Tachycardias in Pregnancy

CHARLES D. JOHNSON, MD, FACC

Pregnancy is associated with an increased incidence of cardiac arrhythmias in women with and without structural heart disease, i.e., pregnancy may have an arrhythmogenic effect. These arrhythmias may be due to the cardiovascular physiology and stress of the condition and to increased sympathetic tone or sensitivity exacerbating preexisting underlying cardiovascular disease, or to *de novo* stimulation of arrhythmias in women without organic heart disease. Thus, the increased propensity for arrhythmias during pregnancy may result from the hemodynamic, autonomic, hormonal and neurohumoral, and emotional changes. The augmented physiologic volume and heart rate too, may play some role.

Tachyarrhythmias during pregnancy comprise: atrial premature contractions (APC's) and ventricular premature beats (PVC's) which are common, sinus tachycardia, various supraventricular tachycardias (SVT's), including atrial fibrillation (AF) and flutter, ventricular tachycardia (VT) and tachycardias associated with the Wolff-Parkinson-White (WPW) syndrome. An arrhythmia during pregnancy may have the potential to increase maternal and fetal morbidity. Moreover, it may cause significant maternal symptomatology (1-5).

The cases of three gravid females in our institution whose pregnancies were complicated by tachycardias are reported.

CASE I

This 34-year-old female, Gr 3 P2, 6 months pregnant, was seen at another hospital because of chest pain, palpitations and shortness of breath. An electrocardiogram (ECG) there revealed a wide-complex tachycardia (WCT). Later, she was referred to the University Hospital and diagnosed as a 34 weeks pregnancy.

Physical examination revealed a grade 1/6 systolic ejection murmur at the 2nd left interspace (IS), a S_3 gallop and no rales. Echocardiograms showed ejection fractions (EF) of 30%, 40% and 50%, hypokinesia, dilatation of the left ventricle and mitral regurgitation. Subsequently, metoprolol 25 mg b.i.d. was prescribed and the patient improved markedly. At 38 weeks gestation she underwent induction and vaginal delivery, and bilateral salpingectomy. The baby was normal, with an uneventful neonatal period.

ECG's are illustrated in figures 1. A subsequent ECG was within normal limits. At 9-weeks post-delivery the patient reported no further symptoms. However, an

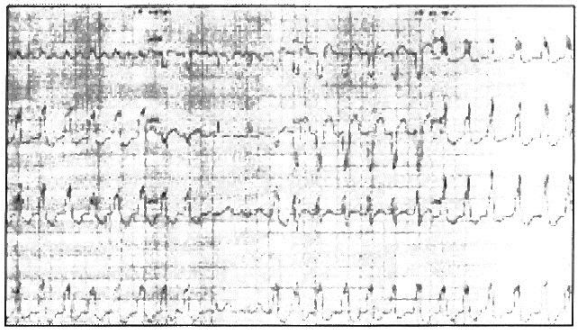

Figure 1 A. WCT. VT, origin right ventricular outflow tract (left bundle branch block-LBBB with a normal vertical axis). Atrioventricular (AV) dissociation. Heart rate (HR) ± 145 bpm.

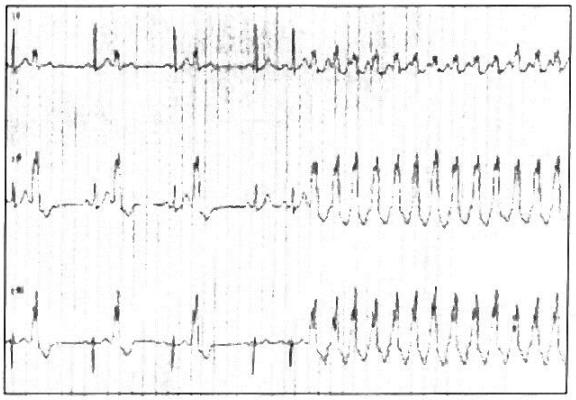

Figure 1B. PVC's in bigeminy on the left, and VT of identical morphology on the right of the illustration. HR= 174 bpm.

From the Department of Medicine, Cardiology Section, University of Puerto Rico, Medical Sciences Campus, San Juan, Puerto Rico

Address for correspondence: Charles D. Johnson, MD, Department of Medicine, Cardiology Section, University of Puerto Rico, Medical Sciences Campus, PO Box 365067, San Juan, Puerto Rico 00936-5067.

