INTRACTABLE DIPLOPIA

SHAYNE BROWN DipAppSc(Cumb), DOBA

INGRID ATKINS AssocDip in Orthoptics(Cumb), DOBA

Co-authors: Margaret Doyle DOBA; Julia Kelly DOBA; Frances Merrick DOBA; Ann Stephens DOBA; Sandra Tait DipAppSc(Cumb), DOBA
Sydney Eye Hospital

Abstract

Four patients presented with apparent spontaneous diplopia. The diplopia was diagnosed subsequently as "intractable". These cases are discussed with special reference to the possible aetiology of "intractable" diplopia.

INTRODUCTION

Diplopia is a disturbing symptom which can be caused by a paresis of one or more of the ocular motor nerves, by mechanical restrictions or by decompensation of a heterophoria. Treatment of diplopia involves investigation of the possible cause, as diplopia can be the presenting sign of an underlying serious condition, and correction by means of surgery, prisms, orthoptic exercises or a combination of these treatment forms. Occasionally patients have diplopia which is unable to be eliminated. Four such patients presented with apparent spontaneous diplopia which was diagnosed subsequently as "intractable".

METHOD

All patients (three female, one male) had routine ophthalmological and orthoptic examinations. Detailed ophthalmological and orthoptic histories were available for each patient.

RESULTS

The common characteristics of these patients were:

1. The onset of diplopia in the teenage years was apparently spontaneous and intractable.
2. In each case there was a history of early onset of convergent strabismus. Initially, two patients presented with constant convergent strabismus and two with intermittent convergent strabismus which later decompensated and became constant.
3. None of the patients had a history of dense amblyopia. Two had equal vision and two had a small degree of amblyopia — 6/12, 6/6 the other 6/4, 6/9.

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4. None had demonstrable fusion at the time that the diplopia became troublesome. Careful examination of the medical histories revealed that fusion had never been elicited in the cases of constant strabismus. In the two cases which presented as intermittent strabismus, their fusion ability had been unstable at best and now could not be demonstrated.
5. At the onset of the diplopia none of the patients had a demonstrable suppression scotoma. It cannot be deduced from the histories whether a suppression scotoma was ever present or whether the ability to suppress had been lost, in the cases of constant strabismus. However the lack of suppression can be explained in the two cases of intermittent strabismus as they had undergone active suppression treatment to improve their fusional control.

DISCUSSION

The characteristics of this group suggest that the intractable diplopia was the result of a lack of or a loss of the ability to suppress.

What then is the mechanism of suppression? Burian and von Noorden[1] considered that suppression, and by extension amblyopia, represented a loss of the rhythm of binocular rivalry; Fells[2] asks "can facultative suppression become obligatory and is this the neurological basis of functional amblyopia? Ikeda,[3] on the other hand suggests that amblyopia and suppression are different entities. She has found that amblyopia is caused by poor foveal stimulation, but admits that the physiological basis of suppression is still unknown. The authors wish to stress that it is unwise to apply results of animal experiments to human conditions, but Ikeda's hypothesis that there is a different physiological basis for amblyopia and suppression is an attractive one.

This study was unable to offer any further explanation as to the mechanism of suppression. However, comment can be made on the spontaneity of the diplopia. Contrary to expectations diplopia had been noted in all cases at an early age but had become symptom producing in the teenage years. This can be explained in the two cases who had active suppression treatment as children. In retrospect this treatment probably contributed to the intractable diplopia. As these patients had no demonstrable fusion and no amblyopia, suppression was the only mechanism by which diplopia had been avoided.

In the other cases no reason could be found to account for the absence of suppression. Kilmister and Smith[4] suggested that trauma could disturb a patient's "central cortical suppression" mechanism but none of these patients had a history of trauma, nor of emotional stress, illness, change in glasses or a change in the angle of strabismus. This may suggest, and the authors would agree with Hugonnier,[5] Burian and von Noorden[1] and Duke-Elder,[6] that the onset of symptoms could be related to the patients' personality type rather than to any change in the ocular status.

The personality of the patients may also explain why the various methods of treatment have been unsuccessful. Various forms of occlusion, including opaque contact lenses, have been tried but all patients complained that their field of vision was reduced. The most successful form of treatment has been to encourage the patients to try to ignore the diplopia and to stress that the diplopia is not a sign of a more sinister problem. While helpful, this method is far from satisfactory. Hypnosis (Canadian Orthoptic Meeting 1982) has been suggested as an alternative form of treatment and may be beneficial for this small but distressed group of patients.

CONCLUSION

In conclusion it is felt that the loss or absence of suppression was the cause of the intractable diplopia in these patients. It is apparent that suppression is an important natural defence mechanism against disruption to the normal binocular sensory adaptions, therefore orthoptists should be particularly discerning about the presence of true fusion before carrying out vigorous suppression treatment because it seems

that contrary to common belief children do not always learn to resuppress easily once it has been eliminated.

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