showed no ischemic ST changes with exercise testing and has been free of any anginal symptoms for more than two and one half years of clinical followup experience.

The close temporal association of the administration of isoproterenol coupled with acute myocardial necrosis strongly suggests that isoproterenol can, in appropriate doses, produce myocardial necrosis in man.

This report does not indicate a new hazard in the use of catecholamine bronchodilators in standard prescribed doses. Indeed, low doses of catecholamines over long periods may actually have a beneficial effect on the myocardium. Rather, it should remind the physician to obtain serial ECGs on patients using a high dosage of catecholamines, regardless of age. When ECG abnormalities are found, serial serum enzyme measurements need be obtained along with clinical assessment of possible myocardial disease. In addition, patients should be warned that failure to respond to the usual dosage of isoproterenol indicates a worsening of their condition, which requires prompt contact with their physician.

ACKNOWLEDGMENT: We wish to thank doctors Karlman Wasserman and Irwin Ziment for reviewing the manuscript.

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Intermittent Functional Closure of Patent Ductus Arteriosus in a Ten-Month-Old Infant; Hemodynamic Documentation*

Ira W. DuBrow, M.D., Elizabeth Fisher, M.D., Alois Hastreiter, M.D.

We describe a ten-month-old patient in whom intermittent patency and closure of the ductus arteriosus was observed clinically, and in whom this observation was uniquely demonstrated at the time of cardiac catheterization. The hemodynamics of this patient and the physiology of ductal closure are discussed.

Delayed closure of the ductus arteriosus is known to occur during early infancy, particularly in those born prematurely. Although documented in the literature, closure after infancy is rare. Intermittent closure is a well-known phenomenon to those who care for critically ill newborns. This communication describes a ten-month-old infant in whom episodes of spontaneous closure of a large patent ductus arteriosus were observed, and in whom this could be uniquely documented at the time of cardiac catheterization.

Case Report

The patient is a 7.7 kg, ten-month-old black boy who was the 1 kg product of a 19-year-old primigravida whose pregnancy was terminated at seven months by spontaneous labor and delivery. The baby developed respiratory distress syndrome at 12 hours and was treated with oxygen, alkali and intravenous fluids. A murmur was first heard at four days of age. Signs of congestive heart failure never developed. He recovered and upon discharge at two months, no murmur was detected.

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The infant was presented at the University of Illinois at age four months with bronchiolitis. After the acute phase, a soft continuous murmur was observed at the left infraclavicular area. No apical rumble was heard and the pulses were not bounding. Follow-up examination, two weeks later, demonstrated findings compatible with a large shunt, as evidenced by a hyperdynamic precordium, bounding pulses, a loud apical S₃, a diastolic rumble, and a loud continuous murmur at the upper left sternal border. Chest x-ray examination disclosed cardiomegaly and increased pulmonary vascularity. The ECG demonstrated left ventricular hypertrophy. The infant was seen on several occasions because of lower respiratory tract infections. The cardiovascular findings alternated from those of patent ductus arteriosus with a large left-to-right shunt to entirely normal. He was scheduled for cardiac catheterization at seven months; however, the procedure was cancelled due to a silent left lower lobe infiltrate. No murmur was heard at this time and the arterial pulses were normal. He was studied at ten months of age.

**Cardiac Catheterization**

The catheterization findings are summarized in Table 1. Early in the procedure the aortic, pulmonary artery, and left ventricular end-diastolic pressures were normal (Fig 1a and 1c). There was no step-up in O₂ saturation from superior vena cava to left pulmonary artery. Auscultation at that time revealed no murmur. Ascending aorta acid-base status was normal and the Pₒ₂ was (93 percent saturation) 66 mm. Upon administering oxygen by hood, the Pₒ₂ rose to 150 mm. Later in the procedure wide aortic pulse pressure and elevated left ventricular end-diastolic pressure were noted (Fig 1b and 1d). In addition, oximetry demonstrated a 22

**FIGURE 1a (left).** Ascending aorta and left pulmonary artery. Note aortic pulse pressure is 30 mm. The shunt was not demonstrable at this time. **FIGURE 1b (right).** The aortic pulse pressure is wide, 60 mm. The shunt was demonstrated at this time.

**FIGURE 1c (left).** Left ventricle and right ventricle when the shunt was not demonstrated. End-diastolic pressures are normal. **FIGURE 1d (right).** Left ventricle and pulmonary capillary wedge when the shunt was demonstrated. Note marked increase in left ventricular end-diastolic pressure.
Figure 2. Angiogram with contrast material injected into the proximal descending aorta (Desc Ao). The long fusiform patent ductus arteriosus (PDA) and dilated main and left pulmonary arteries (MPA, LPA) are visualized.

percent step-up from right ventricle to left pulmonary artery. Auscultation disclosed a loud continuous murmur at the upper left sternal border and a loud apical S2.

Aortogram demonstrated a long fusiform ductus that narrowed at its junctions with main pulmonary artery and aorta (Fig 2). Main pulmonary artery injection showed dilated pulmonary arteries, negative blanching at the superior aspect of main pulmonary, dilatation of left atrium and left ventricle and reopacification of main pulmonary artery from aorta. These features were indicative of a large shunt. At surgery, a long tapering patent ductus arteriosus was ligated and divided.

