DIZZINESS

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DIZZINESS is a symptom that is encountered several times a week in any general medical out-patient department yet it remains a notorious diagnostic puzzle. What does a patient mean by "dizziness"? What particular associated symptoms and signs can help in the diagnosis? How can one explain and treat dizziness in a patient who has no abnormal physical signs? This paper is an account of the authors' attempt to answer these questions from their experience over fourteen months in a medical clinic.

Even in health the word "dizziness" may refer to a number of different sensations:

1. The sense of rotation which persists after rapidly spinning around several times.
2. The dimness of vision and weakness of the legs resulting from postural hypotension after getting up quickly from a low chair on a warm day.
3. The fear engendered by looking down from a height to the ground beneath.
4. The confusion which results from attempting too difficult or too numerous intellectual problems. This is the origin perhaps of the closely related "mazy" derived from "being in a maze".

Thus dizziness is applied to physical, emotional or intellectual disturbances whose common denominator seems to be a loss of order or stability, a disruption of the pattern in which the individual is aware of his surroundings and their relation to him, whether these refer to his physical orientation in space, his emotional equilibrium or intellectual clarity.

Review of the Literature

There have been remarkably few reviews of dizziness over the past fifteen years and comparison between them is difficult. Over this period new disease concepts have appeared: basilar artery insufficiency, for example, and positional nystagmus of benign paroxysmal type which were not recognised in the earlier reports. Reviews may describe cases from neurological clinics or medical out-patients, or ear, nose and throat clinics, so that the patients are not comparable. Finally

the definition of dizziness on which the composition of the series primarily depends varies from one author to another.

De Weese (1950) reviewed the causes of dizziness in a large number of patients without giving figures. The importance of general medical causes such as anaemia, aortic stenosis and many others was emphasized. The diagnosis of psychogenic dizziness was not made unless there were other signs of neurosis.

Cawthorne (1957) reported more than 2,500 patients with dizziness which he defined as an hallucination of movement. All patients with anxiety or phobic reactions were excluded. In 85% the cause was in the labyrinth or in the peripheral vestibular apparatus comprising Meniere's disease, vestibular neuronitis, paroxysmal positional vertigo and perilymphitis. Eight per cent of patients had lesions in the central nervous system. In 7% the site could not be determined but he postulated a circulating toxin which had affected some part of the vestibular system.

Williams and Corbin (1958) reviewed 632 patients who presented with dizziness, defined as an unreal sensation of movement. Meniere's disease comprised 17% of the total, psychosomatic vertigo was the diagnosis in 7% and no final diagnosis was reached in 8%.

The review by Macrae (1960) of 400 patients with dizziness from a neurological clinic is of interest because 27% of the patients were deemed to have psychogenic dizziness. Meniere's disease comprised 20% of the total and 8% of the patients remained undiagnosed.

Barber (1964) surveyed 1,028 patients from an ear, nose and throat clinic. Meniere's disease was found in 15%; 9% of the patients had psychogenic dizziness and in 28% the aetiology remained obscure.

Method

The patients were selected from those attending two medical out-patient clinics at Hope Hospital, Salford, or who were seen by one of us (J.H.E.) in their homes over fourteen months from the 1st August, 1963. All patients who made a prominent or presenting complaint of dizziness, giddiness, mizziness, vertigo or a similar symptom which could be regarded as a sense of dysequilibrium were studied in detail.

TABLE 1

GENERAL MEDICAL CAUSES OF DIZZINESS

Islet-cell Adenoma of Pancreas ...	...	...	...	1
Iron Deficiency Anaemia ...	...	...	...	3
Diabetes Mellitus ...	...	...	...	3
Polycythaemia Rubra Vera ...	...	...	...	1
Functional Hypoglycaemia ...	...	...	...	1
Aortic Stenosis ...	...	...	...	1
Hyperthyroidism ...	...	...	...	1

Results

During the fourteen months of the study 100 patients were examined. The eventual conclusion about each patient was grouped into one of four categories: general medical disease, labyrinthine disease, central nervous disease and a considerable group in which no organic disease could be found to account for their dizziness.

General Medical Disease

In 11 patients dizziness was a prominent feature of some general medical disease as listed in Table 1. The dizziness in these patients was variously described as a sensation of movement or a feeling that they would fall. No patients complained of rotatory vertigo.

