirely arrested; or secretion getting the ascendancy; or an unusual transudation of serum taking place from the veins of the chylopoietic viscera,—the same result must follow in each case, namely, an accumulation of fluid in the peritoneal cavity.

Many cases of dropsy are met with, not only in the peritoneum, but also in the pleura, tunica vaginalis, and other serous cavities,

which can only be attributed to defect or arrest of the normal absorption process, or a hypersecretion from the healthy membrane, because the serous membrane itself presents nothing abnormal to our senses, nor can any pathological process be discovered outside of it adequate to account for the effusion: and finally, when the fluid has once or twice been removed by tapping, or otherwise, it does not re-accumulate.

When an inflammatory process is present in the peritoneum, fluid is effused into its cavity because the blood-vessels in an inflamed part are more pervious, and allow fluid to pass through them more readily than healthy blood-vessels will do.

Independent of inflammation, the most common cause of ascites is some impediment to the current of blood returning from the abdominal viscera to the heart. This may be considered under two heads: (1)—Direct mechanical obstruction affecting the portal circulation; and (2)—Cardiac and pulmonary diseases affecting the general circulation.

Direct mechanical obstruction may affect the portal circulation either within or without the liver. Within the liver the disease which most commonly produces this effect is cirrhosis; the contraction of the abnormally proliferated interlobular connective tissue obliterating numerous capillary branches of the portal vein, and so cutting off the connection between the veins of the abdominal viscera and the right side of the heart. The effects of this are, hyperæmia of the tributaries of the portal vein, with transudation of the fluid portions of the blood into the peritoneal cavity and arrest of absorption.

The same results follow obstruction to the trunk of the portal vein outside the liver, or obstruction of the inferior vena cava at its junction with the hepatic veins or within the thorax. In the case of the trunk of the portal vein the most common causes of its obstruction are tumors, growing either in the hepato-duodenal ligament, from the under surface of the liver or from a neighboring organ; chronic peritonitis, or pylethrombosis. Mechanical obstruction of the hepatic veins or inferior vena cava is rarely met

with, but may arise from the pressure of a tumor in connection with the liver or some neighboring organ (aortic aneurisms, cancerous tumors of the retroperitoneal glands), as well as extensive exudations in the left pleural cavity.

Diseases of the heart and lungs which impede the veinous circulation produce stasis of blood in the portal system and so lead to ascites. Anasarca of the lower extremities, however, usually precedes the abdominal dropsy in these cases, the latter symptom being most likely to arise when persistent hyperæmia has developed grave structural changes in the liver,—the Ramose Atrophy of some authors.

Inflammatory processes in the peritoneum are very liable to be attended with more or less ascites; the blood-vessels of the inflamed part allowing a freer transudation of fluid through their walls than healthy ones will do. Chronic peritonitis, whether it be a sequence of the acute form or otherwise, is especially liable to be associated with ascites, and here the operation of paracentesis is very prone to favor the reaccumulation of the ascitic fluid. Local peritonitis, caused either by cancerous or other tumors, tubercular deposits or spirit drinking, as in some cases of hepato-peritonitis, may give rise to ascites, and in the case of the latter disease it may not always be possible, during life, to differentiate it from that produced by cirrhosis of the liver.

Of diseases of the general system, leukæmia is frequently associated with ascites, which latter may reach a high grade if the enlarged spleen compress the trunk of the portal vein. Extreme anæmia and general debility likewise tend to the production of ascites.

Amongst the causes of a more general character which may produce ascites are, exposure to cold or wet, and the suppression of profuse habitual discharges.

The ascitic fluid varies very much in its physical characters. Usually it is thin and watery, colorless, or of a slight yellow tint, clear and transparent, and alkaline in reaction. It may, however, be colored by blood or bile, or rendered turbid by the admixture

of inflammatory products, pus or lymph. Albumen is usually present in large quantity. The specific gravity of the fluid is usually about 1015, but is subject to great variation in this respect.

Signs and Symptoms.

The signs whereby fluid in the abdomen is recognised are, (1) Enlargement or swelling; (2) Dulness on percussion; and (3) Fluctuation.

