PAPERS AND ORIGINALS

Are breast-fed babies still getting a raw deal in hospital?

PHYLLIS CULLEY, PAMELA MILAN, CLAUDIA ROGINSKI, JOHN WATERHOUSE,
BEN WOOD

British Medical Journal, 1979, 2, 891-893

Summary and conclusions

After preliminary validation of test weighing under ward conditions the fluid intake and weight gain of 39 breast-fed and 25 artificially fed infants were compared. All were fed every four hours for six feeds a day, and the breast-fed infants received dextrose supplements only. The average intake and weight gain of the breast-fed group was significantly less than that of the group fed artificially.

When cows'-milk supplements are withheld from breast-fed infants a four-hourly regimen provides insufficient stimulus to lactation for their needs in the first week of life. If more than lip service is to be paid to the mother who desires to breast-feed in hospital, early and more frequent feeding should be practised.

Introduction

Despite successful demand feeding in hospital being reported in 1952¹ fairly rigid feed schedules by the clock are still widely practised on the grounds of conformity to ward routine and efficiency. Since lactation increases in response to suckling widely spaced, infrequent feeds may reduce the intake of breast-fed infants compared with those fed from the bottle.

Adequate data on the consumption of milk by infants fed

exclusively from the breast are not available²; we therefore decided to compare breast-fed and bottle-fed infants subjected to the clock-feeding regimen prevailing at this hospital during 1975-6. The fluid intake and weight gain of wholly artificially fed (AF) infants were compared with those of breast-fed (BF) babies given only dextrose as a supplement.

Methods

Preliminary study—The intake of AF babies may be measured direct from the bottle, whereas the intake of BF infants must be deduced from the weight gain after each feed. We were unable to find any reported assessment of the accuracy of test weighing, so it was necessary to reconcile the two methods by test weighing AF infants and comparing the intake as measured from the bottle. A total of 115 AF infants selected at random were studied for single feeds supervised by one of us (PM). They were weighed before and after feeds on an Avery balance scale accurate to 5 g and the difference was recorded. The amount taken from the bottle was measured direct to 1 ml. The figure gives the results, which showed a correlation coefficient of 0.83. This was thought to be sufficient to allow the comparison to proceed.

Standard feeding schedule—The BF infants were put to the breast within four hours of birth and then fed four-hourly for five feeds, starting with three minutes each side on the first day and increasing daily to 10 minutes each side on the fifth day. They were given a night feed of 5% dextrose until the fifth night, when mothers were asked if they wished to be woken to breast-feed. The AF infants were also fed about four hours after delivery and then four-hourly six times a day to supply 40 ml/kg on day 1, 60 ml/kg on day 2, and attaining 150 ml/kg on day 5. The feed was Cow and Gate Plus, reconstituted to provide 650 kcal/l (1000 kcal $\approx$ 4.2 MJ), and the volumes were calculated for the nearest 500 g of birth weight. The milk kitchen supplied bottles in 15 ml multiples, so that there was an opportunity for individual preferences, which usually erred on the generous side.

Results

Forty-two BF infants selected at random entered the study, accurate records of test weighing for all feeds from the third to fifth days being obtained for 39. Thirty babies received dextrose for all their night feeds, and 9 (23%) went to the breast on the fifth night

Birmingham Maternity Hospital, Queen Elizabeth Medical Centre,
Birmingham B15 2TH

PHYLLIS CULLEY, MB, CHB, medical assistant in paediatrics
PAMELA MILAN, SRN, SCM, research sister
BEN WOOD, DM, FRCP, consultant paediatrician (now senior lecturer in
child health, University of Southampton)

Department of Social Medicine, University of Birmingham,
Birmingham B15 2TT

CLAUDIA ROGINSKI, BSC, statistician
JOHN WATERHOUSE, MA, PHD, reader in medical statistics

Differences in weight after single feeds in 115 infants fed artificially. Some infants were test weighed on more than one occasion.

at their mothers' request. Twenty-five AF babies had their intake measured direct from the bottle, and both groups were reweighed on the sixth day for comparison with their birth weight. All infants had normal bowel function, and there were no significant differences in birth weight and gestation between the two groups.

