Termination of Supraventricular Tachycardia Complicating the Wolff-Parkinson-White Syndrome with External Countershock

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The termination of various refractory supraventricular and ventricular arrhythmias by external electric countershock has been reported by Zoll and Linenthal. We are unaware, however, of any reports of supraventricular tachycardia complicating the Wolff-Parkinson-White (WPW) syndrome.

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Figure 1

The upper two rows of this figure were obtained just prior to countershock. The rate is 200 beats per minute with aberrant intraventricular conduction due to WPW. The third row, recorded after the countershock, shows the classical WPW syndrome with a short P-R interval and the slurring on the upstroke of the QRS. The ST-T segment and T-wave changes are in all probability secondary to the WPW as well as to the effects of digoxin and quinidine.
treated in this manner and thus thought it worth while to report the following case. This case is also of additional interest because of the occurrence of rare ventricular fusion complexes between impulses originating in the sinoatrial and atrioventricular nodes.\textsuperscript{2-3}

Case Report

The patient, a 17-year-old man, was first hospitalized at the Indiana University Medical Center in 1952 at the age of 6. The patient had tachycardia for 10 days prior to admission that failed to respond to digitalis, cholinergic drugs, and quinidine. During hospitalization, the digitalis was continued and an additional 1.4 Gm. of quinidine was administered over a 20-hour period with conversion of the tachycardia to sinus rhythm. At that time the WPW syndrome was first recognized.

The second admission was in 1957, when the patient was 12 years old. Tachycardia had been present for 9 days prior to admission and an unknown amount of quinidine had been administered. Following admission 0.4 Gm. of quinidine was given every 2 hours for five doses with conversion of the tachycardia to sinus rhythm. The electrocardiogram again revealed the WPW syndrome. The patient was discharged on a maintenance dose of 0.2 Gm. of quinidine three times daily. This dose was continued until his most recent admission.

The patient was admitted for the third time on May 10, 1962, with tachycardia that began the previous evening (fig. 1). While in the hospital the apical rate varied from 180 to 220 beats per minute. The blood pressure was obtainable only by palpation at about 80 to 90 mm. Hg. The patient complained constantly of nausea and vomited frequently. During the 4 days following hospitalization therapy included vasopressor agents, a total of 2.6 mg. of digoxin, and 6.6 Gm. of quinidine. By the fourth day the patient's condition was precarious. He continued to vomit, was unable to retain fluids, and exhibited signs of digitalis and quinidine intolerance. On May 14 the patient was taken to surgery where under general anesthesia with concomitant use of a muscle relaxant external countershock was applied from an A-C defibrillator with a defibrillating current of 5 amperes for 0.25 second with an output of 480 volts.\textsuperscript{6} One shock was required to restore sinus rhythm with WPW pattern (fig. 1). The appearance of permanent sinus rhythm with WPW conduction was preceded by short runs of AV nodal rhythm. During this interplay of the two pacemakers, ventricular fusion complexes between impulses originating in the SA and AV nodes were recorded (fig. 2).

Summary

A case that demonstrates the feasibility of using external electric countershock for termination of paroxysmal supraventricular tachycardia complicating the Wolff-Parkinson-White syndrome is presented.

\textsuperscript{*}General Electric.

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The occurrence of fusion complexes between impulses originating in the sinoatrial and atrioventricular nodes was observed.

References

Amyl Nitrite and Angina Pectoris

Few things are more distressing to a physician than to stand beside a suffering patient who is anxiously looking to him for that relief from pain which he feels himself utterly unable to afford. His sympathy for the sufferer, and the regret he feels for the impotence of his art, engrave the picture indelibly on his mind, and serve as a constant and urgent stimulus in his search after the causes of the pain, and the means by which it may be alleviated.

Perhaps there is no class of cases in which such occurrences as this take place so frequently as in some kinds of cardiac disease, in which angina pectoris forms at once the most prominent and the most painful and distressing symptom. This painful affection is defined by Dr. Walshe as a paroxysmal neurosis, in which the heart is essentially concerned, and the cases included in this definition may be divided into two classes.

In the first and most typical there is severe pain in the precordial region, often shooting up the neck and down the arms, accompanied by dyspnea and a most distressing sense of impending dissolution. The occurrence and departure of the attack are both equally sudden, and its duration is only a few minutes.

In the second class, which from its greater frequency is probably the more important, though the pain and dyspnea may both be very great, the occurrence of the attack is sometimes gradual, and its departure generally so; its duration is from a few minutes to an hour and a half or more, and the sense of impending dissolution is less marked or altogether absent.—T. L. Brunton. “Use of Nitrite of Amyl in Angina Pectoris.” The Lancet, 2: 97, 1867.
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