Migraine: Impact on quality of life (QOL)

Two articles study large populations (rather than patients referred to specialists) to assess how migraine affects the general population.

Terwindt et al. (p. 624) studied 6491 Dutch adults. Ten percent had migraines; over half had not been diagnosed by a physician. Both asthma and musculoskeletal pain were more frequent in migraineurs. Migraine had a greater deleterious effect on QOL than did asthma.

◆ Lipton et al. (p. 629) studied US and UK populations for the occurrence of migraine and depression, assessing the effect of both on QOL. Migraine and depression frequently coexisted and both had a major deleterious effect on QOL. ◆ The accompanying Editorial by Becker and Ware (who developed the RAND 36—the gold standard QOL instrument) (p. 610) reviews how QOL is measured and why it is of great importance as new treatments are pursued.

Essential blepharospasm: PET localization

Hutchinson et al. (p. 673) previously used PET to define brain regions of abnormal metabolic activity in idiopathic torsion dystonia (ITD). Here, in essential blepharospasm (EB), they find abnormally increased metabolic activity in the pons and cerebellum and hypometabolism during sleep in frontal eyelid control regions. Patients with EB also shared some abnormalities with patients with ITD.

Hydrocarbon solvent exposure a risk factor for PD

Pezzoli et al. (p. 667) used case-control methods to study a 990 patient cohort with PD. Solvent

(e.g., n-hexane, toluene, trichloroethylene, trichloroethane) exposure correlated with disease severity and earlier onset of disease. Exposed patients required higher dosages of levodopa and had a reduced response to apomorphine.

Alpha-2 macroglobulin (A2M) gene in AD

There are a number of simple gene mutations that cause early onset AD and a growing number of genes associated with an increased incidence of AD. The A2M gene is a logical gene because it codes for a protease inhibitor that influences β -amyloid in AD. Polymorphisms of A2M have been linked to AD in earlier studies. Koster et al. (p. 678) studied a large population of patients with late-onset AD and did *not* find an association. Meta-analysis of studies to January 2000 also rejected an association.

Duchenne dystrophy (DD): Which method of ventilatory support is best?

Nomori and Ishihara (p. 698) report a new technique using a small tube (mini-tracheostomy) for respiratory support of patients with DD. They document the success of the method in a small number of patients with DD as well as in MG. Three Editorials comment on the article and the general problem. ◆ Reynolds and Mendell (p. 611) discuss the mini-tracheostomy (and their own) experience to support invasive approaches. ◆ Bach (p. 613) argues from his extensive (although uncontrolled) experience that non-invasive methods are superior. ◆ The Editor (p. 615) concludes that the matter is unsettled. Because there is clinical equipoise, patients deserve

controlled studies to determine which approach is best.

Hereditary spastic paraplegia (HSP)

There are at least seven different genetic loci for autosomal dominant HSP. *SPG4* codes for the protein spastin and is mutated in approximately 40% of patients with HSP. Santorelli et al. (p. 702) report an *SPG4* mutation in a kindred with remarkable diversity in age at onset and rate of progression of signs and symptoms.

IV valproate in patients with hypotension and status epilepticus

Sinha and Naritoku (p. 722) reviewed records of 13 patients, mostly elderly, who had status epilepticus complicated by hypotension. IV valproate was well-tolerated and did not change pulse or lower blood pressure.

Evaluation of first nonfebrile seizure in childhood

All AAN-approved papers are now peer-reviewed as are all *Neurology* articles. The Hirtz et al. (p. 616) practice parameter provides guidelines for diagnostic studies and summarizes the important areas needing further study. EEG is indicated; MRI is recommended in selected specific situations. Further studies are needed to establish the instances where MRI influences treatment sufficiently to be necessary.

A recessive gene causing AD?

Bowirrat et al. (p. 731) report an unusually high frequency of AD in an Arab population with a very high prevalence of dementia. The frequency is not due to an increase in *APOE* $\epsilon 4$. A recessive gene is likely to be responsible for the disorder—thus far, no recessive genes causing AD have been identified.

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