specificity and 100% sensitivity). Cross-validation tests confirmed that the functions were accurate in their classifications.

A tool such as this, which helps to diagnose probable AD at an early stage, could aid early intervention and also improve understanding of the various aspects of brain function.

*Pippa Murdie*

**Original article** Chapman RM *et al.* (2006) Brain event-related potentials: diagnosing early-stage Alzheimer's disease. *Neurobiol Aging* [doi: 10.1016/j.neurobiolaging.2005.12.008]

## Link between newborn encephalopathy and autism spectrum disorders?

Researchers have reported a possible link between newborn encephalopathy (NE) and autism spectrum disorders (ASDs) such as autism and Asperger's syndrome.

This population-based study in Western Australia enrolled 276 full-term infants with moderate to severe NE and 564 randomly selected full-term controls. Sociodemographic, IQ, behavioral, language and neurodevelopmental data on each participant were collected by questionnaires and medical records at enrollment, and by a series of tests at 3 years and 5 years of age. Participants who survived for at least 5 years were included in the subsequent analyses.

Children with NE were 5.9 times more likely to be diagnosed with an ASD than were controls—12/239 children with NE and 5/563 controls were diagnosed with an ASD. Children with NE and an ASD were more likely than controls to have a history of bleeding, trauma or infection *in utero* (odds ratio 4.7), and were also more likely to have had a birth defect diagnosed at 2 years of age (odds ratio 11.2).

The authors state that children with a history of NE should be considered to be at risk of experiencing an ASD, and should be followed up appropriately to enable early intervention if necessary. The association between NE and ASDs might be attributable to common risk factors between these conditions or a shared underlying defect in fetal neurodevelopment, or, alternatively, NE might cause or be the first sign of an ASD in newborn children.

*Rebecca Ireland*

**Original article** Badawi N *et al.* (2006) Autism following a history of newborn encephalopathy: more than a coincidence? *Dev Med Child Neurol* **48:** 85–89

## Gefitinib treatment for brain metastases in patients with *EGFR* gene mutations

Gefitinib—a tyrosine kinase inhibitor specific for the epidermal growth factor receptor (EGFR)—might have antitumor activity in non-small-cell lung carcinoma (NSCLC) metastases to the brain, and the effectiveness of this treatment might be related to mutations in the *EGFR* gene. Shimato *et al*. investigated the relationship between the presence of *EGFR* mutations and the response to gefitinib of patients with brain metastases.

In this retrospective study, the brain tumor response of eight patients who were treated with gefitinib for NSCLC brain metastases was evaluated by MRI. *EGFR* gene mutations were assessed by gene sequencing. After considering the effect of earlier radiotherapy on metastases, the response of patients to gefitinib was assessed. Three patients had an effective objective response and three patients had no objective response; efficacy was not assessable in the remaining two patients because of a continued response from the completion of radiotherapy through to gefitinib treatment. Objective responses lasted 4–18 months and *EGFR* mutations were identified in all patients with effective objective responses, but not in any of those without objective responses ($P = 0.036$).

The authors conclude that there might be a relationship between *EGFR* mutations and the efficacy of gefitinib treatment in NSCLC patients with brain metastases, and that this could explain why gefitinib treatment is effective in only a subgroup of patients. Further, prospective trials are needed to test whether *EGFR* mutations can predict response of brain metastases to gefitinib.

*Kate Matthews*

**Original article** Shimato S *et al.* (2006) *EGFR* mutations in patients with brain metastases from lung cancer: association with the efficacy of gefitinib. *Neuro-oncol* [doi: 10.1215/15228517-2005-002]

## Genetic polymorphism affects timing of levodopa-induced dyskinesias in PD

Dyskinesias, a common complication of levodopa therapy administered for Parkinson's