Correspondence

method. It has been generally accepted² that human fusional response to retinal image disparity contains 2 components: a motor component in the form of compensatory, vergence eye movements and a sensory or nonmotor component described by the extent of Panum's fusional areas. Objective measurements of cyclofusional response, which utilised binocular eye movement monitoring devices, revealed a substantial sensory component in cyclofusional response.³⁻⁶ The magnitude of this sensory component is a function of stimulus complexity and of the visual angle subtended by the stimulus. Subjective methods such as the one used by Sen *et al.* may be used to measure the overall amplitude of cyclofusional response, but cannot be used to measure the amplitude of either the motor or the sensory components individually, because the method is unable to distinguish between the 2 components. Therefore Sen *et al.* could not have measured *torsional vergence* (a term reserved exclusively for the description of the motor component) as was indicated in their paper, but only the overall cyclofusional response. I think that the interesting results of Sen *et al.* may be better appreciated subsequent to this correction in terminology.

Biomedical Center,
Northwestern University and
Division of Ophthalmology,
Evanston Hospital,
2145 Sheridan Road,
Evanston, IL 60201, USA.

ANDREW E. KERTESZ

References

- 1 Sen DK, Singh B, Mathur GP. Torsional fusional vergences and assessment of cyclodeviation by synoptophore method. *Br J Ophthalmol* 1980; **64**: 354-57.
- 2 Parks MM. *Ocular Motility and Strabismus*. New York: Harper Row, 1975: 40.
- 3 Crone RA, Everhard-Halm Y. Optically induced eye torsion. *Albrecht von Graefes Arch Klin Ophthalmol* 1975; **195**: 231-9.
- 4 Kertesz AE, Sullivan MJ. The effect of stimulus size on human cyclofusional response. *Vision Res* 1978; **18**: 567-71.
- 5 Sullivan MJ, Kertesz AE. Binocular coordination of torsional eye movements in cyclofusional response. *Vision Res* 1978; **18**: 943-49.
- 6 Sullivan MJ, Kertesz AE. Peripheral stimulation and human cyclofusional response. *Invest Ophthalmol Visual Sci* 1979; **18**: 1287-91.

SIR, We were very interested to read Dr Kertesz's comments about our paper.¹ We agree that the subjective method used by us measured the overall amplitude of cyclofusional response. We are aware that Parks² has used the term 'fusional vergence' to mean the motor component of fusion. But our impression is that the term 'torsional fusional vergence' is more often used to mean the overall amplitude of cyclofusional response rather than only the motor component. Parks³ used a subjective method and stated that the normal person had an incyclovergence of 6° to 10° and an excyclovergence of 8° to 12°. Bullock and Bredemeyer⁴ reported incyclovergence to be 6° to 10° and excyclovergence to be 4° to 8°. In view of the fact that the motor component of cyclofusion is small⁵ or absent⁶ we presumed with justification that what Parks,³ Bullock and Bredemeyer,⁴ and Kramer⁷ meant by 'torsional fusional vergence' was nothing but the overall cyclofusional response

and not merely its motor component. Since we wanted to compare our data with those of Parks,³ Bullock and Bredemeyer,⁴ and Kramer⁷ we retained the term 'torsional fusional vergence' to avoid confusion.

V/4 MAM College Campus,
Kotla Road,
New Delhi 110002,
India.

D. K. SEN

References

- 1 Sen DK, Singh B, Mathur GP. Torsional fusional vergences and assessment of cyclodeviation by synoptophore method. *Br J Ophthalmol* 1980; **64**: 354-7.
- 2 Parks MM. *Ocular Motility and Strabismus*. New York: Harper Row, 1975: 40.
- 3 Parks MM. *Ocular Motility and Strabismus*. New York: Harper Row, 1975: 65.
- 4 Bullock K, Bredemeyer HG. *Orthoptics: Theory and Practice*. St Louis: Mosby, 1968: 75.
- 5 Ellerbrock VJ. Inducement of cyclofusional movements. *Am J Optom Physiol Opt* 1954; **31**: 553-66.
- 6 Kertesz AE, Jones RW. Human cyclofusional response. *Vision Res* 1970; **10**: 891-6.
- 7 Kramer ME. *Clinical Orthoptics. Diagnosis and Treatment*. 2nd ed. St Louis: Mosby, 1953: 480.

Oculocardiac reflex

SIR, A recent article¹ by Drs Apt and Isenberg described the usefulness of the oculocardiac reflex in identifying and recovering a lost muscle. The authors make the observation that a chronically lost muscle may be difficult to identify visually, 'often blending into the surrounding tissues.' They also correctly point out that the 'area to be explored often has adhesions and extraneous tissue.'

It is not clear to me how the surgeon can be certain that he is not pulling on nonmuscular scar tissue which has adhered to the true muscle, thereby producing a vagal response. This false muscle identification would lead to surgery (recession, resection, advancement) on nonmuscular tissue, which would not correct the patient's strabismus. Also, in a recent paper by Drs Parks and Bloom² it was demonstrated that in certain cases the muscle capsule may remain attached to the surgical insertion site on the sclera, while the muscle itself slipped back within the capsule; surgery performed on the nonmuscular capsular tissue was ineffective in correcting these patients' motility problems. The oculocardiac reflex was not investigated in this study. However, it is reasonable to expect that, since the capsule remained intimately attached to its muscle, pulling on the capsule would stretch the muscle and produce the oculocardiac reflex.

