NONPROPAGATED electrical activity of diseased myocardial cells has been well demonstrated, particularly in canine\(^1\) and human ischemic myocardium.\(^2\) The role of such activity in the modulation of paroxysmal dysrhythmia in man, although strongly suspected, is undefined. We describe the reproducible termination of multiple episodes of sustained ventricular tachycardia (VT) by a single, nonpropagated pacing stimulus delivered during the effective refractory period of the right ventricle.

**Case Report**

GF was a 57-year-old man with ischemic heart disease, an old anterior myocardial infarction and severe left ventricular dysfunction. He had been treated for 2 years with various antiarrhythmic drugs for several episodes of VT and ventricular fibrillation that occurred within 1 month after the infarction. At the time of the study, the patient was receiving no antiarrhythmic therapy. An atrioventricular sequential permanent pacemaker had been implanted several months earlier, and amiodarone had been discontinued for 3 weeks because of severe neurotoxicity. The patient was taken to the electrophysiology laboratory unsedated and, after local anesthesia (1% lidocaine) of the right femoral area, a #7F (USCI) quadripolar electrode catheter was advanced into the right ventricle under fluoroscopic control. A #5F cannula was placed in the right femoral artery for monitoring the systemic blood pressure. The distal pair of poles of the electrode catheter was connected to a programmable stimulator (Bloom DTU-101), and the proximal pair was used for electrogram recording. Leads I, aVF and V\(_1\) of the surface ECG, the right ventricular (RV) electrogram (filtered between 100 and 500 Hz), the stimulation artifact and the blood pressure tracing were displayed on an oscilloscope (Electronics for Medicine VR-16) and recorded on photographic paper at speeds of 25–100 mm/sec. Programmed stimulation of the right ventricle was performed using rectangular pulses of 2 msec duration, at an initial current strength of twice end-diastolic threshold (1 mA). The protocol of stimulation included single, double and triple extrastimuli applied during sinus rhythm and during ventricular paced rhythm at cycle lengths of 600, 500 and 400 msec, incremental ventricular pacing to a cycle length of 400 msec (limited by hypotension), and ventricular burst pacing to a minimum cycle length of 300 msec. The stimulation was initially performed at the RV apex and continued at the RV midseptum. After the induction of multiple episodes of sustained VT, the patient received i.v. procainamide to a total dose of 1500 mg, and programmed stimulation was repeated.

### Results

**RV Apex Stimulation**

Programmed stimulation of the RV apex failed to induce any tachydysrhythmia.

**RV Septal Stimulation**

**Before Procainamide**

Sustained VT at a cycle length of 405 msec was reproducibly induced by triple extrastimulation of the RV apex. The RV electrogram and the arterial pressure were recorded simultaneously.

**After Procainamide**

Sustained VT at a cycle length of 405 msec was reproducibly induced by triple extrastimulation of the RV apex. The RV electrogram and the arterial pressure were recorded simultaneously.

---

From the Cardiology division, Jewish Hospital of St. Louis, Washington University School of Medicine, St. Louis, Missouri. Address for correspondence: Rodolphe Ruffy, M.D., Cardiology Division, Jewish Hospital, 216 South Kingshighway, St. Louis, Missouri 63110. Received June 14, 1982; accepted July 19, 1982.

RV midseptum. Fourteen consecutive episodes were initiated in the same fashion, and all were identical in rate and QRS morphology in the three surface ECG leads. No episode ended spontaneously. Because the stimulator failed to sense the rhythm properly, precise timing of premature stimulation during VT could not be achieved. However, prolonged periods of random underdrive pacing before procainamide infusion resulted in innumerable ventricular captures that never terminated VT. All episodes were terminated by a non-propagated stimulus delivered in the effective refractory period of the right ventricle (fig. 1). The window of VT termination was narrow, all successful stimuli being 140–165 msec after the onset of the preceding RV electrogram. The effectiveness of the premature stimulus in terminating VT was also current-dependent (fig. 2). No episode ended with a stimulus of 1 mA. Four episodes ended with 2-mA pulses delivered 140–155 msec after the last spontaneous electrogram. The window of termination was widened to its maximum of 165 msec by stimuli 3 mA and higher.

After Procainamide

After 1500 mg of procainamide (serum level 6 μg/ml), VT remained inducible by the same technique; its cycle length became 425 msec, and the morphology was unchanged. However, in contrast to the preceding testing period, all episodes ended with a premature ventricular capture as soon as random underdrive pacing was initiated.

Discussion

The ability to terminate VT by low-energy DC shocks delivered in the ventricular refractory period by an electrode catheter has been demonstrated in animal experiments and clinical studies. This report extends these observations to a nearly physiologic pulse strength delivered from a close bipolar electrode. Our case also relates to the previous report by Wellens et al. of VT termination by a single atrial premature impulse without overt ventricular capture.

The importance of the electrode placement in our ability to identify this rare phenomenon is supported by the failure of RV apical stimulation to induce VT, in contrast to the reproducible initiation of the dysrhythmia from the midseptum. It is tempting to hypothesize that the tip of the pacing electrode was fortuitously placed on, or very near, a critical anatomic site responsible for sustaining VT. From a basic electrophysiologic standpoint, our observation, as often is the case, does not distinguish reentry from abnormal automaticity as the underlying mechanism of VT. Whereas most of the ventricular myocardium was refractory at the time of premature stimulation, the site of origin of the VT had probably sufficiently recovered its excitability to be depolarized by the artificial pulse; this explanation is applicable to a reentrant mechanism using a portion of the RV septum, and thus would be comparable to the example provided by Wellens et al., as well as to an automatic oscillator triggered by triple extrastimulation and extinguished by a critically timed single premature pulse. Short of a regenerative depolarization, the terminating stimulus could have produced enough local electrotonic activity to stop the propagation of a reentrant wave front or extinguish a triggered focus.

