

Management of Cardiac Arrhythmias and Conduction Disorders

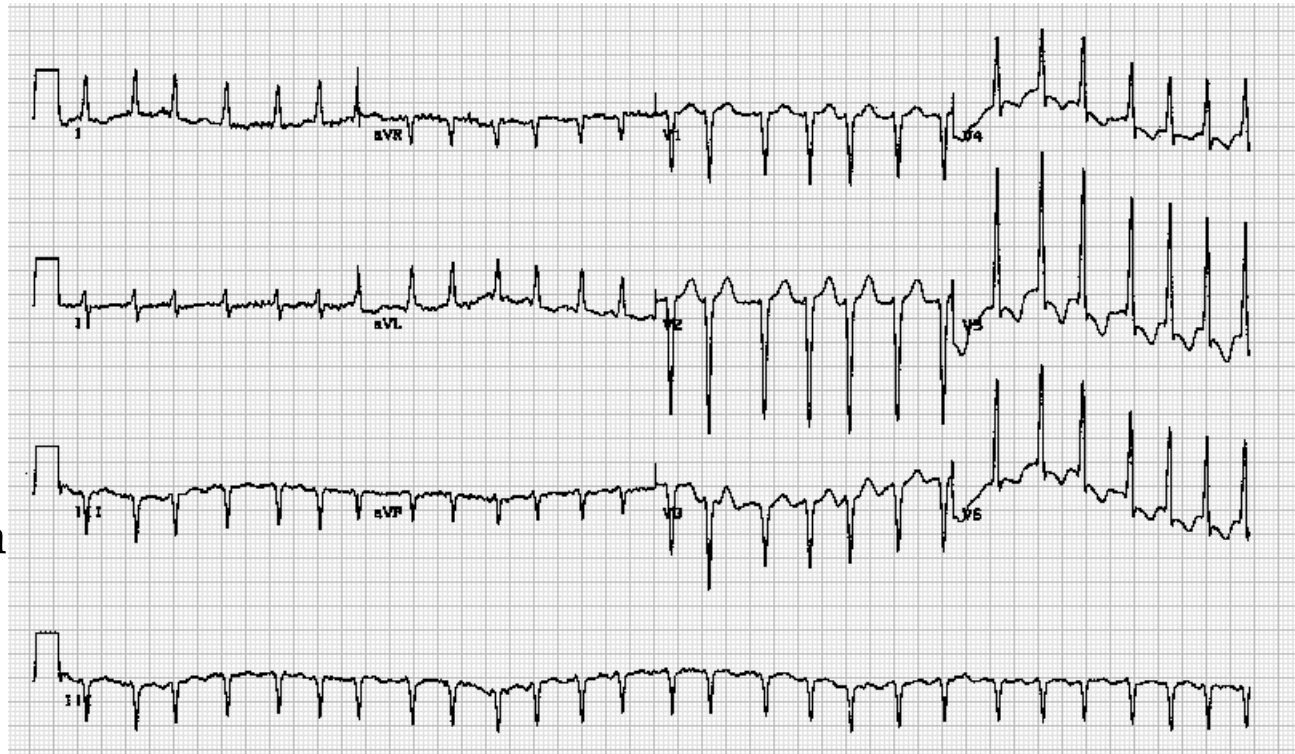
Robert J. Chilton, DO, FACOI

No Disclosures

Management of cardiac arrhythmias and conduction disorders

Case 1

- ▣ JR is a 76 y/o male who presents to your office with breathlessness and heart palpitations that are recent in onset.
- ▣ PMH: hypertension, hyperlipidemia
- ▣ Current meds: HCTZ 12.5 mg/day and atorvastatin 40 mg/qd
- ▣ Labs
 - K=3.8
 - LDL=75
- ▣ Physical
 - BP 138/78
 - No S3
 - Lungs-clear
 - Ext-no edema



Does the patient require thyroid testing?

