During 1949-83 there were 1134 registrations for childhood leukaemias. The age adjusted rate ratios (study areas versus the rest of New Zealand) were not significantly raised during or after the greatest influx (1951-6; fig 1). For ages 0-4, 5-9, and 10-14 the rate ratios were under 1.8, and none was significantly raised. There was no significant heterogeneity in the rate ratios between time periods for any age group, but for ages 5-9 and 10-14 there were few registrations in the study areas. Subsequently we ran analyses for the 15 years 1949-63. The age adjusted rate ratio was 0.84 (95% confidence interval 0.50 to 1.44). The age specific rate ratios were: 0.92 (0.45 to 1.87) for ages 0-4 years, 0.97 (0.40 to 2.37) for ages 5-9, and 0.37 (0.06 to 2.43) for ages 10-14.

The study areas were very rural in the early 1950s, but prior population mixing would have occurred in Rotorua through tourism. Nevertheless, the age adjusted rate ratio (1949-63) was not raised in any of the three areas (including Matamata and Whakatane, where tourism was negligible).

Comment

This study does not provide support for Kinlen's hypothesis, and there are several possible explanations. There were few affected children in the study areas, so a small effect might have been missed. Bias may have been introduced by unmeasured confounders. Community size and population density are important in deter-

mining whether an infectious disease will maintain itself,⁴ and differences between the communities in this study and those studied in the United Kingdom may have produced differences in transmission of infection and different results. The relevant infectious agent may even have been absent from New Zealand in the 1950s. Alternatively, the reported relation between population mixing and childhood leukaemia could have non-infective explanations.⁵ Leaving speculation aside, the only firm conclusion of this study is that it does not support the population mixing hypothesis.

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Conflict of interest: None.

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Rupture of the uterus: a review of 32 cases in a general hospital in Zambia

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Rupture of the uterus is a major cause of maternal and perinatal deaths.¹ This study aimed to identify the risk factors associated with uterine rupture and to determine maternal and fetal outcomes.

Subjects, methods, and results

Between 1 July 1993 and 30 June 1994, 32 cases of uterine rupture were recorded at Mansa General Hospital, Zambia. This 412 bed secondary care centre receives referrals from hospitals, dispensaries, and health centres within Luapula province (population 160 000). During the study period there were 1858 deliveries, including 98 caesarean sections.

Table 1 shows the age and parity of women with uterine rupture. Age ranged from 15 to 46 years (mean 23 (SD 7) years), with a significantly higher proportion of teenagers than among all women giving birth (12/32 (38%) v 321/1858 (17%); $\chi^2=8.86$, df=1, $P<0.01$). Parity ranged from 1 to 11 (mean 2 (SD 2)). The study group contained a higher proportion of nulliparous women (14/32 (44%) v 501/1858 (27%); $\chi^2=4.4$, df=1, $P<0.05$) but not multiparous women (14/32 (44%) v 778/1858 (42%)) than overall.

Twenty patients with uterine rupture (63%) had not been booked for delivery in hospital. Rupture occurred during labour in 30 cases (94%). Rupture was associated with cephalopelvic disproportion in 24 cases (75%). Three women had previously had a caesarean section. Two ruptures occurring in hospital were due to oxytocic stimulation and assisted breech delivery with undiagnosed mild hydrocephalus. Postmortem examination of two patients who arrived in moribund condition showed a macerated fetus with its placenta outside the uterus in the abdominal cavity in one and a complete rupture in the lower uterine segment complicated with bladder injuries in the other.

Laparotomy (performed in 30 cases) showed complete uterine rupture in 21 patients (70%), located in the lower uterine segment in 16, and bladder injury in seven (23%). Procedures were carried out in 30 cases: subtotal hysterectomy in 14 cases (47%); caesarean section with uterine repair in eight (27%); uterine repair only in eight. Bladder injuries were repaired in five cases.

Postoperative mortality was high (14/32; 44%) and mainly due to sepsis (11 patients; 79%). Higher mortality was associated with nulliparity (79%), teen age (71%); and anaemia, sepsis, and shock on admission (64%). Sepsis and maternal mortality increased with time since the rupture and the distance between hospital and the patient's home or referring centre. Postoperative mortality was similar for patients booked or not (5/10 (50%) v 11/20 (55%)). One late complication, a vesicovaginal fistula, was due to bladder necrosis caused by prolonged fetal head compression before operation.

Mortality did not differ significantly in patients who underwent hysterectomy and those treated by uterine repair (5/14 (36%) v 11/16 (69%); $\chi^2=3.28$, df=1, $P>0.05$). Fetal mortality was 100%.

