

# Arrhythmias in Pregnancy

- one case report and current recommendations from a cardiological perspective -

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## Abstract

*We present a case of atrial fibrillation in a pregnant woman (known with Wolff-Parkinson-White syndrome) treated with direct current cardioversion. The risk of arrhythmia in pregnancy is related to fetal hypoperfusion, with possible teratogenic effect. Termination of atrial fibrillation episodes must always be attempted in pregnancy. Also, for acute treatment of supraventricular tachycardia during pregnancy we can use vagal maneuvers, adenosine or metoprolol.*

**Keywords:** WPW syndrome, supraventricular arrhythmia, radiofrequency ablation

Wolff-Parkinson-White (WPW) syndrome is a cardiac abnormality characterised by the presence of abnormal conductive tissue between the atria and the ventricles that is often associated with frequent episodes of supraventricular tachycardia. We report the case of a 31 years old woman known with WPW syndrome being pregnant - gestational age 6 weeks. She came in emergency room with a high cardiac rate (aprox 250 bpm, irregular- diagnostic: atrial fibrillation with very high ventricular rate), pale, and with low blood pressure (70 mmHg systolic).

Although the patient had countless more arrhythmic episodes in the past (starting from childhood, probably all supraventricular tachycardia, most of them short lasting), she has no active prophylactic therapy. Also she has never been offered the option for radiofrequency ablation before the pregnancy. This is the method of choice for curative results, but must be done before pregnancy because of significant radiation exposure. In this case direct current (DC) cardioversion was performed with prompt restoration of sinus rhythm. Because transient fetal arrhythmia has been reported it was suggested that DC cardioversion should be done with fetal rhythm monitoring. This was not the case however due to small age pregnancy. Now the patient is stable and asymptomatic (without antiarrhythmic treatment) and the course of pregnancy was normal, with normal foetal development (a recent ultrasound scan was normal).

It was a case of extreme tachycardia due to atrial fibrillation in a patient with an accessory pathway. The treatment decision was taken because of the haemodynamic compromise. In all forms of sustained tachycardia which are not tolerated haemodynamically

(mother's cerebral and systemic hypoperfusion resulting in fetal hypoperfusion), emergency DC cardioversion must be performed. We can use drug therapy only in cases with good haemodynamic status and cardioversion if drug treatment fails or is not tolerated. The risk of arrhythmia in pregnancy is related to fetal hypoperfusion, with possible teratogenic effect.

Termination of atrial fibrillation episodes must always be attempted in pregnancy because anticoagulants - which are teratogenic in the first trimester - should be avoided. We consider that the patient has no indication of prophylactic antiarrhythmic treatment (with limited efficacy in patients with accessory pathway). Future management of the patient will be offering the catheter ablation procedure. The patient is now stable and asymptomatic and her pregnancy is within normal limits.

The incidence and severity of tachyarrhythmias may increase during pregnancy because of autonomic and haemodynamic changes related to pregnancy and of the patient's increased awareness. The concern for fetus' safety is a complicating factor during treatment. Teratogenic risk is the highest in the first eight weeks. Premature atrial beats are noticed in about half of pregnancies but they are generally well tolerated<sup>[1]</sup>. Sustained arrhythmias are rare (2-3 per 1000 patients), but with frequent symptomatic exacerbation. The major concern is the potential adverse effects on fetus (especially in the first 8 weeks) since antiarrhythmic drugs cross the placental barrier. Physiological changes in pregnancy can affect bioavailability of drugs so careful monitoring and dose adjustments are necessary.