- ▣ **Overt thyroid dysfunction** can be the sole cause of AF and may predispose to AF-related complications. In recent studies, hyperthyroidism or hypothyroidism was found to be **relatively uncommon** in AF populations, but subclinical thyroid dysfunction may contribute to AF
- ▣ **AF occurs in 10–25% of patients**, with **hyperthyroidism especially in men and the elderly**. Treatment is aimed primarily at restoring a euthyroid state, which may be associated with a spontaneous reversion to sinus rhythm.



Hyperthyroidism

Propranolol

Less negative inotrope

Class I

1. Administration of a beta blocker is recommended to control the rate of ventricular response in patients with AF complicating thyrotoxicosis, unless contraindicated. (*Level of Evidence: B*)
2. In circumstances when a beta blocker cannot be used, administration of a nondihydropyridine calcium channel antagonist (diltiazem or verapamil) is recommended to control the ventricular rate in patients with AF and thyrotoxicosis. (*Level of Evidence: B*)
3. In patients with AF associated with thyrotoxicosis, oral anticoagulation (INR 2.0 to 3.0) is recommended to prevent thromboembolism, as recommended for AF patients with other risk factors for stroke. (*Level of Evidence: C*)

What is heart rate target?

Table 2. Recommendation for Rate Control During Atrial Fibrillation

2011 Focused Update Recommendation	Comments
Class III—No Benefit	
1. Treatment to achieve strict rate control of heart rate (<80 bpm at rest or <110 bpm during a 6-minute walk) is not beneficial compared to achieving a resting heart rate <110 bpm in patients with persistent AF who have stable ventricular function (left ventricular ejection fraction >0.40) and no or acceptable symptoms related to the arrhythmia, though uncontrolled tachycardia may over time be associated with a reversible decline in ventricular performance. ³ (<i>Level of Evidence: B</i>)	New recommendation

Resting HR below 110



RACE II (Rate Control Efficacy in Permanent Atrial Fibrillation) trial of 614 patients with permanent AF

Primary endpoints were death from cardiovascular causes, hospitalization for heart failure, stroke, systemic embolism, bleeding, and life-threatening arrhythmias.

The 3-year estimated cumulative incidence of the primary outcome was 12.9% in the lenient-control group and 14.9% in the strict-control group

N Engl J Med. 2010;362:1363–73

Table 4. Recommendations for Use of Dronedarone in Atrial Fibrillation

2011 Focused Update Recommendations	Comments
Class IIa	
1. Dronedarone is reasonable to decrease the need for hospitalization for cardiovascular events in patients with paroxysmal AF or after conversion of persistent AF. Dronedarone can be initiated during outpatient therapy. ²⁹ <i>(Level of Evidence: B)</i>	New recommendation
Class III–Harm	
1. Dronedarone should not be administered to patients with class IV heart failure or patients who have had an episode of decompensated heart failure in the past 4 weeks, especially if they have depressed left ventricular function (left ventricular ejection fraction $\leq 35\%$). ³⁰ <i>(Level of Evidence: B)</i>	New recommendation

Increased mortality after dronedarone therapy for severe heart failure. N Engl J Med. 2008;358: 2678–87.

Considerations for diagnosis and initial management

Recommendations	Class ^a	Level ^b
The diagnosis of AF requires documentation by ECG.	I	B
In patients with suspected AF, an attempt to record an ECG should be made when symptoms suggestive of AF occur.	I	B
A simple symptom score (EHRA score) is recommended to quantify AF-related symptoms.	I	B
All patients with AF should undergo a thorough physical examination, and a cardiac- and arrhythmia-related history should be taken.	I	C
In patients with severe symptoms, documented or suspected heart disease, or risk factors, an echocardiogram is recommended.	I	B
In patients treated with antiarrhythmic drugs, a 12-lead ECG should be recorded at regular intervals during follow-up.	I	C

