

7. Nursing

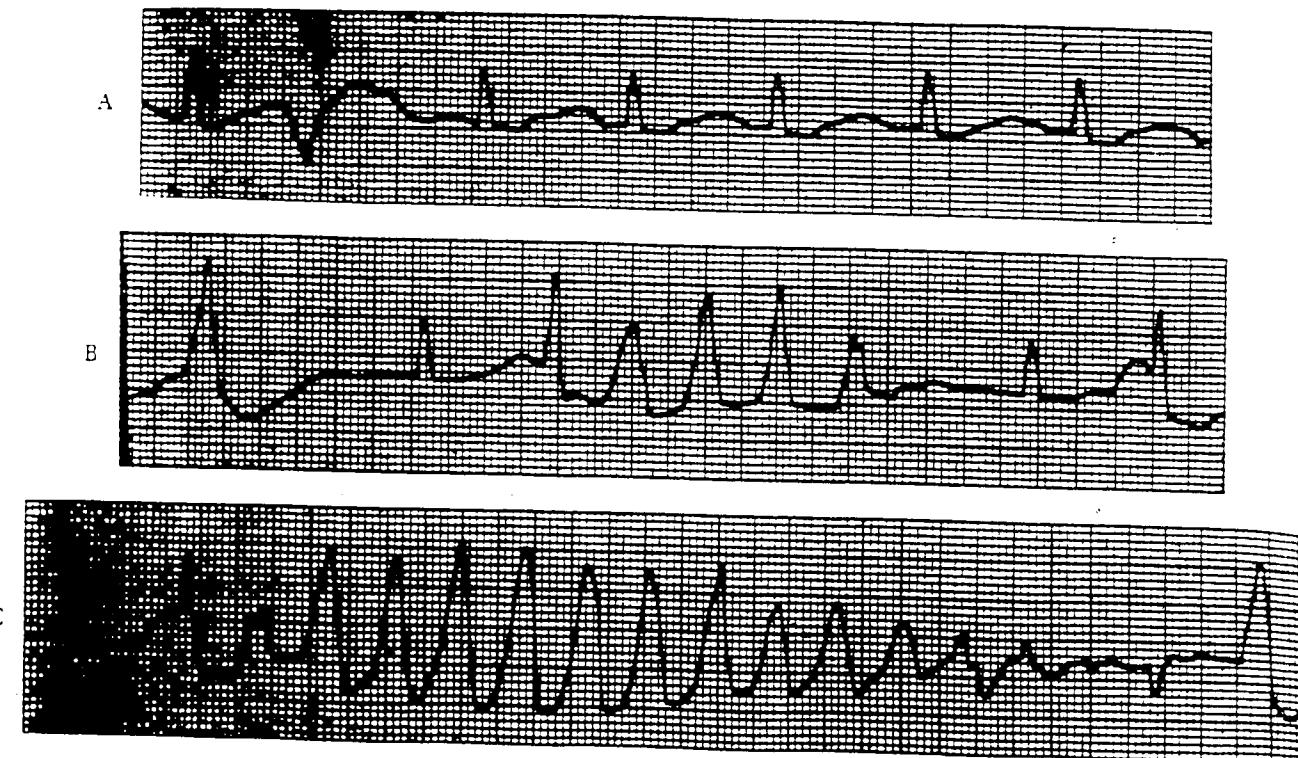
- a. Assess the patient - verify the rhythm as V-fib (make sure it's not a loose lead)
- b. Precordial thump if observed
- c. Get help, notify MD stat
- d. ACLS or defibrillate if skill verified by institution
- e. Code

