and separate pulmonary veins entering into separate corresponding atria.

Because of levoisomeric liver, hepatomegaly was out of proportion and abdominal protruberance persisted significantly even after removal of the giant spleen. A meticulous search must be made to remove all spenules in such cases of hemolytic anemia with polysplenia syndrome. Abdominal wall closure should be done accurately to avoid problems of wound dehisence or incisional hernia because of enlarged levoisomeric liver.

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Celphos Poisoning

Recently aluminium phosphide (Celphos) taken by persons accidentally or with suicidal intent has resulted in large number of deaths. We are reporting one such case.

A six-year-old boy was admitted with complaint of ingesting aluminium phosphide (Celphos) tablet, pain in epigastrium 6 hours prior to admission, vomiting and altered sensorium two hours after the ingestion. There was no history of bleeding from any site, chest pain, breathlessness and diarrhea. The patient was brought to the hospital by his father who revealed that the mother along with 4 children (aged between 3 to 10 years) had taken Celphos tablets after an altercation with him. The exact number of tablets consumed by each child and mother was not known. While three children died after half an hour of ingestion, the mother succummbed eight hour later.

On examination, the pulse was 128/min regular, respiratory rate 46/min and blood pressure 80/60 mm Hg. He was drowsy, restless and pupils were semidilated but reacting to light. Cardiovascular examination showed no abnormality except tachycardia. Rest of the systems were normal.

Treatment included gastric lavage with 1 : 5000 solution of potassium permanganate, intravenous fluids, magnesium sulphate as antidote and oxygen inhalation. The condition of the patient deteriorated further in the form of fast and feeble radial pulse, blood pressure 56 mm Hg systolic and irregular respiration. At this time, the patient was put on intravenous dopamine infusion and hydrocortisone. This therapy also failed and the patient expired after 3 hours.

Celphos is used as a grain preservative and on exposure to moisture, liberates phosphine gas causing severe gastrointestinal irritation, cardiovascular collapse and death(1). Recent reports suggest a steady increase in cases of aluminium phosphide poisoning in our country. Chugh *et al.*(2) described 228 cases of Celphos poisoning in a 6 years period. Majority of cases belonged to younger generation and the intention was suicidal in 60% of cases with

77.2% mortality. Puranin *et al.*(3) found 39 cases of Celphos poisoning in one year period, majority of them young. Except one, all cases were suicidal with 72.73% mortality. The presenting clinical features were profuse vomiting, epigastric pain, restlessness, drowsiness and shock. Our case too had similar clinical features.

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Fanconi's Anemia

Fanconi's anemia is a rare familial disorder inherited as autosomal recessive condition with variable penetrance and characterised by pancytopenia, marrow hypoplasia or aplasia, multiple skeletal and visceral anomalies and chromosomal aberrations.

A female child was born of primigravida at full term in May, 1982. There was no history of consanguinity or drug intakes, exposure to radiation and exanthematous fever during antenatal period. At birth left thumb was absent and right thumb was rudimentary. Since the age of 4 years she presented with gradually increasing pallor and weakness and poor gain in weight and height. She also had hematuria and hematemesis after which there was history of easy bruisability off and on. Both parents and one younger sib were normal. Examination revealed short stature, small face, microcephaly, microphthalmia, marked pallor, absence of both thumbs, and hepatosplenomegaly. There was pancytopenia and marrow hypoplasia. *X*-ray showed absence of phalanges and metacarpals of both thumbs. Abdominal ultrasound was normal. The diagnosis of Fanconi's anemia was made on the basis of above phenotypic features and investigation. Since then patient is receiving androgen (Oxymetholone) and regular blood transfusion.

Fanconi(1) in 1927, in his original article described a familial type of aplastic anemia. Since then Fanconi's anemia has been widely reported. In these cases spontaneous or induced chromosomal anomalies have been demonstrated which may aid in prenatal(2) and postnatal(3) diagnosis of Fanconi's anemia. In our case chromosomal study was not performed because of non-availability of such facility. The patients are at increased risk of developing leukemia and solid tumors(4).

These patients demand the attention of pediatricians to search and provide some definite modality of therapy, *i.e.*, true bone marrow stimulants, bone marrow trans-