Appearance of Ventricular Ectopic Rhythm During Carotid Sinus Pressure*

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The observation to be discussed in this report illustrates how carotid sinus pressure may enhance impulse formation in the ventricle.

A 55-year-old housewife with a 15-year history of paroxysmal tachycardia was examined by one of us three hours after the onset of an attack. Similar attacks which usually appeared after retiring would last only a few minutes. She described “a funny sensation as if fading away” along with a quivering or palpitation inside. The attacks occurred often in the lateral position and disappeared on change of position. They were accompanied by profuse salivaion — a not too rare, although little known, symptom of paroxysmal tachycardia. Previous examination did not reveal any sign of organic heart disease and the electrocardiogram showed normal sinus rhythm with a normal P-R interval and right bundle branch block.

In Fig. 1A, the electrocardiogram during the attack shows a supraventricular tachycardia with a rate of 187 beats per minute. It is recorded in lead II. With carotid sinus pressure, the tachycardia terminated within a few seconds to be followed after a pause of only 0.44 second by a ventricular tachycardia. The ventricular extrasystoles appear in groups of two (“extrasystoles in groups”). The short intervals measure 0.25 second. This cycle length corresponds to a rate of 240 beats per minute. Tachycardia of this type may be attributed to a regular rapid formation of impulses in an ectopic center with a 3:2 exit block. Upon release of the carotid sinus pressure, the ectopic ventricular beats promptly disappeared. Repeated attempts of carotid sinus pressure a few minutes later failed to elicit ventricular ectopic beats. Fig. 1b shows such failure (the tracing is slightly distorted by artifacts).

The high rate of 240 ectopic beats per minute is unusual. Yet rates up to 500 per minute have been found in canine experiments during vagal stimulation. Such rates during carotid sinus pressure (or vagus stimulation) manifestly may lead to ventricular fibrillation, as has been observed, both in the laboratory and clinic.

In order to explain this action of carotid sinus pressure on the ventricles, where it has been postulated that no vagus fibers and direct vagal effects exist, it was assumed that acetylcholine formed in the atria during vagus stimulation reached the ventricles. As is well known, acetylcholine exerts the dual effect of inhibition and enhancement of ectopic impulse formation. In our observation, this action is unlikely because the diastole preceding the ventricular extra beats is prolonged only from 0.32 to 0.44 second, a difference merely of 0.12 second. A more likely explanation would seem the presence of vagal fibers as an exceptional occurrence in the ventricles of the mammalian heart as found by Mitchell and Tcheng.

Of interest is the observation that a few seconds after the end of the attack of paroxysmal tachycardia, carotid sinus pressure performed during regular slow sinus rhythm did not induce the appearance of ventricular extrasystoles. Conceivably the paroxysmal tachycardia, as every rapid rate, increased the negative after-potential.

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in a single ventricular fiber which begins to fire off impulses as soon as threshold strength is reached and the higher impulses are inhibited by the carotid sinus pressure. With this explanation, the ectopic ventricular beats appear passively because of the inhibition of the higher center. Against this assumption speaks the fact that the interval between two single ectopic beats originating in the ventricle is shorter than that between two single beats of the tachycardia. A combination is possible of both factors, the release of acetylcholine and the inhibition of the higher centers. It is known that the process of firing off impulses may be self-sustaining and continue once it had started.

Also noted in laboratory experiments and clinical practice is the awakening of impulse formation in mammalian ventricles by carotid sinus pressure, which is unusual. This case should be added to the recently reported cases, showing ectopic ventricular tachycardia and even ventricular fibrillation during carotid sinus pressure, the Valsalva experiment or eye ball pressure.

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