Intake compared with sixth-day weight—In the BF group intakes on the third, fourth, and fifth days were compared separately and in aggregate with the sixth-day weight expressed as a percentage of birth weight. On analysis the mean fifth-day intake of 134 g/kg (SE 6.11) showed as good a correlation with sixth-day weight as the aggregates, so the fifth-day intake only was taken for further comparison. To permit direct comparison with the AF babies the intake of the BF group was converted from g/kg to ml/kg using our observation and that of Macy³ that 1 ml transitional milk weighs 1.035 g on average. This gave a mean intake of 129.6 ml/kg (SE 5.90) from the breast on the fifth day. There were no significant differences between the infants of primiparous and of multiparous women as regards either intake or weight.

BF compared with AF infants—The mean fluid intake and mean weight gain showed highly significant differences ($P < 0.01$ and $P < 0.001$ respectively) between the BF and AF infants (table). The BF group averaged 129.6 ml/kg, while the AF babies, who were scheduled for 150 ml/kg on the fifth day, actually received on average 160 ml/kg, suggesting that the schedule underestimated their capacity. The energy content of the two regimens could not be compared, as individual sampling was not practicable and the "crematocrit"⁴ not then available. Nevertheless, as the artificial feed provided 650 kcal/l while the dextrose given to the BF babies provided 200 kcal/l the difference in energy intake was even greater than that of the fluid.

Fifth-day fluid intake and sixth-day weight in breast-fed and artificially fed babies. Values expressed $\pm$ SE

		Mean fluid intake (ml/kg/day)	Mean sixth-day weight as percentage of birth weight
Breast-fed		129.6 $\pm$ 5.90	95.7 $\pm$ 0.54
Artificially fed		159.7 $\pm$ 6.12	99.7 $\pm$ 0.49
Probability		$P < 0.01$	$P < 0.001$

Discussion

While rapid weight gain in later infancy is not necessarily desirable, in the neonatal period a slow return to birth weight, a possible increased incidence of jaundice,⁵ and the threat of

delayed discharge from hospital increase a breast-feeding mother's anxiety with the result that milk yield falls, and some become so discouraged as to give up.

Breast-fed babies gain weight more slowly than those fed from the bottle,⁶ and Fomon *et al.*⁷ showed that this difference was apparent by the eighth day of life. Fomon also stated² that adequate data on volume of milk consumed by normal infants fed exclusively from the breast were not available. Our paper provides such data and confirms the slower weight gain even by the sixth day in breast-fed infants.

The question arises whether breast-fed babies are underfed or artificially fed babies overfed. That our artificially fed group were scheduled to receive 150 ml of cows'-milk formula per kg by the fifth day and actually received 160 ml/kg on average suggests that so far as their appetite was concerned their capacity might well have been as great as that of the bottle-fed series of Fomon *et al.*,⁸ who had a median intake of 199 ml/kg on the eighth day when fed *ad libitum*. When we consider that the breast-fed babies in our series averaged only 129 ml/kg on the fifth day there is some support for the belief that they were receiving suboptimal amounts of fluid and energy. It appears that their relatively infrequent visits to the breast failed to induce early lactation such as that reported in Dundee,⁹ where mothers breast-fed two-hourly for the first 48 hours.

The problem was accentuated in our study by the strict avoidance of cows'-milk supplements, which are often given in some centres to top up and speed discharge in so-called breast-fed babies. Thus in the series of Dahms *et al.*¹⁰ "breast-fed" infants received two full formula feeds and four breast-feeds in the 24 hours; at Queen Charlotte's Hospital, London,¹¹ nearly 80% of breast-fed infants received complementary feeds; in Newcastle¹² cows'-milk supplements were given "as necessary"; while in the series of Fomon *et al.*¹³ breast-feeding mothers were provided with a cows'-milk supplement, although not all elected to use it. As Fomon pointed out, the term "breast-feeding" needs to be carefully defined in any report on the subject.

