

See-saw nystagmus

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See-saw nystagmus is a rare ocular abnormality, only 15 cases having been recorded to date (Maddox, 1914; Rucker, 1946; Larsen, 1957; Jensen, 1959; Smith and Mark, 1959; Mark, Smith, and Kjellberg, 1960; Lourie, 1963; Schurr, 1963; Kinder and Howard, 1963; Slatt and Nykiel, 1964; and Daroff, 1965). As initially described by Maddox (1914), the eye movements consist of regular reciprocating oscillations in which one eye rises while the other falls. Torsion of the eyes accompanies the vertical movements so that the axis of the rising eye intorts while that of the falling eye extorts.

Nine of the reported cases of see-saw nystagmus have been associated with tumours in the region of the optic chiasm and diencephalon. These lesions do not entirely explain the pathogenesis of the see-saw movement, however, since in other cases brain-stem or ocular lesions were described instead. Furthermore, in innumerable parasellar lesions see-saw nystagmus does not occur. Much still remains to explain the pathophysiology of see-saw ocular movements.

The purpose of the present communication is to present an additional case in which see-saw nystagmus was observed in a patient with bitemporal hemianopia and abnormal vestibular responses. Attention is directed to several of the previously reported cases in which abnormal vestibular responses were also noted. A mechanism is suggested by which see-saw nystagmus is produced by various

combinations of visual, vestibular, or other lesions rather than by a lesion in a single anatomical site.

CASE REPORT

HISTORY A 65-year-old woman was well until 1957 when she noticed the insidious onset of visual impairment in both eyes. In 1963 she was admitted to another hospital where radiographs revealed enlargement of the sella turcica; examination of the visual fields demonstrated bitemporal hemianopia. Neurological examination was otherwise within normal limits and lumbar puncture revealed no abnormalities. An endocrine evaluation showed normal thyroid function but markedly diminished urinary excretion of 17-ketosteroids and 17-hydroxy-corticoids. After steroid replacement therapy was instituted, a right frontal craniotomy was performed and a chromophobe adenoma partly removed and the optic chiasm decompressed. The patient was given a course of radiation therapy to the pituitary region and placed on a regimen of cortisone acetate, thyroid extract, and diphenylhydantoin sodium.

Following surgery the patient's vision improved, but in 1964 further visual deterioration was noted (Fig. 1). In February 1965 the patient was admitted to Passavant Memorial Hospital because of complaints of increasing impairment of vision and 'pulling sensations' lateral to her eyes. In addition she complained of dizziness on turning rapidly. She was unaware of oscillopsia. The patient had gained 20 lb. in the year before admission and had discontinued her medications eight months previously.

On physical examination the blood pressure was

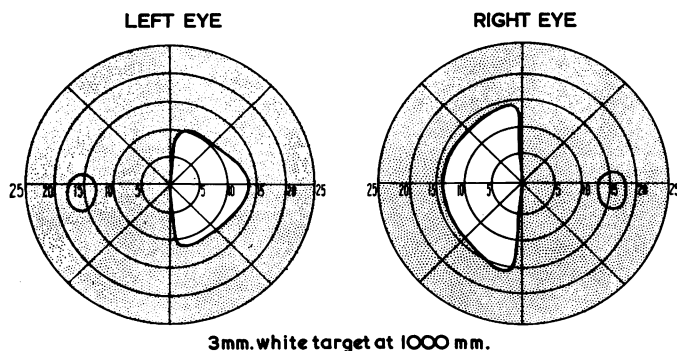


FIG. 1. *Central visual fields in 1964, determined with tangent screen using a 3 mm. white object at 1 metre.*

210/120 mm.Hg. The general physical examination was normal except for a moderate degree of obesity.

The patient showed a mild intellectual deficit although she was orientated in all spheres. She expressed paranoid ideas about dealings of foreign governments and involvements in 'the atomic business'. On confrontation, gross bitemporal visual field defects involving central vision were demonstrated. Visual acuity uncorrected was 20/80 in the right eye and 20/300 in the left. Both eyes could be corrected to 20/60-1. Tangent screen examination showed dense bitemporal hemianopsias with a 3 mm. white object and some general shrinking of the remaining visual field. Ophthalmoscopic examination showed moderate disk pallor bilaterally. The pupils were equal and responded to light and to accommodation.

