Section of Neurology

President—JULIAN TAYLOR, C.B.E., M.S., F.R.C.S., F.R.A.C.S.

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DISCUSSION ON FAINTS AND FITS

Sir Charles Symonds: The symptom common to fits and faints is loss of consciousness which, by definition, is of rapid onset and brief duration. By definition again the word "faint" commonly implies a loss of consciousness due to a temporary failure of the cerebral circulation of postural or reflex causation, not the result of heart disease. This definition would exclude from our consideration attacks caused by heart block or other cardiac disorder, and also those due to hypoglycæmia.

The word "fit" may be used to indicate that the loss of consciousness is attended by convulsions, or in a larger sense to include an attack or seizure of any kind that is epileptic. The wider meaning is clearly more appropriate. When, therefore, I speak of fits I shall mean epileptic attacks. The definition of epilepsy is not easy, but Hughlings Jackson's physiological concept of "occasional, sudden, excessive, rapid, and local discharges of grey matter" will serve—perhaps as well to-day as when it was first written. The confirmation of this hypothesis by the E.E.G. is indeed remarkable.

Having regard to these definitions there appears to be a distinction between fit and faint of fundamental importance. In the fit the disorder of function is at the onset local: it is somewhere—and it may be almost anywhere—*in* the brain. It is therefore to be expected that the symptoms of a fit will show great variety, depending upon the parts in which the discharge arises. That this is abundantly true is confirmed by clinical experience. The number of epileptic variants is legion. In the differential diagnosis it is often possible in a moment to decide that an attack is a fit, not a faint, by reason of its local sign, generally the aura described by the patient, sometimes the observation by others of particular movements or localized spasm. But there are many varieties of epileptic attack in which there is no clinical evidence of local origin, or at any rate none which distinguishes the attack absolutely from a faint. The aura is present in little more than a half of the attacks of idiopathic epilepsy. The spread of the discharge in a convulsive attack is often so rapid that the movements are at once generalized, and, even more important, the epileptic discharge may begin in those parts of the central nervous system which are also involved in fainting, a subject to which I shall return later.

By contrast with the local origin of the epileptic attack there is in a faint involvement of the brain as a whole. This does not, of course, mean that the brain is equally affected in all its parts at the same time by a particular degree of anoxia. There seems to be an order of susceptibility so that some parts give out before others. The retina, for example, fails early and so do the centres for postural tone. Dimness of vision and loss of equilibrium, therefore, happen in a faint before loss of consciousness. Thus in a faint, if the onset of anoxia is not too abrupt, there is a certain order of lost function which is characteristic. This is true of most faints but there are striking exceptions. In the disorders known as postural hypotension and carotid sinus syncope, which both fall within my definition, the onset of the faint may be abrupt, and this is also true in some cases of reflex—I mean emotional—fainting. The sudden onset of pallor, unconsciousness and falling then closely resembles the symptoms of a minor epileptic attack. The resemblance is even more striking when a faint is attended by convulsive movements. It is well known that these occur when cerebral anoxia is of sufficient degree and duration. They were recorded in 7% of a series of 362 faints in blood donors, whose symptoms were analysed in a Medical Research Council Report. There is no doubt that in a faint of reflex origin ventricular standstill may occur and when this happens loss of consciousness ensues within 5 to 10 seconds, and if the heart stops for 15 or 20 seconds there are convulsive movements.

This happened in a patient whose case I have reported elsewhere, a young man who was liable to faint at the sight or thought of illness or even when examined by a doctor. While serving in the Army he was referred to Captain Michael Ashby with the diagnosis of epilepsy confidently made by a medical officer who had witnessed an attack. While Captain Ashby was examining the patient he found that his pulse was beating about once in 3 seconds. I quote verbatim his note on what followed. "I had only time to take three more beats when it stopped completely, confirmed by auscultation, for 45 seconds, during which he had a fit, rolling eyes, rigidity, and a few convulsive movements. The pulse stopped before the fit by about 10 seconds." This was an extraordinarily complete observation of cardiac standstill in a faint. The electrocardiogram in this case was normal, and in a subsequent attack induced by Dr. Denis Williams while an E.E.G. was recorded there was no evidence of epileptic activity, so that I do not think there can be any doubt that this man's attacks were faints.

To return now to the subject of epileptic variants, there is little doubt that the discharge may be localized in those parts of the brain, wherever they may be, upon which the state of consciousness depends. The epileptic attack often consists of nothing else but a loss or disturbance of consciousness. When together with this loss there is loss of postural tone with falling, the symptoms of the attack are indistinguishable from those of a very sudden faint. But if there is loss of consciousness without falling I believe we are right in supposing the attack to be epileptic, for in a faint the two go together.

Other epileptic variants which may resemble faints are those which have their origin in visceral centres. There is little to be said on this subject which has not already been stated by Kinnier Wilson (1928) in his classic paper. Pallor, nausea, palpitation, and dizziness separately or in combination may usher in an epileptic attack, or may occur as its sole manifestations.

A woman aged 20 was referred to me for attacks which had begun three years before. She would suddenly feel sick and dizzy, with buzzing in her ears and inability to hear clearly. If possible she would lie down for a minute or two and would then feel quite well. She had never fallen and was positive that she did not lose her senses. Attacks had been witnessed by her parents. In one of them she was driving a car and said to her father "Oh, Daddy take the wheel". She turned pale and had recovered in a minute or two. On another occasion she was in the garden with her mother, said she felt faint, lay down on the grass for a minute, and then said she was all right. Later, however, she had an attack while alone in her bedroom, beginning in exactly the same way. She went to lie down, but lost her senses and was found unrousable twenty-five minutes later, having bitten her tongue and passed water. Enquiry revealed that for the past four years about three times a year she had woken in the morning with a headache, a sore tongue and sometimes a wet bed, feeling as if she had had a nightmare. Her subsequent story has been that of epileptic attacks, almost but not quite controlled by phenobarbitone and epanutin.

