autoantibody profile, platelet count, clotting screen, tests for Australia antigen, antistreptolysin O titre, *Cryptococcus neoformans* titre, serum fibrin degradation products, haptoglobins, and creatine phosphokinase. Widal tests confirmed a good immune response to the vaccine with titres of 1/1280 for *Salmonella typhi* H and O antigens and 1/640 for *Salmonella paratyphi* A and B H antigens.

He was given an infusion of isotonic saline and 250 mg of intravenous frusemide. Urine flow started two hours later. The urine was normal in colour and microscopy showed granular casts in only one of six samples examined. No white cells or red cells were found in any of the specimens, though a trace of protein was found in two. The patient's condition steadily improved and he became asymptomatic over the next few days. Within two weeks his renal and hepatic function tests gave normal results.

Comment

A careful search was made to find a cause for acute renal failure other than the vaccine. But all relevant tests gave negative results, and the possibility of contamination of the vaccine was excluded by the uncomplicated vaccination of two other subjects from the same phial of vaccine.

An immune complex glomerulonephritis in a patient previously exposed to the antigen could have occurred, but the rapid onset of the illness and the laboratory results make this unlikely. Dehydration was not a prominent feature of the illness and seems an improbable mechanism for the renal failure. Neither would dehydration explain the hepatitis. Myoglobinuria as a cause of renal failure is characterised by dark urine and raised serum creatine phosphokinase concentrations, both of which were absent in this case.² The hepatorenal syndrome is unlikely in that liver function was not severely impaired and the condition usually has a poor prognosis.³

In many cases of acute renal failure the actual mechanism remains obscure,⁴ but this patient's acute intrinsic renal failure seems most logically explained by an episode of acute renovascular shutdown of indeterminate cause but related to a reaction to the vaccine.

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Bottle feeding, early gastroenteritis, and inflammatory bowel disease

Breast milk contains several factors,¹ including immunoglobulins, that may protect particularly against gastrointestinal infection. In addition there has been interest in the role of IgA in antigen exclusion² early in life. Some patients with ulcerative colitis are sensitive to milk,³ and Acheson and Truelove⁴ reported a significant deficiency of breast-feeding in patients with ulcerative colitis compared with controls. In the light of reports of infectious agents, possibly acquired early in life, being associated with Crohn's disease⁵ we have examined the pattern of breast-feeding in these patients as well as in patients with ulcerative colitis and in controls.

Patients, methods, and results

We questioned 57 patients with Crohn's disease and 51 with ulcerative colitis. Patients with proctitis and those whose mothers were dead or unavailable were excluded. For each patient two age- and sex-matched healthy controls were selected. A questionnaire giving details of place of birth (town, country, home, hospital), occupation of father at time of birth, breast-feeding or bottle feeding, length of time of breast-feeding, order in family, history of gastroenteritis in first six months of life, and a history of

atopy was completed. Breast-feeding was considered to have ceased when any other food was started.

The results (table) showed that 29.9% of patients with ulcerative colitis compared with 11.8% of controls had never been breast-fed ($P = 0.005$). The difference in the two groups was lost in patients who were breast-fed for two weeks or more. No such differences appeared in the Crohn's disease group. Nevertheless, there was a significant difference in a history of early gastroenteritis in patients with Crohn's disease—10.5% as compared with controls 0.9% ($P = 0.005$). No significant relationship was found with place of birth, social class, or order in the family. The incidence of atopy did not differ in the bottle-fed and breast-fed patients.

Results of inquiry into history of breast-feeding or bottle feeding in patients with ulcerative colitis or Crohn's disease

	No of patients ("n")	No of hospital controls ("n")	No of normal controls ("n")	Total controls ("n")	Significance*
Ulcerative colitis ..	51	51	51	102	
Mean age ..	36.7	36.5	36.7	36.6	
Male ..	17 (33.3)	17 (33.3)	17 (33.3)	34 (33.3)	
Female ..	34 (66.6)	34 (66.6)	34 (66.6)	68 (66.6)	
Bottle fed ..	15 (29.4)	6 (11.8)	6 (11.8)	12 (11.8)	$P = 0.005$
Gastroenteritis ..	4 (7.8)	0 (0)	1 (2.0)	1 (1)	NS
Crohn's disease ..	57	57	57	114	
Mean age ..	33.3	32.9	32.8	32.9	
Male ..	22 (38.6)	22 (38.6)	22 (38.6)	44 (38.6)	
Female ..	35 (61.4)	35 (61.4)	35 (61.4)	70 (61.4)	
Bottle fed ..	11 (19.3)	11 (19.3)	11 (19.3)	22 (19.3)	NS
Gastroenteritis ..	6 (10.5)	1 (1.7)	0 (0)	1 (0.9)	$P = 0.005$

*Fisher's exact test, patients vs hospital + normal controls.

Comment

The results for ulcerative colitis are in agreement with those of Acheson and Truelove.⁴ No such effect was seen in Crohn's disease. The observation that only a short period of breast-feeding was necessary to eliminate the effect in ulcerative colitis suggests that whatever factor is responsible was operative only shortly after birth. Thus either bottle feeding is harmful or breast-feeding protective. The harmful effect of bottle feeding might result from sensitisation to cows' milk early in infancy, possibly as a result of increased permeability to macromolecules. Bottle feeding might also alter bacterial flora at a time when sensitisation to bacterial antigens may occur. This may be relevant to the possibility that ulcerative colitis results from an abnormal immune response to enterobacteriaceae.

Patients with Crohn's disease had a significantly increased incidence of gastroenteritis within the first six months independent of bottle feeding. A possible explanation for this finding in Crohn's disease is that a pathogenic infection occurs which persists, becoming manifest as Crohn's disease only later in life. Subacute sclerosing panencephalitis and chronic hepatitis B are two examples of an abnormal response to a viral infection with a prolonged latent period. Rotavirus infection is a well-recognised cause of infantile gastroenteritis, and there is some evidence for its possible association with Crohn's disease,⁵ but a persistent carrier state for this virus has not been shown.

Our findings suggest that an event associated with bottle feeding occurs within the first two weeks of life which predisposes to the development of ulcerative colitis later in life in some individuals. Similarly, an event related to gastroenteritis but independent of breast-feeding or bottle feeding may predispose to the later development of Crohn's disease.

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