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LECTURE ON FAINTS AND FITS*

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That a large and variegated assortment of clinical conditions may first become manifest by a passing loss of consciousness with or without convulsions is a well-known fact. Diagnosis, particularly some time after a single attack, often presents considerable difficulty. It is therefore useful to collect and correlate the results of recent investigations and advances in this particular field, particularly in the hope that a simple classification may lead to a clearer understanding of the mechanisms involved, and thus facilitate accurate diagnosis and appropriate treatment. In such a wide subject it is only possible to discuss certain aspects which illustrate the problem as a whole and provide a basis for the investigation of the individual clinical case.

Just as there is no entirely satisfactory definition of a faint, though it is rightly attributable to peculiar circulatory maladjustments, so also even greater difficulties beset the exact definition of a fit. Convulsive movements in the form of facial twitchings or muscular jerkings of the limbs may be observed in the severer fainting attacks, and in fits generalized or local clonic movements confined to a limb or muscle group may dominate the clinical picture. Convulsive movements are common to both, but it is equally justifiable to employ the term "fit" to designate those attacks in which muscular movements are absent and an unnatural motionlessness is the prominent feature (Wilson, 1928). Similarly certain fits may occur without loss of consciousness and the convulsive movements be local or widespread. For these reasons I do not attempt to define either the faint or the fit, recognizing that the one merges at times almost insensibly into the other. I propose to limit this discussion to attacks characterized by sudden and transient weakness, loss of muscular control, a greater or less degree of amnesia if not actual unconsciousness with or without convulsive movements.

Clinical Groups

The recognition of cerebral ischaemia as a cause of the convulsive state led gradually to the differentiation of attacks of unconsciousness into two broad groups—the syncopal, due to transient circulatory defects, and the epileptiform, primarily of cerebral origin (Russell, 1909; Sutherland, 1927). Actually between these two somewhat artificial groups there exists a wide gulf—a gulf bridged by the autonomic nervous system. Recent investigations indicate the importance of this link between the central nervous system and the viscera, autonomic reflexes exercising directly and indirectly a dominant role in the control of the circulation and in the maintenance of consciousness. It has long been known that pressure exerted on the carotid arteries in the neck is capable of

slowing the heart, and by the work of Hering (1927), Heymans (1929), and their associates it has been conclusively demonstrated that, concentrated at the dilated bifurcation of the common carotid artery—the carotid sinus—a rich nervous anastomosis exists in which lie various sensory receptors reflexly regulating heart rate and blood pressure. This outpost of the autonomic system transmits afferent reflexes to the medulla via the glossopharyngeal nerve, the main efferent channel being over the vagus nerve to the periphery. Recent studies indicate that a hypersensitive state of the carotid sinus reflexes is responsible for certain forms of syncope (Weiss and Baker, 1933), and though the mechanism varies in different instances yet it is sufficient to indicate the importance of the autonomic system in the regulation of the conscious state. Recognizing that cerebral and general circulatory mechanisms are closely interwoven, it seems justifiable and, even if not very exact, at least helpful from the point of view of practical medicine to submit a classification consisting of three broad groups according to the dominant mechanism:

1. Seizures resulting from a disturbance of the autonomic nervous system.
2. Syncope the result of an intrinsic disturbance of the heart beat.
3. Seizures more directly of cerebral origin, due to local structural or general metabolic defects.

Autonomic Nervous System

A study of the common syncopal attack is sufficient to indicate a disturbance of the usual balance between heart rate and blood pressure. Beginning with a sense of uneasiness, muscular weakness, and restlessness, the symptoms gradually advance until the victim becomes aware of a vague abdominal discomfort associated with nausea, which increases the feeling of insecurity. Lightheadedness, blurring of vision, and loss of power in the limbs, accompanied by a feeling of coldness and sweating, are usually recognized before the final collapse. Lewis (1932, 1918-20) has shown that these symptoms are associated with a progressive fall in blood pressure, often a decrease in the heart rate, and has suggested the term vaso-vagal attack to describe the underlying mechanism, cardiac inhibition, sudden hypotension, and diminished cerebral blood flow being the predominant features (Starr and Collins, 1931). Unconsciousness may last a few minutes, during which time examination reveals a striking pallor, particularly of the face, low blood pressure, a weak or almost imperceptible pulse, usually regular but slow in rate—50, 40, or 30 beats a minute or less being commonly recorded. Recovery is gradual, slower than the onset, and in the succeeding hours leaves the victim listless and unsettled.

