Earlier studies conducted in Mumbai and Punjab reported that 40.2% and 20.5% of children 5-14 years were underweight(2,3). Stunting was observed in 45.1% of the children. A comparatively lower proportions of children were wasted (13.1%) for their age indicating that acute (severe, but short term) undernutrition is less common in children. However, earlier studies conducted in Kerala on children (5-12 years) in 2000 and 2001 have reported the prevalence of wasting as 53.3% and 57.6% respectivel(4,5). The results of the present study revealed that 52.5% of children were suffering from one or the other form of under nutrition, possibly due to inadequate diet and chronic infections. Strengthening of the school health services and creating awareness among parents about the nutritional requirements of their children is required.

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Practical Approach to Neonatal Analgesia

We read with interest the article, "Practical approach to neonatal analgesia"(1). The authors have dealt with many aspects of neonatal analgesia. Surprisingly, there is no section on the assessment of pain in neonates.

Many validated pain measures are currently available to assess pain in both term and preterm infants. Behavioral and physiological alterations of neonatal pain are incorporated in these pain measures. Some of the well-validated pain scores include the Neonatal Facial Coding System, the Objective Pain Scale, CRIES (crying, requirement for oxygen, increase in heart rate, blood pressure, facial expression and sleeplessness), Premature Infant Pain Profile (PIPP) and the Neonatal Infant Pain Scale(2).

The approach to neonatal analgesia could have been better summarized. The authors have underplayed the role of fentanyl, alfentanyl and sufentanyl in neonatal analgesia by quoting various side effects. However, chest wall and glottic rigidity are most often seen with bolus doses; and fentanyl and its congeners can be safely used as small frequent doses (0.5 to 10 μg/kg) or as infusions (1-5 μg/kg/h)(2). EMLA is not an effective analgesic for heel lancing and the side effect of methemoglobinemia with repeated usage in preterms has not yet been adequately studied(3).

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Glucose-6-Phosphate Dehydrogenase Deficiency with Bilateral Cataract

Various studies from India have shown association between glucose-6-phosphate dehydrogenase (G-6-PD) deficiency in erythrocytes and age-related cataract(1,2). However, its association with childhood or congenital cataract has not been well studied. An 8-year-old boy presented to us with history of bilateral cataract developing at age of 2 years, which was operated at 3.5 years; and pallor requiring 3 blood transfusions at ages 3.5, 4.5 and 8 years. He had received multiple antibiotics including co-trimoxazole for various pharyngeal infections. His mental development was normal, and there was not history of seizures. Family history was negative for early-onset cataract or hemato-logical disorders. On physical examination, patient had bilateral aphakia; mild icterus and pallor. There was no dysmorphism, signifi-cant lymphadenopathy or hepatospleno-megaly.

Laboratory investigations revealed hemoglobin 8.4 g/dL, WBC count 6000/mm^3, platelets 386,000/mm^3, MCV 101.6 fl, MCH 30.4 pg, MCHC 30 g/dL, red blood cell (RBC) count 2.76 million/mm^3 and absolute reticulocyte count 11%. Peripheral smear showed macrocytic erythrocytes with polychromasia. Total bilirubin was 3 mg/dL; direct bilirubin 0.7 mg/dL; serum creatinine 0.6 mg/dL; liver enzymes normal; hemoglobin electrophoresis normal; direct Coombs' test negative; and bone marrow examination revealed erythroid hyperplasia. Methemo-globin reduction test showed defi-ciency of G6PD enzyme in erythrocytes. On quantitative analysis, G6PD was 93.7 mU/10^9 erythrocytes (normal: 118-144 mU/10^9 erythrocytes). Screening for galactosemia in view of early-onset bilateral cataracts was negative with absence of reducing substances in urine.

The patient had congenital nonspherocytic hemolytic anemia due to G-6-PD deficiency with episodic drops in hemoglobin most probably due to cotrimoxazole exposure. G6PD deficiency results in reduced glutathione activity in the lens, making the lens more prone to oxidative damage and resultant aggregation of lens proteins. The development of bilateral cataract at the age of 2 years in a develop-mentally normal child may have been due to the