Withholding other milk feeds from breast-fed infants originated from the belief that it stimulated hunger so that they tended to suck more effectively and so increase lactation. It has been continued because some authorities^{14 15} believe that even small amounts of heterospecific milk may reduce the anti-infective and hypoallergenic effects of breast-feeding, while even a short period of breast-feeding may protect against ulcerative colitis in adult life.¹⁶

If cows'-milk supplements are to be avoided and adequate nutrition maintained in the neonatal period, early and more frequent suckling, as practised in Dundee, followed by demand feeding, which has been shown to be feasible in Sheffield and Oxford,¹⁷ should be extended to many more maternity units such as ours in Birmingham, where less rigidity is now practised. This might allay some of the misgivings of women who are pressing for home delivery, where less rigid schedules are likely to prevail than in many hospitals at present.

One of us (PM) was in receipt of a nursing research grant from the West Midlands Regional Health Authority and from the Central Birmingham District Medical Research Endowment Fund. We much appreciate Dr Brian Wharton's advice and Professor Colin Normand's encouragement in preparing this paper.

Requests for reprints should be addressed to: Dr Phyllis Culley, Birmingham Maternity Hospital, Queen Elizabeth Medical Centre, Edgbaston, Birmingham B15 2TH.

References

- Illingworth, R S, *et al*, *Lancet*, 1952, **1**, 683.
- Fomon, S J, *Infant Nutrition*, p 22. Philadelphia, Saunders, 1974.
- Macy, I G, *American Journal of Diseases of Children*, 1949, **78**, 589.
- Lucas, A, *et al*, *British Medical Journal*, 1978, **1**, 1018.
- Wood, B S B, *et al*, *Archives of Disease in Childhood*, 1979, **54**, 111.
- Stewart, A, and Westrop, C, *British Medical Journal*, 1953, **2**, 305.

- ⁷ Fomon, S J, *et al*, *Acta Paediatrica Scandinavica*, 1971, suppl No 223, p 1.
⁸ Fomon, S J, Owen, G M, and Thomas, L N, *American Journal of Diseases of Children*, 1964, **108**, 601.
⁹ Salariya, E M, Easton, P M, and Cater, J I, *Lancet*, 1978, **2**, 1141.
¹⁰ Dahms, B B, *et al*, *Journal of Pediatrics*, 1973, **83**, 1049.
¹¹ Campbell, N, Harvey, D, and Norman, A P, *British Medical Journal*, 1975, **2**, 548.
¹² Eastham, E, *et al*, *British Medical Journal*, 1976, **1**, 305.

- ¹³ Fomon, S J, *et al*, *Acta Paediatrica Scandinavica*, 1970, suppl No 202, p 1.
¹⁴ Bullen, C L, and Willis, A T, *British Medical Journal*, 1971, **3**, 338.
¹⁵ Gunther, M, *et al*, *International Archives of Allergy*, 1962, **21**, 257.
¹⁶ Whorwell, P J, *et al*, *British Medical Journal*, 1979, **1**, 382.
¹⁷ Cruse, P, Yudkin, P, and Baum, J D, *Archives of Disease in Childhood*, 1978, **53**, 76.

(Accepted 3 August 1979)

De novo acute infection and reactivation of hepatitis B virus in established cirrhosis

A THEODOSSI, S P W WILKINSON, B PORTMANN, Y WHITE, A L W F EDDLESTON, ROGER WILLIAMS, A J ZUCKERMAN

British Medical Journal, 1979, **2**, 893-895

Summary and conclusions

Five patients with cirrhosis proved by biopsy had clinical, biochemical, and serological evidence of an acute hepatitis B infection. In two the illness was fulminant and led to death. Only one patient completely recovered. Serological markers for the hepatitis B virus were absent before the onset of the acute illness in four patients, which suggested that a de novo infection had been acquired as a result of recent transfusions of blood or blood products. The fifth patient, who had Goodpasture's syndrome, had antibody to the core of hepatitis B virus, indicating previous exposure to the virus; his acute hepatitis may have been related to immunosuppressive drug treatment, which may have reactivated a dormant virus infection.