Hearing was bilaterally diminished with air conduction better than bone conduction. The tongue deviated slightly to the right. The patient's gait was unsteady and she staggered to either side on tandem walking. The Romberg test was negative and cerebellar function was entirely normal. The remainder of the neurological examination, except for the ocular movements, was entirely within normal limits.

OCULAR MOVEMENTS When the patient stared straight ahead, particularly into a light sufficiently bright to prevent her focusing, intermittent bursts of three or four see-saw movements were observed. As the left eye intorted and rose, the right eye extorted and fell and vice versa. See-saw movements occurred spontaneously but were intermittent. In addition, they could be readily elicited by tilting the patient's head slightly to either side, and they occurred regularly after she blinked or moved her eyes slightly in any direction. There was no consistent change in amplitude of see-saw nystagmus on upward or downward gaze. At no time were the see-saw movements continuous. In addition, on right lateral gaze transient horizontal nystagmoid jerks were noted; none occurred on gaze to the left.

Caloric stimulation according to the method of Fitzgerald and Hallpike (1942) was carried out, irrigating each ear with water at temperatures of 30° and 44°C. An imbalance between the two vestibular systems was

noted, with responses on the right significantly greater than on the left and a directional preponderance to the right (Fig. 2).

LABORATORY DATA The haemogram and sedimentation rate were normal. Except for a few leucocytes per high-power field the urine analysis was normal. Serological tests for syphilis were negative. A chest radiograph was normal. An electrocardiogram showed evidence of an old posterior myocardial infarction. Fasting blood sugar, blood urea nitrogen, calcium, phosphorus, alkaline phosphatase, serum sodium, chloride and carbon dioxide contents of the blood were all normal. The serum potassium level was 3.3 mEq./l. Protein-bound iodine was 3.5 µg.%. Tri-iodo-thyronine sponge-uptake was 28% (normal 25-35%), and radioactive iodine uptake was 5% at 24 hours. After thyroid-stimulating hormone radioactive iodine uptake was 9%. 17-Ketosteroids and 17-ketogenic steroid excretions were both 1.7 mg./24 hours; following A.C.T.H. stimulation they both rose to normal levels. Urinary gonadotrophins were too diminished to be detected by the mouse-uterine weight method. A glucose tolerance test was normal.

An electroencephalogram showed a severe slow wave abnormality in the right frontal area probably projected from deep subcortical regions. Skull radiographs showed a markedly enlarged sella measuring 2.2 × 1.5 cm. on lateral projection. There was evidence of an old osteoplastic craniotomy in the right frontal region. A pneumoencephalogram (Fig. 3) demonstrated the presence of a mass arising from the sella and protruding into the floor of the third ventricle, obliterating the chiasmatic cistern. In addition there was mild generalized dilatation in the ventricular system.

COURSE IN HOSPITAL Following the diagnostic studies the patient was given thyroid extract and hydrocortisone replacement; during this time her vision deteriorated further until she could barely see hand movements with the right eye. Three weeks after beginning replacement therapy, the patient underwent a right frontal craniotomy for further removal of the recurrent pituitary tumour. At operation a tumour mass was seen extending beyond the

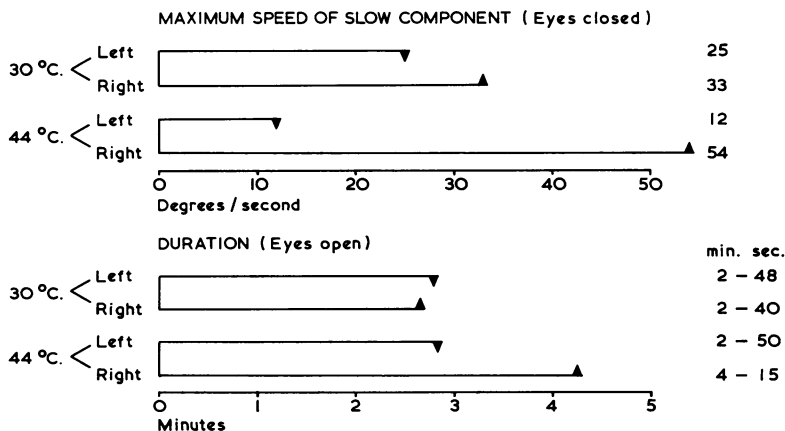


FIG. 2. Record of caloric vestibular studies indicating significantly greater responses on the right and a directional preponderance to the right.

