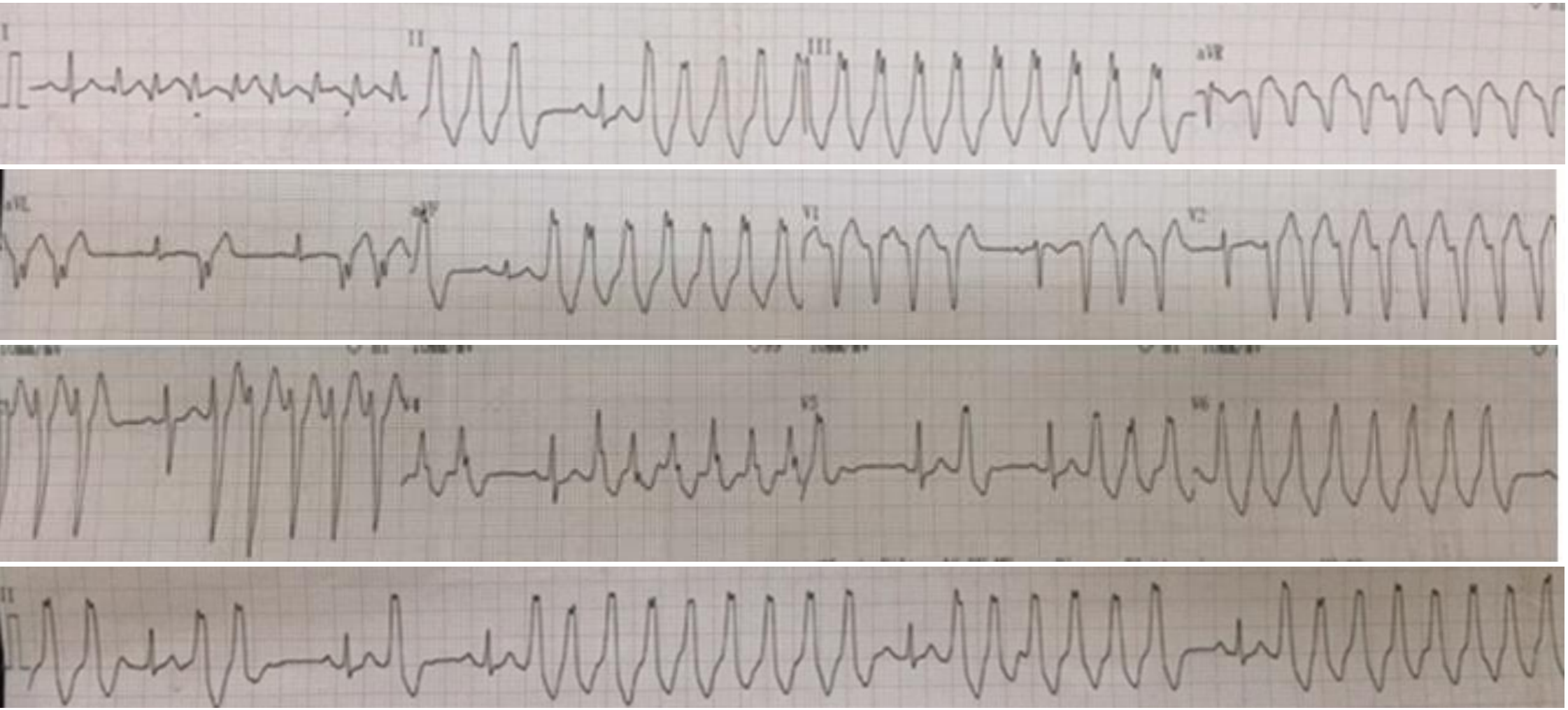
Non-sustained wide QRS complex tachycardia in pregnant woman

