glaucomatous change (StratusOCT was introduced in 2001 and glaucoma, specially when treated, is a slowly progressing disease). Furthermore, no progression algorithms are currently available for OCT beyond linear regression analysis of changes in measurements over time. The difference between clinically significant change and short-term and/or long-term measurement variability for OCT measurements has not been determined, although arbitrary cut-offs based on image resolution have been suggested.¹² Other imaging technologies have incorporated sophisticated progression analyses that are yet to be incorporated in the OCT (to be fair, these algorithms developed by other manufacturers have not been well tested). Finally, it is not clear whether the current image registration techniques used by OCT will allow sensitive detection of change.

Overall, although OCT can clearly identify thinner RNFLs in glaucomatous eyes compared with healthy eyes, the usefulness of the StratusOCT normative database has not been well established in patients suspected of having glaucoma (ie, the population of most interest). In addition, the ability of OCT measurements to detect glaucomatous change has not been demonstrated. Cutting-edge advances in OCT technology^{13–16} such as high-resolution spectral/Fourier domain OCT and OCT-confocal scanning laser hybrids are theoretically promising (and exciting), but it will be

some time before these technologies have been scrutinised to the point that their clinical use can be recommended by evidence-based studies. In general, it is important that OCT and all imaging technologies be used in conjunction with careful clinical examination when making diagnostic and treatment decisions for glaucoma.

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VIDEO REPORT

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Apraxia of lid opening

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ABSTRACT

We describe eyelid movement abnormalities in an 80-year-old man with apraxia of lid opening (ALO), resulting from involuntary levator palpebrae inhibition (ILPI) and pretarsal orbicularis oculi (OO) contraction. He was unable to open his lids at will following closure. Attempted eye opening resulted in forceful contraction of the frontalis muscle, backward thrusting of the head and lengthened lid closure. The inability to reopen the lids was not evident during spontaneous reflex blinking and he had no difficulty in keeping the lids open once they had been manually lifted up. There were no episodes of involuntary drooping of the eyelids or spasmodic contraction of the OO causing involuntary eyelid closure. Pursuit eye movements were not restricted, the vestibulo-ocular reflex was preserved and both horizontal and vertical saccades were normal. Despite the clinically visible persistence of pretarsal OO activity, treatment with botulinum toxin injections in the pretarsal and preseptal portions of the muscle did not reduce his difficulty in initiating lid elevation but he found some benefit using lid crutches. ALO is thought to be due to an abnormality in the supranuclear control of eyelid movement. ILPI can present either isolated or combined with blepharospasm. The excitatory levator palpebrae response necessary to lift the lids up is likely to be in very close connection with the OO antagonistic inhibitory response. Alterations in one or another pre-motor structure may result in inability to raise the lids due to inhibition of the levator palpebrae as well as persistence of the pretarsal OO.

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