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Simple syncope occurs under a variety of circumstances. The erect posture, long hours without food, convalescence after debilitating illnesses, hot stuffy rooms, or exposure to cold and damp all play a part, but there is evidence to suggest that the emotional tone of the victim at the moment plays an important part in the precipitation of the attack. A hundred onlookers may see a child run down in the street, but perhaps only one will faint. Received by one or other of the special senses the stimulus itself does not necessarily cause fainting, but the emotional idea—often disgust or loathing—dominant in the mind of the subject at the moment may be the main determining factor in the initiation of these circulatory reflexes, which produces a sudden change in the mechanics of cerebral blood flow. A slow heart beat in itself is not harmful, as witness the rate of 28 or 34 maintained for years in cases of heart-block. Similarly a low blood pressure of perhaps 90 or 100 mm. Hg may be entirely symptomless, but an abrupt alteration from a high to a low level in pulse rate or blood pressure seldom passes without the production of symptoms.

The slow pulse and particularly the abrupt loss of vasomotor tone which form the essential features of simple syncope are in effect a transient disturbance of the reflex control of the circulation exerted by the autonomic nervous system. This suggests that just as receptor organs situated at the bifurcation of the carotids are capable of receiving and transmitting afferent stimuli to the medulla with resulting cardiac depression and lowered vasomotor tone, so also through similar but unrecognized channels the medulla may be influenced from higher cerebral and other centres with a resulting disorganization of these usual circulatory reflexes constantly passing through this busy regulatory area.

Carotid Sinus Syncope

More conclusive evidence of the influence of the autonomic nervous system is afforded by the recent studies of Weiss (1933) and Ferris *et al.* (1935), who have revealed in a group of people liable to fainting attacks a peculiar sensitivity of the carotid sinus to mechanical pressure in the neck. That it was possible, by pressing with the fingers on one or other sinus, to reproduce constantly in a matter of seconds an exactly similar train of symptoms as those for which the patient sought advice yields very good evidence of a hypersensitive state of the sinus mechanism.

Fainting, it was discovered, resulted from one or more of three distinct mechanisms: (a) a marked cardiac asystole or sudden slowing of the pulse, with or without a marked fall in the arterial blood pressure; (b) a striking fall in the blood pressure without essential cardiac slowing; (c) a cerebral type associated with a striking degree of facial pallor, but without alteration in the heart rate or blood pressure level. The origin of the fainting and convulsions in the first two groups associated with bradycardia and hypotension is clearly attributable to a sudden cerebral ischaemia, and is directly comparable to the observed facts recorded in simple syncope. The experimental data indicate such a close parallelism between these two forms of sinus syncope and the simple vasovagal attack that they are almost certainly of a similar nature. Paralysis of the parasympathetic nerve endings with atropine abolished the cardiac, but not the peripheral, vascular changes. Both forms were prevented by the previous administration of adrenaline.

Arteriosclerosis or hypertension was a common clinical finding, but a local mechanical cause for the increased sensitivity was demonstrable in nine of the fifteen cases

studied, a small tumour pressing on the sinus in three instances. Furthermore, operative removal of the tumour and section of the sinus nerve supply abolished both the spontaneous and the induced attacks. It is well known that tumours in this situation are often associated with syncope (Cassidy and Page, 1928), and from the studies of the Boston workers the mechanism seems clear.

