NWOCC

NORTHWEST OHIO CARDIOLOGY CONSULTANTS

Cardiac Arrhythmias

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2013 OAPA Conference

EPNO

Emergency Physicians of Northwest Ohio

Objectives

- Review differentials of cardiac arrhythmias
- Discuss the most common arrhythmias
- Review the latest treatment guidelines for each
- Review EKG's of selected arrhythmias

Cardiac Arrhythmias

Tachycardic

Wide Complex QRS > 120ms

Regular:

* V. Tach
* ST, SVT, A. Flutter with Aberrancy

Irregular:

* AFib, MAT, A.Flutter with Variable conduction with Abarrency
* V. Fib
* Tosades de points

Narrow Complex QRS < 120ms

Regular:

* Sinus Tachycardia
* A. Flutter
* SVT
* WPW

Irregular:

* A. Fib
* A . Flutter with variable conduction
* MAT

Bradycardic

Wide Complex QRS > 120ms

Regular:

* CHB

Irregular:

* AFib with BBB with Slow AV nodal conduction

Narrow Complex QRS<120ms

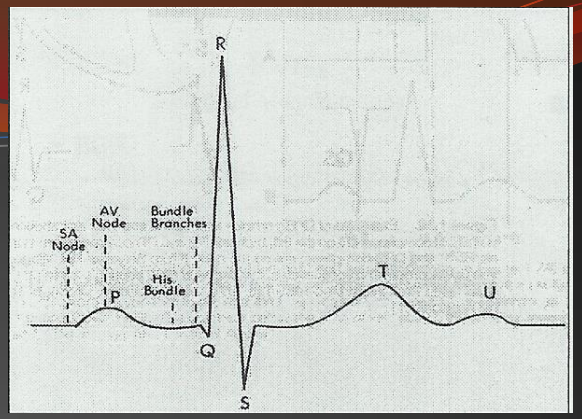
Regular:

* Sinus Brady
* 1st degree AVB
* 2nd degree Type 1 AVB

Irregular:

* 2nd degree Type 2, Weinkebach
* Afib with Slow conduction

Normal Values



- **P Wave** - Atrial Depolarization
- **PR Interval** - AV node Conduction
- **QRS Complex** – Ventricular Depolarization
- **ST Segment** – Plateau of all ventricular action potentials
- **T Wave** – Repolarization of Ventricular cells
- **U Wave** – Follows T wave, etiology unclear

Regular Narrow-Complex Tachycardias

- **Sinus Tachycardia:** Normal P-waves, HR usually <150 bpm
- **Proxysmal Atrial Tachycardia (PAT):** P-wave Morphology is different from sinus (may be inverted) *or* absent
- **Atrial Flutter:** Large "saw-toothed" *flutter-waves,* +/– variable AV-block
- **AV-Nodal Re-entrant Tachycardia (AVNRT):** Most common form of PSVT, +/– P-waves
- **AV Re-entrant Tachycardia (AVRT):** A common form of PSVT, +/– P-waves, + accessory pathway

Irregularly Irregular Narrow-Complex Tachycardias

1. **Atrial Fibrillation**: **No** recognizable P-waves

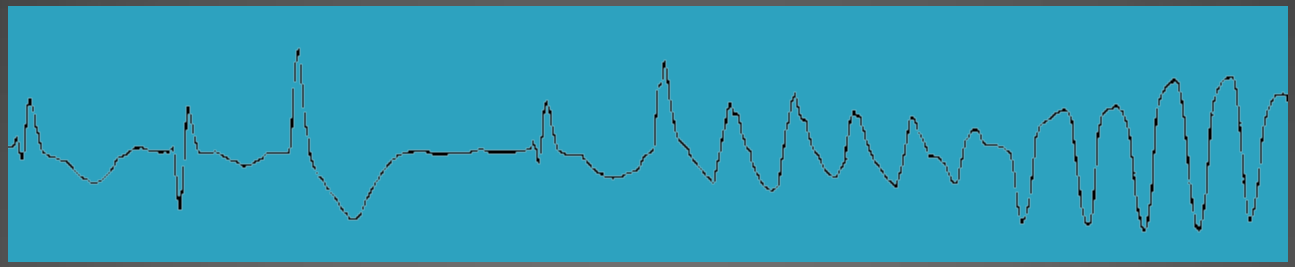
Irregularly Irregular Ventricular Rhythm

2. **Multifocal Atrial Tachycardia (MAT)**: Three (3) consecutive P-waves with different morphologies, usually associated with COPD

3. **Any "regular" SVT with variable AV-block**:
   Examples: PAT *or* a. flutter with variable AV-block

Ventricular Tachycardia

A 60 year old man with Ischaemic Heart Disease.



Polymorphous ventricular tachycardia (Torsade de pointes).

- **This is a form of VT where there is usually no difficulty in recognising its ventricular origin.**
- **wide QRS complexes with multiple morphologies**
- **changing R - R intervals**
- **the axis seems to twist about the isoelectric line**
- **it is important to recognise this pattern as there are a number of reversible causes**
  - **heart block**
  - **hypokalaemia or hypomagnesaemia**
  - **drugs (e.g. tricyclic antidepressant overdose)**
  - **congenital long QT syndromes**
  - **other causes of long QT (e.g. IHD)**

Polymorphic VT – Torsades de pointes

- Causes:
  - Family History of Congenital Long QT
  - Hypomagnesiumia
  - Hypokalemia
  - Congenital long QT syndrome
  - Female gender
  - Acquired long QT syndrome (causes of which include medications and electrolyte disorders such as hypokalemia and hypomagnesemia)
  - Bradycardia
  - Renal or liver failure
- Treatment – Magnesium
  - Mexilitine, Esmolol, isoproterenol (for Brady induced torsades)

VA & SCD Related to Specific Populations

Examples of Drugs Causing Torsades de Pointes

Frequent (greater than 1%)*

- Disopyramide
- Dofetilide
- Ibutilide
- Procainamide
- Quinidine
- Sotalol
- Ajmaline

Less Frequent

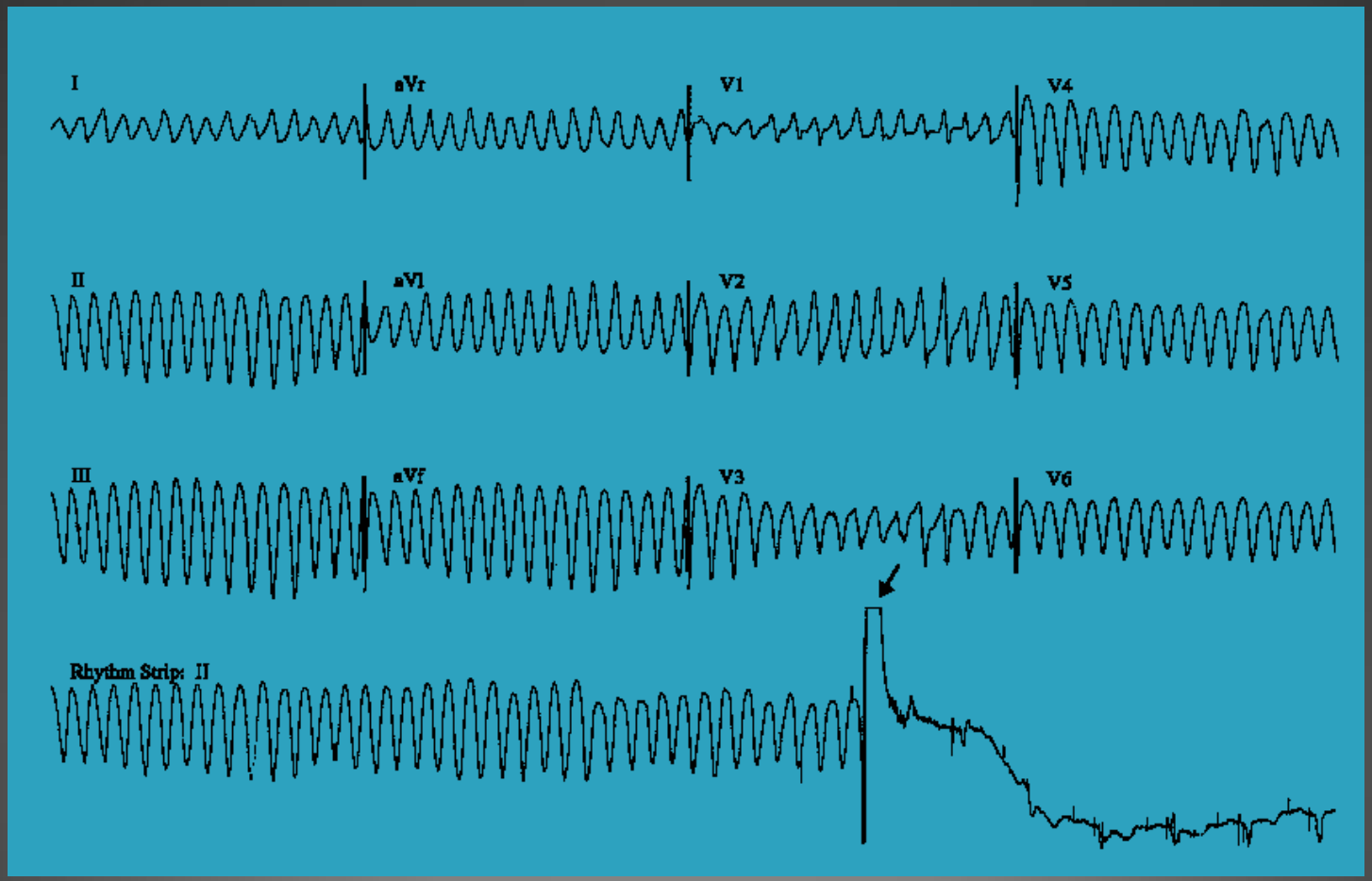
- Amiodarone
- Arsenic trioxide
- Bepridil
- Cisapride
- Anti-infectives: clarithromycin, erythromycin, halofantrine; pentamidine, sparfloxacin
- Antiemetics: domperidone, droperidol
- Antipsychotics: chlorpromazine, haloperidol, mesoridazine, thioridazine, pimozide
- Opioid dependence agents: methadone

* (e.g., hospitalization for monitoring recommended during drug initiation in some circumstances)

Adapted with permission from Roden DM. N Engl J Med 2004;350:1013-22.

Ventricular Tachycardia

A 36 year old lady with recurrent blackouts.



Implantable cardioverter defibrillator
Most of this 12-lead recording is polymorphic ventricular tachycardia but, in the rhythm strip, the large deflection (arrowed) is the defibrillator discharging.
Following the defibrillation a dual chamber pacemaker can be seen.

Clinical Presentations of Patients with VA & SCD

- Asymptomatic individuals with or without electrocardiographic abnormalities
- Persons with symptoms potentially attributable to ventricular arrhythmias
  - ♥ Palpitations
  - ♥ Dyspnea
  - ♥ Chest pain
  - ♥ Syncope and presyncope
- VT that is hemodynamically stable
- VT that is hemodynamically unstable
- Cardiac arrest
  - ♥ Asystolic (sinus arrest, atrioventricular block)
  - ♥ VT
  - ♥ Ventricular fibrillation (VF)
  - ♥ Pulseless electrical activity

Epidemiology of VA & SCD

Classification of Ventricular Arrhythmia by Disease Entity

- Chronic coronary heart disease
- Heart failure/LV Dysfunction
- Congenital heart disease
- Neurological disorders
- Structurally normal hearts
- Cardiomyopathies
    - ♥ Dilated cardiomyopathy
    - ♥ Hypertrophic cardiomyopathy
    - ♥ Arrhythmogenic right ventricular (RV) cardiomyopathy

VT Causes

- Common causes:
  - Coronary artery disease with myocardial infarction
  - Nonischemic cardiomyopathy
  - Infiltrative disease (*eg*, sarcoidosis, amyloidosis)
  - Infectious disease (*eg*, viral myocarditis, Chagas disease, Lyme disease)
  - Inflammatory diseases that affect the myocardium (*eg*, systemic lupus erythematosus, rheumatoid arthritis, giant cell myocarditis)
  - Digitalis toxicity (bidirectional ventricular tachycardia)
  - Mitral valve prolapse
  - Electrolyte (notably potassium and magnesium) abnormalities
  - Structural, toxic, or metabolic derangement affecting the homogeneity of ventricular repolarization (*eg*, prolonged QT syndromes, Brugada syndrome), most often associated with torsade de pointes, polymorphic ventricular tachycardia, or ventricular fibrillation
  - Arrhythmogenic right ventricular dysplasia
  - Blunt chest trauma
- Rare causes:
  - Congenital myocardial defects (*eg*, tetralogy of Fallot, pulmonary stenosis) previous corrective surgery for congenital heart defect)
  - Marfan syndrome with aortic dissection
  - Torsade de pointes is caused by certain drugs (*eg*, haloperidol, erythromycin, quinidine, and methadone, among others) or by inherited defects in cardiac ion channels (*eg*, cardiac channelopathy)
  - Carbon monoxide poisoning

VT - Risk Factors

- **Risk factors**
  - Ischemia
  - Cardiomyopathy
  - Heart failure
  - Cocaine use
  - Use of certain medications, such as quinidine, phenothiazines, and tricyclic antidepressants
  - Congenital heart disease
  - Surgical repair of congenital heart defects
  - Primary and metastatic malignancies involving the heart muscle
  - QT prolongation and Marfan syndrome in neonates
  - Trauma
  - Pericardial inflammation

