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Episodic Giddiness

W. B. Matthews[1] writes, "There can be few physicians so dedicated to their art that they do not experience a slight decline in spirits on learning that their patient's complaint is of giddiness." This is partly because the term covers such a variety of symptoms and partly because even in the best hands a considerable proportion of cases will defy exact diagnosis.

Fortunately true episodic vertigo, with its sudden onset, sense of rotation of self or surroundings, vomiting, and ataxia, is sufficiently vivid for most patients, including children, to leave no doubt about its nature, if not its cause, and considerable advances have been made in the last 20 years in the differentiation of the different types. The diagnosis "acute labyrinthitis" should be made with great caution. Though local aural disease undoubtedly causes vertigo, and a chronic ear infection may be of great importance in this respect, in the absence of such local disease vertigo may arise in any part of the complex vestibular system from the labyrinth itself to its most central connexions in the temporal lobes, and it is its very complexity which adds to the difficulty. Ménière's disease, so familiar a term, is too readily diagnosed. It is a condition mainly of middle age, in which relatively brief episodes of vertigo are accompanied and usually preceded by tinnitus and progressive deafness, and each attack may be ushered in by an increase in the distortion of hearing and tinnitus.[2] Audiometry and caloric tests will show both loss of cochlear and canal function, and the phenomenon of loudness recruitment[3] is most likely to be demonstrable. Terms such as "pseudo-Ménière's disease" probably cover so wide a variety of symptoms that they should be avoided.

Vestibular neuronitis, a disorder described by M. R. Dix and C. S. Hallpike,[4] affects all age groups but mainly younger patients, may be preceded by an infective illness, and consists of a gradual or a sudden onset of vertigo, vomiting, and ataxia severe enough at times to give a sensation of "blacking-out," prostrating the patient for hours or days and leaving a sense of vertigo on movement for many weeks. Some patients do not show the same episodic onset, but all are quite free from cochlear symptoms, and caloric tests show canal paresis, which may be bilateral. The condition is benign. It occurs sporadically, but a very similar condition, well described by M. S. Harrison,[5] may occur in epidemic form. The benign paroxysmal vertigo that L. S. Basser[6] drew attention to, affecting children in the age group 3–8, has the same characteristics. It also has an excellent prognosis, but is often undiagnosed because the child is not allowed to tell his own story, which can be very vivid.

But it should be borne in mind that, in children and adolescents in particular, vertigo without any of the other features of labyrinthine disturbance may be an epileptic manifestation arising from the temporal lobe. The attacks are short-lived. Loss of consciousness, if it occurs, is a vital clue, and even without frank unconsciousness a clear-cut memory of the details of the attack makes this diagnosis much less likely. The caloric tests show no canal paresis; electroencephalography can give the

diagnosis. E. R. Bickerstaff[7] coined the phrase "basilar artery migraine" for migrainous attacks preceded or accompanied by symptoms of disturbed function of the brain stem. His patients were mainly adolescent, but vertigo, followed by severe headache and vomiting, may be part of a basilar migrainous aura without the full picture of dysarthria, visual impairment, ataxia, and peripheral dysaesthesia. Vertebrobasilar deficiency of a structural type is the most common cause of vertigo in later life, particularly if occurring under conditions when the blood supply from the vertebral artery may be embarrassed, such as a sudden change to an upright position or excessive twisting of a spondylotic spine, while more prolonged vertigo can follow pontine infarction. Other signs of disturbance of the brain stem are in this case likely, but not invariable.

The relationship to posture is important in all forms of vertigo, but "benign positional vertigo" gives a clear-cut clinical picture. The patient becomes giddy when first lying down at night, or on first turning over in bed. The condition is relieved by sitting up and carefully repeating the motion, but it may occur again on sitting up in the morning. Also without cochlear features, it can be reproduced by simple tests during bedside examination[8] and may follow a variety of illnesses, including head injuries. As vertigo is a feature of brain stem disease, such as vascular accidents, multiple sclerosis, tumours of the posterior fossa, and acoustic neuromata, there is always anxiety that such conditions may be overlooked. But as a purely isolated presenting symptom, without any other features to suggest the nature of the lesion, it is very uncommon except in vertebrobasilar insufficiency. However, if there are any other such features, radiology of the skull and the internal auditory meati and audiometry and caloric testing should certainly be carried out if cochlear or brain-stem function is also thought to be involved.

There remains the most difficult group of all, in which the aetiology is most uncertain. M. J. Eadie[9] has recently tried to clarify it by a study of 62 patients suffering from episodic non-positional giddiness which was not due to ear infection, vertebrobasilar insufficiency, or Ménière's disease. Seven corresponded to the criteria for vestibular neuronitis; 8 were due to epilepsy; 34 were thought part of an attack of migraine; and 13 remained undiagnosed. It was thought that some of the last group might be migrainous, without headache; others might be examples of Ménière's disease without cochlear features; and others might be added to the group of vestibular neuronitis, for though this term suggests an infective origin it is in fact merely a syndrome, certainly a reproducible syndrome, but of a nature as uncertain now as when it was first described.

The differentiation of episodic giddiness depends primarily on accurate history taking, supported by the increasingly sensitive tests of cochlear and vestibular function. But, in the present state of our knowledge, despite all these there will still remain the group of patients who will continue to cause the physician that "slight decline in spirits."

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6 Basser, L. S., *Brain*, 1964, **87**, 141.
7 Bickerstaff, E. R., *Lancet*, 1961, **1**, 15.
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Progressive Patient Care

That the most seriously ill patients in a ward should be nursed in beds close to the sister's office has been accepted in most British hospitals for many years, and was certainly advocated by Florence Nightingale. The grouping of patients into different wards according to their needs, the logical extension of this arrangement, was widely practised in American military hospitals and later adopted with enthusiasm by geriatricians.[2] The increasing need to concentrate specialized equipment and services into units for intensive care, recovery, and coronary care, the shortage of skilled nursing staff, and the rapidly rising cost of inpatient treatment are now forcing many doctors and administrators to accept the concept of progressive patient care and to abandon the old, rigid allocation of hospital beds among individual consultants.[3]

A recent survey[4] suggests that between 1% and 2% of acute hospital beds are needed for intensive care (much higher figures being accepted in the U.S.A.), while 27% of patients could be given hostel-type accommodation with minimal nursing and medical care. It was also suggested that as many as 22% of patients in hospital did not need to be there at all if use were made of community services. This confirms that there is often a serious lack of liaison between the hospital and the local welfare services and of appreciation of what can be done for convalescent patients at home.[5]

Most British hospitals still fall short of the ideal of "the right patient in the right bed with the right services at the right time."[6] It was therefore encouraging to read the account of the experiment in progressive patient care being conducted by R. Hartley and his colleagues in a small hospital.[7] This involved the grouping of 8 wards of 189 beds into intensive care, intermediate, and homeward bound units, the extra nursing and service needs of the first being largely found by reducing the provision made for the last.

The success of this experiment must largely be due to the co-operation of medical and nursing staffs and their willingness to accept much more flexible roles than their accustomed ones. Many other difficulties are likely to be met in carrying out such a scheme—the need, for instance, to provide specialized equipment and highly trained staff for both coronary and intensive-care units while keeping them physically separate, as well as for recovery units, which most American advocates of progressive patient care prefer to keep separate also.[8] Moreover, the very varying workload of medical and surgical wards,[9] even in the "intermediate"

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