Letters to the Editor

Neonatal Pericardial Tamponade from a Silastic Central Venous Catheter

Central venous catheters (CVC) form an integral part of management in most neonatal intensive care units. They are used for monitoring of central venous pressures (CVP), hyperalimentation and for long term intravenous access. Surgically placed central venous lines have been largely replaced by the use of the more pliable, peripherally sited silastic central venous catheters. However the use of these catheters is still associated with complications. This baby developed a fatal pericardial tamponade as a result of a silastic central venous catheter.

A female 630g preterm infant was born vaginally at a gestation of 24 weeks to a 18yr old woman with a history of ante partum hemorrhage two weeks prior to delivery. The baby was ventilated from birth for Respiratory Distress Syndrome. Just one dose of Exosurf was required and her oxygen requirement settled at 30%. Her blood pressure was very labile and she was commenced on infusions of Dopamine and Dobutamine at 6 mcg/kg/min each to maintain normal blood pressure. An echocardiogram done on day four showed no patent ductus arteriosus and a bicuspid aortic valve with trivial aortic regurgitation. She had been commenced on antibiotics at birth in view of her respiratory distress. All cultures sent at birth were sterile. To facilitate venous access a per-Q-cath silastic central venous catheter was inserted via the ante cubital vein on day 3. Check chest *X*-ray confirmed the tip of the catheter to be in the superior venacava (SVC). Head ultrasound revealed a small grade II intraventricular hemmorrhage on the left side. The baby was stable on minimal ventilator pressures and in a FiO_2 of 30% from day 3.

On day 6 she had suddden onset of recurrent episodes of desaturations. Her oxygen requirement increased to 70%. Cold light examination did not show any evidence of a pneumothorax or a pneumomediastinum. She continued to have adequate chest movement at minimal pressures and a CXR showed no air leaks or consolidation and a normal cardiac shadow. A septic work up was performed and she was started on Vancomycin and Ceftazidine. This was followed shortly afterwards with an episode of severe bradycardia and hypotension. Inotoropes were increased to maximum support, endotracheal tube was changed and a rapid infusion of colloid was given. However the response to these mesures was transient. She had a cardiac arrest from which she could not be revived. Consent for autopsy was obtained from the parents.

Autopsy showed a large pericardial effusion compressing the heart. A total volume of 5 ml was aspirated from the pericardial cavity which when compared to the heart weight of 4.4g was significant enought to interfere with cardiac filling. There was no evidence that the catheter tip had perforated the cardiac wall. Apart from a small peritoneal and pleural effusion and a small left sided germinal layer hemmorrhage the rest of the post mortem was unremarkable. Cell count of the pericardial fluid was zero and culture was sterile. All ante mortem and post mortem cultures were sterile. Retrospective review of the last CXR showed that the long line had migrated and that the tip of the catheter now lay within the cardiac shadow.

Pericardial tamponade is a rare complication of central venous catheters. It occurs in 1% of all adults with central venous catheters and despite recognition has a high mortality of approximately 80%(1). This complication has also been seen in neonates especially with the use of polyethylene and other stiff catheters used for umbilical catheterization and percutaneous central lines(2,3). Although rare, recent reports have shown that pericardial effusions can occur even with the use of the pliable silastic catheters in extremely premature low birth weight infants(4,5). As in adults, this complication is associated with a high mortality rate which varies from 66% to 100%(2,5).

The exact pathogenesis of pericardial effusions is not clear. Perforation of the cardiac wall by the catheter tip during catheter insertion is responsible in some of the cases. In others where there is delayed onset of effusion, it has been postulated that the catheter tip lying within the heart comes in repeated contact with the endocardium. This repeated trauma leads to endocardial damage and thrombosis. The catheter then adheres to the endocardium and erodes through the myocardium leading to extravasation and pericardial effusion. These erosions may be very small and may be missed on post mortem studies. In our patient during insertion the tip of the catheter had been placed high in the SVC. However, the last CXR showed the tip to have migrated to lie well within the cardiac shadow. The repeated mechanical trauma combined with the hyper-tonicity of the infusate could have resulted in thrombus formation, tethering to the endo-cardium and then erosion of the myocardium.

Symptoms of catheter related pericardial effusion are typically sudden in onset. In case reports the most frequently reported symptoms have been desaturations with unresponsive hypotension and bradycardia(4). Some babies have presented with a sudden cardiac arrest. Effective management requires a high index of suspicion and a rapid diagnosis. An echocardiographic diagnosis would be helpful but in its absence a diagnostic pericardial tap should not be delayed. It may prove to be life saving. The volume of fluid aspirated may be very small. It has been shown that a volume of 11.4 ± 1.5 ml/kg body weight (mean ± SD) is enough to result in tamponade(4).

