The Journal of Laryngology and Otology
July 1998, Vol. **112**, pp. 622–627

Interpretation of the dilated pupil during endoscopic sinus surgery

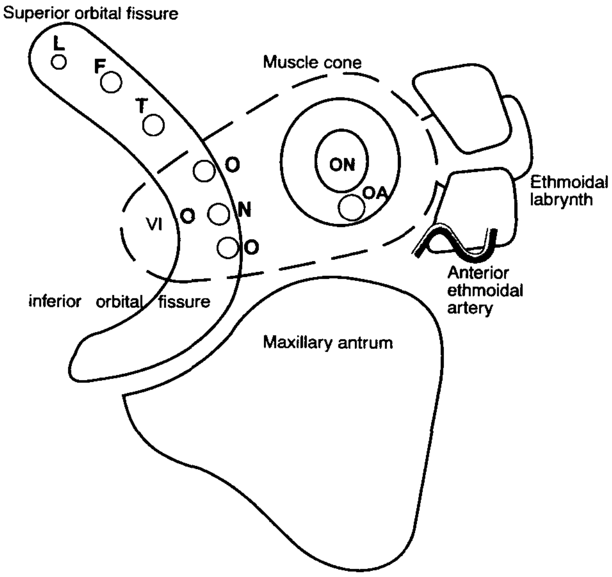
J. D. T. MASON†, R. J. HAYNES*, N. S. JONES*

Abstract
This article describes the anatomy of the visual pathways and how they should be assessed under anaesthesia. The differential diagnosis of asymmetrical pupils is illustrated with clinical examples and a strategy as to how they should be examined.

Key words: Vision; Endoscopy; Nasal cavity; Sinusitis; Visual pathways; Vision disorders

Introduction

There is a recurring nightmare for the endoscopic sinus surgeon that the patient will be blind at the end of the procedure. The optic nerve, orbital fat and the anterior ethmoidal artery are all vulnerable to damage (Figure 1).



L: lacrimal, F: frontal, T: trochlear, VI: Abducens, O: Superior and inferior divisions of the oculomotor nerve, N: Nasociliary nerve, ON: Optic nerve, OA: Ophthalmic artery

FIG. 1

Diagrammatic relationship of nerves which could potentially be damaged during endoscopic sinus surgery.

Various attempts have been made to monitor the eye function during endoscopic sinus surgery. Light emitting diode goggles (Raudzens, 1982) and fitted contact lenses (Wright *et al.*, 1973) have been used to deliver stimuli and measure evoked potentials. Palma and Guadingino (1990) suggested that visual evoked potential monitoring is useful for surgery on lesions near the optic chiasm. However, the optic chiasm can be damaged without detectable loss of visual evoked potentials (VEPs) (Allbright and Sclabassi, 1985). Visual evoked potentials are very sensitive to anaesthesia, temperature and blood pressure (Palma and Guadagnino, 1990). Visual evoked responses are very variable in the operating room and flash VEP are not useful for intraoperative monitoring under general anaesthesia as they require a cortical response (Cedzich *et al.*, 1987; Jones, 1997).

These objective measures of eye function during sinus surgery are so fickle that it remains an immutable maxim that the eyes should always be uncovered during endoscopic sinus surgery. The lids should be elevated intermittently to look for an afferent pupillary defect. If the pupils are of unequal size then this physical sign has to be interpreted carefully, as it may be the relatively innocent and short-lived effect of pharmacological agents such as cocaine and adrenaline (Figure 2). An asymmetrically dilated pupil may be a sign of damage to the occulomotor nerve by an increase in intra-orbital pressure due to an expanding haematoma, though it is very unlikely to be due to the latter without obvious proptosis. Optic nerve damage will not cause a dilated pupil but will cause an afferent pupillary defect. There is a common misconception that damage to the optic nerve will cause a dilated pupil.

From the Departments of Otorhinolaryngology – Head and Neck Surgery and Ophthalmology*, University Hospital, Nottingham, and the Department of ENT–Head and Neck Surgery†, Royal Bolton Hospital, Bolton; UK.
Accepted for publication: 6 May 1998.

