Letters to editor

National Physicians Survey 31.1% of ophthalmologists in Canada who responded indicated that they planned to reduce their weekly work hours in the next 2 years.3

Waiting times for ophthalmic care is felt to be a national problem. The federal and provincial governments recently transferred $5.5 billion to try to shorten wait times in problem areas, one of which is cataract surgery. Although this initiative is certainly shortening cataract waiting lists, it is also causing longer delays in many parts of the country for care for other vision-threatening conditions. Imagine what the public outcry will be if we maintain the training status quo and the ratio of ophthalmologists to patients over 65 drops from our current 1:4301 to 1:7576 in 2021.4

Finally, we agree with their concern about the uneven distribution of ophthalmologists across the country. If we don’t increase our supply of ophthalmologists, however, then we anticipate that the disparity may get even worse. If that were to happen, then governments may again try to legislate where physicians can practise, as they tried unsuccessfully many years ago in British Columbia. Therefore we feel that for the good of the country it is best if we increase the number of ophthalmology residency training positions.

REFERENCES


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doi: 10.3129/can j ophthalmol.07-053

Nature's shell game revealed: evidence for non-Newtonian laterality of macular vision (Ocular integration in the human visual cortex. Vol. 41 (5))

Dear Editor,

J.C. Horton’s defence of the Newtonian view of spatial representation in vision as one based on “sheer logic” is incorrect.1 The facts are that movement of electrical signals from one hemisphere of the brain to the other takes time and that the laterality of such transfers is governed by hard-wired callosal directionality of motor and sensory signals unique to each individual. We see this revealed by the “lead” of one hand on the other when a “simultaneous” button press is intended by the subject.2,3 Thus, for example, it takes longer for right-handed subjects to move their eyes to the left of the midline than it does to do the same to the right side and by an interval equal to the interhemispheric transfer time (IHTT).

Another manifestation of this same phenomenon (i.e., the laterality of macular vision) is the occurrence of macular sparing in lesions affecting the nondominant occipital lobe.4 Lesions affecting the visual cortex of the major hemisphere (the command centre, where macular vision occurs) are associated with cortical blindness, although documentation of this aspect is less voluminous that of lesions affecting the nondominant hemisphere (probably because of its association with visual agnosia).

It appears, therefore, that Nature has played an 8-way shell game with those engaged in understanding the laterality of vision in humans: 2 hemispheres and 4 kinds of laterality (2 neural and 2 behavioural).2,3

The most dramatic display of 1-way callosal traffic connectivity is the immediate remediation of “hemianopia” by using the nondominant hand in lesions affecting the minor hemisphere. According to the non-Newtonian model sketched here, this is the result of the excitatory influence of commands arising from the major hemisphere (left, in most people) when moving the left hand, which awakens the injured dormant (right) hemisphere into activity. Severing callosal connectivity between two hemispheres results in nondominant paralysis despite preservation of the motor cortex in the opposite (right) hemisphere.

The Newtonian doctrine of semi-decussation of the optic nerves at the chiasm has left many questions unanswered, specifically those related to Traquair’s anterior junction syndrome (central scotoma in one eye and temporal field loss invading the vertical meridian in the other) as well as those related to absence of callosal connectivity between the occipital lobes, as documented in cortical EEG recordings by the Chilean neurosurgeon Aranda and colleagues.”

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To sum, the evidence that macular vision is the province of the occipital lobe of the major hemisphere, as defined above, is overwhelming.2,3,5

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Acute hypertension due to phenylephrine eyedrops in a newborn

A 5-month-old girl, weighing 6.7 kg, experienced acute severe hypertension during general anaesthesia while undergoing treatment of a posterior synechia. Her past medical history revealed that she had been born by vaginal delivery at 40 weeks of amenorrhea. Her birth weight was 3280 g, and her Apgar score was 8 at 1 minute and 10 at 5 minutes. Consultation before anaesthesia showed a child in good health with a blood pressure of 85/55 mm Hg manually controlled.

Anaesthesia was induced by inhalation of sevoflurane. Alfentanil (180 µg) and atropine (0.125 mg) were injected intravenously before orotracheal intubation with a cannula number 4. Expiratory fraction of sevoflurane was between 4% and 5% to maintain anaesthesia.

For 30 minutes, systolic blood pressure was maintained between 70 and 90 mm Hg and the heart rate between 120 and 140 beats/min. Systolic blood pressure then suddenly increased to 180 mm Hg while the heart rate varied in the previous normal range. While we were checking the intubation, ventilator, and blood pressure device, the surgical procedure ended. We noticed a generalized redness of the child’s body that lasted 5 minutes. The child was awakened, and her systolic blood pressure slowly decreased to 95 mm Hg after 90 minutes and stayed in the normal range for 24 hours. Clinical examination revealed no neurologic or cardiac side effects. Results of an electrocardiogram were normal.

Investigation showed that the patient had received 6 drops of 5% phenylephrine (Neo-Synephrine). These drops were absorbed through the lachrymal duct and reached the blood circulation, where they exerted their systemic effects.

Shinomiya et al reported a case of renal failure in a low-birth-weight infant that was caused by instillation of eyedrops containing phenylephrine.1 Fraunfelder et al reported systemic effects with pledgets including 10% phenylephrine.2

In the literature, eyedrops of phenylephrine are not clearly forbidden in newborns. Nonetheless, we have removed phenylephrine eyedrops from our local protocol for both newborn and child.

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Giant cell arteritis presenting with hemianopic visual field loss

A 69-year-old woman presented with headache, dizziness, and jaw fatigue (especially while chewing). She specifically denied any scalp tenderness, and her past medical history was significant only for hypertension. Her erythrocyte sedimentation rate (ESR) was 54 mm/h, raising a suspicion of giant cell arteritis (GCA). She was started on prednisone therapy, but within a few days she became severely ill and nauseated. She developed episodic ataxia, weakness, and inability to appreciate objects in the left part of her visual field. Cranial magnetic resonance (MR) imaging, including diffusion-