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Non-invasive detection of beat to beat variation in stroke volume during ventricular tachycardia. A case report.

The authors present the case of a 69 years old patient in whom non invasive recordings of Doppler aortic flow-velocity curves, the electrocardiogram and either M-mode echocardiograms of the mitral valve, aortic root or jugular venous pulse were obtained during a spontaneously occurring episode of ventricular tachycardia. Following electrical cardioversion to sinus rhythm, these same parameters were again recorded.

According to the relationship of the S wave to the P wave oil the electrocardiogram during ventricular tachycardia, the ventricular beats were divided into three groups: group 1 - beats preceded by inappropriately timed P waves; group 2 - beats following appropriately timed P waves; group 3 - beats unrelated to P waves.

Appropriately timed atrial contractions were associated with stroke volumes (group 2) considerably larger than the observed when the atrium contracted against closed atrioventricular valve (group 1) or when no P wave was present before the ventricular beat (group 3).

The data suggests that patient's relative absence of symptoms during ventricular tachycardia even in the presence of significant left ventricular disfunction may have been related to the presence of atrio-ventricular dissociation during VT allowing the frequent occurrence of appropriately timed atrial contractions.

Patients with idioventricular rhythms and A-V dissociation may present prominent phasic variations in arterial pressure and stroke work which have been related to alteration in the temporal relationship between atrial and ventricular contractions [1-4]. Using a Doppler catheter-flowmeter system[5], Benchimol and associates were able to demonstrate variations in beat to beat aortic flow velocity often as high as 80% in patients with ventricular tachycardia[6]. Although Benchimol and Desser stated that these variations were unrelated to P-R or R-P intervals [7], it is reasonable to assume that an appropriately timed atrial contraction may significantly augment ventricular filling during ventricular tachycardia. On the other hand, inappropriately timed atrial contractions may be deleterious to ventricular performance. Experimental animal studies suggest that the "cannon" A waves which result from atrial contractions against closed atrioventricular valves impair venous return, therefore lowering the diastolic filling of the ventricle during the next cardiac cycle and consequently reducing both cardiac output and arterial blood pressures [8,9]. During ventricular tachycardia with atrio-ventricular dissociation, atrial contractions may occur at various phases of the cardiac cycle and exert a varied range of hemodynamic effects. We had the opportunity of studying the effect of the timing of atrial contraction on the beat to beat changes in stroke volume in a patient with ventricular tachycardia and atrioventricular dissociation using a pulsed echo-Doppler system.

The patient is a 69 year old which male who was transferred to Mount Sinai Hospital for control of recurrent episodes of ventricular tachycardia, refractory to conventional antiarrhythmic therapy. The patient had a history of arteriosclerotic heart disease sustaining an antero-lateral myocardial infarction three years prior to admission. He made an uneventful recovery and was relatively asymtomatic until approximately one year prior to admission when he noted palpitations. Ventricular tachycardia at a rate of 150 beats per minute was noted on the electrocardiogram and although well tolerated for up to 24 hours without chest

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pain, hypotension or dyspnea, he required cardioversion on several occasions. He has been treated with both Quinidine and Pronestyl but was not well controlled on these medications and he had intolerable side effects.

On admission to this hospital, the patient was in normal sinus rhythm. Blood pressure was 140/70 mm Hg, and the pulse rate was 60 per minute and regular. Physical examination was essentially normal, except for a displaced and paradoxical PMI and a soft S3 at the apex. Laboratory examination was essentially normal. Chest X-ray revealed the heart to be midly enlarged and the lung fields were clear. Electrocardiogram revealed sinus bradycardia at a rate of 55 beats per minute. Significant Q waves were present in leads I, aVL, V2-V6 which was compatible with his previous anterior wall myocardial infarction. Electrophysiological studies were carried out and the tachycardia was diagnosed as ventricular in origin, of the re-entrant type. Cardiac catheterization revealed severe triple vessel disease and a large left ventricular anteroapical aneurism with a severely depressed ejection fraction (EF = 0,30). The end diastolic left ventricular pressure was markedly elevated.

