Pericardial tamponade by Holter ventriculoatrial shunts

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Ten cases of cardiac tamponade caused by pericardial perforation by a Holter catheter in the treatment of hydrocephalus by ventriculoatrial drainage are reported. These 10 cases occurred in an 8-year practice of two surgeons in a period during which approximately 1800 Holter shunts were inserted and an approximately equal number of operations for lengthening of the distal catheter were performed. The etiological factors, the clinical picture, and the diagnosis are discussed. Cardiac perforation is caused either by forceful introduction of the distal catheter during a shunt revision procedure or by too long a distal catheter that ulcerates through the cardiac wall. Enlargement of cardiac silhouette after shunt insertion is virtually diagnostic of a pericardial effusion. If undiagnosed, this condition was invariably fatal, but if diagnosed in time, immediate treatment was always curative. The distal catheter should be shortened, but suture of the cardiac perforation itself is not necessary.

KEY WORDS hydrocephalus ventriculoatrial shunts cardiac perforation pericardial effusion cardiac tamponade

COMPLICATIONS of Holter shunts have been reported in a large number of cases and after long periods of follow-up. Our own complications have been presented and discussed previously. We are concerned here with pericardial tamponade following insertion or revision of the Holter shunt, a rare but lethal complication if not diagnosed and treated promptly.

This complication has been reported on three occasions and briefly mentioned in three publications.

Clinical Material
The present report was obtained from the study of hydrocephalic patients treated at the Westminster Children’s Hospital, London; The Hospital for Sick Children, Great Ormond Street, London, and Queen Mary’s Hospital for Children, Carshalton, where over a period of 8 years more than 1800 Holter ventriculoatrial shunts have been inserted and an approximately equal number of operations for therapeutic or prophylactic lengthening of the distal catheter have been performed. Ten cases of pericardial tamponade occurred. These form the basis of our study of the etiological factors, clinical picture, diagnostic methods, treatment and prevention.

Results

Etiological Factors
The first consideration was the type of distal catheter used and whether its introduction was difficult or forceful. A Holter type “A” catheter was used in each of the 10 cases; it was forcefully introduced in two of
After reviewing the x-ray films taken following insertion of the shunt or revision of the distal catheter, it was obvious that malposition of the tip of the catheter was an important causative factor, occurring in seven cases.

The two cases due to forcible introduction followed operation for repeated obstruction of the distal catheter, which was finally inserted with difficulty on a stilet and under x-ray control. The two children died 21 hours and 7 days after the operation respectively. At postmortem examination pericardial tamponade with cerebrospinal fluid was found. In the first case, the catheter was in a false passage outside the jugular vein; it did not enter the cavity of the right atrium but passed through a papillary muscle, so that its tip reached an area of ecchymosis 1 cm in diameter over the outer aspect of the right ventricle. In the second child it was found that the catheter was draining into the pericardium, having perforated the wall of the superior vena cava. The operative and postoperative x-ray films showed that the catheter was apparently within the cardiac silhouette, but examined in retrospect its course could be considered abnormal.

In the remaining cases, the introduction of the catheter was done without any difficulty, either during primary insertion (four cases) or at lengthening of the distal catheter. Except for one case, the films for which have since been lost, all the distal catheters were long and their tips lay in an abnormal position within the cardiac silhouette.

In one, the tip of the catheter after primary insertion was in the upper part of the inferior vena cava, and 1 month later, when the child developed signs of tamponade, the tip was just in the right atrium pointing posteriorly.

In another child, the tip of the catheter immediately after revision was in the right ventricle, but 4½ months later the tip had passed through the atrial wall.

In a third child, the catheter, during primary insertion, was placed low in the inferior vena cava. (This was done by means of electrocardiographic electrode in a small group of children as an attempt to overcome the problem of growth, but proved unsuccessful.) Four months later the catheter had curled back on itself, passing anteriorly, probably into the right ventricle (Fig. 1); 4 months after this observation the child died from pericardial cerebrospinal fluid effusion. At postmortem examination, the tip was in the right ventricle in a pocket between the papillary muscles, and a pinhole orifice was present in the myocardium at this point.