Cerebral Syncope of Sinus Origin

The cerebral type of carotid sinus syncope, in which fainting occurs without alteration in heart rate or blood pressure, has also been carefully investigated (Ferris *et al.*, 1935). Earlier observations suggested that these seizures resulted from reflex vasoconstriction of the cerebral vessels, but further experiments designed to influence cerebral blood flow cast a doubt on the earlier explanation. In spite of the previous administration of adrenaline or histamine, pressing on the sinus as readily induced attacks, and measurements of jugular blood flow failed to yield evidence of a generalized cerebral anaemia during the seizure. Weiss and his co-workers tentatively suggest for the regulation of the conscious state the presence of a special area or centres in the brain closely related to the autonomic system. Whether in the cerebral form of syncope the afferent reflex from the sinus travels over the same pathways as those responsible for the cardiac depression and hypotensive forms is difficult to decide—probably not. At all events, cerebral syncope was not prevented by atropine, and this suggests more purely sympathetic channels.

In a proportion of cases considerable benefit was obtained by stripping the coats of the carotid artery on the more sensitive side. It is yet too early to judge of the permanence of the results, but a recurrence of spontaneous attacks was observed in three of ten cases operated on. Fortunately hypersensitivity of the sinus mechanism is not necessarily permanent, and much good was done several patients by measures directed to the improvement of their general health. Patients presenting this form of syncope apparently fall into a distinct clinical group. Weiss points out that in virtue of their unstable heart rate, easy fluctuations in blood pressure, and low metabolic rate they might be vaguely classified as examples of vasomotor neurosis or instability. Carotid sinus syncope is a definite entity, and when the problem of repeated syncopal attacks is encountered in practice firm pressure applied first on one side and then on the other for ten or twenty seconds will form a useful clinical test for sinus hypersensitivity. Further, careful palpation of the neck may reveal a local mechanical cause for the recurring seizures.

Postural Hypotension

This peculiar variety of fainting occurs but rarely and has certain characteristic features which clearly indicate a disorder, more or less permanent, of the autonomic system. It is, however, uninfluenced by carotid sinus stimulation. Originally described and carefully investigated by Bradbury and Eggleston (1925) little has been added to our knowledge by subsequent observers. Its leading symptom is readily induced fainting on assuming the upright position. There is a marked and abrupt drop in the systolic and diastolic pressures when the patient stands erect, and this is associated with actual syncope or marked weakness and giddiness. The symptoms are accentuated in hot weather. Two other observations serve to give a clue as to the nature of the disorder. These are a local or generalized deficiency of sweating and the failure of the heart to increase markedly in rate when the patient stands erect. Adrenaline, while it augments

the heart rate, does not restore peripheral vasomotor control, and fainting is still apt to appear after its administration. The heart rate is uninfluenced by atropine. As an explanation for these findings it is reasonable to postulate a decreased activity of the autonomic nervous system amounting to a virtual paralysis of the peripheral vasomotor constrictor fibres. It was also observed that the amount of urine excreted varied with posture, being considerably less in the erect posture, a finding attributable to the decreased renal blood flow when the patient was erect. The extraordinary dependence of the blood pressure upon the influence of gravity is illustrated by the findings of Chew, Allen, and Barker (1936) in one of their patients.

	Recumbent	Sitting	Standing
Blood pressure ...	Systolic, 140 Diastolic, 80	Systolic, 80 Diastolic, 50	Systolic, 40 Diastolic, 35
Pulse rate	64	64	68

Drugs are of little value in influencing the course of the disease. Ephedrine in 50-milligramme doses even as often as every two or three hours does not always prevent attacks (Chew *et al.*, 1936). It is difficult to determine the site of the lesion, but realizing the capacity of the splanchnic area it is conceivable that destruction of the coeliac ganglion or involvement of the lumbar sympathetic roots might account in some measure for the symptoms.

