bulboventricular spur is formed, projecting between the common ventricle and the bulbus cordis. Normally, this large spur is absorbed into the ventral wall of the ventricle and is reduced to the bulbo-atrial spur, while at the same time the bulbus cordis shifts and grows to the left to form most of the right ventricle. Concurrent with both the above events, the muscular portion of the interventricular septum is formed. It has been postulated that the haemodynamic consequences of a developmental arrest at the time of ventricular septation may in turn give rise to the various other findings which occur with the single ventricle such as rudimentary chamber, transposition, and corrected transposition of the great vessels (Harley, 1958).

Campbell, Reynolds, and Trounce (1953) reported that partial pulmonary obstruction improved prognosis. The proband, in contrast to her sib with a normal-sized pulmonary valve, lived 7 months, possibly because pulmonary stenosis limited the pulmonary blood flow.

Environmental factors such as drugs or viruses are known to cause congenital heart malformations, but in general these teratogens cause multiple system anomalies, and affect only one member of a sibship. In the present family the occurrence of two sibs with such strikingly similar, isolated, and rare heart malformations is strong evidence for genetic determination. The most likely modes of genetic transmission appear to be an autosomal recessive trait or a discontinuous polygenic trait.

**Summary**

Two sisters with single ventricle are presented as evidence for genetic determination of this malformation. Embryogenesis of single ventricle is reviewed.

**References**


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**Endocarditis in the Neonatal Period**

Vegetations on the heart valves of newborn infants are rare at necropsy. Hudson (1965) in his textbook mentions the presence of non-bacterial vegetations on the heart valves in the neonatal period only in the context of Lambi's excrescences. In diseased valves, these contribute to the general roughening of the contact margin, and may build up as a granular and friable mass of platelets and fibrin. The underlying valve shows no cellular reaction and the whole lesion resembles the terminal or cachectic vegetations seen in the elderly and the degenerative verrucal endocarditis of Allen and Sirota (1944). While these lesions are usually papillary, some may be more warty and may vary from the size of a pinhead to several mm in diameter. Hudson does not recognize infective endocarditis in this age group, though three fairly well-documented cases can be found in the literature, as well as one rather incompletely recorded case (see Table). We here report a further case.

**Case Report**

The infant was the third child of a Rhesus positive mother aged 29 years, who developed pre-eclamptic toxæmia at 37 weeks, and was delivered by lower segment caesarian section at 39 weeks. The mother had chronic bronchitis with poor respiratory function, but suffered no acute exacerbations during the pregnancy. Two previous pregnancies had terminated in forceps deliveries; the first infant (male, 2300 g) died aged 1 week from intracranial haemorrhage, the second (male, 4500 g) died during delivery. The present infant (male, 3133 g), who cried early, but then went limp and was resuscitated, appeared normal with a clear chest and normal heart sounds. By 24 hours, the infant had developed respiratory distress and began to have apnoeic attacks. Chest x-ray was consistent with hyaline membrane disease and the infant was maintained on intermittent positive pressure respiration with oxygen until death at 40 hours.

At necropsy the only significant gross findings were confined to the thorax. The lungs were fully expanded and a dull cherry red colour. There was no evidence of pulmonary embolism or infarction, and no broncho-
The mitral valve showed friable yellowish vegetations 4 × 4 × 5 mm on each cusp (Fig. 1). Similar smaller vegetations were also present on the ring of the closed interatrial foramen. The tricuspid, pulmonary, and aortic valves were normal. The ductus arteriosus was just patent. The only other abnormal finding was some areas of haemorrhage in the medulla of both kidneys.

**Histologically** the vegetation was composed of platelet and fibrin thrombus attached to a valve cusp which contained a moderate mononuclear infiltrate (Fig. 2). The endocardial lining cells were deficient and the elastic tissue on the atrial aspect of the cusp was distorted. No bacteria or viral inclusions could be identified. Small emboli of platelet/fibrin thrombus could be identified in arterioles in both renal medullae adjacent to the areas of haemorrhage. No other embolic phenomena were noted. Histological examination of the lungs confirmed the presence of hyaline membrane with focal atelectasis. Culture of the valve vegetation was negative.

