all led me to regard the case as one probably of tuberculosis. It was the high leucocyte count, furnished me at the last moment, that led me to suspect suppuration and to counsel operation. With so many factors suggesting tuberculosis, I even now think it just possible that the pneumococcal inflammation was secondary to a previous tuberculous affection of the pericardium, although I have to admit that there was nothing found *post mortem* to justify such a supposition.

The double failure to find pus with the exploratory needle, even with the child under full anaesthesia, is further proof of the unreliability of the procedure in numerous cases. The failure with the needle, too, may well tend to upset a diagnosis that was otherwise well established, and in the present instance made me momentarily hesitate about sanctioning further operative measures. The diphtheritic condition of the larynx, discovered *post mortem*, of course precluded any chance of success from the operation; but it may be said that the condition had not revealed itself by any signs, and could not have been diagnosed during life.

This is the fourth case of purulent pericarditis I have had operated on, all without success. Three of the patients died within a few days after operation. In a fourth case, however, recently reported by Mr. Rowlands and myself,² the child lived for four months after operation, and here the death was accelerated by the contraction of measles and bronchopneumonia at a time when recovery seemed hopeful. All my cases, too, it may be claimed, were much younger than the generality of those recorded. The results are certainly not encouraging so far as to operation in very young children. Still, as death is practically certain without it, I would not be deterred by past experience from giving such children the slight chance that operation affords in any future cases I may come across.

REMARKS BY MR. ROWLANDS.

The peculiar narrowness of the costo-xiphoid angles in this emaciated child, together with the absence of any fullness there, made me decide against adopting the epigastric operation in this case. I therefore removed the sternal end of the seventh left costal cartilage, but this gave very little room and exposed the edge of the left pleura. It was easy to remove a portion of the adjoining cartilaginous sternum; this gave ample room and a good view of the pericardium, and a safe and quick access to it. The sac was opened at the lowest extremity of its anterior surface, and thus at a good place for drainage.

Moreover, it was found possible to pass a finger into the various recesses of the cavity, and to introduce a tube behind the heart into the oblique sinus with comparative ease. The above points and especially the patency and directness of the aperture established strongly recommend the sternal route for young children. In our former case the fifth left costal cartilage and a piece of the sternum at that level were removed; but I am confident that the lower incision is a better one. Instructions were given to turn the child over on to the prone position several times a day for drainage, but unfortunately the child succumbed to his several diseases on the second day following the operation. Irrigation was not adopted at the operation, partly because Dr. Coutts reminded me of Mr. Parker's case, in which Mr. Parker believed irrigation to have proved fatal owing to pressure on the auricles due to failure to provide a good exit for the fluid; but the chief reason against irrigation was to avoid delay in the critical condition of the child.

REFERENCES.

¹ BRITISH MEDICAL JOURNAL, January 2nd, 1904. ² Ibid.

WATER SUPPLY AND SEWERAGE FOR MANILA.—When Manila passed into the control of the United States Government there were no water-pipe connexions in the houses. The residents were compelled to carry water from public fountains at the street corners. There was no connexion with sewers. The refuse was carried to inlets, which carried the sewage away only when flushed by rain. During the last two years the United States Government has ditched the sewers so as to flush them from the river, and it now proposes to spend £500,000 on the construction of a modern system of sewerage. The sum of £300,000 has been appropriated for a new water supply. The water, according to the new plans, will be taken from the Mariquina river, about fourteen miles above Manila.

AN "Osler" Club has been formed in New York, each member of which pledges himself to retire from business when the majority of his fellows declare that he should be "Oslerized."

THE HEPATIC FACTOR IN BILIOUSNESS.

By FRANCIS HARE, M.D.,

Late Consulting Physician, Brisbane Hospital; Visiting Physician, Diamantina Hospital for Chronic Diseases, Brisbane; Inspector-General of Hospitals, Queensland.

