troublesome gout, and (by all carefully checked criteria) tolerating the drug very well, have had to revert to other therapy. 2 of our patients have already had an attack of gout since restarting a thiazide. The only real justification for the sudden total ban would be if the reaction, now that it is recognised, could not be predicted by reasonable monitoring of liver function. We hope that information about this will become available quickly and that, if at all possible, the drug will again become available for use in hyperuricæmic patients who need a diuretic.

Wellcome Medical Research Institute,
University of Otago Medical School,
Dunedin, New Zealand

F. O. SIMPSON
HENDRIKA J. WAAL-MANNING

APNŒA AND UNEXPECTED CHILD DEATH

SIR,—A conference in Sheffield last July discussed the use of apnœa alarms as a possible means of preventing "cot deaths", and concluded that a controlled trial should be done in the next five to ten years. I would recommend greater urgency.

The value of a device to warn parents that their child is not breathing in time for them to restart the breathing before brain damage develops seems to depend on three things. Does the cot death population contain an unusually high proportion of infants who have had previous apnœic episodes? Are infants who have an apnœic attack severe enough to require resuscitation in danger of further apnœic spells, one of which may prove fatal? Do apnœa alarms work?

Since 1970 I have interviewed over 150 cot death parents in Auckland. 6 said that their child had had one or more previous episodes of apnœa. The parents had usually sought medical advice and been reassured that their baby was normal. Other workers have disturbing figures for subsequent deaths in babies who have had apnœic spells. Shannon et al.[2] describe 11 such infants of whom 3 subsequently died at home and Steinschneider[3] recorded 2 deaths in five "apnœic spell" infants. Guilleminault et al.[4] report the death of 1 of their "near miss" study group 30 h after polygraphic monitoring. A few weeks ago, a New Zealand general practitioner told me that he had been called on two consecutive days to a farm. On the first occasion the baby had been found cyanosed and not breathing but revived when he was picked up and shaken. The doctor saw an apparently healthy infant and reassured the parents. The following morning the baby was dead. We do not know the death rate for all babies who have experienced apnœic spells but it is apparent that some of them die suddenly and unexpectedly (as cot deaths).

Many workers have used apnœa alarms successfully for home care of near miss cot death infants.[5] For the past three years the Auckland Cot Death Society has lent Lewin type[6] apnœa alarm mattresses to 29 parents, of all educational levels; 25 were for babies who had had at least one apnœic spell requiring resuscitation and who had been judged, by monitoring of respiration or by X-rays and observation, to be at risk of further episodes.[7] Most were determined obligatory nasal breathers. The parents, before being lent the alarms, often sat up all night watching their baby. All have found the mattress alarm a great comfort: they can sleep knowing that if their baby stops breathing an alarm will sound. The alarm is usually set to ring after 15 s of apnœa.

The mattress alarms are cheaper than the impedance type and are reliable. They have been used in cars, prams, and boats. The alarm sounds if the mattress is flat, if the batteries are flat, if the baby rolls off the mattress or pulls out the plug—or if the baby is not breathing. The parents are taught resuscitation—wake the baby, pull the tongue forward, give mouth-to-nose breathing. Borrowers are given emergency numbers to ring if there is any trouble with the mattress or the baby. At least half of the mattress users have been alerted to apnœa in their baby and some infants have been resuscitated many times. Parents often stop using them when the alarm has not sounded for 4–6 weeks and the infant is 6–8 months old. The infants then respond promptly by mouth breathing when their noses are occluded.[6]

In 3 cases we have lent alarms to parents of previous cot death babies to allay excessive fears. The reassurance the alarm provides may prevent a breakdown in the mother's mental health.

We cannot yet prevent the apnœa—but we can in some cases ensure that the parents are warned in time to apply effective resuscitation and in this way lower the incidence of cot death.

Post Graduate School of
Obstetrics and Gynæcology,
University of Auckland,
Auckland, New Zealand;
and Department of Health, Auckland

SHIRLEY TONKIN

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NURSING JAUNDICED BABIES ON LAMBSKIN

SIR,—Since the letter from Scott and Richards in *The Lancet* a year ago[1] and a B.B.C. *Horizon* report on their work on television last November, the University of Otago has received requests for the unpublished work by M. Powley showing that babies in incubators on lambskin rugs made fewer gross movements than did babies on cotton sheets. This work was carried out by a psychology student (M. P.), supervised by senior lecturer in psychology (P. N.) and a neonatal pædiatrician (P. B.).

Washable sheepskin rugs for nursing bedridden patients have been available in New Zealand for many years but it was not until the late 1960s that similar rugs were produced for babies. The Parents' Centre, in collaboration with the Wool Research Organisation and the manufacturers (G. L. Bowron and Co. Ltd), pioneered the use of babycare lambskin rugs and tried to discover whether babies on lambskins cried less, and got fewer colds and rashes, than did babies on standard bedding.[2] The results were encouraging but inconclusive, because most mothers in the sample wanted to use lambskins. We wanted to carry out a well controlled experiment and one of us (P. B.) suggested using jaundiced babies in the special care baby unit at Queen Mary Maternity Hospital, who must lie naked in an incubator receiving phototherapy for several days after birth. The thirteen jaundiced babies, born between May and September, 1976, were alternately assigned to lie on babycare lambskin (4 boys, 3 girls) or on hospital cotton sheets (4 boys, 2 girls). All babies wore a blind to protect their eyes from the light, and the babies on lambskin had a napkin under their buttocks to reduce soiling of the rug and a cloth under their heads to prevent loose fibres getting in their mouths. The babies were fed at different times depending on their weight and tolerance to food.

