An "overtrained" ophthalmologist responds

EDITOR,—As one of the most “overtrained”1 ophthalmologists in the United Kingdom at the present time, I was delighted and stimulus to read the excellent, erudite, and witty commentary by James Acheson.2 I think that the idea that lies at the heart of the matter is, as Mr Acheson himself puts it, “It all depends on what you mean by training…”3 Surely one of the driving reasons behind the length of all specialist training in the UK has always been the high demands of the service commitment of the senior house officer and registrar grades alike. Until the issue of doctors’ numbers can begin to be tackled at a meaningful level in the UK we shall forever have the push-pull politics of service versus training. It is still worth pointing out that we have the lowest number of doctors per capita in the developed world, bar only Greece and Albania. It is also very true that the standards of ophthalmology training in the UK are regarded very highly by trainees from overseas, who regularly come to the UK to complement and polish off their training. However, they come mainly for subspecialty training and often go to super-specialist regional centres, where they act as fellows, often in a somewhat privileged position. They are able to benefit from the high level of internationally re-nowned expertise in their chosen field that the UK is still able to provide. We in the UK face a rather unique situation, in that super-specialist fellowship training is quite rightly becoming the norm while still being outside the national Calman training programme. This sends a very mixed message about its value to the powers that be. It is also far from easy for every trainee to find a suitable fellowship and funding.

So, on the one hand the length of training could be shortened by tackling the issue of service versus training demands, and on the other hand perhaps training could formally be lengthened to ensure that British ophthalmologists are able to stay at the forefront of their chosen fields in the international arena. We all await developments with interest.

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Biometric aspects and comparison with published papers

EDITOR,—In their discussion on central corneal thickness determined with optical coherence tomography in glaucoma in the BJO, Bechmann et al4 mention the results of Ehlers et al5 and compare them with the results of Whitacre et al6 without regarding generally accepted principles of interpretation.6 Some biometrical considerations will be found in the following.

In the paper by Ehlers et al in figure 4 the correlation coefficient between the correction value and corneal thickness is 0.768 at n = 29. In the comparable figure 2 of Whitacre et al no correlation coefficient is given at n = 15. This coefficient was calculated by us after digitalising the data points. It equals 0.51. According to Klemm,7 (page 97) the estimate of regression is extremely unreliable and thus useless at r < 0.6. The data of Ehlers et al, therefore, are much more convincing than the data of Whitacre et al. This fact does not reduce the merit of Whitacre, who brought the problem of corneal influence on tonometry to our notice.

It escaped the attention of Bechmann et al that figure 4 of Ehlers et al and figure 2 of Whitacre et al differ fundamentally from figure 2 in the paper by Wolfs et al.8 Furthermore, regarding the results of the Rotterdam study, the figure 4 of Ehlers et al shows the correction value according to corneal thickness and in figure 2 of Whitacre et al the ordinate shows the measurement error according to corneal thickness. These two ordinates (Ehlers et al and Whitacre et al) differ by sign and show the result of subtraction of intracamerally measured IOP and application tonometry values. The ordinate in figure 2 of the Rotterdam study, however, shows the correlation value after application tonometry. This is a fundamental difference that absolutely forbids a comparison. The Rotterdam study does not provide a correlation coefficient of the data shown in figure 2, which may be interpreted by points of view. We have similar data and have calculated the coefficient of correlation r = 0.17. Therefore, in this case it may be concluded that the estimate of regression is playing with figures only (Klemm,7 page 97).

In summary, the data of Ehlers et al currently show the association of measurement error and corneal thickness in the most convincing way. Bechmann et al have (erroneously) seen a small influence of central corneal thickness in IOP measurement in the literature they attribute an important part to corneal thickness in the diagnosis and understanding of various types of glaucoma. It can be concluded from the context that the authors treat corneal thickness as a new quantity in the diagnosis of glaucoma, comparable with optic disc parameters. They have nicely shown different values of corneal thickness in the various types of glaucoma. However, they do not believe that corneal thickness influences applanation tonometry. Therefore, they have to explain their findings in a more complicated way. The psychologist and philosopher Watzlawick9 (page 67) states that we prefer declaring undeniable facts (which are inconsistent with our explanation) to be untrue or unreal instead of fitting our explanation to these facts. The application of biometric knowledge in judging the data of Whitacre et al10 and a reinterpretation of figure 2 of the Rotterdam study11 may fit the opinion of the authors to the most likely explanation12 that corneal thickness influences the results in applanation tonometry to a clinically relevant degree, and that recommends the application of OCT in the diagnosis of glaucoma if available.

