Ventricular Tachycardia during Cardiac Catheterization of Patient with Wolff-Parkinson-White Syndrome

Report of a Case Showing Effects of Atropine Sulfate

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Cardiac catheterization of a patient with probable Wolff-Parkinson-White syndrome was attempted to clarify the possibility of coexistent congenital heart disease. Introduction of the catheter into the heart induced a multifocal ventricular tachycardia which endangered the life of the patient. After quinidine and other drugs had failed to control the arrhythmia that persisted for nine hours, a prompt and gratifying conversion to supraventricular tachycardia, and subsequently to sinus rhythm, followed the intravenous administration of atropine sulfate.

The Wolff-Parkinson-White syndrome consists of an abnormally short P-R interval, usually prolonged duration of QRS complexes, and history of repeated episodes of paroxysmal tachycardia. Variations of QRS complex have been observed during tachycardia, following exercise or atropine sulfate, or even while patients were at rest. The paroxysmal tachycardias have been generally supraventricular, especially auricular, occasionally auricular fibrillation, and rarely paroxysmal ventricular tachycardia. Although the Wolff-Parkinson-White syndrome is usually unassociated with organic heart disease, rheumatic disease has been observed not infrequently. Sudden death of patients with this syndrome has warranted a guarded prognosis.

Short paroxysms of ventricular tachycardia are known to occur occasionally during cardiac catheterization and to stop promptly following partial withdrawal of the catheter tip. Two cases of Wolff-Parkinson-White syndrome, subjected to cardiac catheterization, have been reported by Ferrer and associates. Each exhibited paroxysms of supraventricular tachycardia without apparent hazard to their welfare. The following case is reported because it presents two features of interest: the occurrence of repeated paroxysms of ventricular tachycardia with multifocal characteristics in a patient with cardiac enlargement and Wolff-Parkinson-White syndrome, and the striking benefit of intravenous atropine sulfate in the control of this arrhythmia when quinidine and other medications had failed.

Case Report

A 26 year old Italian man has had repeated attacks of paroxysmal tachycardia since 1929. He has had slight cyanosis, clubbing, and splitting of the first sound with a harsh systolic murmur at the apex transmitted to axilla. He also had low grade fever for a prolonged period of time during childhood.

Since then he has had repeated attacks of paroxysmal tachycardia, three or four times a year. Often the attacks could be controlled by ocular pressure. A chest film showed cardiac enlargement, and fluoroscopy revealed slight prominence of the left auricle, and no hilar dance.

An electrocardiogram taken in September, 1949 (Fig. 1, left) showed moderate left axis deviation, sinus rhythm, short P-R interval of 0.10 second, and deformed and slurred QRS complex indicative of the Wolff-Parkinson-White characteristics resembling type 1 of Burch and Kimball. The Q-T interval was 0.44 second, and the K (Q-Tc), 0.459. The R waves were prominent in Leads AVL and V6. Stethocardiograms revealed a systolic murmur at the apex, presystolic sound at the aortic area following the P wave of the electrocardiogram and an early diastolic de-
crescendo murmur along the left sternal border in the third and fourth intercostal spaces. The arterial blood oxygen saturation was 84 per cent with PO₂ 49 mm. Hg; the hemoglobin was 20.3 Gm. per 100 cubic centimeters.

Cardiac catheterization was performed with a double catheter (sampling tube and intracardiac electrode combined) inserted into the right arm vein. Under fluoroscopic control, the catheter was readily passed into right atrium to the region of the tricuspid valve. Immediately the patient complained of palpitations and tachycardia. Neither ocular pressure nor carotid sinus pressure were effective. He rapidly became apprehensive, restless, and ashen gray with a cyanotic tint, and perspired profusely; the blood pressure was unobtainable. The peripheral pulse at the wrist was irregular, faint, and about 50 to 70 per minute. A very rapid irregular rate was observed at the apex of the heart. The legs were raised to aid venous return and oxygen therapy was instituted. Electrocardiograms revealed multifocal ventricular tachycardia with a rate of 220 to 250 per minute (Fig. 1, right B). Quinidine sulfate, 0.4 Gm., given by mouth, and followed in half an hour by 0.6 Gm. quinine dihydrochloride intravenously, was without any effect. Short intervals of bizarre low-voltage complexes suggestive of incipient ventricular fibrillation were observed frequently during almost continuous recording of a chest lead electrocardiogram on a direct-writing instrument. The patient never lost consciousness although he showed signs of cerebral anoxia. Nine hours later, after futile efforts using morphine sulfate (0.016 Gm.), magne-

