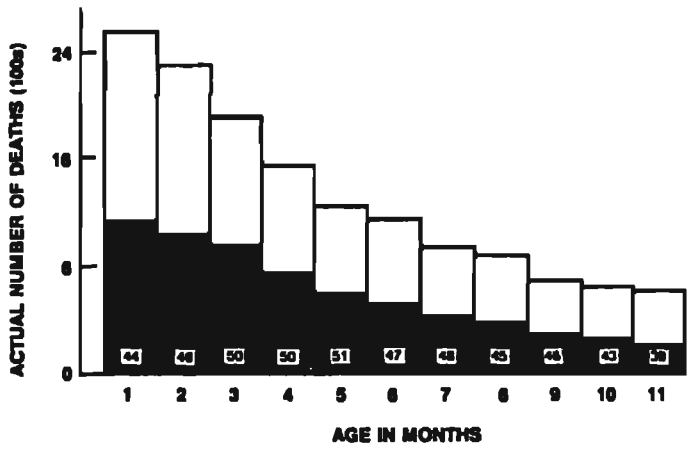
Letters to the Editor

POST-NEONATAL MORTALITY IN THE THIRD WORLD

SIR,—Dr Campbell and colleagues' article on pneumonia in Gambian children (Nov 19, p 1182) appeared a week after an editorial (Nov 12, p 1117) in which you emphasise the importance of dividing infant mortality into neonatal and post-neonatal. A further subdivision might be valuable. In 1979 Cross[1] introduced the concept of "lethal day$_{50}$", which is the day within the first year by which half the infant deaths have occurred. In Britain LD$_{50}$ moved back from 91 days in 1901 to 3 days in 1975. Cross drew conclusions of practical importance about the cause of this change.

It has long been the dogma in nutritional circles that the danger period for infants in the Third World is the second 6 months of life. The peak incidence of diarrhoeal disease is at around 9–12 months, continuing into the second year, presumably because of the child's increased exposure to contaminated food and a contaminated environment. The relative mortality, compared with that in developed countries, is far higher in the second than in the first year of life. As a consequence, enormous effort has been devoted to programmes targeted to the preschool child. I must bear my share of responsibility for this, having been one of the first to propose second-year mortality as an index of malnutrition.[2]

However, if one looks at absolute numbers, the picture is completely different. Data from the Pan American Health Organisation study[3] of infant and child mortality in Latin America and the Caribbean (figure) show that about two-thirds of post-neonatal deaths occur between 1 and 6 months.[3] About half these deaths were attributed, by retrospective diagnosis, to diarrhoeal disease, despite the fact that most children were being breast-fed, at least for the first 3 months or so. A similar pattern has been found in other Third World countries where statistics are available.



Post-neonatal deaths by month.

Source: PAHO survey of child mortality in Latin America and the Caribbean.[3] Black areas = number (with %) of deaths attributed to diarrhoeal disease.

Only in the past few years has serious attention been given to respiratory infections as a major cause of early post-neonatal mortality. These infections seem not to be well understood. The peak incidence of measles apparently occurs later.

If more attention had been given to Cross' concept, the time-lag in attacking this problem might have been reduced and our ideas about infant and child mortality in the Third World might have been better balanced.

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NUTRITIONAL IMPROVEMENT WITHOUT BETTER HEALTH: SANTA MARIA CAUQUÉ, 15 YEARS LATER

SIR,—In 1978, Dr Leonardo Mata published a longitudinal study done by the Institute of Nutrition of Central America and Panama (INCAP) between 1964 and 1972 in Santa Maria Cauqué, a rural community in Guatemala.[1] This study supported an interaction between malnutrition and infectious diseases. In Santa Maria Cauqué over those eight years malnutrition, infectious diseases, and infant and child mortality were highly prevalent.

From Sept 1, 1986, to Aug 31, 1987, Santa Maria Cauqué was revisited. Length and weight were collected in over 95% of all live births. A cross-sectional study on breast milk intake in all exclusively breastfed male infants less than 2 months of age, similar to one in 1977,[2] was carried out. Information on mortality and causes of death was obtained. All the data were collected by trained personnel with the same standardised procedures used in the original longitudinal study.

