EMERGENCIES IN GENERAL PRACTICE**SUDDEN GIDDINESS**

BY

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Perhaps the value of unhurried history-taking is nowhere better exemplified than in the case of the patient who complains of sudden giddiness. In most cases a proper analysis and interpretation of the complaint can be achieved by taking a careful history and performing a clinical examination. Ancillary methods of inquiry complete the clinical evaluation but are not essential.

Needless to say, the first requirement is to listen carefully to what the patient has to say about his symptom. Is it giddiness? Or is it something else? The patient who has experienced sudden giddiness usually has little difficulty in conveying to his doctor the fact that there has been some upset of equilibrium. The difficulty is not that giddiness goes unrecognized by doctor or patient, though this may happen, but that some other sensation is misleadingly described or interpreted as giddiness. So terminology becomes important—patient and doctor must know in what sense they use certain words. And perhaps no word is more misused than "giddiness."

Nature of Giddiness

Giddiness is a sensation of disturbed equilibrium. It is not just "faintness," "queer feelings," "muzzy feeling," "light-headedness," "floating," "sick feeling," or any of the many unpleasant sensations that some people experience under certain circumstances, such as on heights or in busy city streets. The person who experiences giddiness nearly always experiences a sensation of movement. His senses are deceived into seeing or feeling movement of one sort or another. His accustomed sense of correct orientation within his environment is temporarily disturbed. This abnormal sensation of movement is not necessarily one of rotation (vertigo). Vertigo is, of course, one of the commonest forms of giddiness; there is a sensation of turning (*vertere*, to turn), either in self, of self, or of the environment. Thus the patient may feel a swirling sensation in his head; he may feel that his whole body is spinning; he may see or feel his surroundings are turning about him. Vertigo is often used in this strict sense—to denote that the sensation of movement is one of rotation. But there is nothing to be gained by adhering to this definition, for we know that other equally distressing forms of giddiness, of identical aetiology and significance, may be characterized not by a revolving sensation but by a sense of falling through space, or by some movement of self or surroundings in any other plane. For example, the patient may describe how the ground rocks beneath him or appears to rush up towards him. He may see walls or objects tilting this way or that; the ceiling may look as if it is falling in or the pavement as if it were swaying. Or, bewildered, the patient may only be able to exclaim, "Nothing will stay still."

A given patient in different attacks of giddiness usually has much the same experience; the pattern of his vertigo remains the same, varying only in intensity and duration. Less often the actual illusions of movement vary in different episodes. Occasionally patients with paroxysmal vertigo and its characteristic accompaniments do not seem to experience a sense of movement of any sort. They may stagger, fall, and vomit, but afterwards deny that their giddiness consisted in any abnormal sensation of movement.

Whatever form the giddiness actually takes the patient will try to clutch hold of something or sit or lie down. He

may close his eyes in an attempt to shut off the distressing impressions of movement. Pallor, sweating, nausea and vomiting, even diarrhoea, may follow. The patient not only feels very ill and prostrated but his appearance may be alarming. The total picture may suggest some catastrophic illness or food-poisoning, not a transient upset in one ear. If the vertigo is abrupt and severe the patient will fall to the ground, perhaps staggering about in a drunken manner at first. He may injure himself trying to get upstairs to bed; he may be concussed. Occasionally he may go down like a log, without warning, but later he may recollect feeling a sensation of movement as he fell. At other times he seems just to "fold-up" as if all sense of posture had left him; he drops to his knees in an inexplicable fashion.

Sudden attacks of vertigo are usually spontaneous and irregular in occurrence; they may appear at any time of the day or night, when awake or when sleeping. In one variety the attacks are provoked by certain movements or the adoption of a certain posture. Turning the head or body, changing from the horizontal to the upright position, lying on one side, bending down, or looking upwards are examples of such movements. Consciousness is rarely affected. Between attacks the patient may feel perfectly well in respect of his sense of balance, but some patients may feel a more or less continuous state of unbalance. Rarely the doctor is consulted before any acute episode is experienced. The vagueness of the sensations may suggest a diagnosis of neurosis. Vertigo may therefore present as paroxysmal vertigo, postural or positional vertigo, or as chronic vertigo.

Aetiology

Correct orientation of the body requires information provided by the retinae, the proprioceptors of the muscles of the eyeballs, neck, trunk, and limbs, and above all by the two labyrinths. The co-ordination of the afferent impulses derived from these sources is effected by the vestibular nuclei and the posterior longitudinal bundle in the brain stem, by the cerebellum, and by the temporal lobes. Vertigo may therefore result from a lesion of the sensory end-organs, of the afferent paths, or of the central mechanisms. In practice nearly 90% of cases of vertigo are due to labyrinthine lesions.

