plunger of the syringe will keep the balloon inflated (Fig 2).

When persistent intrabronchial bleeding occurs, the Fogarty catheter is passed through the T tube into the inner channel of the bronchofiberscope. After visualizing the bleeding site, the balloon is inflated so that the bleeding area is occluded. The area around the bleeding site is then lavaged and aspirated to determine the presence of further bleeding. The balloon should tamponade the bleeding area for approximately 3 to 5 minutes. The inflated balloon fits snugly in the main bronchi (Fig 3), or may be directed into an upper lobe bronchus (Fig 4).

The Fogarty catheter may be transferred to other locations in the tracheobronchial tree by moving the bronchofiberscope upward or downward. The occluding balloon is very durable and seldom perforates; but if the balloon should break, only 3 ml of sterile saline solution would escape into the bronchus and the fragments would adhere tightly to the catheter.

With severe, critical bleeding, the Fogarty catheter balloon is used as a blocker until surgical intervention is available. After the bleeding site is localized, the balloon is inflated to tamponade the area. The proximal end of the catheter is clamped with a hemostat and the hub is cut off. A straight pin plug inserted into the catheter will maintain the pressure in the inflated balloon when the hemostat is removed. The bronchofiberscope may be removed by slipping it over the Fogarty catheter, leaving the inflated balloon in place. The catheter may then be removed at surgery, or at an appropriate time, by removing the pin plug which permits the balloon to deflate.

Other indications for the use of this blocker are the control of bleeding from a biopsy site and the control of severe hemoptysis in patients who are inoperable due to severe bilateral lung disease or terminal malignancy. The above technique may also be applied to persistent bleeding when rigid bronchoscopy is performed. This technique has been used in a limited number of cases but appears to be of sufficient importance to be reported at this time.

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REFERENCES


Right Juxtaposition of the Atrial Appendages*

Rajamma Mathew, M.B.B.S.; Robert Replagate, M.D.; Otto G. Thilenius, M.D.; and René A. Arcilla, M.D.

We present an infant with right-sided juxtaposition of atrial appendages who had open heart surgery for ventricular septal defect and patent ductus arteriosus. Of 12 cases thus far reported, ventricular d-loop was observed in nine, and normal position of great vessels in four. Contrary to previous views, this condition may not be accompanied by severe conotruncal anomalies.

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RIGHT JUXTAPOSITION OF ATRIAL APPENDAGES 483
Juxtaposition of the atrial appendages is a rare anomaly characterized by both appendages adjoining each other and lying either to the left of, or the right of, the great vessels. It is generally associated with other major cardiac defects. Juxtaposition to the left is far more common than to the right, their comparative incidence being approximately 6:1. In 1968, Melhuish and Van Praagh reviewed 42 cases of which 3 were of the right juxtaposition type. To our knowledge, 11 cases of right juxtaposition have thus far been reported (Table 1). This report describes an additional case accompanied by a large ventricular septal defect and a patent ductus arteriosus (no. 12 in Table 1). Transposition of the great vessels, a usual accompanying anomaly, was not present.

Table 1—Summary of Cardiac Findings in Right Juxtaposition of Atrial Appendages (12 cases)

<table>
<thead>
<tr>
<th>Case, Age, Sex, Reference</th>
<th>Ventricular Loop</th>
<th>VSD</th>
<th>ASD</th>
<th>Relations Great Vessels</th>
<th>Other Cardiac Anomalies</th>
<th>Extracardiac Anomalies</th>
</tr>
</thead>
<tbody>
<tr>
<td>(1) 20y, M</td>
<td>d-loop</td>
<td>present</td>
<td>NK</td>
<td>d-TGA</td>
<td>Infundibular stenosis; hypoplasia of aorta</td>
<td>NK</td>
</tr>
<tr>
<td>(2) 21y, M</td>
<td>d-loop</td>
<td>present</td>
<td>absent</td>
<td>Aorta anterior and to right of PA</td>
<td>Double outlet RV; coarctation of aorta; PDA; atypical coronary artery distribution</td>
<td>Kyphoscoliosis</td>
</tr>
<tr>
<td>(3) 3 wks, F</td>
<td>d-loop</td>
<td>present</td>
<td>present</td>
<td>PA</td>
<td>Aorta anterior and to right of PA</td>
<td>Tricuspid stenosis; double outlet LV; preductal coarctation; absent IVC; absent caval drainage to RA</td>
</tr>
<tr>
<td>(4) 4 wks, M</td>
<td>l-loop</td>
<td>present</td>
<td>present</td>
<td>l-TGA</td>
<td>Bicuspid pulmonic valve</td>
<td>Double outlet RV; mitral atresia with rudimentary LV; atypical coronary artery distribution</td>
</tr>
<tr>
<td>(5) 14y, M</td>
<td>d-loop</td>
<td>NK</td>
<td>large</td>
<td>d-TGA</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>(6) Stillborn, M</td>
<td>d-loop</td>
<td>absent</td>
<td>present</td>
<td>normal</td>
<td>—</td>
<td>Multiple congenital anomalies</td>
</tr>
<tr>
<td>(7) NK</td>
<td>d-loop</td>
<td>present</td>
<td>NK</td>
<td>normal</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>(8) 2 hrs, M</td>
<td>l-loop</td>
<td>present</td>
<td>present</td>
<td>l-TGA</td>
<td>—</td>
<td>Hypoplastic LA, left-sided tricuspid valve and left-sided RV; infundibular and valvar pulmonic stenosis</td>
</tr>
<tr>
<td>(9) 3d, F</td>
<td>l-loop</td>
<td>present</td>
<td>present</td>
<td>l-TGA</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>(10) Stillborn, F</td>
<td>d-loop</td>
<td>absent</td>
<td>present</td>
<td>normal</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>(11) 1d, F</td>
<td>d-loop</td>
<td>malrotated</td>
<td>present</td>
<td>present</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>(12) Present case, M</td>
<td>d-loop</td>
<td>present</td>
<td>absent</td>
<td>normal</td>
<td>—</td>
<td>—</td>
</tr>
</tbody>
</table>