I think it fairly certain that the little attacks, which had been diagnosed as faints, were, in fact, epileptic.

One cannot mention visceral epileptic variants without referring to what Gowers (1907) called vaso-vagal attacks, a designation which has been widely misunderstood, mainly because the term vaso-vagal attack was much later used by Sir Thomas Lewis to describe fainting. Gowers used it for prolonged attacks comprising symptoms which he thought must be referable to a disturbance in the medullary centres. "The attacks", he states, "are never really brief, they seldom last less than ten minutes and more often continue for half an hour or more." The symptoms, according to Gowers, might include epigastric discomfort, respiratory distress, substernal discomfort, coldness, pallor and often a sense of impending death. These attacks are, I believe, quite rare and are not to be confused with so-called anxiety attacks. Kinnier Wilson in the paper already mentioned suggested that they were epileptic variants of visceral type, arguing that their extended nature was no insuperable obstacle in the way of acceptance of the hypothesis.

I have had under my care at hospital a woman who had attacks of this kind at infrequent intervals occurring during the day and lasting an hour, each being followed during the subsequent night by a major epileptic attack in her sleep.

It is of course the duration of vaso-vagal attacks which makes them unlike epilepsy but as an example of the manner in which a single visceral symptom of epilepsy may vary in duration I take substernal pain.

A man began to have attacks at the age of 29 and had half a dozen in the next five years. There was a sudden onset of what was described as a vice-like pain between chest and back, which rapidly increased in severity and after a few seconds he became unconscious for five minutes. In one attack he bit his tongue. Dr. Denis Williams on two occasions found an abnormality in the E.E.G. indicating a long-standing benign lesion of the left hemisphere.

A woman aged 37, who had previously suffered from migraine, had her first epileptic attack while at a theatre. It was preceded for an hour by a pain in her chest like indigestion. She then told her husband she was going to faint and had a fit in which she bit her tongue and passed water. On a subsequent occasion she experienced substernal pain all day, then felt a sudden rush from her stomach to her head (an epigastric aura) and had a fit.

The transition from this case to the next, which is an example of Gowers' vaso-vagal attacks, is I think easily followed.

A married woman, aged 25, began to have infrequent and causeless attacks which were all of the same kind. There was a sudden onset of substernal pain like toothache, everything started to swim, and her head sweated. She lay down and remained in this state (the substernal pain continuing) for about two hours with a feeling as if she were going to die. There were two more episodes of this kind in the next three months. Finally she had a more serious attack which brought her to see me. She was on holiday taking the air before breakfast when there was a sudden onset of substernal pain with sweating at the temples and sound coming and going. She just had time to say to her husband "Look out" before she fell unconscious. He stated that she was pale with stertorous breathing, stiff for the first quarter of a minute, them limp, and remained unresponsive for five minutes. She was confused for ten minutes afterwards and had no memory of being carried up to bed. After she recovered her senses she continued to have substernal pain for two hours and during this period felt giddy if she attempted to sit up. Neither I nor any of my colleagues could find anything wrong with this woman. E.C.G., E.E.G., fasting blood sugar, blood sugar curve, response of pulse-rate and blood pressure to posture and effort were all normal.

This has been a digression from the main theme of our discussion. Vaso-vagal attacks are not faints, nor are they in any ordinary sense fits, but the observations which I have put forward support Kinnier Wilson's argument that they may be epileptic.

Perhaps the most important distinction between faint and fit is the setting of the attack. Faints very rarely occur except in the erect posture. If an attack occurs after long unaccustomed standing, or as the result of sudden emotion or pain, in a hot and stuffy atmosphere, or after loss of blood, it may be presumed to be a faint unless there is evidence to the contrary. If it occurs without any of these causes it is probably a fit. Yet again, however, the distinction is imperfect. Epileptic attacks may be precipitated by various causes of which emotion is one. The malaise of infection may be another. In the following case both precipitants were evident.

The patient was a man of 61, whom I saw for an entirely different complaint, and the details which follow were obtained from his past history. He had as he said "fainted" on many occasions. The first was in his teens when he was taken to the doctor with influenza, the second at 19 after a cycle accident. He was not injured, but very much upset, and lost his senses suddenly fifteen minutes later. The next occasion was a year afterwards when he went to have a tooth out, and, feeling, as he said, in a blue funk, lost his senses and fell before getting into the dentist's chair. In this attack he passed urine. There was a similar episode at the dentist's a year later. Again he passed water. There were no more attacks precipitated by emotion, but on about a dozen occasions he had fainted at the onset of a febrile illness, influenza or bronchitis. The sequence was always the same. He would begin to yawn and continue yawning for ten to twenty minutes. If he could keep walking about this might end without further incident, but if he gave way and sat down his vision would suddenly become grey and he would lose his senses for a minute. In almost all these attacks he passed urine.

These attacks, I believe, were epileptic for two reasons. The first is the occurrence of involuntary micturition in nearly all the attacks. This may occur in a faint if the bladder happens to be full at the time, but its regular occurrence always suggests an epileptic discharge involving the centres for micturition. The second reason is the prodromal yawning, recognized by Gowers (1901) and again by Kinnier Wilson (1928) as a precursor of epileptic attacks. I have several examples of this in my own notes of undoubted epileptics.