F. Torsade de Pointes - singular episode
Torsades de Pointes - plural episodes

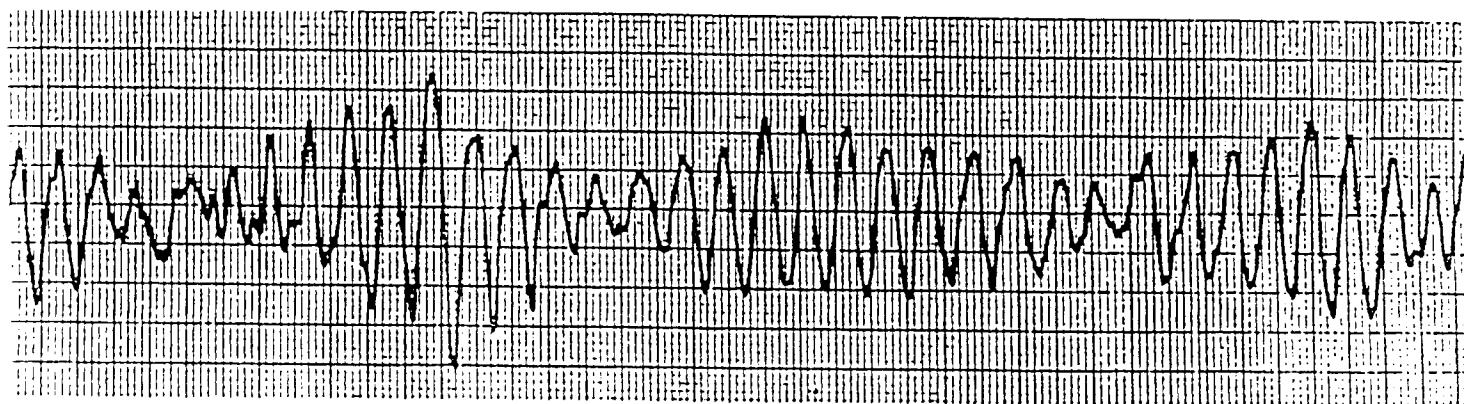
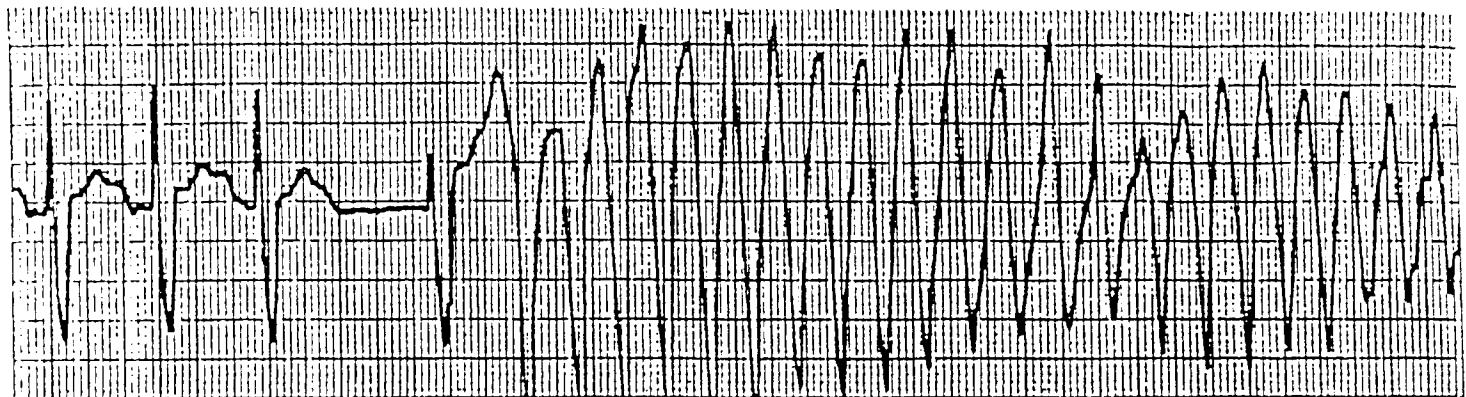
1. Polymorphous but organized V tach often mistaken for V fib
2. Occurs against background of
 - a. Prolonged QT intervals
 - b. Ventricular bigeminy with long coupling intervals
 - c. Certain precipitating causes and therapeutic behavior
3. Mechanism: Unknown, but theories include
 - a. Reentry
 - b. Multiple completing activation sites
 - c. Increased dispersion of repolarization associated with certain conditions that cause long QT intervals. (Example: hypocalcemia prolongs QT to the same extent as congenital QT syndrome but does not increase risk of V tach. Hypocalcemia prolongs action potential uniformly and in congenital QTS action potentials are prolonged only in areas of the ventricle (Surawicz))
 - d. After depolarizations (Rosen)
4. Conditions which may lead to Torsades de Pointes
 - a. Drug related (idiosyncratic or dose related)
 - (1) Quinidine procainamide, disopyramide
 - (2) Amiodarone