This brief review of recent work serves to illustrate the fact that identical symptoms can be produced in a variety of ways. In each and all the activity of the autonomic nervous system is disturbed in varying degree. By way of contrast it is perhaps a little surprising that syncope is so uncommon after the administration of amyl nitrite. The explanation probably lies in the fact that in this instance hypotension is to some extent compensated by tachycardia. The fact that slow heart action and fall in blood pressure coexist in simple syncope indicates a disturbance in the reflex control of the circulation, a transient failure to stabilize pressure and heart rate. A penalty of the erect posture, these forms of syncope, indicating a maladjustment of the autonomic system, are seldom observed other than in the upright position. The force of gravity constantly puts a burden upon the fine adjustment of the circulatory mechanism. It is also worth noting, from a diagnostic viewpoint, that the syncopal attacks just described are unassociated with a sense of rotation. Giddiness is common enough as a premonitory symptom, but true vertigo is not a feature of the autonomic forms of syncope.

Syncope of Cardiac Origin

Under this heading may be grouped those cases in which the major defect lies in a sudden change in the dynamics of cerebral blood flow consequent upon an intrinsic disorder of the heart beat. The outstanding example is the Stokes-Adams seizure associated with certain forms of heart-block. Attacks may vary in severity, from the mildest associated with only a transient giddiness or unsteadiness to the most critical, in which during a long period of ventricular asystole lasting three or four minutes muscular twitchings with cessation of respiration and urinary incontinence may occur. Under these circumstances consciousness may not be regained for several hours or days after the return of the heart beat. To witness the Stokes-Adams attack in all its dramatic sequence is an unforgettable event in clinical experience.

The literature contains no entirely adequate definition of the term "Stokes-Adams syndrome." Neither Adams (1827) nor Stokes (1846) defined a syndrome, nor did they offer an explanation for their clinical observations. It was Huchard (1893), unaware of the earlier observations of Morgagni (1761) or Spens (1793), who made the original attempt to marshal the relevant facts and defined the triad of slow pulse with syncope and epileptiform attacks as the "maladie d'Adams ou de Stokes-Adams." It is best to confine the term Stokes-Adams seizure to those forms of syncope associated with a defect, transient or permanent, in the conducting mechanism of the heart beat.

The recognition of this type of syncope is not difficult when attacks are witnessed or when, in the intervals between them, a persisting bradycardia suggests the presence of complete heart-block. As a rough-and-ready clinical test the inhalation of amyl nitrite is of help in elucidating the nature of a bradycardia when graphic methods of recording the heart beat are unavailable (Gilchrist, 1934b). As a general rule amyl nitrite does not appreciably alter the ventricular rate when the block is complete, though there are exceptions. Atropine does not yield a reliable test for the presence of complete block (Gilchrist, 1933).

The great majority of sufferers from complete heart-block are seldom disturbed by seizures, though at the time of the total severance of the bundle of His, when the ventricles are for the first time starved of the auricular leadership and momentarily hesitate in the development of their independent rhythm, one or more seizures may be seen. When seizures do occasionally recur they are as a rule readily controlled by ephedrine (Gilchrist, 1934a).

Ventricular Arrest with Intermittent Complete Block

On the other hand there is a group of cases in which fainting attacks due to a transient paralysis of ventricular activity are common. These people do not suffer from a persisting chronic block, but only from time to time does the bundle of His cease to function. As a general rule, perhaps for weeks or months, the heart beats in normal rhythm with no impairment of auriculo-ventricular conduction; suddenly without warning a varying period of ventricular asystole occurs, accompanied by a faint of corresponding severity. This event is followed by independent ventricular beating—complete heart-block—for a brief space of time, minutes or hours, and then almost as suddenly normal rhythm is resumed and the pulse rate rises from 25 or 30 to 70 or 80 or more a minute. It is important to note that in this variety complete block is only evident for a short time after the Stokes-Adams attack, and that even immediately before the seizure there is no evidence of partial block. I believe that this form of heart-block, which affects middle-aged or elderly subjects, is often mistaken for senile or arteriosclerotic epilepsy. It is certainly more common than the number of recorded cases would indicate. Carter and Dieuaide (1923) collected eight cases, and since then only ten others have been reported. During the last few years I have had under my personal observation eight cases of intermittent block with Stokes-Adams seizures.

The diagnosis is not difficult if the absent pulse is noted during the attack or the steady slow rate for a short time thereafter, but the presence of normal rhythm in the intervals between seizures and the frequent absence of all other cardiac symptoms may lead to some confusion in making the diagnosis, as the following case indicates.

