Short report

Psychophysical assessment of a patient with Tolosa-Hunt syndrome

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SUMMARY Patients may complain of impaired vision and yet exhibit no abnormalities when tested with conventional tests of visual acuity. Some sensitive psychophysical tests used by psychologists and neurophysiologists to examine the function of the normal visual system are beginning to gain recognition as useful tools in clinical situations. They are particularly sensitive indicators of damage to the visual pathways. The results are presented of one such application which characterises the visual defects of a patient with a suspected diagnosis of Tolosa-Hunt Syndrome.

Clinical History

In 1979 the patient, aged 51 years, presented with a history of 2 episodes of pain in and around the left orbit and upper gum. Diagnosis was thought to be an unusual presentation of migrainous neuralgia. By January 1980 there had been two further episodes of painful ophthalmoplegia with diplopia. This pain was predominantly peri-orbital and there was 3rd and 6th cranial nerve paresis and ptosis on the left side. His visual fields were full and Snellen acuity was normal. A carotid angiogram showed narrowing of the entire intracavernous section of the left carotid artery, and elevation of the syphon. The CT scan (fig 1) shows a mass lesion in the left orbital apex surrounding the optic nerve, and involvement of the superior orbital fissure. Full blood count, ESR, urea, electrolytes, protein electrophoresis were all normal. Prednisone treatment resolved the headache and improved the ocular pareses.

A diagnosis of Tolosa-Hunt syndrome¹ was confirmed at the Moorfields Eye Hospital. By May 1981 there was no diplopia, although the patient still complained of transient lack of clarity in the left eye. Central field analysis now showed a left para-central scotoma despite a visual acuity of 6/5. Steroid treatment had been stopped and the CT scan was now normal. Visual evoked responses to checkerboard pattern reversal showed a delay across the left visual pathway with a major P2 potential at 124 ms. The right side had a normal latency at 109ms. The patient continued

Fig 1 CT scan showing high attenuation area in orbital apex taken 18 February 1980.

to complain of poor vision in the left eye when psychophysical measurements were made in June 1981.

Measurement of contrast sensitivity.

The limits of normal spatial vision can be characterised by measuring the contrast sensitivity to sinusoidal gratings of different spatial frequency. Periodic exchange of the dark and light bars produces a flickering grating. Different flicker-rates or temporal frequencies produce specific changes in the shape of the spatial contrast sensitivity curve as can be seen in

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fig 2(a). The fall-off in sensitivity at low spatial frequencies has been shown to be most marked at low temporal frequencies;² the high frequency fall-off is quite constant in form for all temporal frequencies. Contrast sensitivity at low spatial frequencies improves with high temporal frequency and the curve shows a monotonic decline in sensitivity with increasing spatial frequency.

Patients with known and sub-clinical visual pathology often show losses of contrast sensitivity³ which can be restricted to a narrow range of spatial frequencies. Glaucoma for example commonly depresses sensitivity to low spatial frequencies.⁴ Some patients with multiple sclerosis lose sensitivity to medium spatial frequencies⁵ and high spatial frequency losses of sensitivity can be seen with multiple sclerosis⁶ and in macular disease.⁷ Of particular importance to this paper is the finding that an abnormal contrast sensitivity function can be found in patients who nevertheless have a quite normal Snellen acuity.

Method

Vertical sinusoidal grating patterns were generated by computer on a television display screen using similar techniques to those described by Schade.⁸ The contrast of gratings could be varied continuously without changing the space-averaged luminance, which was fixed at 200 cd m⁻² (photopic). Contrast sensitivity was measured using a self-paced yes/no staircase procedure. The patient was seated in a dental chair at a viewing distance of 4 metres and measurements were made monocularly. The non-test eye was covered with a translucent occluder to prevent dark adaptation of that eye. He attended three separate sessions at weekly intervals so that contrast sensitivity could be tested at temporal frequencies of 1, 5 and 15 Hz.

Results

The contrast sensitivity functions for each temporal frequency can be seen in fig 2 (b), (c), (d). At a temporal frequency of 1 Hz (fig 2 (b)), a clear loss of sensitivity is apparent at low to medium spatial frequencies in the patient's left eye when compared with sensitivity of his right eye which falls within the normal range. This deficit increases further at 5 Hz (fig 2 (c)). At a high temporal frequency of 15 Hz (fig 2 (d)), the enhancement of sensitivity at low spatial frequencies so obvious in the unaffected eye and control eyes, is not found in the affected eye. At high spatial frequencies his contrast sensitivity is virtually unimpaired relative to normals and his cut-off spatial frequency is normal in both eyes. This explains why his visual acuity, as measured by Snellen charts remains normal (6/6).

Fig 2 (a) Spatial contrast sensitivity functions for 24 naive subjects at three different temporal frequencies. The unfilled circles show the curve at 1 Hz, filled circles at 5 Hz and the unfilled squares at 15 Hz. (b), (c), (d). Spatial contrast sensitivity curves for normals with error bars (hatched area), patient's right eye (filled circles) and patient's left eye (unfilled circles). Graphs (b), (c), and (d) show the contrast sensitivity at temporal frequencies of 1, 5 and 15 Hz respectively.

Conclusions and discussion

The selective low spatial frequency deficit in this patient, suggests two main points. Firstly a mechanism must exist that is responsible for detection of these particular stimuli, and secondly this mechanism appears to be susceptible to damage in the Tolosa-Hunt Syndrome.

Recent electrophysiological work on non-human primates has shown that there are physiologically distinct classes of neurons within the visual pathway. There is for example an apparent anatomical separation of different classes of cells in the parvocellular and magnocellular layers of the lateral geniculate nucleus.⁹⁻¹¹ Parvocellular cells have smaller receptive fields¹² and thus have superior spatial resolution to the magnocellular cells.^{9,10} They also have slower conducting axons^{9,13} and lower contrast sensitivity.^{10,11} Our patient's visual deficit is highly suggestive of damage to the large, fast conducting fibres which project from the retinal ganglion cells to the magnocellular layers of the lateral geniculate nucleus. This conclusion is reinforced by the finding that increasing temporal frequency did not improve sensitivity in the affected eye. It is also consistent with the VER measurement which showed a latency delay in the left eye.

Patients with glaucoma and normal acuity reveal losses similar to those of this patient.¹⁴ A plausible suggestion for the similarity of deficit in both problems is that these conditions produce a compressive lesion, intra-ocularly in the case of glaucoma and extra-ocularly in Tolosa-Hunt Syndrome. The neurons with large, fast conducting axons would presumably be most susceptible to this sort of damage. Contrast sensitivity measurements substantiate the patient's complaint that vision is poor in one eye. It also provides us with a non-invasive method of establishing the possible underlying neuropathology of different disease processes such as the Tolosa-Hunt Syndrome.

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