tained. Our arterial blood gas and pH determinations are performed in an IL 313 blood gas analyzer. Very strict calibration techniques are followed, and the values have been accurate; therefore I must assume that the venous CO₂ values were, in general, falsely low. Even accepting the reported venous CO₂ values as true, it can be observed from the Table that, in general, the venous CO₂ and the estimated arterial carbon dioxide tension (Pco₂) increased concurrently.

In summary, compensatory hypoventilation and hypercapnia may be the consequence of acute or chronic metabolic alkalosis in patients with azotemia. Cerebrospinal fluid alkalosis appears to be the most likely explanation for the compensatory hypoventilation.

REFERENCES

Right Aortic Arch with Coarctation of the Aorta *

Herman L. Price, LTC, MC, and Richard M. Schieken, Maj, MC

The first case of right aortic arch with mirror-image type branching and coarctation of the aorta is presented. The patient has Turner's syndrome. Severe valvular aortic stenosis is also present. Genetic and embryologic explanations for the unique association of these anomalies are suggested.

Rare cases of right aortic arch with coarctation of the aorta have been reported,1-3 all of which were associated with an aberrant left subclavian artery. This type of arch anomaly ordinarily is not associated with congenital intracardiac lesions. We are reporting a patient with mirror-image right aortic arch, coarctation of the aorta, valvular aortic stenosis, and Turner's syndrome. Mirror-image right aortic arch, unlike right aortic arch with aberrant left subclavian artery, is frequently associated with intracardiac anomalies. However, this is the first reported case of mirror-image right aortic arch in association with coarctation of the aorta.

CASE REPORT

This 13-year-old white girl was the product of a full term uncomplicated pregnancy and was born by breech delivery. Her birth weight was 8 lbs 11 ozs. A heart murmur was noted first at four months of age. A diagnosis of Turner's syndrome was confirmed by chromosomal analysis. During the first six years of life, observers noted a gradual decline in her peripheral pulses. However, she had no cardiovascular symptoms.

When first seen in our clinic she was 6½ years of age and presented as a slightly obese white child with the phenotypic features of Turner's syndrome. Her radial pulses were questionably decreased and the femoral pulses were definitely decreased. Blood pressures in the arms were 100 to 200/90 to 100 mm Hg; but no blood pressure measurement could be obtained in the lower extremities. The heart size was normal. There were no lifts, but there was a systolic thrill over the base of the heart which radiated to the suprasternal notch and both carotid arteries. The second heart sound was narrowly split and of normal intensity. A grade 4/6 harsh sys-
tolic ejection murmur was heard best at the upper right sternal border and radiated widely. There was an ejection click at the apex. No hepatosplenomegaly, cyanosis, clubbing or edema were observed.

Her electrocardiogram suggested left ventricular hypertrophy as manifested by prominent posterior forces in the mild precordial leads and ST-T wave changes. The PA and lateral chest x-ray films demonstrated right aortic arch, normal heart size and normal pulmonary vascularity (Fig 1,2).

At eight years of age right brachial artery arteriotomy and transthoracic ventricular puncture were accomplished. With simultaneous left ventricular and right subclavian artery pressures being recorded, the total peak systolic pressure gradient was about 50 mm Hg (Table 1). A cineortogmm was obtained via the right subclavian artery. Because the right subclavian artery was the last vessel originating from the arch and the catheter could not be passed beyond the proximal subclavian artery, visualization of the aortic root was not accomplished. A subsequent right heart catheterization with a right atrial forward biplane angiogram demonstrated a normal right heart study; and the angiogram confirmed a mirror-image type right aortic arch, dilatation of the ascending aorta, a left innominate artery, a right descending aorta, and coarctation of the aorta near the origin of the right subclavian artery (Fig 3,4).

The patient underwent resection of the coarctation of the aorta in November 1965 and the angiographic findings were confirmed. The coarcted segment involving the right subclavian artery was essentially a diaphragm with a functional lumen of 6 mm in diameter. A 1.5 cm segment was resected and an end-to-end anastomosis was effected. Her early postoperative course was complicated by mild systolic hypertension. Her discharge evaluation revealed blood pressure to be unobtainable in the right arm and 140/70 mm Hg in the left arm and both legs. She had a systolic thrill and a grade 4/6 systolic ejection murmur at the base of the heart.

Because of easy fatigability and a persistently abnormal electrocardiogram, left heart catheterization via left axillary artery was performed in September 1971 and demonstrated left ventricular pressure of 230/8 and aortic pressure of 112/75 mm Hg which gave a peak systolic aortic valve gradient of 118 mm Hg (Table 1). Aortic valve commissurotomy was accomplished in December 1971 and was followed by an uncomplicated postoperative course. However, she had no obtainable blood pressure or pulse in either arm.

Except for more deeply inverted precordial T waves, her electrocardiogram was unchanged from her preoperative electrocardiogram and continued to suggest left ventricular hypertrophy.