In 1986–87 infants were heavier and longer from birth to 6 months of age than infants measured during 1964–72 (table). In 1964–72 42% of babies weighed 2500 g or less compared with 31% in 1986–87. Daily breast milk intake was higher in 1986–87 than in 1977, and the mothers were heavier and taller than lactating mothers studied earlier.

This improvement in nutritional status in Santa Maria Cauqué over the past 15 years contrasts with the stagnation in morbidity and mortality rates. Infectious diseases continue to be a major cause of demand of health services. Infant mortality was 98 per 1000 live births in 1986–87 and 96 per 1000 between 1964 and 1972. The main causes of death in 1964–72 were acute respiratory infections, whooping cough, measles, acute diarrhoeal disease, congenital malformation, and prematurity. The 11 infant deaths in 1986–87 were due to dehydration secondary to diarrhoeal disease (4), acute respiratory infection (4), and sepsis, prematurity, and congenital malformation (1 each).

Why did nutritional status improve in this community without parallel improvements in the health indicators? Since 1972 Santa Maria Cauqué has seen the introduction of tap water and electricity and increased asset holdings. However, the major change took place after the 1976 earthquake when most families began to participate in an agricultural (cash crops) cooperative. Total family income increased, accompanied by opportunities for employment for agricultural workers.[3] This change might explain the nutritional improvements.

INFANT AND MATERNAL CHARACTERISTICS SANTA MARIA CAUQUÉ, SACATEPEQUEZ

—	1964–72		1986–87	
	No	Mean (SD)	No	Mean (SD)
Infant weight (kg)				
Birth	430	2·6 (0·4)	107	2·7 (0·3)
3 mo	375	5·0 (0·8)	118	5·2 (0·7)
6 mo	374	6·3 (0·8)	104	6·5 (0·8)
Infant length (cm)				
Birth	430	45·7 (2·2)	107	47·1 (1·5)
3 mo	186	55·3 (2·7)	118	56·0 (2·0)
6 mo	184	60·5 (2·7)	104	62·1 (2·3)
24 h breast milk intake (ml)	20	615 (123)	20	772 (106)
Maternal weight (kg)	20	48 (5)	20	52 (8)
Maternal height (cm)	20	144 (5)	20	145 (8)
Maternal arm circumference (cm)	20	25 (1)	20	27 (2)

Whooping cough and measles had disappeared as causes of mortality in 1986–87, probably reflecting the impact of vaccination campaigns and health technology, but acute respiratory infections and diarrhoeal diseases persist. It seems that economic improvements, while positively affecting nutritional status in the short run, may not necessarily modify health. Health and educational activities should be integrated to community

development programmes—if they are not, then improvements in nutrition will not necessarily be accompanied by better health, and vice versa.

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ACUTE TREATMENT OF SEVERE HAEMORRHAGIC GASTRITIS WITH HIGH-DOSE SUCRALFATE

SIR,—We report management of severe haemorrhagic gastritis with sucralfate in 10 patients.

4 patients were admitted to our surgical intensive care unit (ICU) with acute upper gastrointestinal bleeding. 4 other patients had this complication during their stay in the ICU while on stress ulcer prophylaxis with H_2-receptor antagonists. The other 2 ICU patients were on prophylaxis with sucralfate when acute gastrointestinal bleeding occurred. 6 of the patients were on ventilatory support. In all patients endoscopy revealed a severe diffuse haemorrhagic gastritis but no bleeding gastric or duodenal ulcer.

The patients received a high dose of H_2-receptor antagonist (600 mg ranitidine or 2000 mg cimetidine, daily) and pirenzepine (50 mg, daily) to suppress almost all acid secretion. However, bleeding continued or worsened over the next 2–3 days. All patients needed between two and six units of packed red cells during the 24 h before we started sucralfate. 3 of the patients were scheduled for emergency gastrectomy because of the duration and severity of the bleeding.

