Cohen, A., and Way, B. J. (1962). *Australasian Annals of Medicine*, **11**, 189.
Foss, G. L., Perry, C. B., and Wood, F. J. (1956). *Quarterly Journal of Medicine*, **25**, 185.
Greenspan, E. M. (1949). *Archives of Internal Medicine*, **83**, 271.
Huth, E. J., Maycock, R. L., and Kerr, R. M. (1959). *American Journal of Medicine*, **26**, 818.
Huth, E. J., Webster, G. D., and Elkington, J. R. (1960). *American Journal of Medicine*, **29**, 586.
Kaltreider, H. B., and Talal, N. (1969). *Annals of Internal Medicine*, **70**, 751.
Leeson, P. M., and Fourman, P. (1967). *American Journal of Medicine*, **43**, 620.
McCurdy, D. K., Cornwell, G. G., and DePratti, V. J. (1967). *Annals of Internal Medicine*, **67**, 110.
Milne, M. D. (1963). In *Diseases of Kidney*, ed. M. B. Strauss and L. G. Welt, p. 786. Boston, Little, Brown.
Morris, R. C. (1969). *New England Journal of Medicine*, **281**, 1405.
Morris, R. C., and Fudenberg, H. H. (1967). *Medicine*, **46**, 57.
Morris, R. C., Sebastian, A., Morris, E., and Ueki, I. (1968). *Journal of Clinical Investigation*, **47**, 70a.
Pines, K. L., and Mudge, G. H. (1951). *American Journal of Medicine*, **11**, 302.
Read, A. E., Sherlock, S., and Harrison, C. V. (1963). *Gut*, **4**, 378.
Seedat, Y. K., and Raine, E. R. (1965). *South African Medical Journal*, **39**, 595.
Shearn, M. A., and Tu, W. H. (1965). *American Journal of Medicine*, **39**, 312.
Shearn, M. A., and Tu, W. H. (1968). *Annals of the Rheumatic Diseases*, **27**, 27.
Smith, P. M., Middleton, J. E., and Williams, R. (1967). *Postgraduate Medical Journal*, **43**, 439.
Talal, N., Zisman, E., and Schur, P. H. (1968). *Arthritis and Rheumatism*, **11**, 774.
Wilson, I. D., Williams, R. C., and Tobian, L. (1967). *American Journal of Medicine*, **43**, 356.
Wrong, O. (1965). *Journal of Clinical Pathology*, **18**, 520.
Wrong, O., and Davies, H. F. (1959). *Quarterly Journal of Medicine*, **28**, 259.
Zisman, E., Buccino, R. A. Gorden, P., and Bartter, F. C. (1968). *Archives of Internal Medicine*, **121**, 118.

Preliminary Communications

Respiratory Syncytial Virus Infection of the Newborn

British Medical Journal, 1970, **3**, 146–147

Summary: In an outbreak of respiratory syncytial (R.S.) virus infection in a maternity hospital the respiratory illness was of a mild nature and the virus was not found in infants without respiratory symptoms. This confirms the suggestion that R.S. virus can infect infants at a very early age. Rapid diagnosis was achieved by applying the direct fluorescent antibody technique to cells in nasal secretions. This proved to be more sensitive than culture techniques where there was delay between the onset of respiratory symptoms and submission of specimens to the laboratory.

Introduction

That newborn babies may sometimes suffer from a relatively mild respiratory illness due to respiratory syncytial (R.S.) virus was recently suggested as an essential part of a hypothetical explanation of the pathogenesis of bronchiolitis in infants (Gardner, McQuillin, and Court, 1970). Evidence to support this suggestion has been found during the investigation of an outbreak of respiratory infection among babies at the Princess Mary Maternity Hospital, Newcastle upon Tyne. This is the first report of an outbreak of R.S. virus in a maternity hospital in the neonatal period, and the mild nature of the clinical illness contrasts sharply with the devastating effects of introducing other viruses into maternity nurseries—for example, parainfluenza I (Sendai) and Coxsackie B virus (Kuroya, Ishida, and Shiratori, 1953; Javett *et al.*, 1956). R.S. virus has been isolated from infants with respiratory infections in a premature nursery (Berkovich and Taranko, 1964), though in this series their ages ranged from 1 to 6 months.

