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.

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ON THE CLOSURE OF TRAUMATIC DEFECTS OF THE SKULL.*

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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.

quately protects the brain beneath. The patient is not only especially liable to cerebral injury from blows on the head, but severe intellectual disturbances are apt to supervene. It is true that this statement is opposed to the opinion held by Professor Kocher³ and Berezowski,⁴ neither of whom believes that such defects have injurious consequences. Indeed, Kocher is of opinion that they tend rather to prevent than produce serious cerebral symptoms, such as epilepsy. But, on the other hand, there is an increasing record of cases, which shows that though it is true that large traumatic defects may be unattended by serious consequences, yet at times severe cerebral symptoms do develop, which strongly point to such a cause. There is König's⁵ historic case of a young man who, in consequence of a severe injury to the head, sustained an extensive cranial defect of the left temple and parietal region. Over this area the pulsations of the brain could be distinctly seen. The man, who previous to the injury was typically healthy, gradually became dull and apathetic to a marked degree, and suffered from frequent epileptic attacks. All these symptoms were permanently cured on the closure of the defect by an osteoplastic operation.

Von Bergmann also regards such defects, especially in young people, as directly predisposing to epileptic attacks. He quotes an interesting case⁶ where a child, after a compound fracture of the frontal bone produced by the kick of a horse, was left with a pulsating defect. Three years after the injury the child was subject to attacks of headache, giddiness, and fainting. The defect in the bone was closed by a plastic operation, and the child at the time of writing, more than six years after, was perfectly well; von Bergmann has recently published three other similar cases, which he cured in a like manner. Bunge⁷ is also of opinion that these defects are apt to give rise to grave trouble. Out of 13 cases observed by him, severe symptoms developed in 9, and in 4 of these epileptic attacks were present. In his cases the older the defect the more marked were the symptoms.

But, it may be asked, Are the symptoms observed in these cases really due to the defect in the cranial wall, or are they due to other causes, such as injury to the cortex, or adhesion of the brain to the dura mater or scalp? Although it is possible, nay probable, that frequently the symptoms observed are due to one or other of these latter causes, rather than to the presence of the defect, there are nevertheless many cases recorded which cannot be explained in this manner. These are cases where the dura mater was unopened, either at the time of the injury or at the plastic operation for the defect, and where the simple closure at once and permanently relieved the symptoms.

There is no lack of methods by which the defects may be made good, whether at the primary operation or subsequently at a second operation for that purpose. It is well known that the circular piece of bone removed by the trephine may be replaced either entirely or in small fragments. The re-implanted portions of bone generally heal in without any trouble. They do not themselves become living parts of the skull, but are generally absorbed, new bone being laid down in their place. Where such portions of bone are not available, other measures have been suggested. For example, the implantation of decalcified bone has been effected by Senn;⁸ but it has since been shown that this kind of bone is rapidly absorbed, fibrous tissue, and not true bone, taking its place. Barth⁹ says that this is due to the absence in the decalcified bone of lime salts, which are essential in any graft laid down with a view to producing true bone. He himself uses charred bone; and there are numerous cases recorded by him and others¹⁰ showing the efficiency of this method. Mertens¹¹ advocates the implantation of bone which has been boiled. The superiority of this method over the former arises from its more rapid and more easy preparation, and its freedom from the objection of brittleness. Other surgeons have made use of grafts of living bone, removed from distant portions of the body. The anterior surface of the tibia,¹² or portions of bone removed from the lower animals may be used for this purpose.¹³ In other cases plates of ivory,¹⁴ silver, or celluloid¹⁵ have been inserted, and successfully healed in. All these methods have the same drawback—namely, that of utilizing foreign bodies to close the opening. They therefore cannot be used unless the wound be in a perfect aseptic condition. If it should not heal per primum the bone graft or inserted plate will not be tolerated. In many of the reported successful cases of the implantation of silver or celluloid plates the plate has eventually caused irritation, and has had to be removed.

A method which is free from the disadvantages arising

from the implantation of foreign bodies, whether of celluloid, boiled bone, or other material, is that which is known in Germany as the "König-Müller method."^{16 17} It is the one which I adopted in the two cases which I wish now to record.

Rather more than five years ago a young man, aged 22, was struck violently on the head by a swing-boat, and sustained a large compound comminuted fracture of the occipital bone. He was brought to the Queen's Hospital, where I removed a large number of small bony fragments, leaving a defect in the skull of 3 sq. in. The dura mater was only slightly torn, and no gross lesion of the cortex of the brain could be detected. In consequence of the dirty nature of the wound and the damage done to the scalp, no attempt at bone-grafting could then be entertained. The patient made an uninterrupted recovery, and in a few weeks left the hospital with the intention of resuming his work. I then lost sight of him for two years, when he turned up in the out-patient room, complaining of severe headache, augmented by any movement of the head, dizziness, especially when stooping, and epileptic fits, which were increasing in frequency and severity. The headache and giddiness began to develop some six months after the injury; and a few months later mild epileptic attacks were noticed. At the site of the former injury there was a well-marked depression, about 2½ in. in diameter. The brain could plainly be seen pulsating beneath the scalp. As the symptoms were increasing in severity and completely prevented the man from following his employment, I admitted him to the hospital with a view to closing the defect. Under an anaesthetic I turned up a large horseshoe flap of the scalp so as to fully expose the defect in the skull. I then refreshed the bone margin of the defect. (The dura mater was not opened.) When this had been done I made another horseshoe incision to one side of the first flap to outline another flap of the same dimensions as the first one, but having its base downwards. This flap differed from the former one, inasmuch as in raising it I took with it a thin layer of bone from the outer table of the skull. This was done by means of a broad chisel. The two flaps were then transposed; this second one, with the thin layer of bone adhering to its under surface, was placed so as to cover the cranial defect, and the first one to take its place. The wound healed per primum, and the patient was able to leave the hospital in a fortnight's time.

