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Letters

A blind spot tester

Sir,—Enlargement of the blind spot is a sensitive indicator of papilloedema.[1-5] Unfortunately, the size of the blind spot determined by perimetry is influenced by the nature of the test object and the subject's visual acuity so that an enlarged blind spot on perimetry may be difficult to interpret.[6] We describe a simple bedside method for determining blind spot size which can be used as a non-invasive index of intracranial pressure. The blind spot tester consists of a series of fixation points and laterally placed test objects which replicate the blind spot. The subject, with one eye covered, is instructed to look at the fixation point corresponding to the smallest test object and then to move the blind spot tester to and fro until the test object is totally hidden by his blind spot. The procedure is repeated with successively larger test objects until the test objects become too large to be totally obscured by the blind spot. The largest test object which can be hidden in the patient's blind spot serves as a measure of the blind spot size.

The tester's construction is based upon prior determinations of the shape and location of the blind spot. The blind spot is a vertical ellipse with a height/width ratio of approximately 1·4: 1·0.[6] It is located $15{\cdot}5° \pm 1°$ laterally from the fixation point.[3 6] The blind spot is centred with 64% of the long axis below the horizontal meridian.[2 3 6] The separation between a focal point and test object on the blind spot tester is the product of the distance that the tester is held from the subject and the tangent of angular separation between the fixation point and the blind spot. We chose a working distance of 30 cm between the patient and tester. Consequently, the separation between the fixation points and test objects was 8·3 cm. Because of variation in the angular separation between the fixation point and the blind spot, the actual distance between the patient and the blind spot tester at which the test object projected on the retinal blind spot was 30 ± 1 cm.

Thirty-five patients (70 eyes) with normal blind spot size on tangent screen perimetry were tested to determine the normal range. The mean solid angle subtended by the blind spot was $0{\cdot}0052 \pm 0{\cdot}0012$ (SD) steradians, and the blind spot height was $5{\cdot}4° \pm 7°$ (SD). The test usually took less than five minutes to perform. The blind spot sizes were rechecked at a later date in 20 patients. Initially the mean solid angle subtended was $0{\cdot}00547 \pm 0{\cdot}00077$ steradians; on rechecking it was $0{\cdot}00547 \pm 0{\cdot}00079$ steradians. Blind spot size determined by the BST was relatively independent of environmental illumination or the subject's visual acuity so long as the subject could clearly see the fixation point and test objects of the BST. A second group, of 46 patients with papilloedema, were evaluated with the BST. Their blind spot sizes were all larger than 0·013 steradians. For each patient the blind spot of each eye was enlarged to a similar degree. Three patients had a pseudotumour cerebri treated with serial lumbar punctures. There was a good correlation between the lumbar CSF pressure and the blind spot size just prior to the lumbar puncture (figure). The blind spot size did not decrease immediately after the lumbar puncture, but when the patients were retested 12–24 hours later the blind spot size had decreased. In six other patients with unilateral optic neuritis, the blind spot was enlarged for the involved eye, but was normal for the uninvolved eye.

Our preliminary results suggest that the blind spot tester is a quick and simple method for measuring the blind spot size of co-operative patients at the bedside or in the office. This test is similar to the method used by Mariotte and others to demonstrate the existence of the blind spot,[7] but this is the first report in which this technique was used to measure blind spot size. The advantages of the blind spot tester compared to perimetry in determining the blind spot size are that it can be used quickly at the bedside, and little examiner experience is required. For optimal results, the tester should be used under uniform conditions of good lighting and with the patient's visual acuity corrected. The blind spot tester is not accurate in conditions in which the blind spot is translated with respect to the point of fixation, such as severe hyperopia or myopia.[8] The presence of a normal sized blind spot may help to distinguish conditions which mimic the ophthalmoscopic appearance of papilloedema from true papilloedema.[1]