- (3) Psychotropic drugs (phenothiazines, tricyclic antidepressants)
 - (4) Vasodilators (lidoflazine, prenlyamine)
 - (5) Organophosphate poisonings
- b. Bradydysrhythmias
- (1) SA nodal disease with severe sinus bradycardia
 - (2) Complete heart block with slow idioventricular rhythm
- c. Electrolyte disturbances
- (1) Hypokalemia
 - (2) Hypomagnesemia (rare)
- d. Intrinsic cardiac disease
- (1) Ischemia
 - (2) Myocarditis
- e. Congenital syndromes
- (1) Jervell and Lange-Nielsen syndrome
 - (2) Romano-Ward syndrome
- f. Liquid protein diets
- g. Central nervous system
- (1) Subarachnoid hemorrhage
 - (2) Pneumoencephalography
 - (3) Intracranial trauma
5. Clinical implications
- a. Recognition of predisposing conditions
 - b. Early detection of warning signs

- (1) Prolonged QT interval
 - (2) Relative change in QT interval
 - (3) Bizarre T wave aberration following extrasystole pause
6. ECG recognition
- a. Long QT interval (QT >0.50 sec or prolonged by >33%)
 - b. Tall U wave distorting the T wave
 - c. Undulating pattern to the tachycardia (may be seen in 1 lead and not another)
 - d. Initiated by long-short cycle sequence
 - e. Rhythm - irregular
 - f. Rate 150-250 (may be 300) BPM
 - g. Late initiating PVC (coupling interval of >0.50 seconds)



The onset of torsades de pointes. A, A prolonged QT-U with an R-on-T. B, Frequent PVCs with a run of ventricular tachycardia. C, Torsades de pointes.



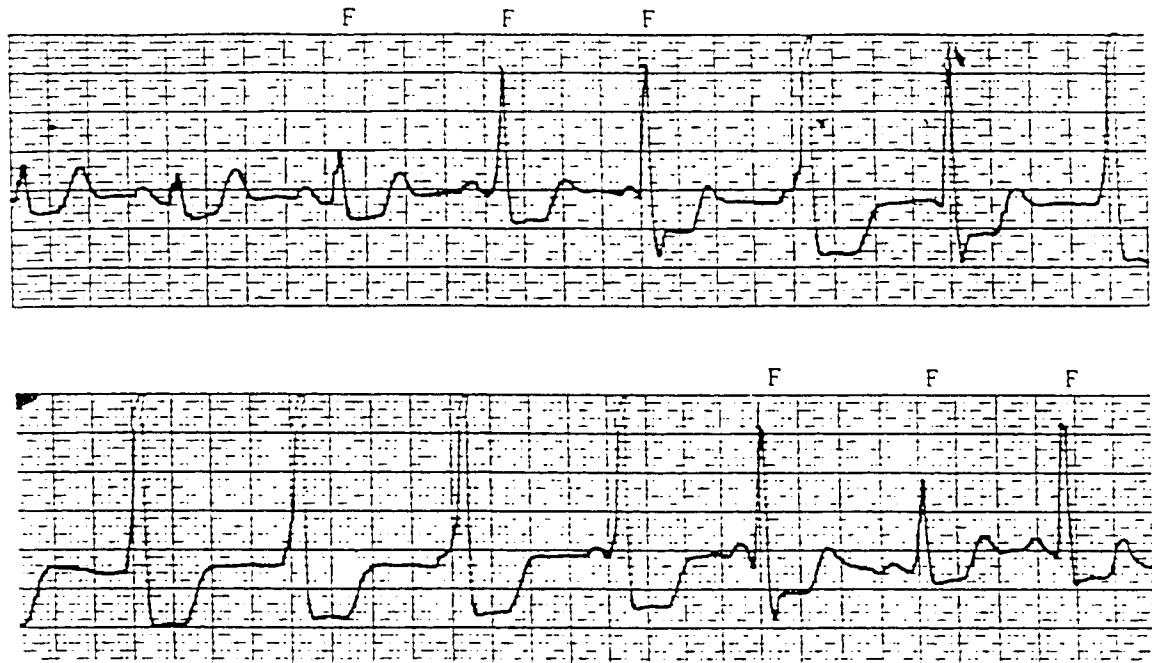
Torsades de pointes (continuous strip). Note the prolonged QT interval and the undulating-spindle look to the pattern. (Courtesy Dr. Alan Lindsay, Salt Lake City, Utah.)

