the age of 3 months. Generalized weakness (proximal more than distal) and muscular fasciculations were present. A neurogenic pattern in electromyography and microscopically proved muscular atrophy (quadriceps femoris) supported a diagnosis of SMA. Nerve conduction velocity was normal as was the histology of a nerve biopsy specimen. Creatine kinase was moderately elevated (220 U/l). Molecular analysis showed a homozygous deletion in the SMN gene confirming the diagnosis of SMA.

Additionally, the boy showed a balanced translocation t(4q; 10q) (qter; q24.1) which is also present in his healthy mother. The father is healthy. The patient is the youngest in a sibship of four. A 9-year-old girl is healthy. The second child died at the age of 6 months from pneumonia in Kosovo. The third pregnancy ended in the second trimenon. The third child died at the age of 7 days with an unbalanced chromosomal aberration (partial trisomy 10, partial monosomy 4) and is published elsewhere [4, case 2]. The SMN gene locus of the other children was not investigated.

Considering the rarity of SMA (1:10000 to 1:25000) its concurrence with the above balanced translocation may not be coincidental. Assuming a causal relationship, there might be contributory factors on the chromosome regions 4qter or 10q24.

References

1. Cobben JM, Steege G van der, Grootscholten P, Visser M de, Scheffer H, Buys CHCM (1995) Deletions of the survival motor neuron gene in unaffected siblings of patients with spinal muscular atrophy. Am J Hum Genet 57: 805–808
2. Hahnen E, Forkert R, Marke C, Rudnik-Schöneborn S, Schönling J, Zerres K, Wirth B (1995) Molecular analysis of candidate genes on chromosome 5q13 in autosomal recessive spinal muscular atrophy: evidence of homozygous deletions of the SMN gene in unaffected individuals. Hum Mol Genet 4: 1927–1933
3. Lefebvre S, Burglen L, Reboullet S, Clermont O, Burlet P, Viollet L, Benichou B, Cruaud C, Millasseau P, Zeviani M, et al (1995) Identification and characterization of a spinal muscular atrophy determining gene. Cell 80: 155–165
4. Pfeiffer RA, Jünemann A, Lorenz B, Sieber E (1995) Aplasia of the optic nerve in two cases of partial trisomy 10q24-ter. Clin Genet 48: 183–187
5. Rodrigues NR, Owen N, Talbot K, Ignatius J, Dubowitz V, Davies KE (1995) Deletions in the survival motor neuron gene on 5q13 in autosomal recessive spinal muscular atrophy. Hum Mol Genet 4: 631–634
6. Rudnik-Schöneborn S, Forkert R, Hahnen E, Wirth B, Zerres K (1996) Clinical spectrum and diagnostic criteria of infantile spinal muscular atrophy: further delineation on the basis of SMN gene deletion findings. Neuropediatrics 27: 8–15
7. Rudnik-Schöneborn S, Wirth B, Hahnen E, Kreiß-Nachtsheim M, Zerres K (1996) Spinale Muskelatrophien – klinische Formen und Stand der molekulargenetischen Diagnostik. Med Genetik 8: 179–186
8. Velasco E, Valero C, Valero A, Moreno F, Hernández-Chico C (1996) Molecular analysis of the SMN and NAIP genes in Spanish spinal muscular atrophy (SMA) families and correlation between number of copies of ^{c}BCD541 and SMA phenotype. Hum Mol Genet 5: 257–263

G. Hofner (✉) · L Nährlich · H. Lauffer
Klinik mit Poliklinik für Kinder
und Jugendliche der Universität
Erlangen – Nürnberg,
Loschgestrasse 15,
D-91052 Erlangen, Germany
Tel.: 09131/853118

B. Scharrer
C. Rudin
P. W. Nars

Transumbilical venous access with small diameter silastic catheters in very low birth weight infants

Received: 1 July 1996 and
in revised form: 4 April 1997
Accepted: 8 May 1997

Sir: Central venous access is necessary to provide very-low-birth-weight (VLBW) infants with sufficient amounts of fluids and calories. In order to reduce discomfort related to a peripheral transcutaneous insertion method we evaluated a technique of insertion of the same small-diameter transcutaneous silicone catheters (Nutricath "S", 0.3 mm inner and 0.6 mm outer diameter, 30 cm length, Code 2184, Vygon, France) through the umbilical vein, using a conventional polyurethane umbilical vein catheter (1.7 mm outer diameter, 38 cm length, Ref: 8888–160226 argyle, Sherwood Medical, Belgium) as an introducer. This technique has been introduced by Amato et al. [1], who performed a similar study in 1992.

For the procedure both catheters are filled with 5% dextrose and inserted under strict sterile conditions at the bedside. After insertion the ubilical vein catheter is cut off at 1 cm above the umbilical stump and the small diameter catheter is then pushed forward through the lumen to a presumed central position and the polyurethane catheter withdrawn simultaneously. After fixation with small strips of adhesive surgical tape (Steristrip 3 M, St. Paul, USA), the correct position of the catheter tip is radiographically verified using contrast media (0.5 ml of preheated Lopamiro 300, Bracco, Italy).

Between 1990 and 1993 35 neonates with a mean birth weight of 1.114 (650–1490) g and a mean gestational age of 29.5 (27–34) weeks were provided with transumbilical small-diameter silicone catheters at a mean age of 2.7 (1–5) days after delivery. All except one patient required total or partial parenteral nutrition including fat emulsions due to prematurity and/or intolerance of gastro-intestinal feeding. They also received intravenous antibiotics.