Therapies for VA

- **Antiarrhythmic Drugs**

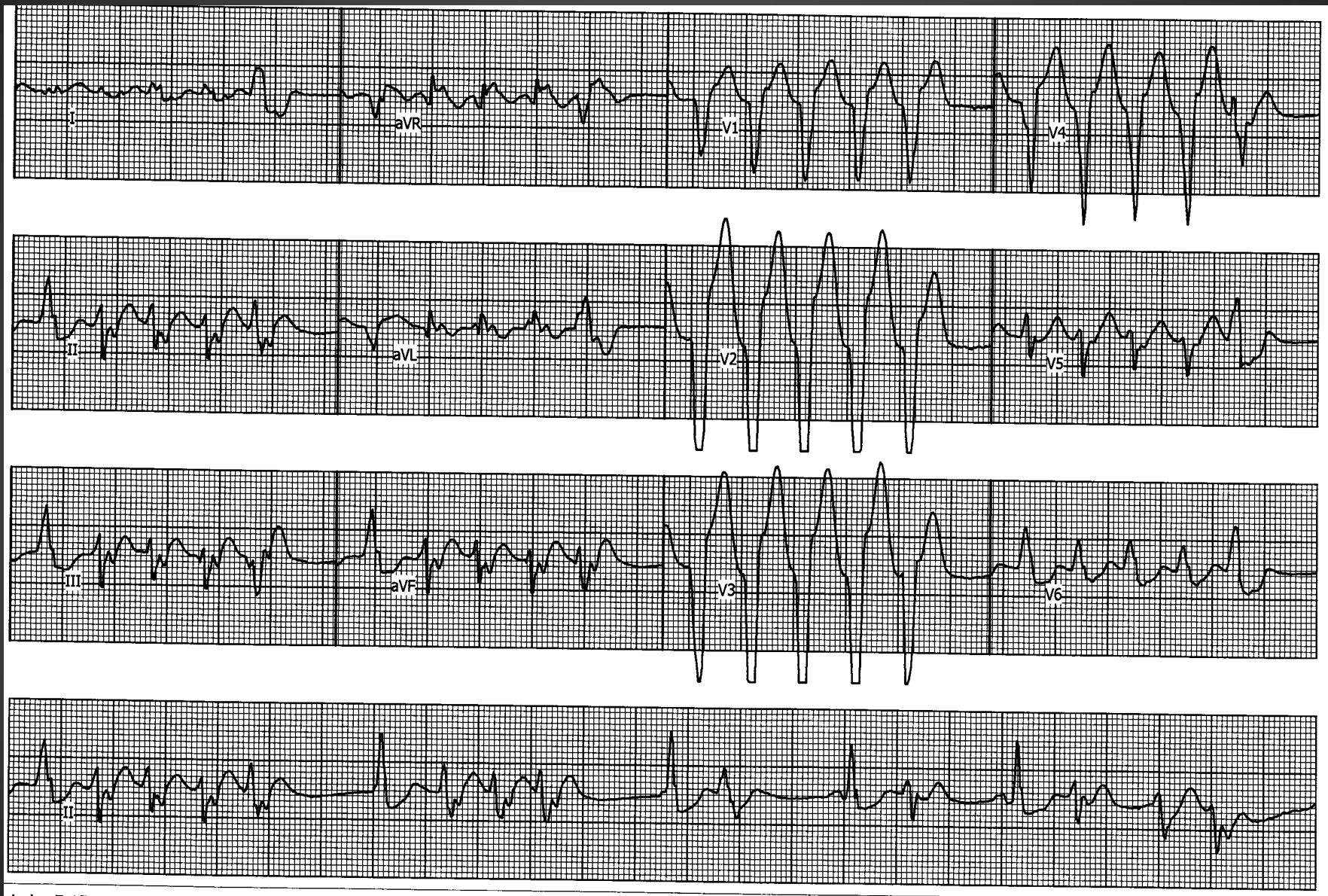
- ♥ Beta Blockers: Effectively suppress ventricular ectopic beats & arrhythmias; reduce incidence of SCD
- ♥ Amiodarone: No definite survival benefit; some studies have shown reduction in SCD in patients with LV dysfunction especially when given in conjunction with BB. Has complex drug interactions and many adverse side effects (pulmonary, hepatic, thyroid, cutaneous)
- ♥ Sotalol: Suppresses ventricular arrhythmias; is more pro-arrhythmic than amiodarone, no survival benefit clearly shown
- ♥ Conclusions: Antiarrhythmic drugs (except for BB) should not be used as *primary* therapy of VA and the prevention of SCD

Therapies for VA

Non-antiarrhythmic Drugs

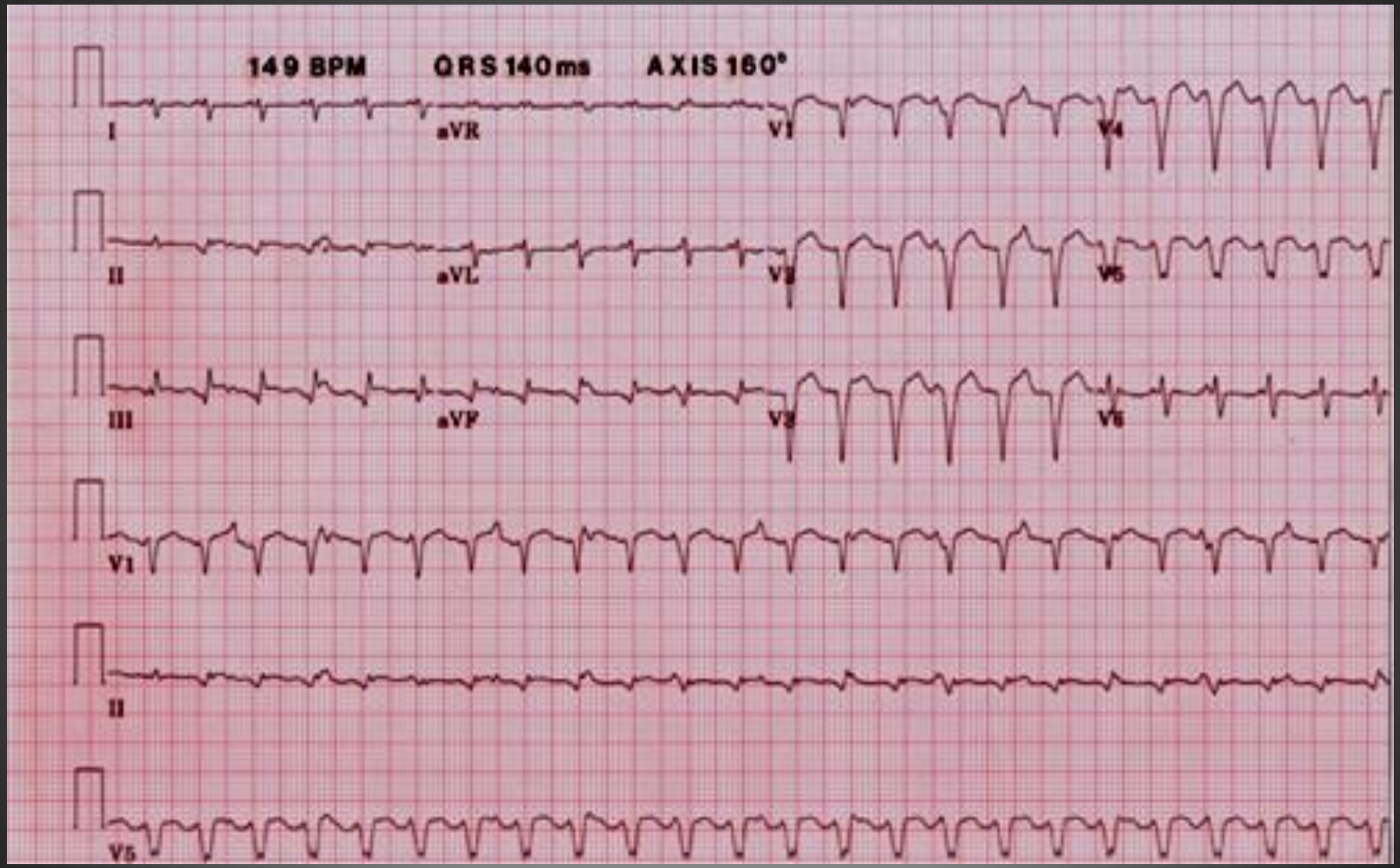
- Electrolytes: magnesium and potassium administration can favorably influence the electrical substrate involved in VA; are especially useful in setting of hypomagnesemia and hypokalemia
- ACE inhibitors, angiotensin receptor blockers and aldosterone blockers can improve the myocardial substrate through reverse remodeling and thus reduce incidence of SCD
- Antithrombotic and antiplatelet agents: may reduce SCD by reducing coronary thrombosis
- Statins: have been shown to reduce life-threatening VA in high-risk patients with electrical instability
- n-3 Fatty acids: have anti-arrhythmic properties, but conflicting data exist for the prevention of SCD

