

cal prolonged QT syndrome is strongly promoted by excessive adrenergic influence, such as might have arisen from the patient's septic, dehydrated state.³ Intravenous adenosine possesses a significant short-lived antiadrenergic effect.⁶ However, in this case any potential beneficial anti adrenergic effect was overridden by adenosine-induced bradycardia. Moreover, there is the indication that the antiadrenergic effect of adenosine would favorably rectify the sympathetic imbalance and dispersion of repolarizations

associated with adrenergic-dependent prolonged QT syndrome.³ An effect or role of adenosine in the mechanism of early afterdepolarizations has not been established. In summary, extreme caution should be used when administering intravenous adenosine and other agents that promote bradycardia in the presence of a prolonged QT interval. Unlike the present case, this situation is most likely to arise during the concomitant use of antiarrhythmic drugs known to prolong the QT interval.³ Intravenous adenosine should not be administered in the presence of established prolonged QT syndrome.

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WPW syndrome during pregnancy: Increased incidence of supraventricular arrhythmias

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Some authors^{1,2} have suggested that the incidence of supraventricular tachyarrhythmias (SVT) is increased dur-

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ing pregnancy in otherwise healthy women. Other reports^{3,4} have indicated that pregnancy may also predispose asymptomatic patients with preexcitation to supraventricular tachyarrhythmias. We report three clinical patients with Wolff-Parkinson-White (WPW) syndrome in whom the incidence of tachyarrhythmias increased during pregnancy.

Patient No. 1. A 21-year-old woman was first admitted to LAC+USC Medical Center during her thirty-ninth week of pregnancy with severe palpitations, lightheadedness, and near syncope. The patient's medical history was traceable to March 1986, when she sought medical advice for chest pain, exertional dyspnea, lightheadedness, and a syncopal episode that occurred while she was taking a sauna. A complete evaluation was performed and revealed subaortic membranous stenosis with a gradient across the left ventricular outflow tract of 65 mm Hg. The patient had electrocardiographic (ECG) evidence of preexcitation; however, no arrhythmia could be documented. The symptoms were attributed to the subaortic membranous stenosis and she was referred for surgery. The subaortic membrane was surgically resected and the gradient decreased postoperatively to 16 mm Hg. During follow-up she was relatively asymptomatic until 1987, when she complained of a syncopal and a near syncopal episode occurring during emotional upset. A 24-hour ambulatory ECG monitoring failed to confirm any arrhythmias. Subsequently, with the exception of occasional "sensations" in her chest, she was asymptomatic until November 1989, when she became pregnant. During her pregnancy, the patient experienced four syncopal episodes and several near syncopal episodes. She also reported having a substantial increase in the frequency of episodes of "fluttering in her chest" associated with lightheadedness, which lasted from a few seconds to several minutes. Twenty-four-hour ambulatory ECG monitoring performed during the third trimester showed preexcitation and 20 runs of SVT, with the longest run lasting 194 beats at a rate of 170 beats/min. The maximum SVT rate was 242 beats/min. The patient entered in her diary several episodes of dizziness and palpitations; these episodes were associated with SVT with rates ranging from 220 to 242 beats/min. During this admission, the patient was monitored in the cardiac intensive care unit where she continued to have short runs of SVT associated with palpitations. The patient was started on quinidine and atenolol, with complete suppression of arrhythmia and relief of symptoms. Forty-eight hours later she left the hospital against medical advice. Subsequently, she stopped the medications, and 3 weeks later (at 42 weeks of pregnancy), she delivered a 3080 gm female neonate with an Apgar score of 8 and 9 at 1 and 5 minutes. Postpartum, the patient left California and was lost to follow-up.

