radiographic appearances these were attributed to trauma. In contrast, only 8·7% of our patients with alcoholic liver disease had detectable rib fractures. This difference may reflect the different populations studied. Patients with alcoholic liver disease admitted to the liver unit usually present with symptoms of chronic liver disease; they are rarely severely dependent on alcohol,[5] and only 5% report previous episodes of trauma. Alcoholic patients presenting to general gastroenterology wards or to alcoholism units with psychosocial problems relating to their alcoholism[2] are perhaps more likely to be severely dependent and to have suffered trauma during accidental falls or fights or as a result of withdrawal fits. Although cirrhosis was present in 63% of the group with alcoholic liver disease compared with 18% of the group with non-alcoholic liver disease, there was no association between the presence of fractures and the severity of liver disease.

The low sensitivity of fractures on chest *x* ray films for diagnosing alcoholism in patients with liver disease in the present study indicates that chest radiography has limited application in detecting patients with alcohol problems. When fractures are present they may alert the radiologist and clinician to the possibility of alcoholism, but the diagnostic yield is not sufficiently great to justify the use of chest radiography when it would not otherwise be undertaken.

We thank Dr B Portmann for reviewing the liver biopsy specimens. This study was supported in part by the Department of Health and Social Security and the Brewers' Society, London.

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(*Accepted 27 October 1983*)

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Postperinatal mortality among infants discharged from special care units

With improving neonatal care more babies of low birth weight survive into infancy. Although much is made of the reduction in perinatal mortality, little is known of the long term survival of babies discharged from intensive care units. We report on the mortality among babies discharged from one such unit.

Patients, methods, and results

We studied all postperinatal deaths (from one week to one year) that occurred among children born from 1 January 1977 to 31 December 1981 to parents resident in the Southampton health district including those who were resident outside the health district at the time of death (data from the Office of Population Censuses and Surveys). Name, age, sex, cause, and place of death were detailed in each case. For babies who had been admitted to the special care unit the following information was obtained from hospital records: date of birth, type of pregnancy (single or multiple), birth weight, duration of stay in unit, reason for admission, and treatment given.

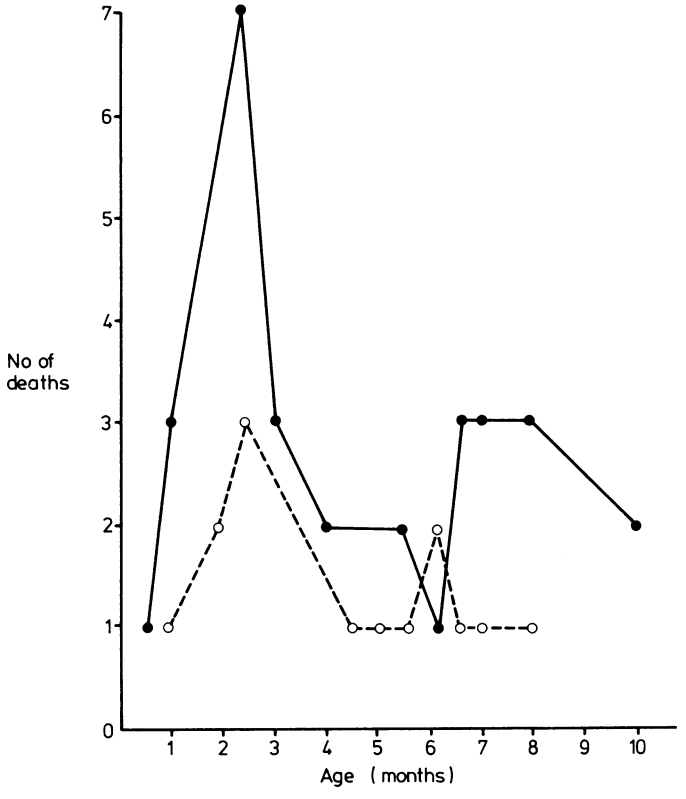
Of 2840 babies discharged from the special care unit, 30 (10·6/1000) died postperinatally, compared with 132 (6·3/1000) out of 20 939 babies not admitted. Deaths after discharge from the unit thus accounted for 18·5% of all postperinatal deaths and consisted of 20 (67%) sudden deaths at home and 10 (33%) in hospital, the ratio of boys to girls being 6:4.