Nonsustained Monomorphic VT

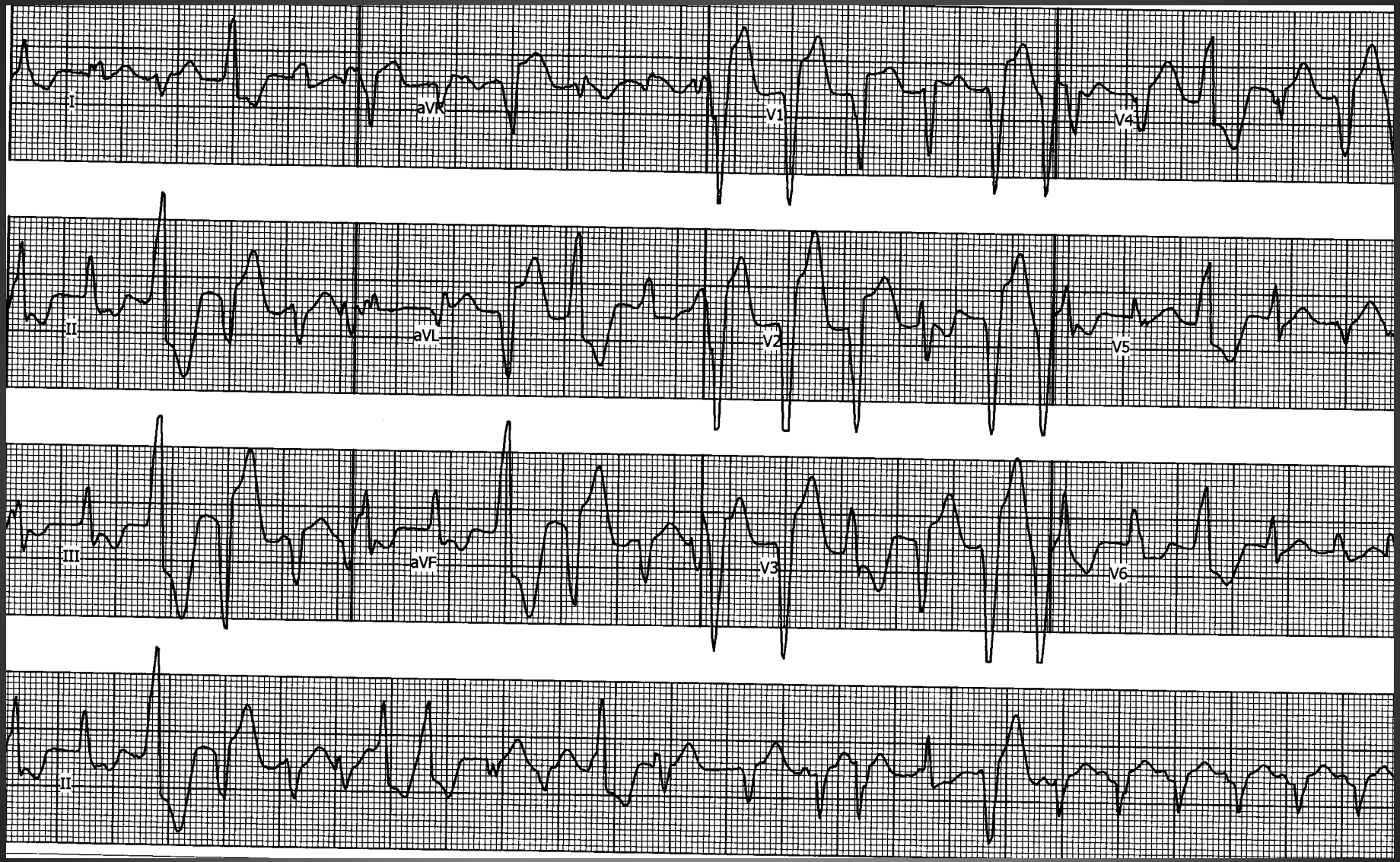


Sustained Monomorphic VT

72-year-old woman with CAD

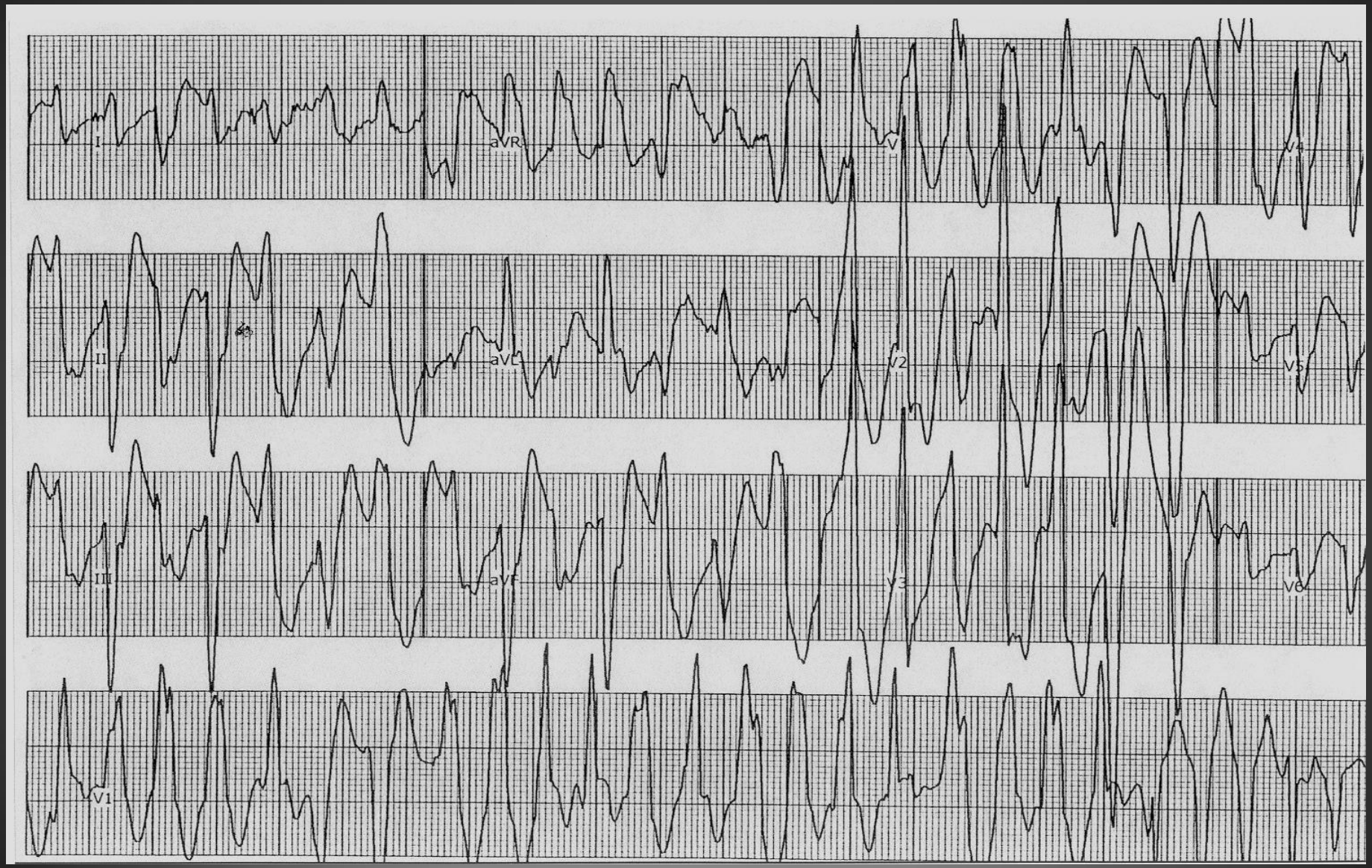


Nonsustained Polymorphic VT

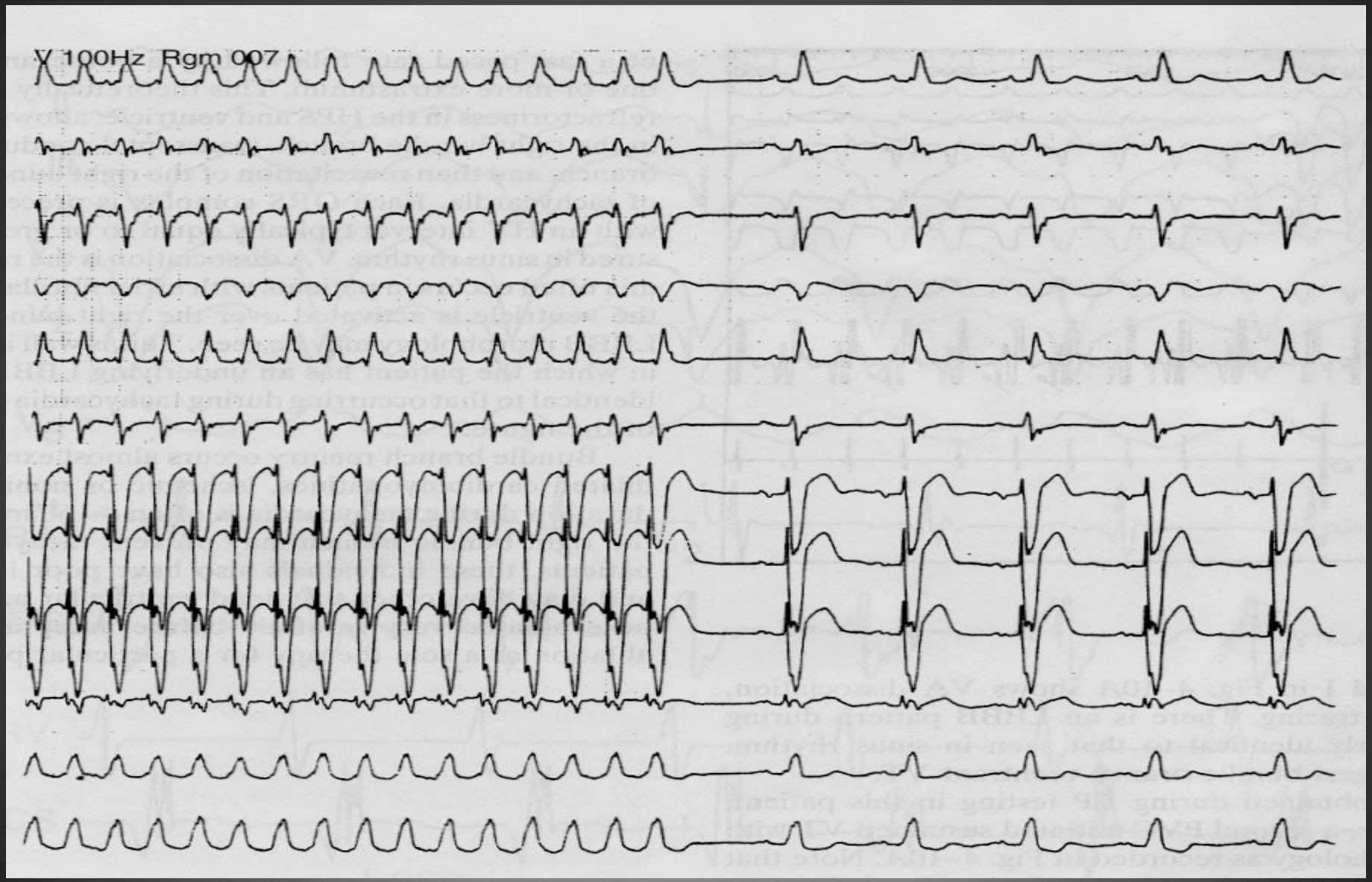


Sustained Polymorphic VT

Exercise induced in patient with no structural heart disease

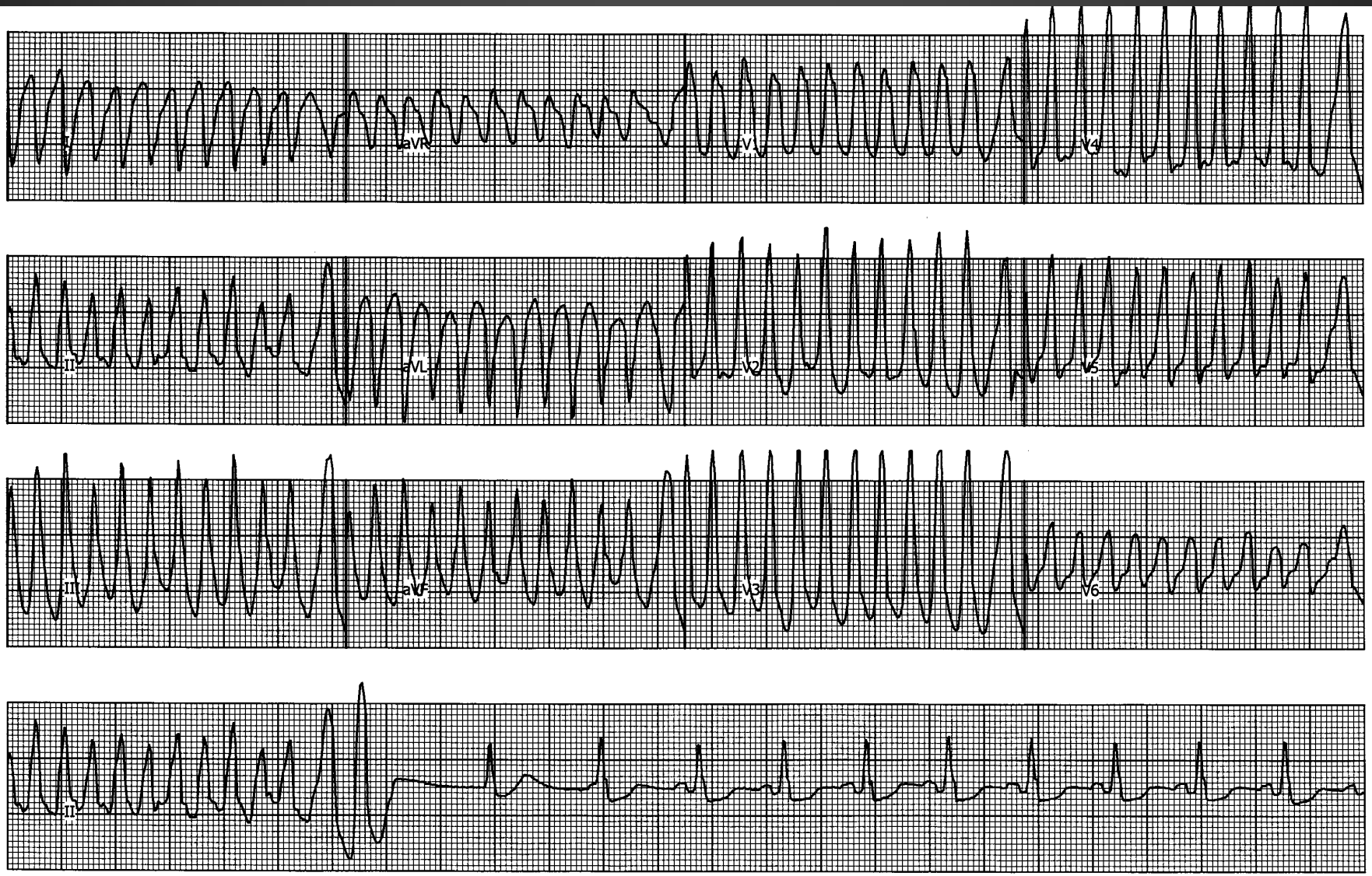


Bundle Branch Reentrant VT

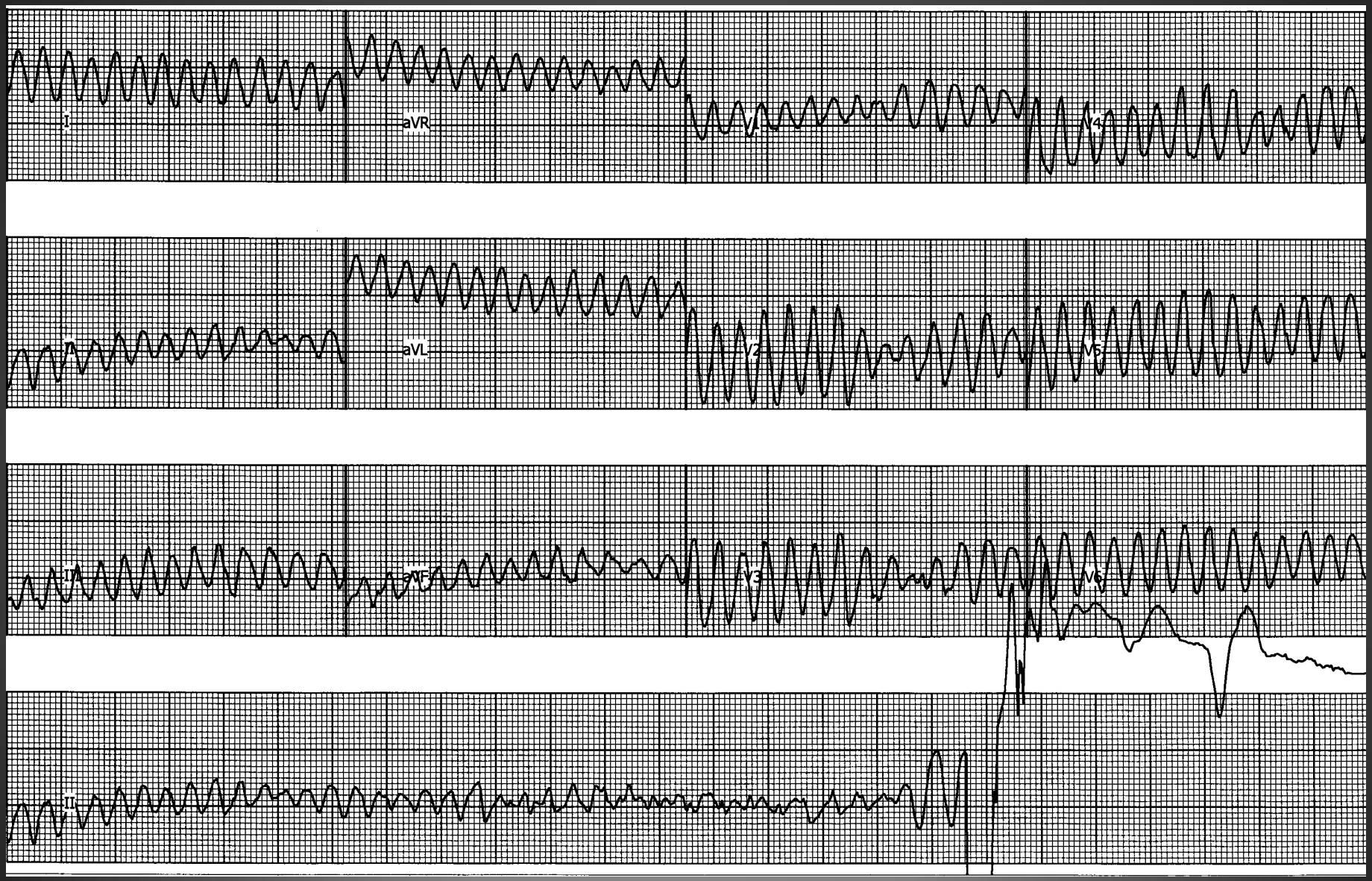


Ventricular Flutter

Spontaneous conversion to NSR (12-lead ECG)