Patient No. 2. A 31-year-old woman, gravida 3, para 2, was evaluated for severe episodes of palpitations that started during her twenty-sixth week of pregnancy. The episodes were sudden in onset and termination, lasting from a few minutes to half an hour, and were associated with irregular pounding in her chest, dizziness, dyspnea,

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and an aching pain in the back of her neck. The frequency of episodes were approximately once a week. Her past medical history was significant for preexcitation discovered incidentally 5 years before during a routine preoperative examination for retinal detachment repair. Before her third pregnancy, the patient was completely asymptomatic. Her family history was significant for preexcitation observed in her first son. There was a family history of sudden death. Her cardiac evaluation revealed mild mitral and tricuspid regurgitation but no valve prolapse. Twenty-four-hour ambulatory ECG monitoring showed 123 runs of atrial tachycardia, with the longest run being 50 beats. The maximum SVT rate was 182 beats/min. Transtelephonic monitoring documented atrial fibrillation, with the shortest R-R interval being 260 msec. The patient was given procainamide (slow release, 3 gm/day), with complete relief of symptomatology. The patient delivered at term a 4607 gm male infant with an Apgar score of 7 and 8 at 1 and 5 minutes. The infant's ECG did not reveal preexcitation. Postpartum, procainamide was discontinued and the patient remained free of any symptoms up to 8 months offollow-up.

Patient No.3. A 21-year-old obese woman with a history of asymptomatic preexcitation presented to an emergency room at an outside hospital with severe palpitations and chest pain. Her ECG showed orthodromic atrioventricular reciprocating tachycardia with a heart rate of 200 beats/min. The SVT was terminated with intravenous verapamil. Subsequently during her pregnancy, she had four other episodes of severe symptomatic SVT requiring treatment with intravenous verapamil in the emergency room. Between these severe attacks she had frequent episodes of palpitations lasting up to 30 minutes. The patient was treated with oral verapamil, 80 mg during the attacks, and with atenolol, 25 mg/day. At 39 1/2 weeks of pregnancy, she was admitted at LAC+USC Medical Center with a diagnosis of pregnancy-induced hypertension, preeclampsia, and amnionitis. Three days later, she delivered a healthy 3500 gm male baby with an Apgar score of 7 and 9 at 1 and 5 minutes. Five days after delivery, the patient had another SVT attack that was treated successfully with verapamil. During the following 2 1/2-year period after pregnancy, in which she did not take any antiarrhythmic drugs, she had only three episodes of palpitations that terminated spontaneously.

Comments. The incidence of pre excitation in the normal population ranges from 0.01 % to 0.3 %. The incidence of paroxysmal tachycardias in the young adult population with preexcitation is approximately 10%, and it increases with age up to 36%.⁵ The exact incidence of WPW syndrome during pregnancy is not known, but some reports^{3,4} have indicated that pregnancy may facilitate the onset of tachyarrhythmias in patients with previous asymptomatic pre excitation. Gleicher et al.³ in a report of three patients, suggested that pregnancy may predispose asymptomatic patients with preexcitation to tachyarrhythmias. McKenna et al.⁴ described a 27-year-old pregnant woman with WPW syndrome who had her attacks of tachyarrhythmias (atrial fibrillation and reentrant atrioventricular tachycardia) during two subsequent pregnancies. Before her first pregnancy and during the interval between the next pregnancy, she was asymptomatic. In the present report, patient No.1 had intermittent symptoms before her pregnancy. However, during pregnancy she experienced an increase in the frequency and severity of her SVT paroxysms. Patient No.2 experienced her first attack during the twenty-sixth week of her third pregnancy. She denied any symptoms before her third pregnancy and became totally asymptomatic after the pregnancy. The absence of symptoms during her previous two pregnancies is of interest. It is possible that "short runs of tachyarrhythmias may have been overlooked because of the lack of symptoms; on the other hand, she was older during the third pregnancy, which may lead to an increased incidence of tachyarrhythmias.⁵ Patient No.3 was completely asymptomatic before her pregnancy. During gestation she had numerous episodes of palpitations and five documented symptomatic SVT paroxysms requiring treatment in the emergency room. Following pregnancy, she had only three episodes of palpitations over a period of 2 1/2 years.

An increased incidence of episodes of paroxysmal SVT during pregnancy has also been observed in an obstetric population without preexcitation.^{1,2} Szekely and Snaith¹ reported on six pregnant women in whom paroxysmal SVT occurred only during pregnancy. Mendelson² summarized 82 cases of paroxysmal SVTs during pregnancy. Some patients experienced their first attack during pregnancy, while others with a history of prepregnancy arrhythmias usually had an increase in the frequency, duration, and severity of episodes. More recently, Panja et al.⁶ observed four cases of atrial tachycardia that developed during pregnancy. Hubbard et al.⁷ reported another case of atrial tachycardia that occurred only during pregnancy.