7. Treatment

- a. Lidocaine may cause and/or control Torsades de Pointes
- b. Magnesium sulfate 2 gm IV over 1 minute (10 cc of 20% solution) followed with IV infusion 3-20 mg/min
- c. Beta-blockers: Deleterious in drug induced Torsades de Pointes but have favorable effect in congenital prolonged QTS
- d. Cardioversion

8. Nursing concerns
 - a. Recognize conditions which predispose patient to Torsades de Pointes ectopy, that is, any prolonged QT interval
 - b. Recognize Torsades de Pointes ectopy
 - c. Identify and withhold medication known to prolong QT until evaluated by physician
 - d. Of course, notify MD immediately
 - e. ACLS
- G. Accelerated Idioventricular Rhythm (AIVR)
 1. Area of altered automaticity originating in the ventricle paces the heart at a rate less than 100 beats per minute and greater than 60 beats per minute
 2. Mechanism - altered automaticity. Gradual increase in rate overtakes sinus rhythm.
 3. Causes
 - a. Increased myocardial irritability
 - b. Coronary artery disease, ASHD, RHD, digitalis toxicity
 4. Clinical significance
 - a. In the face of acute MI, AIVR does not affect outcome or prognosis
 - b. In face of digitalis toxicity, the mechanism is triggered activity and if not recognized and corrected, adversely affects patient outcome
 - c. May be a lifesaving escape rhythm
 5. ECG characteristics
 - a. Similar to ventricular tachycardia in morphology. QRS complex over 0.12 seconds.
 - b. Difference is rate
 - c. AIVR may not be evident if it is slower than intrinsic sinus rhythm, as ventricular ectopic focus is reset with each conducted sinus impulse
 - (1) May begin and end with fusion beats

- (2) May have AV dissociation
- (3) May be evident only when sinus impulse slows
- (4) Or, if faster than sinus rhythm may usurp sinus rhythm



Accelerated idioventricular rhythm. Note the fusion (F) beats at the onset of the tachycardia and at the end of the tracing.

6. Treatment

- a. None if patient is hemodynamically stable - assess and observe patient.
Usually self limiting.
- b. Correct underlying conditions
 - (1) Assure digitalis level is therapeutic
 - (2) Correct conditions which increase myocardial stretch, such as CHF
- c. Caution in use of Lidocaine - may suppress lifesaving rhythm
- d. Overdrive atrial or AV sequential pacing may be of value if rhythm is associated with clinical or hemodynamic instability due to loss of AV synchrony

7. Nursing interventions

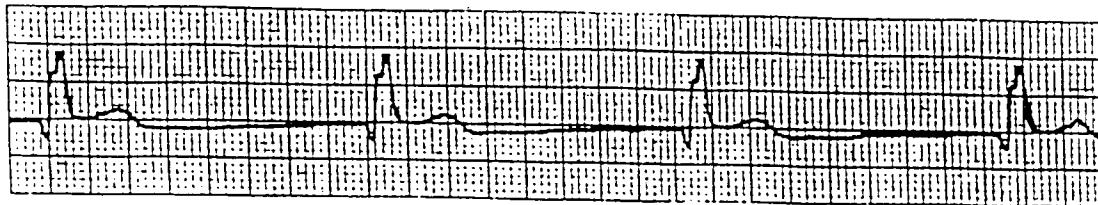
- a. Identify the rhythm and document with rhythm strip

- b. Assess patient for signs/symptoms of cardiac output
- c. Assess patient for tissue perfusion
- d. Notify MD of rhythm and patient status
- e. If appropriate, prepare for pacemaker
- f. Assess if drug induced suppression of higher pacemaker

H. Idioventricular Rhythm (Ventricular Escape Rhythm)

- 1. Escape rhythm originating in the ventricle with a rate of 20-40
- 2. Mechanism: Normal rate of automaticity for ventricular cells
- 3. Causes
 - a. Coronary artery disease - myocardial ischemia or infarct
 - b. Excessive vagal stimulation, particularly in face of MI
 - c. Drugs, which suppress higher pacemakers
- 4. Clinical significance
 - a. Escape rhythm may appear in severe sinus bradycardia or AV block and may be lifesaving
 - b. Not considered a stable escape rhythm
- 5. ECG characteristics
 - a. Slow - natural rate of automaticity for the ventricle 20-40 BPM
 - b. Usually regular
 - c. Morphology: similar to ventricular premature beats
 - (1) Wide, bizarre QRS
 - (2) AV dissociation
 - (3) May see fusion beats

(4) T wave opposite repolarization



Slow idioventricular rhythm.