Comment

Because of inaccessibility of essential health information, lack of transportation, poor access to fixed health care services, and shortcomings in the supervision and monitoring of home deliveries, uterine rupture is common in rural and illiterate women.^{2 3} Those most

Table 1—Age and parity of women with uterine rupture, 1 July 1993 to 30 June 1994, Mansa General Hospital, Zambia

Age (years)	Parity										
	0	1	2	3	4	5	6	7	8	9	10
15-19	12										
20-24	1	3	3	2	1						
25-29	1	1	2	1							
30-34						1	1				
35-39										1	1
≥40							1				

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affected are primigravid teenagers living far from hospital and relying on a traditional birth attendant who is unable to diagnose cephalopelvic disproportion.

When labour is prolonged and delivery delayed, herbal preparations with oxytocic and analgesic components are given orally by traditional birth attendants or inserted into the vagina, leading to uterine hypertonia, ruptured uterus, and fetal death.⁴ This is less frequent in multiparous women, where cephalopelvic disproportion is rare and labour proceeds quite quickly. Mortality was similar in booked and unbooked patients because many women attempted to deliver at home with traditional midwives, whose hazardous practices can lead to uterine rupture, sepsis, and shock.

The health education of rural people, the training and supervision of traditional birth attendants, and availability of transport may reduce the incidence of uterine rupture in remote areas. Particular attention to

regulation of training and practices of traditional birth attendants is required. Maternal prognosis may improve if women marry later and if family planning and antenatal and delivery care are available near women's homes and are affordable.

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Effect of inadvertent intradermal administration of high dose percutaneous BCG vaccine

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In the United Kingdom it is recommended that all children receive BCG vaccination to prevent development of tuberculosis.^{1,2} The standard route of administration is intradermal. In neonates a 10 times more potent percutaneous preparation is used, a small volume being injected to a depth of 2 mm with a modified Heaf gun. The two preparations have similar packaging and labelling and so may easily be confused.³ We report the outcome of inadvertent intradermal administration of the stronger percutaneous product and define the relation between the development of skin lesions and the dose of vaccine.

Subjects, methods, and results

Thirty two school children aged 11-14 received 0.1 ml of BCG vaccine intradermally at a routine school immunisation session. Nineteen received the preparation for percutaneous administration, which contains 50-250 million colony forming units per ml. Thirteen received the correct preparation, which contains 8-26 million colony forming units per ml (Evans Medical, Leatherhead, England). The error occurred because of the similar packaging and labelling of the two products. All parents and children were informed of the error and all agreed to a period of close observation. All observations were performed by the same observer (MMM). The study was not blind since a component of the consultation was to discuss any concerns with children and parents.

The area of induration increased progressively and was significantly greater in the group who had received the percutaneous vaccine intradermally (fig 1). Mean diameters were 0.96 cm (range 0.8-1.3, $P<0.02$ by Student's *t* test) at two weeks, 1.15 cm (0.9-1.5, $P<0.01$) at three weeks, and 1.23 cm (1.0-1.6, $P<0.01$) at six weeks; the mean diameter had decreased to 0.91 (0.6-2.2) cm by 15 weeks. By contrast, the mean diameter of the induration in those receiving the correct intradermal vaccine was 0.83 cm (0.7-0.9) at two weeks, 0.83 cm (0.6-1.1) at three weeks, and 0.77 cm (0.5-1.1) at six weeks.

The sequence of symptoms was itchiness, soreness, and discharge. These were similar in both groups, although discharge was more common at six weeks in the group given the percutaneous vaccine (nine out of

Fig 1—Mean (SE) diameters of skin lesions after intradermal BCG vaccine and percutaneous vaccine given intradermally

15 children v one out of 10 children). Ten of the 15 children given the percutaneous vaccine were examined at 28 weeks. They complained of no symptoms, and mature scars had formed (mean diameter 1.2 cm (range 0.8-2.0)).

Comment

This study emphasises the requirement for clear packaging and labelling of drugs. If subjects inadvertently receive the percutaneous BCG vaccine intradermally they may, however, be reassured that the lesion will resolve over a few months without treatment. Although the lesions were sometimes painful, the induration was larger, and discharge was more common after six weeks when the high dose preparation was given intradermally. The lesions had started to resolve at 15 weeks.

The results also suggest a clear dose-response relation in vivo between the size of the inflammatory response and the dose of vaccine. We do not know whether there is a relation between the size of the inflammatory response and the protective effect of BCG or the size of the scar.

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Conflict of interest: None.

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