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

We thank Ms Kong, of the Dept of Ophthalmology, for performing the perimetry of the normal patients. The authors will provide a photocopy of a sample blind spot test along with each reprint. This work was partially supported by an NINCDS Teacher-investigator Award (NS 00458) to Dr Ruff.

References

Latent idiopathic torsion dystonia provoked by thyrotoxicosis

SIR,—Chorea occurring in thyrotoxicosis is usually remits as the over-active thyroid is controlled, but occasionally it persists. 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 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 ino 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 torsional dystonia and thyrotoxicosis, the latter being confirmed biochemically (T4 244 nmol/L, T3 5.09 nmol/L, TTI 382). Thyroid microsomal and thyroglobulin antibodies were present in high titre. The thyrotoxicosis was treated with carbimazole (10 mg thrice daily, reduced 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 chair-bound. 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. 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 occurrence 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.

References
Cimetidine induced postural and action tremor

SIR,—Cimetidine, an H₂ receptor antagonist used in the treatment of peptic ulceration has been reported to cause a number of central nervous system side effects.1 We have recently seen three patients who developed tremor on cimetidine which resolved when the drug was discontinued, but recurred on a rechallenge.

Case 1 A 68 year old male with chronic obstructive airways disease and polycystic kidneys developed a marked coarse postural and action tremor (8–10 Hz) of the upper limbs four days after commencing cimetidine (800 mg orally daily) for treatment of upper gastrointestinal bleeding. There were no associated focal neurological signs, and on stopping the drug the clinical features resolved over three days leaving a mild residual action tremor, which the patient admitted to having had for some years. The patient was rechallenged with intravenous cimetidine (200 mg) under controlled conditions, the tremor being recorded by transducer. Within 10 minutes the tremor became exaggerated, with superimposed myoclonic jerks. The tremor resolved within five minutes after propranolol 2 mg intravenously.

Case 2 A 72 year old female with known ischaemic heart disease was treated for 10 days with cimetidine (1 g orally daily) and metoclopramide (total dose 70 mg) for a pyloric ulcer. She developed a marked postural and action tremor (8–10 Hz) of the upper limbs, which on clinical examination was associated with increased tone in the arms. The abnormal signs resolved within 48 hours of stopping the cimetidine and metoclopramide. A controlled rechallenge with intravenous cimetidine (200 mg) 72 hours after stopping the drug resulted in recurrence of the tremor, which was abolished by propranolol (2 mg) intravenously. Transducer recordings were again obtained.

Case 3 A 46 year old female patient who was receiving cimetidine (1 g orally) for peptic ulcer formation developed severe tremor of all limbs. The tremor diminished when the cimetidine was discontinued by her general practitioner. A week later a clinical diagnosis by thyrotoxicosis was made (serum thyroxine 246 mmol/l) and the cimetidine was restarted. The tremor again worsened markedly, but was diminished by the addition of propranolol (40 mg twice daily) to her therapy.

An extrapyramidal syndrome has previously been reported in one patient who was receiving cimetidine,2 but he was also receiving metoclopramide and droperidol, drugs known to produce extrapyramidal effects. Two of the present cases are elderly patients in whom an underlying postural and action tremor was markedly aggravated by cimetidine. It appears that other side effects of cimetidine are more common in the elderly.3 In case 3 a thyrotoxic tremor was considerably intensified by cimetidine. The increase in tone initially noted in the second patient was probably due to metoclopramide.5 We have given intravenous cimetidine (200 mg) to healthy adults under controlled conditions and have not produced tremor.

The mechanism for the effect of cimetidine is obscure. On rechallenge, tremor was associated with myoclonic jerks in the first patient, but not in the other two. Myoclonic jerks have been noted in patients with cimetidine-induced confusional states.1 It is not clear whether the effect of cimetidine in producing tremor is due to a central or peripheral action. The confusional state caused by cimetidine has been attributed to H₂-receptor blockade in the CNS4 and the myoclonus also suggests a central effect. Propranolol is thought to act peripherally in reducing physiological tremor.4 However, the effect of propranolol on cimetidine-induced tremor does not necessarily indicate a peripheral origin. The abolition of the tremor by beta-adrenergic blocker suggests cimetidine has aggravated an underlying physiological or essential tremor.5 Excitation of physiological or essential tremor by cimetidine suggests that histaminergic pathways are normally involved in the suppression of tremor.

We would like to thank Dr PG Leggat, Dr RB Thomson and Professor J Grimley Evans for allowing us to study patients under their care, and Professor MD Rawlins for his advice and encouragement.

References


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Copies of the tremor recordings are available on request.