1a

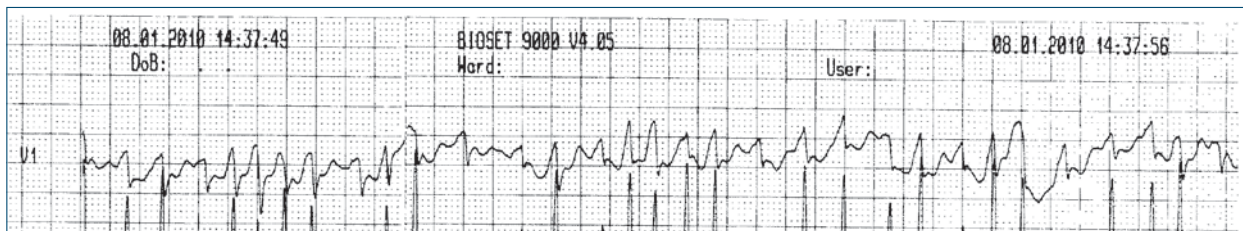
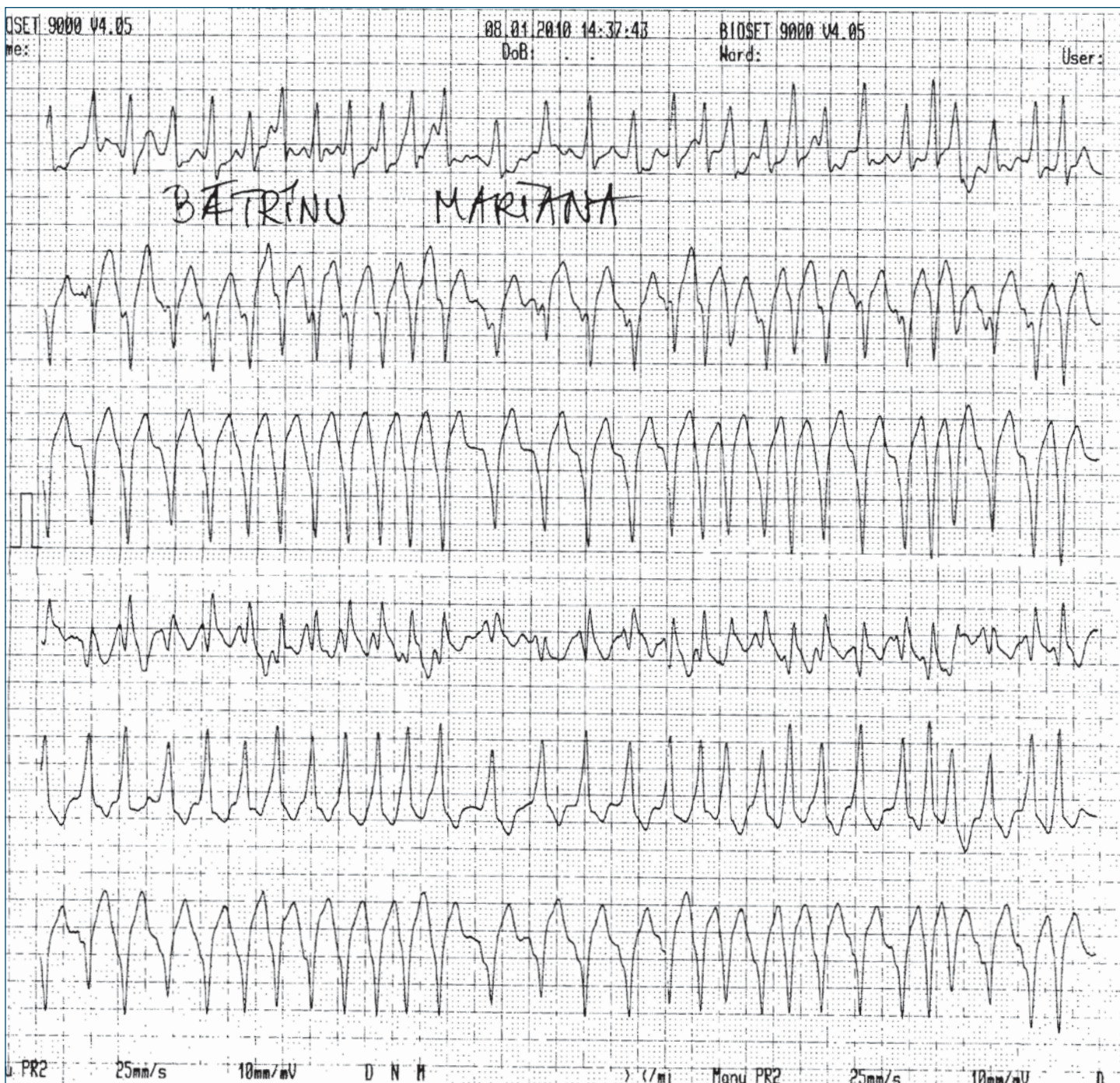
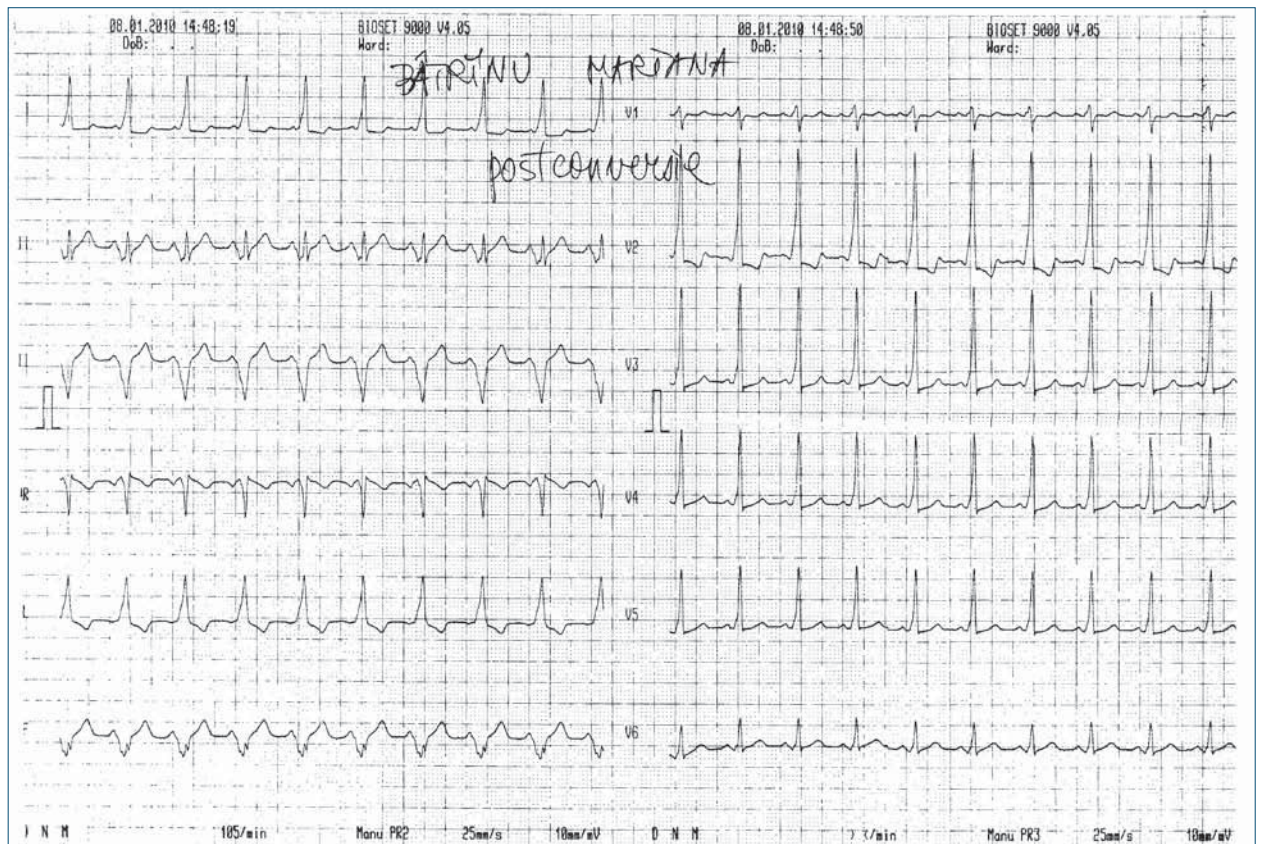


