supratentorial), 4 neuroectodermal, 2 teratoid rhabdoid, 2 choroid plexus, 1 meningioma, and 1 teratoma. Median age of presentation differed by lesion location, but not duration of symptoms. Raised intracranial pressure was more than twice as prevalent with posterior lesions and increased head circumference. Seizures occurred in 9 (50%); the tumor was supratentorial in 67% and infratentorial in 17% (p=0.04). Torticollis occurred in 4 (67%) of infratentorial and none of supratentorial tumors (p<0.01). Total resection was performed in 47%, and CSF shunt was more frequent with infratentorial tumor. Adjuvant chemotherapy was given in 44%, and radiotherapy in 17%, mainly in infratentorial tumors. Eight survived, 7 with supratentorial tumor, 5 to adulthood. Six are functionally independent. (Mehrotra N, Shanji MF, Vassilyadi M, Ventureyra ECG. Intracranial tumors in first year of life: the CHEO experience. **Childs Nerv Syst** Dec 2009;25:1563-1569). (Respond: Dr Michael Vassilyadi, Division of Neurosurgery, The Ottawa Hospital, Ottawa, Canada. E-mail: vassilyadi@cheo.on.ca).

COMMENT. In this young age group (<1 year of age), seizures occurred in 50% of patients, mainly with supratentorial tumors. In a study of 291 children with intracranial tumors treated at the Mayo Clinic over a ten-year period, seizures occurred in 17%; the tumor was supratentorial in 62% and infratentorial in 38%. Average age at seizure onset and at diagnosis was 4.9 and 6.7 years, respectively, in patients with supratentorial, and 4.8 and 5.1 years in those with infratentorial tumors. EEG was of localizing value in 75% of supratentorial tumors (88% of cortical tumors). A generalized dysrhythmia, maximal in the occipital regions and compatible with a lesion in the posterior fossa, was present in 44% of patients with infratentorial tumor. A delta pattern, indicative of an expanding lesion, occurred in 57% patients. (Millichap JG et al. The electroencephalogram in children with intracranial tumors and seizures. **Neurology** 1962;12:329-336).

NEUROMUSCULAR DISORDERS

ASCORBIC ACID IN CHARCOT-MARIE-TOOTH DISEASE

Ascorbic acid has been shown to reduce demyelination and improve muscle function in a transgenic mouse model of Charcot-Marie-Tooth disease (CMT1A). Aberrant expression of the myelin protein 22 gene, PMP22 is the cause of CMT1A, and large doses of ascorbic acid are shown to inhibit cAMP-mediated stimulation of human PMP22 expression. A 12-month, randomized, double-blind, placebo-controlled study of ascorbic acid in 117 adult patients compared to 62 receiving placebo found no significant difference between groups in neuropathy scores. Doses of ascorbic acid were 1 g and 3 g daily. The occurrence of adverse events did not differ between groups. (Micallef J, Attarian S, Dubourg O, et al. Effect of ascorbic acid in patients with Charcot-Marie-Tooth disease type 1A: a multicentre, randomized, double-blind, placebo-controlled trial. **Lancet Neurology** Dec 2009;8:1103-1110). (Respond: Dr Olivier Blin, CHU La Timone, Marseille, France.E-mail: Olivier.blin@ap-hm.fr).

COMMENT. Similar negative results were obtained in a placebo-controlled trial of ascorbic acid (30 mg/kg/day) in 81 children with CMT1A (2-16 years of age). (Burns J, Ouvrier RA, Yiu EM, et al. **Lancet Neurol 2009;**8(6):537-544).