Non-invasive measurements - During a spontaneously occurring episode of ventricular tachycardia, simultaneous recordings of Doppler aortic flow-velocity curves (ATL 500 A Pulsed Echo Doppler - Advanced Technology Laboratories, Seattle, Washington), the electrocardiogram and either M-mode echocardiograms of the mitral valve, aortic root or jugular venous pulse were obtained and recorded on an Irex physiological recorder at a paper speed of 100 mm per second.

Following electrical cardioversion to sinus rhythm, these same parameters were again recorded.

The Doppler transducer was positioned at the suprasternal notch and its ultrasonic beam aimed at the ascending aorta. The settings on the machine were kept constant throughout the exam. Since aortic flow can be derived from velocity by multiplying the integral of aortic blood velocity by the average cross-sectional area of the aorta, the area under the Doppler curve should be proportional to stroke volume for that beat. In our laboratory, Steingart et al.,[10] studying variations in the integrated area under the Doppler flow velocity tracing, have shown a high correlation ("r" values of greater than 90) with variations, in stroke volume measured by electromagnetic flowmeters placed around the ascending aorta of open-chested dogs.

Each Doppler flaw curve was traced over several consecutive beats during ventricular tachycardia and in sinus rhythm after cardioversion using a sonic Graf-pen digitizer. The areas under the flow curves were obtained by integration of these curves using a PDP 11/70 computer.

The nadir of the S wave (the most precisely identifiable feature of the ventricular complex during ventricular tachycardia) was used as a reference point for the measurement of atrio-ventricular intervals. Atrial contractions related to P waves occurring less than 110 ms after an S wave always occurred when the mitral valve was closed and were associated with "cannon". A waves on the jugular venous pulse tracing (fig. 1). These P waves were considered as "inappropriately timed".

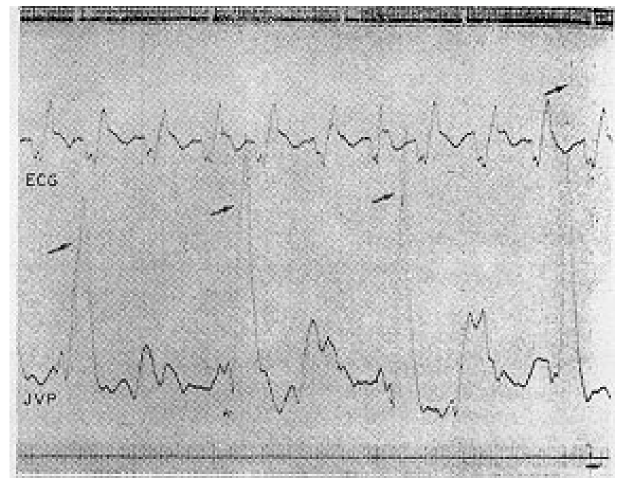

Fig. 1 - "Cannon" A waves during ventricular tachycardia. The upper trace is the ECG and the lower is the jugular venous pulse JVP. The arrows point to large "cannon" A waves.

P waves occurring more than 160 msec after an S wave were assumed to correspond to "appropriately timed" atrial contractions since during this period the mitral valve was always opened. These P waves were associated with significant volume changes in the left atrium, being followed by abrupt posterior movements of the aortic root[11].

P waves occurring between 110 and 160 msec: after an S wave were excluded from analysis (5% of all beats analyzed) since during this time an atrial contraction could find the mitral valve either opened or closed. Consecutive QRS complexes without a P wave in between were categorized as corresponding to "beats unrelated to P waves".

Therefore, according to the relationship of the S wave on the electrocardiogram during ventricular tachycardia, the ventricular beats were divided into three groups (Group I -beats preceded by inappropriately timed P waves and group III - beats unrelated to P waves). Following cardioversion thirty consecutive beats were analyzed in sinus rhythm with less than 10% variation in the integrated areas between beats (fig. 2).

