Atrial Dissociation: An Electrophysiologic Finding in a Patient With Transposition of the Great Arteries

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Complete interatrial block with two independent atrial rhythms is demonstrated by intracardiac electrograms in a patient with transposition of the great arteries who had undergone a Mustard operation. The atrial conduction abnormalities produced by the Mustard procedure are related to damaged anatomic interatrial connections and are similar to conduction abnormalities produced in experimental models of interatrial block.

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Electrical dissociation of the atria, first described by Schrumpf (1) in 1920, remains a controversial electrocardiographic finding. Most published examples (2–4) of this phenomenon consist of sinus rhythm and a slower, independent ectopic rhythm that does not conduct to the ventricle. However, White (5) questioned the existence of atrial dissociation in humans and Katz (6) pointed out that this finding could be explained by artifacts.

To date, conclusive proof of the existence of complete interatrial block has been lacking, except in patients with cardiac transplantation (7), in whom intracardiac recordings have demonstrated two independent rhythms from the recipient and donor atria. We document another cause of independent atrial rhythms in a patient with transposition of the great arteries who had undergone a Blalock-Hanlon septectomy, followed by a Mustard procedure and subsequent atrial baffling revision. Atrial conduction abnormalities, caused by these operative procedures, are probably related to damaged anatomic interatrial connections and are similar to conduction abnormalities produced in experimental models of interatrial block.

Case Report

A 20 year old woman was admitted to Yale-New Haven Hospital after being resuscitated by paramedics. Her past medical history included transposition of the great arteries, Blalock-Hanlon atrial septectomy during infancy and a Mustard operation at age 6. At age 16 she had a syncopal episode and atrial flutter was documented. After conversion to sinus rhythm, intermittent bradycardia occurred and a ventricular demand pacemaker was inserted. Cardiac catheterization demonstrated a 3:1 left to right interatrial shunt. At reoperation several holes were closed at the junction of the baffie and the atrial wall. The patient was maintained on digoxin and did well until the present admission.

Physical examination was unremarkable. The digoxin level was less than 0.5 ng/ml. The 12 lead electrocardiogram, unchanged from previous tracings, demonstrated sinus tachycardia, right-axis deviation and right ventricular hypertrophy with repolarization changes.

Electrophysiologic study. Electrophysiologic evaluation was performed in the nonsedated, postabsorptive and drug-free state. Sinus rhythm was present with an AH interval of 80 ms and an HV interval of 40 ms. Sinus rhythm was present with an AH interval of 80 ms and an HV interval of 40 ms. Programmed atrial and ventricular stimulation did not induce tachyarrhythmias.

Simultaneous systemic venous atrial and pulmonary venous atrial intracardiac recordings were obtained using a quadripolar electrode catheter at the low right lateral border of the systemic venous atrium and a bipolar electrode catheter within the pulmonary venous atrium. The latter catheter was passed retrograde from the aorta through the morphologic right ventricle to the pulmonary venous atrium. A third catheter was positioned in the apex of the morphologic left ventricle (Fig. 1). Two regular independent atrial rhythms were recorded (Fig. 2), one from the systemic venous atrium and the other from the pulmonary venous atrium. The systemic venous atrial rhythm was faster (cycle length 570 ms)
and conducted 1:1 to the ventricle. The pulmonary venous atrial rhythm (cycle length 650 ms) did not conduct to the ventricle or the other atrium. Placement of the pulmonary venous atrial catheter in a second distant location within the pulmonary venous atrium confirmed the independent rhythm.

Recordings of both atrial rhythms during isoproterenol infusion (Fig. 3) demonstrated a progressive reduction in the cycle length of the systemic venous and pulmonary venous atrial rhythms, with persistence of both the interatrial block and lack of conduction from the pulmonary venous atrium to the ventricle. Incremental pacing of the systemic venous atrium during the baseline state (Fig. 4) and during isoproterenol infusion did not produce depolarization of the other atrium or affect its rhythm. Incremental pulmonary venous pacing at 10 mA demonstrated total lack of electrical penetration to the systemic venous atrium. However, as only a bipolar catheter could be positioned in the pulmonary venous atrium, doc-

Figure 1. Chest radiograph demonstrating quadripolar electrodes at the systemic venous atrium (SVA) and the morphologic left ventricle (LV). A bipolar electrode catheter is located at the pulmonary venous atrium (PVA).

Figure 2. Baseline electrophysiologic recording during spontaneous sinus rhythm. Surface electrocardiographic leads I, aVF and V1 with intracardiac electrograms from the systemic venous atrium (SVA), pulmonary venous atrium (PVA) and morphologic left ventricle (LV) are shown. Two independent atrial rhythms are recorded, one from the systemic venous atrium (Asv) and the other from the pulmonary venous atrium (Apv). T = time lines; V = ventricular electrogram.

Figure 3. During isoproterenol infusion (1 μg/min), electrophysiologic recording demonstrates a reduction in cycle length of both the systemic venous atrial (Asv) and pulmonary venous atrial (Apv) rhythm with persistence of both the interatrial block and lack of conduction from the pulmonary venous atrium to the ventricle. Abbreviations as in Figure 2.

Figure 4. Systemic atrial pacing at a cycle length of 500 ms during the baseline state did not produce depolarization of the pulmonary venous atrium or affect the pulmonary venous atrial rhythm. s and arrow = stimulus artifact; other abbreviations as in Figure 2.
...umentation of pulmonary venous atrial capture was not possible. Ventricular pacing at rates slightly above the atrial rates demonstrated ventriculoatrial block to both atria.

**Discussion**

Interatrial conduction occurs preferentially through the anterior internodal pathway to Bachmann’s bundle (8,9). The anterior internodal pathway originates at the anterior aspect of the sinus node and curves around the superior vena cava and anterior aspect of the right atrium. There it divides into two bundles, Bachmann’s bundle, which continues to the left atrial wall and the second, which continues to the atrioventricular (AV) node. Two other potential pathways between the right and left atria exist. There are inconsistent sparse connections between the septal portions of the internodal tracts of the right atrium to the left atrium. In addition, at the region just above the AV node, the three internodal tracts of the right atrium join with the myocardial fibers from the left side of the atrium (9).

Complete interatrial block has been produced experimentally in animals by direct crush injury to the septum (10,11) or by interfering with the atrial blood supply (12). Isolated interruption of only Bachmann’s bundle does not cause complete interatrial block, but rather interatrial conduction delay (8). In humans, complete interatrial block has been noted in cardiac transplantation (7) when the recipient sinus node remains in its original location and is separated from the donor atrium by a suture line.

**Role of Mustard operation.** One might expect that the Mustard operation would create the conditions under which two independent atrial rhythms could coexist. In this operation (13), the atrial septum is excised and a pericardial baffle is sutured to the atrial wall to redirect the pulmonary venous circulation into the morphologic right ventricle and the systemic venous circulation into the left ventricle. The atrial internodal pathways are commonly either excised or damaged during the atrial septectomy. Isaacson et al. (14) found all three internodal pathways damaged in 12 of 14 patients. Lewis et al. (15) described fibrosis in the region of the anterior and posterior internodal tracts associated with the sutures. Damage to the atrioventricular node has been observed and is thought to be related to its proximity to the fixation of the baffle. In our patient, damage to interatrial connections could also have occurred during the Blalock-Hanlon procedure or revision of the Mustard baffle, or both.

To our knowledge, atrial dissociation has not been previously described in patients with transposition of the great arteries (16) who have undergone a Mustard operation. The anatomic lesions creating atrial dissociation in these patients are remarkably similar to those used experimentally (10) to produce this phenomenon.

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**References**