6. Treatment

- a. Treat cause of the underlying bradycardia or block
- b. Do not suppress the rhythm with antidysrhythmics
- c. Pacemaker if symptomatic
- d. BLS/ACLS if appropriate (See Algorhythm)

7. Nursing interventions

- a. Identify and document rhythm
- b. Assess patient for cardiac output and tissue perfusion
- c. Notify MD stat
- d. BLS/ACLS as necessary
- e. Prepare for pacemaker

I. Ventricular Asystole - (synonyms: cardiac standstill, agonal heart, dying heart, asystole)

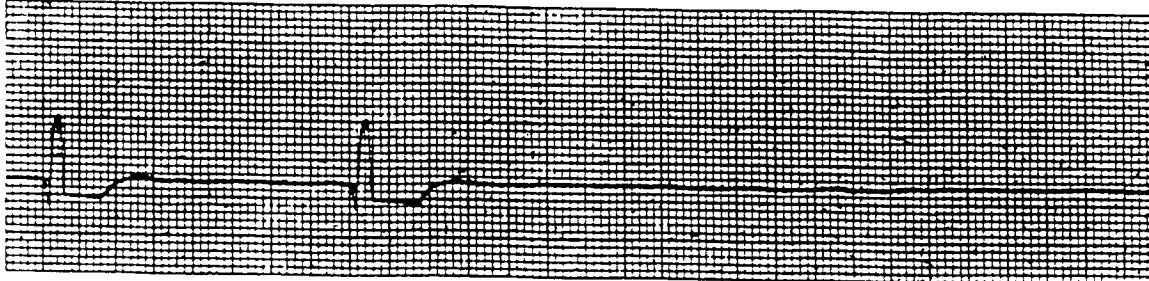
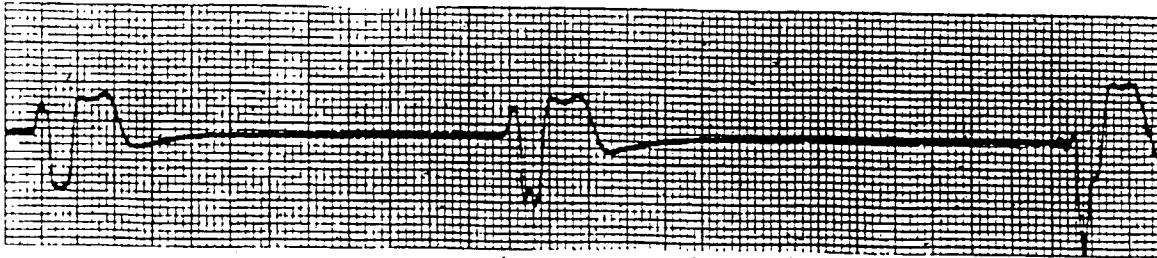
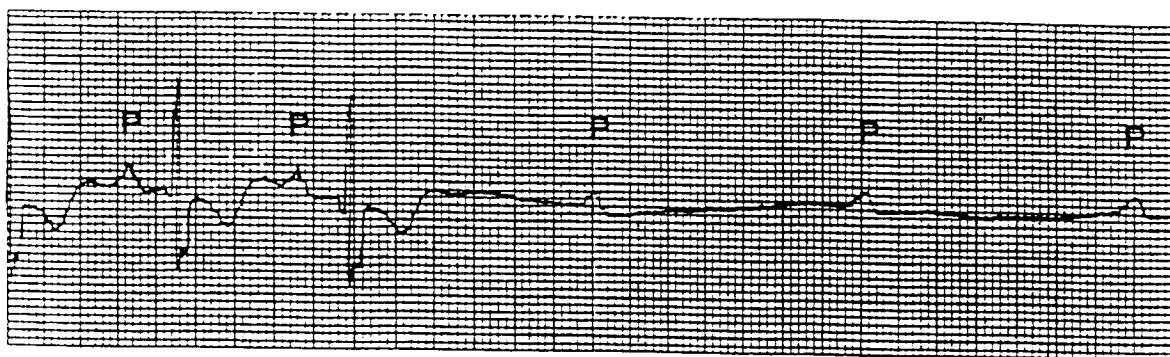
1. No ventricular activity
2. Mechanism - ventricles are not stimulated and do not contract
3. Causes
 - a. CAD - end stage
 - b. End stage/unsuccessful code
 - c. Terminal rhythm