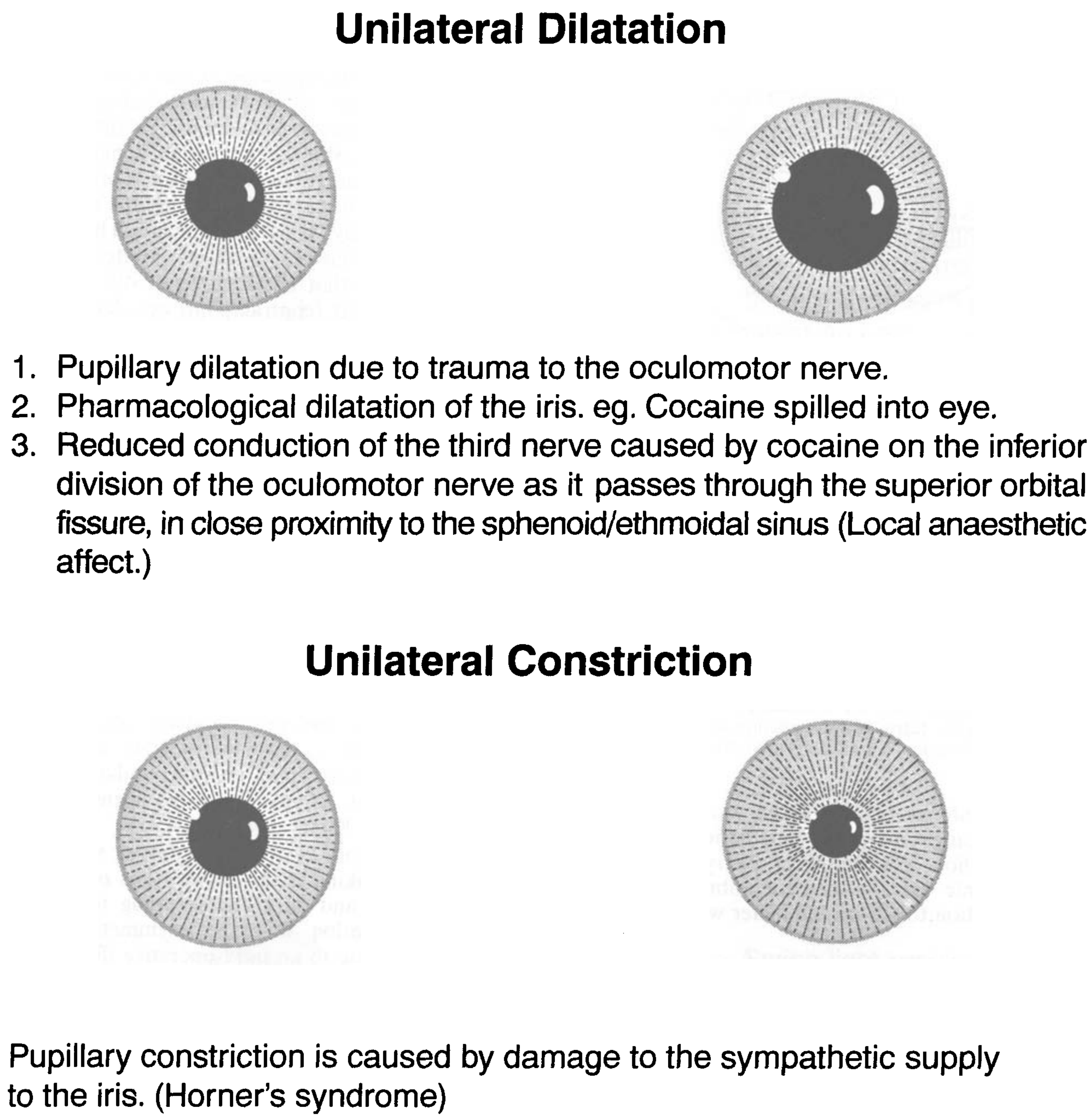


FIG. 2

Acquired causes of assymmetric pupils during endoscopic sinus surgery.

The size of the pupil depends on the balance between the sympathetic and parasympathetic systems acting on the iris. Sympathetic stimulation acting via the superior cervical ganglion and sympathetic fibres carried on the internal carotid artery cause the pupil to dilate. Parasympathetic stimulation via the third cranial (oculomotor nerve), the ciliary ganglion and the short ciliary nerves cause the pupil to constrict.

