infraumbilical transverse incision was given. An ileal loop with peritoneal sac was entering one deep inguinal ring from inside. It was pulled out from the inguinal canal from within by gentle blunt dissection. There was a perforation of antimesenteric border of this ileal loop approximately 1.5 cm in size. The edges of perforation were freshened and it was closed transversely in two layers. Rest of the bowel and solid viscera were normal.

The abdomen was closed in layers. A penrose drain was put in the scrotal wound. Unfortunately, the child died on the 2nd post-operative day due to septicemia.

Although incarceration and strangulation are the common complications of inguinal hernia, spontaneous fecal fistula formation in these cases is very rare. A variety of causes for the development of fistulas of the gastrointestinal tract have been described, but the great majority of external fistulas follow operative procedures performed in the GIT or other intra-abdominal organs. In one of the largest series of 81 fistulas of GIT, 98.5% were postoperative. Even among the 14 occurring spontaneously in this series, all but one were internal and none followed inguinal hernia(2).

Inguinal hernia in children is almost invariably indirect and is a congenital abnormality due to failure of the obliteration of processes vaginalis. Incarceration and strangulation are common in infants than in older children(3). The pathogenesis of spontaneous fecal fistula formation is obscure but may be due to a part of ischemic intestine getting adherent to the scrotum which later gives way resulting in fecal fistula formation(1). Although various unusual complications of inguinal hernia like testicular infarction, rupture of stomach(4), appendicular abscess(3) are on record, spontaneous fecal fistula formation is extremely rare. Review of the World literature reveals only one previous case of spontaneous fecal fistula in a 3½-month-old boy(1).

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Antenatal Supplementation Effect on Iron Status of Infants

In their recent editorial, Drs Lokeshwar and Manglani have mentioned the effect of maternal iron deficiency on newborn iron stores(1). They have cited studies from Western centres suggesting that cord ferritin levels are lower in infants born to an iron deficient mother.

Two studies from our own country have however shown conflicting results. Agarwal *et al.*(2) in their study of 30 anemic mother-infant pairs showed that there is a direct correlation between maternal hemoglobin and cord ferritin, cord serum iron and transferrin saturation. In contrast, a larger study comprising of 95 anemic mother-infant pairs from another centre(3) showed that there was no difference in the cord ferritin in the anemic and non-anemic groups. Both these studies were cross-sectional and interestingly the cord ferritin levels in both were far lower than those quoted in the Western studies.

Both these studies differed from Western studies in that despite maternal anemia manifested by low hemoglobin level, the levels of maternal ferritin were around 25 ng/ml. Kelly *et al.*(4) have suggested that maternal ferritin <10 ng/ml was associated with a lower fetal ferritin level. Perhaps this may explain the divergent results expressed by the Indian studies.

We also wonder whether maternal-fetal iron dynamics are different in our population. A possible conjecture is whether an associated deficiency of a vitamin, mineral or micronutrient may influence this complex relationship. But above all a longitudinal study enrolling women in the antenatal period with follow up of the offspring into infancy may throw some light on the problem.

One of the causes of iron deficiency anemia during pregnancy is hookworm infestation which is rampant in many parts of our country(5). This may cause anemia in the infant in two ways. The first as suggested in the editorial by depleting the maternal iron stores. The second is possibly by transmission of the infestation to the nursing infant. A recent article by Hotez(6) suggests that hookworms especially *Ancylostoma duodenale* are transmitted through the breast milk. The article goes on to suggest that this may be responsible for the hookworm disease seen in infants.

Managing maternal anemia during pregnancy due to hookworm infestation is, however not easy. In view of the possible teratogenic effect of mebendazole on the fetus-routine treatment of hookworm infestation during pregnancy is not recommended. Specific therapy is only indicated when there is inadequate response to vigorous nutritional therapy and supportive care including blood transfusions. The role of specific therapy in the form of pyrantel pamoate would only arise where severe anemia exists with lack of transfusion therapy; as would be the case in rural areas in our country, where the infestation is the heaviest. Mebendazole or pyrantel pamoate with vigorous oral iron therapy is recommended in such situations(7).

Effective management for antenatal anemia in the mother, and adequate treatment of hookworm infestation, if present, would thus obviate anemia in the infant. The problem of lactogenic transmission of hookworm has, however not been clearly defined in our country, and we would be interested to know if anyone has looked at this problem.

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Progeria

With reference to the recent publication on Progeria(1), the authors state that their's is the third reported case from India. The reference cited by them(2), however, relates to only one earlier case. Secondly, and indeed surprisingly, at least two of the cases of progeria recorded in our noted pediatric Indian journals(3,4) have not been taken into account by the authors.

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Essential Drugs

Fungal infections are a common cause of nosocomial infections. With the improved life support measures, antibiotics, steroids and antimetabolites, the incidence of mycotic infections appears to be increasing. Antifungal sensitivity testing is not yet standardised and has a poor correlation with clinical efficacy. The choice of antifungal agents is therefore, made on the basis of clinical experience(1). Hence despite the recent availability of newer agents, Amphotercin B continues to be the sheet anchor of systemic antifungal therapy.

In our country, Amphotercin B is available only under the brand name of Fungisone(2). Of late, this has not been openly available in the market, causing serious therapeutic and management problems. Enquiries with the manufacturing concern (Sarabhai Chemicals) reveal that the shortfall has arisen as a result of certain problems with the import of the