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Major orbital complications of endoscopic sinus surgery

EDITOR,—We read with interest the article by Rene et al13 We would like to clarify a few points regarding endoscopic sinus surgery. Endoscopic sinus surgery is considered by many to be the most exciting development in otorhinolaryngology. The aim is to restore the natural mucociliary clearance mechanism, drainage, and suture of the sinuses by a minimally invasive technique, maintaining as much of the normal anatomy as possible.2 We agree with the authors that the incidence of ocular complications is low and similar to those reported by other non-endoscopic approaches.3 The authors mentioned CT scanning as a preoperative measure to reduce complications; this is a well established practice in all departments that practise endoscopic sinus surgery. Orbital complications are more likely to occur in patients with extensive polyposis especially those who had multiple surgery; however, in a survey of British otorhinolaryngologists4 the overall estimated complication rate was 0.24%. As a matter of fact endoscopic sinus surgery techniques are being used to treat orbital complications such as malignant exophthalmos in thyroid eye disease.5 We believe that the key to avoiding such complications is the adequate understanding of the nasal anatomy endoscopically, which is only achieved through attending specialised workshops that are widely available throughout the country; adequate personal experience is cooperative in paramount importance. If complications are encountered then the immediate termination of the procedure is recommended and an urgent ophthalmological opinion should be sought.

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Posterior canal predominance in bilateral skew deviation

EDITOR,—We were excited to see the recent case report of Park et al regarding the 30 year old man with horizontal locked-in syndrome and disconjugate gaze.1 We were intrigued by the description of his eye movements on attempted horizontal gaze, whereas “when the patient was asked to look to the right side, the right eye moved upward with intorsion, and at the same time, left eye moved downward and extorsion . . . when the patient was asked to look to the left side, . . . the left eye moved upward with intorsion whereas the right eye moved downward with extorsion . . . Magnetic resonance imaging revealed a large ventral pontine infarct. The authors postulated that the lesion caused a disturbance in the neural integration of prurenal inputs to the interstitial nucleus of Cajal.

We believe we can refine further their mechanism for this observed disconjugate gaze based on the anatomy of the vestibular ocular reflect pathways, as it is probably a type of bilateral skew deviation. Each semicircular canal provides excitatory innervation to an extraocular muscle and its contralateral yoke, and inhibitory innervation to the corresponding antagonist extraocular muscles. The otolithic pathways are less well understood but are believed to follow the same pathways as the semicircular canal pathways. Each anterior semicircular canal provides excitatory innervation to the ipsilateral superior rectus and the contralateral inferior oblique muscle, while inhibiting the yoke ipsilateral inferior rectus and contralateral superior oblique muscle. Unilateral injury to these vestibular-ocular pathways produces classically skew deviation with hypertropia of one eye in all fields of gaze, whereas bilateral injury produces alternating hypertropia in side gaze. Bilateral damage to anterior canal pathways causes a posterior canal predominance with bilateral tonic downgaze.

Theoretically, bilateral damage to the otolith-ocular pathways corresponding to those of the anterior semicircular canal should produce the motility disturbance described in the patient reported by Park et al. The disinheritment resulting from such damage would produce posterior canal predominance, and increase torsus to all four depressors (both inferior recti and both superior obliques). Since the vertical action of the superior oblique is more prominent in adduction, the abducting eye should have a relative hypertropia on side gaze (alternating skew on lateral gaze). Likewise, because the torsional action of the superior oblique is more prominent in abduction, dynamic intorsional movements of the hypertropic eye would be seen on attempted adduction.

In this scenario, fundus examination should demonstrate bilateral intorsion in primary position, and detailed motility measurements would show an A-pattern. However, these findings would have been difficult to detect in this patient who could not elevate the eyes above the midline. We believe that bilateral injury to the same pathways may be responsible for A-pattern strabismus and bilateral superior oblique overaction seen in some patients with posterior fossa disease.

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