## Effect of epilepsy and anticonvulsants on pregnancy outcome

### Neuropsychological tests in children

Vinten et al. studied the effect of exposure to antiepileptic drugs in utero in 249 children using neuropsychological tests. Sodium valproate and frequent tonic-clonic seizures during pregnancy carried a potential risk for developmental delay and cognitive impairment.

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### Lamotrigine exposure

Cunnington et al. reported results from the International Lamotrigine Pregnancy Registry. In this 12-year observational study with outcomes of 414 first trimester monotherapy exposures, the risk of major birth defect was 2.9% (95% CI; 1.6 to 5.1%) and similar to the general population.

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### Valproic acid exposure

Wyszynski et al. prospectively studied 149 women taking the antiepileptic drug valproic acid (VPA) as monotherapy during pregnancy. Sixteen children with major malformations were found among the offspring of these women. A four-fold risk was found when they compared this proportion to that of women who used other antiepileptic drugs. Compared to the general population, the risk of having an affected offspring among VPA-exposed women was 7.3-fold.

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### Editorial perspective

*The editorial by Penovich and Gaily accompanying these three papers notes that the results from lamotrigine monotherapy-exposed pregnancies are not alarming and may be somewhat reassuring; but as the authors note, the sample size is too small for definite conclusions. The data support both structural and functional teratogenicity of VPA. However, as the choice of drug is dependent on epilepsy syndrome, there is a possibility that the results from prenatal VPA exposure may be not wholly due to VPA exposure, but may be in part associated with some maternal characteristic related to the epilepsy syndrome. Moreover, none of the three studies provide antiepileptic drug serum concentrations or doses used at conception or during the first trimester to evaluate for a dose-response effect. None provide a pathophysiological explanation for the disturbance in fetal development. Without good animal models for human teratogenicity, the mechanism for the abnormalities remains unknown.*

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## Mitochondrial DNA content in autosomal dominant optic atrophy (ADOA)

Studying patients with ADOA, Kim et al. investigated the alterations of mitochondrial content caused by *OPA1* mutations. Mitochondrial DNA content was attenuated due to mitochondrial damage supporting a pathogenic role of *OPA1* mutations.

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### Optic mitochondriopathies

*The accompanying editorial by Chinnery and Griffiths notes that a visual field defect that affects the macula and the papillomacular bundle—a cecocentral scotoma—points towards an optic neuropathy. Reviewing the hereditary and acquired causes of optic neuropathy, they also note that autosomal dominant optic atrophy or Kjer optic atrophy usually presents in childhood and progresses slowly and symmetrically with retinal ganglion cell loss from the papillomacular bundle. Some families with ADOA have mutations in the* OPA1 *gene. Down regulation of* OPA1 *expression leads to a fragmentation of the normal mitochondrial network, a decrease in the mitochondrial membrane potential, and the release of cytochrome* c *from mitochondria triggering caspase-dependent activation of apoptotis. Kim et al. show that patients with* OPA1 *mutations have a small but significant reduction in the amount of mitochondrial DNA (mtDNA) in their peripheral blood lymphocytes. Some would argue that such a small change would not have a major effect, so that the bioenergetic consequences of the subtle decrease in mtDNA copy number needs to be established.*

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## Intractable seizures after discontinuing AEDs

Peter and Carol Camfield report the long-term follow up of a Nova Scotia population-based epilepsy cohort in which 260 children became seizure free and attempted to discontinue daily medication treatment. Only three of the 260 (<1%) subsequently developed intractable epilepsy that could not be controlled with further medications.

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## Vasoactive medications and SAH-related vasospasm

In this retrospective cohort study, Singhal et al. found that the use of statins and selective serotonin reuptake inhibitors was associated with an increased risk of developing vasospasm after aneurysmal subarachnoid hemorrhage (SAH).

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*continued on page 937*

 

## Posterior thalamic hemorrhage induces "pusher syndrome"

Hemiparetic stroke patients with contraversive pushing use the nonaffected arm or leg to push away actively from the nonparalyzed side. Observing 40 patients with thalamic stroke, Karnath et al. found the disorder typically was caused by hemorrhage (in contrast to infarction) located in the posterior thalamus (in contrast to anterior thalamic lesions).

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## The risk of stroke in migraineurs

In a prospective cohort study of 39,754, women, Kurth et al. found no association between overall migraine, migraine without aura, or non-migraine headache and stroke. However, migraine with aura was associated with a 1.5-fold increase in risk of ischemic stroke.

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## Facioscapulohumeral muscular dystrophy (FSHD) and exercise

Olsen et al. demonstrated a beneficial effect of a 12-week course of aerobic exercise in patients with FSHD.

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## Seizures in MS patients treated with intrathecal baclofen

Schuele et al. observed a higher incidence of epileptic seizures in patients with multiple sclerosis (MS) treated with intrathecal baclofen vs a matched control group (7% vs 1%, $p < 0.05$). Seizures were often temporally related to other factors: fear, baclofen overdose, and surgery.

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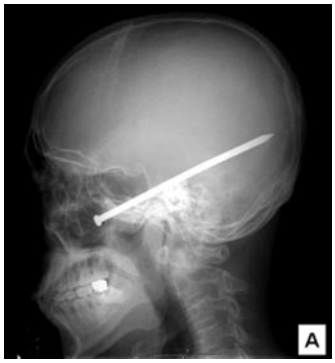
## Lost nail




Seo and Chang report a patient who presented with a 2-year history of seizures after having "lost" a nail 22 years earlier.

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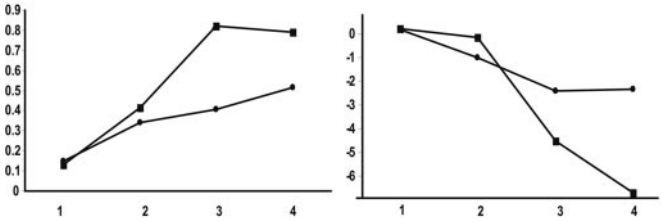
## Vincristine-induced peripheral neuropathy: Worsening after stopping treatment




*Vincistine therapy: high vs low dose. (Left) Log change in vibration perception. (Right) Group strength.*

Verstappen et al. treated 114 patients with malignant lymphoma with chemotherapy regimens including vincristine in two different dose-intensities. Vincristine caused a dose-dependent peripheral neuropathy. Progressive worsening after discontinuing vincristine was seen in nearly a quarter of patients.

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## Stenting for symptomatic basilar artery stenosis

Yu et al. reviewed long-term outcome of stenting for basilar artery stenosis. In this retrospective and uncontrolled study stenting appeared to reduce stroke risk and death. A randomized trial is needed.

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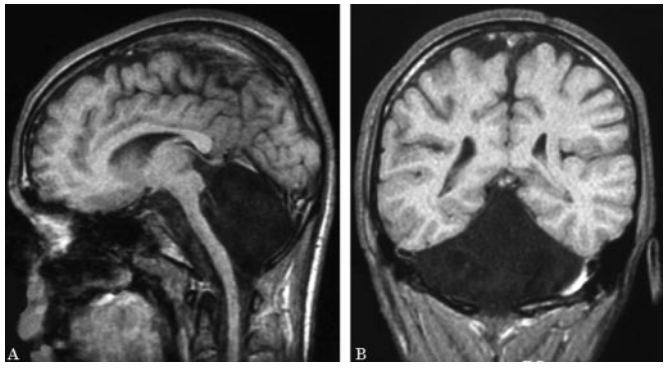
## Cerebellar agenesis




This online-only Neuro*Image* by Titomanlio et al. has a video. A patient without a cerebellum has remarkably little defect.

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