Of the 20 deaths at home, 14 were "cot deaths," three babies had congenital heart disease (one having Down's syndrome), one had suffered perinatal brain injury, and two died accidentally. Fourteen of those who died at home had been low birthweight preterm babies, six of whom were one of twins. Ten of the 14 cot deaths were in low birthweight babies, including all six twins. These 14 cot deaths together with a further 35 among babies who had not been admitted to the unit gave an overall incidence of cot death of 2:1000 live births (4·9:1000 discharged from the unit, and 1·7:1000 others).

Of the 10 babies discharged from the unit who died in hospital, six had congenital deformities, one birth asphyxia, one meningococcal septicaemia, one bronchopulmonary dysplasia, and one hydrocephalus after meningitis; six had been low birthweight infants.

When the duration of stay in the special care unit was calculated the 30 babies who died had spent a total of 951 days in hospital (mean 31·7 days/baby). The 14 presenting as cot deaths stayed 237 days (16·9 days/baby) while the others stayed 714 days (44·6 days/baby).

The figure shows the ages at death (corrected for gestation) of babies discharged from the special care unit, with a peak in postperinatal deaths at 2-3 months.



Ages (corrected for gestational age) at death of 30 babies discharged from special care unit (●—●); 14 cot deaths shown separately (○ - - - ○).

Comment

Babies discharged from special care units continue to have a higher postperinatal mortality than the rest of the population, in spite of the quality of care provided immediately after birth. None the less, the 68% increase in mortality shown in this study compares favourably with the tenfold increase found by Kulkarni *et al*[1] and Sills *et al*.[2]

The pattern of deaths after discharge from the unit was similar to that of postperinatal deaths generally,[3] with a preponderance of boys, a high proportion of deaths at home, and a peak at 2-3 months. This pattern suggests an overlap of factors relating to the outcome of pregnancy and deaths of infants.

A striking feature was the relatively high number of cot deaths after discharge which accounted for nearly a third of all cot deaths. Of the babies presenting as cot deaths, only one had a bradycardic episode and one a hypoglycaemic attack. This strengthens the view that neonatal apnoea and cardiac arrhythmias are not major factors in cot deaths.

Babies discharged from intensive care units contribute appreciably to postperinatal mortality, and particularly cot deaths, in a way not yet completely understood. Further study of physiological mechanisms in this group of vulnerable babies could, however, provide vital clues about deaths of infants generally.

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(*Accepted 24 October 1983*)

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Incidence of cardiac arrhythmias in patients taking slow release salbutamol and slow release terbutaline for asthma

The sustained release β_2 adrenergic agonists terbutaline and salbutamol are prescribed to prevent early morning attacks of asthma. We undertook a study to establish the incidence of cardiac arrhythmias caused by these drugs in a group of otherwise healthy patients with asthma.

Patients, methods, and results

Sixteen patients with asthma (11 men, five women; mean age 39 (range 19-60)) took part in a single blind crossover study lasting 14 days. None smoked or had clinically apparent cardiac disease. Treatment with cromoglycate (two patients) and steroid aerosols (seven) was allowed to continue during the study. One patient was taking a stable oral dose of steroids. All 16 patients were using a salbutamol aerosol before the study.