Anatomy (Figure 1)

The following are the nerves that carry sensory and motor information relevant to the size of the pupil:

(1) The optic nerve carrying sensory information regarding the amount of light striking the retina (Figure 3). This may be damaged in endoscopic sinus surgery as it passes through the lateral wall of the sphenoid or within the orbit. The nerve is particularly susceptible if it is related to an Onodi cell. Damage to the optic nerve may be overlooked unless an afferent pupil defect is specifically tested for, otherwise the pupil size remains the same because of input to the brainstem from the other eye, (assuming the other eye is open – if the other eye is closed or completely blind damage to the optic nerve will result in pupil dilation because the only source of afferent input has been damaged).

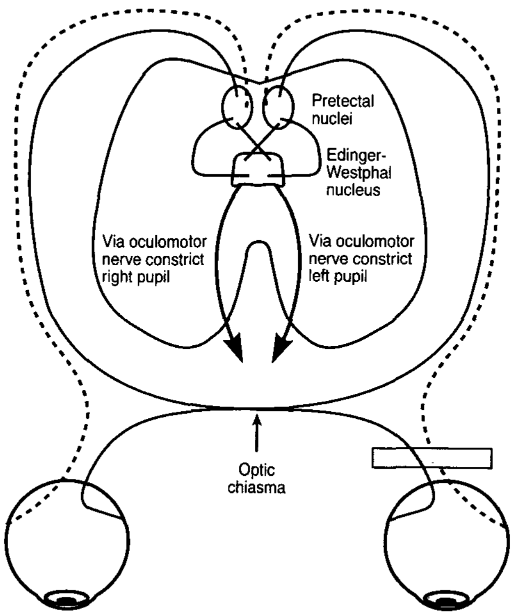


FIG. 3

Neural pathways involved in direct and consensual pupilliary responses, showing why shining a light in one eye causes *both* pupils to constrict. Note the decussation of fibres from the medial half of the retina in the optic chiasma, and from the pretectal nuclei to the Edinger Westphal nucleus.

(2) The inferior branch of the oculomotor nerve carries preganglionic parasympathetic fibres to the ciliary ganglion, and the short ciliary nerves carry postganglionic parasympathetic fibres from the ciliary ganglion to the iris sphincter which constricts the iris.

(3) The nasociliary nerve which crosses the optic nerve, carries sympathetic fibres from the internal carotid plexus to the eye via both the long ciliary nerves and the short ciliary nerves. This can show interindividual variation.

(4) The Edinger-Westphal nucleus (Figure 3) is responsible for the constrictor response to light.

Autonomic nerves are sensitive to peripheral stimulation. Cocaine and adrenaline have sympathomimetic properties. Both these agents can stimulate $\alpha 1$ receptors at the nerve terminal, causing the iris radial muscle to contract and the pupil to dilate (Martin *et al.*, 1996; Ruffolo *et al.*, 1996). Cocaine will also have a local anaesthetic blocking effect on nerve fibres, if applied to the nerve proximal to the smooth muscle motor end plate.

The neural balance that controls the size of the pupil depends on the integrity of the nerves mentioned above. The function of these nerves can be disturbed by direct damage, pressure from an expanding haematoma, pharmacological stimulation or pharmacological suppression. Experimental work on primates suggests that the optic nerve can withstand pressure-induced interruption of axonal transport for eight hours and total ischaemia for two hours (Radius and Anderson, 1981).

Case report

We present a case to show why it is essential to know not only the anatomy of the nerves involved but also the effects of pharmacological agents used. A 37-year-old lady was undergoing fronto-spheno-ethmoidectomy. Shortly after a right sphenoidotomy was performed an adrenaline pack was placed in the sphenoid sinus because of bleeding, the right pupil became obviously larger than the left. The ophthalmologist whose advice was sought, pointed out to the fearful surgeon that the right pupil still reacted to light, and the left (contralateral) eye demonstrated an appropriate consensual and direct pupillary constriction to light. The reaction of the right pupil was sluggish compared to the left but definitely present. On waking from anaesthesia the vision of the patient was normal. It took four hours for the pupil to return to normal size.

