that he did not develop symptoms of angina or syncope characteristic of IHSS, nor marked pulmonary hypertension in association with the ASD.

It may be speculated that these combined lesions are mutually aggravating since decreased compliance of the left ventricle and the resultant increased resistance to left ventricular filling which are characteristic of IHSS would be expected to increase the left-to-right shunt at the atrial level. In addition, decreased left ventricular filling as a result of such a shunt aggravates the left ventricular outflow tract obstruction during systole.

It is now generally accepted that repair of an ASD with left-to-right shunt can be undertaken in adults with acceptable surgical mortality and amelioration of symptoms.\(^2,3\) Septal myotomy and myectomy have likewise proved beneficial for the patient with IHSS who has developed disabling symptoms.\(^4\)

It may be reasoned that closure of the ASD alone could have been performed in this patient with resultant physiologic and symptomatic improvement. In addition, recent clinical observation suggests that patients with IHSS and predominant dyspnea and fatigue are helped less by operative intervention than those with angina, syncope and presyncope.\(^4\) However, since it was impossible to determine which lesion contributed most to his symptoms, the result of less than total correction was uncertain and reoperation would incur additional risk to the patient. Also, isoproterenol, which is commonly used for low output states during the postoperative period, might have been hazardous if the subvalvular obstruction had not been relieved. For these reasons it was elected to perform both ASD closure and septal myectomy in this patient.

In summary, this is the first known case in which relief of the subvalvular obstruction and closure of the secundum ASD have been successfully accomplished in the same patient.

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**Idiopathic Hypertrophic Subaortic Stenosis and Wolff-Parkinson-White Syndrome**

**Changes of Obstruction in Left Ventricular Outflow Depending on the Type of Ventricular Activation**

Juan Angel, M.D.; Juan J. Armendartiz, M.D.; Herminio Garcia del Castillo, M.D.; Ceferino A. Llamas, M.D.; and Carlos Gaust, M.D.

A case of idiopathic hypertrophic subaortic stenosis (IHSS) associated with Wolff-Parkinson-White (WPW) syndrome is studied. On three occasions preexcitation was suppressed with an intravenous injection of ajmaline. The left ventricular outflow tract obstruction (LVOTO) was analyzed; when WPW syndrome activation disappeared, the obstruction decreased. The ventricular depolarization pathway is noteworthy as a factor in the severity of LVOTO in IHSS.

Some authors\(^1,2\) considered Wolff-Parkinson-White (WPW) syndrome a classic electrocardiographic pattern in idiopathic hypertrophic subaortic stenosis (IHSS), while questioning whether it is due to pre-
excitation or to anomalous conduction in the hypertrophic septum. In these cases Schamroth\(^1\) considers the possibility of suppression of questionable preexcitation an important factor in the diagnosis of WPW syndrome when associated with IHSS.

It is the purpose of this report to analyze the relationship between preexcitation and LVOTO in a patient with WPW and IHSS.

**CASE REPORT**

A 64-year-old man complained of exertional dyspnea and substernal chest pain. Physical examination disclosed a bifid carotid pulse with rapid upstroke, an ejection systolic murmur and a hyperkinetic apical impulse.

The electrocardiogram (ECG) showed a short PR interval of 0.11 sec, with a delta wave upright in L1, AVL and V2-V6, the QRS duration was 0.14 sec. In the vectocardiogram...
(VCG) the duration of the slowed beginning of the QRS loop was 0.08 sec.

One hundred mg of ajmaline was intravenously administered twice on different days for ten minutes each time; ECG, phonocardiogram and carotid pulses (CP) were recorded simultaneously (Fig 1). Complete ECG and VCG were recorded before and after ajmaline injections.

During routine heart catheterization the same dose of ajmaline was repeated while recording simultaneous tracings of left ventricular (LV) and brachial artery (BA) pressures.

**RESULTS**

After each injection of ajmaline preexcitation disappeared; PR interval increased from 0.11 to 0.20 sec, QRS duration decreased from 0.14 to 0.11 sec, and the delta wave disappeared. The VCG showed no delta loop.

After the initial two injections of ajmaline, CP was no longer bifid with rapid upstroke, and the systolic murmur decreased when preexcitation disappeared (Fig 1).

When the third dose of ajmaline was given, with catheterization control, the pressures of the few beats before the disappearance of preexcitation remained similar to those before the injection. Simultaneous with suppression of preexcitation, LV peak systolic pressure decreased from 172 to 144 mm Hg, while BA maximal systolic pressure increased from 96 to 105 mm Hg. Consequently the ventricular-arterial systolic gradient decreased from 76 to 39 mm Hg and the arterial diastolic pressure and LV maximal dP/dt remained unchanged (Fig 2).

**DISCUSSION**

In the present case the diagnosis of WPW syndrome is obvious: shortened PR interval, widened QRS complex and delta wave are present, demonstrating the possibility of suppression of these features. The diagnosis of IHSS was documented with right and left heart catheterization and left-ventricular cineangiography.

Observing the results of injections of ajmaline, it is obvious that obstruction decreased at the exact moment that preexcitation disappears. The possibility of a direct effect of the injection of ajmaline by changing contractility or loading can be discarded because obstruction always decreases at the same time and as abruptly as preexcitation disappears. LV and BA pressures immediately preceding preexcitation suppression are identical to those before injection of ajmaline, and changes are produced only with preexcitation suppression.

Some authors speculate regarding changes of ventricular contraction related to an anomalous excitation due to WPW syndrome. In WPW syndrome a murmur disappearing simultaneously with preexcitation is attributed to an early deformation of the left ventricular outflow tract.

In our case the WPW syndrome, suspected as posterior right ventricular activation, produced a stronger LVOTO. Thus the coexistence of WPW syndrome and IHSS may be a factor in increasing the severity of LVOTO, and the ventricular activation pathway should be considered among factors in the severity of obstruction in left ventricular ejection in patients with IHSS.

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**Idiopathic Hypertrophic Subaortic Stenosis Presenting as Cough Syncope**

Carl W. White, M.D., Thomas J. Zimmerman, M.D., and Masud Ahmad, M.D.

In a patient with idiopathic hypertrophic subaortic stenosis, syncope developed as a result of a sustained decrease in aortic pressure induced by severe cough paroxysms. Treatment with propranolol was effective in abolishing the syncopal episodes, by reducing the post-tussive gradient and facilitating a more rapid return to normal of aortic pressure. Post-tussive syncope in IHSS may result from both an unusually strong cough paroxysm and augmented left ventricular outflow obstruction consequent to reflex sympathetic stimulation.

*Asymmetric hypertrophy of the left ventricular outflow tract is characterized by a dynamic muscular obstruction to left ventricular ejection, which may be

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