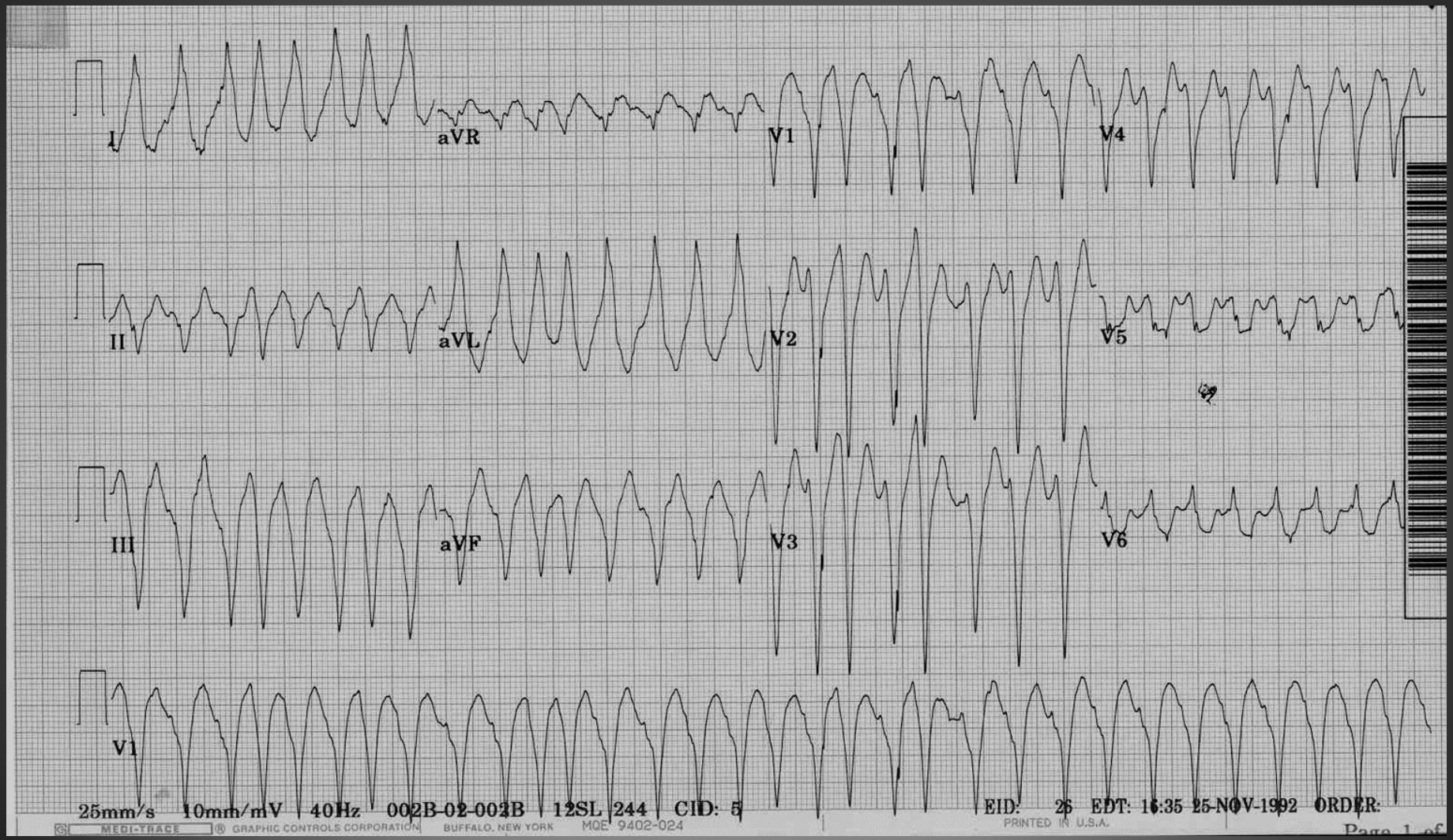


VF with Defibrillation (12-lead ECG)

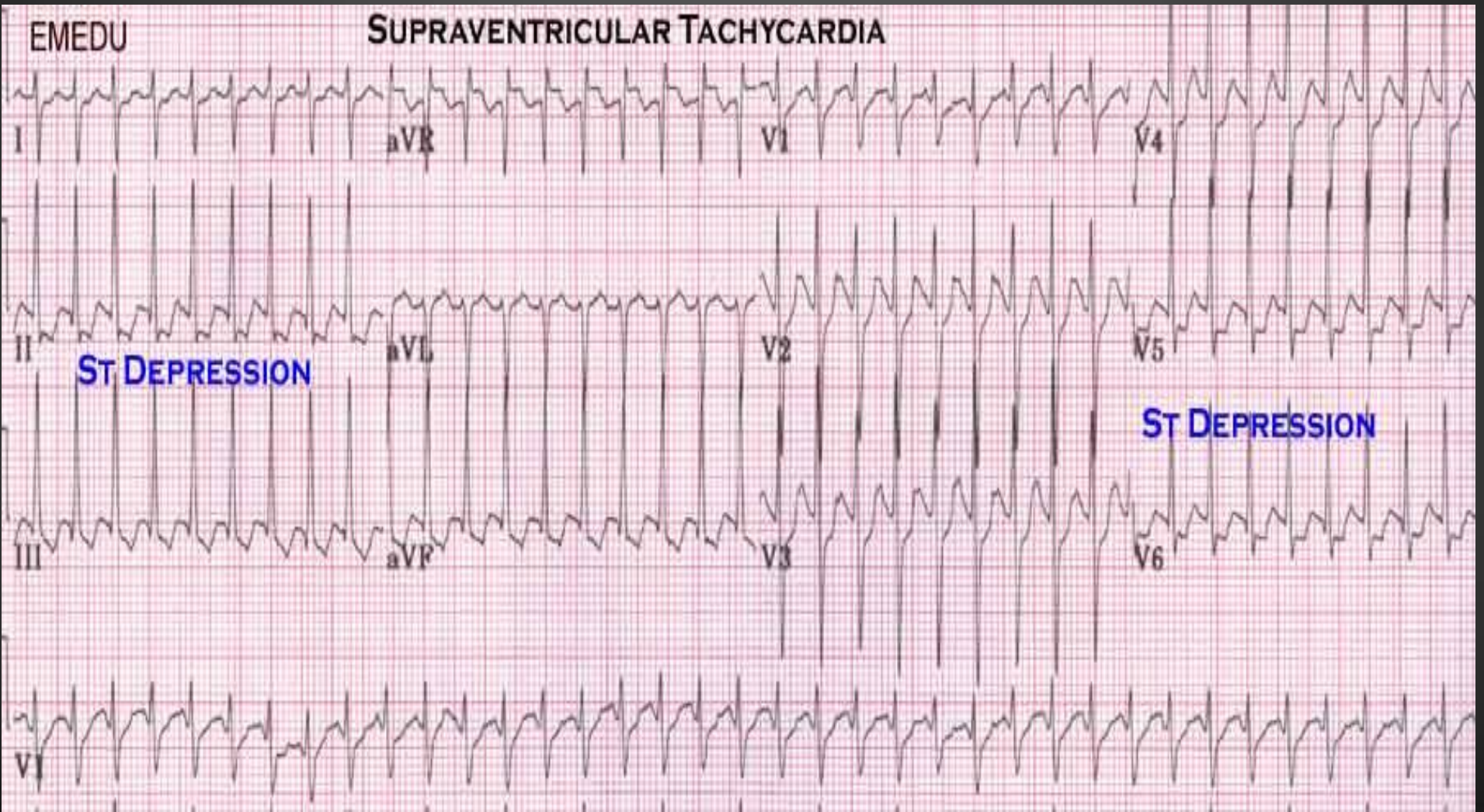


Wide QRS Irregular Tachycardia:

Atrial Fibrillation with antidromic conduction in patient with accessory pathway – Not VT



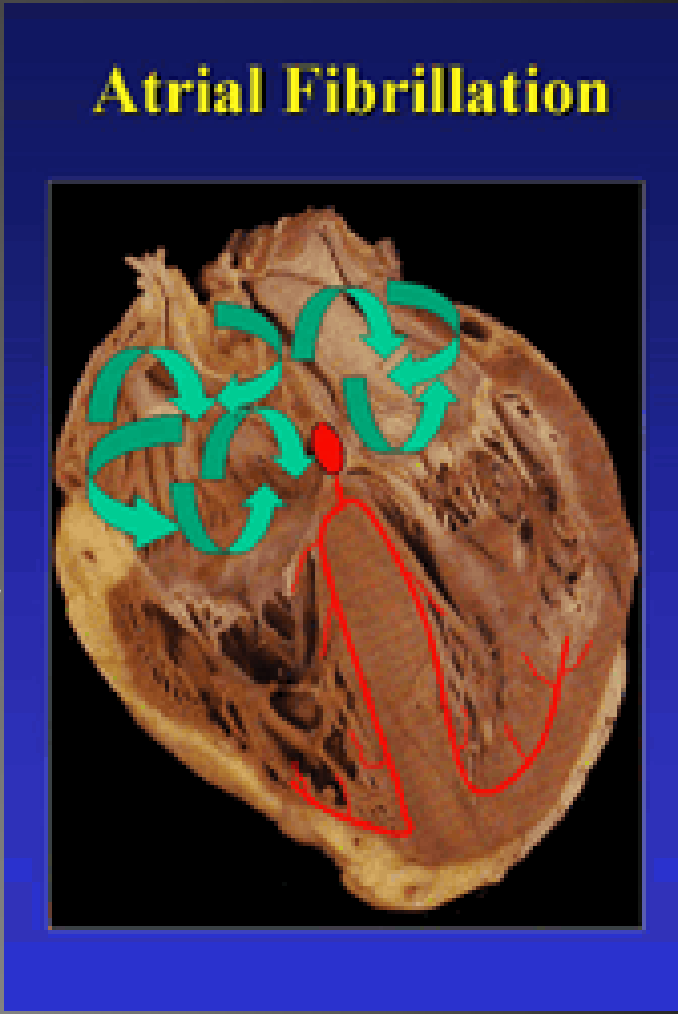
SVT


EMEDU
SUPRAVENTRICULAR TACHYCARDIA
I
aVR
V1
V4
II
aVL
V2
V5
ST DEPRESSION
ST DEPRESSION
III
aVF
V3
V6
V

Atrial Fibrillation

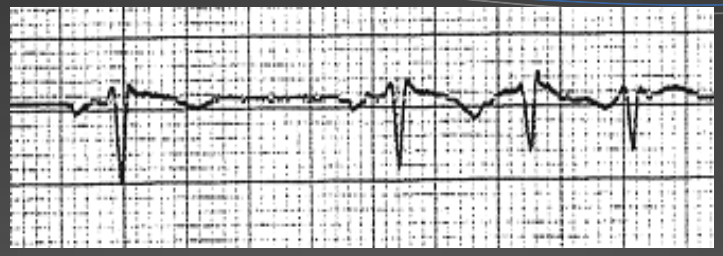
- Characterized by the absence of coordinated atrial systole
- **ReEntrant Waves (Wavelets) Theory**
  - **Small multiple waves initiate in the atrium spreading chaotically to form small circuits of reentrant electrical activity**
- **Atrial Myocyte Theory**
  - **Rapid repetitive impulse generation by atrial myocytes located near the orifice of the Pulmonary veins**
- **Afib begets Afib**
  - **Anatomical remodeling, disruption of electrical circuits, and cellular damage and fibrosis results in permanent Afib**

www.nhlbi.nih.gov/health/health-topics/topics/af/

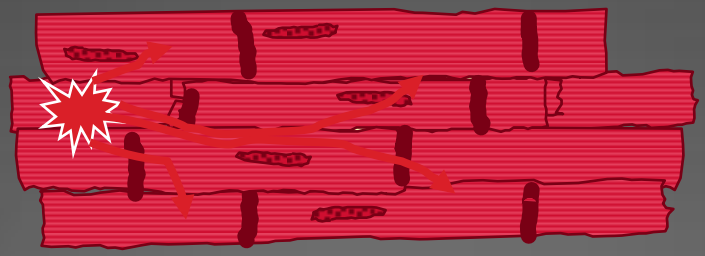


Types of AF

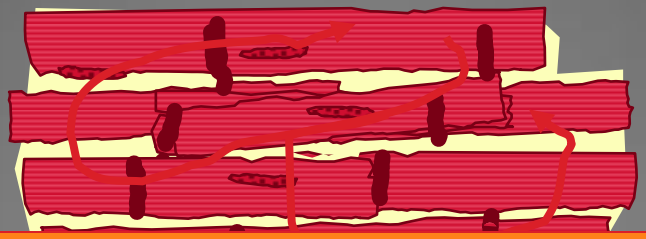
Paroxysmal AF
(< 7 days)



Persistent AF
(> 7 days)



Permanent AF



Triggers

Ectopic foci

Electrophysiologic Remodeling

Chronic Substrate fibrosis

Other Types : Lone AF, AF-CHF, Holiday heart

Stambler et al JCE 2003;14:499
Li, Nattel et al. *Circulation*. 1999;100:87-95

Prevalence

- The most common Chronic Arrhythmia, increasing worldwide
- Incidence Increases with age
  - 4% of pop 60-75 y/o
  - 10% of pop >75y/o
  - 2.2 million US adults
  - Male > Female, Whites>Blacks
- Increased risk (age/disease matched controls)
  - 1.5-2.0 increased mortality
  - Markedly increased risk of embolic events and CHF

Etiology - Cardiac

- **Structural Heart Disease**
  - CAD
  - Mitral Valve Disease
  - Systolic or Diastolic dysfunction
  - Congestive Heart Failure (15-30%)
  - Myocardial Infarction
  - Atrial Enlargement
  - Rheumatic Heart Dz
  - Hypertrophic CMP
- **Other Heart Disease**
  - Pericarditis/Myocarditis
  - Wolff-Parkinson-White Syndrome
  - Sick Sinus Syndrome
  - Congenital Heart Disease
  - Post Coronary Artery Bypass Surgery (40%)
  - Post Valvular Sx
  - Hypertension
  - Diabetes