Blood clots were cleared from the stomach by suctioning through a gastroscope or by saline lavage through a stomach tube. Because of the severity of the bleeding in 2 patients a second large-bore tube was placed near the antral region while the smaller stomach tube was placed in the cardia. Several litres of saline were flushed through the proximal tube and suctioned through the distal tube until no further blood clots could be removed. Then the large-bore tube was removed. 60 ml of sucralfate suspension (12 g) was flushed through the stomach tube which was then clamped. 2 h later the tube was opened and we aspirated as much gastric content as possible. In 6 patients the aspirate was almost clear while the other 4 patients still showed some bleeding. A second dose of 60 ml sucralfate was instilled and the stomach tube again clamped for 2 h. 4 h after the start of the treatment the bleeding had stopped in all patients. During the next 20 h the instillation of 60 ml sucralfate was continued every 2 h. Bleeding did not recur in any patient. On the second day the sucralfate dose was reduced to 30 ml 2 hourly and on the third day to 20 ml. From days 4 to 7, 20 ml sucralfate was administered every 4 h. During the whole period the parenteral administration of H_2-receptor antagonist and pirenzepine was continued.

In 5 patients gastroscopy between days 3 and 7 revealed no pathological findings in the gastric mucosa. The other 5 patients showed only a mild superficial gastritis, usually in the antral region without any sign of active bleeding. No patient needed blood transfusion after the start of treatment.

Sucralfate is a weak antacid, increasing gastric mucosal blood flow, promoting mucosal regeneration, binding bile acids, and inhibiting pepsin. The drug also forms a mucus-like layer on the mucosal surface. The administration of high-dose sucralfate suspension ensures contact of the suspension with virtually the whole gastric mucosa, which may be necessary for successful treatment of haemorrhagic gastritis.

These results are preliminary, but at least 3 of the 10 patients were spared gastrectomy. We do not know whether treatment with sucralfate could be extended to bleeding peptic ulcers.

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FATAL HYDRALAZINE-INDUCED SYSTEMIC LUPUS ERYTHEMATOSUS

SIR,—The systemic lupus erythematosus (SLE) syndrome due to hydralazine is not confined to slow acetylators on high doses.[1-4] The potential hazards of this drug have been understated. We describe here a fatal case of SLE in which circumstantial evidence implicates hydralazine.

A 61-year-old woman presented elsewhere with headaches and was found to have a blood pressure of 240/140 mm Hg. She was treated with labetalol and hydralazine (50 mg twice daily), with good blood pressure control. 3 years later she was admitted as an emergency with an 8 week history of anorexia and tiredness. She had vitiligo, renal failure (serum creatinine 242 μmol/l), normochromic, normocytic anaemia (haemoglobin 5·4 g/dl), purpura, and retinal haemorrhage. There was no evidence of joint involvement. Despite active treatment she died.

Necropsy revealed splenomegaly, pleuropulmonary involvement, and glomerulonephritis. Histological features suggested pulmonary interstitial fibrosis, vasculitis of the skin, focal glomerulonephritis with crescent formation and granular IgG, and complement and fibrin deposition on immunofluorescence. The clinical and pathological diagnosis was SLE. Serum, taken before death, was antinuclear antibody positive (titre 600) by immunofluorescence with rat liver substrate; and negative for antibodies to ribose nucleoprotein and SM antigen. Native DNA binding, by radioimmunoassay was 9% (normal below 10%). Anti-Ro antibodies were positive and anti-histone antibodies were also positive by immunofluorescence.[5] Complement levels were reduced with C3 0·5 g/l (normal 0·75–1·75) and C4 0·08 g/l (0·14–0·54).

Although the clinical features are not typical of drug-induced SLE the lack of significant increase in double-stranded DNA antibodies and positive histone antibodies favour this possibility. This late presentation in a patient with no joint involvement raises the possibility that the SLE was hydralazine induced. Drug-induced lupus usually manifests as mild arthralgia, fever, and weight loss, occasionally with serositis.[6] Doses of 200 mg daily or less were thought to be free from this side-effect, especially in fast acetylators,[7] but recent reports have suggested that the syndrome is seen at lower doses and not just in slow acetylators. Others have drawn attention to glomerulonephritis in patients on hydralazine, which was fatal in two cases.[2,8]

Hydralazine was a useful antihypertensive drug but the advent of the calcium channel blockers has led to a decline in its popularity. It is not possible to prove that hydralazine caused this patient's death. However, we suggest that this drug carries an unacceptable risk and should be regarded as obsolete.

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