Labyrinthine Disease

The diagnosis of *Meniere's disease* or labyrinthine hydrops was made in eight patients who described recurrent episodes of dizziness associated with tinnitus and deafness in the absence of middle-ear disease. Their average age was 53 years and there were five females and three males.

All these patients had at times experienced severe rotatory vertigo, but many described in addition mild attacks which amounted to no more than a sense of unsteadiness. In the severe attacks dizziness lasted for some hours, and the sense of dysequilibrium persisted for several days after a bad attack. Nausea was invariably present and vomiting frequent. In three patients the attacks tended to occur on waking in the morning. Movement of the body and the head was not a significant factor in initiating an attack, but once it had started all patients noticed that such movement accentuated their dizziness.

In addition to deafness patients frequently

complained of some other disturbance of hearing, such as hearing less well in a noisy crowd, or difficulty in understanding speech. One patient could not enjoy music unless his affected ear was stuffed up with cotton wool. These spontaneous and characteristic descriptions were of considerable diagnostic value.

Eleven patients had *positional nystagmus of benign paroxysmal type* (Dix and Hallpike, 1952). This diagnosis depends on a characteristic reaction to placing the head in a certain critical position. The patient is laid flat on his back, his head rotated to one side, care being taken not to occlude the vertebral artery by over-extension of the head. If the test is positive he will remain comfortable for five to 10 seconds and then develop an intense vertigo accompanied by a rotatory nystagmus and sometimes by vomiting and sweating. If the position is maintained the effect wears off after thirty seconds or so. By returning the patient to the sitting position the vertigo and nystagmus will recur after a similar latent interval with less intensity and the nystagmus is in the opposite direction. On repeating the manoeuvre the reaction becomes less severe and soon fatigues.

This phenomenon was first described by Barany (1921) who attributed it to a disorder of the otolith apparatus and this assumption was later confirmed by the pathological studies of Cawthorne and Hallpike (1957). In a patient who died of intercurrent disease they demonstrated degenerative changes in the utricle which were attributed to atheroma of the arterial supply. The condition has also been associated clinically with infective middle-ear disease and may follow a head injury.

There were nine females and two males in this group and their average age was 56 years. In seven patients there was a history of head injury which preceded the dizziness, either immediately or by a short interval. The severity of the head injury as judged by the duration of post-traumatic amnesia varied considerably, but only two of the patients had radiological evidence of a fractured skull. In the remaining four patients the dizziness had appeared out of the blue without any history of middle ear infection or head injury and they were in the sixth and seventh decades of life. In two there was evidence of mild hypertension or a previous arteriopathy and it was presumed that their symptoms were due to atheromatous occlusion of a branch of the vestibular artery.

All these patients had recognised the rela-

tionship between their attacks and change in posture. It could be distinguished from simple postural hypotension by the patients' story that their symptoms came on when they lay down flat, turned over in bed, bent down to put coal on the fire or adopted similar positions which would not challenge their vasomotor reflexes. The other important diagnostic feature was that the dizziness, thus precipitated, did not last for more than a few minutes. It was usually described as rotatory, but this was by no means invariably so, and a description of rotatory vertigo was not essential to the diagnosis.

On examining these patients no abnormality was found elsewhere in the skull or the nervous system. The diagnosis of otolith damage, although it may be suspected on the history, could only be confirmed by executing this postural manoeuvre. The importance of this test in medico-legal examinations of patients with post-traumatic dizziness is clear. Several of the patients in this group were referred with a diagnosis of post-traumatic neurosis. This may indeed have co-existed, but the postural test put the organic basis of their dizziness beyond question.

One patient had a *perilabyrinthitis* secondary to chronic middle-ear disease which responded to surgical treatment. This low incidence of middle-ear infection is probably due to selection, patients with aural pain or otorrhoea being referred to the otology clinic.

Central Nervous Disease

One patient developed dizziness after a course of streptomycin. A negative finding of some interest is that no patient in this series was found to have an *acoustic neuroma*.

In 1949 Dix and Hallpike coined the term *vestibular neuronitis* (Dix and Hallpike 1952) to describe episodes of paroxysmal vertigo without evidence of disease of the central nervous system or the cochlear apparatus. This diagnosis was considered in five patients who described a period of vertigo lasting for from three weeks to six months, accompanied in each case by nystagmus. There was no evidence of cochlear disease, the response to posturing was negative and the caloric tests were abnormal. The course of the disease in three of these patients raised the question of *multiple sclerosis*. Two patients had slight but constant impairment of co-ordination on the right side of the body, indicating a cerebellar disturbance and one patient subsequently developed a retro-bulbar neuritis.