**Discussion**

Heart failure in the newborn is usually due to congenital anomaly involving the chambers, coronary arteries, great vessels, or myocardium, or to infection such as Coxsackie myocarditis. Vegetative endocarditis appears to be a rare finding in this age group. McDonald (1950) ascribed his case to thrombotic vegetations forming on a deformed valve, and proposed that the deformity was a ruptured blood cyst. Plaut (1939) and Plaut and Sharnoff (1935) were more definite and ascribed their cases to some unidentified infective agent, though in both cases search was made for blood cysts. Poynton (1909) has the distinction of recording the first case of acute endocarditis in the

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**Fig. 1.—Heart from the posterior aspect showing the opened left ventricle. Large friable vegetations can be seen on the mitral valve.**

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**Fig. 2.—Valve cusp showing fragments of a platelet/fibrin vegetation on the atrial aspect. There is a mononuclear cell infiltrate in the cusp beneath the vegetation.** (Masson's trichome. × 30.)
neonatal period, but failed to record any histological examination of the valve cusp, apart from the statement that numerous organisms were present within the vegetation. The mother in this case had an attack of rheumatic fever late in pregnancy, and Poynton proposed this as the origin of the valvular vegetations. The present case shows sizeable vegetations on both cusps of the mitral valve with a cellular reaction in the body of the cusp, though no infective agent could be identified. Blood cysts were present on the mitral valve, but there was no evidence of cyst remnants in the base of the vegetations. The atrial dilatation and coronary vein distension are features of cardiac failure, and there was evidence of systemic embolization of fragments of the vegetation to the kidneys. As with the previous recorded cases, with the exception of Poynton’s, there seems nothing in the maternal history to account for the infective process.

Boyd (1965, 1967) described a case (3 and 5, respectively) in which there were firm vegetations on the tricuspid valve, but there was no cellular reaction in the valve cusp and he attributed the vegetations to a generalized fibrin thromboembolic disorder. There are, however, certain similarities between Boyd’s case and the present one. Both mothers had chronic bronchitis, though without acute exacerbation during pregnancy; and both infants had hyaline membrane disease, the membrane being poor in stainable fibrin in each case, possibly reflecting the generalized fibrin consumption. The vegetations in our case were composed of fibrin and platelets, as were also the thrombi in the renal vessels, and there was a cellular reaction in the valve cusp.

Summary

A case of vegetative endocarditis in an infant aged 40 hours is presented. This very rare finding has only been reported previously in 4 cases.

I should like to thank Dr. J. A. Black and Mr. T. Smith, Jessop Hospital for Women, Sheffield, for permission to publish this case.

<table>
<thead>
<tr>
<th>Author</th>
<th>Age</th>
<th>Birthweight (g)</th>
<th>Affected Valve</th>
<th>Other Features</th>
</tr>
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<tbody>
<tr>
<td>Poynton (1909)</td>
<td>2 dy</td>
<td>Unstated</td>
<td>Mitral</td>
<td>Multiple pulmonary emboli</td>
</tr>
<tr>
<td>Plaut and Sharnoff (1935)</td>
<td>$\frac{1}{2}$ hr</td>
<td>1162</td>
<td>Mitral</td>
<td>Multiple pulmonary emboli, hyaline membrane disease</td>
</tr>
<tr>
<td>Plaut (1939)</td>
<td>1 dy</td>
<td>Unstated</td>
<td>Tricuspid</td>
<td>Fibrin thromboembolism, hyaline membrane disease</td>
</tr>
<tr>
<td>McDonald (1950)</td>
<td>17 hr</td>
<td>2135</td>
<td>Tricuspid</td>
<td>Renal emboli, hyaline membrane disease</td>
</tr>
<tr>
<td>Boyd (1965, 1967)</td>
<td>21 hr</td>
<td>2550</td>
<td>Tricuspid</td>
<td></td>
</tr>
<tr>
<td>Present case</td>
<td>40 hr</td>
<td>3133</td>
<td>Mitral</td>
<td></td>
</tr>
</tbody>
</table>

REFERENCES


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Rippling Mattress Worked From Compressed Air Supply

This communication describes the application of a new technique, i.e. fluidic switching, which can improve the manufacture and hence the clinical application of the rippling air-filled mattress. The application of such devices, e.g. the Hawksley Rippling Bed, to the modern therapy of bed sores is well established: the contribution of this note is to draw attention to some interesting new possibilities in the paediatric context.

The rippling mattress described here operates directly from any compressed air supply having a pressure greater than 0·1 kg/cm$^2$ and capable of delivering a flow of 15 l/min (i.e. it is compatible with the standard type of ward and theatre-piped gas outlet point though of course, for reasons of safety, oxygen ought not to be used for this purpose). The air flow to the different mattress segments is controlled entirely by means of fluidic switching elements* mounted under the inflatable part of

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*Plessey Bistable Amplifier type BS: see Fig. 1.*
Endocarditis in the neonatal period.

A M Ward

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