abnormality. Histological, microbiological, and chemical pathological examination yielded unremarkable results, and the sudden infant death syndrome was diagnosed by exclusion.

A few months later at a confidential survey of postperinatal mortality we were informed of the high rectal temperature and the fact that the child had slept in a carry cot placed on an uncarpeted floor equipped with off peak heating controlled with a time switch.

As one of us (MAG) had experience of severe burns sustained by adults lying unconscious on uncarpeted floors with underfloor heating we decided to determine the ambient temperatures reached in a carry cot placed on such a floor. In a maisonette equipped with similar underfloor heating the thermostat was set at 16°C, and temperature was measured with a pen recorder continuously for four days. The recorder was placed in the cot and covered with two blankets as the child had been, though the child had also been wearing a vest, a nappy, and a babysuit. The lowest temperature recorded under the blankets in the carry cot was 31°C at noon, when the heating had been off for several hours. Peak temperatures of 42°C were reached when the heating was on, and temperatures ranged from 34°C to 38°C for several hours at a time. Temperatures where the cot base touched the floor reached a maximum of 44°C and were often above 40°C.

Comment

These findings suggest that placing a carry cot directly on a heated floor is dangerous. We can find no studies or reports of similar accidents. The hazards of placing carry cots directly on floors with underfloor heating should be more widely known. This type of heating is common in council properties throughout the United Kingdom.

We thank Her Majesty's coroner for West Yorkshire for permission to report the case.

- 1 Bacon CJ, Scott D, Jones P. Heat stroke in well-wrapped infants. *Lancet* 1979;ii:422-5.
- 2 Stanton AN, Scott DJ, Downham MAPS. Is overheating a factor in some unexpected infant deaths? *Lancet* 1980;i:1054-7.
- 3 Sunderland R, Emery JL. Febrile convulsions and cot death. *Lancet* 1981;iii:176-8.

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Babywalkers and infant burns

Babywalkers are mobile metal frames supporting a plastic seat that allow infants who cannot yet walk to achieve premature bipedal motion. Increasing evidence suggests that these devices cause unacceptable levels of morbidity. British government figures, in parallel with American trends, show an increase in burns and scalds—the most disfiguring forms of injury—associated with babywalkers. Our own recent experiences confirm this.

Case reports

Between April 1986 and May 1987 five infants, aged 4 to 10 months, presented to the plastic surgery unit at this hospital with burns closely associated with the use of babywalkers (table). Four of the accidents occurred in spite of supervision by an adult at the time of injury. Three patients (cases 2, 1, and 4) required admission with 4%, 6%, and 12% burns, and the last of these needed skin grafting. Two of those admitted later developed hypertrophy of their scars. The fourth child (case 5) was lucky to escape severe injury when his walker ignited on contact with a gas fire, and in the final case healing of a contact burn was complicated by finger contracture.

Details of five infants with burns associated with use of babywalkers presenting to Leicester Royal Infirmary

Case No	Age (months)	Parent present	Mechanisms of injury	Injuries sustained	Treatment	Follow up
1	7	No	Reversed into gas fire	6% Burns to head and neck	20 Days of inpatient care	Hypertrophic scars and hair loss
2	4	Yes	Reversed into gas fire	4% Burns to head	17 Days of inpatient care	Mild scarring to cheek
3	10	Yes	Advanced into oven	Palm burns	Outpatient care	Mild finger contracture
4	9	Yes	Pulled kettle down	12% Burns to head, trunk, and arms	Resuscitation; 24 days of inpatient care; skin grafts on 18th day	Some scar hypertrophy
5	10	Yes	Reversed into gas fire. Walker seat caught fire	Minor burns to head	GP care	Well healed

Comment

An increasing number of patients with serious burns, and other injuries, associated with the use of babywalkers are presenting to hospitals in developed countries.¹ The burns sustained can lead to disfiguring scars in important places; the hands (41%) and the head (33%) are the most common.¹ Babywalkers are an inherent, but widely unrecognised, source of danger for children, who, with the dramatic increase in mobility that babywalkers give them, are becoming the infant equivalent of the adolescent motorcyclists who litter our orthopaedic wards.

Furthermore, strong evidence suggests that babywalkers actually defeat their very purpose by hindering the development of normal movement.⁴ Public information campaigns have had limited effect on home safety⁵ and are therefore unlikely to alter these burn rates. We believe that the continued sale and use of babywalkers should be urgently reviewed.

- 1 Castles M. *Home accident and surveillance systems*. London: Department of Trade and Industry, 1985.
- 2 Anderson JL. *Babywalker injuries: hazard analysis*. Washington, DC: US Consumer Products Safety Commission, 1974.
- 3 Fazen LE, Felizberto PI. Babywalker injuries. *Paediatrics* 1982;70:106-9.
- 4 Ridenour MV. Infant walkers: developmental tool or inherent danger? *Percept Mot Skills* 1982;55:1201-2.
- 5 Minchom PE, Sibest JR, Newscombe RG, Boveley MM. Does health education prevent childhood accidents? *Postgrad Med J* 1984;60:260-2.