This case illustrates that pupil dilatation in isolation is not a sign of damage to the optic nerve. Pupil dilatation will not occur with damage to the optic nerve in isolation, as the parasympathetic supply has not been interfered with.

To make sense of changes in pupillary size and to maximize awareness of any possible eye trauma during endoscopic sinus surgery, the following points are made.

(1) Check the patient's pupillary size prior to surgery. If there is asymmetry, this should be documented. Anisocornia (unequal pupils) is present in 20 per cent of people with normal vision (Bajandas and Kline, 1988). Ask the patient if there is any history of eye complaints or surgery. It is anxiety-provoking if this variation is only noted during surgery and very embarrassing to be told after the operation that the asymmetric pupils thought to be due to an intra-operative disaster are actually congenital.

(2) Note any pre-operative proptosis.

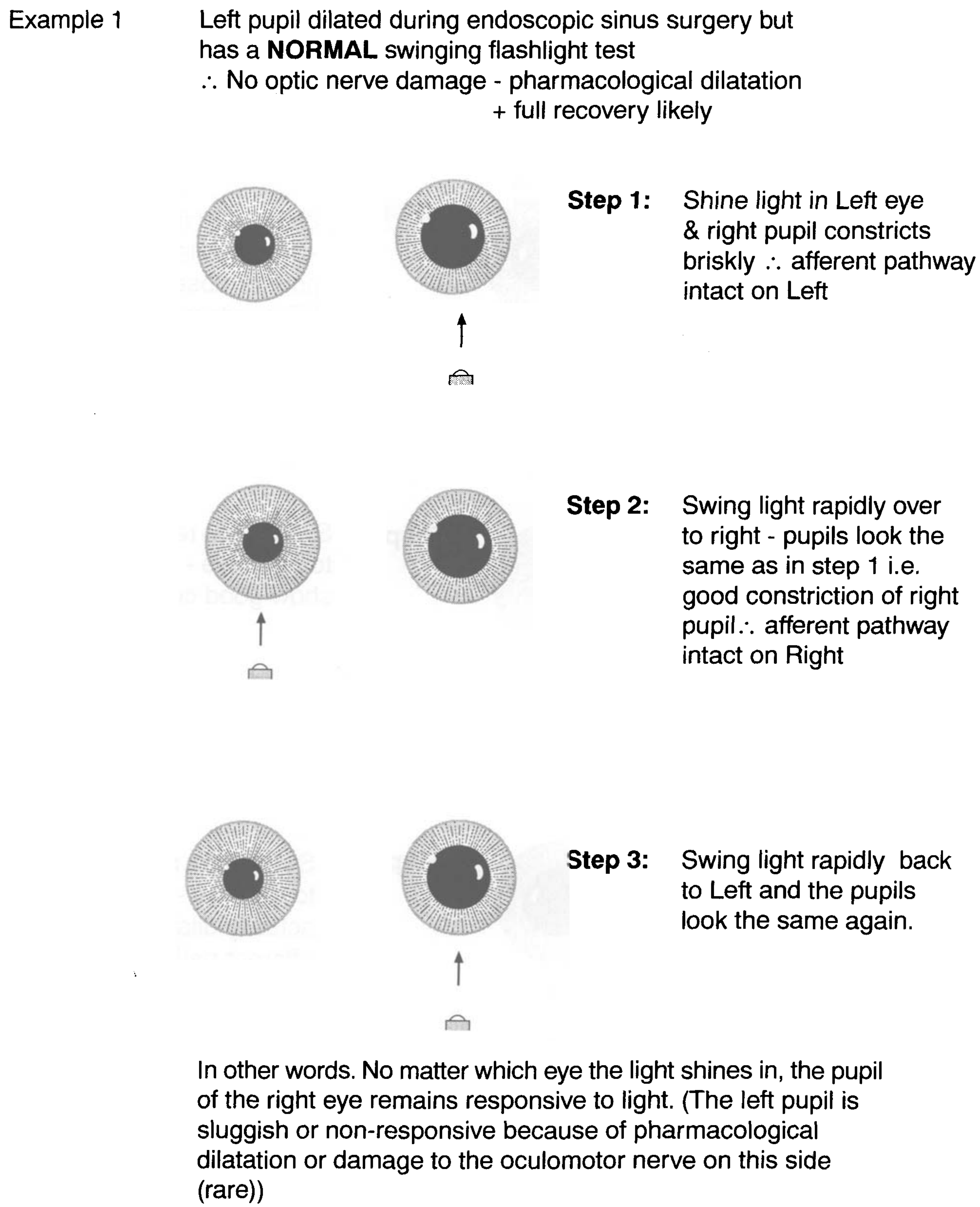
(3) If cocaine or adrenaline spills into the eye when preparing the nose for surgery, it should be noted. Cocaine has a sympathomimetic effect, and therefore dilates the pupil, but it potentially also has a local anaesthetic effect which could also dilate the pupil by paralysing the parasympathetic supply to the iris via the oculomotor nerve.