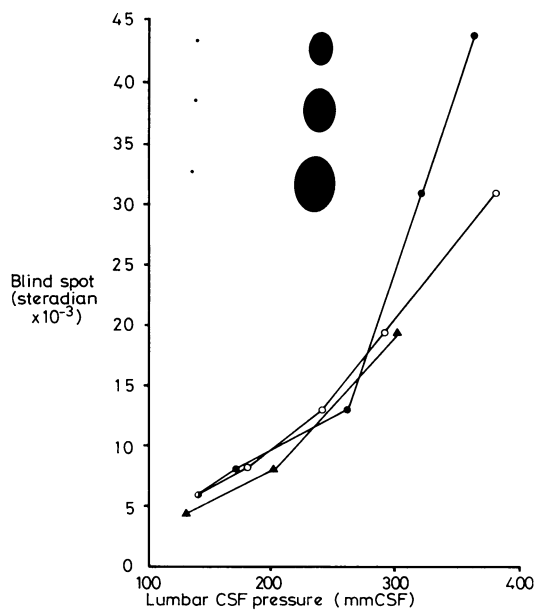

Figure *Correlation between lumbar CSF pressure and blind spot size in three patients with pseudotumour cerebri. The blind spot size was determined immediately before the lumbar puncture was done. The pressure values are opening pressures. Recordings were obtained at intervals of between 12–24 hr. Insert (upper left): Model of a right eye blind spot tester. The actual tester contained more test objects for increased sensitivity.*

The blind spot will be enlarged in patients with optic neuritis[3] and pseudopapilloedema.[9] However, if the eyes are unequally involved then the blind spot will be asymmetrically enlarged suggesting a process other than papilloedema.[2] As shown in this report and in previous studies[1 2 5] serial determinations of blind spot size can provide a useful index of treatment response in patients with increased intracranial pressure.

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Latent idiopathic torsion dystonia provoked by thyrotoxicosis

SIR,—Chorea occasionally occurs in thyrotoxicosis.[1-4] Usually it remits as the over-active thyroid is controlled, but occasionally it persists.[5] We now describe a patient in whom another type of dyskinesia, torsion dystonia, was provoked by thyrotoxicosis.

At the age of 17 years, a Caucasian woman with no Jewish ancestry, developed spasms of her neck, twisting her chin to the right, and intermittent shaking of the right arm. Unfortunately, this typical picture of segmental dystonia was mis-diagnosed as hysteria, and she spent the next year in a mental hospital, during which time the dystonia gradually disappeared. Between 1951 and 1978 she was only aware of rare mild torticollis when under extreme stress, but she did develop mild rheumatoid arthritis in 1965, for which she had been taking prednisolone 7·5 mg daily since 1973. In December, 1978, she again developed spasms of the neck and pronounced shaking of the right arm. In addition, she had lost weight and had experienced night sweats. On examination then she had spasmodic torticollis with the chin deviated to the right and hypertrophy of the left sternomastoid, intermittent repetitive dystonic spasms of the trunk, arching of her back into excessive lordosis and pulling her body to the right, and dystonic spasms of the right arm which was extended and hyper-pronated. In addition, there was exophthalmos, lid lag and a smooth uniform goitre. The clinical diagnosis was of idiopathic segmental torsion dystonia and thyrotoxicosis, the latter being confirmed biochemically (T4 = 244 nmol/L, T3 = 5·09 nmol/L, FTI = 382). Thyroid microsomal and thyroglobulin antibodies were present in high titre. The thyrotoxicosis was treated with carbimazole (10 mg thrice daily, reducing to 5 mg twice daily) but proved very difficult to control. Indeed, she remained thyrotoxic up till April, 1980, when we saw her for the first time (by courtesy of Dr A Hopkins). Then she had severe, exhausting dystonic spasms affecting the neck, trunk and right arm, which rendered her more or less chairbound. Clinical evidence of thyrotoxicosis was again confirmed biochemically (T4 = 172 nmol/L, T3 = 3·05 nmol/L). Eventually her thyroid over-activity was brought under control by a combination of radioactive iodine and carbimazole (10 mg four times a day), and the dystonic abnormal movements gradually settled. On two occasions, however, she stopped drug therapy, with the result that she rapidly became thyrotoxic again, and the dystonic movements became dramatically worse. Now that she is euthyroid, the dystonia is very mild, with a slight tendency for torticollis and scoliosis to the right, and for the right outstretched arm to hyper-pronate.

It has been suggested that chorea in thyrotoxicosis is due to hyperthyroidism causing increased sensitivity of striatal dopamine receptors to dopamine.[4 6] Admittedly, the chorea usually disappears as the thyrotoxicosis is controlled, suggesting a biochemical rather than a structural cause, but this hypothesis does not explain why it is such a rare occurence in hyperthyroidism. Such a mechanism may have been responsible for the activation of dystonia in our patient, although involvement of dopaminergic mechanisms in dystonia is much less obvious than in chorea. However, we believe that some other unknown mechanism is more likely to have been responsible in our patient, for her severe dystonia did not respond to treatment with dopamine antagonists (tetrabenazine 150 mg daily; haloperidol 12 mg daily) while she remained hyperthyroid.

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