Total parenteral nutrition is often necessary for the survival of neonates, but obtaining central venous access can be very difficult. A case is reported in which malpositioning of a 27 gauge central venous catheter resulted in the misdiagnosis of necrotising enterocolitis and hypoglycaemia.

Twins were delivered by emergency caesarean section at 29 weeks because of intrauterine growth retardation. Twin 1 weighed 0.766 kg, well below the 3rd centile, and required ventilation shortly after delivery. At 9 hours of age he was extubated to continuous positive airway pressure, and later a 27G Medex long line (Medex, Rossendale, Lancs, UK) was inserted using a 24G intravenous cannula in the left leg. Total parenteral nutrition was started.

The position of the long line was checked by injecting radio-opaque contrast immediately before x ray examination, which outlined the fine gauge catheter in the abdomen (fig 1). During the second day of life he deteriorated and required reintubation. His abdomen had become increasingly distended and oedematous and there were bile stained aspirates from the nasogastric tube. Also, a reddish mark was noted on the left side of the abdomen. He was treated for early necrotising enterocolitis and started on triple intravenous antibiotics. The following day he was extubated, but his abdomen remained distended and his scrotum had become swollen and tense. There was a localised area (1 cm x 1 cm) of pus, oozing serous fluid to the left of the abdomen (fig 2). His platelet count fell to 73, and an abdominal radiograph showed distended loops and oedematous bowel wall. The area of pus continued to ooze but the abdomen became less distended. A burn was suggested as a possible diagnosis, but no probes had been applied over the area. Microbiological swabs were also negative.

By day 6 the abdomen was improving, but unfortunately the infant had developed a new problem of hypoglycaemia. Confirmed by true blood glucose, the baby had persistent hypoglycaemia resistant to treatment with 15% dextrose, increased fluid volumes, and 10 mg/kg hydrocortisone. He received 15% dextrose at 180 ml/kg/day, equivalent to 18.5 mg/kg/min (normal range 4–8 mg/kg/min). Throughout this time the baby remained asymptomatic. Urine output was 11.6 ml/kg/h, and urine dipstick continually showed 4+ glucose, even in the face of such profound hypoglycaemia. Further doses of hydrocortisone were given, but glucose levels were never greater than 2.0 mmol/l. Hyperinsulinism was considered, and a serum sample was taken to measure insulin levels. Diazoxide was prescribed, but before this the long line site was re-examined. It looked normal and had continued to flush easily; however, a new long line was electrically inserted. One hour after starting 20% dextrose infusion, the blood glucose level was 16 mmol/l, the first satisfactory value in 24 hours.

The problems seen in this infant had resulted from malpositioning of the long line. Although on abdominal radiograph (fig 1), the catheter, outlined with an injection of contrast, appeared to be in the lower inferior vena cava, it was in fact in a superficial abdominal vessel, possibly the inferior epigastric. The abdominal wall oedema and “abscess” (fig 2) were secondary to total parenteral nutrition (TPN) extravasation. This was confirmed when the long line (still in situ) was flushed and the ulcer was seen to ooze fluid immediately. The variable and excessive amount of glucose in the urine was explained by leakage from the abdominal wall flowing down the abdomen and soaking into the nappy.

Abbreviations: TPN, total parenteral nutrition.
The baby made a good recovery, although the extravasation had caused a small full thickness scar which healed well. His developmental progress will be monitored as part of his neonatal follow up, although at no time did he have symptoms of hypoglycaemia and did not have a fit.

**DISCUSSION**

The use of very small peripheral intravenous catheters in neonates has risks which should always be borne in mind. Several malpositioning problems have previously been reported. Erosion of the left ventricle wall by the catheter tip resulting in a tamponade due to TPN in the pericardial space is well recognised. Other problems that have been reported are cardiac arrest due to TPN infiltrating the myocardium and degenerating myocardial fibres, neonatal subdural transudation of TPN, and pleural effusion caused by intrathoracic central line hyperalimentation. Extravasation from catheters in the inferior vena cava has been known to cause TPN ascites and acute abdomen in neonates. In the case of an infant of 30 weeks gestation, the diagnosis was also confused with necrotising enterocolitis and a laparotomy was performed.

In most cases, problems can be avoided by careful assessment of the position of the catheter tip and withdrawing it if necessary.

Medex were not previously aware of the problem of 27G catheters advancing into superficial abdominal vessels. Neonatologists should be reminded of the risks of using these very small peripheral long lines, and all medical practitioners should be reminded of the possibility of resistant hypoglycaemia due to catheter misfunction.

**REFERENCES**