The means of the integrated areas under Doppler velocity curves for these different groups of beats were compared and statistical analysis was performed using the unpaired t-test.

The ventricular and atrial rates during tachycardia were 150 beats per minute and 90 beats per minute, respectively. Following cardioversion, the heart rate was 72 beats per minute. One hundred and fifty six consecutive ventricular beats were analyzed during ventricular tachycardia with less than 10 msec variation in S-S intervals. Eight beats followed

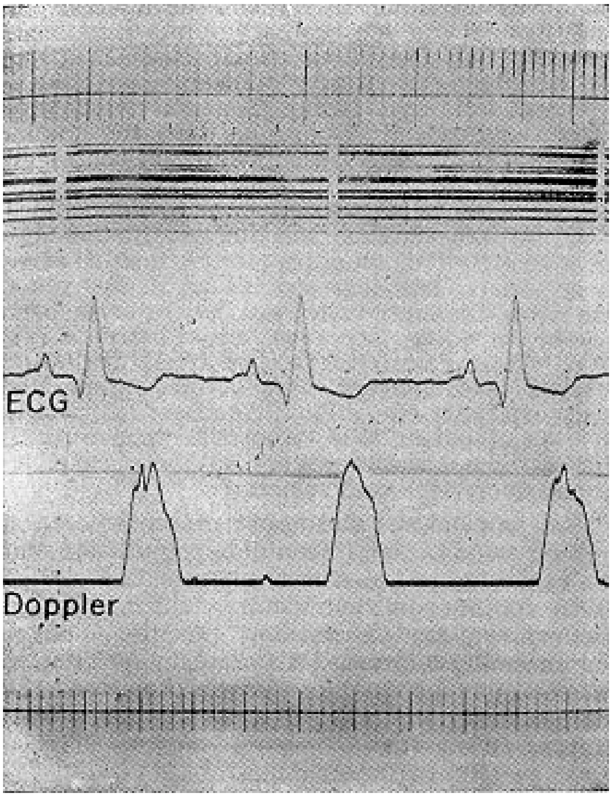

Fig. 2 - Aortic flow velocity during normal sinus rhythm. Upper trace is ECG. Lower trace is Doppler flow curves. Note the similarity of Doppler flow curves with each beat.

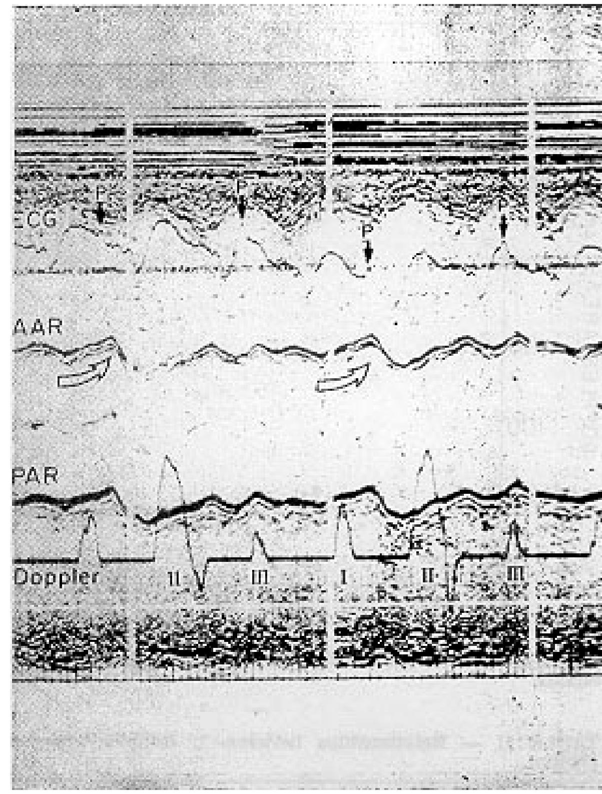

Fig. 3 - Aortic flow during ventricular tachycardia. AAR = anterior aortic root; PAR = posterior aortic root; ECG = ECG trace; Doppler = Aortic flow velocity. P waves are indicated on the ECG and wide arrows point to abrupt posterior motion of AAR in beats following appropriately timed atrial contractions. Note the marked beat to beat variation in aortic flow curves. I = Group I beat, II = Group II beat, III = Group III beat (see text).

