

Failure to thrive has been described in association with chronic intussusception in one previous report.¹ The authors described three children, aged 10 months to 2.5 years, with clinical histories of seven to 18 weeks' duration. Weight loss and emaciation were described together with abdominal pain and vomiting. Vomiting was a late symptom in our case, and abdominal pain absent.

An unusually high incidence of chronic intussusception and subacute intussusception was described in a study of 62 children from Nigeria.⁴ A high incidence of 'painless intussusception (41%)' was reported. Seven patients with an average age of 5.5 years had a chronic history with a symptom duration of three weeks to three years illustrating the increased incidence of chronic intussusception in the older child.¹ Anorexia, weight loss, and frequent mucoid or bloody stools were the prominent symptoms. This contrasts with our case in which the stools were never mucoid and always occult blood negative.

Incomplete intussusception may be associated with diarrhoea,⁵ and it is possible that the diarrhoea in our case was related to a developing intussusception.

Ultrasound has a recognised place in the diagnosis of intussusception. Although the

identified mass appeared to be related to bowel in our patient, the echo pattern was not characteristic enough to allow a specific diagnosis. In view of the chronic history a bowel associated malignancy was considered more likely.

Barium enema is the investigation of choice in the diagnosis of intussusception and in acute intussusception attempted barium reduction is advocated if the patient is clinically fit.⁶ In the child with failure to thrive and no specific features to suggest intussusception, however, a barium meal and follow through will probably be chosen. The diagnosis will be missed if a barium follow through is not performed.

Chronic intussusception is more commonly associated with a predisposing lesion and operative reduction is the appropriate treatment.¹

- 1 Rees BI, Lari J. Chronic intussusception in children. *Br J Surg* 1976;63:33-5.
- 2 Rafinesque FG. *Etude sur les invaginations intestinales chroniques*. Paris: JB Baillière et fils, 1879.
- 3 Macaulay D, Moore T. Subacute and chronic intussusception in infants and children. *Arch Dis Child* 1955;30:180-3.
- 4 Momoh JT, Lawrie JH. Tropical paediatric intussusception—is it a different disease entity? *Ann Trop Paediatr* 1981;1:237-40.
- 5 Ein SH, Stephens CA. Intussusception in 354 cases in 10 years. *J Pediatr Surg* 1971;6:16-27.
- 6 Mackay AJ, MacKellar A, Sprague P. Intussusception in children a review of 91 cases. *Aust NZ J Surg* 1987;57:15-7.

White noise and sleep induction

J A D Spencer, D J Moran, A Lee, D Talbert

Abstract

We studied two groups of 20 neonates, between 2 and 7 days old, in a randomised trial. Sixteen (80%) fell asleep within five minutes in response to white noise compared with only five (25%) who fell asleep spontaneously in the control group. White noise may help mothers settle difficult babies.

Records of intrauterine sounds calm babies and one study found that four out of five infants fell asleep,¹ although the effectiveness of such noise has often been judged solely by the mother.² Quiet sleep in 4 day old neonates was reached significantly sooner and its duration was prolonged by 20% when exposed to continuous white noise.³ We assessed a white noise device, suitable for domestic use and designed to calm babies and promote sleep, by performing a study in which normal neonates were exposed, at random, to a short period of white noise.

Methods

The acoustic output of the commercially available, self contained, battery operated white

noise generator (Babyshh, Egnell-Ameda Ltd) was measured in the acoustic laboratory, Department of Health Supplies Technology Division, Russell Square, London, using the Bruel and Kjoer generating and measuring equipment normally used for assessing deaf aids.

Permission to perform a clinical trial was obtained from the local ethics committee and 40 healthy neonates, born at full term and between 2 and 7 days old, were studied after informed consent had been obtained from the mothers. The white noise device was placed in the cot, between 12 and 20 inches from the baby's head, and either switched on or left off at random according to allocation cards concealed in envelopes. The baby's state was observed continuously for five minutes by a single investigator who noted whether or not the baby was asleep after the four and a half minutes of white noise. Sleep was defined as a state of quiescence with eyes closed and regular breathing.⁴

The first 20 babies had a continuous record of heart rate made during their studies using electrocardiogram electrodes on the chest and an FM7 monitor (Oxford Sonicaid Ltd).

Institute of Obstetrics and Gynaecology, Royal Postgraduate Medical School, Queen Charlotte's and Chelsea Hospital, London

J A D Spencer
D J Moran
A Lee
D Talbert

Correspondence to:
Mr J A D Spencer,
Institute of Obstetrics
and Gynaecology,
Royal Postgraduate
Medical School,
Queen Charlotte's and
Chelsea Hospital,
Goldhawk Road,
London W6 0XG.