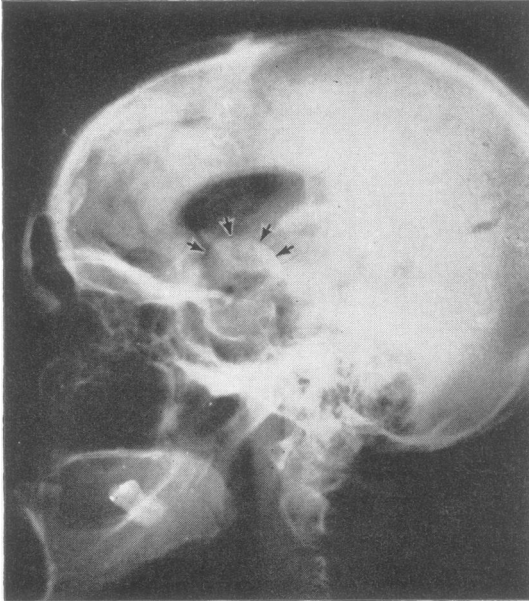


FIG. 3. *Pneumoencephalogram showing a large tumour mass arising from an expanded sella and protruding into the floor of the third ventricle.*

diaphragma sellae, flattening the right optic nerve and pushing the chiasm posteriorly into the floor of the third ventricle. The entire tumour mass was removed and the sella was curetted. Histologically the specimen was identified as a pituitary chromophobe adenoma.

Post-operatively the patient failed to regain consciousness. She became febrile and on the third post-operative day assumed a decerebrate posture upon noxious stimulation. The patient expired on the seventh post-operative day. A post-mortem examination was not obtained.

DISCUSSION

Although only 15 previous cases of see-saw nystagmus have been reported (Table I), three hypotheses have been advanced to explain the curious ocular movements. See-saw nystagmus has been variously attributed to an ocular or visual abnormality; a brain-stem lesion; and a diencephalic lesion.

OCULAR OR VISUAL ABNORMALITIES Lourie (1963), describing a patient with a chromophobe adenoma, noted that covering of one eye suppressed the nystagmus. He concluded that '... a disturbance in the normal tonic influence from the macula on attempted fixation (was) the mechanism of the nystagmus'. The patient of Slatt and Nykiel (1964), suffering from visual loss and see-saw nystagmus following an undiagnosed febrile illness, also showed variation of the nystagmus with alteration of the environ-

mental illumination or of fixation. These authors similarly concluded that see-saw nystagmus was '... ocular rather than central in origin'. Finally, Duke-Elder (1949) has noted that see-saw nystagmus may occasionally occur in miners, where it is presumably of visual origin.

BRAIN-STEM LESIONS Case 1 of Jensen (1959) and case 2 of Daroff (1965) were apparently due to brain-stem vascular lesions. In Jensen's patient cranial nerve findings indicated an incomplete unilateral lesion extending from the medulla to the midbrain. Daroff's case showed evidence only of mesencephalic abnormalities. In neither patient was any visual impairment noted, although visual fields were considered unreliable in the former case.

DIENCEPHALIC LESIONS Because parasellar tumours have occurred in nine of the reported cases of see-saw nystagmus, some authors have considered that the abnormal eye movements are due to damage to diencephalic structures. Although there is no clinical evidence that diencephalic lesions without chiasmatic, *i.e.*, visual, involvement can produce see-saw nystagmus, Daroff (1965) has summarized considerable experimental evidence linking this region to control of vertical and torsional eye movements.