Etiology – Non Cardiac

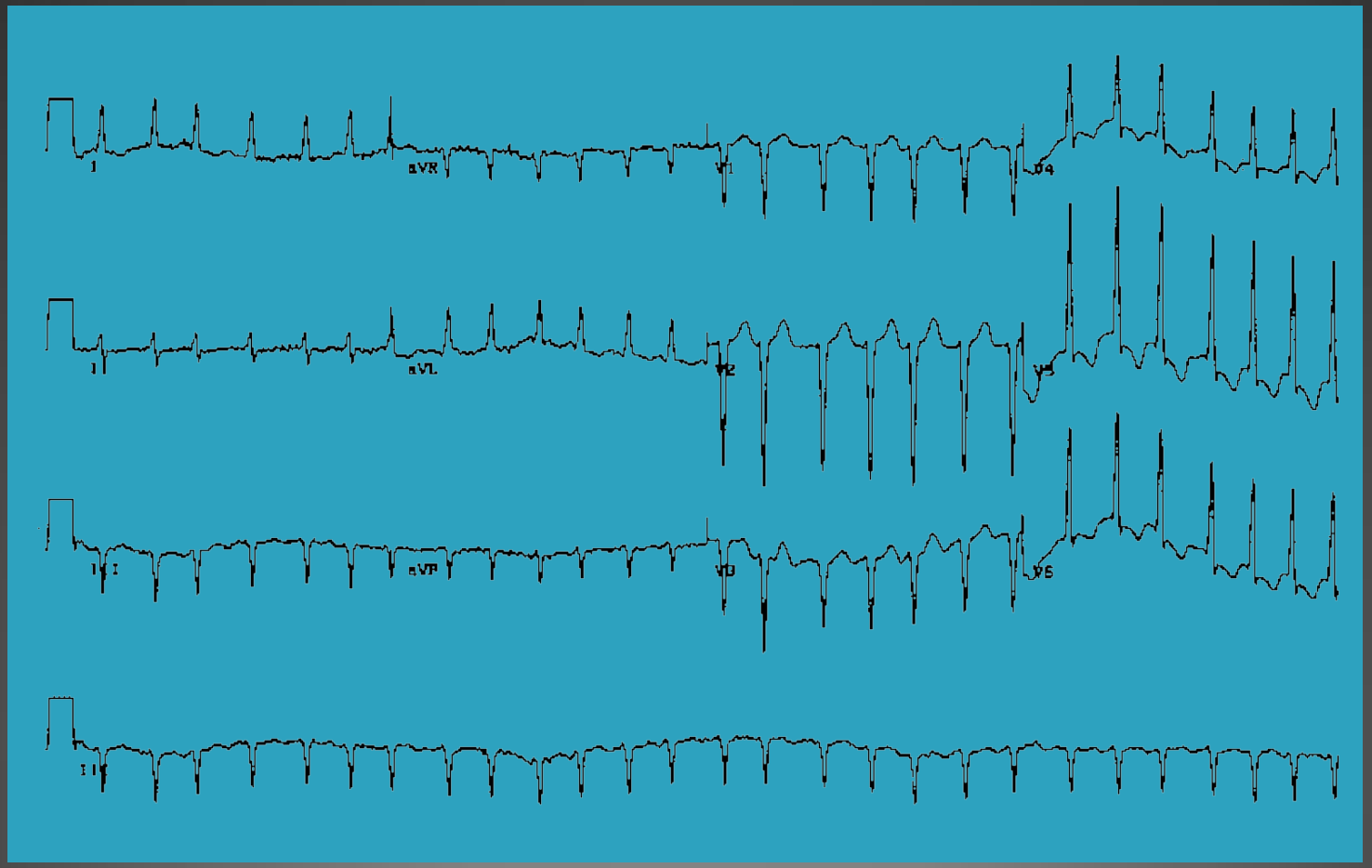
- Acute or Chronic alcohol ingestion (Holiday Heart Syndrome or Alcoholic Cardiomyopathy)
- Hyper or Hypo – thyroidism
- Alteration in vagal or sympathetic tone
- Pulmonary Embolism (PE)
- Sepsis
- Chronic Obstructive Pulmonary Disease (COPD)
- Theophyilline
- Pheochromocytoma
- Lone Atrial Fibrillation (< 10%)

Clinical Presentation

- Asymptomatic / Incidental Finding
- Palpitations
- Skipped Beats
- Bradycardia/Tachycardia
- Lightheadedness
- Malaise/Weakness
- Shortness of Breath
- Anginal Symptoms
- Syncope
- CHF presentation –
  Mostly Diastolic Dysfunction
  Ventricular filling dependent on Atrial Kick
- TIA/CVA
- Thromboembolic event
- Hyper/Hypo-thyroid symptoms

Atrial Fibrillation

A 76 year old man with breathlessness.



Atrial fibrillation with rapid ventricular response

- **Irregularly irregular ventricular rhythm.**
- **Sometimes on first look the rhythm may appear regular but on closer inspection it is clearly irregular.**

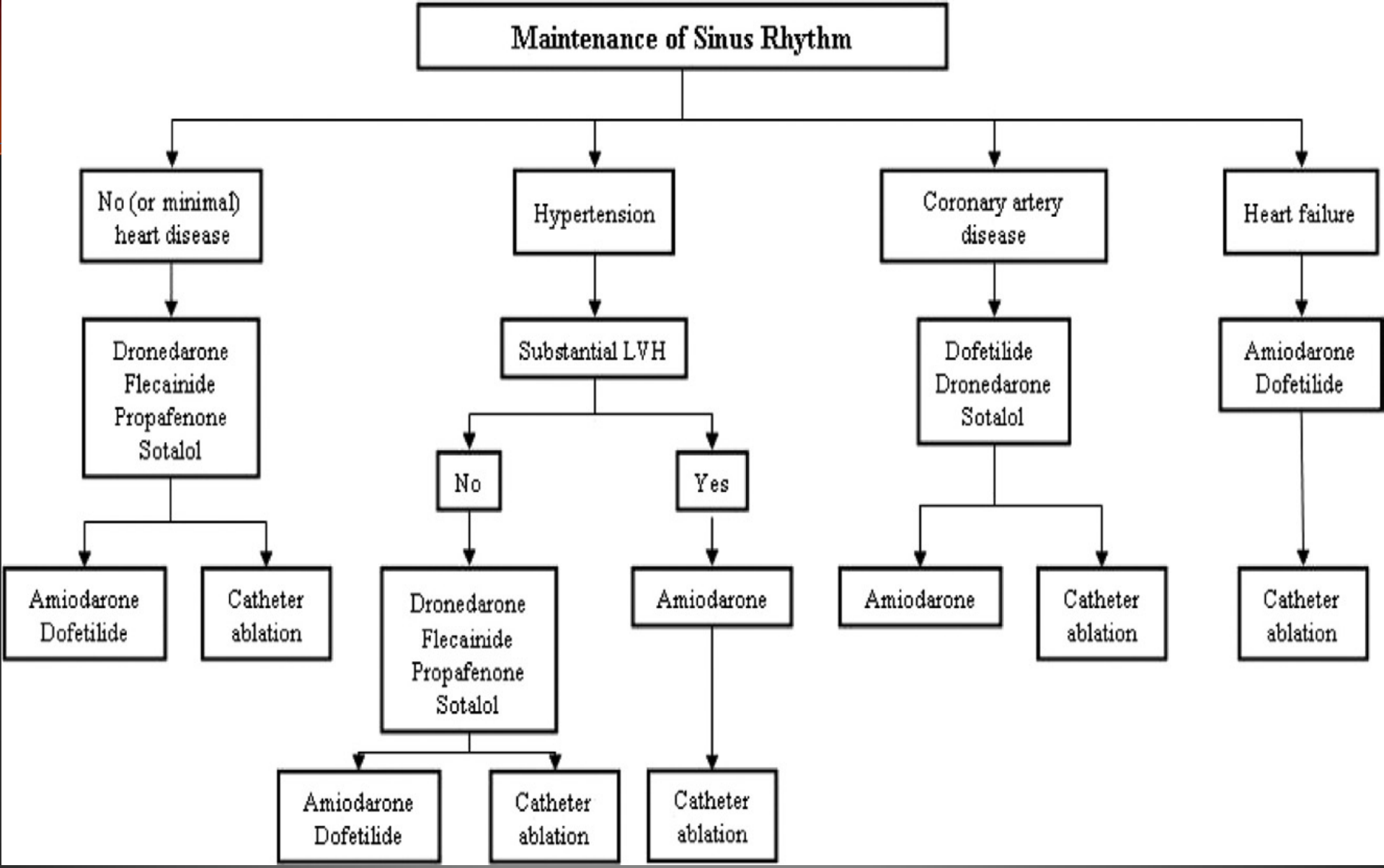
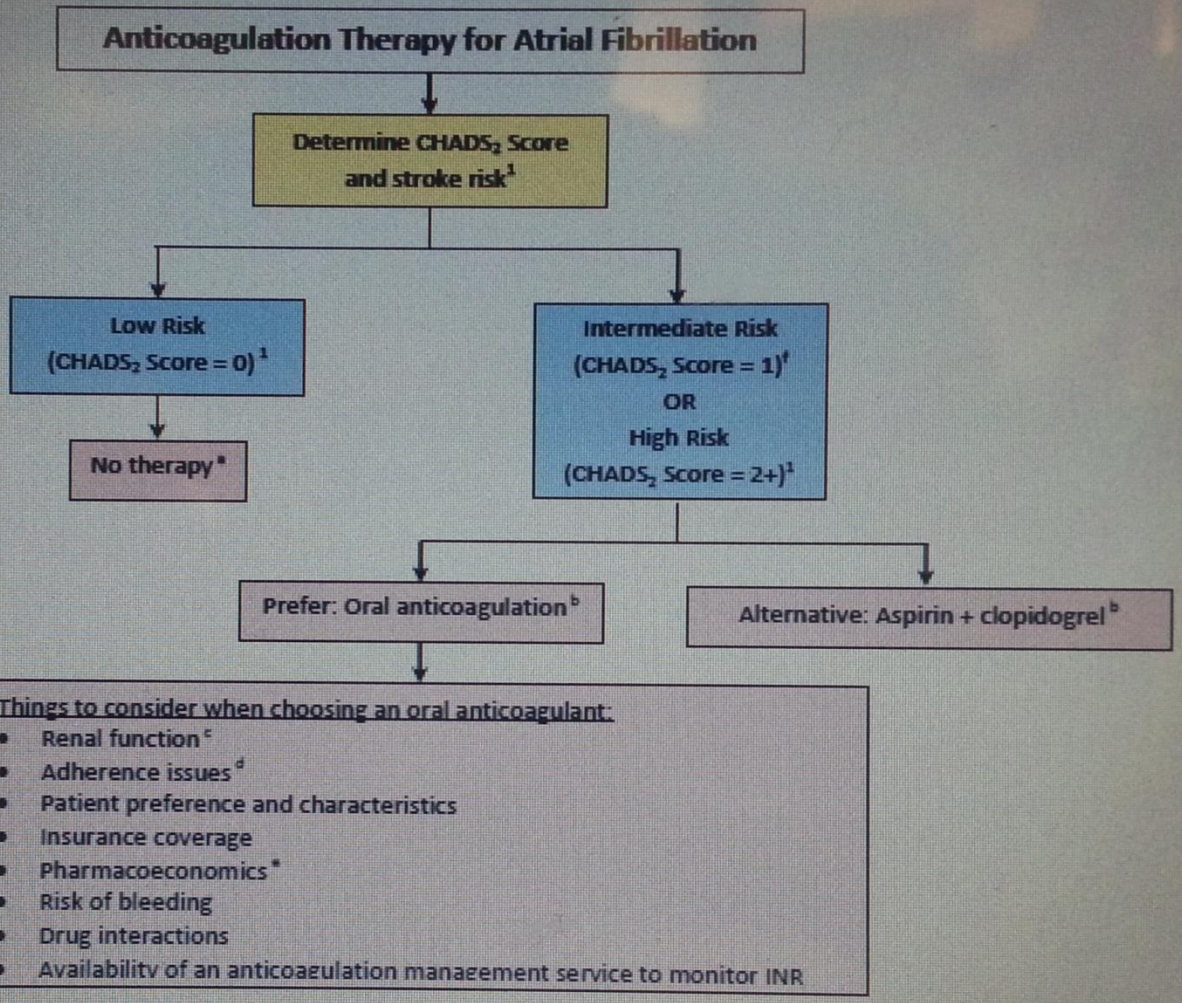


Figure 1. Therapy to maintain sinus rhythm in patients with recurrent paroxysmal or persistent atrial fibrillation. Drugs are listed alphabetically

and not in order of suggested use. The seriousness of heart disease progresses from left to right, and selection of therapy in patients with multiple conditions depends on the most serious condition present. LVH indicates left ventricular hypertrophy. Modified from Fuster et al. (2) (formerly Figure 15 from 2006 Section 8.3.3).



Refer to prescribing information for more complete information.

[a] Aspirin (75 to 325 mg daily) may be used for patients who choose antithrombotic therapy

[b] Oral anticoagulation preferred over no therapy, aspirin alone, or aspirin + clopidogrel. Aspirin (75 to 325 mg daily) + clopidogrel may be used for patients who are unsuitable for or choose not to take an oral anticoagulant (for reasons other than major bleeding).

