become innocuous when undue pressure from the boot has been removed through correcting the alignment of the toe. The plaster of paris splint should be placed direct on to the skin without the interposition of any padding, as recommended by Böhler in his treatment of other fractures.

The rounded flap incision, advised by Sir Robert Jones, with the convexity forward is sometimes preferable, as it allows removal of the poorly nourished skin over the bursa, but I find a straight incision more generally applicable, passing straight through the bursa, the two halves of which can then be peeled off the bone and overlying skin. Usually no skin needs to be sacrificed; the thinned portion having its blood supply less interfered with by a straight incision than by one that fashions a long flap.

J. Stewart of Leeds and M. Brandes of Dortmund, impressed by the disastrous sequels that not infrequently follow removal of the head of the first metatarsal, have resected the base of the proximal phalanx with decidedly better results, since by this measure the tripod of the foot is left intact. But even though less destructive this operation still falls short of the ideal.

The causes of hallux valgus have not been touched upon, since they are well-known, nor is there any necessity to labour the after-treatment. It has been my chief endeavour to show that the operation of cuneiform osteotomy, so strongly advocated by the late Sir Robert Jones, is essentially sound in principle and gives the nearest possible approach to a 
restitutio ad integrum.

On the other hand, the operation of excision of the head of the metatarsal bone violates surgical principles, being crude in conception, ruthless and destructive in execution and leaving behind at the best a far from perfect result, at the worst a condition as disabling as that which the operation set out to remedy.

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**ON FITS AND FAINTS.**

By NORMAN KLETZ, M.B.(Manc.), M.R.C.P.(Lond.),

Physician to Ancoats Hospital; Physician and Hon. Pathologist to Stockport Infirmary.

It is a frequent experience of medical practice to be called upon to explain "attacks" to which a patient has been subjected, and to indicate their nature and significance. Sometimes the "attack" has been personally observed; more frequently its character must be deduced by careful questioning. Whatever their features, such attacks invariably arouse great anxiety, and, it is, therefore, of paramount importance that we should have at least a general knowledge of the possibilities.

Two types will be excluded from this consideration—infantile convulsions and vertiginous attacks. The vertiginous attack has a fundamental character which at once indicates its nature, though the establishment of its cause may well demand very thorough analysis and investigation.

The manifestations to be considered fall into three main groups:—

(1) Those arising primarily in some dysfunction of the nervous system—Fits.
(2) Those dependent upon dysfunction of the circulatory system—Faints.
(3) Hypoglycaemic attacks, etc.

*Based on Post-Graduate Lecture delivered at Ancoats Hospital, Manchester, on October 26th, 1933.*
I.—Fits.

Fits are of the following main types:—

- Epileptic or Epileptiform.
- Pyknolepsy.
- Narcolepsy.
- Cataplexy.

The classical major epileptic fit is a convulsive seizure which may occur abruptly without warning or be ushered in by premonitory symptoms—the aura—of motor, sensory, gustatory, psychic, etc., type. The sufferer is unconscious and passes through a phase of generalised tonic contraction, followed by a phase of more or less generalised clonic movement, succeeded by a variable period of quiescence before recovery occurs and consciousness is restored. The sphincters are frequently relaxed during the attack. Such are the essential features of the major convulsive fit, but all epileptic attacks are not of this type. They vary both in causation and in their manifestations. It is scarcely necessary to emphasize the fact that in many cases epileptiform seizures, particularly of a convulsive type, are dependent upon definite organic causes and structural disease, e.g.:—

- **Trauma** causing fracture of the skull, hæmorrhage or cerebral contusion.
- **Toxic Factors**: Such toxins may be endogenous—products of distorted metabolism as in uremia, eclampsia, cholaemia, or exogenous—lead, absinthe, etc.
- **Vascular**—resulting from spontaneous hæmorrhage, particularly meningeal or sub-arachnoid, or, consequent upon acute hypertension and cerebral oedema. It is probable that the convulsive attacks which occur in acute nephritis are of this type, a point of importance from the aspect of prognosis and treatment.
- **Inflammatory**: Acute and chronic.
  - Meningitis.
  - Encephalitis of various types.
  - Brain Abscess.
- **Neoplastic**: Apart from the Jacksonian fit with its localising value, general convulsive seizures may occur as a symptom of intracranial neoplasms. Such fits are often purely an expression of increased intra-cranial tension. They may, however, be specifically associated with frontal tumours.