In a fourth patient, the catheter, after revision, was found to be bent acutely to the left and was in the same position 1 week later when he developed signs of tamponade (Fig. 2).

In a fifth, after revision, the tip was turned to the right in the right atrium; he developed tamponade 2 weeks later (Fig. 3).

In a sixth case, immediately after primary insertion the catheter was directed toward the posterior wall of the right atrium; 5 weeks later perforation of the atrial wall occurred.

In a seventh case, immediately after an azygos shunt the catheter was long and turning backward; 2 weeks later tamponade developed (Fig. 4).

In the cases described above, where no force was used in the introduction of the distal catheter, the most likely cause was that the tip of the catheter became impacted within the pectinate or papillary muscles and...
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slowly perforated the myocardium as a consequence of normal cardiac contraction.

Clinical Characteristics

The course of illness was shorter and more dramatic in the four fatal cases in which cardiac arrest occurred within a period of 5 to 48 hours from the onset of signs. In the remaining patients, the diagnosis was made within 5 to 11 days.

All the patients had dyspnea and cyanosis, four had hepatomegaly, three abdominal distension, and two peripheral edema. Tachy-
cardia was common, while sinus arrhythmia was recorded once, and muffled heart sounds only twice.

Six children were treated initially for pneumonia and three for heart failure, without any improvement. There was no correlation between the severity of the heart failure and the outcome; nor was there any relationship between the size of the effusion and the severity of the heart failure or outcome. In one patient, 420 ml of cerebrospinal fluid were present without severe distress, yet in two other patients 50 ml were found at post-mortem.

Recovery was rapid, often dramatic, after a pericardial tap, and all the signs disappeared very soon after revision of the distal catheter.

Five children had clinical signs of inadequately controlled hydrocephalus preceding the signs of cardiac failure, although the valve appeared to pump without noticeable difficulty.

**Investigations and Diagnosis**

In five of the six successfully treated cases, the diagnosis was made from the chest films and confirmed by pericardial tap. The sixth was diagnosed when the chest was opened for internal cardiac massage. Two of the fatal cases had no chest films and one had only moderate heart enlargement. In the other cases, the heart was grossly enlarged with a globular shape (Figs. 2 and 4 right), except for one which had a “water bottle” configuration, typical of tamponade.

In one case the diagnosis was confirmed by the injection of 10 ml of Hypaque into the valve; chest films were then taken after pumping the valve. An electrocardiogram was performed in two patients; it was normal in one. The other showed low voltage which returned to normal postoperatively.

**Treatment and Outcome**

The four patients who were undiagnosed died. Three have already been mentioned; the fourth occurred after therapeutic revision of the distal catheter, which was performed under x-ray control. Its position was thought to be satisfactory, but the films are not now available for review. The patient developed respiratory distress 3 weeks later and cardiac arrest within 6 hours; during injection of cal-
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passage during a revision operation unless it is threaded on a fine (and traumatic) stilet. Forceful introduction of the distal catheter is on many occasions unavoidable, and we were surprised to find no similar traumatic complications when we recently reviewed 322 lengthenings of the distal catheter.\(^7\)

Revision under x-ray control can only give a crude picture of the relationship of the catheter to the cardiac silhouette, and none of its actual position.

From the incidence of tamponade with malpositioned catheters, it is obvious that these catheters should be revised or shortened prophylactically.

All the classical clinical signs\(^{10}\) and electrocardiographic changes of pericardial effusion need not necessarily be present. Respiratory symptoms (or any signs of heart failure) in the presence of an enlarged heart shadow after insertion of the shunt or revision of the distal catheter should arouse suspicion, which can be confirmed easily by injecting Hypaque into the valve, pumping the valve briefly, and taking a chest film.

A pericardial tap can be a lifesaving procedure, which should be followed by simple shortening or repositioning of the distal catheter. From the operative and postmortem findings, both in our cases and three reported ones, the slow ulceration of the myocardium needs no repair.

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**References**


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