Postoperative cardiac catheterization in November 1972

Table 1—Summary of Cardiac Catheterization Data (mm Hg)

<table>
<thead>
<tr>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Subclavian artery</td>
<td>110-125/90-95</td>
<td>115/90,95</td>
<td>100/80</td>
</tr>
<tr>
<td>Aorta</td>
<td>105/80,90</td>
<td>100/80</td>
<td></td>
</tr>
<tr>
<td>LV body</td>
<td>160-190/0-10</td>
<td>250/0-30</td>
<td>170/7</td>
</tr>
<tr>
<td>LV subvalve</td>
<td>100/6</td>
<td></td>
<td></td>
</tr>
<tr>
<td>PAW</td>
<td>a=8, v=11.8</td>
<td></td>
<td></td>
</tr>
<tr>
<td>MPA</td>
<td>24/10,15</td>
<td></td>
<td></td>
</tr>
<tr>
<td>RV</td>
<td>24/0-2</td>
<td></td>
<td></td>
</tr>
<tr>
<td>RA</td>
<td>a=4, v=3,2</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

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is asymptomatic. Repeat cardiac catheterization is planned for one year.

**DISCUSSION**

This is the first case report of a patient with both coarctation of the aorta and mirror-image type right aortic arch. Coarctation of the aorta is known to be associated rarely with right aortic arch. To date, all of these patients had a right aortic arch with an aberrant left subclavian artery.

It is of interest to note that this unique association occurred in a child with Turner’s syndrome. Turner’s syndrome is a chromosomal anomaly (XO) frequently associated rarely with right aortic arch. To date, all of them had a right aortic arch with an aberrant left subclavian artery.

Despite the rare occurrence of right aortic arch with coarctation of the aorta, approximately 98 percent of the patients with right aortic arch of the mirror-image type have congenital intracardiac anomalies. The lesions most frequently observed are tetralogy of Fallot and persistent truncus arteriosus.

Embryologic studies have demonstrated that during the second month of fetal life, the heart grows in a caudal direction normally, the left fourth arch assumes the major arterial flow. The anomalous persistent right aortic arch results from the abnormal regression of the truncus arteriosus.

Embryologic studies have demonstrated that during the second month of fetal life, the heart grows in a caudal direction normally, the left fourth arch assumes the major arterial flow. The anomalous persistent right aortic arch results from the abnormal regression of the left eighth dorsal segment. The innominate artery then forms to the left.

Recently, a hypothesis to explain the development of a coarctation of the aorta has been proposed. Rudolph and co-workers have suggested that a slight reorientation of the angle at which the fetal ductus arteriosus meets the aorta with a left aortic arch could account for the development of a localized aortic constriction. They observed that with a right aortic arch and a normal pulmonary artery that the left ductus arteriosus is longer than usual. Therefore, the angle at which the left ductus arteriosus meets the aortic with a mirror-image type right aortic arch might be less conducive to the formation of an aortic coarctation. Despite the fact that the fetal flow patterns which occurred in this child appear to prevent the formation of an aortic coarctation, the genetic predisposition of Turner’s syndrome towards the formation of coarctation prevailed.

**REFERENCES**


**Possible False-Positive Diagnosis of Pericardial Effusion by Echocardiography in Presence of Large Left Atrium**

*Robert A. Ratshin, M.D.,* *McKamy Smith, M.D., F.C.C.P.* *and William P. Hood, Jr., M.D.*

Initial echocardiographic examination in a young woman with cardiomegaly suggested the presence of pericardial effusion. Echocardiographic scanning revealed continuity of the space defined posterior to the posterior left ventricular wall with an enlarged left atrium. Subsequent cineangiographic studies confirmed left atrial enlargement and the absence of pericardial effusion. Scanning, which can be accomplished in most, but not in all patients, is an important component technique of the echocardiographic examination for pericardial fluid. Left atrial enlargement represents a potentially common cause of false-positive echocardiographic diagnosis of pericardial effusion, of which the examiner must be aware.

Diagnostic ultrasound has provided a simple, noninvasive adjunct in the evaluation of the patient in whom pericardial effusion is suspected. The ultrasound examination can be performed at the bedside, usually in a matter of minutes, and the validity of the technique for the diagnosis of pericardial effusion has been confirmed in a number of laboratories. Although the accuracy and reliability of the examination have been established and, although the limitations of the procedure are frequently technical ones which can be overcome by experience, instances of false-positive diagnosis of pericardial effusion, albeit infrequent, must be identified in order that any limitations of the technique may be defined. In the present report, echocardiograms which could be interpreted as positive for pericardial fluid were obtained from a patient in whom angiographic studies demonstrated a significantly enlarged left atrium without evidence of pericardial effusion.

**METHODOLOGY AND CASE REPORT**

A 51-year-old Negro woman was admitted to the University of Alabama Hospital on Sept. 9, 1971 for evaluation of valvular heart disease. The patient was first told of a cardiac murmur at age 14. Although there was no history of rheumatic fever, the patient subsequently experienced an episode of fever and joint pain at 16 years of age, which was confirmed as acute rheumatic fever. At 37 years of age the patient was

*From the Division of Cardiology, Department of Medicine, University of Alabama Medical Center, University Station, Birmingham.

**Instructor in Medicine.

Associate Professor of Medicine.

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Reprint requests: Dr. Hood, University Station, Birmingham, Alabama 35294

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