Attention must also be directed to the epileptiform seizures of general paralysis of the insane and such relatively rare affections as cysticercus infection.

It is, therefore, an obvious statement that at any age whenever fits occur, the history must be carefully considered and a detailed clinical examination undertaken with these specific possibilities in mind in order to establish or eliminate the presence of organic disease. Especially is this so when attacks first manifest
themselves in middle adult life, an age at which syphilis and frontal tumour must be particularly suspected. So far as frontal tumour is concerned, its direct symptoms may be for long so slight and its signs so few that it is perhaps advisable to support clinical evidence by skull X-ray examination and ventriculography before excluding the possibility of its presence. The following case may be quoted in illustration:

"A man, aged 34 years, had two major epileptic seizures in February, 1932. There was no previous history of such attacks, nor any family history of epilepsy or its variants. He was examined by his doctor and a skilled neurologist. No clinical signs were discovered, and, syphilis having been excluded by the appropriate serological tests, a diagnosis of idiopathic epilepsy was made. No further attacks occurred. At the beginning of November, 1932, he commenced to have severe headaches with vomiting and a tendency to drowsiness. He was seen on November 18th, 1932. There was then found slight weakness of the face on the left side, together with diminution of the left abdominal reflex and marked papilloedema. Careful questioning also elicited that between February and November nothing had been noticed except that he was occasionally forgetful. The diagnosis of right frontal tumour was made and confirmed post-mortem, death having taken place before any attempt could be made to operate."

In the majority of cases in which epileptic attacks occur, no organic cause can at any time be demonstrated. Such cases are usually grouped together as idiopathic epilepsy. Though this term has its uses, it must not be regarded as denoting a uniform clear-cut state. Epilepsy is not a disease, nor is it a definite entity. Indeed, it is a matter of general clinical experience that the course of epilepsy is extremely varied and that, whilst in some cases the outlook is serious, others do not carry with them the grave and hopeless prognosis so frequently expressed. Its manifestations, too, are multiple and varied suggesting a similar variation in neural localisation and significance. Hughlings Jackson has expressed the view "that many different epilepsies are grouped under the one term epilepsy; if the paroxysms are different, the seats of the discharging lesion must differ". One should therefore speak not of epilepsy but of the epilepsies. Edwin Bramwell has recently written that "the tendency to speak of the epilepsies is, in itself, an advance, since it expresses the recognition of a variety of conditions which present variations from the diagnostic, prognostic and therapeutic standpoints".

When one comes to consider the various forms of epilepsy it is clear that no one of the characteristics of the classical major attack is essential. For example, unconsciousness is a variable feature; a severe Jacksonian fit may occur without any loss of consciousness, or, the sole representation of a fit may be an occasional, transient "conscious blank", as in petit mal. Even the motor manifestations are inconstant. Fits occur characterised purely by attacks of tonic spasm without any clonic movements whatsoever. Such tonic epilepsy is more frequently the result of focal organic disease.

Another motor variant is myoclonic epilepsy. Myoclonus is an interesting type of involuntary movement, characterised by rapid, shock-like contraction of muscle or muscle groups. It is not by any means necessarily associated with epilepsy. It has occurred as a manifestation of epidemic encephalitis, more particularly involving the rectus abdominis muscles; it also occurs in the chronic multiple form as paramyoclonus multiplex. Myoclonus may, however, recur paroxysmally
as the sole representation of epilepsy. It usually involves a limb producing irregular twitches. It may also be associated with major fits as in the familial myoclonus epilepsy of Unverricht. A not dissimilar condition is that of epilepsia partialis continua in which the movements, though of a twitching character, are constantly present; major fits occur periodically.

Again, fits, though motor, may not be kinetic, that is, may not produce any movements. A patient with or without loss of consciousness may have attacks of motor immobility, either localised or generalised—so called inhibitory or akinetic epilepsy. It will be at once appreciated that in some cases it will be difficult to differentiate such attacks from syncope and hysterical states. A case will be described of such akinetic attacks, associated with other interesting phenomena, in which a tumour of the motor area was found.