There is next to be considered a rare but important group of patients who begin by having attacks in childhood or adolescence which we diagnose confidently as faints, but who go on to have attacks which we are sure are epileptic. The earlier attacks have the usual causes for a faint, but the liability appears excessive, the attacks are more frequent than usual and continue to a later age and when they have continued long enough we are not altogether surprised when we are confronted with the story of an attack this time without cause and characteristic of epilepsy. In these cases then we observe excessive liability to fainting followed by an excessive liability to epilepsy. The word excessive may be equally applied to both. Anyone may faint or have a fit with sufficient cause. Why do some persons have faints or fits with so little cause or no apparent cause? The answer in the case of the epileptic is that there are nervous centres which are unstable. This is the trigger mechanism for the attack. Of the causes that operate to pull the trigger we know very little. In the case of the fainter on the other hand we know a good deal about the causes that pull the trigger, and we know that when the trigger is pulled there is bradycardia or fall of blood pressure to explain the symptoms that follow. But where is the trigger itself? Probably in the central nervous system. Fainting in response to emotion clearly suggests this localization. Again in a faint which results from prolonged or unaccustomed standing what is it that gives way? Surely the reflex mechanisms responsible for maintenance of heart-rate and blood pressure, and where are these except in the central nervous system? In the syndrome of postural hypotension, which provides the extreme example of postural fainting, there is a good deal of evidence for the existence of a trigger mechanism at or near the hypothalamus (East and

Brigden, 1946). It seems probable, therefore, that for faints as well as fits an essential link in the chain of causation is an unstable nervous mechanism. If this be so it would not be surprising if we sometimes found both kinds of instability present in the same person. Nor I think would it be surprising to find that with the passage of time the instability responsible for fainting became less and that responsible for epilepsy greater. This kind of thing sometimes happens in persons who have migraine in their youth and as they grow older exchange it for epilepsy.

The carotid sinus syndrome is also of interest in relation to the occurrence of faints and fits in the same person. In this malady pressure upon the sinus, according to Ferris and others (1935), may produce faints, with bradycardia or fall of blood pressure sufficiently abrupt to cause loss of consciousness and sometimes convulsions: or it may cause these symptoms without any adequate cardiovascular derangement to explain them. Thus it would appear that the specific stimulus in this syndrome may produce either reflex fainting or reflex epilepsy.

The general trend of these remarks has been towards the conclusions, first, that there is no absolute means of distinguishing between a fit and a faint unless the attack can be observed in detail with estimations of pulse-rate and blood pressure before and during the episode, and second, that even though fits and faints may be clinically distinguishable they are closely related in their dependence upon the instability of nervous mechanisms.

REFERENCES

East, T., and Brigden, W. (1946) *Brit. Heart J.*, **7**, 103.

Ferris, E. B., Capps, R. B., and Weiss, S. (1935) *Medicine*, **14**, 377.

Gowers, W. R. (1901) Epilepsy. London.

—— (1907) The Borderline of Epilepsy. London.

Wilson, S. A. K. (1928) *J. Neurol. Psychopath.*, **8**, 223.

Dr. Denis Williams: "Familiar as fainting is, adequately as we seem to know it, there is much in it that we do not know. Our knowledge is enough to obscure our ignorance. The most obtrusive feature of complete cardiac syncope is the loss of consciousness which results, evidently due to the failure of the action of the heart which precedes and attends it. But the loss cannot be the direct effect of the cardiac failure, because consciousness is not the result of the circulation of the blood. To say this is to state an obvious truism, for the two are totally different in nature. The loss must be immediately due to a state of the nerve elements of the brain produced by the change in the circulation. We are apt to overlook this when we think of the process of fainting, but its recognition is of great importance because consciousness may be lost from other causes. It is the most common feature of the epileptic seizure. Not long ago it was thought to be a constant feature; without such loss an attack was said not to be epileptic. We now know that minor attacks are common in which consciousness is only dimmed: sometimes hardly a ruffle on its surface attends the sensation which constitutes the slightest form of attack. Still, in the pronounced form of each condition, in fainting and in epilepsy, loss of consciousness is a dominant feature. The fact is of practical importance in diagnosis, because it often makes their distinction difficult, and sometimes causes a mistake. For another reason, also, the loss of consciousness is important. We do not know in either malady the nature of the process in the nerve elements on which the symptom depends. We do not know whether it is the same in the two or is different. If we can trace any relation between the affections it will constitute some evidence on the question, evidence at least suggestive. For this reason also the study of fainting in relation to epilepsy is important."

Those words were written by Sir William Gowers over forty years ago, and they make it clear that we are discussing the problem in the same kind of way to-day as he did then.

Sir Charles Symonds has suggested that there may be a common link between epilepsy and syncope, although a precise distinction can normally be made between the two conditions upon clinical grounds. In considering any relationship between the two, it is of the utmost importance to keep such a distinction clear, for it is all too easy by elision to recognize a faint as a fit, and vice versa, and so seem to establish a relationship in any one case which does not, in fact, exist. Any experimental study of the relationship between epilepsy and fainting must be based on the certain recognition of an epileptic attack and a fainting attack as entities, and it is because of the failure to do so that so much work must be discarded. The criteria that Sir Charles Symonds has laid down are those which are readily acceptable to most of us, and such criteria should be employed in any experimental or statistical survey of the subject.

Kershman (1949) considered the relationship of "Syncope and Seizures". He took 114 patients who had a history of syncopal attacks or similar episodes without loss of consciousness. All of them had abnormal electroencephalograms between attacks but none of them had had a definite convulsive seizure. 65% had a diffuse dysrhythmia in the E.E.G. and Kershman concluded that "the form of the pathological E.E.G. abnormality and the character of the spells suggested a subcortical and probably hypothalamic origin for the attacks". He concluded that these "syncopal spells" were "a mild form of idiopathic epilepsy" and stated that "these spells were essentially the same as the vagal and vaso-vagal attacks described by Gowers, and the E.E.G. disturbances indicate that they are fundamentally epileptic in character".

At first glance all this might suggest that syncope is, as Kershman has suggested, a special if mild form of epilepsy. But the difficulty is that many of the cases included in his series clearly had epilepsy. Consider the first of his illustrative cases, that of a man who had an attack of unconsciousness while at breakfast, in which he suddenly felt a choking sensation, stood up and fell unconscious. He had urinary incontinence and recovered fifteen minutes later, weak and dizzy. In his next attack he was sitting, he had twitching of the lips and he was again unconscious. There is no doubt that this man was suffering from epilepsy and the absence of major convulsive movements does not in any way influence this fact.

