Histopathological findings in three children with His bundle tachycardia occurring subsequent to cardiac surgery

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This report concerns three children with His bundle tachycardia who died following cardiac surgery. At autopsy the conduction system was examined in detail. In all three, the sinus node was intact and supplied by a well-formed artery. Haemorrhagic tracks were identified invading the penetrating atroventricular bundle. The tracks originated from stitches placed close to the conduction tissue. The hypothesis that disruption of the conduction tissue results in an arrhythmogenic focus is discussed.

Introduction

His bundle tachycardia, otherwise known as junctional ectopic tachycardia, remains a life-threatening complication of surgery used for treating congenital heart disease. The anatomical or pathological basis of this has not previously been documented. Histopathological findings in three children who died with His bundle tachycardia following surgery are discussed.

Case reports

An 11-month-old girl underwent surgical repair of tetralogy of Fallot combined with an atroventricular septal defect. She had sickle cell disease and a congenital balanced translocation of chromosome 3 with 16; this resulted in a wide flattened nose and pointed ears. The surgical repair was considered straightforward but she returned from theatre in His bundle tachycardia which increased in rate to a maximum of 210 beats min⁻¹. She became hypotensive and required dopamine, adrenaline and isoprenaline support. Her chest was splinted open, she was cooled to a central temperature of 31-32 °C and atrial pacing was instituted to restore atrioventricular synchrony (Fig. 1), but repeated cardiac arrests followed and she died 17 h after surgery.

The second patient was an 11-year old boy who underwent surgical correction of tetralogy of Fallot, valvar pulmonary stenosis and closure of an atrial septal defect. The correction was performed using a transjunctival patch to widen the right ventricular outflow tract. On return from the operating room his rhythm was His bundle tachycardia with a maximum rate of 190 beats min⁻¹ (Fig. 2). Dopamine was used to support his blood pressure and amiodarone given to decrease the rate of the tachycardia. The latter was unsuccessful and after a delay of 22 h the child was cooled and an oesophageal electrode positioned to enable atrial pacing to be performed at a rate such that

Figure 1 (a) Leads I, II and III of an electrocardiogram recorded from case 1 showing a His bundle tachycardia. The QRS complexes are wide as a result of right bundle branch block. Atrioventricular dissociation is seen. (b) The same leads of an electrocardiogram recorded during atrial pacing. The atrial paced impulse is conducted to the ventricle and the QRS complex morphology fails to change. Paper speed = 25 mm s⁻¹.

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atrioventricular synchrony was restored. This resulted in an immediate increase in blood pressure of 20 mmHg; a temporary transvenous atrial electrode was then inserted. Despite these measures the child’s general condition slowly deteriorated. His chest was splinted and an adrenaline infusion started but he died 55 h after surgery.

The third child was a newborn male infant (birth weight 3.6 kg) who was noted to be in cardiac failure from the time of delivery. Echocardiography and cardiac catheterization confirmed a diagnosis of a dysplastic mitral and tricuspid valve with severe mitral incompetence. Because of a rapid deterioration in his clinical condition a mitral valve repair was undertaken on the third day of life. Surgery successfully reduced the degree of mitral incompetence but the cardiac rhythm was intermittent complete atrioventricular block. The ventricular escape rate was adequate and pacing was not necessary. However at 12 h following surgery he developed His bundle tachycardia with a maximum rate of 210 beats min⁻¹. He was cooled to a core temperature of between 30 °C. Atrial pacing was conducted to the ventricle, but atrial pacing synchronous with the His bundle was used to restore atrioventricular synchrony without further increase in ventricular rate. Despite this the child remained in low cardiac output. Adrenaline, dopamine and nitroprusside were used but his condition deteriorated and he died 36 h following surgery.

In each case permission for autopsy was given and the gross anatomical cardiac findings confirmed the pre-mortem diagnosis. The conduction system was examined in detail by serial section. In all three patients the sinus node was found to be well formed and supplied by an intact sinus node artery. On examination of the atrioventricular junction in case 1 an extensive area of haemorrhage was found in the atrial septum with streaks of haemorrhage extending towards the atrioventricular node (Fig. 3). The node itself was well formed. Haemorrhagic streaks were traced to the junction between the penetrating atrioventricular bundle and the node further into the distal part of the atrioventricular bundle on the left side of the septal crest. More anterior sections showed damage to the branching bundle due to a deeply placed suture and further damage to the distal portion of the slender right bundle branch on the right side of the septum. The atrioventricular node of case 2 was more extensive than usual with several islands of specialized tissue seen in the central fibrous body. Interstitial haemorrhage could be seen in the common bundle and the proximal part of the branching bundle, while a haemorrhagic track, composed of inflammatory infiltrate, was seen to pass anteriorly, ascending along the fibrous sheath. By tracing the track retrogradely it was found to originate from two sutures placed close to the branching bundle, Fig. 4. In the posterior part of the atrioventricular node in case 3 haemorrhagic incursions were evident that had tracked from the lower part of the atrial septum. More anteriorly, the node was well formed but there was haemorrhage into the superficial layers and invading the compact node near the beginning of the penetrating bundle. The conduction tissues distal to this were undamaged, Fig. 5.