McAlpine, Compston and Lumsden (1955) found a presenting complaint of dizziness in 5% of patients with multiple sclerosis. It is clear that the clinical picture of vestibular neuronitis may be the first episode of disseminated sclerosis. The occurrence of dizziness and nystagmus at this stage, either in isolation or associated with other transient neurological signs, must carry a guarded prognosis.

There were fifteen patients with transient attacks of dizziness who had associated symptoms or signs of *basilar artery insufficiency*. These symptoms correspond to the transient ischaemic attacks associated with atherosclerosis of the vertebral artery and its branches which were described by Marshall (1964). Males and females were equally represented and their average age was 60 years. Seven patients described the dizziness as rotatory; the remaining eight used some other phrase such as "The floor came up to meet me" or "You feel your feet are going up t'brew". In seven patients additional symptoms were referable to other parts of the territory of the basilar or posterior cerebral arteries. Visual disturbances were commonest: a darkening of vision, a loss of vision, a bright light like a pin wheel, blurring of vision, or diplopia. Tingling down one side of the body or coming up from the legs, facial weakness or dysarthria were also reported. Nausea occurred in six patients and two vomited. The dizziness was frequently precipitated by sudden movement such as getting up from a sitting or lying position, looking upwards as in shaving or hanging washing on the line, turning the head quickly and looking down and in going down stairs. Lying down usually produced improvement of the symptoms. In the majority of patients the attack lasted for less than five minutes.

On examination in the interval between attacks abnormal neurological signs were found in all but one patient. Nystagmus occurred in nine patients. Two other patients developed nystagmus on being placed in the recumbent position and this nystagmus continued for as long as that position was maintained. It was therefore distinguishable from the positional nystagmus of benign paroxysmal type by having no latent interval, not exhibiting adaptation and not fatiguing with repeated posturing. Other signs commonly found were cerebellar ataxia, loss of pain and temperature sensation in the limbs and trunk on one side and extensor plantar reflexes. Two patients showed dementia, but above-normal performance on the pro-

gressive matrices was common in the rest of the group.

Two patients had evidence of *internal carotid artery disease*. One had a systolic bruit over the right orbit. Another patient had intermittent attacks of dizziness for a year which ended in a right hemiplegia from which he died. Post-mortem examination showed that the origin of the left common carotid artery was occluded in a ball-valve fashion by a sessile mural thrombus arising from the wall of the aorta.

A further eight patients had dizziness which was presumed to be due to *cerebral arteriosclerosis*, but the nature of their symptoms did not allow one to tell whether the carotid or vertebral system had borne the brunt of the disease. All patients were over the age of 60 years and the sexes were equally represented. The description of their dizziness was similar to that given by patients with basilar artery disease. On examination two of these patients had early Parkinson's disease, but none showed lateralizing neurological signs. Organic mental tests revealed a defect in four patients. Calcification of the internal carotid artery on X-ray was seen in three patients. These findings supported the assumption that there was diffuse cerebral arterial disease.

To complete the description of patients with central dizziness of known cause there were six patients in whom dizziness preceded a period of unconsciousness. In three the dizziness was an *aura to a grand mal seizure*. In the remaining three the nature of the unconsciousness was less clear. These attacks sometimes occurred while kneeling in church or standing in the assembly hall at school. While an epileptic basis for their attacks could not be completely excluded, the circumstances under which they occurred and their brief duration suggested that they were *syncopal* in nature.

Psychogenic Dizziness.

There remained a relatively large group of 32 patients in whom no abnormal physical signs were found, either in the central nervous system or in the vestibular apparatus, to account for their attacks of dizziness. Nor did the pattern of their symptoms fall into any of the recognised causes of vertigo which have been described. On the other hand, in each of these patients there was evidence of some emotional disturbance attributable to constitutional disability, environmental factors or a combination of the two.

Four patients gave a clear history of rotatory

vertigo, but the remaining patients were aware of a sensation of movement, either between themselves and the outside world or within their heads. This was described as a feeling of being pulled forwards, of losing their balance, often associated with a fear of falling. The symptom was sometimes localised within the head and described as a spinning, or a sensation of light headedness, or feeling as though they would faint. In many cases this was accompanied by a sensation of fear or even panic, and weeping was a frequent accompaniment or sequel of the episodes. Three patients described attacks of depersonalisation.