THE pathology of functional diseases—that is, diseases which have no morbid anatomy—is admittedly unsatisfactory. The reason seems clear. Modern medicine is in the main inductive; it relies largely on the study of effects—usually ultimate structural effects; and where such are absent or imperceptible, further progress is liable to be checked. There is only one obvious way out of the impasse: we should add to our accustomed means of investigation a review of functional diseases from the deductive standpoint; in other words, we should apply the known principles of physiological function to the elucidation of the meaning of pathological function. So viewed, many seemingly mysterious functional disorders appear simple. This is true at least of those commonest of functional disorders, bilious attacks and biliousness.

Clinically these affections have been ascribed to prolonged external heat, to deficiency of physical exercise, to excess of sugar and rich carbohydrate food stuffs, and to excess of meat and nitrogenous food generally. Now it will not be difficult to show that all these etiological factors tend to cause at least one effect in common, namely, accumulation of glycogen in the liver.

It has been proved experimentally that accumulation of glycogen in the mammalian liver is favoured by external heat and physical inactivity (Kulz¹). All physiologists are agreed that sugar and other carbohydrates are the chief food stuffs from which liver glycogen is derived. And it can be argued that, like the performance of function generally, the absorption of all food stuffs, and therefore of glycogen-forming material, is dependent upon the supply of proteid. Conformably Foster says that "the greatest accumulation of glycogen is effected, not by a pure carbohydrate diet, but by a mixed diet rich in carbohydrates."²

It is not difficult to form a definite conception of the mechanism whereby accumulation of glycogen to an ultra-physiological degree can give rise to all the clinical phenomena of a bilious attack. The liver cell which is rich in glycogen is large compared with the liver cell which, through previous starvation, contains no glycogen. This has been demonstrated by experiment. Enlargement of the liver cells involves enlargement of the liver lobules, and this of the whole organ, which is "as it were swollen" (Foster³). Now we have only to glance at the well-known representations of the histology of the liver in Quain's or Gray's *Anatomy* to see that enlargement of the liver cells will cause pressure on the intralobular capillaries, and that enlargement of the liver lobules will cause pressure on the interlobular veins. It seems physically impossible that these blood vessels can escape material pressure; and it is certainly impossible that such intrahepatic pressure can be exerted so as not to cause material retardation of the blood flow through the portal vein—a vein in which the blood pressure is extremely low (10 mm. Hg).

We may then assume that glycogenic distension of the liver causes mechanical congestion of the portal venous system behind the liver. Now "there are no valves in the portal vein or in its larger tributaries. . . . Valves are, however, present in the veins of the stomach and of the walls of the intestine; but, according to Hochstetter and Bryant, they early become insufficient, and in the adult to a great extent disappear."⁴ It follows that the mechanical congestion of the main branches of the portal vein immediately behind the liver will directly involve mechanical congestion, or tendency to stasis, in the whole of the alimentary mucosa from the stomach downwards to the junction of the skin and mucous membrane at the margin of the anus.

Such a vascular condition affecting the gastric mucosa will be opposed to that which obtains when food is introduced into the stomach of the hungry animal. Then not only do the arteries of the organ dilate and largely increase the supply of oxygenated blood, but, the passage through the liver being unobstructed by glycogen, the circulation is rapid, so rapid that the blood in the gastric veins continues to preserve its arterial hue (Foster).⁵ Manifestly for the rapid manufacture and discharge of the digestive juices, and for active digestion, a free escape for the blood is as essential as a plentiful supply.

Mechanical congestion will be associated with some swelling. Such, affecting the duodenal mucosa, will probably be sufficient to cause blocking of the orifice of the common bile duct; for, as Lauder Brunton points out, "the pressure under which bile is secreted is very low, so that a very slight obstruction to its flow through the common duct is sufficient to cause its accumulation in the gall bladder and gall ducts."⁸ Blocking of the common bile duct will involve blocking of the pancreatic duct, since the two open by a common orifice. Thus there will be absence from the alimentary canal of bile and pancreatic juice; and it is probable that the vascular condition will preclude the secretion of the gastric and other digestive juices. As a result, there will be cessation of the processes of digestion and absorption, those processes which were primarily responsible for the glycogenic distension of the liver.