Discussion

His bundle tachycardia remains a life-threatening complication of surgical repair of congenital heart disease. The rapid ventricular rhythm with loss of atrioventricular synchrony and subsequent haemodynamic compromise...
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some physicians prefer to call this junctional ectopic tachycardia. Treatment is difficult but early recognition is essential since a poor cardiac output will lead to an increase in adrenergic tone and a consequent increase in the tachycardia rate\(^2\). Further sympathomimetic stimulation by drugs should ideally be avoided but is often not possible. The rhythm is refractory to conventional antiarrhythmic drugs\(^3\,4\). Recently Garson et al. have reported promising results with the use of intravenous propafenone\(^5\). All seven children with postoperative His bundle tachycardia treated with propafenone responded with a fall in tachycardia rate. However, propafenone had to be given slowly to avoid significant falls in systolic blood pressure. The authors found that the hypotension so caused could be reversed by colloid administration and therefore they recommend the total dose of propafenone is given as a series of bolus doses of 0-2 mg kg\(^{-1}\) at 10-15 min intervals for 10 doses with 1 ml kg\(^{-1}\) of colloid after each dose increment. Amiodarone has also been used in postoperative His bundle tachycardia\(^6\). Four of seven children treated with intravenous amiodarone responded with significant slowing of the tachycardia rate. The use of amiodarone was not associated with hypotension. Alternative non-pharmacological management of these patients has been described. A novel approach is the use of hypothermia to control the tachycardia rate\(^7\) and this was employed in each of our cases. Waldo et al. described the technique of paired ventricular pacing to halve the mechanical ventricular rate\(^8\). Atrial pacing synchronized to the R wave has also been used to restore atrioventricular synchrony\(^9\). The restoration of atrioventricular synchrony in this manner would appear

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**Figure 3** A series of photomicrographs from case 1 showing sections through the posterior atrioventricular junction. (a) A large area of haemorrhage is seen above the atrioventricular node and there is a streak toward the right side of the node. (b) A more anterior section, taken through the penetrating atrioventricular bundle, shows a track of haemorrhage (arrows) within the central fibrous body. (c) A section at the level of the ventricular septal defect shows a deeply placed suture adjacent to the branching bundle. (Masson's trichrome stain.)

**Figure 4** A section through the ventricular septum at the level of the branching bundle from the heart of the second case. The boxed area in (a) is enlarged in (b) to show the proximity of the sutures to the conduction axis and the haemorrhagic tracks (open arrows). (Masson's trichrome stain.)

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to increase cardiac output without further rise in heart rate. Despite these strategies, some children continue to die with His bundle tachycardia.

The exact aetiology of His bundle tachycardia occurring subsequent to surgery is unknown. At a cellular level it has been suggested that the mechanism may be that of delayed after-depolarizations. Because of the association of the arrhythmia and surgery involving the upper ventricular septum, it has been presumed to result in some way from trauma to the conduction tissues. However, the natural history of the arrhythmia is spontaneous termination after 3–8 days, if the child survives, suggesting that the damage sustained is not permanent. In all three children described in this report, the penetrating atrioventricular bundle was invaded by haemorrhage tracking from stitches placed close to the atrioventricular conduction system. We suggest that disruption of the conduction tissues so caused may result in an irritable focus. It is interesting that the third child suffered both intermittent complete atrioventricular block and later His bundle tachycardia suggesting that the aetiology of these two tachycardias may be similar. Complete heart block occurring as a consequence of stitches placed within the conduction tissue, has long been recognized as a complication of surgical repair of congenital heart disease. The course of the conduction tissue in most congenital heart disorders has now been outlined and can be avoided. His bundle tachycardia as a consequence of stitches placed near to the conduction tissue, which result in haemorrhagic infiltration of the conduction axis, is less well appreciated as a cause of postoperative morbidity and mortality. Since this rhythm remains extremely difficult to treat, we would emphasize the need for careful avoidance of not only the conduction tissue but also the immediate surrounding area, wherever possible, in order to prevent this complication.

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References