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 1:4:10. 6 It is located 15.5° ± 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 0.0052 ± 0.0012 (SD) steradians, and the blind spot height was 5.4° ± 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 0.00547 ± 0.00077 steradians; on rechecking it was 0.00547 ± 0.00079 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, 2, 4

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.
Letters

The blind spot will be enlarged in patients with optic neuritis and pseu-
dopapillae~ema. However, if the eyes are unequally involved then the blind
spot will be asymmetrically enlarged suggesting a process other than papil-
loedema. As shown in this report and in previous studies serial determina-
tions of blind spot size can provide a useful index of treatment response in
patients with increased intracranial pressure.

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perimetry of the normal patients. The authors will provide a photocopy of a
sample blind spot tester along with each reprint. This work was partially
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Latent idiopathic torsion dystonia
provoked by thyrotoxicosis

SIR—Chorea occasionally occurs in
thyrotoxicosis.1,2 Usually it remits as
the over-active thyroid is controlled,
but occasionally it persists.3 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, de-
veloped spasms of her neck, twisting
her chin to the right, and intermittent
shaking of the right arm. Unfortu-
nately, this typical picture of segmental
dystonia was mis-diagnosed as hystera,
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 experi-
cenced night sweats. On examination
then she had spasmodic torticollis with
the chin deviated to the right and
hype~rophy of the left sternomastoid.
Intermittent repetitive dystonic spasms
of the trunk, arching of her back in'o
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 thyro-
globulin antibodies were present in high
title. 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, ex-
hausting 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). Eventu-
ally her thyroid over-activity was
brought under control by a combina-
tion of radioactive iodine and carbi-
zazole (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 be-
came thyrotoxic again, and the
dystonic movements became drama-
tically 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 dis-
ppears 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
occurrence in hyperthyroidism. Such a
mechanism may have been responsible
for the activation of dystonia in our
patient, although involvement of dopa-
minerigic mechanisms in dystonia is
much less obvious than in chorea.
Moreover, 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 hyper-
thyroid.

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