Fig. 1.

On examination of the part some months afterwards, the previous defect was found to be completely cured by a firm bony plate. The pulsation of the brain could no longer be detected, and apparently the hole in the skull was completely filled in. The patient declared himself so much better that he regarded himself cured. The fits had almost disappeared and the headache and giddiness quite.

I have recently had an opportunity of re-examining the patient, now two and a half years since the plastic operation. Although it leaves nothing to be desired, as regards closing the defect, yet the patient has not entirely lost the liability to epileptic attacks. But they become more and more slight and infrequent. It is, however, to be noted that he has lately become addicted to alcoholic excess, which has always so adverse an influence in cerebral injuries.

Fig. 2.

I have also made use of this mode of bone grafting when operating for the cure of trigeminal neuralgia, by Krause's method, and with complete success. As ordinarily performed a large horse shoe scalp flap is turned down the temporal region, and the bone removed by means of trephine and cutting forceps so as to gain access to the cranial cavity to remove the Gasserian ganglion. Subsequently this leaves a considerable defect in the skull and produces a deep and unsightly depression, of the size of the bowl of a tablespoon, on the side of the temple. To avoid this, Krause advises that the skull be resected by Wagner's method,¹⁸ though he admits that this procedure is apt to take a disproportionate time, and is not, therefore, advisable in elderly or debilitated patients.¹⁹ I have found that, after the horse-shoe flap has been outlined by the scalpel, one can easily and rapidly by means of a broad sharp chisel shave off a thin film of bone adherent to the under-surface of the scalp flap. A small opening can then be made in the denuded bone, and rapidly enlarged to the necessary size by means of gouge forceps. When the ganglion has been removed, the temporal flap, with its adherent scale of bone, is sutured down over the defect. New bone is rapidly laid down from the bone-periosteum flap, and in a few weeks the defect is filled by firm and unyielding bone, and the disfiguring depression is thus obviated.

I think there can be no doubt that where the secondary closure of a bony defect is indicated the best method to use, if available, is that of the König-Müller flap, or some modification of it, for this method allows of many modifications. It does not seem necessary that the bone-periosteum flap should be adherent to the skin flap.²⁰ It may be raised up independently of the skin, merely receiving its blood supply through a small periosteum attachment. Indeed, there is evidence that a wholly detached periosteal graft is able to lay down true bone, and may be used for plastic purposes. F. von Mangolt²¹ has recently published two cases of pseudoarthrosis from loss of bone which were cured by the grafting of detached grafts of periosteum. Though obviously such a method exposes the graft to a far greater liability to necrosis should the wound become infected.

Where it is to be feared that firm adhesions might form between the cortex and the implanted strip of bone, the graft may be turned over so that the periosteal surface lies

against the brain.²² It is, however, by no means certain that the bony surface is liable to form adhesions with the dura mater or cortex. Czerny²³ reports that, having occasion to turn up a König-Müller flap which he had laid down some eighteen months previously, he found that the under-surface of the bone was not adherent to the dura, but that a new lamina vitrea had formed on the under-surface of the bone-graft. Hoffmann²⁴ substantiates this from experiments made by him on certain animals.

Where for any reason it is not possible to make use of the König-Müller method, or any of its modifications, the best homoplastic method is probably that advocated by Mertens, namely, the use of boiled bone. According to Bunge, this only succeeds when the implanted bone is placed in direct contact with osteoplastic tissue. When the dura mater is covered with strong granulations this condition is probably not fulfilled, and absorption of the implanted bone will take place unless the diploë of the cranial bones be refreshed and the engrafted bone comes in direct contact with the refreshed edges of the cranial defect.

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MEMORANDA:

MEDICAL, SURGICAL, OBSTETRICAL, THERAPEUTICAL, PATHOLOGICAL, ETC.

A CASE OF DEATH UNDER CHLOROFORM.

A CASE of death under chloroform occurred at the Edinburgh Hospital for Women and Children, Brunsfield, on January 21st, under the following circumstances:

The patient, a married woman aged 40, was admitted for operation for irreducible umbilical hernia. The hernia had existed for five years, and had for some time brought on attacks of pain and retching after any exertion. There was nothing in the previous history to contraindicate the use of an anaesthetic; in the circulatory system nothing abnormal was detected; in the respiratory, there were some crepitations at the base of the right lung. The examination of the urine showed specific gravity 1015, alkaline, no albumen; precipitate, phosphates. The temperature was normal or slightly subnormal, and the pulse ran about 80. On the fourth day after admission, and after the usual preparation, chloroform was administered to the patient by the house-surgeon. She took the anaesthetic quietly; there was no holding of the breath. The chloroform was dropped steadily on the mask and lowered gradually to within half an inch of the face, and continued for three or four minutes. Then the patient began to breathe heavily and rapidly, and the pupils dilated; there was no stridor, and no pallor. The mask was held further from the face, but not at once entirely withdrawn; then respiration suddenly ceased, chloroform was stopped, and the face became congested. This was within five minutes of the commencement of the administration. The head was lowered immediately, the feet raised, and artificial respiration combined with tongue traction was begun and persevered with for four hours. Hypodermic injections of strychnine and ether were immediately given, followed