Table 1—Cardiac Catheterization Data

<table>
<thead>
<tr>
<th></th>
<th>Ductus Closed</th>
<th>Ductus Open</th>
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</thead>
<tbody>
<tr>
<td></td>
<td>O2 Satur</td>
<td>%</td>
</tr>
<tr>
<td>SVC</td>
<td>59</td>
<td>a = 8, v = 2.3, m = 0</td>
</tr>
<tr>
<td>RA</td>
<td>60</td>
<td>—</td>
</tr>
<tr>
<td>RV</td>
<td>59</td>
<td>30/4</td>
</tr>
<tr>
<td>MPA</td>
<td>—</td>
<td>30/10 m = 18</td>
</tr>
<tr>
<td>LPA</td>
<td>59</td>
<td>26/8 m = 15</td>
</tr>
<tr>
<td>PCW</td>
<td>—</td>
<td>11</td>
</tr>
<tr>
<td>LV</td>
<td>—</td>
<td>110/5</td>
</tr>
<tr>
<td>Ao</td>
<td>93</td>
<td>109/76 m = 87</td>
</tr>
<tr>
<td>Qp/Qs</td>
<td>—</td>
<td>1:1</td>
</tr>
<tr>
<td>*Rp</td>
<td>2.86 μ/m²</td>
<td>—</td>
</tr>
<tr>
<td>Rs</td>
<td>25.6 μ/m²</td>
<td>—</td>
</tr>
</tbody>
</table>

*LVEDP assumed to equal LA m
SVC = superior vena cava; RA = right atrium; RV = right ventricle; MPA = main pulmonary artery; LPA = left pulmonary artery; PCW = pulmonary capillary wedge; LV = left ventricle; Ao = aorta; Qp/Qs = pulmonary-systemic low ratio; Rp = pulmonary vascular resistance; Rs = systemic vascular resistance; LVEDP = left ventricular end-diastolic pressure; LA = left atrium

Comment

The clinical features of patent ductus arteriosus are well known, but many factors governing shunting and closure have been elucidated within the last 20 years. The monograph by Cassels reviews current knowledge on this topic. PO2, acid-base status and responsiveness of the precapillary pulmonary arterioles are important variables regulating pulmonary vascular resistance and therefore shunting. PO2, catecholamines, bradykinin and acetylcholine have been shown to affect ductal constriction. Normal neonatal circulatory changes lead to functional closure and shortly thereafter, anatomic sealing. However, spontaneous closure has been documented clinically later in infancy and rarely in childhood and adulthood. About 670 patient years of clinical observation indicate a spontaneous closure rate of 0.6 percent per annum.

The patient presented was born prematurely and had respiratory distress syndrome, giving him a 15 percent chance of ductus remaining patent as opposed to 0.04 percent in the sea level normal population and 0.72 percent in the high altitude population. PO2 and acid-base status did not seem to be a factor in his hemodynamics. The low PO2 observed when the ductus was functionally closed may be explained by intrapulmonary right-to-left shunting as well as a lower percentage of pulmonary perfusion as a result of the previous pulmonary infections. Upon offering an oxygen-enriched atmosphere the PO2 rose to only 150 mm, thereby lending credence to this explanation. In addition, the pulmonary resistance was normal at the time when the duct was functionally closed. By age ten months, one would expect autonomic control of the cardiovascular system to be quite well developed. One might postulate the following: 1) that position or kinking played a role in varying the amount of shunt through this long fusiform structure that was narrow at its junction with the main pulmonary artery; or 2) that the narrow portion of the ductus adjacent to the main pulmonary artery was capable of constriction mediated by humoral factors.

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References


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Myocardial Coronary Hemangiomatous Tumors in Children*

Rabi Sulayman, M.D.,** and Donald E. Cassels, M.D., F.C.C.P.†

Persistent and severe ST segment and T wave changes associated with an apical diastolic murmur were observed in an asymptomatic five-year-old girl for ten years. Recent development of exertional dyspnea and chest pain prompted a detailed cardiovascular re-evaluation. The symptomatology and the electrocardiographic changes are attributed to an angiographic tumor involving the anterolateral myocardium demonstrated by selective coronary arteriography. The literature regarding primary cardiac vascular tumors was reviewed and the clinical and therapeutic implications were discussed.

Primary cardiac tumors are exceedingly rare particularly in infancy and childhood. The commonest of these primary tumors is rhabdomyoma, and the most un-

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common are primary hemangiomatous lesions.1-3
Anatomically, hemangiomatous tumors differ from a coronary arteriovenous cardiac fistula where one or more coronary arteries are involved, and hemangiomas involving the heart valves or atrial septum are now regarded as cysts of the heart valves or varicosities in the region of the foramen ovale. The greatest number of hemangiomas exclusive of cysts and varicosities have occurred among adults, have been of the cavernous variety, and have been for the most part small, subendocardial in location, and with no infiltrative features.

Uskoff6 in 1893 was the first to describe a case of hemangioi of the heart that involved the left atrium. There are few reports appearing in the literature describing various types of primary cardiac vascular tumors.7 Most of these are pericardial hemangiois and angiosarcomas, and most of the patients reported have been adults who presented usually with cardiomegaly, congestive heart failure, and pericardial effusion.

Recently we studied a 15-year-old girl who has been followed since the age of four years because of T wave and ST segment changes, but had shown no symptoms. Selective coronary artery angiograms revealed a hemangiomatous tumor involving the left coronary artery and the anterolateral myocardium. A brief summary of the history and findings is given.

CASE REPORT

The patient is a 15-year-old girl who was seen in the cardiac clinic first in 1963 at the age of 4½ years. At that time electrocardiographic abnormalities compatible with a myocardial disease were first noted (Fig 1).

She has had a completely normal course of development, was totally asymptomatic, and her physical examination was unremarkable except for a high pitched diastolic murmur

![ECG](image)

**Figure 1.** Patient’s ECG (1963) showing ST segment and T wave changes. All the precordial leads are half standard.

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