Several hypothetical mechanisms have been invoked to explain the increased propensity for arrhythmias during pregnancy. These include hemodynamic, autonomic, hormonal, and emotional changes occurring in the pregnant women. The physiologic volume overload occurring during pregnancy results in an increased left ventricular enddiastolic volume and may lead to an increased myocardial irritability. Heart rate increases steadily during pregnancy, and sinus tachycardia is often seen in normal pregnant women during the third trimester and at term. An accelerated heart rate may promote cardiac arrhythmias by modifying the effective refractory period, velocity of conduction, and spatial dispersion of refractoriness.⁸ An elevated heart rate in patients with WPW syndrome may induce unidirectional block in the reentrant circuit and start atrioventricular reciprocating tachycardias.

Hormonal changes are thought to play an important role in the development of arrhythmias during pregnancy.³ Estrogens increase the excitability and the frequency of action potentials in susceptible tissues such as uterine muscle fiber. The blood levels and the metabolism of catecholamines do not change significantly during pregnancy. However, Roberts et al.⁹ and Metz et al.¹⁰ have shown that

estrogens increase the number of α -adrenergic receptors and adrenergic sensitivity in susceptible tissues (myometrium, platelet). In other tissues (i.e., hypothalamus) β -adrenergic receptors are increased. Therefore sex hormones modulate receptors of hormones and neurotransmitters in various tissues; the consequent autonomic changes and the increased adrenergic sensitivity may potentially play a role in the genesis of arrhythmias by modifying the refractory periods and conduction velocity in the reentrant circuit.⁸ During normal pregnancy, women experience major physical and psychological changes that often require adjustments in relationships and life-styles. These changes lead to various degrees of stress and anxiety.¹² Anxiety about health of the fetus and fears that harm may come to the fetus are almost universal. Stress, anxiety, and fear activate the pituitary-adrenal axis and stimulate the sympathetic nervous system, with potential arrhythmogenic effects.⁸ In summary, it appears that there is an increased propensity for supraventricular tachyarrhythmias in the pregnant population with and without preexcitation. Therefore further prospective studies are needed to clearly document this increased arrhythmia susceptibility during pregnancy, the magnitude of the problem, mechanisms, and the preferred therapeutic approach in these patients.

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Unexpected PR prolongation following nonconducted atrial extrasystoles: A manifestation of concealed reentry

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A supraventricular impulse occurring after a long pause is often associated with improved atrioventricular (A V) conduction. This is because the long pause facilitates complete recovery of the A V junction in such a way that the ensuing sinus impulse, which is relatively late, finds the A V node in a lesser degree of refractoriness when compared with earlier impulses. The phenomenon is evident, for example, in type 1 (Wenckebach) second-degree A V block, where the impulse after the pause occasioned by the block is associated with the shortest PR interval. We report a case in which sinus impulses occurring after pauses, as a result of nonconducted atrial extrasystoles, are associated with an unexpected worsening of A V conduction, resulting in marked prolongation of the PR interval.

The ECG in Fig. 1 (noncontinuous strip of lead VI) was obtained from a 71-year-old man who had had a myocardial infarction at the age of 65 years and was referred for evaluation of effort angina. The patient was taking no medication except transdermal nitroglycerin. The tracing reflects the following: (1) First-degree A V block, which is manifested, for example, by the first six beats in the bottom strip, the PR interval of which measures 0.28 second; (2) one episode of second-degree type 1 (Wenckebach) A V block with a 3:2 conduction ratio (top strip); (3) nonconducted atrial extrasystoles labeled with dots; (4) the first sinus impulse after any nonconducted atrial extrasystole reflects a longer than expected PR interval. For example, in the middle strip a nonconducted atrial extrasystole occurs after two sinus complexes. The ensuing sinus impulse is associated with a PR interval of 0.36 second, which is markedly longer than the PR intervals of both the subsequent and previous conducted sinus impulses.

Worsening of A V conduction after a long pause is an uncommon and unexpected phenomenon. The pause in itself, however, does not appear to be responsible for prolongation of the ensuing PR interval. This is revealed by the fact that the pause occasioned by an episode of Wenckebach A V block (top strip) is followed as expected by a relatively short PR interval of 0.26 second. It is therefore likely that the extrasystole rather than the pause is responsible for prolongation of the PR interval in the subsequent beat. An

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