TABLE
Blood chemistry in 22 artificially-fed infants

	(a) Bottle-fed (n=15) Artificial milk alone*				(b) Bottle-fed (n=7) artificial milk + solid foods*				Difference between (a) and (b)	Combined data (a + b)	
	Range	Mean	SD	SEM	Range	Mean	SD	SEM		Range	Mean (±SD, SEM)
Age (m)	0.5-2.75	1.14	0.7	0.18	1.5-3.0	2.1	0.6	0.23	P<0.001	—	—
Weight (kg)	2.8-4.4	3.5	0.5	0.13	3.5-6.2	4.8	0.3	0.12	P<0.001	—	—
Plasma urea (mg/100 ml)	20-45	25.7	7.8	—	20-45	26.4	9.0	—	NS	20-45	25.9 (±8.0, 1.7)
Plasma protein (g/100 ml)	4.5-6.3	5.3	1.5	0.4	4.9-6.5	5.8	0.6	0.2	NS	4.5-6.5	5.7 (±1.3, 0.27)
Plasma sodium (mEq/l)	130-139	135.4	3.2	0.9	130-141	134.9	3.9	1.5	NS	130-141	135.2 (±3.4, 0.73)
Haematocrit (%)	28-43	37.9	8.3	2.2	29-35	32	2.2	0.9	P<0.001	—	—
Environmental temperature (°C)	20-26	24.5	1.4	0.35	22-27	24.4	1.8	0.7	NS	20-27	25.7 (±1.5, 0.32)

* Different commercially available half-skimmed powdered milks were used at the usual 15% concentration. These milks were either conventional (15) or modified (7). No significant difference was found between the infants fed on unmodified and modified milks.

Venous blood was taken at 11 a.m., after two feeds at 6 a.m. and 9 a.m. Room temperature was noted in each case. Plasma urea was determined by the urease method in a Beckman BUN analyser; plasma sodium, using a flame photometer; total plasma protein by the biuret method; haematocrit by microtube centrifugation. Normal values for our laboratory are: plasma urea ≤ 35 mg/100 ml; plasma sodium 135-140 mEq/l; plasma protein in infants aged less than 3 months 5.0-6.5 g/100 ml; haematocrit 30-45%.

Our data are summarized in the Table. Plasma urea (mean 26 mg/100 ml) lies within normal limits for all but 3 infants (40, 45, and 45 mg/100 ml). The proportion of these high urea levels is small ($3/22=14\%$) and not significant, and is at variance with the findings of Davies and Saunders (75-88% above 40 mg/100 ml), and Dale *et al.* (mean plasma urea ≈ 50 mg/100 ml). Plasma proteins, plasma sodium, and haematocrit in our infants all fall within normal limits, in spite of a fairly high room temperature (mean 26°C) favouring increased evaporative water losses.

Thus the blood urea levels in our artificially-fed infants lie within normal limits. The high levels observed by others may therefore be attributed not to cow's milk in itself, but to some condition associated with artificial feeding, for instance the well-known tendency to prepare a too concentrated milk formulae, as shown by Taitz and Byers (1972). Infants' artificial feeding has its rules, which though simple, are often neglected and have to be constantly recalled. This is a matter of dietary education by physicians, midwives, social workers, and milk manufacturers.

B. MELEKIAN and J. SALET
Hôpital Bretonneau,
2, rue Carpeaux,
75018 Paris, France.

REFERENCES

- Dale, G., Goldfinch, M. E., Sibert, J. R., and Webb, J. K. G. (1975). Plasma osmolality, sodium, and urea in healthy breast-fed and bottle-fed infants in Newcastle upon Tyne. *Archives of Disease in Childhood*, **50**, 731.
Davies, D. P., and Saunders, R. (1973). Blood urea: normal values in early infancy related to feeding practices. *Archives of Disease in Childhood*, **48**, 563.
Taitz, L. S., and Byers, H. D. (1972). High calorie/osmolar feeding and hypertonic dehydration. *Archives of Disease in Childhood*, **47**, 257.

Three periodic diseases as causes of recurrent abdominal pain in childhood

Sir,

In an article in the *Archives* (Christensen and Mortensen, 1975), in three cited articles therein, and in a current textbook of paediatrics (Vaughan, McKay, and Nelson, 1974) psychogenic reactions, irritable colon, and other conditions are considered to be the main causes of recurrent abdominal pain in childhood. No mention is made of three uncommon, repetitive entities characterized by abdominal pain as described in 1948 (Reimann, 1963). These are periodic peritonitis, periodic oedema (hereditary angio-oedema), and periodic pancreatitis (chronic relapsing pancreatitis). All three are heritable, usually begin in infancy, childhood, or adolescence, and have episodes of the respective diseases lasting a day or more that repeat often with remarkable regularity every 1, 2, 3, or 4 weeks, or irregularly, for decades. Between episodes patients are well. Episodes may cease spontaneously for months or years, only to resume at the same or different rate.

Periodic peritonitis, often misnamed familial Mediterranean fever, occurs chiefly in Jews, Armenians, Turks, and Arabs. Episodes of abdominal pain recur often at predictable times, occasionally with pleuritis and arthritis (Bakir and Murtadha, 1975). Colchicine therapy suppresses episodes in most patients and its effect is diagnostic (Reimann, 1975).

Periodic oedema occurs in local areas of the skin and mucous membrane in episodes lasting several hours to days. In some patients abdominal pain occurs synchronously or alone. Unpredictable laryngeal oedema may be fatal unless tracheostomy is carried out. Diagnosis is aided by demonstrating the deficient serum inhibitor of C1 esterase. There is no effective treatment.

Periodic pancreatosis eventually leads to pancreatitis or pancreatic destruction (Williams, Sherman, and Clatworthy, 1967; Sibert, 1975). Diagnosis is aided when there is pain in the back and, in the early stage, by abnormal results of pancreatic functional tests during the episodes only. Surgical treatment is rarely beneficial.

Recognition of these maladies in otherwise healthy children would remove them from the vague group of

transitory abdominal pain usually considered as of psychogenic, epileptic, or other origin.

REFERENCES

- Bakir, F., and Murtadha, M. (1975). Periodic peritonitis. Report of 41 cases without amyloidosis. *Transactions of the Royal Society of Tropical Medicine and Hygiene*, **69**, 111.
- Christensen, M. F., and Mortensen, O. (1975). Long-term prognosis in children with recurrent abdominal pain. *Archives of Disease in Childhood*, **50**, 110.
- Reimann, H. A. (1963). *Periodic Diseases*. Blackwell Scientific Publications, Oxford.
- Reimann, H. A. (1975). Colchicine for periodic peritonitis. *Journal of the American Medical Association*, **231**, 64.
- Sibert, J. R. (1975). Pancreatitis in children. A study in the North of England. *Archives of Disease in Childhood*, **50**, 443.
- Vaughan, V., McKay, R. J., and Nelson, W. E. (1974). (Editors) *Textbook of Pediatrics*, p. 811. Saunders, Philadelphia.
- Williams, T. E., Sherman, N. J., and Clatworthy, H. W. (1967). Chronic fibrosing pancreatitis in childhood: a case of recurrent abdominal pain. *Pediatrics*, **40**, 1019.

H. A. REIMANN
Hahnemann Medical College and Hospital,
230 N. Broad Street,
Philadelphia, Pa., 19102 U.S.A.