(4) When the patient is anaesthetized make a simple digital check of ocular pressure before the operation begins. This does not have a direct bearing on pupil size, but may alert the surgeon to an expanding haematoma if the pressure behind the eye increases per-operatively.

(5) Use the light from the endoscope to check pupillary responses, both direct and consensual, using the swinging flashlight test (Figure 4a and b) – before the operation begins.

(6) Throughout surgery ask the assistant to observe the eyes for any abnormality or 'tugging'.

(7) If a pupil is noted to become dilated during surgery, employ the swinging flashlight test (Figure 4a and b). The direct response of the dilated eye may be sluggish due to pharmacological agents. If you



(a)

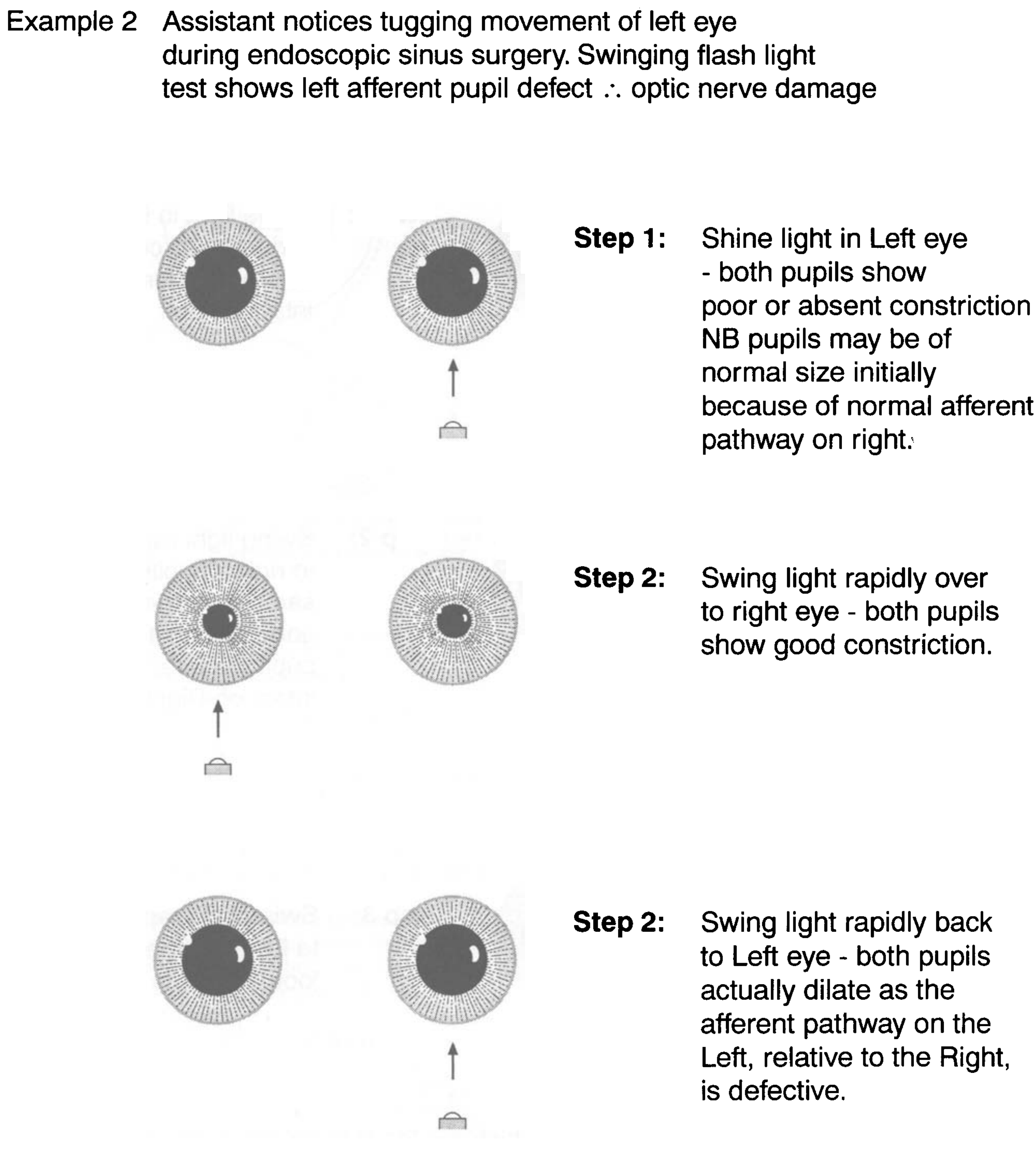
FIG. 4 a and b

The swinging flashlight test to check direct and consensual pupillary reflexes, for a relative afferent pupil defect (examples 1 and 2).

establish rapid and good constriction of the other pupil when light is shone into the dilated eye, this will reassure you that although the pupil is dilated, the optic nerve is still functioning.

These simple manoeuvres should alert the surgeon to any trauma to the optic nerve and prevent any unnecessary concern when a patient develops a dilated pupil. It is important to note that the best test

https://doi.org/10.1017/S0022215100141283 Published online by Cambridge University Press



(b)

to ensure an intact and functioning visual pathway is to check the patient's visual acuity in each eye separately and this should be done when recovery from their anaesthetic allows them to do this.

References

