Scand 2011;124(Suppl 192):83-91). Field defects are bilateral and more pronounced nasally. Reported estimates of pVFDs are 30-50% in adults and 20% in children. Risk increases with increased dosage and duration of therapy. Vision screening is recommended at baseline, every 3 months, and at 3-6 months after discontinuation of therapy.

SEIZURE-INDUCED MIOSIS

Researchers at University of Southampton, UK report an infant with focal seizures secondary to cortical dysplasia who presented at age 4 months with bilateral pinpoint pupils as first feature of right-sided clonic seizure. EEG recording during seizures showed ictal spiking over the left central region. ECG did not show bradycardia, and there were no signs of parasympathetic activation. MRI, obtained after relapse at 13 months of age with 30 seizures daily involving the right upper limb, revealed left frontal cortical dysplasia. A cortical electrographic grid and intracranial EEG demonstrated ictal fast spike activity within the left centroparietal region, over the middle parietal gyrus. The irritative zone was located at the margin and across the area of dysplasia. The child remained seizure-free at 3 years following excision of the lesion. The origin of ictal miosis is considered secondary to activation of a cortical pupillary constrictor zone as opposed to activation of the parasympathetic nervous system through the subcortical central autonomic network, usually resulting in mydriasis. (Sadek A-R, Kirkham F, Barker S, Gray WP, Allen D. Seizure-induced miosis. Epilepsia Dec 2011;52(12):e199e203). (Respond: Dr Ahmed-Ramadan Sadek, Division of Clinical Neurosciences, School of Medicine, University of Southampton, Tremona Road, Southampton SO16 6YD, UK. E-mail: a.sadek@soton.ac.uk).

COMMENT. Bilateral pupillary dilatation preceding, during, or after generalized convulsive or nonconvulsive seizures is common, but ictal pupillary miosis is rare. Autonomic disturbances during seizures (alterations in heart rate, blood pressure, gastrointestinal function) result from seizures within the largely subcortical and brainstem central autonomic network. The UK study demonstrates ictal miosis originating in the middle parietal gyrus juxtaposed by the superior anastomatic vein of Trolard, within the cortical central parietal region.

MITOCHONDRIAL HYPERMETABOLIC NEURONS IN EPILEPSY

Researchers at the University of Calgary and Alberta Children's Hospital, Canada studied histochemically frozen sections of 10 brain resections from 7 epileptic children (ages 2 months to 17 years). None had mitochondrial disease; 1 had tuberous sclerosis and 2 hemimegalencephaly. Preop EEG and intra-operative ECoG defined foci refractory to AEDs. Increased mitochondrial respiratory chain enzymes were demonstrated in individual neurons of hippocampal and neocortex epileptic foci. These intensely staining neurons may indicate functional hypermetabolism and represent a metabolic marker of an epileptic focus. Alpha-B-crystallin was also strongly reactive, demarcating the epileptic focus as a wide field. (Sarnat HB, Flores-Sarnat L, Hader W, Bello-Espinosa L. Mitochondrial "hypermetabolic" neurons in paediatric epileptic foci. **Can J Neurol Sci**