*VSD = ventricular septal defect; ASD = atrial septal defect; NK = not known; d-TGA = d-transposition of great arteries; l-TGA = l-transposition of great arteries; RV = right ventricle; LV = left ventricle; RA = right atrium; LA = left atrium; PDA = patent ductus arteriosus; PA = pulmonary artery; IVC = inferior vena cava.

#age at death

CASE REPORT

A black boy, product of a full-term, uncomplicated pregnancy and delivery, weighed 2.98 kg at birth. At age 8 days, the infant was hospitalized for diarrhea and vomiting, and was discharged 13 days later. At age five months, he was admitted to the cardiology service for congestive heart failure. Physical examination revealed weight 5.5 kg, normal peripheral arterial pulses, no cyanosis, moderate tachypnea and hepatomegaly. Examination of the heart revealed increased right and left ventricular impulses; the PMI was at the left fifth interspace outside the mid-clavicular line. The second sound was split and had an accentuated pulmonic component. A grade 2/6 medium pitch ejection systolic murmur was present at the mid precordial area; in addition, a similar intensity short diastolic rumble was present at the apex. The electrocardiogram and vectorcardiogram revealed
sinus rhythm, frontal QRS axis of +120°, and right ventricular hypertrophy. The chest roentgenograms revealed moderate cardiomegaly, right atrial and combined ventricular enlargement, and borderline increase in pulmonary vascular markings. The base of the heart appeared unusually wide.

He was treated with digitalis, diuretics and oxygen. Cardiac catheterization at age five and a half months revealed ventricular septal defect, pulmonary hypertension and pulmonary systemic flow ratio of 2:1. The intracardiac pressures (in mm Hg) were: right atrium—mean 2; right ventricle—90/6; pulmonary artery—90/45 (68); femoral artery—90/45 (68). Oxygen saturation data revealed (in percent): SVC—64; right atrium—64; right ventricle—83; pulmonary artery—79; left SVC—66; and femoral artery—99. Both superior and inferior vena cava entered the right atrium in a normal manner; in addition, a left superior cava opened into the coronary sinus. Biplane cineangiograms revealed a large ventricular septal defect with left-to-right shunting, juxtaposition of the atrial appendages to the right of the great vessels, and left aortic arch. Both ventricles were dilated and were normally situated; the great vessels arose normally from their respective ventricles. The left atrium was dilated, and no left-to-right atrial shunting was noted. Patent ductus arteriosus was not ruled out.

The baby was observed in the outpatient clinic, but was hospitalized two more times for heart failure and/or pneumonia. Open-heart surgery, using cardiopulmonary bypass, was performed at age 13 months. The juxtaposed atrial appendages were noted to the right of the great vessels (Fig 1A). The aorta and pulmonary artery assumed normal positional relations; the coronary artery distribution appeared normal. The left superior vena cava which drained into the coronary sinus also continued into the (right) superior vena cava via a fair-sized left innominate vein. A large patent ductus arteriosus was ligated; the atrial septum was intact. Through right ventriculotomy, a large solitary infracristal type of ventricular septal defect, measuring 2.2 cm in diameter and situated in the usual paramembranous portion of the ventricular septum, was found and closed with a patch (Fig 1B).

The postoperative course was uneventful. The boy has remained asymptomatic, and has gained 2.2 kg in four months following surgery.

**DISCUSSION**

Although juxtaposition of the atrial appendages is of itself benign, it usually connotes severe congenital heart disease with cyanosis. Melhuish and Van Praagh have emphasized the high incidence of transposition of the great arteries (92 percent), with noninversion of the ventricles in the case of left juxtaposition (95 percent) and inversion of the ventricles in the case of right juxtaposition (100 percent). Other common anomalies include: ventricular septal defect (88 percent); atrial septal defect (78 percent); bilateral conus (77 percent); small or absent right ventricle (71 percent); and pulmonary or aortic outflow obstruction (52 percent, 38 percent).