Jacksonian fits: The general features of this type of fit are familiar; the constant starting point, the spread in accordance with accurate anatomico-physiological localisations, etc. Certain points must, however, be emphasized. Firstly, a Jacksonian fit may be localised to a very limited area of the body. Such attacks can be easily misinterpreted. I have seen such a localised fit confined to one side of the face regarded for some time as a tic. The following case illustrates two points:

"Case. A woman, aged 32 years, between September, 1924, and January, 1925, had four attacks in which she suddenly fell motionless to the ground—akinetic epilepsy. From January, 1925, she began to have paroxysmal attacks of stiffness and contraction in the right forearm and hand—presumably localised Jacksonian attacks. In April, she commenced to have periodic pain in the left frontal region, and when examined showed a slight right sided hemi-paresis with moderate papilloedema. A tumour was removed from the left motor area."

Secondly, Jacksonian fits may be purely sensory and have no motor component.

"Case. A man of 45 years had complained of headache and of paresthesia and numbness occurring paroxysmally with a fixed starting point and a typical Jacksonian spread. There was marked papilloedema and slight hemi-paresis. A cortical tumour was found."

Other types of sensory epilepsy occur; for example, reflex epilepsy, i.e., cases in which some type of epileptic attack follows upon some sensory stimulation—peripheral, auditory, etc. Kinneir Wilson describes a case in which "the hat elastic under a little girl's chin slipped up, hitting the nose; she went off into an epileptic fit. This accident led to the discovery that flicking or tapping the nose always started the attack". Clearly such a case might readily be regarded as hysterical.

Purely sensory fits may also occur. It is a well known fact that in many cases major epileptic convulsions may be preceded by an "aura" of a sensory type—gustatory, auditory, etc. Sometimes such sensory experiences represent the whole fit—so called sensory epilepsy. A particular type of such sensory fit must be specially mentioned—the uncinate fit, a temporal epilepsy. The peculiar importance of this type of fit is its association with organic disease and its value
in localising the lesion to the temporal lobe. The fit is "olfactory". The following case is an illustration:

"A man of 52 years was seen on October 9th, 1933. For three months he had been complaining of paroxysmal headache over the right frontal region. He had also experienced certain attacks: a sickly feeling comes over me and then I smell something which I cannot describe and I do not want to talk about’. He was found to have definite choke of the right disc and slight choke of the left, together with slight weakness of the left face and an extensor plantar response on the left lower limb. This is clearly a case of tumour involving the right temporal lobe."

The above types of epilepsy do not exhaust all the possibilities but merely represent the more important ones.

Pyknolepsy: This remarkable affection occurs in children, first appearing between the fourth and the twelfth years. The attacks are identical with those of petit mal, but their frequency is infinitely greater, occurring even up to a hundred per day. The duration of the period of liability is variable but all cases cease at puberty. The condition is, therefore, a benign one, leaving no physical or mental defect. Unfortunately, no drug or other treatment succeeds in controlling the attacks during the period of incidence.

Two other types of neural attack remain to be mentioned.

Narcolepsy: This is a condition characterised by paroxysmal attacks of "sleep" occurring abruptly in the daytime. During the attack the patient is usually unconscious. Such unconsciousness is not complete but merely of that degree which occurs in natural sleep, namely that he can be roused by those stimuli which would rouse a normal sleeping individual. In other cases, whilst in the attitude of sleep and more or less immobile, the individual is aware of his surroundings. In many of these cases the basis is functional.

"Case. A young married woman, with two children, had also several relatives living in the house. Her domestic difficulties were intense. She began to have narcoleptic attacks. No signs of organic nervous disease were found and enquiry elicited the fact that the 'attacks of sleep' always occurred when affairs were particularly difficult. The lightening of the burden and the improvement in the domestic atmosphere, achieved by removing the relatives, was followed by cessation of the attacks."

In other cases there is definite localised organic disease, involving the sub-thalamic region—inflammatory or neoplastic. Epidemic encephalitis may be a cause. Other cases, however, are idiopathic. The onset may be a sudden or preceded by an aura. The attack varies in duration from a few minutes to hours.

Cataplexy: This relatively rare affection consists of attacks in which there is abrupt loss of use of the limbs, causing the sufferer to fall abruptly to the ground; consciousness is not invariably lost. The essential feature of the cataplectic attack which differentiates it, for example from inhibitory epilepsy is that it is usually provoked by some emotion such as laughing or crying. Cataplexy and narcolepsy often occur in the same individual.