Accepted 4 August 1989

Characteristics and results of the two groups of neonates

	Exposure to white noise		Significance* (p value)
	Yes (n=20)	No (n=20)	
No (%) of vaginal deliveries	12 (60)	14 (70)	>0.5
No (%) of boys	11 (55)	12 (60)	>0.5
Mean (SD) birth weight (g)	3595 (422)	3531 (453)	>0.5
Mean (SD) age when tested (days)	3.1 (1.9)	2.9 (2.0)	>0.5
Mean (SD) interval since last feed (mins)	43.3 (63.2)	34.0 (34.0)	>0.4
No (%) having breast feeds	6 (30)	7 (35)	>0.5
Effect of white noise:			
No who fell asleep	16 (80)	5 (25)	<0.001
No who stayed awake	4 (20)	15 (75)	

*The groups were compared using the two sample *t* test for means and Fisher's exact test for counts. The effect of white noise was assessed by the χ^2 test.

The two groups were compared using the two sample *t* test and Fisher's exact test. The results, after allocation to white noise or no noise, were evaluated using the χ^2 test and expressed as a relative risk. The number of babies studied was based on an expected three fold increase in the number of babies falling asleep (from 25% to 75%) which would be significant at the 5% level with a power of 95%.

Results

When suspended in the open at a distance of 30 cm (12 inches) the sound intensities measured 72.5 dB for the first 30 seconds and 67 dB for the remaining four minutes. Most of the sound energy lay in the spectral band between 500 Hz and 9 KHz.

Randomisation produced two groups of babies similar in a number of characteristics, as shown in the table. The results of allocation to white noise or no noise are also shown in the table where it can be seen that 16 (80%) babies fell asleep when the device was turned on compared with five (25%) who fell asleep in the control group. This difference was significant ($\chi^2=12.13$, $df=1$, $p<0.001$). The relative risk was 3.2, with a 95% confidence interval of 1.5 to 7.0, indicating that more than three times as many babies fall asleep with the use of white

sound than without it. Of the babies still awake in the control group 11 (73%) fell asleep when the device was switched on, this response rate being similar to the initial results. Two babies in each group were still crying after five minutes and these babies all settled after a feed.

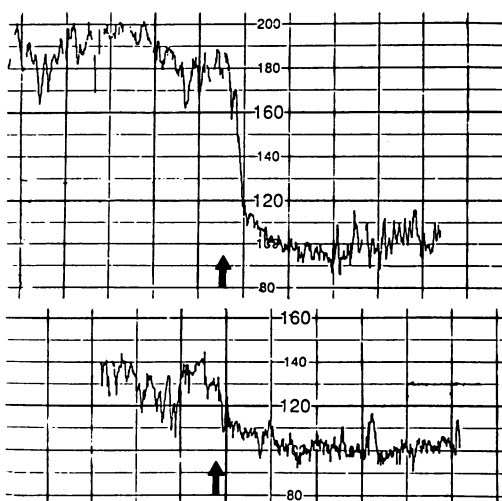
After introduction of the white noise the babies who settled were all asleep within two minutes. The heart rates of the monitored babies who responded settled from between 120 and 180 beats per minute to between 100 and 110 beats per minute, as illustrated in the figure for two of them.

Discussion

The intensities produced by the white noise device used in this study correspond to the noise level of a domestic vacuum cleaner or inside a small saloon car travelling at 50 kph.

This randomised study has shown that, when exposed to white noise, the likelihood of a baby falling asleep is increased more than three fold (25% to 80%). Those that responded did so within a short period of time and before the white noise device ended its emission. Low frequency noise is known to be a more effective inhibitor of behaviour than high frequency sound,⁵ and both continuous and pulsatile sounds have this effect. The noise of hair dryers and vacuum cleaners is known to settle infants and promote sleep. White noise probably acts by masking other external noises thereby removing such arousal stimuli and calming the baby.

We found that white noise promoted sleep only in babies who were not hungry. Two of the babies who did not respond initially settled after a feed. Similarly, two in the control group did not settle when the device was subsequently switched on because they required feeding. Therefore it seems unlikely that use of this device will deprive infants of feeds, should they be required. This is further evidence to suggest that white noise, used for induction of sleep in babies, does not suppress intrinsic stimuli. Total crying time has been found to be related to the method of feeding, such that bottle fed babies cried only two fifths as much as breast fed babies who required complementation by bottle.⁶ Our study of the effect of white noise in promoting sleep in normal neonates suggests it may be of benefit to mothers who have difficulty in settling their baby after a feed.



Two records of neonatal heart rate (vertical divisions = minutes) showing the effect of introducing white noise (arrows). The babies were asleep within two minutes.