This recent paper is quoted to emphasize the fact that no relationship between syncope and epilepsy must be claimed upon the results of statistical or experimental work which does not keep a clear distinction between loss of consciousness resulting from a change in cerebral blood flow (syncope) and that resulting from a primary change in cerebral activity (epilepsy). The clinical basis for this distinction is real.

Keeping clear the fact that there are, on clinical grounds, as well as upon the mechanisms responsible, distinctive differences between fits and faints, I would like to consider the evidences for a common bond between them. There are forms of syncope in which the mechanism of the loss of consciousness is so evident that it is hardly profitable to wonder whether an obscure cerebral mechanism is at work. Such are, for instance, the attacks of orthostatic hypotension resulting in loss of consciousness. There are others, however, in which there is a much closer affinity to epilepsy. I refer to reflex syncope caused by highly specific stimuli, often emotional in their background. The pattern of this disturbance is similar to that of some instances of so-called reflex epilepsy. We have already heard that habitual syncope and epilepsy may co-exist in the same patient, but when reflex syncope is considered the relationship between faints and fits occurring in the same subject may be close. The following are two cases which clearly illustrate this relationship.

Case 1.—An army officer aged 42 was undergoing dental treatment. 1% novocain had been injected, there was no pain and no apprehension. The dental surgeon said the patient suddenly went rigid, his pupils dilated, his eyes were open and he went into a tonic-clonic convulsion, with deviation of the head and body to the left. The convulsions lasted for 5 seconds, then he seemed normal. The patient said he suddenly felt ill, giddy, sweated profusely and then remembered no more until he found himself with his head in a basin. He was confused at that time. Afterwards he was cold and clammy, and the surgeon said that his pulse was thready. Important points are that the subject experienced no apprehension, or pain, that he was half lying throughout, and that the experience of the symptoms of fainting was momentary.

His past history showed that he had had the following kinds of attacks:

(1) In bed just before sleeping, about once a week he had a feeling of apprehension, thumping in the head, pounding of the heart, a hot clammy sensation and a feeling of pins and needles throughout the body. This disturbance seemed to last five minutes. It was never associated with loss of consciousness, incontinence or movement.

(2) In the daytime on sudden bending and rising he had typical postural hypotension with loss of consciousness.

(3) He had had repeated attacks of loss of consciousness in response to the sight of blood, accidents, or bomb casualties.

(4) On hot days, once or twice each summer, he had had attacks of unconsciousness which from the description were syncopal. The symptoms were similar to those of his attacks of orthostatic syncope, but the course of events was more rapid.

His only brother, a leader of a heavy rescue squad in the Civil Defence Services in London during the war, had never been upset by all kinds of horrible sights. Traumatic amputation of a finger did not greatly disturb him. He had lupus of the face and had to make frequent visits to hospital. He always had to be accompanied for treatment, because when he smelt the "smell of a hospital" he would fall to the ground unconscious with only the briefest warning. He might have time to say "I feel funny" then would fall, often hurting himself. He would have a tonic-clonic fit and be confused for a few minutes afterwards. He had never lost consciousness or even felt faint in response to any other stimulus.

The patient was normal on full examination. The E.C.G. and E.E.G. were both normal. Mechanical stimulation of the carotid sinus failed to produce any symptoms.

Here then is an example of a man having orthostatic and reflex syncope, hypnagogic disturbances and epileptic attacks in response to special circumstances, with a family history of epilepsy in response to a highly specific situation.

Case 2.—A young man of 19 hurt his hand slightly in machinery. He was having it dressed by a nurse when he suddenly fell off the chair in a tonic-clonic fit, which the nurse witnessed in detail. He was confused afterwards. He was not pale before the attack and he said he felt no great pain or apprehension while the wound was being dressed.

His past history showed that he was a high-grade defective, three years behind his age, whose physical development was normal. He had had no convulsions in infancy. When 8 he had a tonic-clonic attack after he had cut his finger, and after that he had six attacks at irregular intervals, each precipitated by sight of blood or injury to himself. In each he went pale then blue, fell to the ground and had convulsive movements. In one he was incontinent. He had no attacks of any sort in any other circumstances.

His mother, who was unstable emotionally, had periods of lack of emotional control when frustrated. At the height of her histrionic anger she would suddenly fall down, become cyanosed and have a tonic-clonic fit of short duration. She was usually incontinent in these attacks. The father, a brother and a sister were normal.

On examination, both the patient and his mother were normal. Their E.E.G.s were normal. In this case there was a maternal history of epilepsy in response to emotion, in a boy who had syncopal and epileptic attacks after physical injury.

There are innumerable instances of the presence of syncope and epilepsy in the same person, so that the point need not be stressed. The difficulty is to explain *why* the conditions should exist, and there are very many reasons why it is an over-simplification, and indeed a mistake, to presume that syncope is a minor and special form of epilepsy, as some would wish. It must be recalled that there are many cardiological causes of loss of consciousness, in which the mechanism at fault leads to a fall in cerebral blood pressure. For these such a point of view is clearly untenable.

I would now like to examine a small piece of evidence upon the nature of the common bond between the tendency to faint and the tendency to have fits. The electroencephalograms of four groups of subjects were examined. These groups were made up of large numbers of comparable cases, as in Table I. They comprised: (1) Patients referred for opinion who had had more than one fainting attack. These attacks, from the clinical description given and from the circumstances in which they had occurred, were, in my opinion, faints. In all there had been a fairly long period in which the patient was able to say to himself "I feel faint". This sense of impending loss of consciousness seems to be common to all but the most rapid forms of fainting (such as the Stokes-Adams attack). During this time there was usually a story of pallor, often sweating, coldness, and sometimes nausea. The patient often sought support, and if he failed to find it or to sit down, lost consciousness. In all these cases consciousness was lost. Whether or not convulsive movements followed such a chain of events was unimportant, for an epileptic attack often follows cerebral ischæmia. In none of these patients had there been a story of fits, however brief, which were not heralded by the symptoms of syncope or the circumstances which give rise to syncope.