Case from Dr. Oscar Peralta Inga, Peru

Tracing during the event

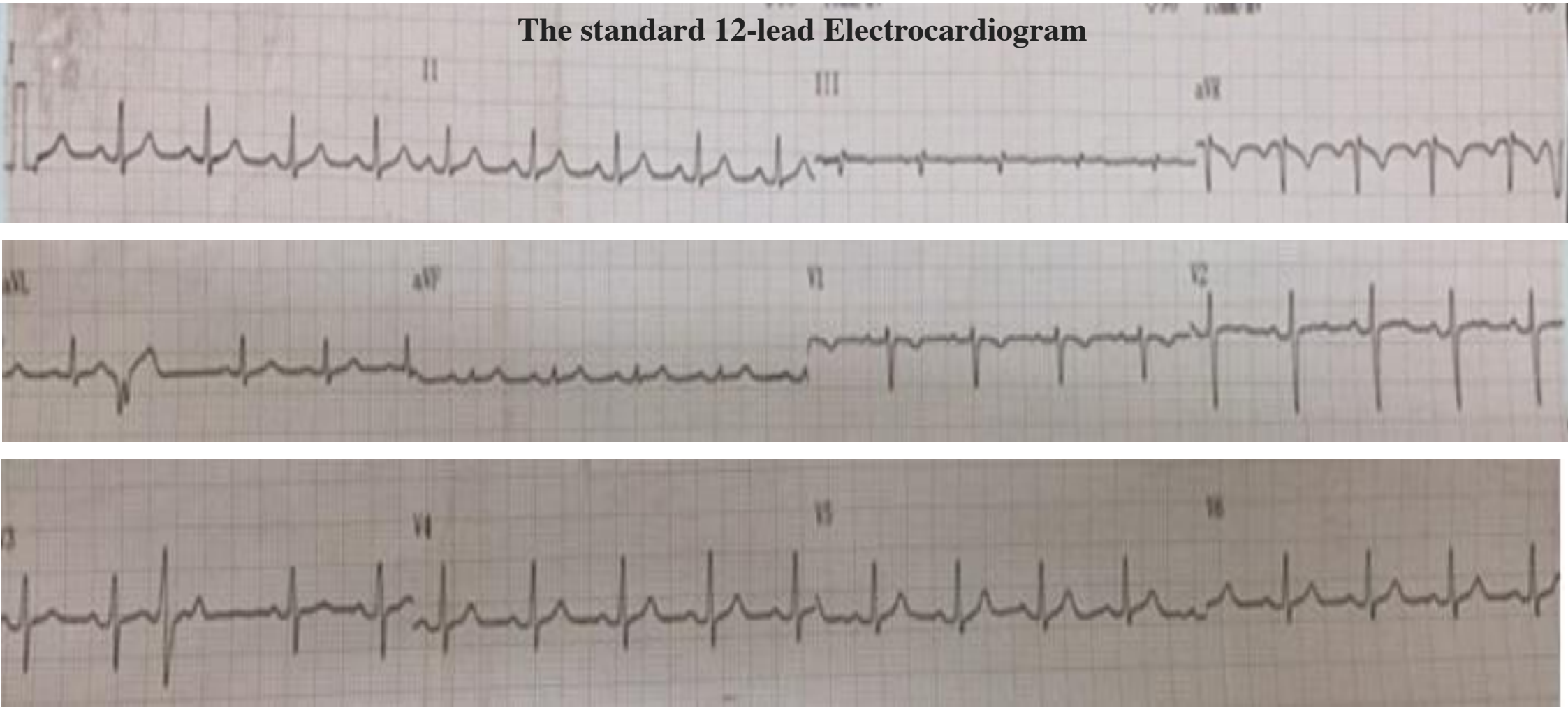


Clinical diagnosis: 26 yo pregnant women with 32 weeks (without hemodynamic compromise).

Which is the diagnosis?

