explanation of the common belief that heredity is a favourable sign in diabetes, which, although true of the more common and obvious dominant variety, is not correct when the condition is inherited as a recessive character.

At present it is not clear whether the inherited factor is itself the determining cause of diabetes, or whether it merely predisposes to the development of the disease by subsequent illness or injury. The occurrence of severe cases without demonstrable cause in early youth and infancy is in favour of the former hypothesis, but the undoubted connexion of diabetes with infections and other recognized causes in some instances indicates that, in later life at least, an exciting agent is often necessary. It is possible that the diabetes of young people may arise from inherited defects of the recessive type, which prevent the development of the organism in a particular direction keeping pace with the increasing demands of the growing body, and, as the inherited factor probably differs in intensity, so the period of life at which the symptoms of diabetes appear also varies. In this way we can account for the severe form met with in infants, the tendency for the disease to develop about the age of puberty, when, as was pointed out by Priscilla White,⁷ it is frequently related to excessive height, and the influence of infections, toxæmias, injury, pregnancy, etc., when growth has ceased. The mild and relatively innocuous form transmitted as a dominant character is an entirely different condition, and, as it is comparatively rarely met with before an age when the vigour of the body is naturally beginning to decline, it would seem to depend upon the development of an inherited tendency to abnormally early failure of some function concerned in carbohydrate metabolism, accelerated, as a rule, by acquired faults of assimilation and nutrition. The notoriously benign type of diabetes so common in certain races, particularly Hindus, Japanese, and Jews, is very likely of this variety, and probably depends upon an inherited dominant strain developed by a lifelong adherence to an improperly balanced diet.

Although there can be no doubt that heredity plays a part in the production of the defects of carbohydrate metabolism, giving rise to glycosuria and diabetes in many instances, there is not as yet sufficient evidence to justify the conclusion that an inherited factor is a necessary basis for their development in all cases. Should further research prove that such a basis exists, it would do much to simplify our ideas of the etiology of diabetic conditions by providing a common bond between the various forms, the differences in symptomatology, severity, and age incidence which occur being then accounted for by the character of the inherited factor and the nature of the exciting cause. Prognosis, too, would be more certain, for it would be largely determined by evidence pointing to the hereditary tendency being of the dominant or recessive type, while the fact that the glycosuria associated with the former is mild and easily controlled, whereas the recessive form is generally progressive and more severe, would be of assistance in formulating an appropriate line of treatment. Even in the present state of our knowledge inquiry into the family history of a patient suffering from glycosuria will often supply information which is useful in treatment, and, by the reassuring prognosis it may suggest, go far to dispel the fear of "diabetes" the discovery of sugar in the urine arouses in so many people.

The main value of the evidence regarding the occurrence of an hereditary factor in defects of carbohydrate metabolism is, however, its bearing on the question of marriage. The marriage of diabetics is clearly inadvisable, and inter-marriage between families where there is even a remote history of the disease is to be discouraged; but as our advice on such matters, although often asked, is rarely taken, the children and grandchildren, even to the third and fourth generation, should be watched and guarded as far as possible from exciting causes likely to develop a latent and inherited defect.

REFERENCES.

- ¹ Lépine: *Diabète Sucre*, Paris, 1909, p. 395. ² *Der Diab. Mellitus*, Wien, 1906. ³ *Arch. Int. Med.*, 1927, 39, p. 67. ⁴ Quoted in *Journ. Amer. Med. Assoc.*, 1922, 2, p. 1934. ⁵ *Journ. Genetics*, xvi, 3, 1926, p. 387. ⁶ *Der Diab. Mellitus*, p. 37. ⁷ *Journ. Amer. Med. Assoc.*, 1927, p. 170.

INJURIES OF THE EAR ARISING FROM FRACTURES OF THE SKULL.*

BY

EDWARD D. D. DAVIS, F.R.C.S.,
Surgeon, Nose, Throat, and Ear Department, Charing Cross
Hospital.

IN view of the increasing frequency of motor and street accidents, a discussion on fractures of the base of the skull from the aurist's point of view should be useful.

Patients suffering from this injury are usually sent for an aural examination some weeks after the accident, when deafness exists, or when the question of compensation arises. Inspection of the ears immediately after the accident would be more valuable and would enable the aurist to give a more accurate estimation of the damage. The additional information so obtained might be helpful in the treatment and prognosis.