Causes of Giddiness**Peripheral Lesions:**

1. Aural:—Ménière's disease; vestibular neuronitis; positional vertigo; trauma; infection (mumps, measles, herpes zoster, meningitis).
2. Ocular:—Diplopia.

Central Lesions:

1. Acoustic neuroma.
2. Brain stem:—Disseminated sclerosis; thrombosis of the posterior inferior cerebellar artery; tumours.
3. Cerebellum:—Tumour; haemorrhage and thrombosis.
4. Cerebral:—Raised intracranial pressure; tumours; arterio-sclerosis; epilepsy; migraine; drugs (barbiturates, phenytoin sodium, streptomycin, etc.).

Preliminary Investigation of the Patient

During an attack of acute vertigo a brief examination is usually possible.

Aural.—Is there any gross impairment of hearing? The external auditory meatus should be examined for wax, evidence of otitis media, or herpes zoster.

Nystagmus.—During an attack of aural vertigo the patient usually lies on the sound side. If he opens his eyes and looks

upwards to the affected ear, nystagmus will be seen. If he is lying on his back, the nystagmus is increased in amplitude when the eyes are turned to the sound side.

Ocular Movements.—Severe vertigo rarely results from diplopia. In most instances of diplopia there is no falling or sickness.

Ocular Fundi.—Raised intracranial pressure may itself cause some vertigo, and posterior fossa tumours will also cause ataxia and vomiting. Swelling of the optic disks and vascular engorgement may already be present.

On recovery a more careful examination can be made.

Aural.—Is there impairment of hearing on either side? Is the deafness conduction (middle ear) or perceptive (nerve) in character? These may be distinguished by the standard tuning-fork tests. **Weber's Test:** When a vibrating tuning-fork is placed on the vertex of the skull, the sound is best heard in the deaf ear in conduction deafness, and in the good ear in perceptive deafness. **Rinne's Test:** Normally air conduction is better than bone conduction, so that a vibrating tuning-fork is better heard when held near the ear than when its base is placed on the mastoid process (the ear being closed by the observer's finger). In conduction deafness the sound cannot be heard by air conduction after bone conduction has ceased to transmit it; in perceptive deafness the reverse is true. Special tests are necessary to distinguish the two forms of perceptive deafness—namely, lesions of the cochlea and lesions of the auditory nerve. Auditory function and vestibular function may be further investigated in hospital.

Ménière's Disease (Recurrent Aural Vertigo : Endolymphatic Hydrops)

Successive attacks of swelling of the membranous labyrinth with eventual irreversible changes are responsible for the well-known sequence of clinical events. Attacks of vertigo, vomiting, and prostration are usually associated with tinnitus and increasing deafness. The vertigo usually lasts several hours at a time, rarely more, and may take any of the forms described above. It is a disorder of middle life and is slightly more common in males; most patients have their first attack before the age of 50. The disease usually affects one labyrinth before the other and appears to involve both cochlear and vestibular components from the start. There is considerable variation in the order of appearance of the three symptoms—vertigo, deafness, and tinnitus. Most commonly vertigo and deafness are first noticed at about the same time. The disease runs a protracted course with phases of activity and quiescence. Thus attacks may appear in groups; for several weeks at a time tinnitus may be loud and persistent, and the hearing distorted or impaired. The patient may feel that one or both ears are "blocked" or "full" and that his sense of balance is "not just right." There may be complaint of headache or "fullness" or "pressure" about the head, and sedentary workers may find concentration difficult. It is during such active phases of the disease that sudden vertigo is especially apt to occur, and the experienced sufferer, by noting such symptoms, may be forewarned of the acute attack.

There is nystagmus with the quick component toward the affected ear, which, as the patient lies on his side, is usually uppermost. The nystagmus subsides with the vertigo and is not usually to be found during intervals of complete freedom. Deafness, on the other hand, tends to be progressive and is of perceptive type. Each attack or group of attacks tends to leave some further impairment of hearing. The classical tuning-fork tests reveal that bone conduction of sound is reduced as well as air conduction.

Vestibular Neuronitis and Paroxysmal Positional Vertigo

The term "vestibular neuronitis" has been applied to a group of cases in which vertigo due to vestibular disturbance is unassociated with any cochlear involvement. There is no tinnitus or deafness but caloric responses are reduced. The affection is seen in a younger age group (30 to 50 years), the vertigo is less paroxysmal, and there may be only one attack. The pathology is not known but it is thought to be in the nature of a selective lesion of the vestibular nerves or

nuclei. It may follow respiratory infection or appear to be associated with focal sepsis; it may account for some forms of so-called "epidemic vertigo."