Ascitic Enlargement of the abdomen is, in its higher grades, very characteristic. It is uniform, and the fluid evinces a tendency to gravitate towards the most dependent parts,—the flanks when the patient is in the recumbent posture,—the inguinal regions when he is standing; and it may even be seen to move when the posture is changed. The skin of the abdomen is tense, smooth and shining, and may present lineæ albicantes due to laceration of its deeper structures. The umbilicus is either stretched and obliterated or protruded more or less considerably, and if a hernia be present at any part of the abdominal wall, its sac is distended with the ascitic fluid. Enlarged and tortuous veins ramifying over the abdomen and converging towards the umbilicus are very significant of ascites due to portal and caval obstruction. The chest appears small in comparison with the enlarged abdomen, and the lower ribs and xiphoid cartilage may become everted through the outward pressure of the fluid. The abdomen feels perfectly smooth and even over its entire surface, and the sensation conveyed to the hand when placed upon it is one of tension rather than hardness.

When a small quantity of fluid is effused into the peritoneal cavity its presence may be difficult to detect. A dull percussion sound and obscure sensation of fluctuation in the flanks or in-

guinal regions may usually be elicited by skilful hands. By placing the patient on his hands and knees, however, the fluid is caused to gravitate towards the umbilicus where it may readily be detected by the dull percussion note.

PERCUSSION.—A dull sound is elicited on percussion over the seat of fluid in the abdomen because the fluid has usurped the place of the tympanitic intestines, which by reason of their lightness float forward. By degrees as more and more fluid accumulates the tympanitic bowel sound is superceded by a dull sound in all the regions of the abdomen except the umbilical, which only becomes dull in the very highest grade of ascites.

As the fluid obeys the laws of gravity, the upper margin of dulness necessarily varies with the position of the patient. Thus, when he lies on his right side this margin is towards his left and *vice versa*, and when he lies with his shoulders elevated the dull margin will usually be found slightly below the umbilicus with a clear tympanitic circular space above it.

In some cases the intestines are bound to the abdominal walls by old adhesions which prevent their floating in the ascitic fluid, and so cause a tympanitic sound to be heard in an unusual position. The possibility of this occurrence must always be borne in mind when forming a diagnosis in obscure cases.

FLUCTUATION is a peculiar wave-like sensation which is communicated to the hand placed upon the abdomen while percussion is made with the fingers of the other, due to motion produced in a fluid in a pent-up cavity. This sign is always best marked when the quantity of fluid in the peritoneal cavity is great; with the walls of the abdomen thin and tense. For the reason already mentioned, change of posture necessarily influences the situation in which fluctuation can be elicited.

It sometimes happens, when the abdomen is tense and distended from another cause than fluid in the peritoneal cavity, that a deceptive sense of fluctuation is elicited, due to vibrations of the abdominal walls. In order to eliminate this source of error

it is necessary that the hand of an assistant should be placed edgewise along the abdomen midway between the hands of the examiner, so as to check the transmission of superficial vibrations.

Besides the above-mentioned ordinary means of eliciting fluctuation, it may also be felt by the finger introduced, into the rectum in the male, or the vagina in the female; and this means of dignosis may with advantage be resorted to in obscure cases. The fluid collects in the recto-vesical space in the male, and in the cul-de-sac between the vagina and rectum in the female. If fluid be present in great quantity, the vagina will be found shortened, and the uterus depressed and flexed. In extreme cases of ascitic distension of the abdomen, the posterior wall of the vagina, or even the uterus itself may be extruded through the vulva.

The symptoms of ascites of a more general character may be divided into (1) those due to the system at large; and (2), those due to the mechanical effects of the fluid accumulation.

The premonitory symptoms of ascites, if any, are of course those of the various diseases which conduce to its development. Sometimes, however, these diseases are very insidious in their incursions, and then it may not be until the second class of symptoms are developed that the patient seeks advice, or that the gravity of his illness becomes apparent. These remarks apply with especial force to the three great causes of ascites, namely, cirrhosis of the liver; chronic peritonitis; and obstruction of the portal vein, slowly developed.