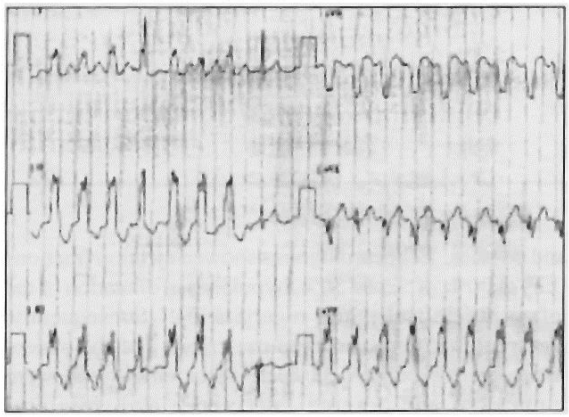

Figure 1C. VT. The fourth beat is a ventricular fusion beat (Dressler beat) and the eighth beat is a sinus captured beat. AV dissociation. HR= 167 bpm left, 162 bpm right.

echocardiogram showed an EF of 47% and left ventricular enlargement (60mm). This case was diagnosed as peripartum cardiomyopathy and VT.

CASE 2

A 22 years old primigravida at 30 2/7 weeks gestation with an 8-days history of occasional shortness of breath with palpitations. The heart rate was 200 bpm, and she was started on verapamil IV, digoxin and propranolol. The rate slowed to 110 bpm, but subsequently the tacycardia recurred, and she was transferred to the University Hospital. A grade 1-2 short systolic murmur was heard at the 2nd left IS. An echocardiogram showed mild left atrial enlargement and 1+ mitral and tricuspid regurgitation. Thyroid tests were normal, the digoxin blood level was subtherapeutic. Propranolol, 40mg q.i.d was started. The patient's ECG's are illustrated in figures 2.

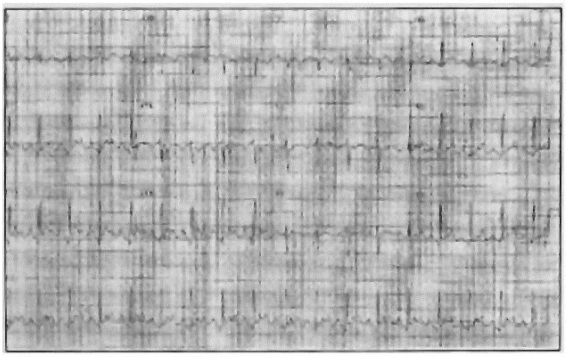

Figure 2 A. SVT. Automatic ectopic atrial tachycardia (AEAT), with 2:1. AV block. Rate: P 210, R 105 bpm. P axis > + 90 degrees.

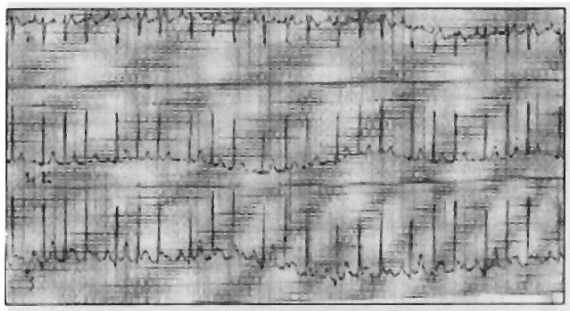

Figure 2 B. AEAT, with 4:3 AV Wenckebach block. P rate 167.

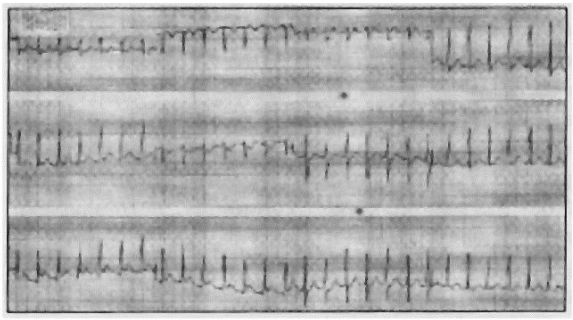

Figure 2 C. AEAT, with 1:1 AV conduction with first-degree AV block. HR 150 bpm.

CASE 3

A 36 years old patient, 7 months pregnant was referred to our institution with palpitations, sweating and lassitude. Past history of a ventricular septal defect repair at the age of 12 years. Her Holter ECG's (Leads II and V_1) are illustrated in figures 3.