(2) Patients with *grand mal* epilepsy, for which no cause had been found, and in whom fainting has not taken place—idiopathic epilepsy.

(3) Patients with a similar story in whom a head injury was thought to have been responsible—traumatic epilepsy.

(4) Normal men and women who had never lost consciousness.

All the subjects were adults, all serving in the Forces, and all were otherwise normal.

The electroencephalograms were divided upon (1) the existence of generalized abnormality of any kind, which placed the records beyond the limits of normal, which excluded at least 12% of the normal population (the standards employed have been described before, Williams, 1941), and (2) upon the presence of runs of abnormal fast waves (Figs. 1 and 2) in both frontal lobes. These disturbances were specially picked out for study because they had so often been seen to occur in patients in whom I had no doubt that syncope and not epilepsy was the correct diagnosis, upon the criteria already outlined.

The results of this division of the cases is shown in Table I.

TABLE I

		Electroencephalogram				
			Non-specific abnormality		Episodes of fast activity	
Diagnosis	No.	No.	%	No.	%
Syncope	68	21	31	13	19
Idiopathic *grand mal* ..	94	54	57	21	22
Traumatic epilepsy ..	68	49	72	14	21
Normal	70	11	16	3	4

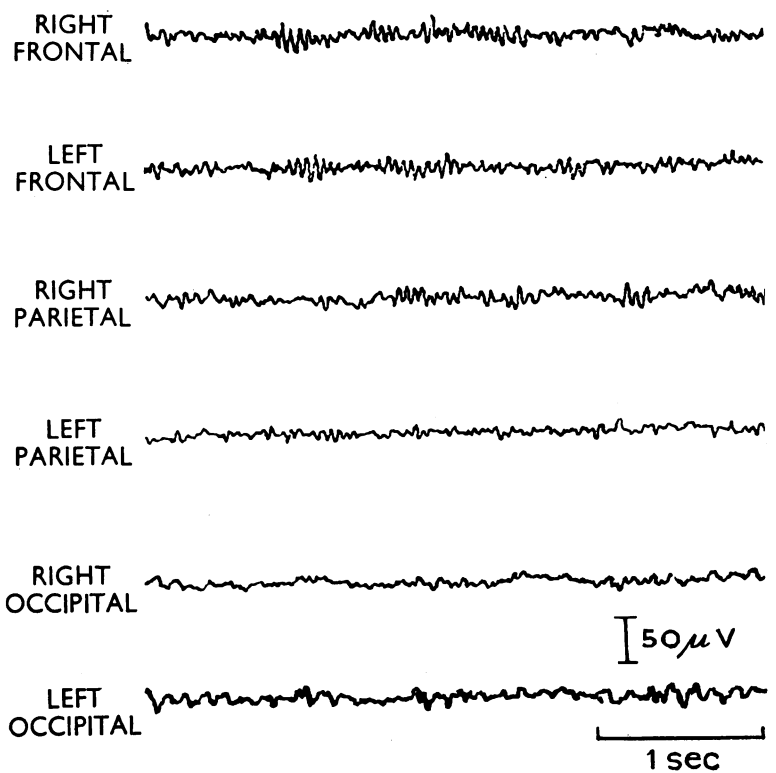

FIG. 1.—Episodes of fast rhythms arising in both frontal lobes in a patient with repeated faints. The electroencephalogram is otherwise normal.

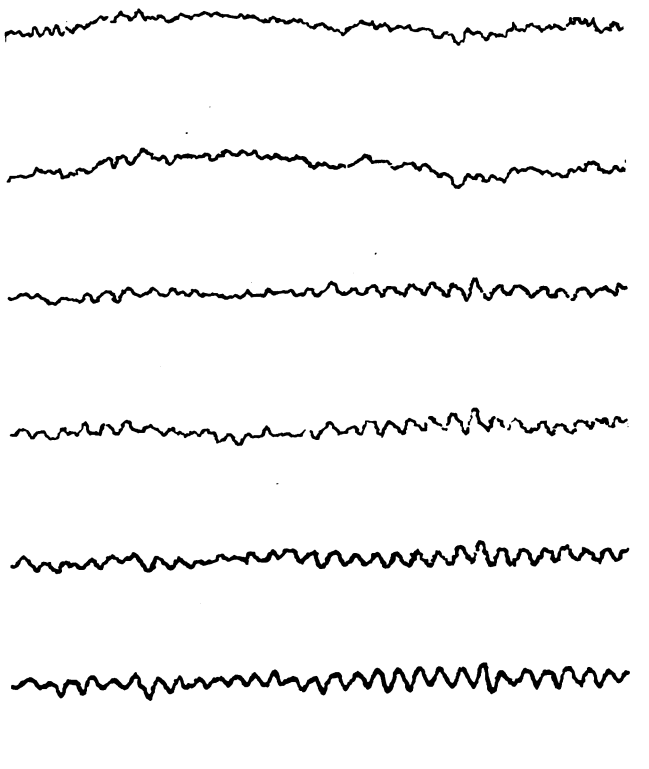

FIG. 2.—A normal electroencephalogram, similar to that in Fig. 1 except that there are no abnormal fast rhythms in the frontal lobes.

This shows that there is a significant increase in abnormality from normals to syncope and the epileptics, but it also shows that the proportion of those showing abnormal fast activity in the frontal lobes is the same in all the abnormal groups (about 20%), while it is only 4% in the normals.

Because of these results, members of the hospital staff were asked confidentially if they had ever had any attacks of any sort, and in 16 instances the subject had fainted more than once in his life, but had always been considered to be normal. None had had epileptic attacks. This small group of 16 otherwise normal fainters were in all other respects exactly comparable to the normal control group, none of whom had fainted more than once. In Table II it will be seen that the proportion with generalized dysrhythmia was the same in the two groups, whereas the proportion showing abnormal frontal fast activity was significantly higher in the small group of "fainters".