[c] CrCl < 15 mL/min: do not use dabigatran[2] or rivaroxaban[3]; CrCl 15-30 mL/min: use lower dose of dabigatran[2]; CrCl 15-50 mL/min: use lower dose of rivaroxaban[3]

9-4-12

Table 2: Recommended Drug Doses for Heart Rhythm Control in Patients with Atrial Fibrillation

Drug*	Dose Form	Loading or Starting Dose†	Maintenance Dose†	Potential Adverse Effects**
Amiodarone[1]	Oral	Inpatient: 1.2 to 1.8 g per day in divided dose until 10 g total or 30 mg/kg as single dose Outpatient: 600 to 800 mg per day divided dose until 10 g total While 10 g desired to see max efficacy, does not have to be completed as an inpatient before fully loaded.[4]	200-400 mg per day	hypotension, bradycardia, QT prolongation, torsades de pointes (rare), GI upset, constipation, phlebitis (IV), photosensitivity, pulmonary toxicity, polyneuropathy, hepatic toxicity, thyroid dysfunction, eye complications See black box warnings for this drug
Dofetilide[1]	Oral	Creatinine Clearance → Dose > 60 mL/min = 500 mcg BID 40-60 mL/min = 250 mcg BID 20 to 40 mL/min = 125 mcg BID < 20 mL/min = Contraindicated	125-500 mcg every 12 hrs, based on renal function. Must be initiated in hospital and patient must be registered to receive this drug. Adjust dose for renal function, body size and age.	QT prolongation, torsades de pointes See black box warnings for this drug
Dronedarone[2]	Oral	400 mg twice daily, with meals	Same as starting dose	bradycardia, heart block, HF, hepatic toxicity, pulmonary toxicity, diarrhea, nausea, abdominal pain, vomiting, asthenia, stroke, death See black box warnings for this drug
Flecainide[1,2]	Oral	200-300 mg[1]‡ When starting a patient on flecainide, it is prudent to do a treadmill stress test after the patient is fully loaded.[3]	50 to 150 mg every 12 hrs[2]	hypotension, atrial flutter with high ventricular rate, ventricular tachycardia, HF Close monitoring of this drug is required. See black box warnings for this drug
Ibutilide[1,2]	IV	1 mg over 10 min; repeat 1 mg when necessary (but risk of proarrhythmia increases)	N/A	QT prolongation, torsades de pointes See black box warnings for this drug
Propafenone[1,2]	Oral	600 mg	150-300 mg every 8 hrs, or sustained release 225-425 mg every 12 hrs	hypotension, atrial flutter with high ventricular rate See black box warnings for this drug
Sotalol[1,2]	Oral	80-160 mg, to a max of 320 mg every 12 hrs, based on renal function Creatinine clearance should be calculated prior to dosing.	Same as starting dose	torsades de pointes, HF, bradycardia, exacerbation of chronic obstructive or bronchospastic lung disease See black box warnings for this drug

*Drugs are listed alphabetically. **Refer to prescribing information for more complete information.

†Dosages given in the table may differ from those recommended by the manufacturers.

‡Insufficient data are available on which to base specific recommendations for the use of one loading regimen over another for patients with ischemic heart disease or impaired left ventricular function.

Notes: AF = atrial fibrillation; BID = twice a day; GI = gastrointestinal; IV = intravenous; HR = heart rate; HF = heart failure; N/A = not applicable.
Click on drug names in table for more detailed usage information for each drug.

Drug*	Dose Form	Recommended Dose†	Major Side Effects
Apixaban (Eliquis®)[1,8]	Oral	5 mg PO twice daily Use lower dose with certain patients or medications.	hemorrhagic event See the black box warnings for this drug
Aspirin	Oral	75 to 325 mg daily	hypersensitivity, Reye's syndrome, GI bleeding, nephrotoxicity
Clopidogrel (Plavix®)[1,2,7]	Oral	75 mg daily with aspirin when warfarin therapy is unsuitable	hemorrhagic event, acute liver failure, anaphylaxis, angioedema, aplastic anemia, agranulocytosis, pancreatitis, Stevens-Johnson syndrome, thrombotic thrombocytopenia purpura See for black box warnings for this drug
Dabigatran (Pradaxa®)[1,2,5]	Oral	150 mg twice daily Use lower dose with renal impairment.	hemorrhagic event
Heparin[1,2]	IV	70 units/kg bolus, then 15 units/kg/hr infusion; adjust dose based on aPTT and hospital's nomogram[2]	hemorrhagic event, heparin-induced thrombocytopenia
Enoxaparin (Lovenox®)[1,2,6]	SC	1 mg/kg twice daily[2] Use lower dose with renal impairment.	hemorrhagic event, heparin-induced thrombocytopenia See for black box warnings for this drug
Rivaroxaban (Xarelto®)[1,4]	Oral	20 mg daily with evening meal Use lower dose with renal impairment.	hemorrhagic event See for black box warnings for this drug
Warfarin (Coumadin® or Jantoven®)[1,2,3]	IV or Oral	Individualize the dose; adjust dose based on INR Target INR = 2.5, 2.0 to 3.0. (with mechanical valve, target INR > 2.5) May need lower doses for Asians; genetic variation in CYP2C9 and VKORC1 enzymes is known; or hepatic impairment.	hemorrhagic event, tissue necrosis, systemic microemboli or cholesterol microemboli See for black box warnings for this drug

*Drugs are listed alphabetically.

Recommended Therapies for Heart Rate and Rhythm Control in Patients with Atrial Fibrillation

Whether a rate control or rhythm control strategy is chosen is very specific to each individual patient. Factors to consider are: ability to tolerate medications, degree of symptoms, degree of functional limitation, occupation, age, and other co-morbidities. While many practitioners may have preferences for a particular strategy, the ACC recommends following the guidelines referenced below[1] and considering referral to a cardiologist with experience managing heart rhythm disorders.

Table 1: Recommended Drug Doses for Heart Rate Control in Patients with Atrial Fibrillation

Drug*	Dose Form	Loading or Starting Dose†	Maintenance Dose†	Potential Adverse Effects**
Amiodarone[1,3]	IV	150 mg over 10 min	0.5-1 mg/min	hypotension, heart block, sinus bradycardia, bronchospasm, HF, pulmonary toxicity, skin discoloration, hypothyroidism, hyperthyroidism, corneal deposits, optic neuropathy, warfarin interaction See black box warnings for this drug
	Oral	800 mg PO daily x 1 week, then 600 mg PO daily x 1 week, then 400 mg PO daily x 4 to 6 weeks, then 200 mg daily	Individual to patient	
Atenolol[2]	Oral	25-100 mg daily	Same as starting dose	hypotension, heart block, bradycardia, bronchospasm, HF
Carvedilol[2]	Oral	3.125-25 mg every 12 hrs (up to 50mg every 12 hrs for patients >85 kg). May use carvedilol sustained release 10-80 mg daily	Same as starting dose	hypotension, heart block, bradycardia, bronchospasm, HF See black box warnings for this drug
Digoxin[1,2]	IV	0.25 mg every 4-6 hrs up to 1 mg	0.125-0.25 mg daily (or orally)	life threatening arrhythmia, perceived color change, heart block, bronchospasm
Diltiazem[1,2]	IV	0.25 mg/kg over 2 min. 2nd bolus can be given if HR > 100 bpm.	5-15 mg/hr	hypotension, heart block, HF
	Oral	Start with a non-sustained release dose 120-480 mg daily. Can switch to a slow-release/extended release dose, which is available and preferred	Same as starting dose	
Esmolol[1]	IV	500 mcg/kg over 1 min	50-200 mcg/kg/min	hypotension, heart block, bradycardia, bronchospasm, HF See black box warnings for this drug
Metoprolol[1,2]	IV	2.5-5 mg bolus over 2 min, up to 3 doses	N/A	hypotension, heart block, bradycardia, bronchospasm, HF See black box warnings for this drug
	Oral	25-100 mg twice daily. May use metoprolol succinate ER 25-200 mg daily	Same as starting dose	
Verapamil[1,2,4]	IV	0.075-0.15 mg/kg over 2 mins. 2nd bolus can be given in 15-30 mins if needed	N/A	hypotension, heart block, HF
	Oral	Start with a non-sustained release dose 120-480 mg daily. Can switch to a slow-release/extended release dose, which is available and preferred	Same as starting dose	

*Drugs are listed alphabetically.

†Dosages given in the table may differ from those recommended by the manufacturers. **Refer to prescribing information for more complete information.

§Amiodarone can be useful to control heart rate in patients with atrial fibrillation when other measures are unsuccessful or contraindicated

Notes: AF = atrial fibrillation; BID = twice a day; GI = gastrointestinal; IV = intravenous; HR = heart rate; HF = heart failure; N/A = not applicable.

Ia	Na/Fast-channel blockers	•Quinidine •Procainamide •Disopyramide	(Na^+) channel block (intermediate association/dissociation)	•Ventricular arrhythmias •prevention of paroxysmal recurrent atrial fibrillation (triggered by vagal overactivity), •*procainamide in Wolff-Parkinson-White syndrome
Ib		•Lidocaine •Phenytoin •Mexiletine	(Na^+) channel block (fast association/dissociation)	•treatment and prevention during and immediately after myocardial infarction, though this practice is now discouraged given the increased risk of asystole, • ventricular tachycardia • atrial fibrillation
Ic		•Flecainide •Propafenone •Moricizine	(Na^+) channel block (slow association/dissociation)	•prevents paroxysmal atrial fibrillation •treats recurrent tachyarrhythmias of abnormal conduction system. •contraindicated immediately post-myocardial infarction.!

II	Beta-blockers	•Propranolol •Esmolol •Timolol •Metoprolol •Atenolol •Bisoprolol	beta blocking Propranolol also shows some class I action	•decrease myocardial infarction mortality •prevent recurrence of tachyarrhythmias
III	Prolong AP and RP	•Amiodarone •Sotalol •Ibutilide •Dofetilide •Dronedarone •(Multaq)	K^+ channel blocker Sotalol is also a beta blocker[7]	•In Wolff-Parkinson-White syndrome •(sotalol:) ventricular tachycardias and atrial fibrillation •(Ibutilide:) atrial flutter and atrial fibrillation
IV	Calcium/slow-channel blockers	•Verapamil •Diltiazem	Ca^{2+} channel blocker	•prevent recurrence of paroxysma supraventricular tachycardia •reduce ventricular rate in patients with atrial fibrillation
V		•Adenosine •Digoxin	Work by other or unknown mechanisms (Direct nodal inhibition).	Used in supraventricular arrhythmias, especially in Heart Failure with Atrial Fibrillation, contraindicated in ventricular arrhythmias.

Amiodarone (Pacerone, Cordarone)	100-400 mg/day	Liver, lung, thyroid, skin,neuro toxicity, interaction with warfarin/digoxin etc
Sotalol (k blocker) (Betapace)	80-320 mg/day	QTc prolongation, Torsades, Renal excretion, CAD pts
Dofetilide (k blocker) (Tikosyn)	500-1000 ug/day (Inpatient load!!!)	QTc prolongation, Torsades, Renal excretion, Drug interactions, CAD/CHF pts
Flecainide (Tambocor)	200-300 mg/day	VT, enhanced AVN conduction
Propafenone (Rythmol)	450 – 900 mg /day	VT, enhanced AVN conduction
Quinidine	600 – 1500 mg /day	VT, enhanced AVN conduction, Last resort in ICD pts
Disopyramide (Norpace)	400-750 mg /day	VT, Last resort in ICD pts
Procainamide	1000-4000 mg /day	VT, Lupus, last resort in ICD pts
Dronedarone (Multaq)	400 mg PO BID	Renal failure, Contraindicated in NYHA III-IV, thyroid/skin toxicities
Ibutilide (k blocker) (Corvert)	1 mg IV (with Mg)	PMVT 3.6-8.3%, Safe only if LVEF >30%

European Society of Cardiology – Guidelines for Management of Atrial Fibrillation Sept 2010

New Anticoagulation Guidelines

CHA_2DS_2-VASc	Score
CHF/LV Dysfunction	1
Hypertension	1
Age >75	2
Diabetes Mellitus	1
Stroke/TIA/Thrombo-Embolism	2
Vascular Disease	1
Age 65-74	1
Sex – Female	1
Total Score Possible	9

European Society of Cardiology – Guidelines for Management of Atrial Fibrillation Sept 2010

Adjusted Stroke Risk

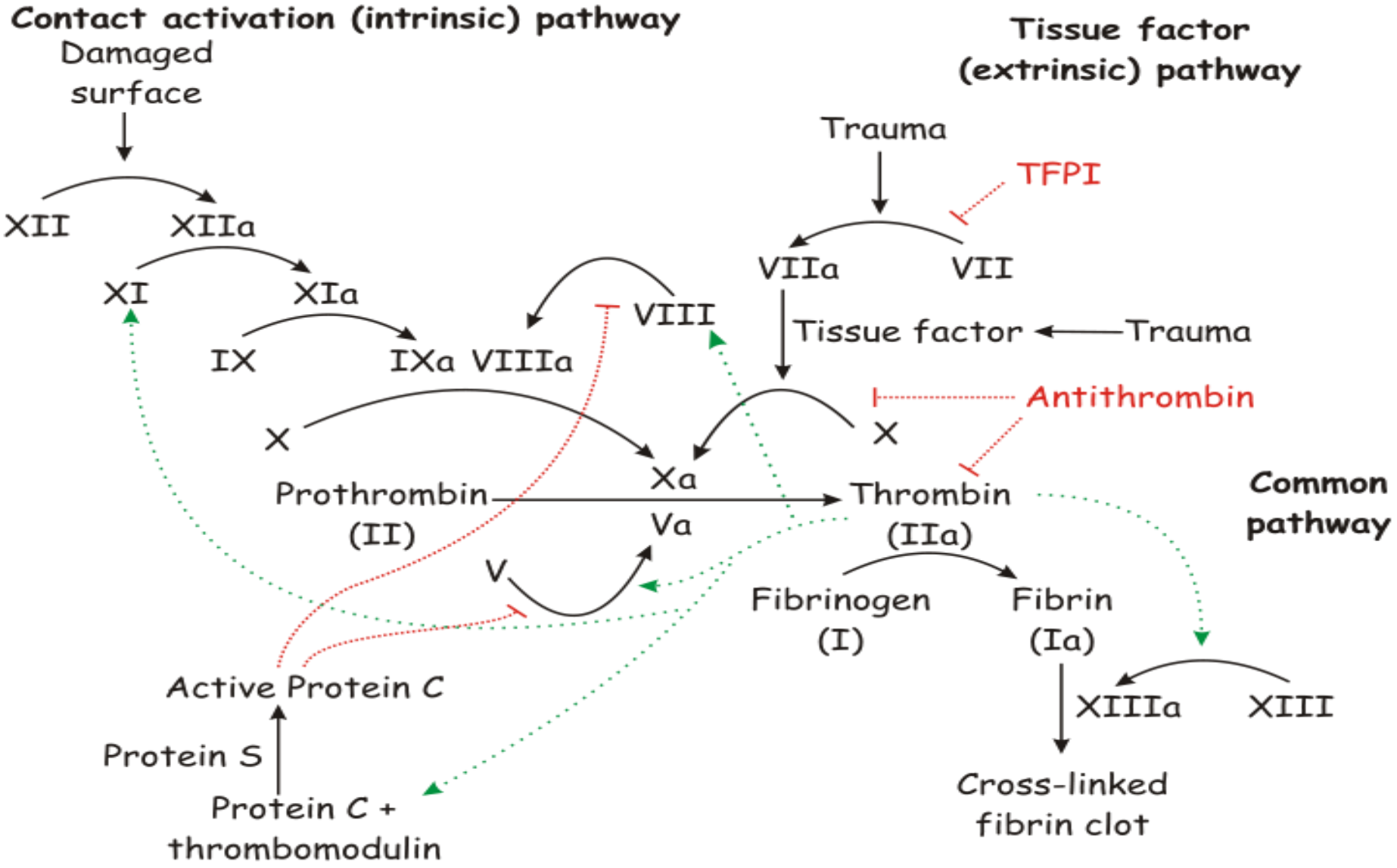
CHA_2DS_2-VASc	% Risk/year	Suggested Tx
0	0%	ASA 75-325 mg
1	1.3%	ASA 75-325 mg
2	2.2%	OAC
3	3.2%	
4	4.0%	
5	6.7%	
6	9.8%	
7	9.6%	
8	6.7%	
9	15.2%	

