metastases were found the liver scan was normal, and in three of the seven the serum LDH value was raised (80, 87 and 290 IU/l). In all 12 of the patients in whom liver metastases were found at autopsy the serum LDH value was elevated, but in 4 of the 12 the liver scan made at the time of the elevated serum LDH value was considered to be normal.

Discussion and conclusion

We believe that this study has provided confirmation of the findings of Einhorn and colleagues,[1] having demonstrated the usefulness of an elevated serum LDH value as an indicator of hepatic metastases in patients with stage IIIB or IV malignant melanoma.[2-5] If a serum LDH value greater than 78 IU/l had been considered an indication for requesting a liver scan, 60 instead of 159 liver scans would have been done; 3 abnormal scans and 1 equivocal scan would have been missed. Since we do not have pathologic confirmation of the proportion of this group of patients who had metastases, it is not strictly correct to speak of test sensitivity or specificity. However, our results do suggest that measuring the serum LDH concentration may be considered as a prerequisite for ordering a liver scan: if the LDH value is normal the probability that the liver scan will be abnormal or equivocal is only 0.04 (4/99), while if the LDH value is elevated the probability that the liver scan will be normal is 0.68 (41/60).

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Severity of lung disease in Indian children

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Recurrent bronchopulmonary infections in North American Indian children are an important social and public health problem in Saskatchewan and adjacent provinces. We have been impressed by the prevalence and severity of these infections, and feel that particular emphasis should be given to measures designed to prevent them. The study described in this paper was conducted to investigate the problem.

Methods

Beginning in January 1974 a 3-year prospective record was kept of children in whom the roentgenographic appearance of the chest became worse during treatment of pneumonia in hospital. One of us (R.L.W.) then reviewed the charts of all children 14 years of age or less who had been admitted to our hospital with pneumonia during the same period. Children with underlying conditions such as heart disease and cystic fibrosis were excluded.

Results

Indian children with pneumonia tended to be sicker. Fever and adventitious sounds persisted more than twice as long as with white children (Table I). The stay in hospital was much longer for Indian children, and this could be explained only in small part by differing social and distance factors. Five times as many Indians had associated diarrhea. Few Indians had been breast-fed.

Of the 21 children in whom the roentgenographic appearance of the chest worsened in hospital, 20 were Indians and 1 was a 12-month-old white boy in whom adenovirus infection developed after rubeola (Table II). None of the 64 white children died, but 4 of the 102 Indian children died, 2 with pneumonia following pertussis, 1 with rubeola and giant-cell pneumonia, and 1 with fulminating primary staphylococcal pneumonia complicated by empyema.

Autopsy of patient 9, who died of adenovirus pneumonia following pertussis at 8 months of age, showed

Table I—Data for 166 children with pneumonia, 1974-76

Variable	Indian (n = 102)	White (n = 64)
Male:female ratio	59:43	32:32
No. of patients with		
Worsening of roentgenographic appearance of chest in hospital	20	1
Pneumatocele	4	0
Death in hospital	4	0
Average duration (d) of		
Stay in hospital	26.9	8.7
Fever after admission	6.6	3.1
Adventitious sounds	17.2	7.6
Proportion of group		
With associated diarrhea	48%	9%
Breast-fed 1 month or more*	11/74	9/40

*Not recorded for some patients.

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much more thickening of the bronchial walls than would have been predicted from the roentgenograms made just before death.

Discussion

Indian children, when admitted to hospital with other problems, commonly have thick bronchial walls; the roentgenographic appearance is similar to that of white children with asthma, cystic fibrosis or immune deficiency. When rubeola or pertussis develops in Indian children, bronchopneumonia almost routinely follows.

These chest infections cause many days of sickness, are a great concern to the parents, and often result in transportation of the child to a distant city hospital in a different culture, where he or she is treated by strangers who speak a different language.

The average Indian child is admitted to hospital five times as often as the average white child. In Saskatchewan, published figures for Indians are available only for 1966, when there were 1148 admissions and 12 387 days in hospital per 1000 Indian infants in their first year of life, compared with only 258 admissions and 2305 days in hospital per 1000 infants in the overall population, including Indians.[1]

A chronic cough and increased susceptibility to chest infections have been among the hallmarks of the underprivileged through the ages. An increased incidence of severe pneumonia is still found among the Maori and Polynesian children in New Zealand,[2] and among the Aborigines of Australia,[3] who also show bronchial wall thickening (W.J.H. Caldicott: personal communication, 1977).

In 1965 Brody[4] reported an apparently increasing incidence of severe lower respiratory tract disease in young Inuit children in Alaska. Of 318 newborns followed prospectively 35% had at least one episode of pneumonia or bronchitis during the first year of life and 16 died from respiratory causes. A follow-up study by Maynard and colleagues[5] in the same area in the winter of 1966 showed a viral association in 75% of attacks. Bronchiectasis, often in the upper lobe of the right lung, following severe lower respiratory tract infections in childhood, is unusually common among Inuit and Indian children in Alaska.[6]

Herbert and colleagues[7] studied prospectively 97 Indian children with 112 episodes of pneumonia that presented to the Charles Camsell Hospital, Edmonton during 1 year. Of these, 71% were between 3 and 18 months of age. Two had a history of more than six previous attacks and 34 had records of three to six previous attacks. Of the 14 children who acquired adenovirus type 3 pneumonia while in hospital with unrelated problems 11 had a chronic productive cough 10 years later.[8] Bronchograms were done in seven and showed widespread airway damage. In our series of children adenovirus infections were present in the one white child and in three of the Indian children in whom the roentgenographic appearance of the chest worsened in hospital.