TABLE II

		Electroencephalogram				
			Non-specific abnormality		Episodes of fast activity	
Normal subjects	No.	No.	%	No.	%
Fainters	16	2	12·5	7	43·7
Others	70	11	15·7	3	4·3

Disturbances of an "epileptic" kind are probably absent from the subjects who faint in this series because of the care which had been taken to divide epilepsy from syncope, but even so there is an episodic disturbance of the kind so often seen in epileptics—particularly those suffering from *grand mal*—occurring in a proportion of people who faint, even though they felt normal at the time of the recording. It would be wrong to assume that this finding indicates that syncope and epilepsy are the same condition but it must indicate that a common factor exists in the mechanism of the two conditions.

The clinical experiences which have been recounted both by Sir Charles Symonds and myself would support the hypothesis that there is such a common factor, and this factor must surely reside in the cerebral hemispheres. It may well be that it consists of the kind of reaction of the hemispheres—the failure to maintain normal activity—in response to deleterious circumstances, whether these consist in changes arising in the hemispheres themselves or elsewhere in the body. That is to say that consciousness is more readily lost in these than in other subjects whether the cause is a fall in blood pressure or an abnormal discharge of neuronal activity in the brain. Certainly in epilepsy some people lose consciousness whenever an epileptic discharge occurs in the E.E.G., while others have numerous "larval attacks" in the E.E.G. without loss of consciousness (Williams, 1950). Certainly also in syncope some people lose consciousness more readily than others with a similar rate and extent of fall in blood pressure. There are at least three mechanisms responsible for the faint:

(1) The special response of the individual to specific circumstance, which may sometimes be a conditioned response to a previously emotional situation.

(2) The resulting disturbance in the cardiovascular regulating mechanisms.

(3) The consequent disturbance in cerebral activity, with loss of consciousness and other changes.

It is in the hemispheres that syncope and epilepsy have common ground, and the similar response of the hemispheres to different abnormal states may well be reflected in the common features seen in the E.E.G.

SUMMARY

Evidence is produced to support the view that one of the factors common to faints and fits is the failure o the individual to maintain consciousness in special circumstances. The view is expressed that although syncope and epilepsy may co-exist in the same family or the same individual, syncope is not a special form of epilepsy. The clinical and experimental knowledge that we have lends support to the view that, although the mechanisms responsible for faints and fits are different, they may occur in the same person because of a common factor in the hemispheres, reflected in the E.E.G. This factor may simply relate to a failure to maintain consciousness under the conditions which exist in the two disorders.

REFERENCES

GOWERS, W. R. (1907) The Borderland of Epilepsy. London.

KERSHMAN, J. (1949) *J. Neurol. Neurosurg. Psychiat.*, **12**, 25.

WILLIAMS, D. (1941) *J. Neurol. Psychiat.* **4**, 257.

—— (1950) *Brit. med. J.* (i), 685.

Dr. Maurice Campbell: The identical word, syncope, is used for trivial fainting attacks and for a manner of death, and should, therefore, be banished from scientific discussion. It might be useful if it was recognized that syncope meant a sudden loss, and fainting a gradual loss of consciousness, but there is no such agreement and it would at present be wrong because syncope is by definition (O.E.D.) "a failure of the heart's action, resulting in loss of consciousness or sometimes in death". Fits are, of course, more often of cerebral origin, but not always: if loss of consciousness from anoxæmia lasts more than a certain time, the familiar sequence of events seen in any epileptiform attack will follow.

More accurate knowledge of attacks of unconsciousness will not be gained without more accurate descriptions and more precise terminology. The speed with which consciousness is lost is a fundamental distinction; and I suggest that the word tachy-apsychia should be used for sudden loss of consciousness, seen characteristically in most Stokes-Adams attacks or in epilepsy, and brady-apsychia for gradual loss of consciousness seen characteristically in most fainting attacks in young people. I am indebted to Mr. E. L. York of New College, Oxford, for the use of these words.

Loss of consciousness concerns the physiologist and neurologist even more than the cardiologist; but there is, perhaps, more knowledge of the provoking mechanism of unconsciousness in disorders of the heart than of the nervous system. The common factor in the former group is the production of cerebral anoxæmia—whether by change of rhythm, including ventricular standstill as in many Stokes-Adams attacks; by standstill of the heart as in a few fainting attacks, or during anæsthetics; by the combination of a slow heart and a fall of blood pressure or by one of these alone, as in most cases of fainting and of the carotid sinus syndrome; or by temporary arrest of the cerebral circulation by venous stasis or difficulty in auricular filling during violent spasms of coughing or after pulmonary embolism.

Broadly speaking, these various cardiovascular causes—and there are many more—can be divided into those where the cerebral circulation is arrested, often but not necessarily, by cardiac or ventricular standstill, and those where it is diminished (as in the severe fall of blood pressure after a large cardiac infarct or with a very rapid paroxysmal tachycardia) when the unconsciousness will generally be gradual.

As a rule, therefore, tachy-apsychia will mean sudden arrest, and brady-apsychia a gradual diminution of the cerebral circulation: there are, however, exceptions, and of course, tachy-apsychia is often cerebral in origin. With arrest of the circulation there is greater danger of the heart failing to start again and so tachy-apsychia has, in general, a much graver prognosis than brady-apsychia.

A patient who has been unconscious can, as a rule, give the physician no direct indication as to whether the cause was cardiac or cerebral, and the decision may be difficult and may call for much knowledge and experience: the symptoms produced in either may be almost identical. It is, in particular, essential to be on familiar terms with epilepsy, in both its major and minor forms.

The duration of unconsciousness is of great importance in this connexion. A short attack, and this means one lasting less than two or perhaps five minutes, may be either cardiac or cerebral in origin. A long attack, lasting more than five or ten minutes, must be wholly or partly cerebral—partly when a short cardiac arrest starts the loss and produces anoxæmic changes in the cerebral cortex that prolong the unconsciousness.