Case I

A lady, aged 57, seen in June, 1934, had been having "giddy turns" once a week, once a month, or at less frequent intervals for four years. They occurred when out walking, when sitting quietly in a chair, and rarely when lying in bed. She described the attack as a sudden feeling of "something coming over her," then fell to the ground and remained unconscious for ten minutes or more. Slight twitching movements of the limbs had occasionally been observed by onlookers. Her son, a doctor, noticed on one occasion that her pulse rate was 30 before she had fully regained consciousness. When on holiday in July, 1933, she had an attack, felt faint, fell, and lay in the street unconscious for nearly half an hour. A consultant who saw her some days afterwards found her pulse rate 70, regular, and electrocardiograms showed normal rhythm. Her blood pressure was then 150 systolic and 70 diastolic, the radial pulses were equal, and the vessel walls healthy. Owing to her obesity it was not possible to determine accurately the size of the heart, but the electrocardiogram demonstrated a marked left-sided preponderance with normal conduction time. A soft systolic murmur was heard in all areas. As no evidence of heart-block was found at the time of examination a diagnosis of simple syncope was made.

On June 4, 1934, I saw her for the first time, and at her home shortly after an attack recorded an electrocardiogram which showed conclusively the presence of complete heart-block. On June 8, suspecting from the frequency of the seizures that the block would be transient, a further electrocardiogram was recorded. This revealed a normal rhythm, the picture being exactly similar to that recorded the previous year (July, 1933) some days after an attack. These facts were sufficient to indicate the mechanism of the production of the syncopal attacks, and justified a diagnosis of intermittent complete block with Stokes-Adams seizures. Adrenaline and ephedrine reduced the frequency of the attacks for a time, but she died a year or so later during an attack.

A further case indicates the benefit which may result from appropriate treatment.

Case II

A schoolmaster led a very active life until at the age of 61, in 1925, he had a sudden attack of unconsciousness while in church. Immediately after the seizure the pulse rate was recorded at 35 a minute. Attacks recurred at intervals of a few weeks, but with the passage of time they increased in frequency until by 1934 he was experiencing as many as six Stokes-Adams attacks a week. Life was made a misery for him. He had no other cardiac symptoms. Seizures occurred at any time, in bed at night, moving about in the house, or when working in the garden. In some he fell unconscious; in others he was momentarily giddy for a few seconds. He knew of no provoking cause. Attacks were sometimes absent for a few days at a time. On other occasions he had as many as four or five in twenty-four hours. Seen in May, 1934, the pulse was regular at 80 a minute, the heart of normal size with no evidence of valvular disease, and the blood pressure 124 mm. Hg systolic and 68 diastolic. There was a marked degree of generalized arteriosclerosis. He was thin and under weight, and had suffered from a mild glycosuria, controlled by diet without insulin, since 1931. The urine was negative. An x-ray of the heart revealed no abnormality, and his electrocardiogram in the interval between attacks was of normal form without any evidence of heart-block. He was advised to take 1/2 grain ephedrine thrice daily. Under this treatment the attacks ceased and there was a great improvement in physical well-being and mental alacrity. In March, 1936, he had two minor attacks. In August, 1936, he had two attacks: these were attributed to the fact that he had forgotten to take his usual dose of ephedrine. Otherwise, during the past two and a half years he has had no attacks, and his improvement in health is remarkable.

In this case the diagnosis was made on the frequency and character of the seizures, the recognition of the slow

pulse immediately after the attack, and the regular normal rhythm at other times. The absence of other circulatory symptoms, such as dyspnoea, precordial pain, or oedema, would appear to be a common feature of these examples of cardiac syncope with intermittent complete block. My experience indicates that there are in all probability different clinical types of intermittent block, and doubtless the mechanism responsible for the abrupt and unexpected cessation of ventricular beating varies in different cases. In some, local lesions in the auriculo-ventricular bundle may be responsible; in others reflex vagal influences, or the ventricles may be limited in their range of rate.