When ascites has lasted for some time, the loss to the system of a large quantity of fluid holding in solution a great proportion of albumen necessarily produces more or less well-marked effects upon the functions of the body. Thus, the skin becomes dry; the bowels are usually constipated, but diarrhœa or dysentery may set in; and debility, anæmia, and wasting of the body progress rapidly, especially if the the fluid be removed by paracentesis.

When the quantity of fluid in the peritoneal cavity is great, its mechanical effects become apparent; a feeling of uneasiness and distension in the abdomen is experienced; and the action of the diaphragm and abdominal muscles being interfered with, more or

less dyspnœa and thoracic breathing are produced. These are most distressing when the patient is in the recumbent posture and after taking food. The action of the heart is also liable to be interfered with, which becomes manifest by palpitation, irregularity, or a tendency to syncope. The patient is obliged to stand or walk with the head thrown back in order to balance the body, as is constantly observed in the advanced stage of pregnancy. When the abdominal distension is excessive, as in cases of very long standing, the patient is unable to stand erect, but moves about with the body bent forward. This I observed in two cases of dropsy from obliteration of the portal vein. The pressure of the fluid on the renal veins and inferior cava may prevent the return of blood through those veins and give rise to albuminurea, scantiness of urine, anasarca of the lower extremities, and enlargement of the superficial veins of the abdomen.

Diagnosis.—By the application of the foregoing means of physical examination it is usually not difficult to determine the existence of fluid in the abdomen, but it is always necessary to go a step farther and to ascertain whether the fluid be free in the abdomen or contained in a distinct cyst.

The diseases which are especially liable to be confounded with ascites are the following; viz. :—

(1)—Flatulence together with a fat and flabby condition of the abdominal walls; but here the tympanitic bowel sound can be elicited in its usual area.

(2)—An Ovarian Cyst.—Obviously this disease is only liable to be confounded with ascites in the female sex. So long as the cyst is small and its outline distinct through the abdominal parieties the diagnosis is easy enough. And so it is also in the case of multilocular cysts by reason of the uneven surface they present on palpation, their greater hardness and resistance to pressure, and the obscure sensation of fluctuation which can be elicited from them. But when a single ovarian cyst is so large as to fill the abdominal cavity, the nature of the disease may be

recognised by the following characters;—the tympanitic bowel sound, if any, is found in the flanks, or in the epigastrium, while the umbilical region gives a dull sound, and the relative position of the clear and dull areas do not vary with the posture of the patient, and the umbilicus, though it may be obliterated, is never distended so as to form a distinct protuberance, except when the disease is complicated with ascites.

The history of the case usually furnishes valuable evidence of the nature of the disease; thus, with ovarian cysts the swelling commences in one or other flank, and for a long time is located more on one side of the abdomen than on the other, and at this period can usually be traced as a distinct tumor taking its origin in the pelvis; and when large it appears to bulge forwards rather than laterally.

(3)—An Hydatid Tumor of the liver, when very large might be mistaken for free ascites, but may be distingished by the swelling having commenced at the upper part and one side of the abdomen before it became general, and by the tympanitic portion of the abdomen not always being at the highest point in every posture the patient is placed in.

(4)—Hydronephrosis.—A renal cyst which has attained very large proportions might be mistaken for ascites, but may usually be distinguished by the tumor being limited to one side, and by the effect of change of posture, hydronephrosis being much less influenced thereby than ascites.

(5)—An Accumulation of Urine in the bladder may simulate ascites when it has reached such a grade as to fill a great part of the abdomen, and the diagnosis is further obscured if the patient assure his medical attendant that he passes fair quantities of normal urine daily, which in fact is frequently the case. Some of our most accomplished surgeons have fallen into the error of tapping a distended urinary bladder under the impression that the fluid was free in the peritoneum, and I need scarcely say the results have been most disastrous to their patients. Such a

mistake is only liable to occur in the case of an old man, the subject of enlargement of the prostate; but this may be obviated by a very simple precautionary measure, namely, by the introduction of a catheter for the purpose of ascertaining the condition of the bladder. This precaution should always be adopted previous to performing paracentesis, especially in elderly men, and for such persons a long prostatic catheter should be employed.