Thus an acute type B viral hepatitis due to either a de novo or a reactivated infection may be superimposed on cirrhosis.

Introduction

Jaundice with biochemical characteristics of hepatocellular injury with established cirrhosis may have several causes. In the alcoholic it is often a manifestation of acute alcoholic hepatitis, and in this and other aetiological groups may indicate decompensation precipitated by sepsis, gastrointestinal haemorrhage, or hepatocellular carcinoma. Patients with cirrhosis may also be at special risk of contracting acute hepatitis, since their management often entails admission to hospital and giving blood transfusions and other parenteral preparations. "Non-A,

non-B" hepatitis acquired by administration of clotting-factor concentrates in patients with cirrhosis has been described,¹ but we are unaware of reports of parenterally acquired acute hepatitis type B infection.

In this report we describe five patients with presumed alcoholic cirrhosis in whom jaundice was related to an acute-type hepatitis B. Four patients had de novo infections, possibly acquired from blood or blood products, while serological findings in the other indicated reactivation of a latent infection.

Clinical findings

In each case the liver disease had been fully assessed in this unit before the acute hepatitis developed.

CASES 1-4

Cirrhosis was present in each case and was attributable to heavy alcohol consumption for many years. None of the patients had had encephalopathy or gastrointestinal haemorrhage, but two (cases 1 and 3) had had ascites, which responded to diuretics; one (case 3), because of appreciable piecemeal necrosis on liver biopsy, was receiving prednisone (20 mg/day). Representative liver function tests before the acute hepatitis indicated moderately well-compensated cirrhosis (table). All patients were negative for the following markers of hepatitis B infection as measured by radioimmunoassay: surface antigen (HBsAg); antibody to surface antigen (anti-HBs); and antibody to core antigen (anti-HBc).

Each patient was subsequently admitted with an acute illness characterised by a prodrome of anorexia and malaise followed by deepening jaundice (maximum bilirubin concentration ranging from 147 to 459 μ mol/l (8.6-26.8 mg/100 ml) (table). Aspartate aminotransferase concentration and prothrombin time also increased (237 to 2050 IU/l and 12 to 26 seconds respectively). On admission the serum was positive for HBsAg. During the preceding two, three, four, and eight months each of the four patients had received blood or blood products—namely, fresh frozen plasma as "cover" for a liver biopsy in cases 1, 2, and 3, and blood transfusions for epistaxis in case 4.

Two patients (cases 1 and 2) rapidly lapsed into hepatic coma and died 12 and two days respectively after admission. The remaining two patients showed less severe signs of encephalopathy (confusion and disorientation) and subsequently improved; in each case the serum became HBsAg-negative. One patient (case 3) was discharged from hospital and found to be positive for anti-HBs five months later. The other patient (case 4), although apparently recovering from the acute hepatitis, later developed other complications including a spontaneous bacterial peritonitis and died four months after admission.

Liver Unit, King's College Hospital and Medical School, London SE5 8RX

A THEODOSSI, MB, MRCP, clinical research fellow
 S P W WILKINSON, MD, MRCP, senior medical registrar
 B PORTMANN, MRCPATH, honorary consultant histopathologist
 Y WHITE, DIPMLT(SA), senior technician
 A L W F EDDLESTON, DM, FRCP, senior lecturer and honorary consultant physician
 ROGER WILLIAMS, MD, FRCP, consultant physician and director of unit

London School of Hygiene and Tropical Medicine, London

A J ZUCKERMAN, MD, FRCPATH, professor of microbiology