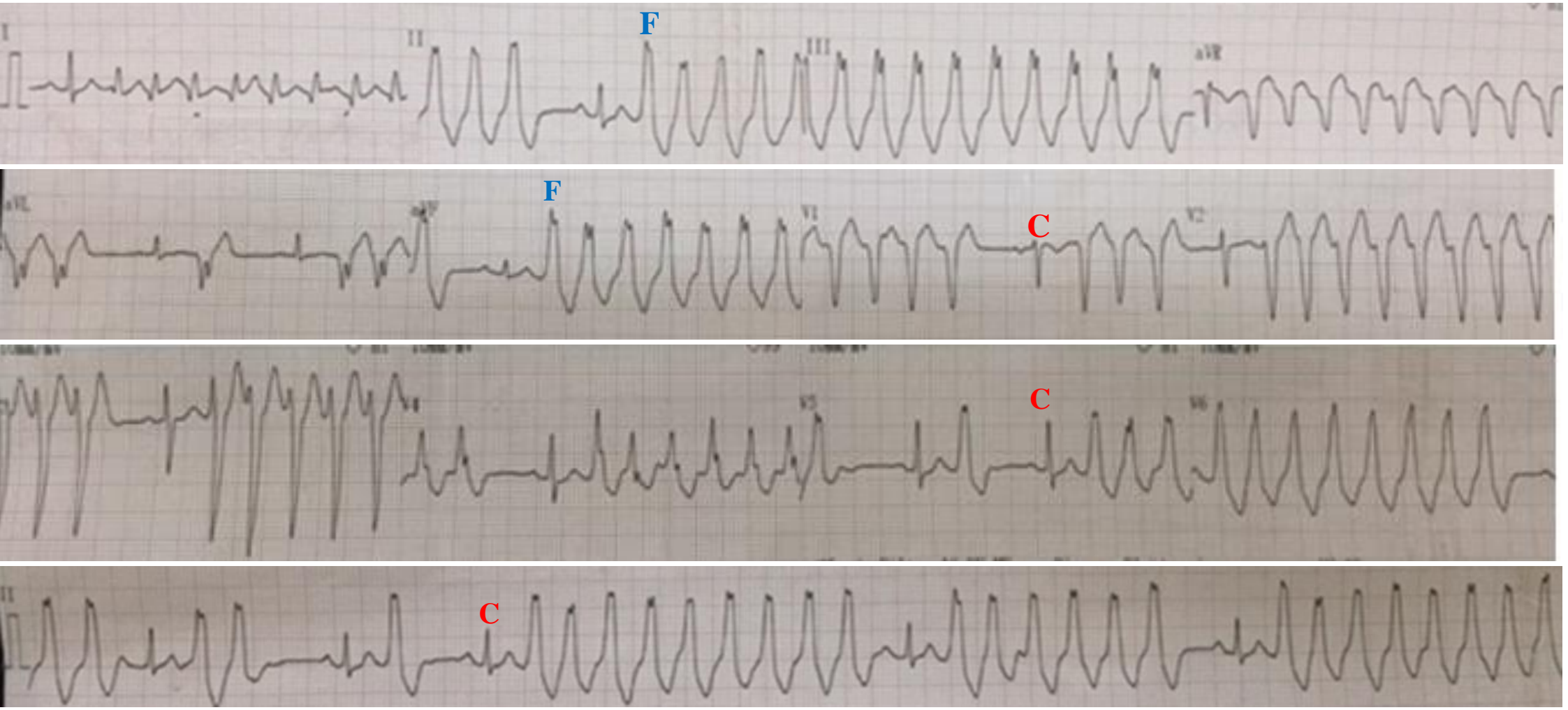
Which is the appropriate approach in this pregnant woman?