4. Clinical implications

- a. Usually pulseless, nonbreathing - clinical death
- b. No BP or palpable pulse
- c. Clinically cannot be differentiated from ventricular fibrillation - must have ECG monitor
- d. More ominous than V-fib

5. ECG characteristics

- a. No ventricular response
- b. May see p waves
- c. Flatline - may see undulation of baseline



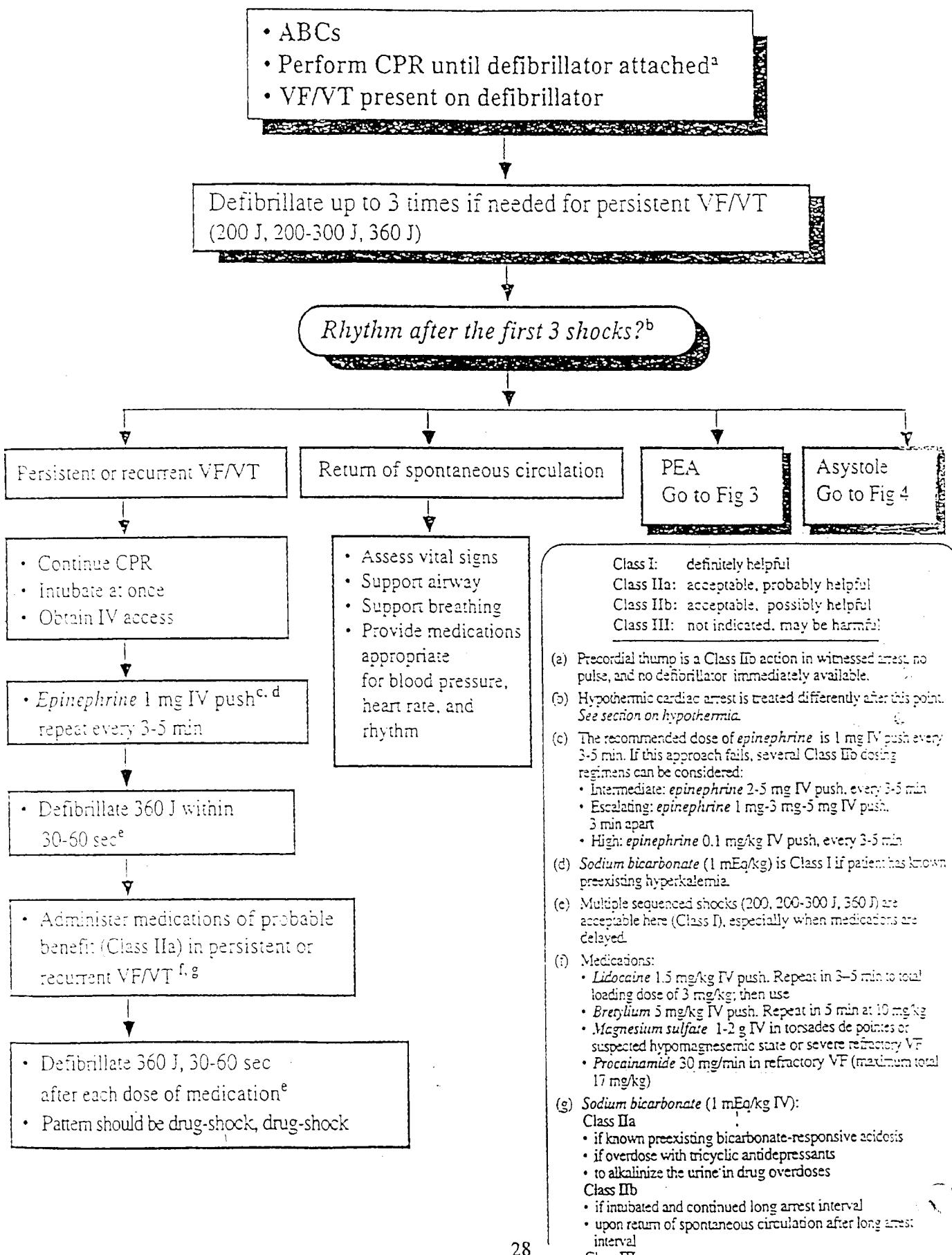
6. Treatment

- a. CPR necessary to keep patient alive
- b. Defibrillation ineffective
- c. May need pacemaker
- d. ACLS algorithm (See Algorithm)