sium sulfate (1.0 Gm.), and a total dose of 2.4 Gm. of quinidine sulfate (plus 0.6 Gm. quinine dihydrochloride parenterally), intravenous 1.0 per cent procaine, and niacinamide, there was no significant change (Fig. 2, upper). Then 1.2 mg. of atropine sulfate was administered intravenously and promptly the bizarre ventricular tachycardia converted to a supraventricular tachycardia which superficially resembled auricular flutter with a 2:1 block and ventricular rate of 146 per minute which lasted six hours (Fig. 2, lower). (It may have been a nodal tachycardia with retrograde depolarization of the auricles.) Gallop rhythm and snapping of the first heart sound were noted. Finally the tachycardia reverted to a sinus rhythm, with a rate of about 86 per minute, and the color, heart rate, blood pressure and heart sounds were restored to normal. There were no signs of congestive heart failure. He was kept on a maintenance dose of quinidine sulfate, 0.4 Gm. every six hours, for the next few days without any recurrence of tachycardia. Electrocardiographic and pneumocardiographic study after restoration of normal sinus rhythm failed to show any residual effects of the tachycardia. The patient recovered strength, felt well, and was discharged five days later.*

* An exercise tolerance test performed eighteen days after the paroxysmal ventricular tachycardia showed a conspicuous reduction in the physical fitness index from 16 to 10.6 due to tachycardia. Subjectively the patient did not tolerate the exertion as well as before but denied any specific symptoms.
The history is confusing in determining the cause of the cardiac enlargement. The laboratory data are compatible with rheumatic mitral valvulitis, auricular hypertrophy, and pulmonary insufficiency. Presumably the hypoxemia indicates right-to-left shunting of blood from an unrecognized auricular septal defect, or diffuse vascular anastomoses of the lungs. Unfortunately the ventricular tachycardia precluded a definitive diagnosis. The persistent arrhythmia did not respond to reflex vagal stimulation. Significant quantities of quinidine, niacinamide, magnesium sulfate, morphine sulfate,* as well as procaine hydrochloride (up to 350 mg. intravenously) failed to revert the arrhythmia. However the intravenous adminis-

* Sabathiel found that morphine sulfate, 0.01 to 0.04 Gm. intravenously, was effective in controlling paroxysmal ventricular tachycardia in 9 out of 10 patients.
treatment of 1.2 mg. of atropine sulfate was promptly followed by a conversion to supraventricular tachycardia, either auricular flutter with 2:1 block, or nodal tachycardia with reentrant auricular depolarization. This in turn converted to sinus rhythm with a normal rate in another six hours. Fortunately there were no sequelae after recovery indicative of myocardial injury or ischemia. This feature has been observed in other reported cases when the initial impression of myocardial infarction could not be supported by subsequent events. Nevertheless, the alarming severity of the circulatory collapse observed in this patient stood in marked contrast to the not infrequent episodes of fleeting paroxysms of ventricular tachycardia recorded during manipulation of the intracardiac catheter in other patients.

The mechanism of the beneficial action of atropine sulfate in converting a ventricular to a supraventricular tachycardia is largely speculative. It is known that atropine may prevent the occurrence of ventricular tachycardia during cyclopropane anesthesia or epinephrine induced tachycardia in dogs.16 Wilbur and co-workers believed that atropine acts on the “acetylcholine-cholinesterase” mechanism to prevent reflex vagal-induced premature systoles, paroxysmal ventricular tachycardia, and fibrillation. Further experimental studies in dogs by Lenel and associates17 indicated that ventricular tachycardia is induced by hyperexcitability of the idioventricular pacemakers in the presence of epinephrine, or by stretching of the myocardium due to a sudden increase in pressure, or by vagal inhibition of the higher pacemakers. Bilateral vagotomy always stopped the paroxysm, and neither epinephrine nor vagal stimulation alone was capable of inducing a paroxysm in the vagotomized animal. Furthermore, ventricular tachycardia can be inhibited by pentobarbital anesthesia, dibenamine, and to some extent morphine.18

**Summary**

An unusual case of multifocal paroxysmal ventricular tachycardia occurring during cardiac catheterization in a patient with cardiac enlargement and probable Wolff-Parkinson-White syndrome is reported. The etiology of the heart disease was not established, but features indicative of rheumatic valvulitis and congenital septal defect could not be excluded. The tachycardia was accompanied by profound circulatory collapse and did not respond to quinidine sulfate by mouth, or to quinidine dihydrochloride intravenously. Intravenous atropine sulfate (0.0012 Gm.) promptly converted the ventricular tachycardia to a supraventricular tachycardia resembling auricular flutter, which changed to sinus rhythm with a normal rate. The possible actions of atropine are briefly discussed.

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