The standard 12-lead Electrocardiogram

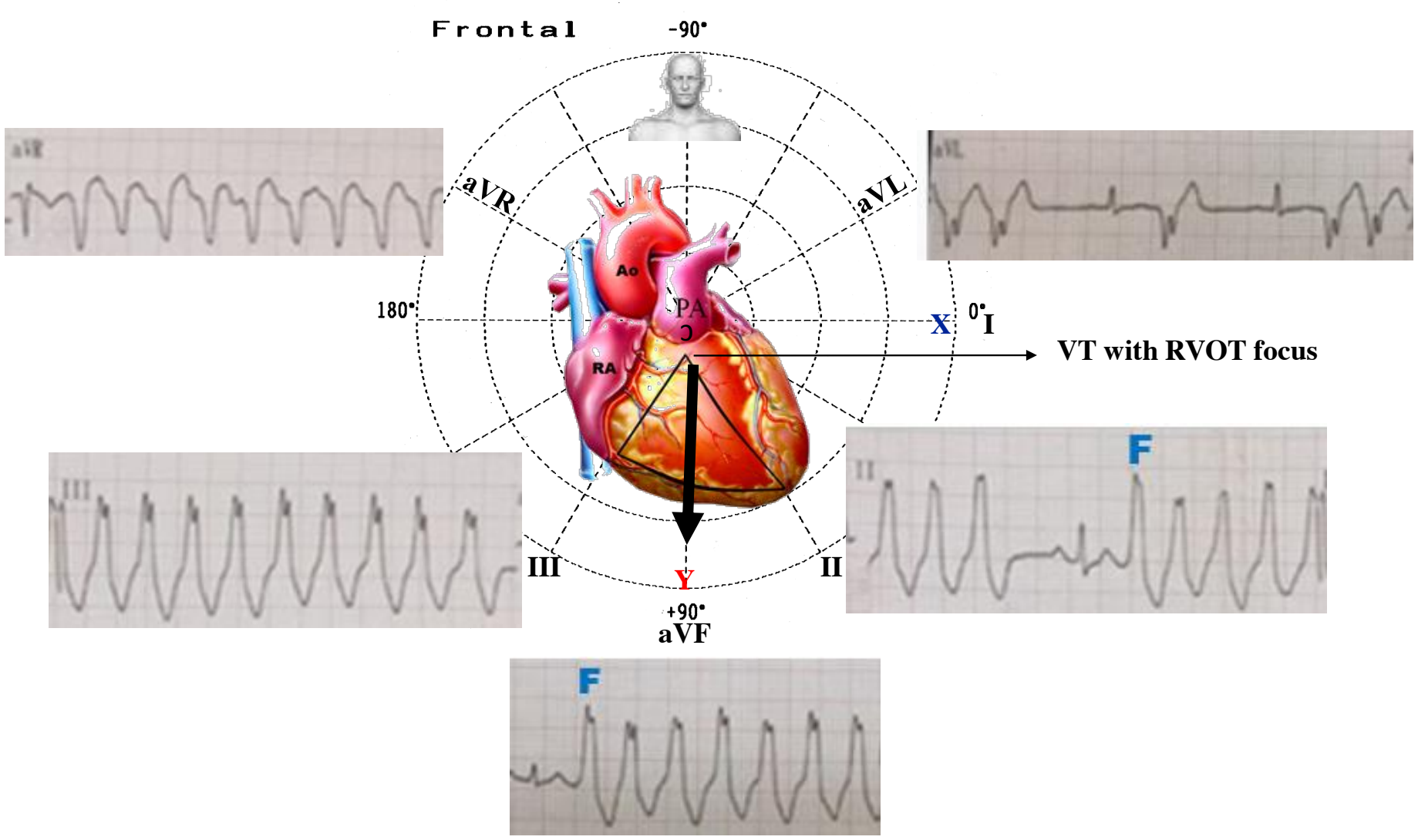


Stardard 12-lead basal ECG

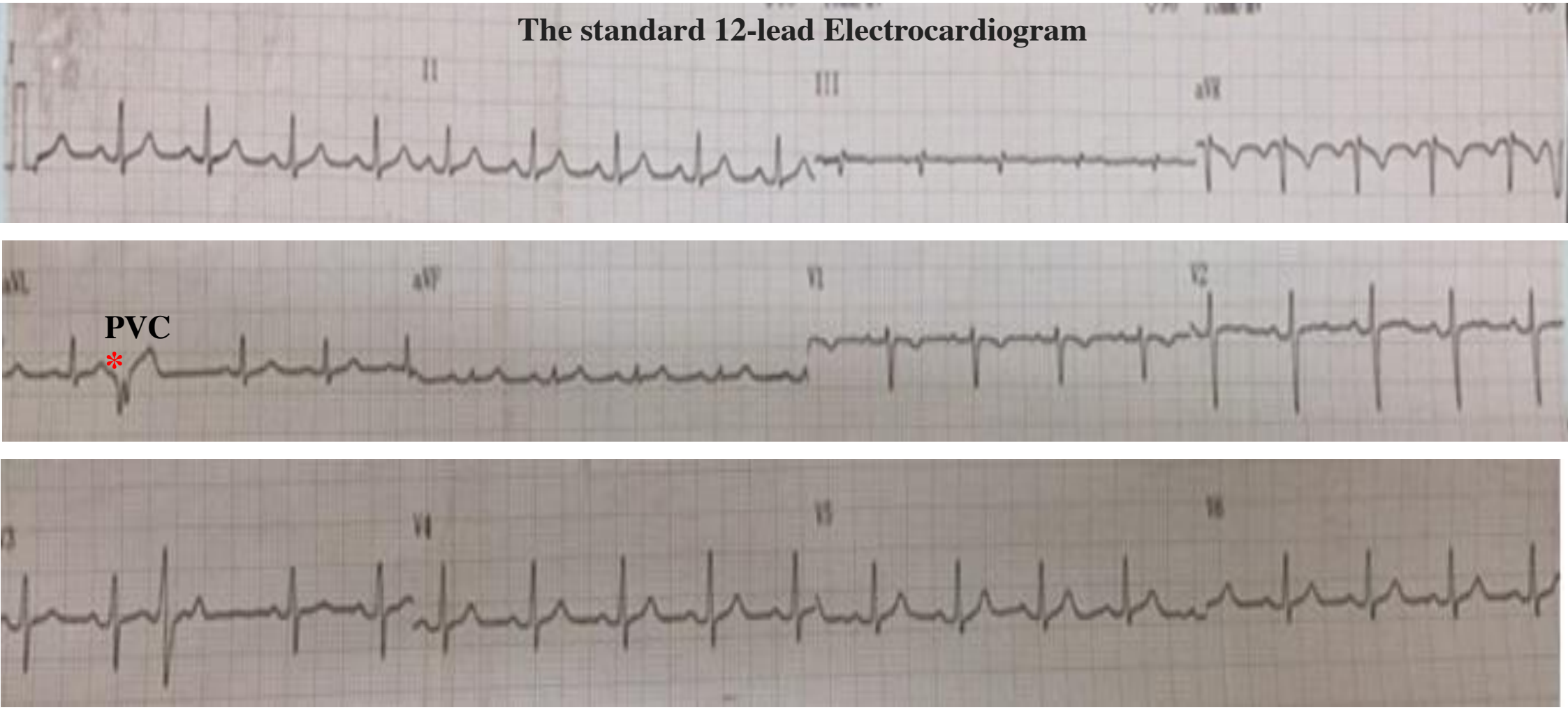
Which is the clinical and ECG diagnosis during the event and basal?
Which is the appropriate approach in this pregnant woman?



