TOXIC AMBLYOPIA IN A PATIENT RECEIVING ETHCHLORVYNOL AS A HYPNOTIC*

BY

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ETHCHLORVYNOL is a non-barbiturate hypnotic which is now widely used in Great Britain and has been used extensively in the United States of America for the past 7 years. The drug has proved to be relatively free from side-effects and since no report could be found in the literature of toxic amblyopia resulting from its use, this case was considered to be worth recording.

Case Report

A man aged 56 years had been previously known to the unit as an in-patient in November, 1962, prior to laminectomy for prolapsed intervertebral discs at the level of L3-L4 and L4-L5. At that time, a full neurological examination revealed no abnormal features other than those associated with the above condition. In particular, his corrected vision was normal on testing with a Jaeger card. In the past the patient had not had any significant illnesses. He smoked from eight to ten cigarettes daily and had never at any time smoked a pipe. From the time of the patient’s original admission until he noted his visual disturbance, he did not drink any alcohol and previously his consumption was very moderate.

During the patient’s convalescence after the laminectomy, he developed metatarsalgia causing him to require an analgesic. Early in January, 1963, “Palfium” (dextromoramide) was prescribed, one tablet (5 mg.) daily until the end of February, when he ceased to require an analgesic. In January he also started to take “Serenesil” (ethchlorvynol), at first one capsule (500 mg.) and then two capsules (1,000 mg.) as a hypnotic each night. This he continued taking until the end of March 1962, when he noticed that his vision was abnormal. At that time he had had no drug other than ethchlorvynol during the preceding months. He first noted a change in colour appreciation, in that blue colours appeared to be more vivid, whereas green, red, and brown hues were dull. At the same time, he began to have some difficulty in reading typescript at work. It is of interest that the patient then stopped taking his hypnotic for a period of 5 days, thinking that there might be some causal relationship between the drug and the visual disturbance. However, his doctor assured him that no such toxic effect had been recorded and he therefore continued to take ethchlorvynol in the same dosage for a further period of time. The patient himself felt that, during the 5 days without the drug, his vision remained static, and that there was then further deterioration when it was recommenced.

The patient attended the Eye Out-patient Department on April 6, 1963.

Examination.—The vision was subnormal, the best corrected visual acuity being 6/18 Snellen and scotomatous reading of N 12 Standard Test Type. Ophthalmoscopy showed no abnormality of the optic discs or macular areas. Examination of the central field revealed a small fairly dense bilateral central scotoma.

In view of the history of drug-taking and the ocular findings, the patient was referred to the Neurological Out-patient Department, where he was seen on April 24, 1963, and then complained of further deterioration in reading vision.

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**Treatment.**—He was advised to take no further "Serenesil" and was admitted to hospital on April 29, 1963. A full neurological assessment revealed no other abnormal features. He was given a course of Cytamen, 1,000 µg. daily for 10 days, and after 5 days noted subjective improvement. He was discharged home on May 19, 1963.

At a second ocular examination carried out on May 14, 1963, the visual acuity had improved to better than 6/9 and N6 in the right eye; and to 6/6 part and N6 in the left eye. There was no evidence of pallor of the optic discs and the macular areas appeared normal. Central field examination revealed only some relative loss to a 3 mm. red target within 5° of fixation.

At this stage of marked improvement to routine clinical testing, a detailed examination of colour vision function was also done. Testing showed a gross acquired dyschromatopsia of the extreme protonomalous type E.P.A. accompanied by a loss of discrimination in the yellow-blue and blue-green axis. The details are set out in the Table.

<table>
<thead>
<tr>
<th>Table</th>
<th>COLOUR VISION OF A MALE PATIENT AGED 55</th>
</tr>
</thead>
<tbody>
<tr>
<td>Test</td>
<td>Date of Test</td>
</tr>
<tr>
<td></td>
<td>16.5.63</td>
</tr>
<tr>
<td>Ishihara</td>
<td>3 misreadings; Total mistakes 16 not read at all 19/24</td>
</tr>
<tr>
<td>Tritan Plates</td>
<td>Usual misreadings of red-green defectives</td>
</tr>
<tr>
<td>Anomalouscope 1° subtend/ Red-Green Equation</td>
<td>Matched yellow of 583 mμ as 582 mμ to 643 mμ, i.e. matching range of 57 mμ or 130 just perceptible steps (5° subtend measurement—no improvement)</td>
</tr>
<tr>
<td>Yellow-Blue</td>
<td>Matching range—91 just perceptible steps (5° subtend—16 just perceptible steps)</td>
</tr>
<tr>
<td>Green-Blue</td>
<td>Matched Blue/Green of 496 mμ as 510 mμ to 474 mμ, i.e. matching range of 36 mμ or 85 just perceptible steps (5° subtend—512-488 or 35 just perceptible steps)</td>
</tr>
</tbody>
</table>

It is significant that these extremely fine tests of visual function gave perfectly normal results when repeated after an interval of 4 months, indicating complete recovery from the toxic effects of the drug.

**Comment**

Iatrogenic toxic amblyopia may present either with changes in the peripheral visual fields, as exemplified by quinine amblyopia, or with a scotomatous loss of central vision which may be associated with peripheral neuropathy.

The clinical features we have described are those one would expect to find in a central form of toxic amblyopia due to drugs. The bilateral central field defect was a true central scotoma rather than the centrocaecal defect typically associated with tobacco/alcohol amblyopia.
The striking results derived from the anomaloscope and parallel tests indicate the value of this technique, not only in the assessment of functional recovery, but also as a possible method of "pre-clinical" diagnosis in the earliest stage of toxic amblyopia. This would be of undoubted benefit in the prevention of irreversible central visual loss.

It seems reasonable to attribute the occurrence of toxic amblyopia in this patient’s case to the taking of ethchlorvynol, in view of the time relationship between the taking of the drug and the appearance of symptoms and the regression of effect on withdrawal.

The course of Cytamen was given empirically on the basis of its use in tobacco/alcohol amblyopia in the hope that it might be helpful. It is obviously not possible in this isolated case to assess exactly the part played by Cytamen in aiding or producing the reversal of the toxic effect.

We felt that this case was worth recording because it may be that, in rare instances, patients have an idiosyncrasy to ethchlorvynol and may, as a result, develop toxic amblyopia. Fortunately in this case, on cessation of the drug, the amblyopia was reversible and complete functional recovery appears to have been obtained.

We wish to thank Dr. J. K. Slater and Prof. G. I. Scott for permission to publish this case and for their advice and encouragement in preparing the report. We are also greatly indebted to Mr. R. Lakowski, who carried out the full colour testing.

BIBLIOGRAPHY