The majority of the fractures of the base of the skull involve the middle fossa, and this proportion is to be expected when the base of the skull is viewed from below and it is seen that a line of weakness extends through both Glaserian fissures and the Eustachian tubes to the foramina lacera media, with the sphenoidal sinus as a connecting link. The two halves of the skull are more or less cemented together by the union of the basi-occipital and basi-sphenoid, and the fracture of the middle fossa usually follows this weak line wholly or in part. The thin roof of the canal of the tensor tympani and Eustachian tube, and that of the tympanum and mastoid antrum, lie in this line and are almost invariably involved in this fracture. The extent of the injury varies considerably with the violence and site of application of the blow received. Comminution of the thin bone of the tegmen sometimes happens, and spicules of bone are subsequently found in the auditory meatus. It is the middle ear which is practically always damaged in patients who survive, and the capsule of the internal ear or labyrinth is rarely fractured. Only a few fatal cases of great violence show fractures of the internal ear, and J. S. Fraser,¹ in 1916, recorded, with microscopical sections, four instances in which the fractures involved the labyrinth capsule in two and the middle ear only in the remaining two. The two middle-ear cases survived for a variable period and then died of meningitis, but the labyrinth cases were quickly fatal.

The external auditory meatus is often involved in the fracture when the line is through the Glaserian fissure, and the fracture appears to split the meatus longitudinally, dividing it into an upper and a lower half, or, more commonly, a crack in the roof of the meatus is caused. In exceptional cases, where great force has occurred, the fracture has extended across the upper or anterior surface of the petrous bone into the internal auditory meatus. A smashing blow on the mastoid process has been known to fracture off the mastoid from the wedge of the petrous bone with injury to the lateral sinus. Fractures of the posterior fossa commence in the thin bone of the cerebellar fossa, and radiate into the lateral sinus groove of the same side; they then pass, either along the groove for the inferior petrosal sinus, or across the petrous pyramid near the inner edge of the lateral sinus, through the cancellous bone surrounding the mastoid antrum and semicircular canals, and behind and external to the internal auditory meatus. The various lines of fractures of the base of the skull can be seen in the museums of the Royal College of Surgeons and the medical schools; the tendency is for the fractures to take those lines indicated previously.

A moderate blow on the temporal fossa may produce a radiating fracture which cracks the roof of the middle ear, and blood effuses into the tympanum without rupture of the drum. No blood escapes into the auditory meatus, but the typical bluish drum can be seen on examination. Four such cases have been observed, and the only indication was deafness. This lesion would not have been detected without an aural examination, and, moreover,

* Read in the Section of Laryngology and Otology at the Annual Meeting of the British Medical Association, Cardiff, 1928.

in such a case blood can occasionally be seen in the orifice of the Eustachian tube if the nasopharynx is examined with a mirror. These patients completely recovered their hearing in three weeks, when the blood in the middle ear had disappeared and the drum was normal. More commonly the upper half of the drum is ruptured and blood freely escapes into the auditory meatus and down the Eustachian tube to the nose and pharynx. If it is accompanied by the leaking of cerebro-spinal fluid, the fracture is a severe and dangerous one, opening up the subarachnoid space, and all such cases in my experience have been fatal. It is also significant that in the few recorded cases where the internal ear has been fractured the escape of cerebro-spinal fluid has been present. A detailed and thorough investigation of the injuries to the middle and internal ear arising from fractures of the skull has been made by J. S. Fraser, and he gave at the Otological Section of the Royal Society of Medicine in 1916 a full description of the lesions found in a series of cases.

When haemorrhage from the ear is profuse and prolonged it is an indication that the middle meningeal artery or lateral sinus has been ruptured and urgent operative treatment is called for. The auditory meatus is sometimes a vent for this extradural haemorrhage, and should never be firmly plugged. Fortunately the lateral sinus is not often ruptured, and the pressure of the brain and the increased intracranial tension are sufficient to arrest venous bleeding. Haemorrhage from both ears is always an unfavourable sign, and the mortality is 66 per cent.²; whereas in cases of haemorrhage from one ear the mortality is 39 per cent. The average mortality of all cases of fracture of the base of the skull is about 40 per cent.

In those cases where a middle-ear suppuration has existed before the injury, the probability of a meningeal infection is very great and the prognosis is correspondingly unfavourable. In some cases the injury to the ear is followed by a local suppuration which materially adds to the damage, and spicules of bone from a comminuted fracture of the roof of the middle ear and meatus may be found. Mastoid suppuration has also been known to follow a fracture, and such a complication is liable to lead to a meningeal infection.³ Fraser reports a case of otitis media and meningitis which followed twelve months after the fracture of the skull. The injury undoubtedly facilitated the spread of the infection to the meninges. Some of these cases of local suppuration when seen late cannot be distinguished from the ordinary chronic middle-ear suppuration occurring without injury, hence the necessity for examining these patients as soon after the accident as convenient. The history of the absence of deafness and ear disease previous to the injury must be verified when the question of compensation arises.

