A 41-year-old female came to the emergency room because of an episode of palpitations. She told us that she had suffered from similar episodes for 1 year, usually lasting \( \approx 20 \) minutes, with spontaneous termination. The incidence was twice a month, usually in relation to exercise or emotional stress.

She came because the current episode had lasted \( \approx 45 \) minutes. On physical examination, the only abnormal finding was “canon a waves” on inspection of the neck. Her 12-lead ECG (Figure 1) showed a tachycardia of \( \approx 130 \) bpm. The tachycardia could be terminated by carotid sinus massage (Figure 2). After the conversion to sinus rhythm, an echocardiographic examination showed normal cardiac volumes and function.

The following questions should be answered:

1. What kind of tachycardia was present?
2. What is the most likely mechanism of the tachycardia?
3. What would be the best therapy?

**Discussion**

(1) During the tachycardia, there is atrioventricular dissociation. The sinus P waves are indicated by arrows in Figure 1. Some of these P waves are conducted to the ventricle.
The tachycardia QRS complex is 110 ms wide and has an incomplete right bundle branch block configuration.

These findings led to the diagnosis of a ventricular tachycardia (VT) originating in the specific interventricular conduction system. The QRS width, configuration, and axis during the tachycardia suggest an origin in or close to the main stem of the left bundle branch. Unfortunately, the origin remains uncertain because of the lack of an electrophysiological study. Idiopathic VTs arising from the interventricular conducting system typically have a right bundle branch block morphology and a left (common form) or a right (uncommon form) axis, depending on the fascicle involved. Tachycardias with a left bundle branch block configuration and a right or a normal axis, based on an origin in the left upper ventricular septum, have also been described. To the best of our knowledge, idiopathic VTs with a typical right bundle branch block configuration and a normal axis suggesting an upper septum origin as well, as in our case, have never been described before.

The tachycardia seems to be originating in or close to the main stem of the left bundle branch. During the tachycardia, the “capture” beats do not reset the tachycardia, making a reentry mechanism unlikely. However, a reentrant circuit with a short excitable gap or a microreentrant focus is not excluded. Abnormal automaticity might be suggested by the fact that the tachycardia usually occurs during high sympathetic tone (exercise and emotional stress), supported by the underlying sinus tachycardia during the VT and the relatively high sinus node frequency after carotid sinus massage (CSM). On the other hand, the termination by CSM is compatible with triggered activity as the mechanism. As pointed out by Lerman et al, those tachycardias are based on triggered activity, are catecholamine sensitive, have a cAMP-mediated mechanism, and do respond to CSM, adenosine, calcium antagonists, and β-blocking agents. The fact that these tachycardias are sensitive to adenosine and verapamil makes an automatic mechanism less likely. The patient did not want an invasive electrophysiological study, and therefore the diagnosis could not be confirmed.

As pointed out under 2, termination and prevention of this type of tachycardia can be accomplished by vagal and different pharmacological means. The efficacy of oral verapamil in preventing relapse is variable. Our patient was treated with a β-blocking agent because exercise and emotional stress seemed to be a triggering mechanism. This resulted in complete prevention of the arrhythmia. Left ventricular upper septal VTs exhibiting left bundle branch block morphology have been successfully ablated before. Our patient had typical incomplete right bundle branch block morphology,
indicating a more proximal origin in the upper septum or close to the main stem of the left bundle branch. Therefore, we believe that catheter ablation in our patient would have been a procedure with a very high chance of creating complete atrioventricular block.

**Conclusion**

A patient with an idiopathic ventricular tachycardia and electrocardiographic features indicative of an origin in or close to the main stem of the left bundle branch resulted in a QRS width of only 110 ms. The most likely mechanism was triggered activity based on a cAMP-mediated mechanism.

**Disclosures**

None.

**References**


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