Clinical diagnosis: 26 yo pregnant women with 32 weeks (without hemodynamic compromise) **ECG diagnosis:** Nonsustained repetitive monomorphic ventricular tachycardia (NSMVT) with typical left bundle branch (LBBB) pattern from Right ventricular outflow tract (RVOT). Frequent premature ventricular contractions(PVCs), capture (**C**) and fusion beats(**F**) Positive QRS complexes in inferior leads and negative in aVR and aVL: RVOT focus. Recognition of this type of VT, has important practical value: we must know how to distinguish idiopathic VT from supraventricular tachycardia with aberrancy (SVT-A) and VT of the ARVC/D since treatment will be very different.



Positive QRS complexes in inferior leads II, III and aVF and negative in aVR and aVL: RVOT



Base ECG only isolated ventricular premature beats with a short coupling interval(coupling interval less than 400 ms), and SVPC. Absence of inverted T waves in right precordial leads (only V1), RV conduction delay(parietal block), epsilon wave on leads V1-V2. QRS duration of right precordial leads (V1-V2 V3) = left precordial leads(V4-V5-V6).

Outflow tract ventricular tachycardia's (OT VT) comprise a subgroup of idiopathic VT that are predominantly localized in and around the right and left ventricular outflow tracts. OT VT are the most common form of idiopathic VTs and originate, in more than 80-90% of cases, from the RVOT. They manifest at a relatively early age (30-50 years, range, 6 to 80 years such us this woman) with equal distribution between sexes in left ventricular outflow tract VT (LVOT VT) whereas RVOT VT is more common in females. The typical presentation of these arrhythmias consists of: NS repetitive MVT. This is the most common form (60-90%). It is characterized by frequent PVC, right ventricular couplets and salvos of non sustained ventricular tachycardia (NSVT) with left bundle branch block morphology and inferior QRS axis. These extrasystoles occur more often during the day than at night, at rest or following a period of exercise and are transiently suppressed by sinus tachycardia. They may diminish or disappear with exercise during stress testing. Paroxysmal, exercise-induced sustained VT. This VT may be initiated during exercise or recovery. Exercise stress testing is frequently uses to initiate and evaluate RVOT VT, but is not clinically helpful in most cases. Most patients present with palpitations, less frequently with dizziness and a minority of patients present syncope. Initial evaluation must include, Structurally normal hearts, ECG and echocardiogram are usually normal, but MRI may show abnormalities of the RV in up to 70% of patients, including focal thinning, diminished systolic wall thickening and abnormal wall motion.

Origin in the RVOT/LVOT (common embryonic origin). RVOT VT should be distinguished from ARVC/D. VT in ARVC/D may have morphologic features similar to RVOT VT but does not terminate with adenosine. In ARVC/D, the ECG typically shows inverted T waves in right precordial leads and when present, RV conduction delay(parietal block) with an epsilon wave, best seen in leads V1-V2. The differential diagnosis of RVOT VT also includes tachyarrhytmias associated with atriofascicular fibers (Mahain fibers) and VT occurring in patients after repair of Fallot's tetralogy. ECG recognition. RVOT VT is associated with a characteristic ECG morphology of LBBB with inferior axis such us this cases. Anterior sites in the RVOT shows a dominant Q-wave or a qR complex in lead I and a QS complex in aVL. Pacing at the posterior sites produce a dominant R-wave in lead I, QS or R-wave in aVL and an early precordial transition (R/S = 1 by V3).

