may provide a useful means of screening for Fabry disease.

Original article Gupta SN *et al.* (2008) Skin-impedance in Fabry disease: a prospective, controlled, non-randomized clinical study. *BMC Neurol* **8:** 41

Postnatal administration of anticonvulsant and sedative agents affects neurogenesis

The γ-aminobutyric acid subtype A ($GABA_A$) receptor agonists phenobarbital and diazepam, used as sedatives or anticonvulsants in newborn babies, and the *N*-methyl-D-aspartate (NMDA) receptor antagonist MK801 suppress early postnatal neurogenesis, a new study has revealed.

Stefovska *et al.* studied the effects of MK801, phenobarbital and diazepam on cell proliferation and neurogenesis in neonatal rat brains during the first three postnatal weeks. Wistar rat neonates were injected either with one of these agents or with saline solution (control); neurogenesis and cell proliferation were assessed by means of cell-type-specific markers and a compound that labels DNA during cell division.

Compared with the brains of the control rats, the numbers of proliferating cells were reduced in rat brains after treatment with MK801, phenobarbital or diazepam; the greatest reduction was observed in rats treated with MK801. Numbers of new cells in the brain were also reduced following administration of the NMDA antagonist or $GABA_A$ agonists. This effect was not, however, a result of apoptosis; the authors instead suggested inhibition of proliferation as the causal factor. Postnatal neurogenesis, confined to the dentate gyrus in the brains of the study rats, was inhibited in the rats treated with MK801 and phenobarbital. Rats aged 6 months that previously received postnatal phenobarbital had impaired learning and reduced memory capacity in comparison with their saline-treated counterparts.

In light of these results, Stefovska and colleagues suggest that caution should be used when considering treatment with NMDA antagonists or $GABA_A$ agonists in obstetric, neonatal or pediatric patients.

Original article Stefovska VG *et al.* (2008) Sedative and anticonvulsant drugs suppress postnatal neurogenesis. *Ann Neurol* **64:** 434–445

Gene variants linked to atrial fibrillation are also associated with risk of ischemic stroke

Gretarsdottir and colleagues have found that gene sequence variants previously linked to risk of atrial fibrillation are also associated with ischemic stroke.

To identify single nucleotide polymorphisms (SNPs) associated with ischemic stroke, a genome-wide study of an Icelandic population was carried out, in which 1,661 patients with ischemic stroke and 10,815 individuals free of cardiovascular disease (controls) were genotyped. A total of 310,881 SNPs were tested for association with overall ischemic stroke and with the three main phenotypes of ischemic stroke—large-artery atherosclerosis, cardioembolic stroke, and small-vessel disease. The top 120 SNPs for each phenotype tested were selected for replication in two European study groups. Further replication of two of these SNPs that have previously been associated with atrial fibrillation (rs2200733 and its neighbor rs10033464, both on chromosome 4q25) was performed in two additional European study groups.

Both of the SNPs were found to be strongly associated with cardioembolic stroke, a finding that the authors explain is reasonable, as cardioembolic stroke is often a complication of atrial fibrillation. The rs2200733 SNP was also linked to overall risk of stroke; this relationship suggests that some patients might be misdiagnosed with noncardiogenic stroke as a result of undiagnosed atrial fibrillation.

Detection of gene-sequence variants associated with atrial fibrillation risk could help in the detection of stroke subtypes and might allow preventative treatment to be carried out, Gretarsdottir *et al.* conclude.

Original article Gretarsdottir S *et al.* (2008) Risk variants for atrial fibrillation on chromosome 4q25 associate with ischemic stroke. *Ann Neurol* **64:** 402–409

Pediatric and adult glioblastoma have different gene expression profiles

A study conducted in India has found that pediatric glioblastoma multiforme (GBM) has a genetic expression profile distinct from that of its adult counterpart.