Standstill of the heart or of the ventricle for from 5–10 seconds causes sudden loss of consciousness. Arrest of the cerebral circulation for from 5–10 seconds by a pressure cuff round the neck (Rossen *et al.*, 1943) causes sudden loss of consciousness, so this is further evidence that both act in the same way. Rossen *et al.* fixed a cuff round the neck that could be inflated to a pressure of 600 mm. Hg. In normal young men fixation of the eyeballs and blurring of vision were the first signs, nearly always within 5–7 sec. Loss of consciousness followed 1 sec. later, and the cuff was then released. Mild generalized convulsions followed but only lasted 6–8 sec. Consciousness was regained 4–10 sec. after the cuff was released. Longer periods of arrest of the cerebral circulation up to 100 sec. were produced in 11 patients with schizophrenia; all regained consciousness in 30–40 sec. and there were no after-effects.

In unconsciousness due to cerebral anoxæmia spasmodic movements or fits are dependent on the depth and duration of unconsciousness and not on its cause: they may, therefore, occur in attacks of all sorts. They follow 15–20 sec. after the arrest of circulation.

Arrest of the cerebral circulation for two minutes by standstill of the heart in disease or during anæsthesia or for 100 sec. in experiments on man is compatible with perfect recovery.

Standstill of the heart for 5 minutes is generally permanent, i.e. the patient is dead. In the rare cases where the heart starts again after longer than this, anoxæmia will have caused irreversible damage to the brain, especially to the cortex; and the first sign of this will often

be a longer period of unconsciousness. Where the cerebral arteries are diseased as in many cardiac cases and most Stokes-Adams attacks, a shorter period than five minutes may produce cerebral damage from which it is not possible to recover.

Standstill of the heart for more than two but less than five minutes will produce cerebral damage from which it may be possible to make a perfect recovery. The first sign of this cerebral damage may be a more prolonged period of unconsciousness. This means that even in a cardiac case that has been unconscious for many minutes, it is of great prognostic value to know when the circulation returns, i.e. when the heart starts beating. Ultimately this period of between two to five minutes may be defined more precisely though there will always be great danger about recovery in such cases. In clinical records of heart standstill periods of from 20 to 80 sec. are not often exceeded and these are well within the limits of complete recovery.

Table I shows the variety of conditions to be remembered and indicates whether tachy-apsychia or brady-apsychia is likely to be found.

TABLE I.—CARDIAC CAUSES OF LOSS OF CONSCIOUSNESS

I.—Standstill of heart, including long sino-auricular block. *Tachy-apsychia* if not preceded by IIA.
- A. Neurogenic (vagal)
  - (1) with normal heart.
  - (2) with heart disease.
- B. Myogenic, with heart disease.
- C. Anæsthesia.

II.—Slowing of heart with fall of B.P. *Brady-apsychia.*
- A. *Fainting attacks.*
- B. Carotid sinus syndrome.

A and B always neurogenic but may occur with heart disease.
- C. Postural hypotension } Fall of blood pressure only.
- D. Severe hæmorrhage }

III.—Ventricular standstill. *Tachy-apsychia.*
- A. *Stokes-Adams attacks* (one form) (difficult if block paroxysmal). Always with heart disease.
- B. Neurogenic cases without previous heart block.

IV.—Tachycardia with changes of rhythm.
- A. Arrest of circulation. *Tachy-apsychia.*
  - (1) Paroxysmal ventricular fibrillation.
  - (2) Paroxysmal ventricular tachycardia.

  If with heart block 1 and 2 are *Stokes-Adams* attacks. And rarely from 3, 4 or 5 below.
- B. With slowing of circulation. *Brady-apsychia.*

  Some cases of 2 above.
  - (3) 1 : 1 auricular flutter.
  - (4) Onset of auricular fibrillation.*
  - (5) Paroxysmal auricular fibrillation or flutter.*
  - (6) Paroxysmal supraventricular tachycardia.*
  - (7) Repeated extrasystoles.*

* Only in those specially prone to fainting.

V.—Failure of the left ventricle.
- A. With cardiac infarction (which also causes other varieties).
- B. With angina pectoris.
- C. With aortic stenosis. Often with exertion and of serious import.
- D. With aortic regurgitation. Not serious unless syphilitic.
- E. With acute pulmonary œdema.

VI.—Failure to fill the left auricle.
- A. With pulmonary embolism.
- B. Tachy-apsychia from coughing (whooping cough, chronic bronchitis and alcoholics).

VII.—Failure to fill the right auricle.
- A. Fainting after unaccustomed exertion.
- B. A factor in ordinary faints, especially standing.
- C. ? postural hypotension.

VIII.—With cyanosis (asphyxia).
- A. With congenital heart disease.
- B. With Cheyne-Stokes respiration.

TABLE I (*continued*)

IX.—Unclassified.
- A. Hypertensive crises.
- B. With labyrinthine vertigo (falling common, unconsciousness rare).
- C. Gowers' syndrome (vaso-vagal).
- D. Cerebral effect of carotid sinus pressure without change of rate or fall of blood pressure.
- E. Cerebral embolism.
- F. Hypoglycæmic attacks.

[The speaker quoted examples of some of these varieties, especially with Stokes-Adams attacks, with aortic stenosis, with sudden attacks of coughing, and the many varieties with cardiac infarction.]

REFERENCE

ROSSEN, R., KABAT, H., and ANDERSON, J. P. (1943) *Arch. Neurol. Psychiat.*, **50**, 510.

Dr. M. N. Pai: Nearly every case of faints, fits and "blackouts" which does not respond to immediate treatment is likely to be referred to a psychiatrist on the assumption that treatment along psychiatric lines is indicated. On account of the socio-economic and medico-legal implications the psychiatrist who handles such a case is assuming a much greater responsibility than the neurologist unless he first satisfies himself as to the accuracy of the diagnosis. A subsequent diagnosis of epilepsy (made by the psychiatrist) may shatter the patient's faith in doctors, make him more hypochondriacal and induce in him a state of secondary anxiety which may again obscure the initial symptoms.