LVOT VT is suggested by LBBB morphology with inferior axis with small R-waves in V1 and early precordial transition (R/S = 1 by V2 or V3) or RBBB morphology with inferior axis and presence of S-wave in V6. Aortic sinus cusp origin is sometimes difficult to differentiate from RVOT VT because both are so close to each other. Coronary cusp origin has to be though when we fail an ablation in the RVOT , ECG shows a LBBB inferior axis morphology with taller monophasic R-waves in inferior leads and an early precordial R-wave transition by V2-V3. A broader R-wave duration and a taller R/S wave amplitude in V1-V2 favored VT arising from the aortic cusp.

Source Josep Brugada.

NSVT was an independent predictor of mortality, but ventricular couplets appeared to be equally predictive (1). Couplets and/or NSVT were detected in 62.7% of the study population, with a 50.8% mortality rate. The remaining 37.3%, without couplets or NSVT, had a lower mortality rate of 26.3%. The sensitivity of NSVT in relationship to SCD or total death varies among several studies, ranging from 31% to 71% (2,3). The positive predictive value is low, ranging from 20% to 50%, although the negative predictive value has been cited as 72% to 93%. There is a long history of intervention trials designed to reduce mortality in high-risk patients with PVCs or NSVT. The CAST Trial was a groundbreaking, double-blind, randomized study that demonstrated that suppression of PVCs and NSVT markers of risk are not necessarily appropriate targets for therapeutic interventions. Randomized, controlled trials have used NSVT, often documented by AECG, to identify patients who should undergo EPS and further treatment if VT was inducible (4,5). These studies showed significant 50% to 60% reductions in mortality in the ICD-treated groups, but intervention was based on EPS. PVCs and NSVT. Although the AECG can reliably record the presence of PVCs and NSVT, the day-to-day reproducibility of the frequency of these arrhythmias is poor (≥2). In the 1970s and 1980s, observational studies demonstrated that PVCs (≥ 10 or more PVCs per hour) and NSVT as recorded by an AECG in post-MI patients were risk factors for subsequent mortality (6,7).

1. *Doval H.C., Nul D.R., Grancelli H.O.;GESICA-GEMA Investigators Nonsustained ventricular tachycardia in severe heart failure. Independent marker of increased mortality due to sudden death. Circulation. 94 1996:3198-3203.*
2. *Crawford M.H., Bernstein S.J., Deedwania P.C.; ACC/AHA Guidelines for Ambulatory Electrocardiography. A report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (Committee to Revise the Guidelines for Ambulatory Electrocardiography). J Am Coll Cardiol. 34 1999:912-948.*
3. *Holmes J., Kubo S.H., Cody R.J., Kligfield P.; Arrhythmias in ischemic and nonischemic dilated cardiomyopathy: prediction of mortality by ambulatory electrocardiography. Am J Cardiol. 55 1985:146-151.*
4. *Moss A.J., Hall W.J., Cannom D.S.;Multicenter Automatic Defibrillator Implantation Trial Investigators Improved survival with an implanted defibrillator in patients with coronary disease at high risk for ventricular arrhythmia. N Engl J Med. 335 1996:1933-1940.*
5. *Buxton A.E., Lee K.L., Fisher J.D., Josephson M.E., Prystowsky E.N., Hafley G.;Multicenter Unsustained Tachycardia Trial Investigators. A randomized study of the prevention of sudden death in patients with coronary artery disease. N Engl J Med. 341 1999:1882-1890.*
6. *Bigger J.T. Jr., Fleiss J.L., Kleiger R., Miller J.P., Rolnitzky L.M.; The relationships among ventricular arrhythmias, left ventricular dysfunction, and mortality in the 2 years after myocardial infarction. Circulation. 69 1984:250-258.*
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Data suggest that ectopy beyond 10 PVCs per hour does not convey a further increase in risk (1). It has also been suggested that PVCs are an independent predictor of mortality, whereas NSVT may not be a predictor. The initial studies described patients without reperfusion, but a similar relationship has been observed (although with somewhat reduced risk) in the era of thrombolysis and acute reperfusion. In GISSI-2 study (2), mortality was 5.5% at 6 months for patients with > PVCs per hour compared with 2% in those with less frequent PVC. The positive predictive value of PVCs after MI for predicting cardiac arrhythmic events or death generally ranges from 5% to 15%, with a negative predictive value of 90% or more (3). When combined with reduction of LVEF, PVCs becomes a stronger risk factor for mortality. In the EMIAT TRIAL, among postinfarction patients with LVEF ≤40%, mortality was higher in patients with frequent or complex arrhythmias on AECG than in those without (20% versus 10%) (4). Patients with nonischemic cardiomyopathy are at increased risk of SCD and frequently have high-grade PVCs and NSVT (5); however, the relationship between arrhythmias on AECG and cardiac arrest is much less clear than in the case of ischemic cardiomyopathy (3). Observational trials make up the majority of data available, and NSVT is used more commonly than PVCs for risk stratification, likely in relation to the high frequency of VPBs in this population. The GESICA trial, which included a majority of patients with nonischemic cardiomyopathy, confirmed the prevalence of ventricular arrhythmias on AECG in patients with HF and LVEF ≤35% (6). Patients with nonischemic cardiomyopathy are at increased risk of SCD and frequently have high-grade PVCs and NSVT (5); however, the relationship between arrhythmias on AECG and cardiac arrest is much less clear than in the case of ischemic cardiomyopathy (3).