In addition to the above-mentioned diagnostic distinctions valuable evidence may be furnished of the nature of the fluid accumulation by the withdrawal of a small quantity of the fluid by means of the aspirator, or even an ordinary hypodermic syringe, for the purpose of chemical examination. The fluid thus withdrawn will be found in the case of simple ascites to be serous, of about 1015 specific gravity, and containing a large quantity of albumen. This albumen is of the ordinary character,—coagulates by heat and will not re-dissolve in double its volume of boiling acetic acid. The albumen contained in the fluid from an ovarian cyst, on the other hand, known as metalbumen and paralbumen, coagulates by heat but is re-dissolved, or converted into a gelatiniform liquid by boiling in double its volume of strong acetic acid. The fluid from a hydronephrosis or a distended urinary bladder will contain urea, while that from an hydatid cyst will be found devoid of urea or albumen but strongly impregnated with common salt, and the microscope will probably reveal echinococcus hooks in it.

Diagnosis of the causes of Ascites.—Having assured ourselves of the fact of the presence of fluid in the peritoneum the next point to determine is its probable cause. In order to do this the whole clinical history of the case must be enquired into, and the heart, lungs, liver, and kidneys examined for evidences of disease. If the ascites result from heart or kidney disease it is always preceded by œdema of the lower extremities; but when the liver or portal vein is the seat of obstruction, fluid first appears in the abdomen, and subsequently in the lower extremities as a result of compression of the inferior cava and functional derangement of the kidneys.

The diseases, other than those of the liver and portal vein, which may produce ascites, and which it is frequently necessary to differentiate from the latter are: Simple Dropsy of the peritoneum; Acute, Chronic, and Tubercular Peritonitis; Cancer of the peritoneum; and Colloid Disease of the peritoneum.

Simple Dropsy of the peritoneum arising without any apparent cause in a person addicted to intemperate habits would constitute a condition difficult to diagnose from ascites due to cirrhosis of the liver. The chief points to which attention must be paid when forming a diagnosis are, the absence of symptoms of liver disease, pain or tenderness of the abdomen, and also of pyrexia. As simple dropsies usually recover speedily under judicious medical treatment, cases of the kind are soon cleared of any doubt as to their nature.

Acute Peritonitis in a small proportion of cases is accompanied by a distinct effusion of fluid, but such a condition, it is hardly possible to confound with ascites due to liver or portal disease. The chief points by which the nature of the disease may be recognised, are its acute character; great pain and tenderness in the abdomen; pyrexia, preceded or followed by rigors; frequent small wiry pulse; pinched and anxious expression of the face; restlessness; a tendency to collapse combined with signs of impaired respiration and stagnant circulation. There are also vomiting, obstinate constipation and gaseous eructations. The patient lies with the hips and knees flexed, and the legs drawn well up in order to relieve abdominal tension.

Chronic Peritonitis is by no means an uncommon disease. It may occur under various circumstances. 1.—It may arise as a sequel to one or more attacks of acute peritonitis, either general or local, but especially the latter. 2.—It may become associated with ascites from liver or portal disease, but especially so when paracentesis has been several times resorted to for the removal of the fluid. 3.—Cancerous and other tumors of the abdominal organs and ulceration of the stomach and bowels, by their

irritating effects may produce chronic inflammation in the peritoneum. 4.—The disease may occur in persons who are the subjects of chronic Bright's disease or rheumatism.

However produced, chronic peritonitis usually leads to adhesions and thickenings of the serous membrane. Organs are united to each other, to the abdominal wall, the mesentery or omentum, or matted together and bound down to the spine in an inseparable mass. In the interstices so formed fluid accumulates, sometimes in considerable quantities. Occasionally fluid is present in enormous quantities and free to move about.