Paroxysmal Tachycardia

A diminished cerebral blood flow may result from an excessive ventricular rate when the volume output per beat is greatly diminished. In these cases mild syncopal attacks or epileptiform seizures occasionally occur, and are usually preceded by a sensation of violent and rapid palpitation (Barnes, 1926; Clarke, 1907). In my experience fainting from this cause is more common in the later age groups, when sclerosis of the cerebral arteries might be expected. Younger people, the subjects of paroxysmal tachycardia, seldom complain of syncopal attacks. In either group the seizures are more often seen if the paroxysmal heart action begins when the subject is in the erect posture. It should be remembered that digitalis is a potent cause of paroxysmal tachycardia when used to excess in unyielding cases of congestive heart failure. Strangely enough, in this instance palpitation is seldom noticed by the patient, but the abnormal rhythm should be suspected when there are fits or faints during the treatment of congestive failure.

There are other cardiac causes for faints. After coronary thrombosis the acute fall in blood pressure may sometimes be responsible, or the damage done to the muscle may account for the development of a ventricular tachycardia with epileptiform seizures at the time of development of the abnormal rhythm. I have seen several examples of this cause of fainting and some wonderful recoveries under quinidine therapy. Syncope occasionally accompanies angina of effort. Valvular lesions are, however, rarely associated with syncope. I agree with Levine (1936) that of the valvular defects aortic stenosis rather than regurgitation is the commonest form associated with syncope. Clinical evidence leads to the conclusion that it is not so infrequent a lesion as pathological observations might indicate.

Syncope in the mind of the layman is closely associated with cardiac disease. Fortunately in young people organic vascular damage is as a rule easily excluded, but the pendulum has swung too far, and in my opinion it is justifiable to assert that fainting attacks in middle-aged or elderly people are a not uncommon symptom of cardiac disease, sometimes the only one. Even though physical signs are lacking, yet a careful clinical history and simple observations directed to the rate of the heart may give a valuable clue to diagnosis and suggest successful treatment.

Cerebral Seizures

The features of the major epileptic attack are well known. Petit mal is occasionally mistaken for a simple faint—but the more gradual onset, the longer period of unconsciousness, and the slower recovery favour simple syncope. Hysterical fits are now so infrequent that difficulty in diagnosis seldom arises (Bramwell, 1936). Convulsions may be seen in such a variety of conditions—Walker (1936) has listed over sixty different states in which

fits are of frequent occurrence—that it is not surprising that one common denominator applicable to all and sundry has not yet been found. The recent work of Gibbs, Davis, and Lennox (1935) supports the view that the fit is accompanied by an altered conduction of the nerve impulse and increased irritability of the nerve cells, alkalosis and waterlogging being factors of importance in the initiation of the unknown mechanism of the fit (Lennox, 1931; Lyon and Dunlop, 1933). Oxygen lack has its advocates, but in my opinion the convulsions of the Stokes-Adams seizure are not strictly comparable to the fit of idiopathic epilepsy. Having personally witnessed a large number of Stokes-Adams seizures of all grades of severity, my experience is that in grand mal the clonic stage lasts longer, and the sustained violence of the generalized muscular efforts sharply distinguishes it from the irregular jerking movements of the limbs commonly witnessed in the Stokes-Adams attack. Further, the convulsions of heart-block are not preceded by an aura, and the automatic behaviour, common enough after an epileptic fit, does not appear. Incontinence of urine may occur in either condition. In spite of the fact that inspection of the brain of epileptics at the initiation of an attack shows a cessation of pulsation in the pial arteries (Penfield, 1936), usually widespread over the hemisphere even though the seizure involves only one part of the body, the available evidence indicates that cerebral anaemia in itself is not the cause of epilepsy. So far as the cerebral circulation is concerned epileptic people have a peculiar vasolability (Penfield, 1936), but there is no proof that vasomotor phenomena actually cause the fit. Oppenheimer and Fishberg (1928) hold that the fits of eclampsia, lead poisoning, and glomerulonephritis are attributable to local vasoconstriction with high blood pressure, but the cause of the vasomotor change remains a mystery. Epilepsy of symptomatic origin is probably most often the result of cerebral tumour, particularly when situated in the frontal, parietal, or temporal lobes (Walker, 1936; Gibbs, 1932). Neurosyphilis and hypertension with vascular disease are next in importance. Epilepsy is not uncommon even several years after penetrating wounds of the dura, and the recent careful studies of Penfield (1936) lend hope to the view that surgery will accomplish more for the subjects of traumatic epilepsy in the future. The recognition of cysticercosis as a not infrequent cause of fits is of more than academic importance (Dixon and Smithers, 1934). Modern methods of investigation lend increasing support to the view expressed by Oppenheim (1911): "We must always be on guard when the epilepsy develops during later life."