1. *Denes P., Gillis A.M., Pawitan Y., Kammerling J.M., Wilhelmsen L., Salerno D.M.;CAST Investigators. Prevalence, characteristics and significance of ventricular premature complexes and ventricular tachycardia detected by 24-hour continuous electrocardiographic recording in the Cardiac Arrhythmia Suppression Trial. Am J Cardiol. 68 1991:887-896.*
2. *Maggioni A.P., Zuanetti G., Franzosi M.G.; Prevalence and prognostic significance of ventricular arrhythmias after acute myocardial infarction in the fibrinolytic era. GISSI-2 results. Circulation. 87 1993:312-322.*
3. *Crawford M.H., Bernstein S.J., Deedwania P.C.; ACC/AHA Guidelines for Ambulatory Electrocardiography. A report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (Committee to Revise the Guidelines for Ambulatory Electrocardiography). J Am Coll Cardiol. 34 1999:912-948.*
4. *Julian D.G., Camm A.J., Frangin G.;European Myocardial Infarct Amiodarone Trial Investigators Randomised trial of effect of amiodarone on mortality in patients with left-ventricular dysfunction after recent myocardial infarction: EMIAT. Lancet. 349 1997:667-674.*
5. *Kron J., Hart M., Schual-Berke S., Niles N.R., Hosenpud J.D., McAnulty J.H.; Idiopathic dilated cardiomyopathy. Role of programmed electrical stimulation and Holter monitoring in predicting those at risk of sudden death. Chest. 93 1988:85-90.*
6. *Doval H.C., Nul D.R., Grancelli H.O., Perrone S.V., Bortman G.R., Curiel R.;Grupo de Estudio de la Sobrevida en la Insuficiencia Cardiaca en Argentina (GESICA). Randomised trial of low-dose amiodarone in severe congestive heart failure. Lancet. 344 1994:493-498.*

Observational trials make up the majority of data available, and NSVT is used more commonly than PVCs for risk stratification, likely in relation to the high frequency of VPBs in this population. The Gruppo de Estudio de la Sobrevida en la Insuficiencia Cardiaca en Argentina (GESICA) trial, which included a majority of patients with nonischemic cardiomyopathy, confirmed the prevalence of ventricular arrhythmias on AECG in patients with HF and LVEF ≤35% (4).

In patients with nonischemic cardiomyopathy and CHF, LVEF ≤35%, and ventricular arrhythmias (NSVT or an average of 10 or more PVCs per hour), DEFINITE demonstrated a trend toward improvement in overall survival and a reduction in arrhythmic events with ICD therapy. The mortality rate of the non-ICD group was 7% per year, but no comparison group of patients without ventricular arrhythmias was reported.

Conclusions There is abundant information linking the detection of ventricular arrhythmias (PVCs, NSVT) on AECG in post-MI patients with LV dysfunction for risk assessment for SCD. Use of the AECG in this setting has been classified as a class IIb recommendation (1); however, the incremental risk stratification provided by this finding in patients with LVEF ≤35% is unclear (2). On the other hand, patients with LVEF between 35% and 40% may warrant AECG recording to assess for NSVT, because this group has been shown to benefit from an ICD if VT is induced at electrophysiological study. Patients with preserved left ventricular function after MI are generally at low risk, and current data suggest that they would not benefit from undergoing risk stratification with AECG recording. Finally, in patients with dilated cardiomyopathy, DEFINITE (3) required the presence of PVCs or NSVT on AECG, whereas SCD-HeFT (2) did not; thus, the utility of AECG for risk stratification in this population remains unclear.