Because of the frequent coexistence of conotruncal anomalies, it has been suggested that this condition may constitute a characteristic syndrome somewhat comparable to, although not as clear cut as, the asplenia syndrome. Its occurrence as an isolated anomaly, ie, in the absence of any other intracardiac defect, has not observed.

![Figure 1](http://journal.publications.chestnet.org/pdfaccess.ashx?url=/data/journals/chest/20964/ on 06/26/2017)

**Figure 1.** Heart at the time of surgery. A. Frontal view with the chest open. Note both atrial appendages lying to the right of the aorta. Ao-aorta; PA-main pulmonary artery; laa-left atrial appendage; raa-right atrial appendage. B. Right ventricle opened after total circulatory arrest, demonstrating a typical infracristal septal defect, vsd-ventricular septal defect; cs-crista supraventricularis.

![Figure 2](http://journal.publications.chestnet.org/pdfaccess.ashx?url=/data/journals/chest/20964/ on 06/26/2017)

**Figure 2.** Schematic diagram of embryogenesis in juxtaposition of atrial appendages (Modified after Dixon).
been described, with the possible exception of the still-born infants reported by Becker and Becker and by Dusek with right-sided juxtaposition. In both instances, multiple extracardiac anomalies were present.

Wenner proposed that left-sided juxtaposition is due to underdevelopment of torsion of the primitive cardiac tube. Dixon later suggested that right-sided juxtaposition is due to overdevelopment of the torsion process of the primitive tube. Thus, left juxtaposition has been assumed to be due to undertorsion, and right juxtaposition to overtorsion of the primitive cardiac tube. However, this generalization appears to be applicable only for the heart with the usual ventricular d-loop (non-inverted ventricles). In the presence of ventricular l-loop (inverted ventricles), one may have to postulate the converse mechanism, namely, undertorsion of the primitive cardiac tube for right juxtaposition, and over-torsion for left juxtaposition (Fig 2). The latter could explain the rare occurrence of left juxtaposition in ventricular l-loop as reported by Lieberson.

Unlike the very high incidence of transposition of the great arteries in left juxtaposition, only 8 of 12 cases with right juxtaposition have shown this abnormality of the great vessels. Our case, and those of others, demonstrated that this abnormality of the atrial appendages may not necessarily be accompanied by complex intracardiac anomalies.

**REFERENCES**


**Severe Hemolysis with a Fabric-Worn Cloth-Covered Aortic Valve Prosthesis**

*Claus A. Pierach, M.D., Hans R. Baur, M.D., and Joseph C. Kiser, M.D.*

A patient developed severe hemolytic anemia one year after insertion of a cloth-covered aortic valve prosthesis (Starr-Edwards No. 2320). The cloth over the three struts was disrupted but showed coverage with mostly organized collagen. Hemolysis stopped after replacement with a porcine heterograft. Fabric wear seems to augment the hemolysis in patients with cloth-covered artificial valves.

Hemolysis after prosthetic heart valve insertion is not rare. If clinically significant, a malfunction of the valve should be suspected and if possible corrected by operation. We describe a patient in whom disruption of the cloth over the three struts of an aortic prosthesis seemed to have augmented the hemolysis, necessitating its replacement.

**CASE REPORT**

A 20-year-old man was admitted to Northwestern Hospital on December 7, 1973, complaining of shortness of breath and dizziness on exertion for the past two months. The patient had undergone commissurotomy for a stenotic bicuspid aortic valve in 1961. On January 16, 1973, a Starr-Edwards cloth-covered aortic prosthesis size 9 (model 2320) was inserted because of evidence of aortic stenosis. The patient subsequently was placed on oral anticoagulants and later on iron substitution.

Physical examination on admission showed a pale, slightly icteric young man in no acute distress, with blood pressure of 140/70 mm Hg. Examination of the chest revealed a well healed mid-sternal scar. An apical and a suprasternal thrill and a loud systolic ejection murmur were heard. The clicking of the prosthetic ball was not appreciably metallic. No diastolic murmur was audible. The remainder of the physical examination was noncontributory.

Complete blood count revealed: hemoglobin level of 8.9 gm/100 ml, hematocrit 20.3 percent, red cell count 1.99 million/cu mm with 33.7 percent reticulocytes and a white blood cell count of 4,800/cu mm, with a normal differential count. Numerous schistocytes were seen on the blood smear. His total bilirubin measured 2.3 mg/100 ml, his lactate dehydrogenase (LDH) 5500 mU/ml (N<200 mU/ml). He was haptoglobinemic. His otherwise normal urinalysis showed an iron content of 13 mg/24 hr (N<0.15 mg/24 hr).

On January 17, 1974, he was operated upon again. At operation, the aortic prosthetic suture line was found to be intact. The valve was replaced with a 23 mm porcine heterograft (Hancock). The patient was discharged eight days later on no medication. On February 15, 1974, he had a

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