It is easy to conceive that the condition of the digestive organs described will be manifested clinically by anorexia, nausea, and vomiting; and that in some cases the retro-hepatic congestion will relieve itself by diarrhoea. Anorexia will preclude fresh intake. Vomiting will cause compression of the liver and distended gall bladder between the abdominal wall and the diaphragm; thus the retained bile will be ejected into the duodenum, and will usually find its way into the stomach, whence it may be vomited. Diarrhoea, in addition to relieving the portal congestion, will hasten the alimentary current, and so tend to restrict further absorption. Meanwhile the ever-present combustion in the tissues will, by creating a demand for fuel, gradually reduce the amount of glycogen deposited in the liver. So the glycogenic distension will be relieved, the hepatic obstruction removed, the mechanical stases dispersed, and convalescence established. The whole paroxysm may be regarded as a well-organized and most salutary strike against the absorption of food in excess of the requirements of the organism; it is, moreover, a strike which attains its object in the most direct manner possible.

Harley divides biliousness into (1) the acute form, due as a rule to some distinct error of diet, such as a surfeit of plum pudding; (2) the subacute (recurrent) form, due not to isolated indiscretions in diet but to continued habitual over-indulgence; and (3) the chronic form, usually met with in adults, especially old residents of the tropics, and extending, it may be, to months in duration.⁷ Conformably, we may speak of (1) acute glycogenic distension of the liver due to a large sudden irruption of glycogen-forming material into the portal venous system; (2) subacute or recurrent glycogenic distension of the liver, in which there is a continued absorption of glycogen-forming material in excess of consumption, and a consequent progressive accumulation of hepatic glycogen, which attains its maximum at more or less regular intervals; and (3) chronic glycogenic distension of the liver, in which recurrent relief fails, and in which consequently the organ remains more or less continuously packed with glycogen.

It has already been pointed out that the etiological factors commonly regarded as responsible for biliousness concur in tending to cause accumulation of glycogen in the liver; and it will not be difficult to show that factors, most of which are admittedly capable of dispersing biliousness, concur in tending to reduce the amount of glycogen in the liver. Some of these may be regarded as therapeutic—namely, abstention from food, physical exercise, climate, purgatives, restriction of meat and nitrogenous food stuffs, and restriction of carbohydrates. Others must be regarded as pathological—namely, pyrexia and glycosuria.

Therapeutic Factors capable of Dispersing Glycogenic Distension of the Liver and Biliousness.

Starvation empties the liver of glycogen (Foster⁹), and Harley says he has frequently cured his "bilious patients with a day or two's starvation diet." Many bilious patients have discovered for themselves the curative influence of temporary abstention from food.

Physical exercise markedly reduces the amount of glycogen in the liver (Manché⁹), and an increase of physical exercise is almost invariably prescribed in all kinds of biliousness.

Biliousness is proverbially more common in the tropics, and many bilious tropical residents may be rapidly relieved by a trip to a cool or cold climate. Other things equal, such climates, by promoting combustion, will reduce the amount of glycogen in the liver.

Deposition of hepatic glycogen depends *inter alia* upon the absorption of glycogen-forming material from the alimentary canal. Absorption occurs mainly in the intestine, hence

purgatives of any kind will restrict absorption by hastening the intestinal current.* But since the nutritive value of the intestinal contents diminishes progressively from above downwards, the greatest restriction of food absorption from purgatives will follow such as act mainly upon the higher portions of the canal. Such a purgative is calomel, which, according to Lauder Brunton, has a "peculiar stimulant action on the duodenum and ileum."¹⁰ This action was recognized by Murchison, who adduced in support the fact discovered by Radziejewski—that leucin and tyrosin, products of pancreatic digestion, which in ordinary circumstances are decomposed in the bowel, appear in the faeces after the administration of mercurials.¹¹ Now calomel is probably the purgative which has been found most useful in biliousness. But its usefulness will not be limited to its indirect relief of portal congestion through reduction of glycogenic distension.

