The Effects of Ventricular Tachycardia on Carotid Artery Blood Flow Velocity

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Abstract: Utilizing the Doppler flowmeter catheter, right carotid blood velocity was measured during episodes of catheter-evoked and pacemaker-induced ventricular tachycardias. Such tachyarrhythmias uniformly diminished peak carotid blood velocity, which in the case of catheter-evoked episodes reduced phasic blood velocity by 50%. At driving rates greater than 140 per minute, the average peak carotid blood velocity was 25% to 50% lower during ventricular when compared with atrial pacing. There was a direct correlation between the ventricular pacing rates and the percent decline of peak carotid blood velocity. Such arrhythmia-related carotid blood velocity deficits may account for syncopal episodes in subjects so affected.

Methods

Nineteen patients comprised the study group. There were 14 men and five women whose ages ranged from 15 to 62 years, with a mean age of 46 years. Five subjects were normal and the remainder had heart disease, thought to represent heart disease. All diagnoses were made on the basis of complete right and left heart catheterization, noninvasive and invasive studies, ECG and exercise tolerance testing, and clinical assessment. Of the 19 patients, 14 had heart disease. In the abnormal group there were 11 patients with coronary artery disease and one with aortic insufficiency. Normal subjects were studied because of the presence of chest pain or systolic murmurs originally characterized phasic carotid artery blood flow velocity, we noted that in a few patients, supraventricular and ventricular tachycardias diminished the amplitude of blood velocity wave-forms. We report here in detail the abnormalities of carotid artery flow velocity noted in human subjects during catheter and pacemaker-induced ventricular tachycardias.

It is well recognized that episodes of seizure, syncope, confusion and agitation can be based on cerebral ischemia secondary to various forms of cardiac arrhythmias. In an earlier report which characterized phasic carotid artery blood flow velocity, we noted that in a few patients, supraventricular and ventricular tachycardias diminished the amplitude of blood velocity wave-forms. We report here in detail the abnormalities of carotid artery flow velocity noted in human subjects during catheter and pacemaker-induced ventricular tachycardias.

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Results
CAROTID ARTERY FLOW VELOCITY DURING SINUS RHYTHM

Carotid artery flow velocity under stable conditions during sinus rhythm has been described previously. Carotid artery flow velocity wave-forms were characterized by a large systolic component followed by a smaller diastolic fraction.

EFFECTS OF CATHETER-INDUCED PAROXYSMS OF VENTRICULAR TACHYCARDIA

During short episodes of irregular ventricular tachycardia, peak systolic and diastolic carotid artery flow velocities varied greatly. When the episode of ventricular tachycardia was preceded by at least two sinus beats, the first beat of the tachycardia resulted in a complete absence of phasic systolic flow velocity in 80% of the cases. Only 50% to 70% of subsequent QRS complexes generated systolic flow velocity waves. There was no direct relationship between preceding R-R interval and peak carotid artery flow velocity during all beats of the tachycardias. The last or next to last beat of the tachycardia usually resulted in greater carotid flow velocity than other beats of the arrhythmia. In general, the lowest peak flow velocities were associated with diminished levels of phasic right ventricular pressure. However, many beats which did not produce phasic carotid flow velocity did result in phasic systolic right ventricular pressures (fig. 1). These relationships also were evident during recording of left ventricular apexcardiograms, with external evidence of forceful left ventricular contraction at times resulting in negligible systolic carotid artery flow velocity (fig. 2). In 50% of cases there was a carotid artery flow velocity “overshoot” during the first few sinus beats following cessation of the tachycardia. The average decline of systolic carotid artery flow velocity resulting from catheter-evoked

![Simultaneous lead II of the electrocardiogram (LH), right ventricular pressure (RV) and carotid artery flow velocity during four short episodes of catheter-induced ventricular tachycardia in a 39-year-old man with mitral insufficiency. Note that only 50% of QRS complexes of the tachycardia are associated with appreciable phasic systolic carotid flow velocity waves. The last or next to last beats of the tachycardias result in greater peak carotid flow velocities when compared with other beats of the arrhythmia. Some beats produce substantial systolic right ventricular pressures yet no phasic systolic flow velocities.](image-url)
ventricular tachycardia in the 19 patients was 50% as shown in figure 3.

Average reduction of peak right carotid flow velocity produced by ventricular tachycardia in the study group. There is a significant (P < 0.001) mean decline of peak flow velocity which averages 50% for the study group. The mean control and tachycardia-related carotid flow velocities (± 1 standard deviation) are indicated.

**FIGURE 2**

Simultaneously recorded lead II of the electrocardiogram (LII), tricuspid area phonocardiogram (TA), right ventricular pressure (RV), left ventricular apexcardiogram (ACG) and right carotid artery flow velocity in a 44-year-old man with coronary artery disease. Note that some beats of the tachycardia result in substantial right ventricular pressures and left ventricular systolic impulses with negligible associated systolic carotid artery flow velocities. (SFW = slow filling wave.)

