

that he should have adequate time to keep in close contact with all the mothers and perhaps to assist the sister in the instruction of nurses. It is noteworthy that Illingworth *et al.* found the services of two registrars to be necessary at the Jessop Hospital for Women, Sheffield.

Where supervision is not adequate self-demand is likely to degenerate into regular feeding, with "a little drop in between if he cries." This happened in our ward not supervised by the registrar.

We wish to thank Professor T. N. A. Jeffcoate and Mr. C. H. Walsh for permitting us to make a trial of this method of feeding in their wards, and also to express our appreciation of the willing co-operation of the nursing staff.

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ACUTE LABYRINTHITIS

BY

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In the *Lancet* of February 9, 1952 (p. 299), it was suggested that there was prevalent at the time an unusual epidemic characterized by the sudden onset of vertigo and nystagmus. Nystagmus is described as "a rapid oscillatory movement of the eyes, independent of the normal movements, which are not affected" (Law, 1945). Vertigo, the causes of which are many, depends upon "a disturbance of the sense of equilibrium" (Farquar Buzzard, 1945), and can be divided into general vertigo and special vertigo. In the latter, objects appear to move, or the patient may himself tend to fall in a definite direction. Nystagmus has been classified (Russell Brain, 1947) into several distinct groups: (1) defective or abnormal retinal impulses; (2) abnormal labyrinthine impulses; (3) lesions of the cervical spinal cord; (4) lesions involving the central paths concerned in ocular posture; (5) weakness of the ocular muscles; (6) congenital abnormality of unknown aetiology; and (7) hysterical. In the present series the nystagmus fell into group 2, and was labyrinthine in origin.

Labyrinthitis has been described in two forms (Tidy, 1945): (1) Ménière's disease, and (2) Ménière's symptom-complex, in which there occur tinnitus, attacks of vertigo, nausea, and progressive deafness. Ménière's disease has three main classes of symptoms (Purves-Stewart, 1945): first, violent giddiness and reeling caused by involvement of the semicircular canals and vestibular mechanism; then with involvement of the cochlear auditory apparatus, tinnitus, and nerve deafness, incomplete at first but fluctuating in intensity; and, thirdly, cold, clammy sweat, and nausea and vomiting of bulbar phenomenon, due to affection of the adjacent medullary centres. Finally, Ménière's disease usually occurs in later years, and is relatively uncommon.

The six cases recorded below have all been seen in routine practice within the last year. They are my own patients, visited in the usual manner. They all reside within a radius of one mile from my surgery.

Case 1

A man aged 58 was first seen at my surgery on the evening of April 12, 1951, with a history of severe vertigo of sudden onset from early afternoon. In standing and walking he was assisted by his wife, and was literally falling

around the room. His oral temperature was normal, and his pulse, although fast, was regular and of good volume. No pathological condition was found in the ears, and examination of the heart and lungs provided no clue to his condition. His blood pressure was 180/90 mm. Hg. There was gross rotating nystagmus, but no other abnormality was found in the central nervous system. He was seen at his home on April 13, when there was little change in his general condition. Nystagmus was produced by opening the eyes and attempting to focus on an object, or on very slight movement of the head. When the nystagmus was present the patient had intense vertigo, which produced vomiting, sweating, and a cold clammy skin. On the 14th he had improved, and on the 16th could sit up in bed without vertigo. On the 19th he was able to attend the surgery, but complained of "lightness of head," so that he did not feel well enough to return to work until April 30.

On May 7 he was examined by an otorhinologist, who reported: "This case suggests an acute labyrinth irritation, which has not recurred since. The right Eustachian tube is completely blocked." On June 4 an audiometric test showed slight impairment of both auditory nerves. Some 11 months later the patient was well and symptom-free.

Case 2

A married woman aged 73 suffering from pernicious anaemia had been receiving regular liver therapy, and her last blood report showed 5,000,000 red cells and 98% Hb. I first saw her on November 10, 1951, when she was in bed in a shocked state, suffering from intense vertigo and sickness. It appeared that she arose as usual at 6.30 a.m., fed her fowls, and had breakfast. At about 8.30 a.m. she was bending over her sink preparing the lunch, when suddenly she was overcome with intense vertigo, vomiting following shortly afterwards. She was carried to her room, and was lying in bed, fully clothed, terrified to move or to open her eyes. Rotatory nystagmus was gross, and she was violently sick and dizzy when attempting to sit up or to move her head. There was no pyrexia, and, apart from the nystagmus, there were no abnormal findings in the central nervous system. I visited her daily for some days, during which her recovery was progressive, and on November 16 she was able to walk down the stairs unaided. I have seen her several times since then, and she has remained perfectly well.

Case 3

This patient was a man aged 61. I visited him at his home on December 17, 1951, and his syndrome was similar to that of Cases 1 and 2. He was finally seen at his home on December 24, when he was able to move about the house. He did not return to work until January 14, 1952, because of feeling "light-headed" on sudden movement. His daughter was, at the same time, confined to bed with a severe tonsillitis. Unfortunately I did not have a throat swab taken, but the case responded to penicillin injections, and the pathological state suggested infection by the haemolytic streptococcus.

Case 4

A man aged 56 went to work on January 10, 1952, feeling fit. He was writing on the blackboard, when suddenly he found that it had begun to dance before his eyes. He got into his car and drove four miles to his home. When I visited him shortly after his return he had slight rotatory nystagmus on moving his head. The next day the vertigo and the nystagmus had gone and he felt quite fit. He returned to work on January 14, and has had no recurrence of his complaint.