Bleeding Risk HAS-BLED Score

HAS-BLED Characteristic		Score
Hypertension		1
Abnormal Renal Function		1
Abnormal Liver Function		1
Stroke - Hemorrhagic		1
Bleeding		1
Labile INR's		1
Elderly > 65y/o		1
Drugs/Etoh	1 pt each	1-2
Total Possible		**9**

- If score ≥ 3, pt is at "High Risk" Needs regular follow up

Coagulation Cascade



Anticoagulants

- **Vitamin K antagonist**
  - Coumadin/Jantoven- Warfarin
- **Factor Xa Inhibitors** – Approved for prevention of Thromboembolic events related to AFib
  - Xarelto - Rivaroxaban
  - Abixiban – Not yet on the market but we are anxiously awaiting it
- **Direct Thrombin Inhibitor**
  - Pradaxa - Dabigatran

Vit K Antagonist

- Vit K is essential for hepatic synthesis of Factors II, VII, IX, and X, as well as Protein C and S
- VKA – therefore stops the coagulation process on multiple levels, which is useful in the prevention of clots
- Complications
  - Narrow therapeutic window
  - Frequent monitoring
  - Easily affected by Vit K rich foods and medications
  - Metabolized in the cytochrome P450, CYP2C9

Warfarin

- 5 prospective randomized controlled clinical trials 3711 non valvular Afib patients
- 60-86% Risk Reduction of Thromboemolic Event compared to Placebo
- Incidence of major Bleeding 0.6-2.7%
- INR range 1.4-4.5
- Peak Anticoagulation affect 72-96 hours
- Duration of action 2-5 days
- Reversed with Vit K and Fresh Frozen Plasma

Factor Xa Inhibitors

- Mechanism of Action - Block activity of clotting Factor Xa.
- Factor Xa is generated in the extrinsic and intrinsic coagulation pathways, which activates prothrombin and thrombin which triggers the final components of the coagulation pathway to form clots

Advancements in Anticoagulation

- Factor Xa Inhibitors – Apixaban
- **AVERROES: Efficacy and Safety Endpoints**
- **Average CHADS2 Score in ARISTOTLE Trial = 2.1**

Endpoint	Apixaban	Aspirin	Hazard Ratio (95% CI)	*P* Value
Stroke or systemic embolism (% per year)	1.6	3.7	0.45 (0.32 - 0.62)	< .001
Mortality (% per year)	3.5	4.4	0.79 (0.62 - 1.02)	.07
Major bleeding (% per year)	1.4	1.2	1.13 (0.74 - 1.75)	.57
Intracerebral bleeding (n)	11	13	NA	NA
First cardiac hospitalization (% per year)	12.6	15.9	0.79 (0.69 - 0.91)	< .001

	Rivaroxaban	Apixaban	Dabigatran	Warfarin
Brand name	Xarelto®	Eliquis®	Pradaxa®	Coumadin®
Manufacturer	Bayer HealthCare and Janssen Research and Development, LLC	Bristol-Myers Squibb and Pfizer	Boehringer Ingelheim	Bristol-Myers Squibb
Mechanism of action	Factor Xa inhibitor	Factor Xa inhibitor	Thrombin inhibitor	Vitamin K antagonist
Prodrug	No	No	Yes	No
Bioavailability, %	~80[75]	~66[76]	7.2[77]	~100[78]
Protein binding, %	92–95	87	35	99[79]
Coagulation monitoring required	No	No	No	Yes
T_{max}, h	2–4[75,80]	0.5–2[81]	1.25–1.5[82]	Slow onset (peak anticoagulant effect may take up to 96 h after administration)[83]
$T_{½}$, h	7–11[50]	Average 12.7[81]	7–17[82]	~40[83]
Main mode of elimination	Renal/hepatobiliary[84]	Renal/faecal[81]	Renal[85]	Renal[78]
Drug interactions	Potent inhibitors of CYP3A4 and P-gp[86]	Potent inhibitors of CYP3A4[87]	Potent inhibitors of P-gp[87]	Multiple drugs, dietary vitamin K[78,88]
Antidote	No	No	No	Yes
Typical effective dose	10 mg od (VTE prophylaxis)* 15 mg bid (days 1–21) followed by 20 mg od (DVT treatment/prevention of recurrent VTE)† 20 mg od (AF)‡	2.5 mg bid (VTE prophylaxis)*	150 mg or 220 mg od (VTE prophylaxis)* 75 mg or 150 mg bid (AF)‡	INR-guided
Approved indications	Approved for VTE prevention after elective hip or knee replacement in adults, for prevention of stroke and systemic embolism in patients with non-valvular AF, and for treatment of acute DVT and prevention of VTE recurrence	Approved for VTE prevention after elective hip or knee replacement in adults	Approved for VTE prevention after elective hip or knee replacement in adults and for prevention of stroke and systemic embolism in patients with non-valvular AF	Approved for VTE prevention and treatment, prevention and/or treatment of the thromboembolic complications associated with AF and/or cardiac valve replacement, and secondary prevention after MI

Xarelto - rivaroxaban

- Indicated for Non Valvular Atrial Fibrillation
- Use of drugs that interact with CYP3A4 Inhibitors may increase bleeding risk
- NO Known Reversal Agent
- ROCKET AF Trial – 7111 patients
- Average CHA2DS2 Score = 3.5
- Xarelto relatively equal risk of Stroke Prevention compared to Coumadin
- Major Bleeding 5.6 (Xarelto) Vs. 5.4 (Warfarin)

Direct Thrombin Inhibitor

- Thrombin plays a central role in thrombus formation through its conversion of fibrinogen to fibrin and activation of platelets as well as amplifying its own generation by feedback activation via factors V, VIII, and XI.

Pradaxa - dabigatran

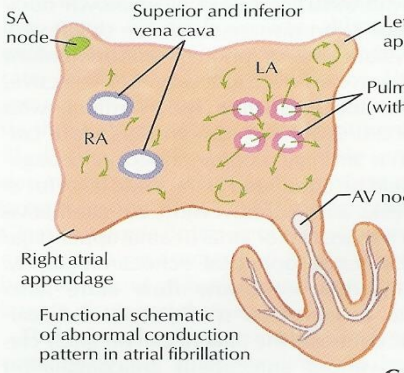
- RE-LY Trial 18, 113 patients
- 1.11% risk of Stroke with 150mg dabigatran vs 1.69% on warfarin (significant)
- Risk of Major Bleeding 3.36 on Warfarin and 3.11 on dabigatran (not clinically significant)
- New England Journal of Medicine September 17, 2009
- Average CHA2DS2 Score = 2.1

Surgical and Percutaneous Tx of AFib

- **Corridor Procedure** – Surgical pathway from SA to AV node
- **MAZE Procedure** – Incisions to isolate and interrupt abnormal re-entry circuits
- **Radiofrequency Catheter Ablation** – Isolates foci of early depolarizing atrial cells around the pulmonary veins
- **AV Node Ablation/Permanent Pacemaker Implantation** – SN Dysfunction, Tachy-Brady Syndrome, Excessive Bradycardia d/t medications.

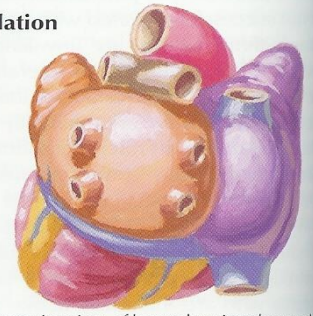
Intra Operative Surgical Techniques

Figure 19-3 Surgical Management of Atrial Fibrillation



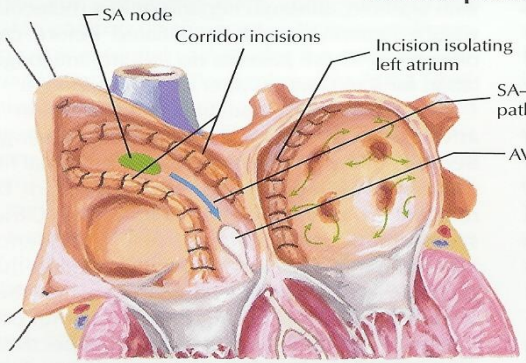
Functional schematic of abnormal conduction pattern in atrial fibrillation

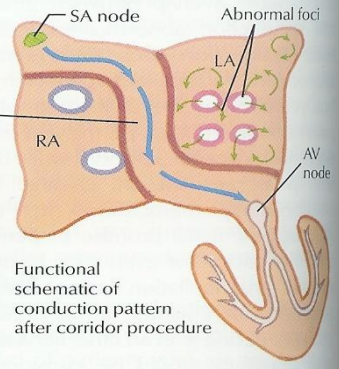
Atrial fibrillation



Posterior view of heart showing abnormal electrical foci and re-entry circuits

Corridor procedure

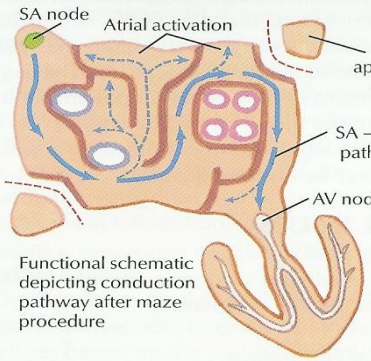




Functional schematic of conduction pattern after corridor procedure

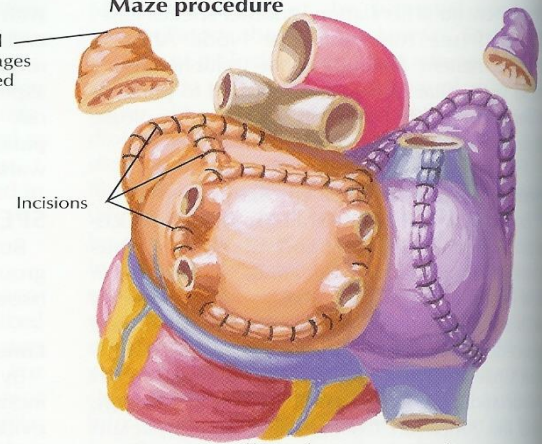
Corridor procedure isolates SA—AV nodal pathway from abnormal foci but does not halt fibrillation or restore AV synchrony

JOHN A. CRAIG, MD © ICON



Functional schematic depicting conduction pathway after maze procedure

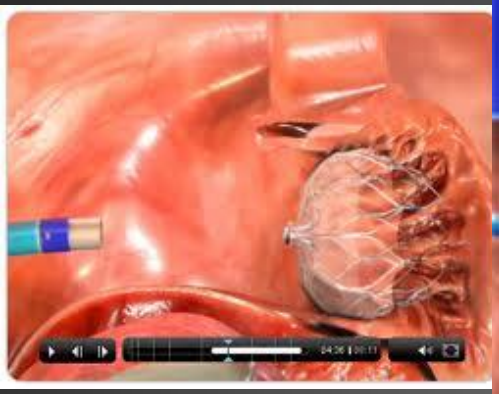
Maze procedure



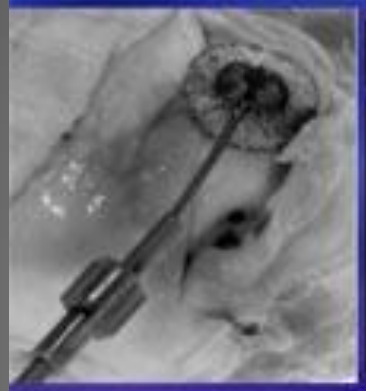
Posterior view of heart showing pattern of incisions to isolate and abolish abnormal conduction

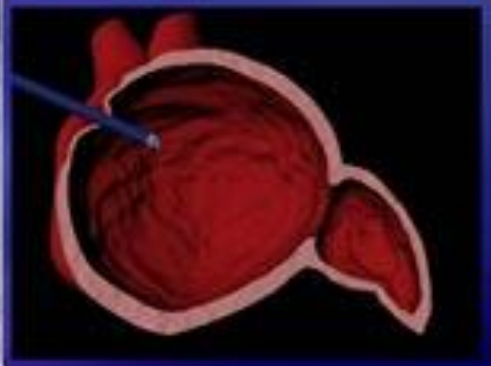
Incision pattern of maze procedure isolates and interrupts abnormal re-entry circuits and provides a single pathway from sinus node (SA) to AV node and simultaneously activates both atria, abolishes fibrillation, and restores AV synchrony

New Treatments – Watchman Device



LAA Occlusion Devices





- No need for LONG-TERM anticoagulation
- Can be done in moderately sedated patient

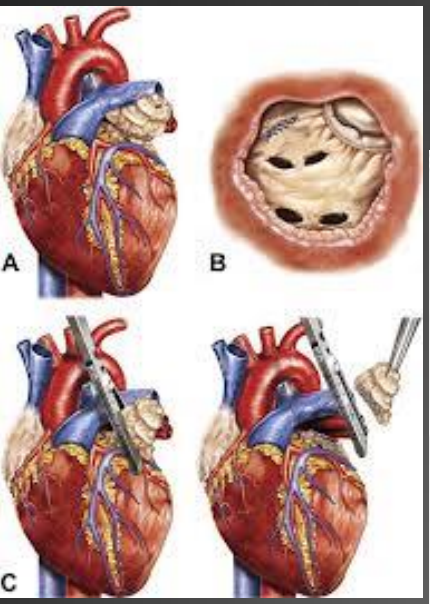
BUT:

- Need for transseptal puncture
- Foreign body in the central circulation:
  - Erosion/migration
  - Perforation/effusion
  - Infection
  - Need for anticoagulation!
- Need to size device to ostium
- Complex LAA – incomplete closure

MAYO CLINIC

Holmes DR, Jr. American College of Cardiology; March 2009; Orlando, Fl.