Figure 1 a, b. ECG at presentation in the emergency room, ventricular fibrillation with very rapid pace

1b



**Figure 2.**  
Postconversion  
ECG typical  
for WPW



**Table 1**

Treatment recommendations for supraventricular tachycardia in pregnancy <sup>(1)</sup> adapted)

<b>Acute conversion</b>	Vagal maneuver Adenosine DC cardioversion Metoprolol
<b>Prophylactic therapy</b>	Digoxin Metoprolol (if possible, not in the first trimester)

**Table 2**

Safety of some antiarrhythmic drugs in pregnancy <sup>(2)</sup> adapted)

Antiarrhythmic drug	FDA classification
Class IA - Quinidine	C
Class IA - Quinidine	C
Class II - Metoprolol	C
Class III - Sotalol - Amiodarone	B D
Class IV - Verapamil	C
Digoxin	C
Adenosine	C

Women with known symptomatic tachyarrhythmias should be recommended catheter ablation before pregnancy. In patients with mild symptoms and normal hearts no medical treatment is required. If the symptoms are intolerable or if hemodynamic compromise occurs, antiarrhythmic drug therapy can be used. For acute conversion of atrioventricular node-dependent tachycardias (the most frequent in clinical practice, including patients with Wolf-Parkinson-White syndrome) the first-line treatment should be vagal maneuvers and, if they do not work, intravenous adenosine. If not curative, we can use intravenous beta-blockers (mainly metoprolol). If they all fail or when hemodynamic compromise occurs, DC cardioversion has to be used. Emergency and elective DC cardioversion is safe in all the stages of pregnancy because the risk of inducing fetal arrhythmias is small<sup>(2)</sup>. The amount of electric current reaching the fetus is insignificant; besides, the fetus has a high fibrillation threshold.

For prophylactic therapy we can use digoxin (one of the safest antiarrhythmics, but its efficacy has never been demonstrated) or metoprolol (best avoided in the first trimester). Digoxin can be used for atrial flutter and fibrillation, with no adverse effects on the fetus. Digoxin crosses the placenta; its levels must be monitored during pregnancy as maternal toxicity can be dangerous to the fetus<sup>(3)</sup>. Cardioselective

beta-blockers are theoretically preferable because they interfere less with vasodilatation and uterine relaxation. The potential for intrauterine growth retardation has been reported for propranolol and atenolol, especially when given in the first trimester. All beta-blockers cross the placenta. From the evidence available to date, beta-blockers do not seem to be teratogenic following exposure in the first trimester. Some studies report an increased risk of intrauterine growth restriction (IUGR), particularly with longer treatment and higher doses; however, the underlying maternal disease may explain this. Theoretically, IUGR could also be mediated by lowering blood sugar with a beta-receptor blockade. However, postnatal growth and development do not seem to be affected<sup>[3]</sup>.

The experience with propafenone is very limited, although no adverse effects on the fetus have been

Table 3

Abbreviated FDA pregnancy risk classification

<b>Category A</b>	Controlled studies show no risk
<b>Category B</b>	No evidence of risk
<b>Category C</b>	No studies in pregnancy
<b>Category D</b>	Positive evidence of risk

reported. The use of amiodarone should be restricted to life threatening arrhythmias. Amiodarone contains high levels of iodine and can lead to congenital goitre or hypothyroidism; thus it should not be a first-line drug in pregnancy. Sotalol has been used for both maternal and fetal arrhythmias; it may cause IUGR, so serial ultrasound is indicated<sup>[3]</sup>. ■

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