was equal to that for 18-year olds. Injured players had greater playing experience, and defense and wings accounted for the highest percentage of injuries.

Injuries occurred primarily during competition (82%) and breakout plays accounted for 23% of head injuries. Colliding with another player (35%) and hitting the boards (20%) were the major mechanisms of injury. Playing hockey to allay tension and aggressions resulted in a risk of concussion four times that of alternative motivations such as enjoyment of the game, a scholarship for college, or peer relations. The use of the face mask may have promoted a more aggressive style of play and also increased risk of concussions and other injuries. The authors advocate elimination of body checking, the cushioning of boards, and the use of breakaway goal posts. (Gerberich SG, Finke R et al. Child's Nerv Syst 1987; 3:59-64).

COMMENT: These statistics are alarming. Other studies have shown that one-third of all hockey related injuries have occurred in children aged 5 to 14 years. Epidemiological studies are needed in the younger age groups and Little Leagues. Coaches should be aware that the development of vertebrae and adjoining cartilage is incomplete in children and susceptibility to neck injury is greater than in adults. Coaches, players and parents should be better educated regarding potential risks of serious injuries associated with collision forces to the head and neck in young hockey enthusiasts and aggressive behavior should not be condoned.

Other sports-related CNS injuries in children and adolescents reviewed by Lehman LB (Postgrad Med Sept 15 1987; 82:141) include closed head and cervical spine injuries associated with boxing, wrestling, judo, karate, gymnastics, football, soccer, and rugby. The most important step in reducing cervical spine injury has been the elimination of "spearing", which involves the use of the helmeted skull as a battering ram. Episodes of mild cerebral concussion, a frequent occurrence in competitive sports, are deleterious. Recently, the cheerleaders "pyramid" formation has been implicated in a number of neurological injuries and even deaths.

DEMYELINATING DISEASES

MULTIPLE SCLEROSIS

Nine MS clinics from the Canadian MS study group collaborated in a retrospective study employing questionnaires about the MS populations and with particular reference to cases with onset before age 16 years. Childhood MS was more frequent in girls and their overrepresentation was even greater in the following subgroups: those with sensory initial symptoms, complete recovery from initial episode, a nonprogressive clinical course, and lower disability scores. Conversely, boys were overrepresented in subgroups of patients with no recovery from the initial episode and progressive course.

MS in girls has an early onset, is usually heralded by sensory symptoms that frequently remit completely, has a relapsing-remitting course and a slow progression. Boys with MS have a poorer prognosis and progressive course, usually related to late onset of the disease. The familial incidence was 28%. CSF showed normal IgG levels in 59% and

abnormal oligoclonal bands in 82%. (Duguette P, Murray TJ et al. Multiple sclerosis in childhood: Clinical profile in 125 patients. J. Pediatr 1987;111:359-63).

COMMENT: MS is probably more common in children than we suspect and the diagnosis should be considered especially in girls with initial sensory or visual symptoms that remit completely and later evolve in a relapsing-remitting manner. An onset at 2 years of age is the earliest case report (Bejar, Ziegler Arch Neurol 1984;41:881). The abrupt rise in incidence that coincides with puberty may be related to hormonal factors. Analysis of data from a Faroe Island epidemic of MS suggested a 2-stage process in the pathogenesis of MS: 1) acquisition of an exogenous factor such as infection and 2) the onset of host factors related to pubescence that allow the pathogenesis to proceed (Fischman HR. AM J Epidemiol 1981;114:244).

Oligoclonal bands in the CSF are the best single laboratory test for the presence of abnormal IgG in patients suspected of having MS. A combination of oligoclonal band and IgG synthesis tests is 97% sensitive for probable and definite MS. (Bloomer LC, Bray PF. Clin Chem 1981;27:2011). NMR imaging differentiates between gray and white matter and is superior to CT in the diagnosis of MS. (Young IR et al. Lancet 1981;2:1063).

DEGENERATIVE AND METABOLIC DISEASES

SPINOCEREBELLAR DEGENERATION

Two children, an 8-year old boy and a 6-year old girl, with progressive ataxia, dysmetria, hypoactive or absent deep tendon reflexes, equivocal plantar response, and sensory impairments, were investigated by pathologists and neurologists at the University of Vermont, Burlington, VT, and University of Saskatchewan, Saskatoon, Canada. The diagnosis of SCD was established by the clinical course and laboratory tests that were normal for arylsulfatase, amino acids, phytanic acid etc.

Rectal biopsy specimens were examined ultrastructurally by electron microscope and by a laser microprobe mass analyzer (LAMMA). Clusters of acicular osmophilic inclusions in the mitochrondria of neuronal somata were consistent with crystals of calcium hydroxyapatite (CHA). The calcific nature of the deposits was confirmed by LAMMA. Similar mitochrondrial inclusions were found in 10% of smooth muscle cells but not in skeletal muscle and nerve biopsy specimens. Tissue from control subjects had no mitochrondrial acicular deposits. It has been suggested that the calcium overload may interfere with mitochondrial enzyme activity by disrupting oxidative phosphorylation. (Munoz DG, Emery ES III, Highland RA. Mitochondrial hydroxyapatite deposits in spinocerebellar degeneration. Ann Neurol 1987;22:258-263).

COMMENT: An abnormal oxidative phosphorylation in muscle mitochondria of patients with Freidreich's ataxia (FA) was previously demonstrated by Stumpf DA et al (Neurology 1982;32:221); mitochondrial malic enzyme activity was 10% of control level in FA fibroblasts. Also, glutamate dehydrogenase deficiency has been noted in cultured skin fibroblasts and leukocyte homogenates of patients with spinocerebellar syndrome (Plaitakis A et al. Ann Neurol 1980;7:297). These studies and the present report may eventually lead to carrier