New Treatments – LARIAT Snare



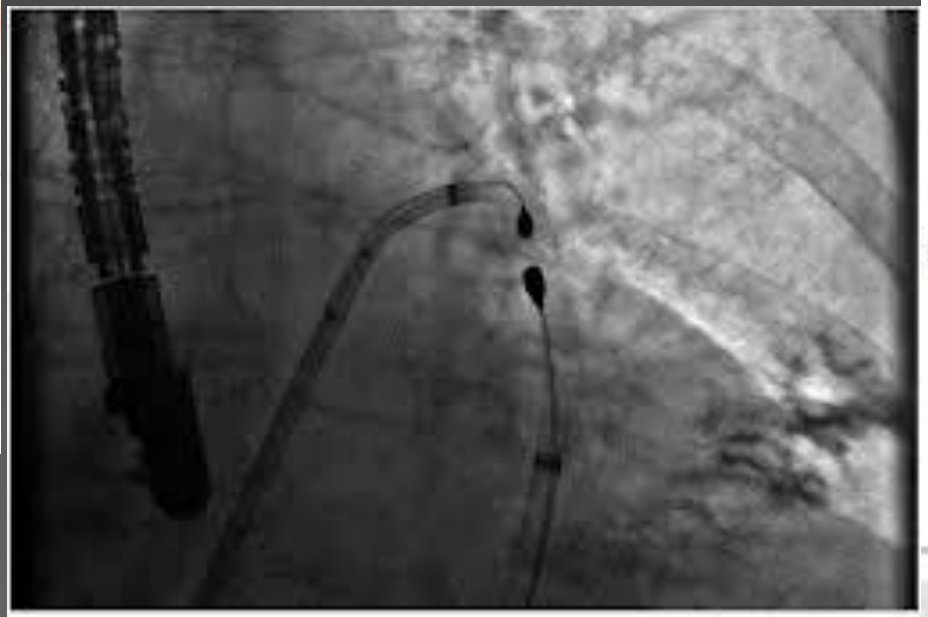


Figure 3: Pericardial magnet-tipped wire introduced and approaching intra-appendicular body-wire.

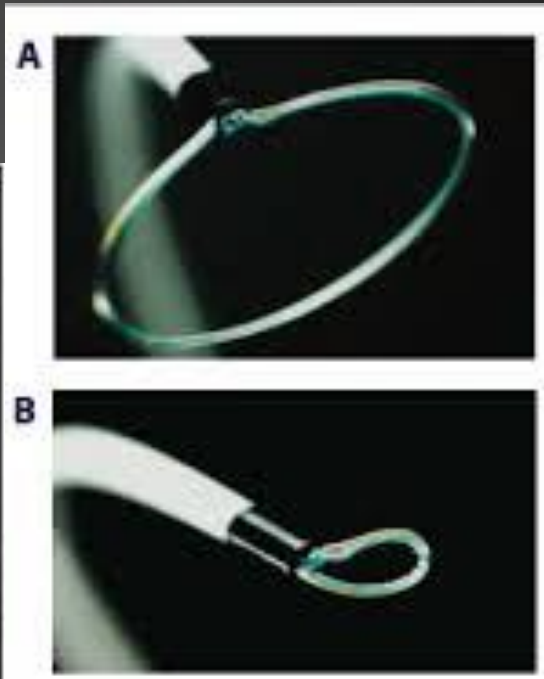
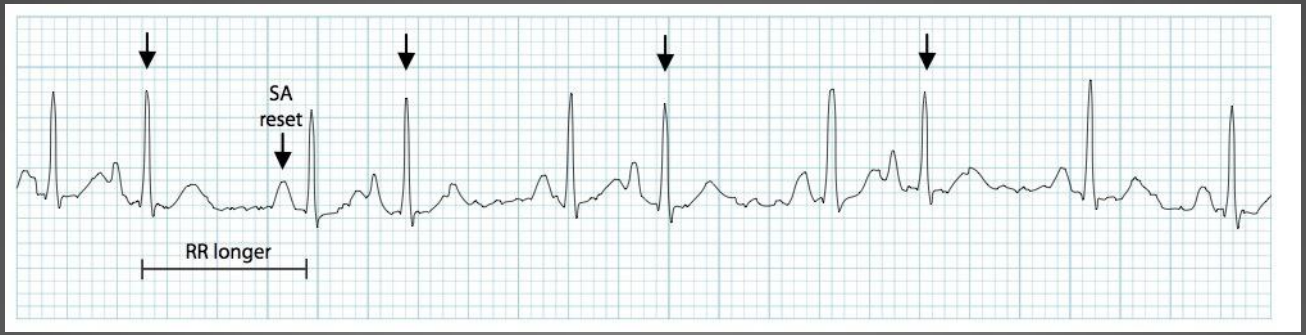


Figure 2: The LARIAT snare is supplied with a pre-loaded, pre-knotted polyester suture. The top panel shows the device open, prior to advancement over the LAA. The bottom panel shows it after tightening of the snare, prior to final closure of the suture.

Sinus Arrhythmias – APC's



APC's

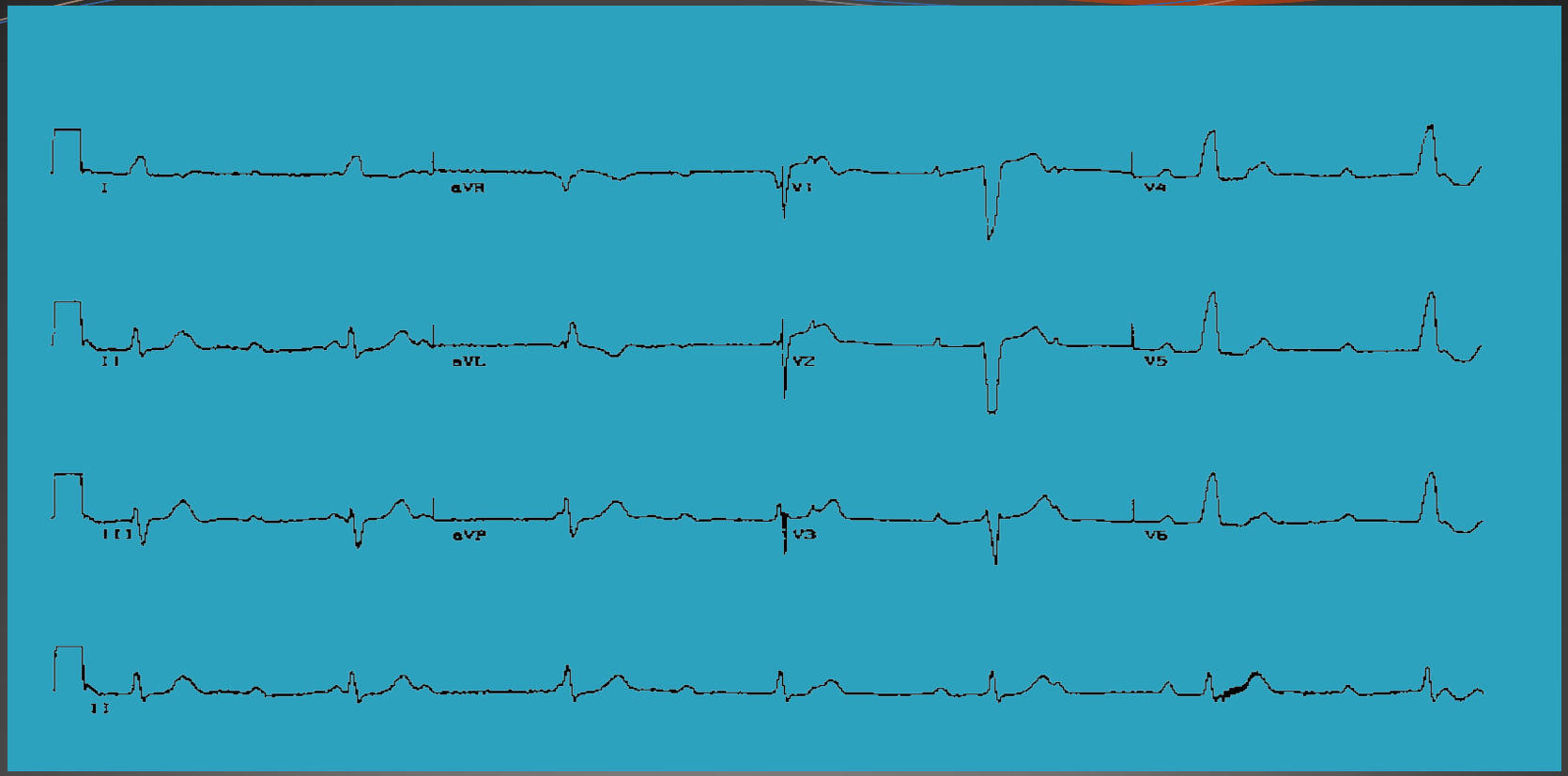
- **Atrial Ectopics**
  - Arise from ectopic pacemaking tissue within the atria.
  - There is an abnormal P wave, usually followed by a normal QRS complex.
- **Electrocardiographic Features**
  - An abnormal (non-sinus) P wave is followed by a QRS complex.
  - P wave typically has a different morphology and axis to the sinus P waves.
  - The abnormal P wave may be hidden in the preceding T wave, producing a "peaked" or "camel hump" appearance — if this is not appreciated the PAC may be mistaken for a PJC.
  - PACS arising close to the AV node ("low atrial" ectopics) activate the atria retrogradely, producing an inverted P wave with a relatively short PR interval ≥ 120 ms (PR interval < 120 ms is classified as a PJC).
  - PACs that reach the SA node may depolarize it, causing the SA node to "reset" — this results in a longer-than-normal interval before the next sinus beat arrives ("post-extrasystolic pause"). Unlike with PVCs, this pause is not equal to double the preceding RR interval (i.e. not a "full compensatory pause").
  - PACs arriving early in the cycle may be conducted aberrantly, usually with a RBBB morphology (as the right bundle branch has a longer refractory period than the left). They can be differentiated from PVCs by the presence of a preceding P wave.
  - Similarly, PACs arriving very early in the cycle may not be conducted to the ventricles at all. In this case, you will see an abnormal P wave that is not followed by a QRS complex ("blocked PAC"). It is usually followed by a compensatory pause as the sinus node resets.

APC's

- **Patterns**
  - PACs often occur in repeating patterns:
  - **Bigeminy** — every other beat is a PAC.
  - **Trigeminy** — every third beat is a PAC.
  - **Quadrigeminy** — every fourth beat is a PAC.
  - **Couplet** – two consecutive PACs.
  - **Triplet** — three consecutive PACs.
- **Clinical Significance**
  - PACs are a normal electrophysiological phenomenon not usually requiring investigation or treatment.
  - Frequent PACs may cause palpitations and a sense of the heart "skipping a beat".
  - In patients with underlying predispositions (e.g. left atrial enlargement, ischaemic heart disease, WPW), a PAC may be the trigger for the onset of a re-entrant tachydysrhythmia — e.g. AF, flutter, AVNRT, AVRT.
- **Causes**
  - Anxiety.Sympathomimetics.Beta-agonists, Excess caffeine, Hypokalaemia., Hypomagnesaemia, Digoxin toxicity, Myocardial ischaemia

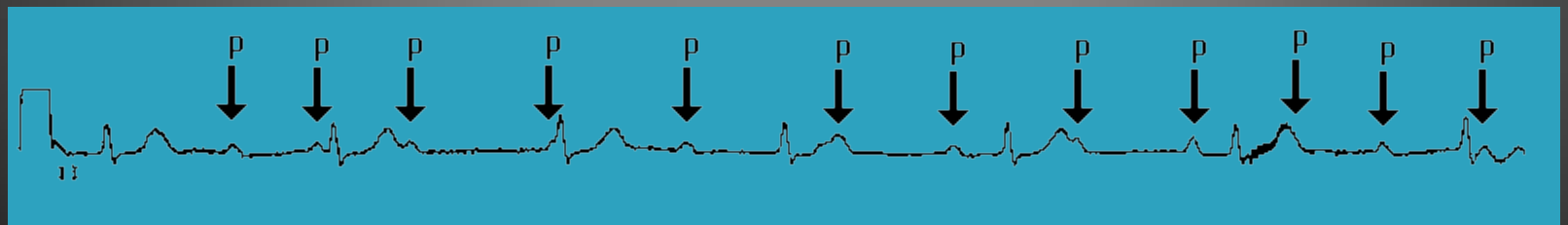
Wagner GS Marriott's Practical Electrocardiography (11th edition), Lippincott Williams & Wilkins 2007

CHB - A 70 year old man with exercise intolerance.



Complete Heart Block

- **P waves are not conducted to the ventricles because of block at the AV node.**
- **The P waves are indicated below and show no relation to the QRS complexes.**
- **They 'probe' every part of the ventricular cycle but are never conducted.**
- **The ventricles are depolarised by a ventricular escape rhythm.**



Thank you

- Questions?