

LETTER

Is the 'Act FAST' stroke campaign lobeist? The implications of including symptoms of occipital lobe and eye stroke in public education campaigns

The 'Act FAST' public education campaign was introduced to reduce the time to presentation of patients with stroke, and thereby increase the opportunity for thrombolysis and rapid initiation of secondary prevention. The campaign was launched in the UK in February 2009 and has subsequently been adopted in a number of other countries, including the USA and Australia. One significant limitation of the 'Act FAST' campaign, however, is that the acronym excludes any reference to the symptoms of sudden visual loss, which may be a manifestation of embolic disease either in the anterior or posterior circulation. One solution would be to modify the acronym to include vision: 'Act VFAST' (act very fast).

FAST has been shown to have good diagnostic sensitivity (>75%) when used by ambulance staff,¹ and other authors have suggested alternative acronyms to further increase this sensitivity.² However, these study designs involve patients who have already been presented to hospital and it is therefore not possible to determine false-negative rates: in the case of vision, it is not known how many patients with embolic visual changes do not make it to acute services for review. If patients with vision loss are not aware that this can be a symptom of stroke, they may not present in the first place. One potential way to improve this is to increase recognition within the community that vision loss can be a manifestation of stroke. Interestingly, there is also evidence that when matching descriptions of stroke symptoms to patient experience of those symptoms, 'loss of vision' or 'sudden decrease in vision' had much higher rates of patients identifying that this description exactly matched their experience.²

Visual symptoms from embolic disease may manifest through occlusion of the retinal or posterior circulation vessels: 5–10% of ischaemic strokes in the USA involve the posterior cerebral artery (PCA) or its branches.³ A population-based study on all types of TIA using active ascertainment of the secondary

event found the risk of subsequent stroke was 9.9% at 2 days and 13.4% at 30 days.⁴ Further, the subset of patients with visual manifestations of embolic disease experience a longer delay in diagnosis: this often precludes thrombolysis and delays the initiation of secondary prevention.⁵ This is in part because screening tools used in emergency departments are less sensitive for posterior circulation than anterior circulation events,⁶ but also because poor public awareness of the visual manifestations of stroke results in patients being less likely to present urgently.⁷

Binocular visual loss from embolic disease relates to occlusion of the posterior circulation; although mortality from PCA stroke is low, morbidity is high. Cortical blindness is as disabling as any other cause of blindness, and hemifield visual field loss often precludes driving and may significantly impair reading, activities that are strong influences on independence and quality of life.⁸ Unless patients are aware that sudden visual loss can be caused by stroke, the delay in presentation will preclude thrombolytic treatment, and yet evidence is now emerging that those with posterior circulation strokes benefit from this treatment.^{9 10}

Monocular visual change from embolic disease relates to occlusion of the retinal arteries. While there is limited evidence for thrombolysis for this indication,¹¹ early investigation and diagnosis would allow prompt initiation of secondary prevention. This is particularly important given that patients with visual symptoms are more likely to experience a delay in time to initiation of antithrombotic therapy compared with patients with hemispheric TIA.¹²

Early diagnosis would also minimise the delay to carotid endarterectomy (CEA) when this is deemed appropriate. Large RCTs have confirmed the benefit of CEA in patients with stroke and TIA and high-grade carotid stenosis,¹³ with the caveat that subgroup analysis suggests individuals enrolled for retinal TIA have a lower 3-year risk of ipsilateral stroke.¹⁴ The decision about surgery involves balancing the risks associated with surgery (5.8% of perioperative stroke in good surgical centres, including 2% risk of major stroke and 0.6% death)¹³ against those of medical management alone (10% risk of ipsilateral stroke at 3 years).¹⁴ There is some suggestion that risk stratification may be useful in deciding which patients are most likely to benefit from surgical intervention.¹⁴

The most appropriate timing of CEA is also contentious, however there is evidence that the net benefit may favour early intervention: although early CEA confers an increased risk of perioperative complication, if CEA is delayed beyond 12 weeks in symptomatic patients with North American Symptomatic Carotid Endarterectomy Trial (NASCET) 50–99% stenosis, there is little benefit to the patient in terms of long-term stroke prevention.¹⁵

For patients with visual symptoms that may be embolic, stroke and TIA services are already set up to provide rapid assessment and intervention if required. Many are open 7 days per week, and have rapid access to relevant investigations. Most importantly this means imaging the carotids, but it also includes ECG (and its interpretation) and echocardiography. When intervention is required, they are set up to provide vascular secondary prevention, including anticoagulation where appropriate, lifestyle advice and they then have links to vascular surgery departments for patients who need prompt endarterectomy. Stroke and TIA services therefore include a well-resourced infrastructure that has almost everything that is needed for these patients.

Broadening the message to 'Act VFAST' would potentially come with some costs. As was the case with the initiation of the FAST campaign, extending to VFAST would not only increase the number of referrals of patients with stroke, but also those with stroke mimics.

Differentiating strokes from their mimics is, however, already a key role for stroke services: the existing process triages patients with Bell's palsy, dysphasic migraine and functional weakness. Differentiating embolic from non-embolic visual conditions would therefore be a natural extension. The most common visual mimic of embolic transient visual loss would be migraine, which may be perceived as either monocular or binocular, and will be identified from the history. In the case of persistent monocular visual loss, most mimics of central retinal artery occlusion are evident on direct ophthalmoscopy: anterior ischaemic optic neuropathy and retinal vein occlusions both show signs of a swollen optic disc, and retinal detachment affecting the central vision entails a markedly abnormal fundus. Diagnosing each of these conditions early is still of benefit to patients, who would then receive prompt condition-appropriate treatment. Close collaboration with ophthalmology services throughout this process would be imperative.

There will also be a concern as to the appropriateness of visual referrals to acute stroke units and the potential for these services to be overwhelmed. It may be necessary to first assess the benefit of improved opportunities for thrombolysis and timely TIA work up against the cost of additional inappropriate referrals. This could be achieved by trialling the VFAST campaign in a defined geographical location, as was the case with the initial FAST campaign.¹

Much of the current delay in initiation of appropriate treatment in patients with visual loss due to embolic disease relates to the delay in being seen by an appropriate clinician. Visual symptoms may herald embolic disease and the only way to create the possibility of rapid assessment and appropriate treatment is to make the general public aware of this. Instead of encouraging patients to 'Act FAST', we should be encouraging them to 'Act VFAST'.

Mitchell Lawlor,^{1,2} Richard Perry,^{3,4} Gordon T Plant^{1,4}

¹Moorfields Eye Hospital, London, UK

²Save Sight Institute, University of Sydney, Sydney, New South Wales, Australia.

³Hyperacute Stroke Unit, UCLH, London, UK

⁴The National Hospital for Neurology and Neurosurgery, London, UK

Correspondence to Dr Mitchell Lawlor, Department of Neuro-Ophthalmology, Moorfields Eye Hospital, 162 City Road, London EC1V 2PD, UK; mitchell.lawlor@moorfields.nhs.uk

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