A peripheral paralysis of the facial nerve has been recorded in about 46 per cent. of fractures of the middle fossa of the skull. It arises in the more severe type of injury, and in a few cases the eighth nerve is involved as well; but in the small number of cases of facial paralysis seen by me the eighth nerve escaped injury. The facial nerve is involved by the fracture as it passes through the facial canal, particularly near the geniculate ganglion. The onset of the paralysis is almost immediate, but if it is delayed this is probably due to haemorrhage into the facial canal, when recovery of the paralysis is more rapid. All the patients seen with paralysis recovered even after long intervals.

The degree of deafness arising from fractures of the skull shows considerable variation. In a number of cases no deafness was reported, and twenty-five cases seen by me on account of deafness were all of the middle-ear type. The four patients in whom effusion of blood into the middle ear was the only detected lesion all showed marked middle-ear deafness when first seen; they completely recovered their hearing with the disappearance of the blood at the end of three weeks, and it was then impossible to detect any lesion. Another case seen twelve months after a severe fracture of the skull, in which the sixth, seventh, and part of the third cranial nerves were paralysed, revealed only a slight degree of middle-ear deafness. The patients in whom fracture was followed by local suppura-

tion and inflammation suffered most loss of hearing; the deafness also became permanent. Any improvement in hearing usually occurs within about eight weeks from the date of the injury, and any deafness remaining after that period is permanent. I have not seen a case of genuine internal-ear deafness arising from concussion alone or from a head injury with such signs of fracture described previously. It is probable that the subjects of injury to the internal ear all die.

The treatment of these injuries to the ear is not always one of masterly inactivity, and a careful watch should be maintained for the onset of suppuration. Absolute rest in bed for a long period is essential, and the less that is done to the ear the better. The head is raised and a number of pillows are allowed. The auditory meatus is gently cleaned with spirit; a piece of sterile wool is lightly placed in the orifice and changed frequently. If there is any discharge of cerebro-spinal fluid the skin should be dried and smeared with an antiseptic ointment such as the dilute yellow oxide of mercury. Packing of the meatus with gauze, and, of course, syringing and drops, are to be firmly deprecated. If suppuration arises the whole aspect of the case is changed and vigorous methods should be adopted to establish free drainage. The drum should be incised if necessary, and there should be no hesitation in performing a simple mastoid operation at the slightest indication. The mastoid operation must be conducted gently, preferably by a motor burr or drill to avoid the jarring of the mallet and chisel.

The early recognition and treatment of suppuration is essential to the prevention of the dangerous meningeal or intracranial infection.

REFERENCES.

- ¹ Fraser, J. S.: *Trans. Roy. Soc. Med.*, Section of Otol., November, 1916.
- ² Rawling, L. Bathe: *The Surgery of the Skull and Brain*.
- ³ Harrison, W. J.: *Journ. Laryngol. and Otol.*, May, 1928, p. 341.

UNCOMMON ILL EFFECTS OF SALVARSAN TREATMENT.*

BY

W. J. O'DONOVAN, M.D.,

Physician to the Skin Department, London Hospital.

FATALITIES from salvarsan therapy do not stand in the front of the minds of the present generation of venereal specialists as they did amongst those who first used this drug with trepidation, and hope, in 1911. For when the arsenobenzols were originally introduced their use became associated with a formidable list of ill effects.

Experience has not demonstrated any certain method by which the type of patient unsuitable for salvarsan therapy can be picked out and treated in other ways;¹ neither old age, aneurysm, phthisis, chronic Bright's disease, high blood pressure, nor severe valvular disease of the heart can, in themselves, be considered as contraindications to its use. Ill effects may follow the administration of small doses²; they may occur at the beginning, at the middle, or at the completion of a full course of salvarsan treatment. No brand of manufacture seems to be exempt, though at times a run of toxic effects from the use of any one make compels the physician for expediency to omit this temporarily from his armamentarium. It would be wrong to say that these remedies are safe remedies, though I would be the last to express anything but gratitude for their invention and the highest appreciation of the way in which this country has been saved from an epidemic of syphilis by the energetic establishment of venereal clinics after the war by the Ministry of Health. The rarity of congenital syphilis must be most gratifying to the officials of the Ministry of Health, when they reflect upon this matter.

Dr. E. T. Burke has recently published a treatment of venereal disease (1927), in which four and a half pages are given on untoward effects of salvarsan therapy. The ill effects there referred to are:

"headache, shivering, vomiting, dyspnoea, palpitation, fainting, and so on. During the actual injection there may be nausea,

* A paper read in the Section of Dermatology at the Annual Meeting of the British Medical Association, Cardiff, 1928.