ECG in a Cyanotic 22-Year-Old Woman Who Had a Cardiac Operation in Early Childhood

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The patient’s chief complaint was exertional dyspnea. On physical examination, she had mild symmetrical cyanosis and clubbing and no significant murmur. On chest radiograph, there was striking enlargement of the pulmonary trunk and central pulmonary arteries with peripheral attenuation, marked cardiomegaly with biatrial and left ventricular enlargement (Figure 1). An ECG was recorded (Figure 2).

Figure 1: Posteroanterior (left) and left lateral (right) chest radiographs taken on admission.

What is your diagnosis?
Figure 2: Electrocardiogram recorded soon after admission.

**DIAGNOSIS:** Normal sinus rhythm; biatrial enlargement; right axis deviation of the QRS complex; biventricular enlargement with repolarization abnormality.

The upright P wave before each QRS in leads I, II, and III indicates sinus rhythm. The large (≥ 0.1 mV and ≥ 0.04s) negative terminal portion of the P wave in lead V1 is a sign of left atrial enlargement, and the P wave amplitude ≥ 0.25 mV in lead II suggests right atrial enlargement. The QRS is negative in lead I and positive in lead aVF, indicating right axis deviation, and a QRS more negative in lead I than in aVR suggests that the QRS axis is closer to +120° than to +90°. Several criteria indicate left ventricular hypertrophy: RV ≥ RV; RV > 2.0 mV; and SV + RaVL > 2.0 mV. Right axis deviation in the presence of left ventricular hypertrophy indicates coexisting right ventricular hypertrophy, which diagnosis is supported by the isoelectric QRS in lead V1. Repolarization changes in the lateral precordial leads are signs of left ventricular hypertrophy, and those in the inferior leads suggest right ventricular hypertrophy.

Congenital malformations that can result in cyanosis, increased pulmonary blood flow, and biventricular enlargement on electrocardiogram include transposition of the great arteries with pulmonary hypertension, single ventricle, truncus arteriosus, ventricular septal defect with moderate pulmonic stenosis, and occasionally pulmonary atresia with an intact ventricular septum and a large patent ductus arteriosus or significant tricuspid regurgitation. In addition, cyanotic conditions that have been palliated with a systemic-to-pulmonary arterial shunt have a cause for both increased pulmonary blood flow and left ventricular hypertrophy.

The current patient’s cardiac malformation, pulmonary atresia, was palliated with a Waterston shunt (side-to-side anastomosis of the ascending aorta to the right pulmonary artery). Unfortunately, the anastomosis was too large, pulmonary arterial pressure was identical with systemic arterial pressure, and she has developed Eisenmenger physiology. The palliative Potts shunt (side-to-side anastomosis of the descending aorta to the ipsilateral pulmonary artery) also can result in too large an anastomosis and Eisenmenger’s reaction. In contrast, with a Blalock-Taussig shunt (end-to-side anastomosis of a subclavian artery to the ipsilateral pulmonary artery) the size of the anastomosis and left-to-right shunt is limited by the size of the subclavian artery, and Eisenmenger’s reaction virtually never occurs.

**REFERENCES**


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