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Alcohol and response to treatment of gout

Excessive consumption of alcohol has long been associated with gout in susceptible people.¹⁻³ Although patients with gout are generally advised to stop drinking alcohol or cut down their intake, this advice may not always be followed. We looked at the relation between alcohol intake and the response to treatment of gout.

Patients, methods, and results

Thirty eight patients with gout who had attended this centre in 1987 were identified by our diagnostic index. A history of alcohol intake had been taken at the first visit to the clinic in all cases, and at subsequent visits those patients who were suspected on the basis of their history or laboratory abnormalities (abnormal liver enzyme activities or increased mean cell volume) to be consuming excessive amounts of alcohol were routinely asked about their intake. Twenty one patients were heavy drinkers (30 or more units of alcohol a week), eight were moderate drinkers (less than 20 units weekly), and nine seldom or never took alcohol. Information on current alcohol intake, drug treatment, episodes of gout in the preceding six months, and (when available) the results of biochemical and haematological investigations was taken from the case notes for the patients' most recent attendance at the clinic. The data were analysed by the Mann-Whitney U test and the χ^2 test.

The table shows clinical details and laboratory findings for patients who used alcohol heavily compared with patients who used it moderately or not at all. Patients with a heavy alcohol intake had significantly higher transaminase and γ -glutamyltransferase activities, uric acid concentrations, and mean cell volumes than the patients who used alcohol moderately or not at all. Some of the moderate users and non-users had increased γ -glutamyltransferase activity, which suggested that the stated alcohol intake was not always the true intake. Twenty (95%) of the heavy drinkers had continued to have acute attacks of gout despite

treatment, compared with five (29%) patients who used alcohol moderately or not at all ($\chi^2=18.09$, $df=1$, $p<0.001$). Two of the five moderate drinkers who continued to have attacks of gout had raised γ -glutamyltransferase activity (71 and 141 U/l; normal range <36).

Comment

The Arthritis and Rheumatism Council's information booklet on gout states that gout is probably the most easily treated form of rheumatic disease.⁴ In our study this statement was largely true for the moderate drinkers and those who took no alcohol: only five of the 17 (29%) had regular attacks of gout while receiving treatment. Two of these patients may have had a fairly high alcohol intake as their γ -glutamyltransferase activity was raised. Despite treatment with allopurinol and non-steroidal anti-inflammatory drugs 20 of the 21 heavy drinkers continued to have attacks of gout.

Median (range) clinical and laboratory variables in patients with gout

	Heavy alcohol intake (n=21)	Moderate or no alcohol intake (n=17)
Age (years)	51 (26-67)	54 (48-84)
Sex (M:F)	21:0	14:2
Body weight (kg)	77 (64-97)	80 (61-92)
Duration of gout (years)	5 (2-16)	4 (1-14)
No taking a diuretic	3	5
No taking allopurinol (300 mg daily at time of assessment)	19	14
No taking a non-steroidal anti-inflammatory drug	19	16
Mean cell volume (fl)	96 (89-107)	92 (84-104)*
γ -Glutamyltransferase (U/l)	125 (51-572)	52 (10-141)**
Aspartate aminotransferase (U/l)	39 (14-193)	26 (19-43)
Alanine aminotransferase (U/l)	34 (13-153)	23 (11-75)*
Uric acid (μ mol/l)	500 (230-770)	330 (170-600)**
Creatinine (μ mol/l)	85 (65-370)	100 (75-180)

* $p<0.05$, ** $p<0.001$, Mann-Whitney U test.

Normal ranges—cell volume 78-96 fl; γ -glutamyltransferase <36 U/l; aspartate aminotransferase 12-48 U/l; alanine aminotransferase 3-55 U/l; uric acid 170-460 μ mol/l (men), 110-370 μ mol/l (women); creatinine 50-120 μ mol/l.

The poor response to treatment that was seen in the heavy users of alcohol may have been due to antagonism of the effect of allopurinol, as ethanol contributes to hyperuricaemia both by increasing production of uric acid and by impairing its excretion in the urine.⁵ Poor compliance is another possible explanation of the poor response. In two outpatients uric acid concentrations were 680 and 770 μ mol/l despite treatment with 600 mg allopurinol daily; when these patients were admitted to hospital, however, uric acid concentrations fell rapidly to normal although the patients took only 300 mg allopurinol daily. This suggests that the failure of response had been due to failure to take the drug.

A continued high alcohol intake was associated with an impaired response to treatment of gouty arthritis. Though counselling on reducing alcohol intake was invariably given, it was obviously ignored by the heavy drinkers and management of these patients was a considerable problem. Failure of patients with gouty arthritis to respond to treatment should alert their doctors to the possibility of hidden alcohol abuse.

