milk easily gets into the mouth of the baby and the baby starts sucking. (*Fig. 2*). If this procedure is continued at every feed for 3-4 days, the babies will very easily learn to suck at the breast properly. As the baby sucks, there will be more secretion of milk. For very small babies this is one way of training them and they learn very quickly.

We have tried this method in the past 2 years in 36 babies. Ten babies would not suck for an unknown reason, 7 babies were late starters and 2 babies had cleft lip and cleft palate. Seventeen babies weighed 1 to 1.2 kg, who were small for gestational age.

The other known method to initiate sucking is by using a nursing supplementer (lact aid) which is expensive, not easily sterilizable and not easily available. We found the method complicated and not hygienic due to involvement of a bottle.

This drop and drip method is simple safe and successful and can be taught to the mother, her relatives and peripheral workers. The method has been tried by my colleagues in Davangere successfully in their own patients.

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Acute Glomerulonephritis in *Salmonella typhi* Infection

With reference to this report(1), in the absence of renal histological findings, the diagnosis of acute glomerulonephritis in typhoid fever remains unconfirmed. Moreover, the clinical course of the acute renal failure in the patient was *not* characteristic of acute glomerulonephritis. The child did not have hypertension and had only slight proteinuria (200 mg/24 hour, in a 10-year-old boy). The patient was almost anuric for 8 days, with a blood urea nitrogen (blood urea, as shown in figure) of 260 mg/dl and a serum creatinine of 4.0 mg/dl (the figure shows 8 mg/dl), indicating very severe renal involvement. He "entered a diuretic phase" and the blood urea and serum creatinine levels came down to normal within about 10 days. Such a rapid recovery is typical of acute tubular necrosis, especially if associated with a pre-renal component.

Patients with acute glomerulonephritis who develop acute renal failure have very severe renal histologic lesions, often with glomerular crescent formation, and heavy proteinuria. The recovery is very slow in such cases. Those with extensive glomerular crescentic changes may progress to develop end stage renal disease or be left with significant renal damage.

The patient reported had red cell casts in his urine, which implies the presence of glomerulitis. However, all other features can be better explained by acute tubular necrosis. In any case, no nephrologist will accept the diagnosis of a rare complication without a complete renal histological examination.

The authors have clearly missed the opportunity to make a significant contribution.

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REFERENCE

1. Dhawan A, Marwaha RK. Acute glomerulonephritis in multidrug resistant *Salmonella typhi* infection. Indian Pediatr 1992, 29: 1039-1041.

Reply

We thank Dr. Srivastava for his interest in the case report of acute glomerulonephritis in typhoid fever(1). Reasons beyond our control precluded a renal biopsy. The absence of confirmatory histological findings naturally generates controversy regarding the renal pathology. We would like to reiterate that the presence of red cell casts in the urine and the low serum C3 give credence to a glomerular lesion. Sitprija *et al.*(2) were able to demonstrate immune complex glomerulitis in three unselected cases of typhoid fever with no clinical or laboratory evidence of renal dysfunction and postulated a direct role of *S. typhi* in the pathogenesis of the glomerular lesion. If this is true, then recovery in renal function could parallel the amelioration of other symptoms and signs as was observed in the case reported. We accept criticism of the obvious omission in our case but it may prompt a systematic evaluation of the pathogenetic mechanism(s) of renal dysfunction in typhoid fever and of the course and pattern of recovery in cases with renal failure.

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REFERENCES

1. Dhawan A, Marwaha RK. Acute glomerulonephritis multidrug resistant *Salmonella typhi* infection. Indian Pediatr 1992, 29: 1039-1041.
2. Sitprija V, Pipatanagul V, Boonpucknavig V, Boonpucknavig S. Glomerulitis in typhoid fever. Ann Int Med 1974, 81: 210-213.

Trichuris Dysentery

Infection with whipworm, *Trichuris trichiura* is one of the commonest helminthiases of childhood. In tropical countries where other diseases obscure the source of symptoms, this parasite is usually more or less ignored, with the assumption that it is non-pathogenic(1). That such is not always true is being demonstrated by the following case report.

A five-year-old male child was admitted with history of prolonged diarrhea with blood streaked stool, abdominal pain, tenesmus for the last 3 years. The child had suffered from generalized swelling of the body two years back for which he had received three bottles of blood transfusion. He was also suffering from rectal prolapse. Apart from this, the child was treated outside also with metronidazole, cotrimoxazole, furazolidine, nalidixic acid and Inj. emetine without any appreciable result.

Physical examination revealed a moderately anemic child with weight of 14 kg, height 100 cm and mid-arm circumference 13 cm. No edema or clubbing was observed. Abdominal examination showed