Ventricular Tachycardia in Infancy: Evidence for a Reentrant Mechanism

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SUMMARY Ventricular tachycardia is unusual in infancy. Three infants are described in whom this arrhythmia was documented by electrophysiologic studies. The ability to start and terminate this rhythm by critically timed premature ventricular stimulation suggests a reentrant mechanism. All three patients have remained free of arrhythmias on oral propranolol therapy.

VENTRICULAR TACHYCARDIA is uncommon in childhood but is being reported more often.1-4 We recently studied three infants with no evidence of associated cardiac disease or systemic cause for the arrhythmia in whom ventricular tachycardia could be initiated and terminated by critically timed premature ventricular stimulation.

Case 1

A 3-month-old male was admitted to Children's Hospital Medical Center because of tachypnea. The infant was asymptomatic until 2 days before admission, when his mother noted increased respiratory noise, effort and rate. His pediatrician noted a rapid heart rate and hepatomegaly and referred the child with a diagnosis of supraventricular tachycardia and heart failure. Examination revealed a 5.4-kg baby in moderate respiratory distress with a heart rate of 220 beats/min. Blood pressure was 70/40 mm Hg. He had a grade II/V systolic ejection murmur at the left sternal edge. The liver edge was palpable 3 cm below the costal margin. He had no cyanosis or edema. Chest x-ray revealed mild cardiomegaly with clear lung fields. An ECG showed a ventricular rate of 215 beats/min. The QRS was of a right bundle branch block pattern with marked left-axis deviation. P waves were seen in leads 2 and 3 with a 1:1 relationship to the QRS complex (fig. 1). The initial diagnostic impression was supraventricular tachycardia with aberrant conduction. The infant was treated with digoxin and propranolol, but had no change in rhythm.

Lidocaine, 1 mg/kg, was given intravenously as a bolus and repeated after 15 minutes, with no effect. Digitalis was withheld and DC cardioversion with 30 watt-sec (which exceeds our usual dosage of 1-2 watt-sec/kg) was attempted twice, without interruption of the tachycardia. He was receiving no medication at the time of the intracardiac electrophysiologic study.

Case 2

An 8-month-old male was admitted because of tachycardia noted at the time of a routine baby check. Physical examination revealed a well-developed 8.5-kg male in no distress and was unremarkable except for a heart rate of approximately 200 beats/min. An ECG that was interpreted as paroxysmal atrial tachycardia revealed a wide QRS of a right bundle branch block pattern with a superior frontal plane axis. Distinct P waves were not seen (fig. 2). The patient was treated with digoxin and converted to sinus rhythm within 12 hours. He was discharged on maintenance digoxin, 10 μg/kg per day. Follow-up examination 1 month later revealed an episode of tachycardia with an ECG identical to that taken previously. Digoxin was discontinued and he was admitted for further study.

Case 3

A 15-month-old female was brought to the hospital because of irritability, difficulty maintaining her balance, and increased perspiration for 12 hours before admission. Physical examination revealed a pale, sweating, lethargic child who weighed 10.9 kg. There was slight circumoral and peripheral cyanosis. The heart rate was approximately 250 beats/min. Rales were heard over both lung fields and the liver edge was palpable 3 cm below the costal margin. An ECG revealed a tachycardia with a left bundle branch block pattern at a rate of 250 beats/min. P waves were not clearly seen (fig. 3). Lidocaine, 1 mg/kg, given as a bolus intravenously, resulted in conversion to sinus rhythm. The patient was on no medication at the time of the electrophysiologic study.

Methods

Electrophysiologic studies were performed under ketamine anesthesia after informed parental consent. Case 1 had a #4F bipolar pacing catheter inserted percutaneously into the femoral vein and used for atrial and ventricular pacing. In cases 2 and 3, a #4F bipolar pacing catheter was positioned in the right atrium and another across the tricuspid orifice. Interelectrode distance was 5 mm. Tracings were recorded on a multichannel oscilloscopic recorder (Electronics for Medicine VR-12) at a paper speed of 100 mm/sec using filter settings of 40–500 Hz for electrograms and

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**Figure 1.** Case 1. Ventricular rate of 215 beats/min with a right bundle branch pattern. A 1:1 relationship to a P wave is suggested in lead 3 and V1.

**Figure 2.** Case 2. Ventricular rate of 190 beats/min with a right bundle branch block pattern and superior frontal plane axis. P waves are seen in leads aV1 and aV5, and appear dissociated.
0.2–20 Hz for the surface ECG. Cardiac stimulation was performed using an isolated programmable stimulator (Metronics Model 5325) with a stimulus of 1 msec duration, 1.5 times threshold in the right ventricle and up to 2.5 times threshold in the atrium.

Results

Case 1

Figure 4A illustrates right atrial pacing at a rate of 200 beats/min with atrial capture and narrowing of the QRS and dramatic shift in frontal-plane axis. The tachycardia could be interrupted (fig. 4B) and initiated (fig. 4C) by a premature stimulus delivered to the right ventricular apex 200–210 msec after a sinus impulse.

Case 2

Recordings (fig. 5) revealed intermittent atrioventricular dissociation, ventricular complexes not preceded by His potentials and a capture beat, in which a His potential clearly precedes ventricular depolarization, confirming ventricular tachycardia. The His catheter was positioned at the right ventricular apex and the tachycardia was interrupted by a premature stimulus delivered 190 msec after the onset of the QRS. We could induce ventricular tachycardia by pacing the ventricle at a cycle length of 400 msec and delivering a premature stimulus (S₂) at the same site 290 and 300 msec after the basic stimulus (S₁). The tachycardia could not be induced by premature ventricular stimulation during normal sinus rhythm.