7. Nursing role

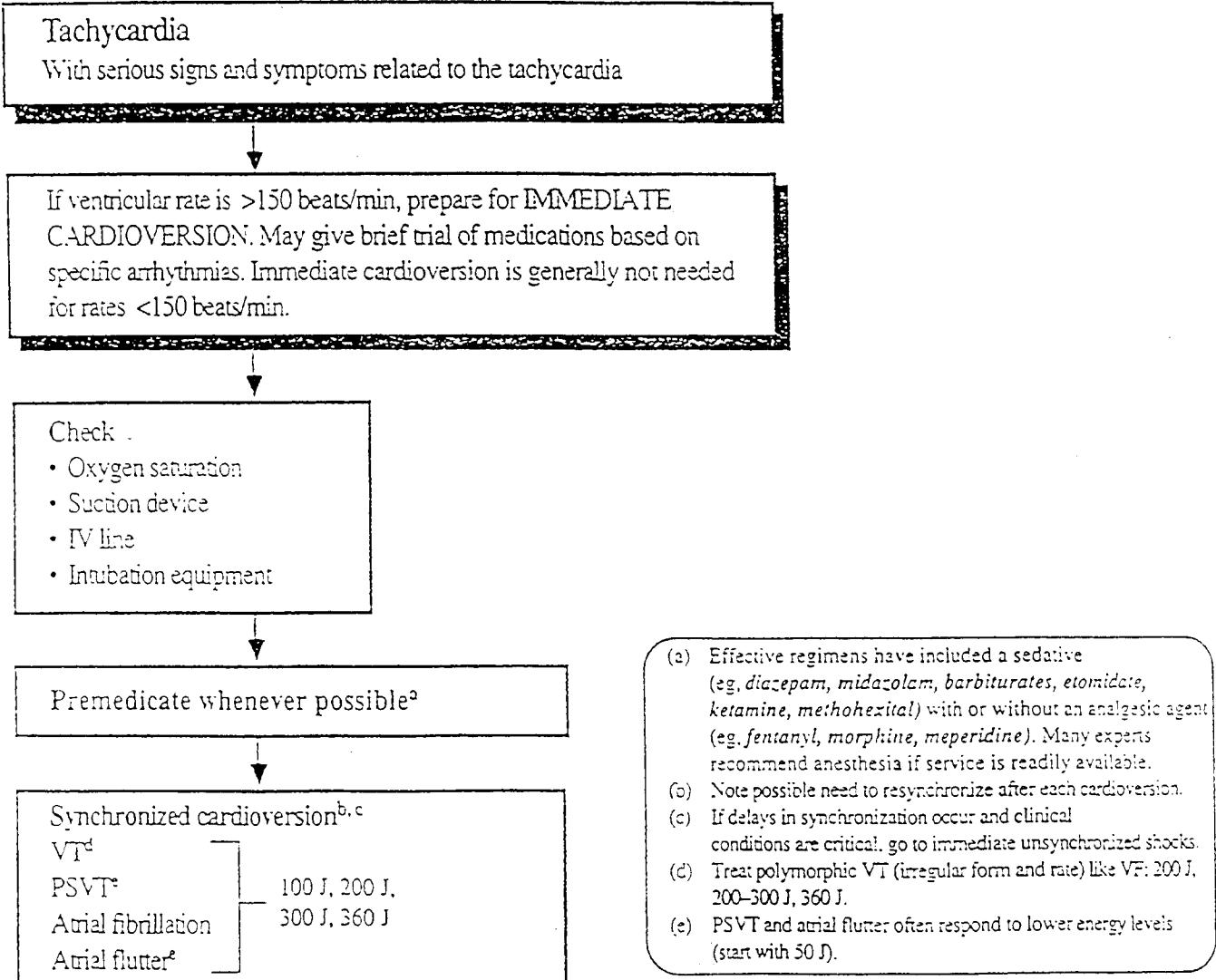
- a. Identify rhythm
- b. Assess patient
- c. CPR/ACLS
- d. Code
- e. Notify MD
- f. Prepare to pace patient