**FIGURE 3**

Average reduction of peak right carotid flow velocity produced by ventricular tachycardia in the study group. There is a significant (P < 0.001) mean decline of peak flow velocity which averages 50% for the study group. The mean control and tachycardia-related carotid flow velocities (± 1 standard deviation) are indicated.

**INFLUENCE OF RIGHT VENTRICULAR PACING**

Right ventricular pacing at driving rates greater than 120 per minute resulted in a decline of carotid artery flow velocity. There was a high degree of correlation between the right ventricular pacing rate and reduction of peak systolic flow velocity (fig. 4). On the other hand, peak diastolic flow velocity was relatively well maintained over a wide range of pacing rates and only declined by 33% at a rate of 180 per minute. In ten subjects, rapid ventricular pacing resulted in peak carotid artery flow velocity alternation. Of interest was the presence of a period of low amplitude irregular flow velocity for five to ten beats prior to the onset of alternation. Peak diastolic carotid artery flow velocity remained relatively constant throughout these periods of flow velocity alternation (fig. 5). The alternation of carotid artery flow velocity was paralleled by similar changes in simultaneously recorded external carotid pulse tracings (fig. 6). During right ventricular pacing, a single patient manifested periods of alternation which were characterized by a large peak flow velocity preceded and followed by lower peak velocity levels. Furthermore, the two low flow velocity beat amplitudes diminished and increased in a cyclic fashion, unrelated to respiration (fig. 7).

**COMPARISON OF RIGHT ATRIAL AND VENTRICULAR PACING AT IDENTICAL RATES**

In eight subjects tested, the mean peak carotid flow velocities were identical during pacing to 120 beats per minute, although the records were more irregular with ventricular pacing. At rates greater than 140 per minute, the average peak flow velocities were 25% to 50% lower during ventricular pacing when compared...
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Heart rate plotted against percent of control peak carotid flow velocity in a patient subjected to 11 episodes of rapid right ventricular pacing at different driving rates. There is a significant inverse correlation ($r = -0.88$) between heart rate and percent control carotid artery flow velocity.

Discussion

Our results, obtained from human subjects, extend and support the previous demonstration of a 40% to 75% reduction of volumetric internal carotid blood flow in dogs subjected to pharmacologically induced ventricular tachycardia. These reductions of carotid artery flow velocity are probably based on decreased diastolic cycle length, reduced left ventricular filling, and diminished stroke output. Phasic alterations of carotid luminal circumference may, in part, also play a role. Interestingly, initial beats of catheter or pacemaker-induced paroxysmal ventricular tachycardia resulted in minimal carotid blood flow velocity while other beats with equal and shorter preceding diastolic cycle lengths near the end of the tachycardia produced appreciable flow velocities. This phenomenon also has been noted during measurement of peripheral arterial flow in human subjects during ventricular tachycardia and probably represents a form of cardiac potentiation.

The dissociation of right heart pressure and carotid flow velocity can be explained on the basis of recording events dependent on left ventricular dynamics with contralateral ventricular pressure. However, there was a poor correlation at times between carotid flow velocity and the magnitude of the apexcardiographical systolic impulse. Insofar as the systolic amplitude of the apexcardiogram reflects changes of left ventricular stroke volume, there appears to be a carotid flow velocity-left heart stroke output “dissociation” during some beats of ventricular tachycardia. This observation might be based on “functional” aortic insufficiency or lack of aortic valve opening associated with these beats. The “overshoot” of carotid artery flow velocity following bursts of ventricular tachycardia, as described here, has been previously demonstrated in other vascular beds and resembles reactive hyperemia, which occurs after obstruction of muscular phasic arterial flow.

Completely irregular flow velocities noted during shorter catheter-induced paroxysms of ventricular tachycardia can be contrasted with sustained pacemaker-driven episodes of ventricular tachycardia comprised of more than ten beats. During most of these latter episodes, peak carotid artery flow velocity was regular or became irregular in an alternating fashion.

CAROTID ARTERY BLOOD FLOW VELOCITY ALTERNATION

Alternation of both aortic and peripheral arterial blood flow velocities as a consequence of rapid cardiac pacing has been previously noted in man. The carotid artery flow velocity alternans seen here was probably based on alternation of left ventricular end-diastolic fiber length and contractility during tachycardia. Of interest was a five to ten beat low peak flow velocity “warming-up phase” prior to the onset of alternation. Although the mechanism accounting for this pre-alternation period is unknown, it possibly represents the critical time interval necessary for cardiac adjustment to the rapid heart rates.

