The feed which was made up at home was analysed and contained sodium 117 mEq/l, glucose 4 g/100 ml, and osmolality 450 mOsm/l. Thus, the sodium concentration was far higher than the 30 mEq/l intended.

The baby was treated by a restricted fluid intake of 100 ml/kg body weight per day given as a solution of 5% glucose in water orally with added potassium chloride. After 36 hours the oedema subsided and blood pressure was 90/60 mmHg. Plasma electrolytes remained normal, 24-hour urine sodium excretion fell to 5 mEq, PRA and PAldo rose to 1225 pgAI/ml per h and 22.8 ng/100 ml, respectively. She remained neurologically normal, was regradied onto milk, and has remained well.

**Discussion**

We report this case to emphasize how the un-supervised addition of salt to infant feeds of any type may give rise to a dangerously high intake of sodium. This baby's 24-hour sodium intake before admission was 117 mEq (about 25 mEq/kg) per day. She was excreting about 4.5 mEq/kg per day and thus was retaining about 15 mEq/kg per day. She had minimal diarrhoea at this time and stool losses of sodium would not be great. If gastrointestinal water losses had been greater, she might well have presented with hypertonic dehydration. Indeed, the latter condition has been reported in association not only with high solute milk feeding but with both commercial and 'home-made' oral electrolyte solutions prescribed for gastroenteritis when either the instructions have not been followed or excess salt added (Colle, Ayoub, and Raile, 1958; Franz and Segar, 1959).

Severe hypertension may well have been avoided by the development of oedema in our patient. The suppression of the renin aldosterone system in this infant, presumably by the exogenous salt and water load, is interesting. We are unaware of a similar observation in a child of this age. We suggest that this degree of suppression may be a factor in the development of hypernatraemia when abnormal water losses occur in infants fed on hyperosmolar feeds. The importance of the renin aldosterone system in control of sodium homoeostasis is well established. If this system is fully suppressed by a high solute intake, there is no reserve capacity to excrete excess sodium.

In conclusion we wish to emphasize that in infants with mild diarrhoea there should be no need to give more than 5 mEq sodium/kg per day. Giving fifth normal saline with glucose at the rate of 150 ml/kg per day would provide the necessary sodium. Unless salt can be given accurately in small amounts, it may be safer to advise glucose and water only.

**Summary**

This paper describes an infant with gastroenteritis, who developed hypertension and oedema after administration of inaccurately prepared oral glucose salt solution. The renin aldosterone system was suppressed in this child and it was suggested that this may be a factor in the development of hypernatraemia when abnormal water losses occur in infants fed on hyperosmolar feeds. Unless salt can be given accurately in small amounts it may be safer to advise feeds of glucose only in infants with mild diarrhoea.

We wish to thank Dr. W. C. Marshall for permission to publish this case.

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**Thymic dysplasia, persistence of measles virus, and unexpected infant death**

After extensive post-mortem investigations of cases of 'cot death' or sudden infant death syndrome, a wide variety of pathological changes has been found, many of doubtful significance. Currently there has been an interest in the histological changes in the conducting tissue of the heart (Ferris, 1973). Unfortunately it is uncertain whether these changes are the cause or the effect of some cases of cot death. Virological studies have shown a greater incidence of isolation of viruses, particularly respiratory, from cot deaths when compared with controls (Ray et al., 1970; Ferris et al., 1973).
The purpose of this report is to draw attention to an unusual thymic deficiency associated with persistence of measles virus, with a normal antibody response, in a case of sudden infant death. A possible relation of the thymic abnormality to the immunology of measles infection is raised.

Although the infant was aged 18 months and therefore does not fit into the generally accepted age range for the sudden infant death syndrome (1 week to 1 year), it may be relevant to the problem of 'cot death'.

**Case report**

An 18-month-old female infant was found dead in her cot at midnight having been put to bed, perfectly well, 4 hours before. A pool of vomit was found around the infant's head and there was some foamy material around the nose and mouth. There was no pillow in the cot and the infant was wearing its day clothes. The infant had a past history of repeated upper respiratory tract infections. 3 weeks before death she had had a typical attack of measles, with rash, from which she had made a complete recovery. She had not been immunized against measles. There was no lymphadenopathy when the child was seen at the time of the measles. She had 2 brothers, aged 6 years and 4 years; both suffered from periodic respiratory infections but were otherwise well.