P waves that occurred 110-160 msec after the preceding S wave and were therefore excluded. The 148 remaining beats were divided into the 3 groups. Forty were placed on group I, 64 on group II and 44 on group III. During tachycardia prominent beat to beat variations in the areas under the Doppler flow velocity curves were recorded (fig. 3). The results of the integrated areas for the three groups of beats analyzed during ventricular tachycardia and of the beats in sinus rhythm (following cardioversion) are shown in table I.

The area under group II flow velocity curves (beats following appropriately timed atrial contraction) was significantly greater than the area in group I and III (beats preceded by inappropriately timed P waves and unrelated to P waves, respectively) (P<.0001). The under group I flow velocity curves was likewise significantly greater than that of group III (P<. 0001). In sinus rhythm the PR interval was constant at 200 msec. The mean Doppler area in sinus rhythm was significantly greater than the mean areas for any of the three groups (P<.0001).

When group II was further analyzed, there appeared to be a marked decrease in Doppler area when the P S interval was less than 80 msec. The largest areas in this group corresponded to P-S intervals measuring 160 to 220 msec. (fig. 4).

In order to estimate the difference in total card output between ventricular tachycardia (VT) and normal sinus rhythm (NSR) the sum of the areas under Doppler flow curves during VT was determined over a one minute interval and compared to that obtained in NSR post cardioversion for a similar one minute interval (table II). It can be seen that the sum of the areas during VT is approximately 15% less than that in NSR.

Table I - Effect of different P-QRS relationships on Dopples áreas.

	Group I	Group II	Group III	Sinus rhythm
Area under Doppler curves (cm. sec) (mean ± SD)	.0752± .0027	.2260± .0831	.0361± .0021	.3037± .0307

The differences among all groups were significant to P < .0001

Comments

In the normal left ventricle, the atrial contribution to filling is approximately 20 to 35% of the stroke volume [12,13] while in heart disease the atrial contribution may be increased [14-16]. Additionally, at various pacing rates, the atrial contribution to cardiac output has been shown to be of greater significance in patients with heart disease when compared to normals, and this was especially evident at ventricular rates above 120 beats per minute [17].

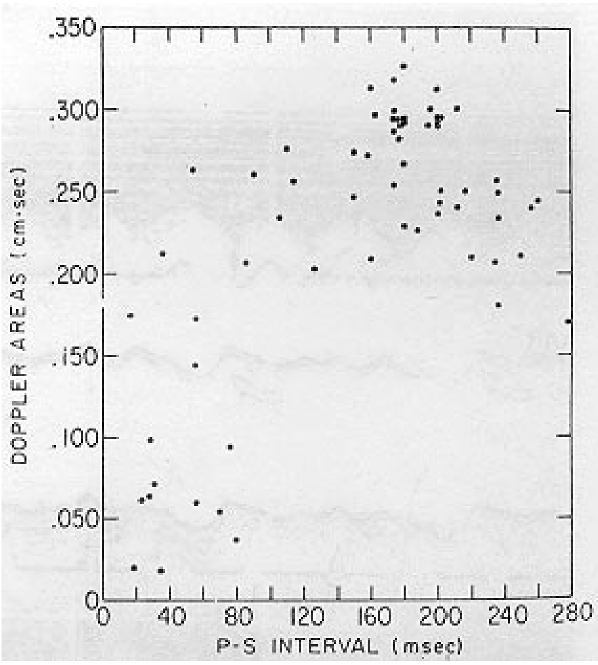

Fig. 4 - Relationship between Doppler areas and P-S intervals.