Through withdrawal of fluid, calomel will directly relieve the retrohepatic portal congestion, the mechanical congestion of the duodenal mucosa, and the tendency to blocking of the orifice of the common bile-duct; and the last will perhaps be favoured by the exaggerated peristalsis of the duodenum induced by the drug. Thus bile will re-enter the intestine, and from its amount will colour the motions in probably greater degree than under physiological conditions. The long-standing difference of opinion between the physician and physiologist as to whether calomel does or does not increase the output of bile, depends, I think, upon the fact that both observers considered only the secretion of bile. The two observations are reconcilable on the above view. Calomel does not increase the secretion of bile, but effects the release of a prior retention. In support, it may be recalled that in some cases of prolonged biliousness the motions passed are light and dark alternately.

Reduction of meat and nitrogenous food stuffs generally is by no means new as a treatment for biliousness. Writing in 1879, J. Milner Fothergill advises "cutting down the albuminoids," and "substituting a dietary of fruit and farinaceous food for meat."¹² He considered that "by such means the work of the liver would be greatly economized, not only as to the storing up of glycogen, but, what is more important, the work of oxidation of albuminoids would be lessened."¹³ The plan is still widely practised, and is moderately successful in many cases. Its success, however, is variously explained. It has been ascribed to diminished absorption of toxins from the intestine and to diminished introduction of the antecedents of uric acid.

On the view that glycogenic distension of the liver to a pathological degree is the essential factor, the success of the plan seems simple. The processes of digestion and absorption are functions of the nitrogenous tissues; and it is a fundamental biological fact that all functions depend upon the supply of proteid. It has been demonstrated by experiment that an animal from which all proteid is withheld dies from starvation, and that despite an ample supply of fats and carbohydrates. This means, of course, that digestion and absorption, amongst other functions, cease to be performed. It is reasonable, therefore, to believe that a large restriction of meat, fish, and eggs—the main sources of the proteid supply—will result in a progressive impairment of the processes of digestion and absorption. Hence the inflow to the portal venous system of glycogen-forming material will become less, and by so much will diminish the deposition of hepatic glycogen. This diminution will be gradual, because the impairment through restriction of proteid, of digestion and absorption, as of all function, is a gradual process. Hence bilious attacks and biliousness generally, treated by this method, may be expected to subside gradually, not suddenly; and clinical experience shows gradual subsidence to be the usual manner of improvement.

But restriction of proteid may prove a double-edged therapeutic weapon. Impairment of functions other than those of digestion and absorption may well ensue. For example, it is admitted that restriction of proteid retards the rate of combustion by the fixed nitrogenous tissues; and there can be little doubt that the function of combustion in the tissues is an important factor in relieving the liver of accumulated glycogen. Hence it is to be expected that restriction of proteid will in some cases fail to relieve bilious manifestations; and this will be found to accord with clinical experience. Many persons treated in this way not only continue

* The excess absorption associated with habitual constipation has been demonstrated experimentally by Baron Oefele (BRITISH MEDICAL JOURNAL, January 7th, 1905, p. 24).

to suffer, but also lose energy, strength, and colour. Such results, however, may often be moderated, if not obviated, by a concurrent increase of physical exercise, whereby the rate of combustion is maintained.

Restriction of Carbohydrates.

