prevalence of neural tube defects in siblings of children with tracheoesophageal dysrhaphism (esophageal atresia with or without tracheoesophageal fistula). In the small samples available in the literature[2,3] they found that of 635 siblings 10 (1.6%) had a neural tube defect. The rate is not significantly larger than that expected by chance, because of the sample size, but it does suggest that further study is needed.

Data available from the Health Surveillance Registry of British Columbia were analysed to see if any evidence could be found of increased risk of neural tube defects in siblings of individuals with tracheoesophageal dysrhaphism. Since 1952 this registry has maintained statistics and other information on congenital malformations and chronic handicapping conditions occurring in British Columbia. Each affected individual may be ascertained by the registry from a variety of sources; the most important for us were hospital discharge diagnoses for infants, physicians' notices of birth and vital registrations of deaths. Discharge diagnoses for children under 7 years of age are submitted to the registry by all public hospitals in the province.

Of all congenital malformations neural tube defects are among those most readily recognized at or shortly after birth. A previous study has shown that virtually all cases of anencephaly and almost all those of spina bifida are ascertained in the first year of life.[4] This means that unusually large numbers of cases of neural tube defects in this population ought not to be missed. In British Columbia the prevalence of neural tube defects is approximately 1.5 per 1000 births. The prevalence of tracheoesophageal dysrhaphism in this province for the period 1952–79 was 1 per 6075 live births; from 1966 on, when important ascertainment sources were added, the rate was 1 per 3824 live births — comparable to the rates quoted in the literature.[5,6]

Of the 167 infants with tracheoesophageal dysrhaphism born in the period 1952–79, 89 were male and 78 were female; 2 (1 of each sex) were stillborn. Additional anomalies were found in 98, but none of these was a neural tube defect. There were 10 siblings registered: 3 had strabismus, 2 clubfoot and 1 each syndactyly, microphthalmia, branchial cleft fistula, celiac disease and cerebral gigantism. None had a neural tube defect or tracheoesophageal dysrhaphism. This is in keeping with the literature evidence suggesting that there is not an increased risk of tracheoesophageal dysrhaphism in siblings of index patients.

In looking at the data available we found no evidence of an increased risk of neural tube defects in the siblings of 167 patients with tracheoesophageal dysrhaphism.

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Legionnaires' disease bacillus in bone marrow

To the editor: Legionnaires' disease has almost invariably been reported as a pulmonary infection; the frequency and extent of involvement of other organs is largely unknown. We encountered a patient with bilateral pulmonary infiltrates in whom the diagnosis of Legionnaires' disease was made by identifying *Legionella pneumophila* in bone marrow aspirate stained by an immunofluorescent dye and was subsequently substantiated by the demonstration of a fourfold rise in the titre of antibodies in the serum against *L. pneumophila* serogroup 2.

Case report

A 49-year-old man with a history of cirrhosis of the liver was admitted to hospital with altered mentation, a low-grade fever and a nonproductive cough of several days' duration. His temperature was 38°C and his pulse rate and blood pressure were normal, but he was confused and disoriented. He displayed asterixis. The sclerae were markedly icteric. Coarse rales were heard at the bases of both lungs, and anasarca was present. The abdomen was tensely distended with ascites, but an enlarged liver and an enlarged spleen could be palpated. The blood leukocyte count was 4.7×10^9/l, with a normal differential count. The serum albumin level was low, at 2.0 g/dl. The prothrombin time was increased, as were the levels of bilirubin and liver enzymes in the serum.

The initial pulmonary diagnosis was aspiration pneumonia. Blood, urine and sputum samples were obtained for culture, and the patient was given cephalothin intravenously for 1 week. No organisms were grown from the samples, but specific media for the culture of *L. pneumophila* were not used. The cold agglutinin titres in the serum were normal, as were the titres of antibodies against common viruses that cause respiratory tract infections. Antibodies to *L. pneumophila* serogroup 2 were demonstrated in a dilution of 1:64.

The patient showed no response to cephalothin therapy. Several tracheobronchial aspirates failed to yield pathogenic organisms in aerobic and anaerobic cultures, including those with charcoal–yeast agar and in Feeley–Gorman medium (performed by the reference laboratory at our hospital), designed to support the growth of *L. pneumophila*.

Since Legionnaires' disease was suspected, a bone marrow aspirate was subjected to direct immunofluorescent staining for *L. pneumophila*.[1] Highly immunofluorescent bacilli were identified when the as-

pirate was stained with antibody against serogroup 2 organisms. Accordingly, erythromycin lactobionate was administered intravenously. The patient's temperature returned to normal 4 days later. The same bone marrow aspirate failed to yield *L. pneumophila* in cultures with charcoal–yeast agar and Feeley–Gorman medium. However, the titre of antibodies in the serum against *L. pneumophila* serogroup 2 was now 1:256.

After 1 month of erythromycin therapy slight clearing of the pulmonary infiltrates was noted. Subsequently massive bleeding from the upper gastrointestinal tract occurred and the patient died. At autopsy a hepatoma was found with metastases to the lungs, chest wall and other organs. Cultures of lung, liver, heart and kidney tissue and of blood failed to yield *L. pneumophila* or other organisms, and direct immunofluorescent staining with antibodies against *L. pneumophila* serogroups did not reveal organisms in these tissues.

Comments

Legionnaires' disease is primarily an infection of the lungs; the organisms have been isolated from lung tissue, pleural fluid and transbronchial aspirates.[1] The extent of extrapulmonary dissemination is unknown. However, intact organisms have been observed in pulmonary hilar lymph nodes[2] and in blood vessels of the heart, kidney and spleen.[3] The fact that *L. pneumophila* has also been isolated from the blood indicates that bacteremia may occur in Legionnaires' disease.[4] Our observations support this notion and also suggest that bone marrow aspiration may provide an additional means of establishing the diagnosis.

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Photosensitivity reaction treated with diphenhydramine

To the editor: A photosensitivity reaction is one of the more clinically annoying and frequent side effects of phenothiazines. In the children I treat it is a common summertime phenomenon. The reaction is not consistently listed as such in the "Compendium of Pharmaceuticals and Specialties"[1] but is described in "Meyler's Side Effects of Drugs"[2] as resembling exaggerated