1 Kelley WN, Fox IH. Gout and related disorders of purine metabolism. In: Kelley WN, Harris ED, Ruddy S, Sledge CB, eds. *Textbook of rheumatology*. Vol 2. 2nd ed. Philadelphia: W B Saunders, 1985:1359-98.

2 Grahame R, Scott JT. Clinical survey of 354 patients with gout. *Ann Rheum Dis* 1970;29:461-7.

3 Reynolds PP, Knapp MJ, Baraf SB, Holmes EW. Moonshine and lead: relationship to the pathogenesis of gout. *Arthritis Rheum* 1983;26:1057-64.

4 Arthritis and Rheumatism Council. *Gout—a handbook for patients*. London: Arthritis and Rheumatism Council, 1986.

5 Faller J, Fox IH. Ethanol induced hyperuricemia: evidence for increased urate production by activation adenine nucleotide turnover. *N Engl J Med* 1982;307:1598-602.

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Virological screening for herpes simplex virus during pregnancy

Studies concerning genital infection with herpes simplex virus during pregnancy have been limited to neonatal morbidity and the frequency of asymptomatic recurrences in American obstetric populations.^{1,3} In the United Kingdom there has been only one report which failed to show asymptomatic viral shedding in a small number of pregnant women with a history of recurrent genital herpes.⁴ There has been no clearly defined policy for screening women at risk of this infection during pregnancy.

In Sheffield we have adopted not only the American practice of screening (from 32 weeks until delivery) women with recurrent disease⁵ but included those who had suffered an initial attack but were free of recurrence before conception and also those whose regular sexual partner had recurrent herpes. It has been our practice to recommend elective caesarean section for all women who have symptomatic or asymptomatic shedding evident within 21 days of the onset of labour.

Patients, methods, and results

All pregnant women attending the department of genitourinary medicine during the study period who fell into the following categories were investigated: (a) those pregnant women presenting with initial genital herpes or who were consorts of men with recurrent genital herpes; (b) those having suffered an initial or recurrent attack of genital herpes before conception. Each woman was screened when possible at 32, 34, and 36 weeks and then weekly until delivery and also during any symptomatic recurrence. On each occasion swabs were taken from the cervix and from any vulval lesion present. These were transported to the laboratory in standard transport medium (viral transport medium No 199) and cultured in human fibroblasts for 14 days.

During 1978-87, 197 women were admitted to the study. All women with genital herpes at term were delivered by caesarean section. These included four women who suffered a recurrence at term after an attack during pregnancy and one who had suffered recurrent disease before pregnancy and who went into premature labour while suffering a recurrence. Among women having suffered initial or recurrent genital herpes before conception eight were delivered by caesarean section for a clinically apparent recurrence at the time of delivery.

In women having an initial attack of genital herpes during pregnancy there was a high probability that caesarean section would be performed. Even when infection occurred before 37 weeks there was a considerable risk of recurrence at term necessitating section (table).

Results of screening for herpes simplex virus during pregnancy and mode of delivery

	Women presenting with an initial attack	Consorts of men with recurrent disease	Women with genital herpes before conception	
			Free of recurrence	With recurrences
No of patients	26	32	77	62
No of pregnancies	26	34	81	71
No (%) having recurrence during:				
1st Trimester (0-12 weeks)	—	—	6 (7.4)	10 (14.1)
2nd Trimester (13-24 weeks)	—	—	7 (8.6)	10 (14.1)
3rd Trimester (25-36 weeks)	—	—	12 (14.8)	24 (33.8)
Term (37-42 weeks)	—	—	6 (7.4)	20 (28.2)
No (%) having initial attack during:				
1st Trimester	4 (15.4)	0	—	—
2nd Trimester	5 (19.2)	0	—	—
3rd Trimester	8 (30.8)	1 (2.9)	—	—
Term	9 (34.6)	4 (11.8)	—	—
No (%) of pregnancies free of recurrence	—	—	61 (75.3)	30 (42.3)
Mode of delivery (No (%)):				
Vaginal	13 (50.0)	29 (85.3)	72 (88.9)	47 (66.2)
Caesarean section for herpes simplex virus	13 (50.0)	4 (11.8)	6 (7.4)	21 (29.6)
Caesarean section for obstetric reasons	0	1 (2.9)	3 (3.7)	3 (4.2)

No other study has considered women who have had only an initial attack of genital herpes before conception. These women were shown in our study to have a significantly smaller risk of recurrence during pregnancy than women with a history of recurrent genital herpes before conception ($\chi^2=17.2$; $df=1$; $p<0.01$). Nevertheless, the risk was still appreciable and not dependent on time since initial infection.

Comment

This study is the first to screen pregnant consorts of men with recurrent genital herpes to assess the outcome in terms of mode of delivery of the