Several factors have been implicated in increasing the risk of pericardial effusion including the type of the catheter, size of the catheter and location of the catheter tip. In a recent case series of 906 central venous catheters over a five year period, polyethylene and polyurethane central venous catheters were responsible for more pericardial effusions (4 patients) as compared to silastic catheters (2 patients)(3). Location of the catheter tip within the myocardium is associated with a high risk of perforation. However catheters with their tips in the SVC have also been reported to result in pericardial effusion(3). Catheters inserted via the neck or the arm veins have been shown to advance into the heart by up to 2 cm with movements of the neck and the arm. An optimum site for such catheters should be high in the SVC so that even with movement the catheter tip does not advance into the heart. However, despite all precautions in positioning the tip, catheters have been shown to migrate. Hence, a diagnosis of pericardial effusion should be considered in all babies who have a sudden unexplained deterioration in their clinical status so that timely pericardiocentesis can be performed.

Rajiv Aggarwal,
Assistant Professor,
Department of Pediatrics,
St Johns Medical College and Hospital,
Bangalore 560 034.
and

Lynette Downe,
Director,
Neonatal Intensive Care Unit,
Nepean Hospital, Penrith, NSW 2750,
Australia.

REFERENCES

1. Dane TEB, King EG. Fatal cardiac tamponade and other mechanical complications of central venous catheters. Br J Surg 1975; 62: 6-10.
2. Agarwal KC, Khan MAA, Falla A, Amato JJ. Cardiac perforation from central venous catheters: Survival after cardiac tamponade in an infant. Pediatrics 1974; 73: 333-338.
3. Keeney SE, Richardson CJ. Extravascular extravasation of fluid as a complication of central venous lines in the neonate. J Perinatol 1995; 15: 284-288.
4. Khilnani P, Toce S, Reddy R. Mechanical complications from very small percutaneous central venous silastic catheters. Crit Care Med 1990; 18: 1477-1478.
5. Necubauer AP. 250 central venous silastic catheters in premature infants less than 1500g. A clinical study of technique and complications. Monatsschr Kinderheilkd 1991; 139: 810-815.

BETARU: An Indigenously Designed Cordless Stethoscope

Despite being a very useful instrument, a conventional stethoscope does have limitations. Auscultating a child in a teaching environment invariably ends up in the child crying by the time the 6th or 7th student examines the child (a problem I came across during my 3rd year MBBS posting in Pediatrics). I therefore developed an indigenous cordless stethoscope (Betaru) to overcome this limitation (*Fig. 1*).

Betaru generates electrical signals enabling a wide spectrum of applications. It represents an advanced stethoscope with enhanced features like volume control and amplification, sound filteration circuits, internet transferable signals, signal recording and assessment facility and a portable cordless phonocardiogram.

Betaru was constructed using FM signals, transmitter, receiver and various amplifying and filtering units. It utilizes the standard type of a chest piece, allowing rotation to the bell/diaphragm position, like a conventional stethoscope. The transmitting unit weighs 70 grams and can be easily gripped in one hand. The Receiving unit is 200 grams which can be clipped onto any pocket or placed along with the head phones. The total manufacturing cost is around Rs. 5000/- with each head set for Rs. 2500/- (excluding research, development and other costs).

The important advantages of Betaru are: (*i*) It is small, compact, handy and cordless giving greater ease and mobility to a doctor; (*ii*) Signals can be stored on a computer or other

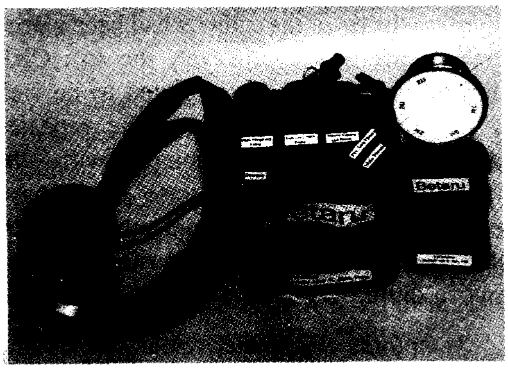

Fig 1. Photograph of the indigenously developed cordless stethoscope. On the left are the headphones, in the middle is the receiving unit and on the right is the transmitting unit.