Materials and Methods

A fine plastic feeding tube (size 5 French), substituted for the much larger distal tube of a standard disposable mucus extractor, was passed through each nostril in turn, mouth suction being used to aspirate secretions from the nostrils and nasopharynx into it. The tube was then detached, placed in a universal container, and transported to the virus laboratory in melting ice. The material, usually consisting of saliva as well as blobs of mucus, was expelled from the tube into a bijou bottle. The blobs were broken up by gentle pipetting and the cellular content washed and centrifuged. The supernatant fluid was used for virus culture and the cellular deposit for fluorescent staining for R.S. virus. The culture techniques and the cell lines used have been previously described (Sturdy, McQuillin, and Gardner, 1969).

Immunofluorescent Staining of Material.—Slides were prepared, fixed, and stained for R.S. virus as described previously (McQuillin and Gardner, 1968; Gardner and McQuillin, 1968). The secretions from these newborn babies were less easy to examine directly than those from older patients, containing fewer cells, but they still proved useful for rapid diagnostic procedures.

The Outbreak

On 1 February a baby who was still in the special care nursery at the age of 7½ weeks because of low birth weight was clinically suspected of suffering from mild bronchiolitis; after two days of a snuffly nose with a clear discharge, he had developed a cough and a slight wheeze. The same diagnosis was independently suggested by a second observer next day, and on the following day his nasal secretions were examined and found to be positive for R.S. virus by the fluorescent antibody technique (later confirmed by culture). During the next six weeks a further 25 babies were investigated—nine of them on two occasions—either because of contact with infected babies or because of respiratory symptoms.

R.S. virus was found in eight babies altogether, of whom seven had at some time been in the special care nursery, and one had gone straight from the labour ward to a normal lying-in ward. All eight babies had respiratory symptoms, starting with a snuffly nose with a clear discharge, at ages between 10 and 52 days (in six cases between 10 and 15 days); all developed a cough between two and seven days later, and three also developed a mild expiratory wheeze. None of the babies was worryingly ill except for one with clinical and radiological pneumonia; *Staphylococcus aureus* was cultured from a cough swab and a persistent conjunctivitis. Two of the three babies who developed a wheeze also developed a cardiac murmur, possibly in the ductus arteriosus; this disappeared, however, as they recovered.

Of the remaining 18 babies not infected with R.S. virus, 14 were investigated because they had been in the same room as a positive case and four because they had developed a snuffly nose at the age of 1 to 5 days (one of these was found to be infected with a rhinovirus).

VIROLOGICAL RESULTS

Altogether, eight infants were infected with R.S. virus, the virus being identified in all cases by direct examination of cells in aspirated material by the fluorescent antibody technique. In six of these patients the virus was subsequently isolated on tissue culture. The two in whom virus was not cultured were babies who had been ill for 5 and 14 days, respectively, before isolation attempts were made. The failure to culture virus in these two infants is not surprising, considering the long interval between the onset of illness and their examination for the presence of virus agents. In 17 older children investigated in this laboratory the presence of R.S. virus was found by direct examination of secretion collected on the day of admission and again seven days later, but culture of virus was achieved from the first specimen only. It appears that virus antigen can still be detected by immunofluorescence even after infectivity has been lost (Gardner and McQuillin, 1970). In one case virus antigen could still be detected in cells of the nasopharynx by the fluorescent antibody technique as long as 18 days after the onset of symptoms.

COMMENT

This report of an outbreak of an R.S. virus infection in a maternity hospital illustrates that mild respiratory illness caused by R.S. virus may occur at a very early age and that maternity units may be an important source of early infection. The mild nature of the respiratory illness contrasted with the severity of illness often caused by R.S. virus in older infants. Even in the young infant this virus was associated with clinical symptoms, and it was not found in children without respiratory symptoms. In the course of the investigation the fluorescent antibody technique, applied to aspirates from these very young infants, proved to be a rapid and efficient means of diagnosing R.S. virus infection.