1. *Crawford M.H., Bernstein S.J., Deedwania P.C.; ACC/AHA Guidelines for Ambulatory Electrocardiography. A report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (Committee to Revise the Guidelines for Ambulatory Electrocardiography). J Am Coll Cardiol. 34 1999:912-948.*
2. *Bardy G.H., Lee K.L., Mark D.B.;Sudden Cardiac Death in Heart Failure Trial (SCD-HeFT) Investigators Amiodarone or an implantable cardioverter-defibrillator for congestive heart failure. N Engl J Med. 352 2005:225-237.*
3. *Kadish A., Dyer A., Daubert J.P.;Defibrillators in Non-Ischemic Cardiomyopathy Treatment Evaluation (DEFINITE) Investigators Prophylactic defibrillator implantation in patients with nonischemic dilated cardiomyopathy. N Engl J Med. 350 2004:2151-2158.*
4. *Doval H.C., Nul D.R., Grancelli H.O., Perrone S.V., Bortman G.R., Curiel R.;Grupo de Estudio de la Sobrevida en la Insuficiencia Cardiaca en Argentina (GESICA). Randomised trial of low-dose amiodarone in severe congestive heart failure. Lancet. 344 1994:493-498.*

Treatment

The decision to treat patients with OT VT depends on frequency and severity of symptoms. Treatment options include medical therapy vs radiofrequency catheter ablation(RFCA).

Acute termination of RVOT VT, can be achieved by vagal maneuver or adenosine (6 mg until 24 mg). Intravenous verapamil (10 mg given over 1 min) is an alternative if the patient has adequate blood pressure. These drugs may suppress triggered rhythms. Cases of hemodynamic instability warrant emergent cardioversion. Chronic management. Long term treatment options include medical therapy and catheter ablation. Medical therapy may be indicated in patients with mild to moderate symptoms. They include beta-blockers, verapamil, diltiazem (rate of efficacy of 20 to 50%)6,7. Alternative therapy include class IA, IC and III agents. RFCA has cure rates of 90% with a recurrence rate of 5% (mainly in the first year). It is the treatment of choice for patients with symptomatic, drug refractory VT, drug intolerance or do not desire long-term drug therapy and it should be strongly considered for the following patients with a potentially malignant form of OT VT:

A) history of syncope;
B) very fast VT;
C) VPCs with a short coupling interval.

Cardiac arrhythmia as a complication of pregnancy can be problematic to maternal health and fetal life and development. RFCA of tachyarrhythmias during pregnancy has been successfully performed in selected patients with limited experience. Techniques to limit maternal and fetal radiation exposure, including intracardiac echo and electroanatomic mapping systems, are particularly important in this setting. Catheter ablations in pregnancy are indicated only in the presence of an unstable tachycardia that cannot be controlled by antiarrhythmic agents.

Specific accommodations are necessary in the care of the gravid patient during catheter ablation.

RFCA in pregnancy are indicated only in the presence of an unstable tachyarrhythmias that cannot be controlled by antiarrhythmic agents. (1)

RFCA can be performed safely with no or minimal radiation exposure during pregnancy. In the setting of malignant, drug-resistant arrhythmia, ablation may be considered as a therapeutic option in selected cases.(2) RFCA must be done without X-ray exposure and the mean fluoroscopy time 42 +/- 37 seconds.

1. *Raman AS, Sharma S, Hariharan R. Minimal Use of Fluoroscopy to Reduce Fetal Radiation Exposure during Radiofrequency Catheter Ablation of Maternal Supraventricular Tachycardia. Tex Heart Inst J. 2015 Apr 1;42 (2):152-4.*
2. *Szumowski L, Szufladowicz E, Orczykowski M, Bodalski R, Derejko P, Przybylski A, Urbanek P, Kuśmierczyk M, Koźluk E, Sacher F, Sanders P, Dangel J,Haissaguerre M, Walczak F. Ablation of severe drug-resistant tachyarrhythmia during pregnancy. J Cardiovasc Electrophysiol. 2010 Aug 1;21(8):877-82.*