The Epileptic Borderland

In this connexion it is worth referring to the peculiar attacks designated by the terms "narcolepsy" and "pyknolespy." In the former sudden short attacks of sleep occur unexpectedly and strange attacks of "cataleptic" paralysis brought on by emotion, particularly laughter, make it an easily recognized if somewhat uncommon malady. Recently Prinzmetal and Bloomberg (1935) have obtained better results in its treatment by the use of benzedrine sulphate than with ephedrine, which hitherto has not proved very satisfactory. Pyknolespy is confined to children. It consists of innumerable attacks, unassociated with mental deterioration, often indistinguishable from petit mal, except perhaps in their frequency and in their resistance to all known forms of treatment. Fortunately the fits invariably end spontaneously by the time puberty is reached.

The syndrome originally described by Gowers (1907), who suggested the term "vaso-vagal attack" to describe

it, might perhaps be renamed in order to distinguish it from the simple syncope or vaso-vagal attack of Lewis. "Vaso-medullary seizure" may be a more apt term, particularly as Ryle (1931, 1934) suggests that this condition is associated with a medullary "storm." Its relationship to epilepsy is obscure and perhaps no more than fortuitous, but in the absence of accurate records of blood pressure, pulse rate, and response to drugs, it is not yet justifiable to include this peculiar state amongst the better-known disorders of the autonomic nervous system.

Arising sometimes unexpectedly, but often after a period of prolonged mental strain or anxiety, the attack is sudden and lasts for ten minutes or half an hour before the symptoms begin to abate. The most striking, urgent, and dreaded of the symptoms is a sense of impending death (Ryle, 1934). With this comes a sense of utter powerlessness and profound weakness, a sense of unreality in the environment, a sinking or floating feeling, and occasionally some precordial discomfort, often distinct dyspnoea, a sensation of choking, or tingling in the extremities. Facial pallor, cold perspiration, and an appearance of grave illness are at once evident. The pulse is generally slow and of poor volume. The attack is not a hysterical phenomenon, though it is often vaguely labelled "nerves" or confusedly designated a "heart attack." In some ways, particularly in its utter helplessness, it resembles the milder symptoms of hypoglycaemia experienced by certain diabetics. So far as I am aware these attacks, alarming in themselves, are never fatal, and, whatever the precise mechanism, reassurance as regards the future, combined with sedative drugs—such as small doses of luminal—does much to diminish their frequency. In diagnosis a careful analysis of the patient's symptoms, the fact that consciousness is maintained, the duration of the attack, and the gradual recovery without convulsions serve to distinguish the condition from either the Stokes-Adams seizure or the epileptic attack. I have seen several examples of this condition, one man developing true idiopathic epilepsy five years after the first vaso-medullary seizure. This sequence is, however, exceptional.

In such a large field I realize that I have only touched on a few aspects of a fascinating clinical problem. My remarks have been based on personal experience of some of the difficulties of diagnosis—difficulties, I believe, which can be largely surmounted by an accurate clinical history, but more particularly by the good fortune of actually witnessing the attack, for accurate clinical observation and detailed but simple facts will solve most clinical problems.