In future winters it is hoped to investigate similar outbreaks, particularly to locate the source of infection and study more closely the nature of the illness caused by R.S. virus in early infancy. In view of previous suggestions on the pathogenesis of bronchiolitis, it would be very important to follow up histories of the subsequent respiratory illnesses in as many as possible of these children who have acquired an R.S. virus infection early in life.

We are indebted to the staff of the Princess Mary Maternity Hospital and of the department of virology for collection and transportation of specimens.

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REFERENCES

Berkovich, S., and Taranko, L. (1964). *Pediatrics*, **34**, 753.
Gardner, P. S., and McQuillin, J. (1968). *British Medical Journal*, **3**, 340.
Gardner, P. S., and McQuillin, J. (1970). Unpublished laboratory data.
Gardner, P. S., McQuillin, J., and Court, S. D. M. (1970). *British Medical Journal*, **1**, 327.
Javett, S. N., *et al.* (1956) *Journal of Pediatrics*, **48**, 1.
Kuroya, M., Ishida, N., and Shiratori, T., (1953). *Yokohama Medical Bulletin*, **4**, 217.
McQuillin, J., and Gardner, P. S. (1968). *British Medical Journal*, **1**, 602.
Sturdy, P. M., McQuillin, J., and Gardner, P. S. (1969). *Journal of Hygiene*, **67**, 659.

Medical Memoranda

A Sign in Gall-bladder Disease

British Medical Journal, 1970, **3**, 147–148

Doctors do not often see the initial stages of acute abdominal disease, for patients delay in sending for medical aid, and doctors cannot get there at once. Yet the earliest symptoms and signs are important.

During a long clinical career I never saw the very beginning of an attack of acute cholecystitis. But I recently corrected this omission when I myself suffered from that disease. An account of my experience may perhaps be of interest.

CASE REPORT

The only previous abdominal crisis I had suffered was an attack of acute gangrenous appendicitis in 1907; an abscess formed and was drained, and later the stump of the appendix was removed at another operation.

For six months before my recent illness I occasionally had attacks of profuse sweating and increased pulse rate that woke me from sleep, but usually passed off within half an hour. They were not accompanied by any pain. A physician found my heart normal.

On 3 April, 1969 I arose about 7 a.m., enjoyed a simple breakfast, and was up and about all the morning. Then, almost exactly at noon, I suddenly felt a dull severe, deep pain in the middle of the epigastrium. I lost all appetite for food and retired to bed. I wondered what might be the cause of the pain, and thought first of coronary thrombosis, but the pulse was normal in every respect, and there were no symptoms to support this diagnosis. My attention was then directed to the abdomen. I palpated the left side and the right iliac region without finding anything abnormal. In the right hypochondrium, however, a surprise awaited me, for in the normal position of the gall bladder was a rounded, tense, and firm swelling, about the size of a small golf-ball. It was not painful or tender. This absence of tenderness probably put all thoughts of acute cholecystitis out of my mind. I decided to phone an experienced medical practitioner, and he arranged to visit me that afternoon. As I rested in bed the epigastric pain became easier and I dozed a little on and off, and did not even once feel if the abnormal swelling had changed. The physician arrived later in the afternoon, examined the abdomen very carefully, but found no abnormal swelling in the gall-bladder area, nor anywhere else in the abdomen. This greatly surprised me, but I was able to confirm that the swelling I had felt had now completely disappeared. Both pain and swelling having now gone it was agreed that there was no need for any special line of treatment.

I remained comfortable and free from pain for four to five hours. But about 9 p.m. I began to have pain in the right hypochondrium and tenderness in the same area. The pain increased in severity, so I phoned the physician, told him of the change, and asked him to pay me another visit on the morrow. The second visit was paid on 4 April. When the physician saw the position of the pain and tenderness, and found I had a slight rise of tempera-