Chest infections are common among treaty Indians, all of whom have some white blood, and among nontreaty Indians and Métis who live in a similar economic and cultural setting. Several factors contribute to this. The average number of persons per house is 6.67 on Saskatchewan Indian reserves, compared with 3.45 for non-Indians. Only 1.4% of Indian homes on Saskatchewan reserves have running water.[9] In a small home the children lack sleep because they stay awake until the adults go to bed and then have to get up early for school. Some Indian homes are inadequately or irregularly heated, and some small homes are smoke-filled from the heating system or from ciga-

Table II—Details of patients in whom the roentgenographic appearance of the chest worsened in hospital

Patient no.	Age (mo)	Sex	Status*	Infection		Outcome
				Primary	Complicating	
1	3	F	T	Pertussis	—	Discharged improved from hospital
2	4	F	T	Influenza A/Victoria	—	Discharged improved from hospital
3	4	F	T	*Staphylococcus aureus*	Septic meningitis	Discharged improved from hospital
4	4	F	T	Rubeola	—	Died
5	5	F	NT	Pertussis	—	Discharged improved from hospital
6	6	M	T	Pertussis	Parainfluenza 3	Pneumatocele, died
7	6	M	T	Pertussis	*S. aureus*	Pneumatocele
8	7	M	T	*Shigella*	Pneumococcus	Empyema
9	8	M	NT	Pertussis	Adenovirus	Died
10	8	M	T	Rubeola	Adenovirus	Discharged improved from hospital
11	9	M	T	Rubeola	Pneumococcus	Discharged improved from hospital
12	11	M	T	Rubeola	Adenovirus, *Staphylococcus, Pseudomonas*	Pneumatocele
13	11	M	T	Rubeola	*Staphylococcus, Klebsiella, Hemophilus influenzae*	Discharged improved from hospital
14	12	M	NT	†	—	Discharged improved from hospital
15	12	M	W	Rubeola	Adenovirus	Discharged improved from hospital
16	14	M	T	Rubeola	Migrating pneumonia, diarrhea	Discharged improved from hospital
17	14	M	T	Pertussis	Rubeola, influenza A, *H. influenzae*	Discharged improved from hospital
18	14	F	NT	*S. aureus*	—	Empyema, died
19	16	F	T	Rubeola	—	Discharged improved from hospital
20	17	F	NT	Rubeola	*S. aureus*	Pneumatocele
21	56	M	T	Rubeola	—	Discharged improved from hospital

*T = treaty Indian; NT = nontreaty Indian; W = white.
†No organism was grown from the patient's specimens.

rettes. The diet may be inadequate, especially relative to physical activity,[10] and 35% of Indian children under 4 years of age have low serum concentrations of vitamin A.[11]

The change from breast-feeding to bottle-feeding is both a social and a biologic problem. The need for the protection offered by the substances in breast milk is even greater in infants with an increased susceptibility to disease.[12] Schaefer[13] has found that, among the Inuit, breast-fed children have much less otitis media than bottle-fed children, and Ellestad-Sayed and colleagues[14] have reported that breast-fed Indian children in northern Manitoba have far fewer lower respiratory and gastrointestinal tract infections than bottle-fed infants, even though the social conditions of the breast-fed infants are, in general, poorer.

The high frequency with which chest infections were associated with diarrhea in the Indian children in our study is of interest. It may indicate a higher susceptibility of both the respiratory and the gastrointestinal systems to infection, or it could relate to a common precipitating cause, such as milk allergy, which appears to be more frequent in Indian infants than in white infants.[15]

Indian children have either normal or, more often, elevated concentrations of serum globulins; the elevation of serum IgA, IgD, IgE and IgG concentrations has been attributed to increased exposure to infections rather than to genetic factors.[16] This suggests a satisfactory humoral immune response; however, Indian children may have a different immune response to some infections, compared with non-Indians. Rubeola was absent among Indians prior to settlement by the white man: such a high proportion of a sparse population would be killed or immunized that the virus could no longer propagate.[17] The lack of many centuries of exposure and selection could, in part, explain the susceptibility of Indians not only to the tubercle bacillus but also to specific viruses, including that causing rubeola, and adenoviruses. Rubeola is known to be more severe in malnourished populations and, in turn, it may precipitate malnutrition.[18]

Sixteen of our 21 patients in whom the roentgenographic appearance of the chest deteriorated in hospital, including 3 of the 4 that died, had a vaccine-preventable disease — rubeola or pertussis — before contracting pneumonia. However, of the 10 such children with rubeola, 6 acquired it before 12 months of age, the age usually advocated for measles vaccination. An active rubeola vaccination program for Saskatchewan treaty Indians is now being offered at the age of 7 months. It is realized that further immunization will be necessary at a later age, but the protection from the devastating effect of measles on young Indian children makes early vaccination justifiable.

It is probable that recurrent pulmonary infections in Indian children might largely be prevented by improvement of living conditions, encouragement of breast milk as the sole food for the first 6 months of life, and more widespread early vaccination.

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continued from page 1111

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