Drs Apt and Isenberg have made an important observation in the treatment of both the lost and the slipped muscles. However, the surgeon must be aware that production of the oculocardiac reflex may represent a false-positive result in the identification of a lost or slipped muscle.

Marshall Taylor Building,
836 Prudential Drive, Suite 555,
Jacksonville,
Florida 32207,
USA.

JEFFREY N. BLOOM

References

- 1 Apt L, Isenberg SJ. The oculocardiac reflex as a surgical aid in identifying a slipped or 'lost' extraocular muscle. *Br J Ophthalmol* 1980; **64**: 362-5.
- 2 Parks MM, Bloom JN. The slipped muscle. *Ophthalmology* 1979; **86**: 1389-96.

SIR, When the ophthalmic surgeon is seeking a lost or slipped extraocular muscle, he often would benefit by additional means of assistance. The use of the oculocardiac reflex as an aid in this quest was suggested, and subsequently proved, in this article as a confirmatory tool. Dr Bloom correctly points out that this test, like most clinical tests, has a certain false-positive rate. As mentioned in our paper, it also has a certain false-negative rate depending on which muscle receives the traction. As described in the case presentations, the intent of the test was to confirm that a tissue, previously dissected and freed of adhesions, is or is not extraocular muscle. One should not adventurously pull on any tissue strand in the field hoping to elicit the reflex since it might be adherent to the muscle at some point.

As with any clinical test, the results of this test should be evaluated only in the context of other data. If one were to pull on muscle capsule devoid of muscle fibres, as described in the paper by Drs Parks and Bloom, and yet elicit the reflex, it would be reasonable to assume that the capsule was somehow attached to the muscle. One should then follow the capsule posteriorly in the hope of finding the true muscle and then repeat the test on the newly discovered 'muscle.' In exploring for a slipped or lost extrinsic eye muscle the surgeon is not likely to confuse muscle capsule with true muscle fibres. Capsule tissue is distinctly thin, almost transparent, and without substance. In fact, it was this observation that led Dr Parks to the notion that capsule rather than muscle must have been sutured to the sclera by the previous surgeon when Dr Parks operated on patients with clinical evidence of a slipped or lost extrinsic eye muscle.

In closing, we would like to add that since the time we submitted our paper for publication we have utilised the oculocardiac reflex in identifying 5 additional lost muscles. Jules Stein Eye Institute,
UCLA School of Medicine,
Los Angeles, California 90024,
USA.

LEONARD APT

SHERWIN J. ISENBERG

Obituary

**Courtenay Hugh Greer, MB, BS,
MRCS, LRCP, FRCPA, FRACO,
FRCPath**

Hugh Greer, pathologist to the Victorian Eye and Ear Hospital, Melbourne, and one time assistant pathologist at the Institute of Ophthalmology, London, died suddenly on 9 March at the age of 68.

Having qualified in medicine at the Charing Cross

Medical School in 1941 and served for several years with the Royal Naval Volunteer Reserve during the second world war, Dr Greer returned to his alma mater to specialise in pathology. There followed a period which encompassed all aspects of clinical pathology at the Whittington Hospital before he took up the post of assistant pathologist at the Institute of Ophthalmology in 1951. Here he rapidly established himself as an authority on the diagnostic problems of ophthalmic pathology.

Thus equipped, he sailed to Australia on Christmas Eve 1954 to take up an appointment he was to hold for the next 25 years. His responsibilities extended to otorhinolaryngology as well as ophthalmology, and before long his opinion in both these fields was canvassed from all parts of the Australian continent and beyond. He was a capable teacher blessed with a straightforward no-nonsense approach and a lively sense of humour, and very many ophthalmologists have reason to be grateful to him for having been given a secure grounding in the pathological basis of their subject. Hugh's skill as a communicator reached a wider circle when he published a deservedly popular primer on *Ocular Pathology* in 1963, its success being evident from the demand for 2 further editions in the subsequent years.

Eligible to retire in 1978, he stayed in post until a successor had been appointed and was able to take up the reins, and it is sad that he was not to enjoy the fruits of his retirement. He leaves a wife, Eileen, 3 sons and a daughter and we extend to them our deepest sympathy.

A. G.

Alan Stanworth, MD, PhD, DOMS

Alan Stanworth died on 17 May 1981 at the age of 61 after a year of illness. He had a remarkable career in ophthalmology, which really began in the Manchester Royal Eye Hospital after he left the Army with the rank of captain. His researches spanned the optics of the cornea, the epidemiology of uveitis, contact lens design, and the recognition of microstrabismus as the real state of many 'cured' squints. He defined the entity of microstrabismus before it was so called and was years ahead of others in that field. He moved to Sheffield in 1960 and was a consultant there, first at the Royal Infirmary and later at the Royal Hallamshire Hospital.

Alan Stanworth graced other aspects of the British ophthalmological scene. He was for many years a council member of the Oxford Congress and was master in 1971 and 1972. He was also a member of the British Orthoptic Council and later chairman of that body. A staunch supporter of the North of England Ophthalmological Society, he was president in 1968.

His talents extended outside ophthalmology. In his younger days he was a runner of considerable reputation; later he was a keen hill walker, and more recently took up horse riding. He was also an excellent pianist.

I had the good fortune to work in a parallel clinic with Alan for the last 11 years and enjoyed his close co-operation on clinical problems and learned much from his store of experience. The many ophthalmologists and orthoptists whom he taught will remember him with gratitude and affection. We extend our deepest sympathy to his wife Carol, their young son, and his daughter June.

I. M. S.