Each of these hypotheses fits some of the cases of see-saw nystagmus but none provides a sufficiently comprehensive mechanism to explain them all. The variation in clinical features documented in Table I defies any simple explanation which would localize a single area of brain as the 'centre' for see-saw nystagmus. An adequate hypothesis should be able to reconcile a number of seemingly inconsistent features that have been noted in the reported cases. The most important of these are:

INCONSTANCY OF VISUAL IMPAIRMENT Visual impairment while usual is not universally present. Bitemporal hemianopia was present in 11 of 13 cases where visual fields were reported; many of the patients with field defects also had severe impairment of visual acuity in one or both eyes. One of the two patients with normal fields had severely impaired visual acuity (Slatt and Nykiel, 1964).

TRANSIENCE OF SEE-SAW NYSTAGMUS IN PATIENTS WITHOUT VISUAL IMPAIRMENT In two cases where no impairment of visual fields or visual acuity could be determined (Jensen, 1959; Daroff, 1965) see-saw nystagmus was transitory. Both of these episodes were thought to follow acute brain-stem infarction.

FREQUENCY OF A PARASELLAR TUMOUR Chiasmatic-diencephalic tumours were found in nine of 13 cases

TABLE I

SUMMARY OF REPORTED CASES

<i>Author and Reference</i>	<i>Visual Defect</i>	<i>Parasellar Tumour</i>	<i>Other Presumed Aetiology</i>	<i>Duration of Nystagmus</i>	<i>Vestibular Abnormality</i>
Maddox (1914)	Bitemporal hemianopsia	?	—	—	—
Rucker (1946)	Bilateral field defect Acuity: OD-2/60 OS-3/60	0	? Toxoplasmosis	> one year	? (tinnitus)
Larsen (1957)	?	+ 'Oligodendrocytoma chiasmatis'	0	Lasting	—
Jensen (1959)					
Case 1	Acuity: 6/18 O.U.; Fields not determined	0	Basilar-vertebral thrombosis	Two days	+ Diminished caloric response, left
Case 2	Acuity: OD-6/18 OS-6/9 fields ? normal (poor cooperation)	0	Alcoholism; left medial rectus paresis	One day?	—
Smith and Mark (1959)					
Case 1	Bitemporal hemianopsia Acuity: OD-20/200 } pre- OS-20/30 } operative OD-20/30 post-operative	+ Epidermoid cyst	0	Five months pre-operatively Ten days post-operatively	+ No response to ice water, Barany chair
Case 2	Improved fields Bitemporal hemianopsia	—	—	—	—
(Dr. F. Walsh) Mark, Smith, and Kjellberg (1960)					
Case 1			See case 1 above		
Case 2	Bitemporal hemianopsia	+ Glioma	0	—	—
Lourie (1963)	Bitemporal hemianopsia Acuity: OD < 3/200 OS-light perception	+ Pituitary adenoma	0	> one year	—
Schurr (1963)					
Case 1	Bitemporal hemianopsia Acuity: 'normal'	+ Craniopharyngioma	0	Years pre-operatively Stopped post-operatively	0 Calorics normal
Case 2	Bitemporal hemianopsia Acuity: OS-6/3 OD-hand movements	+ Chromophobe adenoma	0	Years; transiently relieved for 2-3 weeks post-operatively	—
Kinder and Howard (1963)	Bitemporal hemianopsia Acuity: OD-20/100 } pre- OS-finger-counting } operatively OD-20/40 } post- OS-10/200 } operatively	+ Craniopharyngioma	0	? Years pre-operatively One day post-operatively	—
Slatt and Nykiel (1964)	Fields normal Acuity: OD-20/100 OS-20/200	0	Childhood febrile illness	31 years	No response to ice water, calorics, Barany chair
Daroff (1965)					
Case 1	Bitemporal hemianopsia Acuity: OD-20/200 OS-20/50	+ Chromophobe adenoma	0	Months until death	—
Case 2	Fields normal Acuity: 20/20 O.U.	0	Midbrain infarction	Four weeks	—
Drachman (present report)	Bitemporal hemianopsia	+ Chromophobe adenoma	0	Months until death	Asymmetrical caloric responses

where adequate studies were performed to search for a mass lesion. No other tumours have been associated with see-saw nystagmus.