Before leaving the question of attacks of nervous origin, mention must be made of hysterical attacks which can be so confusing and lead to errors in diagnosis. Such errors are particularly liable to occur unless personal observation of the...
attacks is possible. Apart from any features thus discovered, the circumstances of the attacks and the patient’s general behaviour and mentality may give a clue. So far as apparent major convulsive attacks are concerned, the occurrence of tongue biting and relaxation of the organic sphincters are suggestive of a true epileptic seizure. In many cases differentiation may be for long extremely difficult.

II.—Faints.

The occurrence of faints or syncope attacks is always fraught with great anxiety and is a very frequent cause of patients seeking medical advice. In most cases a faint immediately arouses the fear of “heart disease”. Actually, though such attacks may be associated with serious cardiac affections, this is by no means necessarily so. Indeed, it may be said that in the majority of cases such attacks are not of serious significance. As a general definition, syncope may be described as a loss of consciousness due to deficient circulation through the brain. One important type of cause must be immediately mentioned, namely, cryptic hæmorrhage, such, for example, as may occur in the form of hæmorrhage into the bowel from a duodenal ulcer.

The severity of the faint and the persisting pallor of the mucous membranes, the rapid pulse and failure of reaction will give a clue to the serious underlying cause. In the case of ulcer the subsequent melæna establishes the diagnosis.

Fundamentally, a deficient supply of blood to the brain is due either to vascular factors, diminishing the amount of blood carried to the heart, or cardiac factors, leading to deficiency in output from the heart. Hæmorrhage acts by diminishing the delivery of blood to the heart; it is separately mentioned because of its special significance from the point of view of prognosis and treatment.

It will be immediately apparent that the two types of syncope—vascular and cardiac—have different implications of seriousness. Further, when syncope attacks occur in association with definite organic heart disease, the presence of such heart affection does not enhance the seriousness of the syncope, nor, equally, does the occurrence of the syncope make worse the outlook of the heart condition. Non-serious attacks may occur in cases of organic heart disease, as they do with even greater frequency with a healthy heart. Following Lewis, there are two types of what may be termed vascular syncope:

A.—Postural Faintness: It is a matter of common experience that if we rise abruptly from the recumbent to the erect posture, we may feel a transient light-headedness. This is due to the fact that, in such change of posture, there is a slight fall of blood pressure which is rapidly compensated. In some individuals, and, under certain circumstances, such compensation does not take place sufficiently rapidly, so that faintness, and even transient unconsciousness, may occur. This is particularly so in middle aged and elderly individuals, especially after heavy meals or in an overheated atmosphere. The actual mechanism is that of over filling of the “abdominal reservoir” with consequent temporary inadequate return of blood to the heart. The relationship to change of posture is the essential diagnostic feature of this type of attack.

B.—Vaso-vagal attacks: Most fainting attacks are of this type. They often occur in individuals recovering from some infection or debilitating condition, but they also occur in perfectly healthy individuals.

The actual attack may be precipitated by fatigue, excessive heat, painful stimuli, emotional influence (pleasurable and non-pleasurable), unpleasant tastes,
smells, sights or even loud noises. Frequently, for example, one sees a big, healthy, robust man faint at the sight of blood or at the tiny prick of a fine needle.

The attack may be abrupt, the sufferer falling without warning and even injuring himself; in other cases the onset may be more gradual leading to a gentle slipping to the ground. Often there is a preliminary sensation of a sinking feeling in the abdomen or actual nausea. Blood pressure and pulse rate both fall. There is extreme pallor, flaccidity of the limbs, dilatation of the pupils, loss of consciousness and disappearance of the conjunctival reflex. The respirations are slow and slight clonic movements or twitchings may occur. Sweating is a constant feature at some stage of the attack. The duration is rarely more than a few minutes. The actual mechanism of the attack is that of vagal inhibition and vasodilation.

These vaso-vagal syncopes may well arouse great alarm, but, in actual fact, they are not of grave significance.