Case 3

We could initiate ventricular tachycardia by pacing in the right ventricular inflow area at a cycle length of
400 msec and delivering premature ventricular stimuli to the same site at an S1S2 interval of 200–220 msec. Stimulation of other sites within the right ventricle did not initiate the tachycardia (fig. 6). There was 1:1 retrograde atrial capture during tachycardia and the QRS morphology was very similar to that of the tachycardia that occurred spontaneously (fig. 3). The tachycardia was terminated by a short burst of ventricular pacing at a cycle length of 30 msec shorter than the tachycardia (fig. 7). Intravenous propranolol, 0.01 mg/kg, prevented reinitiation of the tachycardia by premature right ventricular stimulation.
During sinus rhythm, AH and HV intervals were normal in cases 2 and 3. No evidence of preexcitation (Wolff-Parkinson-White syndrome) was noted in any of the patients during sinus rhythm or during atrial pacing or premature atrial stimulation. Blood gases, serum electrolytes and glucose were normal in each case during hospitalization. Patients received continuous ECG monitoring before and after conversion to sinus rhythm. Spontaneous initiation of the tachycardia was not recorded. No arrhythmia was noted after conversion to sinus rhythm. In case 1, heart size became normal after conversion to sinus rhythm and no abnormalities were noted on physical examination, ECG or echocardiogram in each case. Viral studies revealed no evidence of coxsackievirus B or echovirus.

Treatment and Follow-Up

Lidocaine, 1 mg/kg, was given in cases 1 and 3 because of its known effect on ventricular tachycardia and its property of lengthening the refractory period in accessory pathways. Each patient was treated with propranolol, 5 mg four times daily, and has been without clinical and Holter-monitor evidence of tachycardia during follow-up of 1½ years (case 1), 1 year (case 2), and 4 months (case 3).

Discussion

Ventricular tachycardia is reported to be a relatively rare arrhythmia in infancy and childhood. Some have speculated that many of the previously described cases of ventricular tachycardia in childhood are examples of supraventricular tachycardia with aberrancy. This can lead, as noted in case 1, to the incorrect and hazardous therapy with digoxin, the most commonly prescribed treatment for paroxysmal atrial tachycardia. However, without a previous tracing in sinus rhythm for comparison, and the presence of fusion or capture beats, ventricular tachycardia cannot be distinguished from supraventricular tachycardia with aberrant conduction or rapid antegrade conduction via an accessory pathway (Wolff-Parkinson-White syndrome). A 1:1 relationship between QRS and P waves, as seen in the surface ECG trace of case 1, is not helpful in differentiating supraventricular from ventricular tachycardia, as retrograde capture of the atria during the latter is common. The diagnosis of ventricular tachycardia must be considered in every fast rhythm in which the QRS morphology is different in contour from that seen in sinus rhythm.

The clinical presentation of these three infants is noteworthy in that patient 2 was completely asymptomatic during tachycardia. Cases 1 and 3 had signs and symptoms of heart failure. This may have been related to the duration of the arrhythmia, which was uncertain. Cases 1 and 3 may have had hemodynamic deterioration caused by impaired ventricular filling because of contraction of the atria against closed atrioventricular valves as a result of 1:1 retrograde capture of the atria during tachycardia. This was not definitely established in case 1.

The diagnosis of ventricular tachycardia was confirmed in case 1 by normalization of the QRS during ventricular capture with atrial pacing at a rate faster than the tachycardia. In cases 2 and 3 ventricular tachycardia was confirmed by His bundle recordings. In 1972, Wellens and co-workers described initiation and termination of ventricular tachycardia by critically timed ventricular stimuli and attributed this to a reentrant mechanism. The ease of induction and termination of ventricular tachycardia by a single, critically timed stimulus delivered to the right ventricle suggests a reentrant mechanism in our patients. The extrastimulus affected the potential reentrant circuit to set the scene for reentry or interrupt the circuit and thus terminate the tachycardia. Whether these cases are examples of bundle branch reentry is difficult to determine without mapping both bundle branches during the tachycardia.

Case 1 is of interest in that the tachycardia was resistant to lidocaine and DC cardioversion. Atrial pacing and premature ventricular stimulation confirmed the diagnosis and easily interrupted the arrhythmia. We do not know why cardioversion failed to interrupt the tachycardia. It may have been restarted by a premature ventricular contraction that occurred before monitoring could be reestablished after DC cardioversion.

In cases 2 and 3 electrophysiologic studies clarified
the diagnosis and in case 3 strengthened the clinical impression of the efficacy of propranolol. The efficacy of propranolol alone may be due to several factors. Each of our cases had a very narrow echo zone during which premature stimulation initiated ventricular tachycardia. A very slight change in conduction time in the reentrant circuit due to propranolol may have been sufficient to prevent recurrence of the tachycardia. Propranolol may also be effective in suppressing premature ventricular contractions, which initiate the tachycardia, although at least in our three cases, an effect on the reentrant circuit is more likely.

The absence of associated cardiac and/or systemic disease may have been an important factor. Cases of ventricular tachycardia in childhood associated with myocarditis, for example, frequently require multiple drug regimens for their control. Ventricular tachycardia that appears multifocal is also usually more malignant. These cases may represent a subgroup within ventricular tachycardias in infancy. Whether a change in the reentrant pathways responsible for perpetuation of tachycardia will occur with age will be of interest.

We emphasize that in wide QRS tachycardia in infancy, ventricular tachycardia must be strongly considered. If a diagnosis is not possible from the surface tracing and the patient’s condition does not warrant immediate pharmacologic intervention or cardioversion, we recommend electrophysiologic studies that will elucidate the correct diagnosis, may be successful in terminating the tachycardia, and may provide valuable information about the mechanism of the tachycardia and its response to therapy.

References
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