But if, as argued in this article, many of the various clinical conditions embraced in the term biliousness depend upon ultra-physiological distension of the liver by glycogen, then obviously the most rational, because the most direct, method of treatment will consist in restriction of the supply of glycogen-forming material. Glycogen is formed mainly from the carbohydrates, and by cutting down the carbohydrates in a mixed diet we shall relieve glycogenic distension. Conformably this plan has given in bilious attacks and biliousness generally results which are unmistakable. In many cases the change of diet is followed by an almost immediate cessation of recurrent bilious attacks, which remain absent so long as the new régime is maintained. Equally significant is the result of reversion to prior food habits. After a time, varying in different cases from a few days to a few weeks, the attacks recommence, and pursue their old recurrent course, usually at the accustomed intervals. It is true, however, that in some cases, a prolonged period of freedom, attained in the above manner, results in the disappearance of the tendency, even in the face of a moderately full carbohydrate intake. Numerous cases in support could be detailed, but space is lacking.

Pathological Conditions Capable of Dispersing Glycogenic Distension of the Liver and Biliousness.

The most noteworthy of these are pyrexia and glycosuria. Pyrexia markedly reduces the amount of hepatic glycogen (Bernard¹⁴). Probably in most cases it operates to this end in two ways. Always associated with pyrexia is an increase in the output of carbonic acid (Liebermeister, Leyden, Ringer); and this must depend on an increase in the rate of combustion; thus the expenditure of carbohydrate material is increased. Usually associated with pyrexia is a distaste for food, and more or less dyspepsia; thus the income of glycogen-forming material is diminished. At any rate the experimentally-demonstrated fact remains that hepatic glycogen is reduced by pyrexia. Conformably any practitioner can assure himself that the clinical manifestations typical of biliousness remain, for the most part, in complete abeyance during pyrexia of any duration. This is naturally most conspicuous when a prolonged intercurrent pyrexia supervenes in a case of long-standing recurrent or periodic bilious attacks. I know of numerous cases in which recurrent bilious attacks of many years' standing have ceased absolutely throughout the whole course of typhoid fever; and in many of these the hepatic disorder returned during convalescence, and continued to recur thereafter with its old approach to regularity.

What is true of recurrent bilious attacks should be true of chronic biliousness if, as seems probable, this condition often depends primarily upon prolonged glycogenic distension of the liver; any intercurrent pyrexia should have a salutary influence on the hepatic manifestations. On this point I made inquiries of Major D. J. Buchanan, I.M.S., editor of the *Indian Medical Gazette*, who kindly replied as follows: "My attention has not been called to the point you mention about the improvement following intercurrent feverish attacks in liver cases, though when you mention it, I have seen such improvement, though I did not note the connexion at the time." I can myself recall one case in which long-continued bilious symptoms of a distressing nature seemed completely dispersed by a compound fracture into the elbow-joint complicated by considerable septic pyrexia.

Glycosuria has marked inverse relations with biliousness. This is easy of explanation if we accept Pavy's view that the dehydration of portal sugar constitutes the glycogenic function of the liver, and that in glycosuria this function is at any rate inadequate. For, inadequacy of dehydrating power on the part of the liver will manifestly render impossible a pathological degree of glycogenic distension. Conformably Ehrlich states¹⁵ "that very little or no glycogen can be found in fragments of liver withdrawn by a trocar from diabetics during life." And again, it is a fact that recurrent bilious attacks and many other clinical conditions which cannot be here considered, but which, I shall elsewhere argue, depend upon glycogenic distension of the liver, tend to remain in abeyance or to become conspicuously modified, upon the supervention of glycosuria or diabetes mellitus of any kind.

Space is wanting for the enumeration of cases in support, but any physician can assure himself of the truth of the above statement in regard to bilious attacks by going through the previous life-histories of a series of diabetics.