1. Scott S, Richards M. Nursing low-birthweight babies on lambswool. *Lancet* 1979; i: 1028.
2. *Parents' Centre Bull* 1969; **39**: 12.

Crying and activity, measured on a scale from 0 to 5 modified from that of W. H. Bridger and colleagues (*Psychosom Med* 1965; **27**: 123), was recorded during two 15 min periods—the first before a midday feed (between 10.15 A.M. and 1.30 P.M.) and the second before a later feed (between 4.00 P.M. and 8.00 P.M.). Each observation was divided into 15 s periods and the most vigorous activity over each 15 s period was recorded and totalled for the half hour of observation (see table).

RESULTS

Movement	Mean scores		p (t test)
	Cotton sheets (n=6)	Lambskins (n=7)	
None	35·3	45·0	NS
Digit only	7·5	11·3	NS
Ankles or wrist	9·8	13·7	NS
One limb	14·3	20·3	NS
Two or more limbs	23·8	17·6	NS
Limbs and trunk	29·2	12·1	<0·05

This is the same pattern of activity as found by Scott and Richards.[1] Only 2 of the 7 babies on lambskins cried during the observations but 4 out of 6 cried while on the sheets. This difference is not statistically significant although it is in the expected direction.

The special care baby unit has continued to use lambskin rugs for babies in incubators since they were introduced during this experiment.

We are aware of the limitations of this small study but wish to support the findings of Scott and Richards[1] and look forward to the results of their further research.

Department of Psychology,
University of Otago,
Dunedin, New Zealand — MELANIE POWLEY, PAULINE NYE

Department of Pædiatrics,
Medical School,
University of Otago — PATRICIA BUCKFIELD

CIMETIDINE IN HYPERPARATHYROIDISM

SIR,—*The Lancet* occasionally publishes new and useful information which would have been rejected by journals less inclined to leaps from high places. However, you also publish from time to time papers that are dangerously misleading: the article by Dr Sherwood and her colleagues (March 22, p. 616), on cimetidine treatment of primary hyperparathyroidism, falls into this category. This paper is marred by non-sequiturs and misconceptions about parathyroid physiology,[1] the relation of plasma calcium levels to symptoms,[2] and the use and abuse of radioimmunoassays for parathyroid hormone.[3] I would like to point out three serious problems:

(1) Four of the patients were normocalcæmic, and were diagnosed by parathyroid hormone immunoassay. This unprecedented manœuvre[3] seriously calls into question the diagnosis of primary hyperparathyroidism and the value of the assay used.

(2) Serum calcium was "reduced" in all the treated patients, but "hovered" at 11·0–11·4 mg/dl, suggesting that any reductions of immunoreactive parathyroid hormone were unaccompanied by true improvement of the hyperparathyroid state. In other words, the hallmark of primary hyperparathyroidism—hypercalcæmia—was not abolished by the treatment.

(3) I know, through our group's studies in familial primary hyperparathyroidism and the recent paper by Palmer et al.,[4] of at least 18 hyperparathyroid patients in the U.S.A. whose disease was entirely unaffected by cimetidine treatments.

Until better data are available, cimetidine has no place in the clinical management of primary hyperparathyroidism.

Endocrine Research Unit,
Department of Medicine,
Mayo Clinic,
Rochester, Minnesota 55901, U.S.A. — HUNTER HEATH, III

⁂ A late printing error marred the Results section of this paper: line 2 on p. 618 should read ". . . there was *no* rebound hypocalcæmia or tetany". We apologise to Dr Sherwood and her colleagues. —ED. L.

SIR,—Cimetidine controlled the hyperparathyroidism in all 12 patients in Dr Sherwood's series, and it was effective in 8 of 12 other cases.[5] But the comparison made by Sherwood et al. between this drug and methimazole was not pursued far enough.

Cimetidine blocked the secretion of parathyroid hormone (PTH) produced from the adenomas, but it did not block the PTH secreted by the normal parathyroid cells. Indeed, the hypocalcæmia, seen in about a third of cases after parathyroid adenectomy, did not develop postoperatively in any cimetidine-treated patients. This paradox is best explained by the thyroid analogy. Thyroid stimulating immunoglobulins are the usual cause of hyperthyroidism, and a similar immune mechanism very probably induces primary hyperparathyroidism. Serum antibodies to parathyroid cells are occasionally found, but no stimulatory immunoglobulins have been detected. The parathyroids are unlikely to be the exception among the other endocrine glands, however. They are embryologically, anatomically, and functionally close to the thyroid gland. The half-life of PTH is much shorter than that of thyroxine, and the brief survival of PTH in the bloodstream may explain why serum antibodies are rarely found. It may also be the reason why stimulatory antibodies have not been isolated.

There is some indirect evidence for a common immune process affecting both the thyroid and parathyroid glands. Parathyroid hyperplasia may sometimes precede the formation of adenomas, and these are probably not totally autonomous.[6] When Buckle et al.[7] showed that PTH was released from a renal adenocarcinoma, I suggested an immune stimulus as the primary cause:[8] the PTH obtained from the adenomas, might have come from previously bound stimulatory antibody. In sarcoidosis, for instance, 32 cases were recorded with hyperthyroidism, while I have come across another 16 published cases of sarcoidosis with parathyroid hyperplasia or adenomas. Since hyperimmunity is a feature of sarcoidosis, the hypersecretion from both glands may have been generated by local sarcoid granulomas secreting immunoglobulins. 4 of Sherwood's patients were normocalcæmic before treatment, and their high serum PTH levels were probably maintained only at the expense of the normocalcæmia and relative vitamin D_3

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