Table II - Relationships between Σ Doppler areas and rhythm.

	Number of beats	Σ areas (cm. sec)
NSR	75	22,50
VT	148	19,06
Difference		3,44

In the above patient an appropriately timed atrial contraction during VT was associated with stroke volumes considerably larger than the ones observed when the atrium contracted against a closed atrio-ventricular valve or when no P wave was present before the ventricular beat. Although we do not know the exact contribution of his atrial kick in augmenting filling during NSR, it appears to be very important as evidenced by the markedly changing stroke volume in VT. The data also suggests that his relative absence of symptoms during VT even in the presence of significant left ventricular disfunction may have been related to the presence of atrioventricular dissociation during VT, allowing the frequent occurrence of appropriately timed atrial contractions. This is further supported by the fact that the total sum of Doppler flow areas over a one minute period during VT was only 15% less than the sum of areas during one minute of sinus rhythm, i.e. the cardiac output was decreased during VT by only 15% assuming that the area under each Doppler flow curve represents relative stroke volume.

P-S intervals of 160 to 220 msec were associated with the largest stroke volumes. Since the nadir of the S wave was almost 20 msec after the beginning of the QRS complex, the P-S intervals corresponded to P-R intervals of 140 to 200 msec. This interval is consistent with the data of Benchimol and Desser, who found a P-R interval of 200 msec to be the “maximal” ideal for delivering the largest atrial contribution to left ventricular filling [7].

Beats unrelated to P waves (group III) had significantly smaller stroke volume than beats following inappropriately timed P waves (group I). As the vast majority of beats unrelated to P waves followed Group II beats (with large stroke volumes) it is possible that the smaller diastolic volumes of these group III beats (due to greater emptying of the preceding beats) played a more important role in determining small stroke volumes than the postulated deleterious effects of inappropriately timed P waves in group I. The degree of filling of the ventricle after the preceding beat had already been shown to be an important determinant of systolic blood ejection. During atrial fibrillation similar cycle lengths were at times associated with different aortic flow velocities; in particular, peak aortic flow velocities tended to be higher when the preceding beat had produced a relatively low peak velocity [6].

In conclusion, this report presents data on pulsed echo-Doppler techniques to assess beat to beat changes in stroke volume in a patient with spontaneous ventricular tachycardia and atrioventricular dissociation. Despite evidence of markedly depressed left ventricular function at rest, this arrhythmia was well tolerated. The demonstration of enhanced stroke volumes with appropriately timed P waves suggest that on this patient atrio-ventricular dissociation was important in maintaining cardiac output. Further work will be necessary to determine in large series of patients the effects of atrio-ventricular dissociation vs. retrograde atrial activation during ventricular tachycardia, on hemodynamics and clinical manifestations.

Resumo

Os autores relatam os resultados de estudos realizados em paciente do sexo masculino, com 69 anos de idade, portador de disfunção ventricular grave, secundária a cardiopatia isquêmica.

Durante crises espontâneas de taquicardia ventricular registraram-se o eletrocardiograma, o ecograma (modo M) da valva mitral e da raiz da aorta, simultaneamente ao registro da curva de velocidade de fluxo através da raiz da aorta, por método baseado no efeito Doppler. Registraram-se as mesmas variáveis em ritmo sinusal, após cardioversão elétrica.

Dividiram-se os batimentos ventriculares em 3 grupos, de acordo com a relação temporal entre a ocorrência da onda P e do complexo QRS: grupo 1 - batimentos precedidos de sístoles atriais ineficazes; grupo 2 - batimentos precedidos de sístoles atriais eficazes; grupo 3 - batimentos não precedidos de sístoles atriais.

Observam-se extremas diferenças entre os valores do volume sistólico para cada grupo de batimentos, com os maiores volumes sistólicos correspondendo ao grupo II.

A crise de taquicardia ventricular foi bem tolerada pelo paciente, apesar da severa disfunção ventricular, possivelmente em decorrência de ter havido dissociação atrioventricular.

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