Ventricular Fibrillation/ Pulseless Ventricular Tachycardia Algorithm (VF/VT)

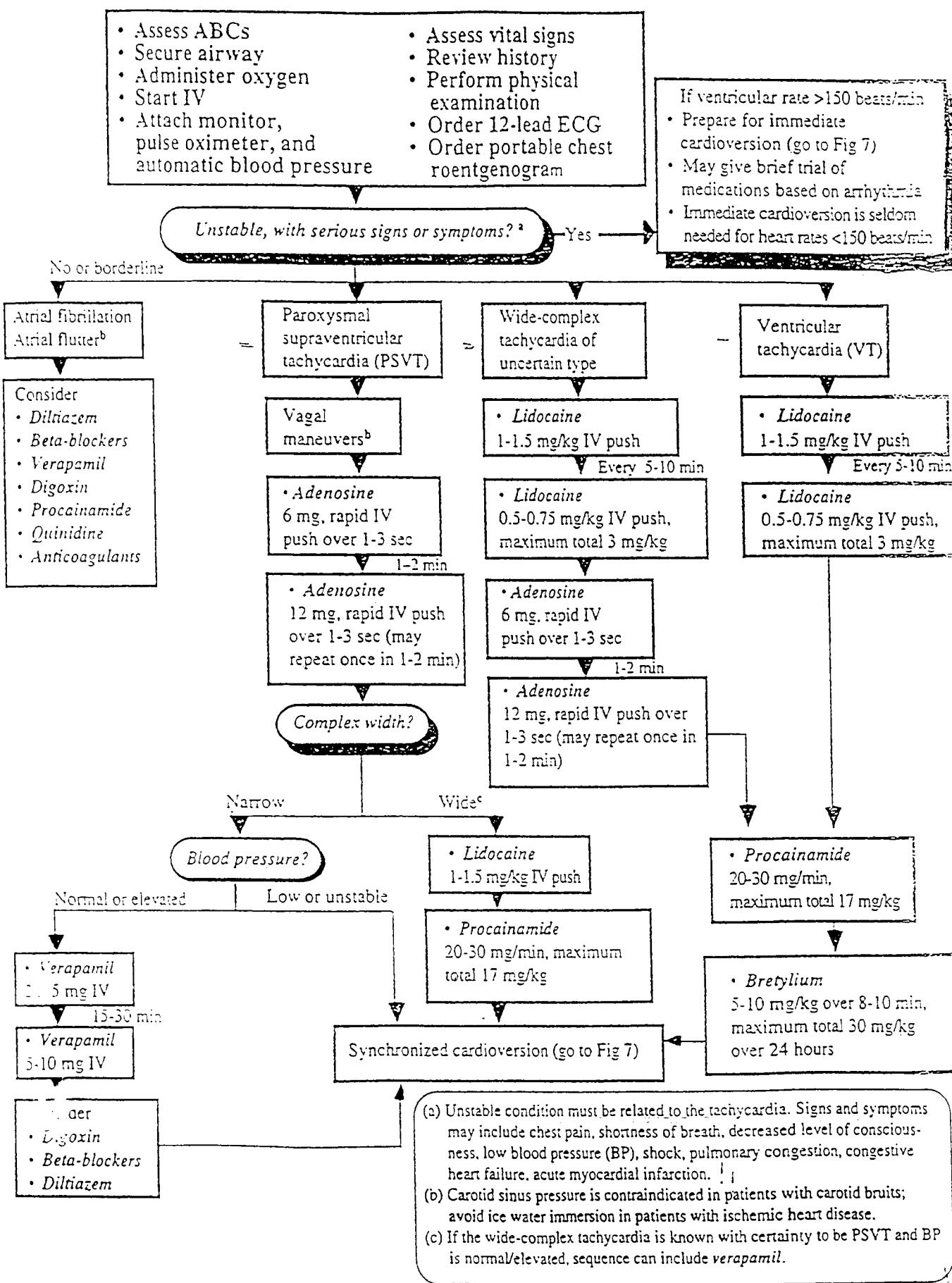


Electrical Cardioversion Algorithm

(Patient is not in cardiac arrest)



Tachycardia Algorithm



Bradycardia Algorithm

(Patient is not in cardiac arrest)