Fluid effusion, resulting from chronic peritonitis may usually be diagnosed from ascites following hepatic and portal disease by a careful attention to the history of the case, the symptoms, and the physical signs. Thus, the patient usually experiences pain or tenderness or both in the abdomen. Pain, when present, is usually of a colicky character, and liable to occur in severe paroxysms. More or less tenderness on pressure is very commonly observed in various localities, where it may be very great. Fever, of a hectic character, increased frequency of the pulse, languor, weakness, wasting and anæmia, with a dry and harsh skin are also usual concomitants of chronic peritonitis.

The physical signs also demand especial attention. As a rule abdominal enlargement, due to effused fluid, is not very considerable, but it may attain enormous proportions with stretching of the skin and protrusion of the umbilicus. More or less want of symmetry, sometimes amounting to marked irregularity of the surface is frequently observed, especially in advanced cases. Palpation reveals an absence of uniformity of resistance in various parts of the abdomen: obscure fluctuation in an area circumscribed by firm or resisting boundaries, and even distinct tumors, more or less irregular in outline, may be felt. Percussion occasionally reveals freely moveable fluid: but as a rule it demonstrates that the fluid is bound to certain localities by more or less solid material, or even tympanitic intestines, and not influenced by change of posture. The diagnosis may be still further strength-

ened by the removal of some of the fluid by means of a small trocar. This may be merely serous, but usually it contains products of inflammation, fibrinous flakes, pus, or even blood.

TUBERCULAR PERITONITIS.—The remarks which have just been made upon the subject of chronic peritonitis apply with equal force to this disease, of which it is merely an important variety.

CANCER OF THE PERITONEUM is comparatively rarely observed as a primary disease. Usually it originates by extension from malignant growths situated in the liver, alimentary canal, retro-peritoneal glands, or sexual organs. Primary cancer of the peritoneum usually results from traumatic causes. It almost always occurs in aged persons; is the seat of more or less pain and tenderness; and produces effusion of usually large quantities of fluid into the peritoneal cavity.

Usually the disease may be recognised by the age of the patient, the existence of pain and tenderness in the abdomen, the presence of primary cancer in other parts of the body, and finally by the development of the cancerous cachexia.

COLLOID CANCER is frequently met with in the omentum. The tumors consist of intercellular spaces of variable sizes, filled with a clear glairy fluid like glue, which contains abundance of granules and large nucleated cells. The disease, like ordinary cancer, is met with in middle or advanced life. Emaciation advances rapidly, and death usually ensues within six months from the commencement of the symptoms.

The abdomen may be greatly enlarged but is not uniform or quite symmetrical. Palpation generally reveals hard irregular masses or distinct tumors; and fluctuation, if present, is often very indistinct. Anteriorily, the abdomen is everywhere dull, leaving a tympanitic space in either flank. Change of posture produces little, if any, effect upon the dull area. A slimy gelatinous fluid may be removed from the tumor by the aspirator, and occasionally a similar fluid is voided *per anum* or from the stomach.

TREATMENT.—Very little need be said in this place as to the treatment of ascites because that has been dealt with when treating of the various diseases of the liver and portal vein of which it forms a prominent symptom. I shall merely give a few hints as to the method of performing.

PARACENTESIS ABDOMINIS.—The instruments, &c., required for the operation are, a medium sized trocar and canula; an india-rubber tube of about the same calibre as that of the canula, about six feet in length, and furnished with a silver tubular plug which fits into the canula when the trocar has been removed from it; a pad of lint; collodion; carbolic oil (1 to 10); a tub or pail; a strip of flannel wide enough to reach from the nipples to the pubes, and long enough to go two and a half times round the abdomen; each end of this bandage must be torn for about one third of its length into four or five tails; some stimulant,—either brandy, wine, or aromatic spirits of ammonia. Two steady assistants are also requisite.