Allbright, A. L., Sclabassi, R. J. (1985) Cavitron ultrasonic aspirator and visual evoked potential monitoring for chiasmal gliomas in children. *Journal of Neurosurgery* **63:** 138–140.

Bajandas, F. J., Kline, L. B. (1988) The pupil. In: *Neuro-ophthalmology Review Manual.* Chap. 8 Slack, New Jersey, pp 113–124.

Cedzich, C., Schramm, J., Fahlbusch, R. (1987) Are flash evoked potentials useful for intraoperative monitoring of visual pathway function? *Neurosurgery* **21:** 709–715.

Jones, N. S. (1997) Visual evoked potentials in endoscopic and anterior skull base surgery. *Journal of Laryngology and Otology* **111:** 513–516.

Martin, P. R., Lovinger, D. M., Breese, G. R. (1996) Alcohol and other abused substances. In *Principles of Pharmacology*. Chapman & Hall, New York, pp 431–435.

Palma, V., Guadagnino, M. (1990) Electrophysiological monitoring of neuronal functions during neurosurgery. *Acta Neurologica* **12:** 481–504.
Radius, R., Anderson, D. (1981) Reversibility of optic nerve damage in primate eyes subjected to intraocular pressure above systolic blood pressure. *British Journal of Ophthalmology* **65:** 661–672.
Raudzens, P. A. (1982) Intraoperative monitoring of evoked potentials. *Annals of the New York Academy of Science* **388:** 389–403.
Ruffolo, R. R., Tendelenberg, U. G., Langer, S. Z. (1996) Chemical neurotransmission: peripheral autonomic nervous system. In *Principles of Pharmacology*. Chapman & Hall, New York, pp 58–62.
Wright, J. E., Arden, G., Jones, B. R. (1973) Continuous monitoring of the visually evoked response during intraorbital surgery. *Transactions of the Ophthalmological Society of the United Kingdom* **93:** 311–314.

Address for correspondence:
N. S. Jones,
Department of Otorhinolaryngology – Head and Neck Surgery,
University Hospital,
Nottingham NG7 2UH.