Within this large group two patients had developed their dizziness after a mild head injury and the symptom was part of a *post-traumatic neurosis*. Both were engaged in litigation and may be said to have had financial interest in the perpetuation of their symptoms. Two other patients gave a long history of chronic anxiety and they had been referred from a psychiatric department for the exclusion of organic disease. Their symptom of dizziness was just one amongst headaches, breathlessness and chest pain that are common in *chronic anxiety*. Once the clinical examination and special tests were found to be normal there was no reason to attribute their dizziness to organic disease.

There were two patients who had experienced attacks of dizziness which arose quite suddenly after childbirth, associated with episodes of depersonalisation and phobia. This syndrome to which Roth (1959) has given the name *phobic anxiety-depersonalisation syndrome*, has achieved some prominence in psychiatric clinics. Both our patients had similar histories and a brief summary of one of them will convey the pattern of the syndrome.

Case Report

A housewife of 48 years who had not experienced any serious ill-health until the age of 29 years when, after the birth of her first baby, she developed a severe post-partum haemorrhage necessitating blood transfusion. Her periods returned some weeks after the baby was born and remained regular and heavy. Within six months of the pregnancy she had become nervous and developed unexplained phobias, particularly of enclosed spaces. She also developed attacks of dizziness which occurred particularly when she was walking in the street. She felt as though she was falling to the right. At times her mind would seem somewhere else: "My mind is here, but my body is not". During this time she had consulted a number of physicians and on each occasion no organic abnormality had been found. When further investigations had been performed, such as a lumbar puncture, she had collapsed with a severe and persistent dizziness after the procedure. It was

curious that in spite of repeated reassurance and her own expressed fear of hospitals, she had persisted in seeking medical advice. In spite of this disability she continued to work all the time.

The diagnostic points in this story are the absence of symptoms until after the post-partum haemorrhage, the association of anxiety and phobia and episodes of depersonalisation aggravated by incidental surgical procedures and a persistently good work record. Fragments of this syndrome were found from time to time in other patients who did not show the complete picture.

The remaining 26 patients in this group were judged to be exposed to circumstances at work or in the home which, while not insuperable, could reasonably be regarded as precipitating symptoms of anxiety in a person so predisposed. In many instances the basic personality was a conscientious and mildly obsessional person with a good work record. The average score for neuroticism on the Maudsley Personality Inventory was significantly raised in this group above those with vestibular or central nervous disease, and above a group of 40 control patients selected at random from the medical and surgical wards. This finding suggests that constitutional factors were also involved.

There were six men and twenty women. The men were all working hard and under some pressure, either at work or in social commitments, or because of illness in their homes. In the twenty females the precipitating cause was related to their age. In young girls it was work at school and the pressure of exams, and in two instances maternal anxiety seemed to play a large part in the patient's coming to the doctor. At a later age the extra work involved in looking after an invalid husband, or taking on extra work for financial reasons was prominent.

In all these patients there was ample evidence of emotional disorder of some kind. Even though no organic cause for their dizziness was found one was reluctant to attribute their symptoms to emotional illness just by association. A search was made for some organic mechanism by which such patients were predisposed to develop dizziness, or at least were more aware of the symptom than normal people.

Study of the case histories of these patients suggested a possible mechanism in some. A common cause was postural hypotension. The anxious person often moves rapidly, jumping up from the sitting position so quickly that his vasomotor reflexes do not operate in time.

The dizzy spells in these patients were often associated with rushing hither and thither at work or in the home. One patient described his attacks as "like stepping off a step suddenly". There was a momentary shift in his surroundings and a sense of nausea. This only occurred when he was working hard. One could not help feeling in listening to his story that this was due to direct stimulation of his labyrinth by the quick activity in which he was engaged. Several other patients gave this kind of history and we came to recognise it as the "rushing around syndrome".

Hyperventilation or involuntary overbreathing was responsible for some of the attacks. The dizziness is accompanied by a sense of oppression in the chest, or breathlessness, dryness of the mouth, palpitation and pins and needles around the mouth and in the fingers. Confirmation of the diagnosis can be made by asking the patient to overbreathe deeply. Within 20 to 40 seconds symptoms similar to the attacks they have been describing will appear. Occasionally a trance-like state may result from which the patient can be released by suggestion. Hyperventilation may also reveal an epileptic tendency where none was expected. Such seizures tend to occur at the end of hyperventilation and the patient should always be told to rest quietly on the couch for five minutes after completion of the test with someone near at hand.

