

### HYPOXAEMIA IN PERTUSSIS

Breathing movements, airflow and arterial oxygen saturation were recorded in 6 infants aged 3 weeks to 7 months with apneic and cyanotic episodes associated with pertussis and compared with 12 healthy controls at the Dept of Paediatrics, Cardiothoracic Institute, Brompton Hospital, and Communicable Disease Unit, St George's Hospital Medical School, London. Infants with pertussis had a greater frequency of apneic pauses, episodes of hypoxemia, and dips in oxygen saturation even during continued breathing movements. Apnea accompanied by mismatch between ventilation and perfusion of the lungs may produce the rapid onset of severe hypoxemia in infants with pertussis. Electroencephalographic recordings during prolonged pauses in inspiratory efforts with hypoxemia in one patient did not show a seizure discharge, but convulsions occurred in association with apnea and cyanosis in 4 patients. The twin sister of one patient, with an identical history of pertussis, died during a cyanotic convulsion while the infants were being brought to the hospital. That the convulsions are secondary to severe cerebral hypoxemia is suggested by these recordings and findings. (Southall DP et al. Severe hypoxaemia in pertussis. Arch Dis Child June 1988;63:598-605).

**COMMENT.** This excellent study and method of investigation offers advantages over epidemiological research in demonstrating the potential hazards of pertussis in small infants and the need for early immunization and less toxic pertussis vaccines. In my experience, I have encountered no cases of convulsions as a result of pertussis in the USA, a reflection of the excellent compliance with immunization programs perhaps, while children with convulsive disorders related temporally to pertussis immunization have been referred to me often for neurologic evaluation and treatment. DPT vaccine was listed as one of 7 drugs most commonly implicated in hospital admissions prompted by adverse reactions (Mitchell AA et al. Pediatrics July 1988;82:24).

### CNS EFFECTS OF COUGHING IN CYSTIC FIBROSIS

Neurological symptoms complicating the paroxysmal coughing associated with cystic fibrosis were studied in 273 patients, aged 10 to 44 years, attending the Cystic Fibrosis Research Center, Divisions of Pediatric Pulmonology and Neurology, Case Western Reserve University School of Medicine and Rainbow and Children's Hospital, Cleveland, OH. Lightheadedness in 47% and headache in 50% of those with paroxysms were the most common neurological symptoms. Confusion, visual loss or dimming, paresthesias, speech disturbance, tremors, paralyzes of extremities and face, and syncope were less frequent, transient abnormalities. The pathophysiology of these symptoms was explained by high intrathoracic pressure, as in cough syncope, that is transmitted to the cerebrospinal fluid, compressing cranial vessels and resulting in a "bloodless brain" or transient cerebral ischemia. In some patients with headache, marked hypoxemia and hypercapnia accompanied the coughing paroxysm and aggravated the symptom, but in most this mechanism was unlikely, and irreversible sequelae were not observed. (Stern RC et al. Neurological symptoms during coughing paroxysms in cystic fibrosis. J Pediatr June 1988;112:909-912).

**COMMENT.** Unlike pertussis, in which paroxysmal coughing, episodic apnea and cyanosis, or disturbed ventilatory perfusion may result in severe hypoxemia and neurological dysfunction, the coughing in cystic fibrosis is rarely accompanied by significant hypoxemia and serious neurological sequelae. A direct effect of B pertussis toxins on surfactant function leading to alveolar atelectasis has been invoked as the cause of hypoxemia in pertussis.

#### LYME DISEASE AND CNS COMPLICATIONS

Four adult patients with chronic meningoencephalitis caused by tick-transmitted *Borrelia burgdorferi* infection are reported from the Depts of Neurology, University of Freiburg, Freiburg, and University of Koln, Koln, Federal Republic of Germany. All patients lived in wooded areas in which the transmitting tick (*Ixodes ricinus*) was widely distributed. IgG antibody titers were higher in CSF than in serum, indicating a specific intrathecal immune response against the B burgdorferi antigen and suggesting neuroborreliosis (Bannwarth's syndrome), although the characteristic painful meningopolyneuritis was absent. MRI showed either multiple lesions of high signal density in the white matter suggestive of MS or evidence of vascular involvement, as in other spirochetal infections, such as meningovascular syphilis. The authors consider the clinical spectrum of neuroborreliosis to be comparable to the different forms of neurosyphilis. Only one patient had a complete clinical remission after intravenous penicillin therapy, in 2 there was no further progression, and one showed no improvement. (Kohler J et al. Chronic central nervous system involvement in Lyme borreliosis. Neurology June 1988;38:863-867).

**COMMENT.** The manifestations of Lyme disease are in 3 stages: Stage I, erythema chronicum migrans, in 80-95% cases, minor flu-like symptoms with headache, fatigue, fever, myalgias, and other signs of disseminated disease such as arthralgias; Stage II, neurological complications in 15% cases, onset at 2-11 wks, with meningitis, Bell's palsy, and peripheral radiculoneuropathy; carditis (8%); and Stage III, arthritis (60%) and chronic neurological syndromes as described above. The most common neurological complication is aseptic meningitis which presents with headache and stiff neck and associated encephalitic symptoms including somnolence, emotional lability, memory loss, poor concentration and behavioral changes. Seventh nerve palsy is seen in 50% patients with meningitis or it occurs alone. Peripheral neuropathies are motor or sensory or mixed. Less common neurological complications include mononeuritis multiplex, transverse myelitis, Guillain-Barre syndrome, chorea, cerebellar ataxia, and pseudotumor cerebri. Maternal-fetal transmission has been described, although no definite link to fetal anomalies has been documented.

Treatment recommended in the literature varies with the age: for children over 8 yrs and adults, oral tetracycline; for children under 8 yrs, phenoxymethyl penicillin. (For details, see Hurwitz S. Contemporary Pediatrics June 1988;74-82; Stechenberg BW. Pediatr Infect Dis J. June 1988;7:402-409). Preventative methods include: (1) avoidance of wooded, grassy areas; (2) use of tick repellants such as Deet and permethrins on clothes; (3) removal of ticks by pulling straight out with tweezers.