INTERLEUKIN-6 CSF LEVELS were increased in 20 infants dying of SIDS in a study reported from the Institute of Forensic Medicine, National Hospital, Oslo, Norway. (Vege A et al. Acta Paediatr Feb 1995;84:193-6). The authors suggest that immune activation plays a role in SIDS, and cytokines in the CNS may cause respiratory depression in vulnerable infants.

An increased postneonatal mortality in lower social groups was explained by an association with SIDS in a study from the Department of Epidemiology, National Institute of Public Health, Oslo, Norway. (Arntzen A et al. Acta Paediatr Feb 1995;84:188-92).

A series of articles and an editorial in a recent issue of JAMA address the roles of sleeping position and passive smoking and tobacco exposure through breast milk in the etiology of SIDS. A major factor relating to a decline in SIDS in Tasmania was a reduction in the prevalence of prone sleeping position of infants. (Dwyer T et al. JAMA March 8, 1995;273:783-789). In contrast, routine prone sleeping position was not associated with an increased risk of SIDS in a Southern California study population. (Klonoff-Cohen HS, Edelstein SH. JAMA 1995;273:790-794). Passive smoking in the same room as infants increased the risk for SIDS in a study at the University of California, San Diego. (Klonoff-Cohen HS, et al. JAMA 1995;273:795-798). An editorial by Willinger M (JAMA 1995;273:818-819) advises that caregivers should follow AAP recommendations, and parents should be counselled that back or side sleep position is one measure to protect their infant from SIDS, but it is not fool-proof.

A CASE OF ACQUIRED "PSEUDO" HYPERTROPHIC NEUROPATHY

A 9-year-old boy with chronic progressive motor-sensory neuropathy beginning in early infancy and reversed by corticosteroid therapy is reported from the Institute of Neurological Diseases, Hirosaki University School of Medicine, Japan. The parents had noticed an awkward gait and frequent falling after learning to walk at 15 months of age. He was in a wheel chair at examination, and he complained of hand numbness. Limb muscles were severely weakened and atrophied, and intrinisic hand muscles totally paralysed. Pes cavus was bilateral. Tendon reflexes were absent. Nerves at elbows and knees and behind the ears were thickened and enlarged. CSF protein was 68 mg/dl. Biopsy of the sural nerve showed edematous swelling, and loss of myelinated fibers, but only occasional onion bulbs. One week after IV methylpredisolone (25 mg/kg/day) for 3 days, followed by oral prednisolone (2 mg/kg/day), numbness in the hands decreased, and sensation and muscle strength improved. Within four weeks, he was walking alone, and posterior auricular nerves were no longer visible. Comparison of EMG and NCS before and after steroids showed that the extremely slow conduction velocities of 2 m/s had increased to 7 to 16 m/s. (Baba M et al. "Pseudo" hypertrophic neuropathy of childhood. J Neurol Neurosurg Psychiatry Feb 1995;58:236-237). (Respond: Dr Masayuki Baba, Department-of Neurology, Institute of Neurological Diseases, Hirosaki University School of Medicine, Zaifu-cho 5, Hirosaki 036, Japan).

COMMENT. Steroid responsive neuropathy in childhood (Byers and Taft. Pediatrics 1957;20:517) was cited as the first reference to this disorder.