In paroxysmal positional vertigo there is usually not only no involvement of the cochlear apparatus but caloric tests are normal. Nystagmus and vertigo are provoked by certain movements or critical positions of the head. It appears to arise from a non-progressive lesion of the otolith apparatus or from one more centrally placed in the vestibular pathways. The nystagmus and vertigo can nearly always be made to "fatigue" by repetition of the particular movement which provokes them in the first place.

Both vestibular neuronitis and positional vertigo are less incapacitating than Ménière's disease and run a milder course, usually terminating spontaneously. Their separation from the condition of endolymphatic hydrops renders further use of the term Ménière's syndrome inappropriate. What Ménière described was a disease *sui generis* of the internal ear.

Injury and Infection of the Labyrinth

Vertigo and distortion or impairment of hearing, or both, may follow a head injury, especially when there has been a fracture through one petrous temporal bone. If impairment of hearing does not draw attention to the internal ear the true nature of the symptoms may not be appreciated. Caloric tests are helpful.

Chronic middle-ear infection sometimes leads to involvement of the bony labyrinth. Vertigo may be acute or chronic and may be aggravated by any increase of pressure in the middle ear. The general health of the patient often deteriorates and aural surgery is necessary. Involvement of the labyrinth may occur during the course of certain acute infections such as mumps, measles, or meningitis. In the Ramsay Hunt syndrome (facial palsy and otitic herpes) vertigo is often a feature.

Posterior Fossa Tumours

In general, tumours in this region present with symptoms of intracranial hypertension or disturbance of balance, or both together. The commonest tumour in this situation is an acoustic neuroma growing in the cerebellopontine angle. Vertigo is a common symptom and it is often paroxysmal as in Ménière's disease. In both conditions tinnitus and nerve deafness are usually present, though not necessarily complained of or related to the vertigo. In the tumour case the complaint of giddiness is probably commoner than the complaint of deafness, while symptoms of intracranial hypertension or of involvement of adjacent cranial nerves may be absent. Differential diagnosis may therefore be difficult. With acoustic neuroma there is often a story of numbness or stiffness on one side of the face and the corneal reflex may be absent. Disturbance of gait tends to be progressive and independent of vertiginous episodes. The tumour patient may recognize that this loss of balance is due to lack of control over his limbs and not to some disturbance in his head. This should be put to him. He may also have noticed that one upper limb is clumsy when there is no question of disturbed balance—as when he is sitting down or reclining. The nystagmus in the tumour case is not restricted to periods of vertigo and its fast component is to the opposite side.

Brain-Stem Lesions

Sudden involvement of the vestibular connexions in the brain stem, as in disseminated sclerosis or thrombosis of the posterior inferior cerebellar artery, provokes vertigo. In disseminated sclerosis there is no evidence of cochlear disease and the vertigo and nystagmus are usually, but not always, accompanied by signs of involvement of other nuclei and tracts. There may be diplopia and sensory symptoms. Recovery is less prompt than in aural vertigo. Thrombosis of the posterior inferior cerebellar artery may lead to ptosis with impairment of pain and temperature sensation on the same side of the face and on the opposite side of the body.

These signs may be noticed by the patient when he shaves or takes a bath.

Tumours of the brain stem are more likely to give rise to a sensation of chronic giddiness with nystagmus. Localizing signs are seldom late in making their appearance.

Management

The management of an acute attack of giddiness is the same whatever its cause. The following description, however, is related to Ménière's disease, as this is by far the commonest cause.

The patient should not be moved unnecessarily during the height of the acute attack. He should be allowed to lie down in a comfortable position of his own choosing as soon as possible. Many patients will later describe that the journey upstairs, dragged there by misguided members of the family, the undressing, and the getting into bed were the worst experience during the illness.

As soon as possible patient and family must be reassured. The episode is alarming, but it will subside in a few hours and the illness is never fatal or paralysing. No drug has a specific effect on vertigo, but phenobarbitone has a general sedative effect and perhaps reduces the sensitivity of the labyrinths. An intramuscular injection of sodium phenobarbitone, 3 gr. (0.2 g.), should be given immediately and oral phenobarbitone, $\frac{1}{2}$ gr. (32 mg.), three times a day, should be continued for some weeks afterwards and during the phases when the patient feels that attacks are "just around the corner." In Ménière's disease seasickness remedies such as hyoscine and antihistamines—for example, promethazine and dimenhydrinate—may be helpful but are in no way specific. Fluids should not be pushed in an effort to correct acidosis or dehydration consequent on vomiting, as this may precipitate further attacks. Fluids should be restricted for a week or two to 2 pints (1.1 litres) in twenty-four hours and salt should be omitted from cooking and at table. There is no certain evidence that this "antiretentional" regime needs to be continued indefinitely. The patient should be advised to adopt it when he suspects a repetition of the attack.