OCCURRENCE OF VESTIBULAR ABNORMALITIES Vestibular abnormalities were found in four of six patients in whom results of caloric (and in some cases rotational) tests were reported.

Thus ocular, chiasmatic, diencephalic, brain-stem, and vestibular lesions have been associated with one or more cases of see-saw nystagmus.

CONCLUSIONS

From a physiological standpoint it is not surprising that see-saw nystagmus has been associated with lesions in these areas, since vertical and torsional ocular movements have been experimentally produced by stimulation or ablation of closely corresponding structures:

Vertical eye movements may be readily produced by bilateral caloric stimulation of the labyrinths (Bender and Shanzer, 1964). Electrically, stimulation of the superior colliculi, the mesencephalic reticular formation, the region of the third ventricle, the head of the caudate nucleus or (with bilateral stimulation) the frontal eye movement area (Brodmann's area 8) may produce vertical eye movements (Cogan, 1956; Szekely and Spiegel, 1963; Bender and Shanzer, 1964; Pasik and Pasik, 1964). Disconjugate vertical movements (one eye rising, the other falling) have been produced by unilateral stimulation lateral to the superior vestibular nucleus (Montandon and Monnier, 1964). Facilitation of nystagmus produced by stimulation in the region of the vestibular nuclei follows destruction of the contralateral 'diencephalic nystagmogenic area' medial to the lateral geniculate body (Montandon and Monnier, 1964).

Torsional eye movements are thought to originate normally in the saccule and utricle (Kompanejetz, 1928; Miller, 1962) and may be produced by tilting of the head. Electrical stimulation of the zona incerta and of the interstitial nucleus of Cajal have also produced ocular torsion in experimental animals (Hyde and Toczek, 1962; Toczek and Hyde, 1963).

Visual control of vertical eye movements takes place via connexions of the occipital lobes with the frontal eye fields (Cogan, 1956). Although no cortical representation of torsional eye movements has been demonstrated, the fact that rotation of the visual environment may produce ocular torsion indicates a probable cortical mechanism for this eye movement as well. Other experimental studies indicate that visual fixation is capable of strongly inhibiting the eye movements induced by electrical stimulation of nystagmogenic areas (Montandon and Monnier, 1964).

This brief summary indicates that numerous areas of the nervous system are concerned with vertical and torsional ocular movements. Yet experimental stimulation or ablation of a single area has not as yet fully reproduced the picture of see-saw nystagmus, combining its torsional and vertical components in a pendular rhythm. It seems likely that the production of see-saw nystagmus may require more than one lesion; this conclusion is in keeping with Bender and Shanzer's statement (1964) that '. . . integration of nervous system activity that results in eye movements . . . occurs at many levels. . . . There may be a system of oculomotor activity but not an anatomic centre subserving such function'.

In the present case visual and vestibular abnormalities occurred in a single patient. Other combined disorders may perhaps be produced by a single tumour mass if it is sufficiently large; a parasellar mass, for example, may compress the chiasma and in addition damage ascending vestibular pathways in the diencephalic region (Spiegel, Szekely, and Gildenberg, 1965).

This concept that combined lesions may be necessary for the production of see-saw nystagmus explains its relative rarity as well as the diversity of lesions that have been held responsible. The importance of specifically visual impairment as a component may be that it permits latent see-saw ocular movements to become manifest when visual inhibition is removed. Similarly, the transitory nature of see-saw nystagmus produced by brain-stem lesions alone may be due to the fact that visual inhibition of ocular movements supervenes after a brief interval.

SUMMARY

A case of see-saw nystagmus is presented in which a vestibular abnormality occurred in combination with bitemporal hemianopia. The neurophysiology of vertical and torsional ocular movements is briefly reviewed. The suggestion is made that see-saw nystagmus may be due to several possible combinations of lesions involving visual, vestibular, or other structures, rather than to a single lesion.

I am grateful to Dr. Cecil Hart who performed the vestibular function studies.

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