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AN INVESTIGATION OF INTERNAL RADON THERAPY

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In this country the use of radium has been largely confined to its local action in the treatment of malignant conditions in which the objective is a destructive or deterrent effect upon the growth. In America and on the continent of Europe radium and other radio-active substances have been administered internally in small doses spread over long periods as therapeutic agents in a wide field of medicine, and considerable claims have been made as to their value. Apparatus capable of producing radio-active solutions for oral administration, injections containing small quantities of radium salts, and radio-active pads for external application have, however, appeared on the market here, and there is some evidence that their use is on the increase. Many of these are available not only to the medical profession but also to the public.

It was thought worth while at this stage, therefore, to investigate certain aspects of the subject under complete hospital routine, and the present paper is an account of the results obtained during the last five years. Although, as will be seen, the therapeutic results have been largely of a negative character, this is thought to be no reason for omitting to publish the data.

The objects of this investigation were: (1) to determine what physiological or biochemical changes might be induced in patients by such comparatively small doses as those generally used; (2) to assess the value, if any, of such treatment upon various diseases; and (3) to ascertain the effects of stronger dosages than those hitherto employed and to find when they become dangerous. Before recording the results of our experiments the following brief survey of what is sometimes known as "mild radium therapy" and of the literature available upon the subject is given.

Mild Radium Therapy

The first disintegration product of radium is the radio-active gas radon. From the point of view of therapy it is important to distinguish clearly between the internal uses of radium salts and of radon.

The salts of radium may be given by the mouth or may be injected intravenously or subcutaneously. Both methods are attended by definite risk. If given by the mouth considerable damage may be done to the mucous membrane of the intestines, especially if the patient undergoing treatment is constipated and the salts are held up. When injected intravenously it is stated (Oloff) that 80 per cent. is retained in the tissues, principally in the bone marrow, and that it disappears from these situations only very gradually indeed. Thus deposits of radium are formed from which radon may be produced for a long time.

There is not enough evidence to determine the exact quantity of radio-active salts which constitutes a lethal dose in man, but it is obvious that the internal administration of radium is attended with the gravest risks. According to Gudzent, a single dose of salt containing the equivalent of 0.5 mg. of radium element can prove fatal. Among the women who have lost their lives working with luminous radium paint the total amount of radium found in the body at the time of death ranged from 0.01 to 0.18 mg. The death of an American citizen was caused by the self-administered and repeated use of a solution containing one-thousandth of a milligramme each of radium and mesothorium per ounce of water.

The introduction of radon into the body, however, presents a totally different problem from that of radio-active salts. Its activity does not last long, declining to one-half in 3.85 days and diminishing to less than 1 per cent. in a month. When radon is taken by the mouth about 90 per cent. of the gas is eliminated through the lungs within an hour or two, and no more is exhaled after five hours. It remains in the urine for a longer time, but very little stays in the body to form "active deposit" (Flinn).

There is, however, another important distinction between the uses of radon and radium salts. Radon is a soluble gas, so that when swallowed it can be absorbed from the alimentary canal, and may, through the circulation, reach every tissue in the body. In this way it may be anticipated that there will be very free action by the alpha rays, which are known to have an intense local effect, not necessarily the same as that produced when radium is applied in platinum or other containers which exclude the alpha rays entirely.

The unit of radon is the millicurie, which is the amount of the gas in equilibrium with one milligramme of radium element. This unit has often been considered much too high for the measurement of very weak concentrations, and the Maché unit has accordingly been substituted; 2,500,000 Maché units are equivalent to 1 millicurie, or 1 Maché unit (M.U.) = 4×10^{-7} millicurie.

Some spa waters are radio-active, and one of the strongest of these is the spring at Joachimstal, which has a content of 600 M.U. per litre. Most radio-active springs contain weaker solutions than this, that of Baden-Baden, for instance, being 104 M.U. per litre. It will be seen, therefore, that the radio-activity of natural waters is exceedingly small, though perhaps not negligibly so.

Artificial radio-active waters can be prepared in very high concentration, but most of the work published on the subject has been done with daily dosage of from 8,000 to 30,000 M.U. per day, usually contained in one litre of water, although some (Bickel) have worked up to