Case 5

A married woman aged 30 awoke on the morning of January 15, 1952, feeling perfectly well. On attempting to get out of bed she had intense vertigo. When seen by a trained nurse about two hours later the vertigo had not decreased and there was extreme nausea. She was reported

to have "fallen back in a faint" each time she attempted to sit up, but she was not completely unconscious, since she was capable of understanding what was said to her. She was cold and shocked and pallid when the vertigo occurred. The next day she was able to sit up in bed without vertigo, and no nystagmus was present. She was "light-headed" for about two weeks after this attack, and sudden movement produced mild vertigo.

Case 6

This case is of special interest in that the patient was a next-door neighbour of Case 4. It is of interest, too, because the onset of his symptoms was just over 48 hours after that of his neighbour. A history of contact is available: on the morning of January 10, 1952, the patient sent for medical aid for Case 4. He then went into his neighbour's house to inform him that the doctor would soon arrive, and spent about five minutes in conversation.

The patient, a man aged 49, went to the cinema with his wife in the afternoon of January 12. To use his own words, he "felt as fit as a fiddle." After about half an hour he suddenly found that the screen was "beginning to wobble." He decided that this was due to the fact that he was sitting nearer to the screen than was his custom. The feeling passed off in a few minutes, only to recur. That too passed off, and he remained for another two hours, until the programme had finished. When he arose to leave the vertigo suddenly returned, so that it became necessary to hold on to his wife lest he should fall. Outside he felt much better and returned home by bus—a distance of about 10 miles. After entering the house the vertigo returned with great intensity, and at one stage it seemed as if the floor on his left occupied the position where the ceiling ought to have been on his right. The whole room seemed to rock and sway, but he himself did not appear to move. In a few minutes he was violently sick and the sickness lasted for about one hour, after which he managed to retire to bed and fell asleep exhausted. The next morning his "stomach was upset," but there was no further vomiting or vertigo. He returned to work on the 14th. On January 31 he stated that he had a slight feeling of falling when he was stepping forward. This lasted two or three days and then passed off. The symptoms were purely subjective, and he did not experience true vertigo.

He first came to see me on February 7 because since his illness on January 12 he had suffered from "palpitation, heartburn, and light-headedness," and seemed to have indigestion and flatulence after meals. Clinical examination showed his heart to be sound, and his blood pressure to be 150/94 mm. Hg. He had had a partial thyroidectomy in 1922 for parenchymatous goitre, otherwise his previous history was of no special interest.

On March 6 he was examined by an otorhinologist, who reported: "There was no obvious cause in the way of a precedent cold, earache, or general disturbance. Both drum-heads appeared normal. In both ears air conduction was better than bone conduction, and the Weber test was negative. Audiometry shows a very slight loss of hearing in the left ear as compared with the right, but the bone-conduction readings are virtually normal for this instrument. With cold caloric tests with water at 60° F. (15.6° C.) on the right, vertigo and nystagmus began after 45 seconds and cleared in 110 seconds. On the left it began at 30 seconds and ceased at 135 seconds. These figures show slightly increased irritability of the left labyrinth, but are really almost normal within the limits of the test."

Discussion

These six cases present as cases of acute labyrinthitis of uncertain aetiology. It is interesting that five of them occurred between November 10, 1951, and January 15, 1952. Another case in the same district was seen by a colleague of mine in January, 1952. In all cases the onset was extremely sudden, the patient feeling perfectly well one minute and having vertigo a moment later. There was

no previous malaise or pyrexia, I was unable to find any aural pathology, and there was no complaint of deafness. The average age of the patients was 54 years, and they all had been, and continue to be, in reasonable health. No treatment was required except rest in a dark room and small doses of phenobarbitone.

There is a great risk in trying to draw a conclusion from a small number of cases followed up for such a short time. I believe, however, that this may prove to be another manifestation of virus disease, the view being suggested by the history of contact between two next-door neighbours who developed similar symptoms within three days. I have not been able to establish any means of contact between the other cases. I suggest, however, that, like anterior poliomyelitis, the disease may be spread by carriers, that sub-clinical attacks occur, and that mild forms of the disease may pass unrecognized.

Summary

Six cases of acute labyrinthitis seen in general practice within one year are recorded; it is suggested that the disease is spread by a virus.

I wish to thank Mr. A. D. Bateman for his report on Case 6.

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A CASE OF PHLEGMASIA CERULEA DOLENS

BY

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The so-called "blue phlebitis," or phlegmasia cerulea dolens, was first described by Trémolières and Véran (1929), and De Bakey and Ochsner (1949) state that since then a further 32 cases have been recorded, mostly in the French literature. Because of the importance of early recognition and treatment of those cases associated with peripheral circulatory collapse, and because it is possibly not widely known that many cases are in fact associated with peripheral circulatory collapse, the following case is put on record.

Case History

A woman aged 67 was admitted to hospital on the evening of December 13, 1951. She had felt quite well until early that morning, when her left leg became swollen; it progressively increased in size, and ached a little in the calves and later in the posterior aspect of the thigh. She remained in bed for most of the day, and at about 4 p.m., some eight hours after the onset, she suddenly collapsed with severe pain in the left groin and hypogastric region associated with vomiting. The pain was intermittent and colicky in type, and she vomited four times. The bowels had been regular until the day previously, but had not been opened for 24 hours. There was slight frequency of micturition, and the patient thought she had been losing weight recently. The past history revealed nothing significant.

On examination the patient was seen to be well-nourished. She was in severe pain and was obviously shocked. Her face was pale and there were beads of cold sweat on her forehead. The pulse was 116 and of poor volume; blood