The “2:1” alternation of carotid artery flow velocity noted in a single subject described here probably represents a transitory state between basal carotid artery flow velocity and classic “high-low” alternation. To our knowledge, this unusual phenomenon has not been previously described in experimental animals or man. In general, it appears that carotid artery flow velocity alternation is a compensatory mechanism whereby peak carotid and other...
Simultaneous lead II of the electrocardiogram (LII), tricuspid area phonocardiogram (TA), right ventricular pressure (RV) and right carotid artery flow velocity recorded during two episodes of pacemaker-induced ventricular tachycardia in a normal 31-year-old man. Irregular low amplitude flow velocity profiles are seen during the first five or six beats of the tachycardias; peak systolic flow velocity alternation then ensues. Peak diastolic flow velocities remain relatively constant during the tachycardias. (PA = right ventricular pacemaker artifact.)

Simultaneously recorded lead II of the electrocardiogram (LII), tricuspid area phonocardiogram (TA), right ventricular pressure, external right carotid artery pulse tracing and right carotid artery blood velocity during right ventricular pacing at a rate of 140 per minute in a 61-year-old woman with primary myocardial disease. There is identical peak systolic alternation on the external carotid pulse and internal right carotid flow velocity recordings. (PA = pacing artifact, 1 = first heart sound, 2 = second heart sound.)
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Simultaneously recorded lead II of the electrocardiogram (LII), tricuspid area phonocardiogram (TA), right ventricular pressure and right carotid artery flow velocity in a normal 31-year-old man during rapid right ventricular pacing. After the eighth beat of the tachycardia, there is the onset of peak flow velocity alternation with a period comprised of two low amplitude beats alternating with a single high amplitude beat ("2:1 alternation"). This was a repetitive phenomenon during longer episodes of cardioacceleration. Note the flow velocity "overshoot" of the first sinus beat following the tachycardia. (PA = pacing artifact.)

Simultaneously recorded lead II of the electrocardiogram, right heart pressures and right carotid artery blood flow velocity in a 50-year-old man with coronary artery disease subjected to right atrial (upper panel) and right ventricular (lower panel) pacing at increasing heart rates. Average peak flow velocities are greater during atrial pacing when compared with ventricular pacing at identical heart rates. Peak flow velocity alternation occurs at a heart rate of 160 per minute during atrial and ventricular pacing. The alternation, however, is more evident during right ventricular pacing. (PA = pacing artifact.)
arterial flow velocities can be partially maintained during rapid heart rates.

ATRIAL CONTRIBUTION TO CAROTID ARTERY FLOW VELOCITY

At heart rates less than 120 per minute, there were no significant differences between peak flow velocities noted during atrial and ventricular pacing. However, at more rapid rates, with shortening of the diastolic filling period, the atrial contribution to ventricular filling, stroke output and peak systolic carotid artery flow velocity was more significant and atrial pacing produced higher peak flow velocity levels. It is noteworthy that atrial tachycardia in experimental animals results in greater volumetric carotid flow than ventricular tachycardia at identical rates.

LIMITATIONS AND ADVANTAGES OF THE METHOD

The Doppler flowmeter catheter utilized in this study measures blood velocity and not volumetric flow. Measurements obtained by means of radial expansion limiting probes placed around peripheral arteries indicate that under most conditions the alterations of arterial flow velocity reflect volumetric flow in these vessels. The qualitative similarities of carotid flow velocities noted during simultaneous catheter and transcutaneous measurement suggest that the latter method may prove useful for noninvasive monitoring of carotid blood velocity under a variety of conditions.

Cerebral vascular resistance, carotid arterial blood pressures and bidirectional blood flow velocities were not measured in this study. The development of a catheter which can accomplish simultaneous estimation of these other important parameters of cerebral blood flow is technically feasible but is not available at the present time. Despite these drawbacks, the flowmeter tip catheter is currently the only device which can directly measure alterations of local carotid artery flow velocity in conscious unanesthetized man. As shown here, ventricular tachycardia results in some unusual and as yet unexplained alterations of carotid artery flow velocity which can only be appreciated by phasic instantaneous measurement.

CLINICAL IMPLICATIONS

The findings described here indicate that ventricular tachycardia can have an adverse effect on carotid artery blood flow velocity. Unexplained episodes of light-headedness and syncope may be based on such arrhythmia-related blood velocity deficits, particularly in subjects with preexisting cerebrovascular disease. The relationship between transient cerebral ischemic attacks and cardiac arrhythmias has recently been stressed. Whether or not the hemic stasis resulting from such diminished flow velocity, in fact, can contribute to the formation of carotid or cerebral arterial thrombosis is currently unknown. In any case, an aggressive approach to the treatment of these arrhythmias seems to be most appropriate.

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