(11.1%) were ascribed to increased intracranial pressure, 23 (10.2%) to trauma, 14 (6.2%) an infectious etiology, 10 (4.4%) to vascular disease, 9 (4%) inflammatory disorders, 6 (2.7%) were congenital, 2 (0.9%) secondary to surgery unrelated to neoplasm, and 1 (0.4%) to radiation necrosis.

Benign sixth nerve palsy and recurrent cases had the following characteristics: a) isolated unilateral abduction palsy; b) without ptosis, papilledema or other neurologic signs; c) normal brain MRI; d) spontaneous improvement; e) without infection, inflammatory or other disease identified. The mean age at evaluation of the 30 (13.3%) benign cases was 3 years. MRIs performed in 28 (93%), and lumbar punctures in 6 were normal, Acetylcholine receptor antibody testing was negative in 11 (37%), and lyme antibody titres, and anti-Gq1b antibody testing were normal. Only one child had some residual abduction deficit at 3-year follow-up. Four patients, including 3 with recurrences, had residual esotropia but full ductions, and all were referred for strabismus surgery. (Mahoney NR, Liu GT. Benign recurrent sixth (abducens) nerve palsies in children. **Arch Dis Child** May 2009;94:394-396). (Respond: Dr Nicholas R Mahoney, Scheie Eye Institute, 51 North 39th Street, Philadelphia, PA 19104. E-mail: nicholas.mahoney@uphs.upenn.edu).

COMMENT. Proposed etiologies for benign sixth nerve palsies include ophthalmoplegic migraine, myasthenia gravis, and inflammation secondary to viral infections or vaccination (Lee MS, 1999). Age and gender are important, the age being younger in the recurrent cases, and girls are affected more frequently than boys. Cases related to vaccination are also prone to recurrence. (Yousuf et al, 2007). The cases in the above study were idiopathic.

METABOLIC DISORDERS

VOLTAGE SENSORS IN HYPOKALEMIC PERIODIC PARALYSIS

Researchers at the National Hospital, Queen Square, London, UK, conducted automated DNA sequencing of the S4 regions of *CACNA1S* and *SCN4A* in 83 patients with hypokalemic periodic paralysis (HypoPP). CACNA1S mutations were identified in 64 cases, and SCN4A or other CACNA1S mutations in 10, including 4 with new mutations. All mutations neutralized arginine residues in S4 segments. The patients with new mutations had the typical HypoPP phenotype: onset of attacks of muscle paralysis in first or second decade, at night or early morning, and low serum potassium. The findings were consistent with the gating pore cation leak hypothesis of HypoPP, and arginine mutations in S4 segments are involved in 90% cases. (Matthews E, Labrum R, Sweeny MG, et al. Voltage sensor charge loss accounts for most cases of hypokalemic periodic paralysis. **Neurology** May 5, 2009;72:1544-1547). (Response and reprints: Prof MG Hanna, Institute of Neurology and National Hospital for Neurology and Neurosurgery, Queen Square, London, WC1N 3BG, UK. E-mail: m.hanna@ion.ucl.ac.uk).

COMMENT. In an editorial, Cannon SC proposes that the remaining 10% of HypoPP families with no identified mutation will also prove to be channelopathies, from a new class of molecular defect or different channel. (**Neurology** 2009;72:1540-1541).