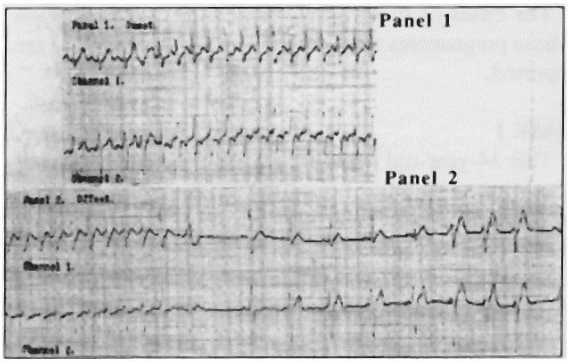

Figure 3 A. Panel 1. The first three beats are minimally preexcited WPW beats. The fourth beat is an APC initiating an Orthodromic AV Reentrant Tachycardia (AVRT), rate 200 bpm. The retrograde r P' wave probably falls in the ST segment.
Panel 2. The tachycardia stops-perhaps one junctional beat-one sinus beat, then concertina beats, followed by one preexcited beat.

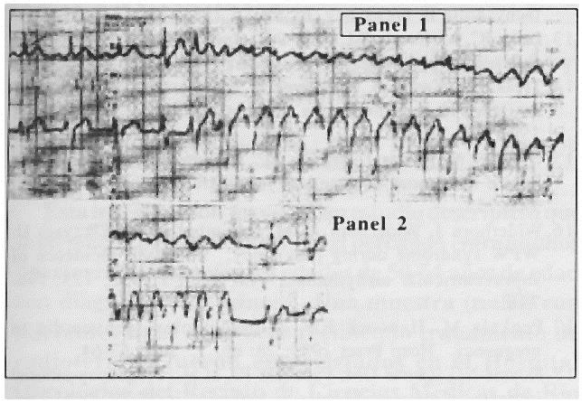

Figure 3 B. Panel 1. The fourth beat is an APC initiating a WCT of differing morphologies, which may be an Antidromic AVRT, HR 188 bpm (however, a preexcited AF and other WCT's associated with the WPW syndrome cannot be excluded without EPS).
Panel 2. The tachycardia, 200-205 bpm, stops, followed by two preexcited fusion beats.

Discussion

Pregnancy is associated with an increased incidence of arrhythmias in women both with and without organic heart disease.

PVC's are common during pregnancy, but frequent or repetitive PVC's are uncommon. VT is uncommon to rare during pregnancy. It can be associated with structural heart disease, including a cardiomyopathy. But generally, VT occurs in the absence of any apparent heart disease (5-10). The most common VT occurring in the patient with a normal heart, whether pregnant or not, originates from the right ventricular outflow tract. It is known as "repetitive monomorphic VT" and is characterized by left bundle branch block morphology and either a normal or right axis deviation (1, 5). VT may also originate from the left ventricular septal region.

Brodsky et al in 1992 (7), found 23 reports of pregnant women with ventricular tachyarrhythmias. The vast majority of these women had no underlying structural heart disease. None had peripartum cardiomyopathy, which is typically associated with congestive heart failure and only rarely with ventricular arrhythmias.

These idiopathic VT's may be adenosine-sensitive and generally they are catecholamine-sensitive. The pathogenetic mechanism may be cyclic AMP-mediated triggered activity as a result of delayed afterdepolarizations-automaticity. Ventricular tachycardia in pregnancy is likely the result of increased sensitivity to catecholamines in patients already predisposed to this condition.

Maternal VT has been reported in association with hypomagnesemia (11). The electrocardiogram in this case also showed a LBBB plus RAD pattern.

Therapy

Beta-adrenergic blockers, such as metoprolol and propranolol, are first-line therapy for idiopathic VT and have been highly effective and generally safe in this type of VT in pregnancy. This beneficial effect derives probably by reducing the myocardial responsiveness to catecholamines (1, 5-7). Case 1 with VT responded readily to low dose ß-blocker therapy.

SVT's are relatively common during pregnancy, especially in the third trimester. Of the major SVT's, typical atrioventricular nodal reentry tachycardia (AVNRT) is the most common. Ectopic atrial tachycardia (Case 2) in pregnancy appears to be uncommon. Ectopic atrial SVT is characteristically persistent and refractory to treatment, but it may respond to propranolol (1, 3, 5, 12-14).
Pregnant patients with the WPW preexcitation syndrome (with accessory bypass tracts) are the second most common cause for paroxysmal SVT's, accounting for 20-30% of these (1, 15).

It has been suggested that pregnancy may predispose asymptomatic patient with the WPW syndrome toward exhibiting supraventricular tachycardias (1, 5, 15-17). Recently, Kounis and associates confirmed an increased propensity for SVT in pregnant women with the WPW syndrome (18). They considered that the increased plasma volume, stress and anxiety and the increased adrenergic sensitivity by estrogens during pregnancy were some of the causative factors.

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