In Ménière's disease the use of vasodilator drugs such as histamine and nicotinic acid and the search for focal sepsis or allergens all have their advocates, but in my experience are not rewarding. The course of the disease is extremely variable and most patients who have been reassured manage reasonably well by restricting their activities, using phenobarbitone, learning to recognize premonitory symptoms, and going to bed. The unexciting nature of such advice may be unacceptable to some patients or their doctors, but in the present state of knowledge it applies to the majority of patients with Ménière's disease. When periods of remission do not occur and attacks cannot be modified by such measures the question of destruction of one labyrinth by alcohol injection, diathermy, or removal of the external semi-circular canal will have to be considered. Section of part or whole of one auditory nerve is a serious intracranial operation which has little part in the treatment of this disease.

When the acute attack of vertigo has subsided a general overhaul, with aural and neurological examination, is advisable. Lesions outside the ear and nervous system seldom produce paroxysmal vertigo.

Next article on Emergencies in General Practice.—"Diphtheria," by Dr. A. B. Christie.

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BIOCHEMISTRY AND CLINICAL MEDICINE

CONTRIBUTIONS FROM THE THIRD INTERNATIONAL CONGRESS, AUGUST, 1955

[FROM A SPECIAL CORRESPONDENT]

The Third International Congress of Biochemistry, held at Brussels from August 1 to 7, was attended by over 2,000 delegates from all parts of the globe, including a large group from the Soviet Union. Some idea of the scope of modern biochemistry can be gathered from the fact that the congress was subdivided into 17 sections, all of which presented a full programme. As a result it was impossible, as at many such international gatherings, to attend more than a small proportion of the meetings in person. While no world-shaking discovery such as would necessitate a major reorientation of current biological theory was announced at the congress, there were plenty of papers of clinical interest, and it is with some of these that this report is mainly concerned.

Metabolic Disorders

In the section of pathological chemistry and immunology, H. HARRIS (London) opened a discussion on amino-aciduria in man. After explaining that a normal adult excretes about 1.1 g. of amino-acids daily, together with 2 g. in the combined form including peptides, and discussing techniques of estimation, he went on to classify the pathological amino-acidurias. He divided them into "overflow" and "renal." In the former category were liver diseases and phenylketonuria, in the second group cystinuria, Wilson's disease, galactosaemia, the Fanconi and similar syndromes, scurvy, rickets, and lead and lysol poisoning. The speaker made it clear that, despite many uncertainties, the main factor in the first category is a raised blood level of amino-acids, and in the second category defects of tubular reabsorption. In another paper, B. H. BILLING and G. H. LATHE (London) summarized their work on jaundiced serum. They have identified three bile pigments in such sera; a fat-soluble one, giving the "indirect" van den Bergh reaction and identified as bilirubin, and two water-soluble pigments giving the "direct" van den Bergh reaction. During the excretion of bilirubin in the bile the pigment is converted to one of these two water-soluble pigments.

A contribution to the curious metabolic disorder of children known as gargoylism was made by L. L. UZMAN (Washington, D.C.). He had isolated from the spleen the storage substance that accumulates in the cells of these patients and fractionated it. The material comprised two components; fraction S consisting of fatty acids, hexoses, hexosamine, hexuronic acid, and neuraminic acid; and fraction P, a sulphate ester polysaccharide of high molecular weight whose sugars are glucose and galactose and which also contains glucosamine and hexuronic acids. Another communication of paediatric interest was given by M. REINER, D. H. HIGGINBOTHAM, and A. PATZ (Washington, D.C.). They had investigated in animals the mode of action of high oxygen levels in causing retrolental fibroplasia in immature retinae. At concentrations of 50% and above oxygen caused vasoconstriction of the retinal vessels. Total obliteration was found at concentrations of 80–100%. But the vasoconstriction was not irreversible until after three days of continuous exposure to high oxygen levels; it was not reversed by adding 5% carbon dioxide to the oxygen.

Protein Chemistry and Hormone Structure

The section of protein chemistry revealed steady progress in many fields. P. H. BELL and R. G. SHEPHERD (Stamford, Conn.) described their work that had led to the purification and structural analysis of β -corticotrophin (A.C.T.H.) and issued the audience with charts showing the complete amino-acid sequence of the molecule of this hormone. C. H. LI (Berkeley, Calif.) produced further evidence showing that the