The marked change in response to premature stimulation produced by procainamide is of great interest, but its discussion extends well beyond the scope of this case report.

Whatever basic mechanism underlies this observation, it may be of far-reaching clinical importance. Indeed, the rapid progress made in the increasingly accurate localization of the ‘‘site of origin’’ of paroxysmal tachydysrhythmias may soon allow the design, in selected cases, of electronic interfaces sufficiently refined to modify the electrophysiologic behavior of part of, rather than the whole heart. The successful termination of VT by low-energy pulses delivered during ventricular refractoriness would combine the advantages of safety (by minimizing the risks of VT acceleration), patient comfort, and increase in battery life.

References

1. Friedman PL, Stewart JR, Wit AL: Spontaneous and induced cardiac arrhythmias in subendocardial Purkinje fibers surviving extensive myocardial infarction in dogs. Circ Res 33: 612, 1973
Primary Pulmonary Artery Sarcoma Diagnosed Noninvasively by Two-dimensional Echocardiography

EDWARD C. WRIGHT, M.D., HARRY A. WELLONS, M.D., AND RANDOLPH P. MARTIN, M.D.

SUMMARY A 21-year-old white male was evaluated in our echocardiographic laboratory for presumed mitral valvular bacterial endocarditis. Electrocardiographic, physical, and initial two-dimensional echocardiographic findings suggested a left-to-right shunt at the atrial septal level. However, injection of contrast saline solution failed to demonstrate signs of an atrial septal defect. Continuation of the echocardiographic study led to the diagnosis of an unsuspected primary sarcoma of the pulmonary trunk, which was rapidly confirmed by computerized axial tomography. Therapeutic interventions were undertaken. This case highlights the usefulness of two-dimensional echocardiography.

PRIMARY SARCOMAS of the pulmonary trunk are very rare. A review of available literature revealed only 67 reported cases. All but 15 of these cases were diagnosed at autopsy. The 15 cases not diagnosed at autopsy were diagnosed at catheterization and angiography performed for a presumptive diagnosis other than tumor of the pulmonary trunk. We report a case of unsuspected primary sarcoma of the pulmonary trunk diagnosed noninvasively by two-dimensional echocardiography.

Case Report

A 21-year-old white male steel worker was referred for evaluation of a heart murmur. Thirteen months previously, he had been successfully treated for right upper lobe pneumonia. Subsequently, he complained of intermittent cough and dyspnea on exertion. Six months before we saw him, he experienced the sudden onset of severe dyspnea and chest pain and a 30-second syncopal episode while running. He did not seek medical attention. Three weeks before referral, the patient was seen by his physician for chills and cough. An apparently new systolic murmur was noted and the patient was hospitalized for possible endocarditis. Evaluation failed to produce evidence of bacterial infection. An M-mode echocardiogram suggested a flail posterior mitral valve leaflet or mitral valve prolapse. The patient was discharged with a diagnosis of an upper respiratory illness, probably viral, and referred to the Cardiac Noninvasive Laboratory at the University of Virginia Medical Center for further evaluation of his murmur and mitral valve apparatus.

Physical examination revealed an extremely thin white male who was dyspneic on minimal exertion. There were no features of Marfan's syndrome. His respiratory rate was 26 breaths/min and his pulse rate was 110 beats/min. The jugular veins were not distended and there was no pedal edema. His lungs were clear. Examination of the heart revealed a normally situated apical impulse and a right ventricular heave. The first heart sound was normal. The pulmonic component of the second heart sound was accentuated and widened physiologic splitting was present. An early to mid-systolic click and a soft blowing mid- to late systolic murmur were present apically. Additionally, a high-pitched ejection murmur that increased in intensity with inspiration was present to the left of the sternum.

A two-dimensional echocardiogram was performed with the patient reclining at 30° elevation in the left lateral decubitus position. The left ventricle was normal in size, wall thickness and function, although the intraventricular septum moved paradoxically. The left atrium was of normal size. The mitral valve demonstrated systolic prolapse. The aortic valve was normal. The right atrium was markedly enlarged. The right ventricle was markedly dilated and hypococontractile. The tricuspid and pulmonic valves were normal. Because a left-to-right shunt was suspected, contrast saline solution was injected intravenously in the left arm. No negative contrast could be demonstrated at either the atrial or ventricular level. No microbubbles passed retrogradely into the inferior vena cava or hepatic veins, thereby excluding significant tricuspid regurgitation. Because the microbubbles cleared slowly from the right ventricle, the right ventricular outflow tract, pulmonary valve and main pulmonary trunk were examined more closely. Just beyond the normal pulmo-
Termination of ventricular tachycardia by single extrastimulation during the ventricular effective refractory period.
R Ruffy, K J Friday and W F Southworth

Circulation. 1983;67:457-459
doi: 10.1161/01.CIR.67.2.457

Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 1983 American Heart Association, Inc. All rights reserved.
Print ISSN: 0009-7322. Online ISSN: 1524-4539

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://circ.ahajournals.org/content/67/2/457

Permissions: Requests for permissions to reproduce figures, tables, or portions of articles originally published in Circulation can be obtained via RightsLink, a service of the Copyright Clearance Center, not the Editorial Office. Once the online version of the published article for which permission is being requested is located, click Request Permissions in the middle column of the Web page under Services. Further information about this process is available in the Permissions and Rights Question and Answer document.

Reprints: Information about reprints can be found online at:
http://www.lww.com/reprints

Subscriptions: Information about subscribing to Circulation is online at:
http://circ.ahajournals.org//subscriptions/