Short report

Gaze-induced laughter

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SUMMARY Pathological laughter was stimulated by pursuit eye movements with a large extramedullary brainstem tumour. Laughter was also evoked by intense direct light. The mechanism by which visual stimuli could induce pathological laughter is discussed.

Inappropriate laughter is an unusual manifestation of central nervous system disease. Such laughter is usually an ictal phenomenon ('gelastic seizure') or a fragment of a pseudobulbar syndrome (Poeck, 1969). Laughter is pathological if it occurs spontaneously, in disproportion to an appropriate stimulus or in response to an inappropriate stimulus (Poeck, 1969; Loiseau *et al.*, 1971; Stearns, 1972). Only Walsh and Hoyt (1969) have described a patient with lateral gaze-induced laughter. A second patient in whom laughter was gaze-induced is reported.

Case report

A 26 year old right handed male was admitted to hospital because of persistent horizontal diplopia, right supraorbital paraesthesias, and right frontal headaches. Between the ages of 10 and 21 years the patient suffered six episodes of loss of consciousness preceded by an emotional distress, abdominal discomfort, shortness of breath, and diaphoresis. Each attack lasted for several minutes without tonic-clonic movements, although urinary incontinence occurred once. No diagnostic testing was initiated and no treatment provided. Diplopia developed three years before admission. It receded spontaneously only to recur two years later. Inappropriate laughter was not recognised by the patient until an ophthalmological examination several days before admission.

The general physical examination was normal. Mentation was normal. Funduscopy, visual acuity and visual fields were normal. The right pupil measured 4 mm and reacted sluggishly to light and accommodation; the left pupil was 2 mm and reacted fully. Movements of the left eye were full while all movements of the right eye were restricted, abduction most severely. Ptosis of the

right lid was observed but without worsening on upgaze. Pursuit and convergent eye movements were usually associated with uncontrolled giggling, most prominent on right lateral gaze. Mirth preceded the act of laughter, and although both were recognised as inappropriate responses, neither could be suppressed by the patient. Attempts to stimulate laughter by voluntary lateral gaze or an optokinetic drum were unsuccessful. Inappropriate laughter could be induced occasionally by intense illumination such as during funduscopy and intermittent photic stimulation during routine electroencephalography. Except for diminished sensation over the ophthalmic division of the trigeminal nerve, the remainder of the neurological examination was normal.

The following endocrine tests were normal: thyroid stimulating hormone, follicular stimulating hormone, serum cortisol, 17-keto- and hydroxyketosteroids, and growth hormone and prolactin responses to levodopa (500 mg) loading.

An audiogram and tympanometry were normal. Routine electroencephalography demonstrated right occipitoparietotemporal 3–5 Hz activity without paroxysmal activity. Homogeneous, fine calcification (40×40 mm) was seen on a skull radiograph in the middle cranial fossa with the floor of the sella turcica slipping sharply and inferiorly to the right without erosion of the clivus. Right carotid and vertebral arteriograms showed a large avascular mass in the right posterior and middle cranial fossas with marked forward displacement of the right internal carotid artery, elevation of the right posterior cerebral and superior cerebellar arteries and medial displacement of the basilar artery to the left. A routine percutaneous brain scan was normal although computerised transaxial tomography outlined a tumour (Figure).

A right temporal craniotomy was performed with subtotal removal of an osteochondroma which displaced the midbrain and pons. Post-

Figure Computerised tomography showing a large calcified tumour.

operatively, the gaze-induced laughter disappeared and has not returned one year later, despite residual right lateral rectus weakness and right lid ptosis.

Discussion

Normal laughter is a complex motor response to a specific and appropriate emotion (Poeck, 1969; Loiseau *et al.*, 1971). The intensity of this facio-respiratory response is influenced by situational factors as well as by the strength and duration of the emotional stimulus. Inappropriate or pathological laughter occurs when the response is not proportional to the emotional stimulus (for example, pseudobulbar syndromes) or when no emotional stimulus is available (for example, hebephrenic schizophrenia). Laughter in the present case was pathological as it did not occur as a response to an emotional stimulus but primarily in response to lateral gaze or convergent eye movements. A feeling of mirth experienced by the patient was not induced by any external emotional stimulus and could not be inhibited.

The 'anatomy of laughter' is incompletely

understood but the hypothalamus has been represented as a possible 'centre' as it receives descending cortical and ascending brain stem input (Martin, 1950). Third ventricular tumours and mechanical stimulation of the hypothalamus during surgery are well established causes of pathological laughter (Martin, 1950; List *et al.*, 1958; Foerster and Gagel, 1934). The present tumour extended into the parasellar region but failed to produce endocrine abnormalities. Other subcortical structures including the basal ganglia and thalamus (Poeck and Pilleri, 1963) may influence the occurrence of inappropriate humour.

The frontal and temporal lobes provide the hypothalamus with appropriate emotional stimulus for the motor act of laughter. Abnormal cortical discharges from these regions may stimulate pathological laughter (the gelastic seizure) (Loiseau *et al.*, 1971; Ames and Enderstein, 1975). This unusual form of epilepsy may arise from within the hypothalamus, but the affective component of the cortical-induced seizure is usually absent. In the present case, laughter did not appear to be of epileptic origin as the mirth experienced occurred without EEG or clinical seizure activity.

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Wilson (1924) postulated a supranuclear pontomedullary 'centre' controlling the facio-respiratory synkinesis necessary for laughter. Interruption of corticobulbar pathways, as in amyotrophic lateral sclerosis or lacunar states, impairs the descending cortical inhibitory control of this 'centre' thereby allowing laughter to become manifest. While there was obvious distortion of the brain stem by the large tumour in this case, no other evidence of a pseudobulbar syndrome was present. Laughter occurred primarily with gaze-associated pursuit and convergent eye movements. Voluntary saccadic eye movement and optokinetic-induced movements were insufficient stimuli. Bright light also evoked laughter, suggesting that various visual stimuli were necessary and not eye movement alone. Occipital corticofugal fibres arising from areas 18 and 19 of the cerebral hemisphere synapse in the collicular area (corticotectal tract) or in the lower brain stem (corticotegmental tract) (Kahn *et al.*, 1955). These tracts may have been compromised by the tumour.

Visual stimuli may activate structures exclusive of efferent occipital lobe pathways that modify hypothalamic activity. In animals, fibres of an opticohypothalamic system enter the lateral hypothalamic area where connections with lateral hypothalamic nuclei and the median forebrain bundle occur (Moore *et al.*, 1968; Scharrer, 1964). Visual stimuli could initiate both the laughter and its affective components by modifying activity of this extensive polysynaptic system. However, the presence of an opticohypothalamic tract in man is still in question. Intermittent photic stimulation alters some hypothalamic endocrine functions in lower mammals (Shimada *et al.*, 1973; Feldman *et al.*, 1971) but not cortisol, growth hormone, or prolactin secretion in humans (personal unpublished data).

The extent of the osteochondroma does not permit accurate localisation of the area responsible for inappropriate affect. A comparison with the case briefly described by Walsh and Hoyt (1969) suggests that brainstem dysfunction is, in part, a prerequisite. Both patients were young males with large extramedullary tumours distorting the brainstem, multiple cranial neuropathies, and altered extraoculomotor functions. In each case, laughter was gaze-induced with no associated electrical or clinical seizure activity. Achari and Colover (1976), recently reported pathological laughter in two patients with brainstem tumours. The absence of seizures and endocrine dysfunction and the cessation of pathological laughter after surgical decompression suggest that the distorted

brainstem was influential in the appearance of pathological laughter in the present case.

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