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Vestibular paresis: a clinical feature of Wernicke's disease

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Wernicke's disease is a well-defined clinical and pathological entity, and its aetiology has been conclusively traced to thiamine deficiency (Phillips, Victor, Adams, and Davidson, 1952). Its essential features are the subacute onset of confusion, ophthalmoplegia and/or nystagmus, and ataxia of stance and gait. It invariably occurs against a background of malnutrition and often chronic alcoholism. Ophthalmoplegia, ataxia, and the global confusion respond readily to thiamine administration, but the patient is often left with a conspicuous disorder in memory (Korsakoff's psychosis), and not infrequently some degree of ataxia when attempting to walk on a narrow base (heel to toe).

In the acute stages of the disease there is frequently a striking discrepancy between the patient's ability to stand and walk and his ability to perform tests designed to assess cerebellar function—that is, heel-to-shin, finger-to-nose, etc. The patient may literally be unable to take even a few steps without support; his legs are rigidly held wide apart, the body pitched forward as he clutches the nearest solid object for fear of falling. In a study of 245 patients with Wernicke's disease by Victor, Adams, Collins, and Silby (to be published), it was found that ataxia of stance and gait occurs in 87%, whereas ataxia of the lower extremities in only 20%.

One interpretation of this finding attributes the ataxia of stance and gait to lesions in the midline cerebellar vermis, and the ataxia of the lower extremities to an extension of the lesion into more lateral areas of the anterior lobes (Victor, Adams, and Mancall, 1959). Vestibular function, which is known to be of paramount importance in the regulation of postural adjustments, has not heretofore been determined. This study was undertaken to evaluate vestibular function in the acute and chronic stages of Wernicke's disease.

MATERIAL AND METHODS

The subjects were 17 chronically malnourished patients admitted to Cleveland Metropolitan General Hospital with unequivocal signs of Wernicke's disease. All showed an initial global confusional state and variable degrees of memory loss as a persistent finding. All patients had nystagmus and severe ataxia of gait as well as signs of mild polyneuropathy. Ophthalmoplegia was seen in 12 patients (bilateral abducens palsies in 11 and total external ophthalmoplegia in one) and lasted from one to seven days. Mild cerebellar signs of the lower extremities could be elicited in nine and of the upper extremities in two. Vertigo was not a complaint in any. Thiamine hydrochloride (100 mg) was administered intramuscularly upon admission to all patients and orally for several days thereafter. All patients were examined neurologically in a serial fashion. Head injuries were not a factor in any of the patients and routine skull films were normal. All patients had been hospitalized throughout the course of this study during which time they received an adequate diet and no alcohol.

Control subjects were 12 chronic alcoholic patients, seven with delirium tremens and five with alcoholic cerebellar degeneration (Victor *et al.*, 1959). The latter group had no impairment in mental function or ophthalmoplegia. Their neurological deficit consisted of mild ataxia of stance and gait with an associated symmetrical ataxia of legs and to a lesser extent of the arms. The onset of the disturbance had been gradual and no discernible episode of Wernicke's disease had occurred. No significant change in the severity of their ataxia was noted during the course of their hospital stay.

Vestibular function was assessed by means of a standard ice water caloric test. The ears were irrigated for 30 seconds with 5 ml. ice water with at least a five minute interval between the testing of each ear. The patient was instructed to fix his gaze on a point straight ahead and the time elapsing between the time of injection and end of the ensuing nystagmus was determined. Past pointing with eyes closed before and after stimulation was evaluated.

Tests were performed on more than one occasion in 14 patients. Four patients were tested only twice; the other 10 were tested on multiple occasions, on alternate days at the beginning and at weekly and monthly intervals later in the course of their illness.

Five patients were tested for directional preponderance by means of a modification of the method of Fitzgerald and Hallpike (1942) in which the ears were alternately irrigated with 75 ml. water at 30 and 44°C.

With one exception, none of our patients had any hearing deficit by history or on routine clinical testing