In elderly people their dizziness could at times be related to an intellectual difficulty in coping with a problem or an emotionally charged situation. A mild dementia was often found on organic mental testing and this almost certainly contributed to their symptoms.

As the importance of emotional factors in the production of dizziness became apparent we reviewed our patients with undoubted labyrinthine and central nervous disease and judged that the psychogenic factors played a part in the persistence of the symptoms in at least 19 out of those 57 patients.

Discussion

In reaching a diagnosis the patient's description of his dizziness was of less help than the precipitating factors and associated symptoms. A rotatory vertigo occurred more often in patients with organic disease, but it was also described by some patients with psychogenic dizziness. On the other hand a non-rotatory hallucination of movement, a sense of swaying from side to side, either of the patient or of his surroundings, was often due to a vestibular disease. Fear and anxiety frequently indicated

psychogenic dizziness, but this occurred as a supplementary factor in many patients with organic vertigo.

The history of the exact circumstances under which the attacks occurred often revealed that the dizziness was brought on by standing upright suddenly as in postural hypotension, or was related to movements of the head as in basilar artery insufficiency, or was precipitated by lying down with the head turned to a certain side, as in positional nystagmus of benign paroxysmal type. A history of dizziness occurring when working under pressure was found in patients with the "rushing around syndrome". A history of shortness of breath or gasping often suggested hyperventilation as a cause. Attacks occurring on attempting to cross a busy street, or while talking in a crowded room often occurred in elderly patients with an organic mental state.

Of the associated symptoms vomiting and loss of consciousness were not found in the patients with purely psychogenic dizziness. Tinnitus and deafness were essential to the diagnosis of Meniere's disease, but they occurred in many other patients, including those with long standing inactive ear disease which was not responsible for their present dizziness. Visual disturbances or symptoms referable to the cranial nerves or limbs were useful in indicating central nervous disease. On examination, evidence of aural disease or central nervous signs relevant to vertigo were of great value in indicating an organic aetiology. Provocative tests, such as posturing the patient for positional nystagmus and hyperventilation were of considerable value and easily performed. A complete general medical examination was essential to exclude a general medical disease.

A feature of this review is the high proportion, one-third of the patients, whose dizziness was deemed to be of psychogenic origin. It seemed that these patients presented the greatest difficulty in diagnosis. One is naturally reluctant to attribute this symptom to a psychiatric cause until an underlying organic disease has been completely eliminated. De Weese (1950) regarded neurotic dizziness as a dangerous diagnosis, which could lull one into a false sense of security. He preferred to classify these patients as undiagnosed and maintain a state of vigilant observation. At first we were instinctively sympathetic to this point of view, but we have come to the conclusion that this approach is unnecessary and even undesirable. Prolonged investigation leads to prolonged un-

certainty and further anxiety and perpetuation of the patient's symptoms if they are so engendered.

It is felt that in patients whose history and clinical examination reveal no relevant abnormality and whose life history indicates a source of anxiety, investigations should be limited to the simplest: a routine examination of the urine, a blood picture and X-ray of chest and skull. It is then our practice to explain to the patient the relationship of his dizziness to his environmental stress, or personal adaptation to his environment, giving examples of any mechanism such as postural hypotension, rushing around or hyperventilation which have been found. At the same time commonsense advice can be given to the patient on the resolution of his problems. If any sedative is necessary phenobarbitone, gr. $\frac{1}{4}$ b.d. or amytal, gr. $\frac{3}{4}$ b.d. is prescribed. Even in patients with undoubted organic disease attention is paid to any obvious psychogenic factors, for there are circumstances under which the patient is as disabled by his anxiety about his organic symptoms as by the vertigo itself. Follow-up of these patients at an interval of two months or so has shown that in those with environmental stress, improvement or complete relief of symptoms occurs, but in patients with evidence of long-standing anxiety, or the phobic anxiety-depersonalisation syndrome, the prognosis is less encouraging.

It is a pleasure to record our thanks to Dr. Hugh Freeman, Department of Psychiatry, Hope Hospital and to Mr. Peter Leeson, F.R.C.S., Department of Ear, Nose and Throat Surgery at Hope Hospital, for their help. We are also grateful to Mr. James Worsley, Principal Psychologist, Springfield Hospital, for many useful discussions. During the course of this investigation one of us received a Grant from the Manchester Regional Hospital Board.

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