**Necropsy** showed a well-nourished female infant with no signs of violence and no signs of a measles rash, eczema, or purpura. The skull, brain, heart, thymus, spleen, and lymph nodes were all normal. There was some milky fluid in the upper respiratory passages and there were focal areas of congestion in the lung parenchyma. The stomach was distended with a recent milky feed. Venous blood was taken for determination of measles antibody titre, slices of lung and tonsil were collected for virus isolation, and a lung swab was cultured for bacteria.

Histologically, there were mild, inflammatory changes in the tracheal submucosa and there was a scanty, patchy, interstitial pneumonitis with acute and chronic inflammatory cells. No giant cells, as described in giant cell pneumonia, were found, and there were no inclusion bodies in the bronchial epithelium. The thymus showed plentiful cortical lymphocytes together with thymic epithelial cells, but sections from 10 blocks of tissue failed to show any mature or rudimentary Hassall's corpuscles. The thymic vasculature was normal and, in particular, showed no evidence of rubella endarteritis (Garcia, Olinto, and Fortes, 1974). The lymph nodes and spleen showed a florid reactive follicular hyperplasia with prominent germinal centres. There were enlarged pleomorphic lymphocytoid cells in the cortical and medullary sinuses with markedly pyroninophilic cytoplasm. No Warthin-Finkeldey giant cells were seen. There was no depletion of lymphocytes in the paracortical zones. Similar abnormal lymphocytoid cells were also identified in the splenic sinuses and portal tracts. Sections of the heart, kidneys, adrenals, salivary glands, thyroid, pituitary, and brain were normal. In particular, no inclusion bodies were seen in the brain. The bone marrow was of normal cellularity.

Complement fixing antibodies to measles virus were present to a titre of 1/256. Measles virus was recovered from slices of lung and tonsil. A lung swab produced a scanty growth of *Streptococcus viridans* and *Neisseria catarrhalis*. Rubella antibody titre was not raised.

**Discussion**

Three unusual features emerge from this case of sudden infant death; persistence of measles virus 3 weeks after the rash, atypical lymphocytoid proliferation in the lymphoreticular system, and isolated deficiency of Hassall's corpuscles.

The isolation of measles virus is normally possible only during the first 3 days after the rash appears; however, in cases of giant-cell pneumonia complicating leukaemia, the virus has been isolated from 2 patients 3 and 4 weeks after the onset of measles (Mitus et al., 1959). This pulmonary reaction occurs in individuals who are unable to manufacture specific antibodies and they often fail to produce a rash. This case clearly does not fall into this category. It is known that a competent, thymic-dependent lymphocyte population is also required by the body to eliminate virus infections. Congenital thymic deficiencies such as Di George's syndrome, Swiss type a-γ-globulinaemia, and Nezelof's syndrome are associated with a marked susceptibility to virus infection. In these cases the thymus shows a deficiency of both lymphocytes and Hassall's corpuscles with variable depletion of lymphocytes in paracortical areas of lymph nodes (Berry, 1970). In a review of neonatal thymic abnormalities, Berry (1968) found 18 cases with deficiency or absence of Hassall's corpuscles, but in all these cases there was additional gross depletion of cortical and medullary thymic lymphocytes. In this case the thymus is normal in size with plentiful cortical lymphocytes and yet completely lacks Hassall's corpuscles. The significance of absent Hassall's corpuscles in relation to the cell-mediated competence of lymphocytes is not known. Their absence may explain the persistence of measles virus in this case, with the inability of the infant to eliminate the infection even in the presence of specific antibody formation. The atypical lymphoid cells are consistent with a reaction to a viral infection; however, an alternative explanation is that these cells represent a subclinical acute leukaemia. Persistent measles virus complicating leukaemia was described by Mitus et al. (1959) but there was no antibody formation. Finally, it is well known that congenital thymic and other immunological deficiency diseases may be complicated by the development of leukaemia and other lymphoreticular tumours.
Summary

A case of sudden infant death in an 18-month female infant is described which occurred 3 weeks after clinical recovery from a typical attack of measles. At post-mortem examination no obvious cause of death could be found apart from some terminal aspiration of vomit. Histologically, the thymus showed selective absence of Hassall's corpuscles with a normal number of cortical lymphocytes. There were many atypical lymphocytoid cells in the lymph nodes, spleen, and portal tracts, and measles virus was subsequently isolated from the lung and tonsil. There was a raised titre of complement fixing measles antibody (1/256).

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