The second great group of faints can be classified as cardiac syncope. Though infinitely less frequent than those already described, cardiac syncope is of more serious significance, in so much as it implies some deficiency of heart function. There are two main types:—

(1) *Those associated with profound slowing of the Heart*. As has already been indicated, slowing of the heart is a constant association of vaso-vagal attacks. Further slowing of various degrees may be caused by numerous factors of non-serious import. The type of condition referred to here is such as occurs in cases of high grade interference with the conductive mechanisms of the heart—auriculo-ventricular block. Cardiac syncopal attacks are especially liable to occur where there is complete block and auriculo-ventricular dissociation leading to periods of profound slowing of ventricular contraction, even to the extent of eight beats per minute, or even phases of temporary ventricular arrest. Such attacks—the so-called “Stokes-Adams syndrome”—vary considerably, both in frequency and severity. When prolonged they are often associated with clonic movements. A period of ventricular arrest exceeding a minute or so is incompatible with recovery.

(2) *Those associated with profound acceleration of the Heart Beat*. To induce syncope a ventricular rate of over 200 per minute is usually necessary. Such rates may abruptly and temporarily occur in attacks of simple paroxysmal tachycardia or auricular flutter. Whether the primary cause be profound slowing or intense acceleration the ultimate effect is a grossly inadequate output of blood from the heart. It will therefore be evident that the recognition and differentiation of the various types of faint depends upon the attendant circumstances, the rate of the heart, the rhythm of its beat, the association of sweating, etc.

### III.—Hypoglycæmic Attacks, etc.

The final type of attack to be considered is the hypoglycæmic attack. It is a well known fact that in diabetics, who are taking large doses of insulin, there is a liability to the development of attacks due to or associated with marked lowering of the blood sugar—so-called hypoglycæmia. Such attacks tend to occur between two to eight hours after a dose of insulin and are relatively abrupt in onset. There may be a feeling of uneasiness, of giddiness, of heat and palpitation, a faintness
and perhaps tremor. If the blood sugar continues to fall there is restlessness and sometimes a state like that of drunkenness. Ultimately the patient becomes unconscious, and, if unrelieved, passes into a state of actual coma.

Similar attacks have been described as occurring spontaneously in non-diabetic individuals. In some such cases a tumour of the pancreas has been found, the mechanism being that of excessive insulin production by the growth. In other cases the liver is at fault. Such abnormal functioning of the liver may be of diametrically opposite types. There may be deficient function leading to inadequate maintenance of glycogen reservoirs or liver over-efficiency, as a consequence of which there is an excessive storage of carbohydrate as glycogen leading to impoverishment of the blood and tissues in sugar. No doubt fainting attacks due to such spontaneous hypoglycaemia are rare but the possibility should be kept in mind in doubtful and unusual cases of recurring faints, particularly with associated features such as those described. The diagnosis can, of course, only be established by estimation of the blood sugar during an attack. In some cases in which a tumour of the pancreas has been found, its operative removal has been successful in controlling the attacks; in other cases some degree of control can be maintained by glucose feeding.

CHRONIC CONSTIPATION IN INFANCY AND CHILDHOOD.*

By ROBERT HUTCHISON, M.D., F.R.C.P.

LADIES AND GENTLEMEN,

I have been asked to lecture to you this morning on Chronic Constipation in Infancy and Childhood. I am afraid it is a dull subject, but there is no doubt that it is an important one in practice, because constipation is not by any means an uncommon trouble in childhood, as it is also in adult life, and it must be admitted that a certain number of cases of constipation in adult life have their origin in neglected constipation in childhood.

It will be convenient, for purposes of description, to speak separately of constipation in infants, and constipation in older children.

Taking infants first, you will remember that the normal infant has three or four stools in the 24 hours; but it would be a mistake to suppose, because a child has fewer motions than that, that it necessarily has constipation, provided always that the motions are normal in character and consistency. There are infants who have only one stool in 24 hours, and yet will be well and will gain weight. This fewness of motions—the consistency remaining normal—is particularly apt to occur in breast-fed children. You will see in books the statement that constipation is a vice of bottle-fed babies. I have not found that statement to be true; this particular scarcity of motions is particularly apt to occur in the breast-fed child. One reason for that probably is that breast milk is so easily digested and so completely absorbed that it leaves little residue; and where breast milk is not too abundant there may be so little intestinal residue left that one motion in the 24 hours is sufficient. Others of the cases, where the motions occur at too long intervals without being otherwise abnormal, are due, I think, to what you may speak of as a sluggishness of the rectal reflex; I think there is no doubt that some little babies have a "lazy

*A Post-Graduate Lecture delivered at the Hospital for Sick Children, Great Ormond Street, London, on October 30th, 1933.
On Fits and Faints

Norman Kletz

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