The rational treatment for many cases of recurrent and other forms of biliousness has been deduced from the pathological views entertained in this article. This treatment consists simply in limiting the intake of carbohydrate material, and sugar is the first food stuff to be excluded. Herein is involved no functional impairment in any direction. The treatment consists merely in cutting down excess of supply. But practically it will be found advisable in most cases to insist also upon an increase in regular physical exercise. This will obviate the necessity for any severe degree of carbohydrate restriction. And it is certain that the patient who precludes glycogenic distension of the liver in part through such a healthy increase of expenditure remains in more robust condition than one who merely reduces his income to the level of a sedentary life; in other words, regular physical exercise confers many incidental benefits of great importance.

There are two further points requiring notice: The first is that the management of biliousness by the means advised is much simpler in the case of patients who are well nourished. To those of the profession who may not be conversant with this treatment but who may wish to make trial thereof I should like to suggest that commencement be made with patients who incline to be or who are corpulent. The second point is that in some cases, though by no means in all, it will be found necessary to provide against an excess of fats not less than against an excess of carbohydrates. Neither of the above points are merely empirical. They are both susceptible of simple explanation on physiological principles. But such explanation would open out a fresh argument and must be deferred.

REFERENCES.

- ¹ *Textbook of Physiology*, Schäfer, 1898, vol. i, p. 918. ² *Textbook of Physiology*, Foster, 1895, p. 751. ³ *Ibid.*, 1895, p. 755. ⁴ *Quain's Anatomy*, 1890, vol. ii, part ii, p. 543. ⁵ *Textbook of Physiology*, 1895, p. 426. ⁶ *Pharmacology, Materia Medica and Therapeutics*, 1885, p. 355. ⁷ *Diseases of the Liver*, 1883, p. 274 et seq. ⁸ *Textbook of Physiology*, 1895, p. 755. ⁹ Quoted by Schäfer, *Textbook of Physiology*, 1898, vol. i, p. 918. ¹⁰ *Pharmacology, Therapeutics and Materia Medica*, 1885, p. 355. ¹¹ *Functional Derangements of the Liver*, 1874, p. 169. ¹² *Practitioner*, 1879, vol. ii, p. 353. ¹³ *Ibid.* ¹⁴ Quoted by Saundby, *Renal and Urinary Diseases*, 1896, p. 299. ¹⁵ *Ibid.*

ON THE CLOSURE OF TRAUMATIC DEFECTS OF THE SKULL.*

By CHARLES LEEDHAM-GREEN, M.B., F.R.C.S.,

Surgeon to Out-patients, Queen's Hospital; Honorary Surgeon, Children's Hospital, Birmingham.

I WISH to bring briefly before your notice the methods by which we can best close large defects in the vertex of the skull, whether produced by traumatism or otherwise, and to record two examples met with in my own experience.

Compound comminuted fractures of the vault furnish a large proportion of these defects, but a certain number follow surgical interference for the relief of cerebral tumour, epilepsy, etc. It is well established that, unless the loss of substance be very small, such defects of the cranium are very seldom spontaneously closed by bone, the bones of the vertex having but little power of repair. As a rule, the lost bone is replaced by fibrous tissue. When the defect is small this tissue is dense and unyielding; so much so that it is difficult to ascertain whether true bone exists or not. The few authenticated cases in which true bone has been formed to any extent can but be regarded as rare exceptions to the general rule.†

Clinical experience proves that the closure by firm fibrous tissue of a small defect, such as is left by the removal of a trephine crown, is generally unattended by injurious consequences to the patient. But it is not always so when the defect is large. For here the fibrous tissue most imperfectly replaces the bone. It is thin, soft, and yielding, and inade-

* This paper was read before the Midland Branch of the British Medical Association. It was forgotten until a short time ago, when, after an interval of some years, I had an opportunity of re-examining both the cases referred to in it. This recalled the paper, and suggested that it might be worth publishing.

† Küster¹ reports a case of necrosis (not syphilitic) of the frontal bone, where, after the removal of a sequestrum (10 cm. by 8 cm.), the defect was completely and spontaneously closed by bone. Goetz² also reports a case of syphilitic necrosis of the skull, where a defect of 13 cm. by 17 cm. was spontaneously closed.