Matters arising

Sir: I was interested to read the elegant account by Gresty et al\(^\text{1}\) of those features of nystagmus which may help to determine whether its cause is congenital or acquired. However, I was surprised that there was no mention of ocular albinism (one of the commonest causes of congenital nystagmus) nor of primary retinal lesions. If I were presented with a patient whose nystagmus seemed out of proportion to the neurological signs, I would first examine the fundi and ask an ophthalmologist to examine the irides for translucency and to perform tests of retinal function.

SARAH BUNDEY

Reference


Gresty replies:

Dr Bunley is quite correct in pointing out the strong association between nystagmus of the congenital type and unequivocal ocular albinism and the presence of this trait would be diagnostically helpful when congenital nystagmus is suspected. It should be noted that it may be difficult for the ophthalmologist to distinguish between the tyrosinase-positive albino and a blonde, e.g. with a congenital type nystagmus,\(^1\) in which case the characteristics of the nystagmus itself assume their importance. As a point of clarification, there is the possible source of confusion in the literature that although several types of nystagmus may be congenital (for example, lantent, pendular nystagmus of an ambylopic eye, wandering eye movements of the blind) the term "congenital nystagmus" is restricted to refer to a nystagmus with an exponentially increasing slow phase velocity.

Reference


Opioid induced unconsciousness reversed by changes in opioid cholinergic and adrenergic function.

Sir: The paper by Goldman et al\(^4\) is of interest. However, although these workers mention the possibility that opioid release may have occurred following the lesion they describe, no further comment is made. We would like to suggest a possible mechanism of action of naloxone in nystagmus and related clinical conditions. It has been suggested that in traumatic brain death and possibly other CNS disorders, (particularly in relation to diencephalic structures) that there is an over-activity of the endogenous opioid system, which leads to loss of consciousness.\(^2\) This paper\(^1\) and those of other workers\(^5\) adds further support for this hypothesis. Furthermore it is possible that the endogenous opioid system causes inhibition of the nor-adrenergic system within the brain by a feed-back loop operating between the locus coeruleus and the arcuate nucleus of the hypothalamus.\(^6\) Decreased adrenergic activity could also lead to decreased cerebral blood-flow.\(^4\) Furthermore, opiates can cause central release of acetylcholine,\(^3\) and activation of a pontine cholinergic site has been implicated in causing unconsciousness.\(^8\)

It is therefore possible that naloxone reversal of unconsciousness following ischaemic damage to the brain-stem, although mediated by the endogenous opioid system, occurs as a result of increased nor-adrenergic and decreased cholinergic activities.

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Goldman replies:

The mechanism by which an endogenous opioid release could influence the level of consciousness was not discussed in our short report. We believe that the naloxone-induced improvement in acutely ischaemic lesions is directly involved in the ischaemic lesion, the effects of naloxone are probably related to the severity of the lesion. In relatively mild lesions, naloxone could perhaps reduce the life-threatening period of unconsciousness and respiratory depression for the reasons suggested.

Reference