The patient, having first passed water, or the bladder having been emptied by the catheter, is brought to the edge of the bed and placed in the recumbent posture with the shoulders raised; a folded sheet or a piece of mackintosh cloth having been previously so arranged as to protect the bed-clothes. The middle of the flannel bandage is then to be applied to the front of the abdomen and the tails so arranged as to interdigitate with each other opposite the spine. An end of the bandage is to be entrusted to each of the assistants with direction that a steady and uniform pull is to be maintained as soon as the trocar is withdrawn. A circular hole is now cut in the bandage at the point at which the abdomen is to be punctured. This is usually done in the linea alba, midway between the umbilicus and the pubes, the operator having first assured himself by percussion that there are no tympanitic coils of intestine in that locality; otherwise a more suitable site for the operation must be selected. The trocar and canula, warmed and oiled, are held under the hand with the end of the handle in the hollow of the palm, the thumb placed upon the rim of the shield of the canula, ready to push it off, and the

index finger firmly pressed against the side of the canula about one inch from its end so as to guard against inserting the trocar too deeply. No preliminary incision is necessary if the instrument is, as it ought to be, in good order; but it is advisable to draw the skin aside from its normal position before introducing the trocar, so as to produce a valvular puncture which will close readily upon the withdrawal of the canula. The trocar and canula are inserted into the abdomen with direct sharp force.

The abdomen having been punctured, the trocar is withdrawn with the right hand and the canula immediately plugged with the left thumb to prevent the escape of fluid. The silver plug of the india-rubber tube is then fitted to the canula, the distal end of the tube being kept submerged in the fluid of the receptacle.

As soon as the fluid has ceased to flow the canula is withdrawn and a pad of lint soaked in collodion is applied over the puncture. Then the corresponding tails of the bandage are brought across the abdomen and firmly tied over the middle line.

Should the patient become faint during the operation it must immediately be suspended, stimulants administered and the head laid low.

APPENDIX.

CHOLECYSTOTOMY.

CHOLECYSTOTOMY consists in making a free opening into the gall-bladder by abdominal incision, so as to permit of thorough evacuation of its contents.

As long ago as the year 1743 this operation was first proposed by Jean Louis Petit in a masterly memoir *(Mémoires de l'acad-roy. de chirurgie Tome I. p. 168.)*, in which he drew a parallel between disorders of the gall-bladder and those of the urinary bladder, and urged the feasibility of the extraction of biliary calculi. From that time the operation appears to have sunk into oblivion until Dr. Hanfield Jones again suggested it.

Notwithstanding this advocacy of the operation surgeons appear to have eschewed it until April 1878, when Dr. Marion Sims boldly carried it out; and although the patient died eight days after the operation, the latter in itself was quite successful as regards the relief of the distressing symptoms, but the *post-mortem* examination revealed that the disease had already advanced too far for the patient's life to be saved.

In June of the same year Kocher *(Correspondenzbl. f. Schweiz Aerzte. viii. Jahrg., 1878, No. 19.)* operated with complete success on an empyema of the gall-bladder as large as a man's head in a woman aged thirty years. Forty-three gall-stones in all were removed.

Again in August 1879, Mr. Lawson Tait of Birmingham *(Lancet, Nov. 15th, p. 730)* removed by abdominal section two large gall-stones, one lying loose and the other impacted in the neck of the gall-bladder, and adherent to the mucous membrane. The latter was removed after a tedious and very difficult operation. The stone and fragments weighed 6.11 grammes. The operation was performed antiseptically under ether. The patient rallied completely in a few hours. Bile continued to flow from the wound for eleven days, and the latter was completely healed in seventeen days. Five weeks after the operation the patient returned home quite restored to health.

In October 1882 Mr. Tait successfully performed Cholecystotomy a second time, sixteen gall-stones being removed from the gall-bladder.—*(British Med. Jour. Jany. 1883.)*

In performing this operation the median line is to be preferred for the abdominal incision because when made away from that line directly over the tumor considerable hæmorrhage takes place. The greatest danger of the operation consists in escape of bile into the peritoneum, which was obviated in Mr. Tait's case by stitching the edges of the wound in the gall-bladder to the upper end of the abdominal wound by continuous sutures, leaving the aperture into the gall-bladder quite open, and closing the rest of the abdominal opening in the usual way. No traction of the abdominal wall was produced by the shrinking gall-bladder.

The circumstances under which the operation appears to be called for are, when the gall-bladder is distended with biliary or purulent fluid together with gall-stones which irritate the mucous membrane. Remembering the brilliant results of ovariotomy, I think we are justified in predicting a successful future for Cholecystotomy.

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