- 1 Murooka H, Tsutoma A, Tsuyoshi S, Yusuo I, Mitsukazu N, Nobuhiro S. Induction of rest and sleep in the neonates by the rhythm of the maternal blood flow. *Nippon Ika Daigaku Zasshi* 1975;42:77-9.
- 2 Callis PM. The testing and comparison of the intra-uterine sound against other methods for calming babies. *Midwives Chronicle* 1984;97:336-8.
- 3 Murray B, Campbell D. Sleep states in the newborn: influence of sound. *Neuropadiatrie* 1971;2:335-42.
- 4 Prechtl HFR. The behavioural states of the newborn infant (a review). *Brain Res* 1974;76:185-212.
- 5 Birns B, Blank M, Bridger WH. Behavioural inhibition in neonates produced by auditory stimuli. *Child Dev* 1965;36:639-44.
- 6 Wood C, Kempson C, Beech C, Brooke OG. An investigation of crying in the newborn in relation to maternal and infant factors. *Br J Obstet Gynaecol* 1987;7:201-5.

Platelet antigens in varicella associated thrombocytopenia

J Winiarski

Abstract

Serum IgG or, predominantly, IgM antibody binding to electrophoretically separated normal platelet membrane protein antigens were detected by immunoblotting in five children with thrombocytopenia associated with varicella. Glycoproteins GPIb, GPIIb, GPIIIa, and other 25-260 kilodalton (kDa) proteins were identified as target antigens, suggesting a transient autoimmune mechanism causing the thrombocytopenia.

Purpura is a well known complication of varicella but the mechanisms causing thrombocytopenia have not been fully elucidated. Thrombocytopenia may be caused by either immune mediated platelet destruction¹ or direct viral interaction with megakaryocytes or platelets.² Circulating platelet binding antibodies and increased platelet associated immunoglobulins have been described,¹ but the antigenic specificity of the putative platelet binding antibodies has remained unknown. Specific antibodies to defined platelet antigens in thrombocytopenia associated with varicella are reported here.

Patients and methods

Blood was drawn from three boys and two girls, aged 1.5 to 11 years, within a week after onset of purpura, that presented three to seven days after the eruption of a varicella exanthem. All children were previously untreated and in good health. Nadir platelet counts ranged from $2-7 \times 10^9/l$. A bone marrow examination was performed only in case 3 who, after seven days of purpura, had slightly decreased megakaryocytes. Platelet associated immunoglobulins were increased in three out of three tested patients. The chicken pox was in all cases uncomplicated apart from thrombocytopenia, which resolved (platelet count $>150 \times 10^9/l$) within nine to 30 days. Serum samples and normal platelet counts were obtained from four of the children one to seven years later. Sera from 24 healthy blood donors were used as negative controls and anti-PI^{A1} sera as positive controls. Separation of

platelet membrane glycoproteins using sodium dodecylsulphate-polyacrylamide gel electrophoresis (SDS-PAGE) and immunoblotting were performed as described elsewhere.³ Briefly, pooled normal platelet membranes were dissolved in SDS and applied to a slab gel together with molecular weight standards. SDS-PAGE was performed under non-reducing conditions using the discontinuous buffer system of Laemmli. The separated proteins were electrophoretically transferred from gels to nitrocellulose membranes and blocked in fat free milk. Membrane strips were incubated with patient or control sera, diluted 1:25. After several washes the blots were first incubated with alkaline phosphatase conjugated protein A or rabbit antihuman IgM and then stained after addition of substrate for the detection of glycoprotein bound antibodies. Glycoproteins were identified on parallel blots by alkaline phosphatase conjugated Lens culinaris lectin which binds to platelet glycoproteins Ib, IIb, and IIIa. To confirm the specificity for platelet surface antigens, sera were absorbed with fresh platelets³ and used in parallel with unabsorbed sera. Sera were also screened with a solid phase platelet membrane enzyme linked immunoadsorbent assay (ELISA).⁴ All positive results were confirmed on repeat testing.

Results

By immunoblotting, platelet glycoprotein binding IgG was detected in sera from three patients and IgM in five patients. IgM corresponded in part to the IgG bands, but additional reactions were noted. The bands comigrated with GPIb (170 kDa), GPIIb (140 kDa), and GPIIIa (95 kDa) in four patients. Other unidentified 25-260 kDa bands were also seen (figure and table). Absorptions of sera with packed platelets eliminated antibody binding to glycoproteins Ib, IIb, IIIa and to the other proteins indicating specificity for native surface antigens. By ELISA, four children were shown to have membrane binding antibodies. In sera collected one to seven years later, the earlier positive reactions were mostly weak or no longer detected (table). In case 4, IgG platelet

Departments of Paediatrics and Clinical Immunology, Karolinska Institute at Huddinge Hospital, Sweden

Correspondence to: Dr J Winiarski, Department of Clinical Immunology, Huddinge Hospital, S-141 86 Huddinge, Sweden.

Accepted 29 August 1989