The transumbilical venous catheters remained in place for a total of 459 (2–45; mean 13). days In 5 patients (18%) the catheter had to be removed prematurely due to complications, such as leakage and extravasation of contrast fluid in the subdiaphragmal area suggesting perforation, transitory colour change on the left gluteal region after dislocation, lack of drainage without proven thombosis, and leakage of the catheter, in one case each. Apart from these one lethal complication occurred. A catheter initially falsely positioned in the right atrium, perforated into the pericardium, causing cardiac tamponade.

Bacterial growth at the catheter tip at removal was found in 6 of 17 (26%) examined cases, coagulase-negative *Staphylococci* (4), *Enterobacter* (1) and *Candida albicans* (1) being isolated.

According to our very limited experience, this simple and painless technique seems to be a promising alternative to provide critically ill VLBW infants with a central venous access necessary for nutritional support and intravenous therapies. The incidence of complications and the frequency of bacterial growth at the catheter tip at removal are comparable to those found in series of percutaneously inserted small-diameter silastic catheters [1, 2]. Despite frequent isolation of pathogens at the catheter tips large series report catheter-related septicaemias to occur in only 1.9%–11% [1–3]. In our series one infant was clinically suspected to have a catheter-related infection, but no pathogen could be isolated from peripheral blood.

In conclusion our results are similar to those of Amato et al. [1] and seem to be comparable with those obtained in large

series of conventionally inserted percutaneous small-diameter silastic venous catheters in neonates [1–6]. However, further and larger prospective studies are needed to validate our data.

References

1. Amato M, Weissmüller A, Hüppi P (1992) Umbilicous venous silastic catheter – a new procedure in low-birth-weight infants. Neonatologica 2:117–20
2. Cairns PA, Wilson DC, McClure BG, Halliday HL, McReid M (1995) Percutaneous central venous catheter use in the very low birth weight neonate. Eur J Pediatr 154:145–7
3. Cathas MK, Paton JB, Fisher DE (1990) Percutaneous central venous catheterization. Three years experience in a neonatal intensive care unit. AJDC 144:1246–50
4. Harms K, Herting E, Krüger T, et al. (1992) Percutane Silastic Katheter bei Früh- und Neugeborenen. Ein Erfahrungsbericht über 497 Katheter in 5 Jahren. ronarbbers Kinderheilk 140:4464–71
5. Neubauer AP (1991) 250 zentralvenöse Silastic-Katheter bei Frühgeborenen unter 1500 g. Eine klinische Studie über Technik und Komplikationen. ronarbbers Kinderheilk 139:810–5
6. Neubauer AP (1995) Percutaneous central iv access in the neonate: experience with 535 silastic catheters. Acta Paediatr 84:756–60

B. Scharrer · C. Rudin · P. W. Nars (✉)
Universitäts-Kinderklinik Basel,
Roemergasse 8, Postfach,
CN-4005 Basel,
Switzerland,
Tel.: 061/691 26 26,
Fax: 061/692 65 55

I. M. Doughty
D. A. Price
N. J. A. Webb

Macroscopic haematuria following immunisation with tetanus toxoid and oral polio vaccine

Sir: Haematuria is not a recognised complication of immunisation with either tetanus toxoid or oral polio vaccines. We report a case of macroscopic haematuria following such immunisation.

The patient was a 15-year-old boy who was physically well and, in the past, had received routine immunisations with no adverse reactions noted. There was no past history or family history of renal disease and no history of recent throat, skin or other infection. Twenty-four hours following immunisation with tetanus toxoid and oral polio vaccines, he became unwell with headache and abdominal pain. Local swelling, erythema and tenderness developed around the site of the tetanus immunisation. Twenty-four hours later, he developed macroscopic haematuria and was admitted to hospital. General examination was unremarkable; blood pressure was normal. Urine microscopy confirmed haematuria. Subsequent investigations included urine culture, full blood count, serum electrolytes and creatinine, serum complement C_3 and C_4, immunoglobulins, plain abdominal X-ray, ultrasound scan and urinary calcium/creatinine ratio. The results of all of these investigations were normal. The haematuria spontaneously resolved and there has been no recurrence.

The episode of haematuria was temporally related to immunisation, and prior to this, there were symptoms suggesting a generalised reaction to one of the vaccines. It is therefore probable that the haematuria occurred as a direct result of immunisation. On enquiry to the U.K. Medicines Control Agency, we learnt that there had been five previous unpublished cases of haematuria associated with tetanus immunisation reported to them (personal communication). In these cases, the onset of haematuria occurred up to 4 days following immunisation in a group ranging in age from 5 months to 65 years. The long-term outcome in this group is unknown. There has been only one previous report of haematuria related to administration of oral polio vaccine. It is clearly important that any similar cases should be reported.

This child was appropriately investigated to exclude common causes of haematuria. In this case renal biopsy was not indicated but it is important to recognise that this child may still have an underlying renal disorder, despite the absence of a past history or family history. Given the apparent infrequent association between haematuria and immunisation, we would suggest that all similar cases still need to be fully investigated and followed up to exclude other more common and significant causes of haematuria.

I. M. Doughty · N. J. A. Webb (✉)
Department of Paediatric Nephrology,
Royal Manchester Children's Hospital,
Pendlebury,
Manchester M27 4HA, U.K.
Tel: +44 161 727 2435
Fax: +44 161 794 5929
email: nwebb@man.ac.uk

D. A. Price
Department of Child Health,
Royal Manchester Children's Hospital,
Pendlebury,
Manchester M27 4HA, U.K.