

Staphylococcal Bacteremia

The report from Chandigarh by Singhi *et al.* (1), noting *Staphylococcus aureus* to be the commonest organism causing bacteremia in highly febrile children with no apparent focus of infection, obviously deserves consideration in planning treatment for such cases. It may be noted that in several Indian studies and in our own report from North Kerala (2), the second common organism for pyogenic meningitis was also *Staphylococcus*. This is in sharp contrast to western literature, where the *Staphylococcus* is a rare cause for both these disorders. The syndromes of bacteremia and pyogenic meningitis are interrelated. In most cases bacteremia is a forerunner of meningitis. Bacteremia is also a distinguishing characteristic of staphylococcal infections (3). Staphylococcal bacteremia is recognized rarely in the absence of a focus of infection; but the focus may be inapparent or exceedingly minor, as a small furuncle (4). The high incidence of skin infections in our tropical climate was suggested as the possible cause

for the common occurrence of staphylococcal meningitis in India (2). In places where staphylococcal meningitis is common, staphylococcal bacteremia will also be common. For these reasons, staphylococcal bacteremia is likely to be common in highly febrile children in other parts of India also.

J. Vincent,

*Department of Pediatrics,
Medical College Hospital,
Round East, Trichur,
Kerala 680 001.*

REFERENCES

1. Singhi S, Kohli V, Ayyagiri A. Bacteremia and bacterial infections in highly febrile children with no apparent focus. *Indian Pediatr* 1992, 29: 1285-1289.
2. Vincent J, Sainaba MK, Rajagopalan KC. Bacterial etiology of meningitis, with special reference to staphylococci. *Indian Pediatr* 1987, 24: 145-151.
3. Sheagren JN. *Staphylococcus aureus*: The persistent pathogen. *N Eng J Med* 1984, 310: 1368-1373.
4. Melish ME. Staphylococcal infections. In: *Textbook of Pediatric Infectious Diseases*. Eds Feigin RD, Cherry JD. Philadelphia, WB Saunders Company, 1981, pp 956-985.

Baby in Arms: A Good Position for Peripheral Venous Cannulation

Setting up of an intravenous line is a common procedure. In patients with dehydration or shock due to low central venous pressure, the veins are collapsed especially when the patient is lying down. The venous

pressure further goes down if during the procedure, the limb gets lifted up above the heart level. On such occasions it can take a lot of attempts and time to set up the intravenous line which may cause immense anxiety to the parents, doctors, and the assisting staff. The continuous crying of the child and at times of the mother makes the matters worse. The parents suffer more if they are

driven out of the procedure room, and have to helplessly listen to the lamenting child.

We no more drive parents out. Doctor and one of the parent sit or stand in front of each other. The parent holds the body up in the arms. In this position, the child's veins of the limbs in the dependent part swell. The venous pressure in the peripheral veins rises depending on the vertical distance between the heart and the vein that one wants to

pierce. Tying a tourniquet further helps. The assistant holds the hand and with aseptic precautions, the cannula is comfortably introduced in the turgid vein by the pediatrician. This procedure is best for the veins on the dorsum of the hand, wrist, and ankle (*Fig. 1*). The veins around elbow can also be taken well.

Experience of last serial about hundred such cannulations shows that one almost



Fig. 1. Photograph showing the position for peripheral venous cannulation.

always gets the vein, and gets it easily. The child does not struggle much as he/she is comfortable in the parent's arms. This facilitates the job. The previously informed parents have no tension, as they witness everything and the job is over in a few minutes. This position obviates the need of a table or a bed or a procedure room for

venous cannulation. It can be done in the consulting room or in emergency, anywhere.

Hemant Joshi,
Archana Joshi,
*Joshi Children's Hospital,
Virar 401 303.*

Glycogen Storage Disease Type II

Glycogen storage disease type II was the first lysosomal storage disease to be recognized. To date, few cases of GSD type IIa have been reported in the Indian literature(1). We report a similar case of GSD type IIa in a 14-month-old child proved by clinical, laboratory and histopathological features.

A 14-month-old first born female child of a non-consanguineous marriage was referred to us for generalized muscle weakness and intractable cardiac failure. She was born of a normal term delivery and was apparently normal at birth. There was no history of diminished fetal movements in the antenatal period. Parents initially noticed paucity of movements of the limbs by around one month of life. The weakness then progressed over the time. She had repeated aspiration of feeds since the age of 3 months. There was profound motor developmental delay since birth, but the social smile was attained at the normal age. There was no family history of a similar illness or any previous abortions in the mother. Examination showed a poorly nourished ill-looking child with central cyanosis and tachypnea. The respiratory excursions were shallow and the pulse was rapid and theready. The child was in a pithed frog position

with minimal body movements and a weak inaudible cry even on painful stimuli. There was marked cardiomegaly. Liver was palpable 3 cm and firm. There was severe hypotonia, areflexia and muscle weakness. The chest X-ray revealed massive cardiomegaly. ECG showed short PR interval, inverted T-waves in the limb leads and tall QRS complexes. The serum CPK level was 222 IU/L (normal range 20-50 IU/L).

The child died 50 hours after admission due to cardiorespiratory failure. A post-mortem skeletal muscle biopsy showed marked infiltration with glycogen. This finding along with the clinical and laboratory features confirmed the diagnosis of GSD type IIa.

GSD type II is a rare inherited metabolic disorder with an incidence of one in more than 100,000(2). Three clinical phenotypes are recognized: the infantile (IIa), childhood (IIb), and the adult (IIc) types. In the infantile form (also known as Pompe's disease) affected infants are usually normal at birth. Skeletal muscle and cardiac involvement become clinically obvious within the first few months of life as muscle weakness and resistant cardiac failure, respectively. Both these progress relentlessly and death occurs before the age of two years due to cardiac and/or respiratory failure.