Matters arising

Sir: I was interested to read the elegant account by Gresty *et al*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

- ¹ Gresty M, Page Nicholas, Barratt Hilary. The differential diagnosis of congenital nystagmus. *J Neurol Neurosurg Psychiatry* 1984;47:936-42.

Gresty replies:

Dr Bunday 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, but with a congenital type nystagmus,¹ in which case the characteristics of the nystagmus itself resume 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, latent, pendular nystagmus of an amblyopic 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 and this is how we use the term. In this context it is not clear whether the nystagmus of all albinos is "congenital nystagmus".

We are aware of no firm data regarding the prevalence of albinism and/or congenital nystagmus in the population. Accordingly, we can only quote from our experience that although all oculocutaneous albinos have nystagmus, patients with idiopathic congenital nystagmus are far more common than albinos with nystagmus.

Although it has been reported that patients with retinal lesions may have nystagmus we are unaware of any eye movement recordings demonstrating that the nystagmus can have an exponentially

increasing slow phase velocity.

Reference

- ¹ Abadi R, Dickinson C. Recognizing an albino. Trans. 1st Int. Cong. Br. Coll. Ophthalmic Opticians. "The frontiers of Optometry". 1984;22-32.

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

Sir: The paper by Goldman *et al*¹ 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 this 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 paper¹ and those of other workers³ 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.^{3,4} Decreased adrenergic activity could also lead to decreased cerebral blood-flow.⁴ Furthermore, opiates can cause central release of acetylcholine,⁵ and activation of a pontine cholinergic site has been implicated in causing unconsciousness.⁶

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.

MA GILLMAN

R SANDYK

8 Highlands House,
173 Louis Botha Avenue,
Orange Grove 2192
S. Africa

References

- ¹ Goldman S, Cordonnier MJB, Sztencel J. Brainstem ischaemia presenting as naloxone-reversible coma followed by downward gaze paralysis. *J Neurol Neurosurg Psychiatry* 1984;47:77-8.
- ² Gillman MA, Lichtigfeld FJ. The opiate system

in traumatic brain death. *Lancet* 1981; 1:890-1.

- ³ Baskin DS, Hosobuchi Y. Naloxone reversal of ischaemic neurological deficits in man. *Lancet* 1981;2:272-5.
- ⁴ Gillman MA, Lichtigfeld FJ. Naloxone and cerebral ischaemia. *Lancet* 1981;2:643.
- ⁵ Beani L, Bianchi C, Siniscalchi A. The effect of naloxone on opioid-induced inhibition and facilitation of acetylcholine release in brain slices. *Br J Pharmacol* 1982;76:393-401.
- ⁶ Hayes RL, Pechura CM, Katayama Y, Povlishock JT, Giebel ML, Becker DP. Activation of pontine cholinergic sites implicated in unconsciousness following cerebral concussion in the cat. *Science* 1984;223:301-3.

Goldman replies:

The mechanism by which an endogenous opioid release could influence the level of consciousness was not discussed in our short report, and Drs Gillman and Sandyk present interesting suggestions on this point. We would like to emphasise that, even in cortical ischaemic lesions, the naloxone-induced improvement seems to imply that subcortical and brainstem regions are not injured.¹ When these regions, related to the endogenous opioid systems, are 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 lifethreatening period of unconsciousness and respiratory depression for the reasons suggested.

Reference

- ¹ Levy RM, Stryker M, Hosobuchi Y. Studies of nuclear magnetic resonance imaging and regional cerebral glucose metabolism in acute cerebral ischemia: possible mechanism of opiate antagonist therapeutic activity. *Life Sci* 1983;33:763-8.