Patients underwent initial 12 hour Holter electrocardiography overnight and were then randomly allocated to receive either salbutamol sustained release tablets 8 mg at night with a salbutamol metered dose inhaler (100 μg/dose) as required or terbutaline sustained release tablets 7·5 mg twice daily with a terbutaline metered dose inhaler (250 μg/dose) as required. Each course of treatment lasted seven days, on the last night of which a 12 hour electrocardiogram was recorded. The patients were then changed to the other drug for the following week, on the last night of which a final 12 hour electrocardiogram was made. All electrocardiograms were analysed on a computerised system (Reynolds Pathfinder) by one of us (RH), who was unaware of which drug treatment (if any) each patient had received. The presence of any cardiac arrhythmia and the maximum heart rate attained during electrocardiography were noted. Patients were also provided with mini Wright peak flow meters and asked to record their peak expiratory flow rate daily on waking and going to bed.

The maximum heart rate during electrocardiography in the 16 patients increased from a baseline mean of 116 (SD 18) beats/min to 126 (19) after terbutaline and 122 (18) after salbutamol, but neither of these increases was significant. Five patients showed arrhythmias on one or more occasions (table). Three patients showed abnormalities on the electrocardiogram before receiving either drug. Five patients showed arrhythmias while receiving salbutamol and three while receiving terbutaline. Ventricular premature beats developed in the same three patients with both sustained release preparations, but the two patients with atrial extrasystoles on their baseline electrocardiograms developed paroxysmal atrial tachycardia only when receiving salbutamol. Palpitations did not occur during the two week study, although one patient had tremor.

Comment

Oral slow release β_2 adrenergic preparations have been reported to cause arrhythmias in patients with chronic obstructive airways disease,[1 2] and the risks of these drugs in patients with ischaemic heart disease or a predisposition to it have been highlighted. Our study shows that arrhythmias may develop with or be exacerbated by both salbutamol and terbutaline in patients with asthma who are otherwise healthy.

There are several possible reasons for the increased incidence of arrhythmias other than the addition of the oral slow release β_2 agonists. Firstly, the patients' asthma may have deteriorated during treatment, leading to hypoxaemia; this may have been the case in two patients. Secondly, serum electrolyte concentrations, particularly potassium concentrations, may have altered. These were not measured as it is not our routine practice to do so before altering treatment with bronchodilators in outpatients. Thirdly, the use of methylxanthines may have had some effect but these were specifically stopped two weeks before the baseline electrocardiography and throughout the rest of the study.

The clinical importance of the cardiac arrhythmias is debatable. The risk of death in patients with coronary heart disease, is reportedly increased in the presence of frequent and complex ventricular premature beats on a six hour electrocardiogram,[3] but none of our patients was known to have coronary heart disease. Further studies are therefore required to elucidate whether the arrhythmias observed with these β_2 agonists are caused or worsened by coexistant hypoxaemia or electrolyte imbalance and whether slow release methylxanthines or nebulised β_2 agonists cause fewer arrhythmias.

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(*Accepted 13 October 1983*)

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Details of patients with asthma in whom arrhythmias were observed either before or during treatment with slow release β_2 agonist preparations

Age (years)	Sex	Time of assessment	Baseline readings: Maximum heart rate (beats/min)	Baseline readings: Peak expiratory flow rate (l/min)	Readings on seventh day of treatment: Terbutaline: Maximum heart rate (beats/min)	Terbutaline: Peak expiratory flow rate (l/min)	Salbutamol: Maximum heart rate (beats/min)	Salbutamol: Peak expiratory flow rate (l/min)
27	F	Evening	132	400	145	NA	145	NA
		Morning		250	Bifocal ventricular premature beats	NA	Single ventricular premature beat	NA
42	M	Evening	104	370	172	370	110	480
		Morning	Single ventricular premature beat	350	Single and paired ventricular premature beats	340	Single ventricular premature beat	410
60	M	Evening	135	200	142	100	115	100
		Morning		150	Frequent bifocal ventricular premature beats	100	Unifocal ventricular premature beats	100
31	M	Evening	134	NA	115	NA	130	NA
		Morning	Atrial extrasystole	NA		NA	Salvos of atrial tachycardia	NA
60	M	Evening	105	370	115	280	150	280
		Morning	Atrial extrasystoles	320		270	Paroxysmal atrial tachycardia	200

NA = Not available.