In the case of a member of the Forces a mistaken diagnosis of epilepsy may bring him some immediate advantages in the form of a discharge from the Forces and perhaps a high-percentage pension for life. The disadvantages are that he may be unnecessarily condemned to an indefinite period of anti-convulsant drug therapy with its occasional risk of intellectual deterioration and may also be precluded from obtaining employment commensurate with his intelligence and experience. There is also the stigma attached to epilepsy and considerable distress may be caused to his relatives.

On the other hand a hasty diagnosis of hysteria merely because the features of a fit do not conform to those of typical epilepsy may be tantamount to accusing the patient of evading his military obligations and civilian responsibilities. Sensible persons and their intelligent relatives resent such a diagnosis. If any Service person is invalided out on account of hysteria he may not only be denied a pension but may also have considerable difficulty in finding suitable employment. The rehabilitation of such persons is taxing the resources of psychiatrists, social workers and officers of the Ministry of Labour.

It is undesirable to diagnose epilepsy on the E.E.G. alone. For instance, in 1940–42 it was thought that an abnormal hyperpnœa response was evidence of epilepsy, quite a large number of patients suffering from hysterical fits and fainting attacks were diagnosed as epileptic and many were discharged from the Services on the basis of erroneous views about the E.E.G. The E.E.G. technique has improved considerably during the war and since about 1944 it has been recognized that an abnormal hyperpnœa response unrelated to the level of blood sugar has no significance.

Knowledge of this subject is now crystallizing and a positive E.E.G. may certainly help in clinching the diagnosis in any doubtful case. When a series of Service and civilian cases complaining of faints or fits were investigated by the E.E.G., it was found that a family history of fits, faints, psychoses and mental defect was commoner in those suffering from epilepsy than in those suffering from hysteria. The E.E.G. was also found useful in spotting epileptic equivalents. In one epileptic patient when the fits ceased there were for four years recurrent attacks of headaches and when these ceased there was periodic epistaxis for which no obvious cause could be found. The E.E.G. showed spikes, and administration of luminal controlled the epistaxis. In another patient with fainting attacks there was paroxysmal tachycardia and the E.E.G. showed paroxysmal outbursts diagnostic of epilepsy. In yet another patient when the fits were controlled there was sudden onset of compulsive-obsessional symptoms, e.g. washing hands; the E.E.G. showed the spike and wave pattern.

A diagnosis of epilepsy should be made with great caution and only after taking into consideration a reliable family history, details concerning previous health obtained from independent sources, careful observation of the fits and the E.E.G. During an attack the one positive physical sign which if present is pathognomonic of loss of consciousness and therefore in favour of a diagnosis of epilepsy is an extensor plantar reflex. Too much dependence on the E.E.G. alone may result in neglect of clinical observation and investiga-

tion with sometimes disastrous consequences. One of the patients sent to me as a case of hysterical faints had well-marked aortic regurgitation, another had advanced lymphosarcoma of the mediastinum and yet another patient was suffering from acute post-vaccinal encephalomyelitis.

Dr. W. Ritchie Russell emphasized that all brains can be made to discharge an epileptic fit, but in the so-called epileptic the threshold is low. In diagnosis there is no substitute for a careful record of what happened during a fit. Fit forms which can easily be posted to someone who saw the attack are of great value and should be widely used in out-patient practice. Elderly arteriosclerotic subjects sometimes have a fit while sleeping in a chair after a meal; this is preceded by pallor of the face and is probably due to cerebral anæmia. Those who suffer from postural syncope should be made to realize that they can prevent fainting if they at once stoop say to adjust their shoe-lace, of if they voluntarily contract strongly the abdominal and thigh muscles.

Dr. Sheila Sherlock: During fainting there is not only release of posterior pituitary extract but other evidence suggests that adrenaline is also liberated. Moreover, there is a re-distribution of blood in the body; the flow through the limb muscles increasing and that through the splanchnic area diminishing. All these events are apparently the effects of fainting. What is uncertain is the exact stimulus that sets this train of events in motion. For instance, is the abnormal rhythm which Dr. Denis Williams observed in the electroencephalogram a result of the faint rather than related to its causation?

Dr. Denis Williams, in reply to Dr. Sherlock: as the abnormal rhythms are present when the patient is in a normal state of consciousness they are presumably related to the basic cause of the instability, and are not an effect of the fainting.

Dr. Charles Baker: A not uncommon, but often unrecognized, cause of loss of consciousness is coughing. This was first described by Charcot in 1876 as laryngeal vertigo, with analogy to labyrinthine vertigo; and these cases have also been classified as laryngeal epilepsy. When my interest was aroused in this syndrome I had no difficulty in finding 9 cases; all were males, all were overweight, and chronic bronchitis and emphysema were common features. These features in the clinical picture were confirmed by a review of a fairly extensive literature, predominantly French (Baker, 1949). The mechanism both of faintness and loss of consciousness from coughing would appear to be a diminished flow of blood to the heart due to increased intrathoracic pressure when the glottis is closed; recent reports of cardiac catheterization during straining and coughing and positive pressure breathing tend to confirm this explanation. This is different from the carotid sinus syndrome. Emphysema would tend to augment the raised intrathoracic pressure owing to the absence of the elastic recoil of the lung; obesity would contribute to the diminished return of blood to the heart from the inferior vena cava by the loss of abdominal musculature; the occurrence in males is explained by the simple fact that men cough harder than women. The prognosis is good and the attacks are not dangerous though one man was reported as killing a pedestrian when he lost consciousness from coughing while driving his car; he was saved from a charge of manslaughter on the medical evidence.

REFERENCES

BAKER, C. G. (1949) *Guy's Hosp. Rep.*, **98**, 132.

CHARCOT, J. M. (1876) *Gaz. Med. Paris*, **5**, 588.