European Heart Journal (2010) 31, 2369–2429



Points

Cardiac failure-1

Hypertension-1

Age>75-1

Stroke-2

Table 7 CHADS₂ score and stroke rate

CHADS ₂ score	Patients (n=1733)	Adjusted stroke rate (%/year) ^a (95% confidence interval)
0	120	1.9 (1.2–3.0)
1	463	2.8 (2.0–3.8)
2	523	4.0 (3.1–5.1)
3	337	5.9 (4.6–7.3)
4	220	8.5 (6.3–11.1)
5	65	12.5 (8.2–17.5)
6	5	18.2 (10.5–27.4)

CHADS 2 score ≥ 2, chronic OAC

CHADS 2 [cardiac failure, hypertension, age>75, diabetes, stroke (2 points)] (SPAF)

JAMA 2001;285:2864–2870

Rate control recommendations

Recommendations	Class ^a	Level ^b
β -Blockers are recommended as first-line therapy to control the ventricular rate in patients with heart failure and low LVEF.	I	A
Where monotherapy is inadequate for heart rate control, digoxin should be added.	I	B
In haemodynamically unstable patients with acute heart failure and low LVEF, amiodarone is recommended as the initial treatment.	I	B
If an AP is excluded, digoxin is recommended as an alternative to amiodarone to control the heart rate in patients with AF and acute systolic heart failure.	I	C



Pre-post Op Afib

Recommendations	Class ^a	Level ^b
Oral β -blockers are recommended to prevent post-operative AF for patients undergoing cardiac surgery in the absence of contraindications.	I	A
If used, β -blockers (or other oral antiarrhythmic drugs for AF management) are recommended to be continued until the day of surgery.	I	B
Ventricular rate control is recommended in patients with AF without haemodynamic instability.	I	B
Restoration of sinus rhythm by DCC is recommended in patients who develop post-operative AF and are haemodynamically unstable.	I	C
Pre-operative administration of amiodarone should be considered as prophylactic therapy for patients at high risk for post-operative AF.	IIa	A



Ablation of atrial fibrillation

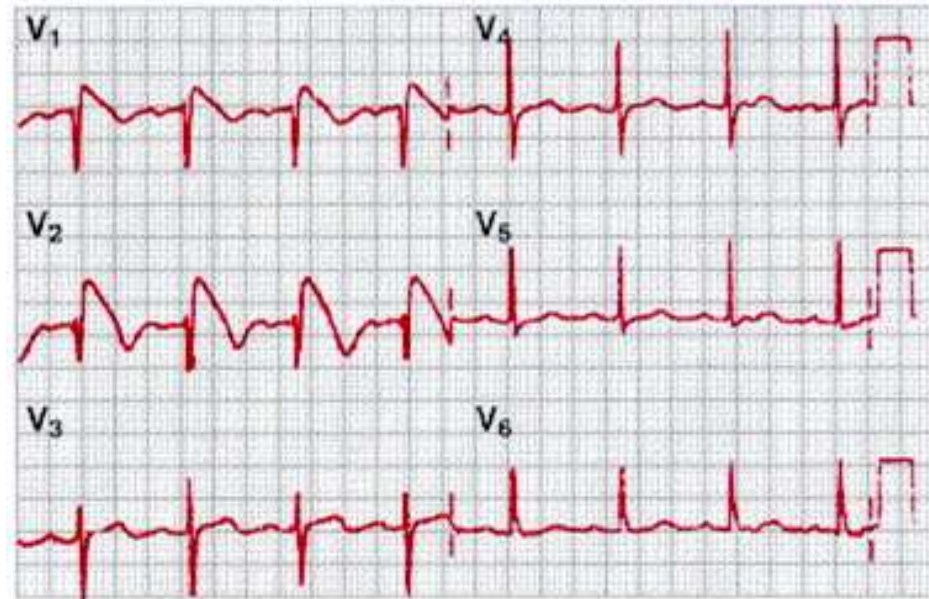
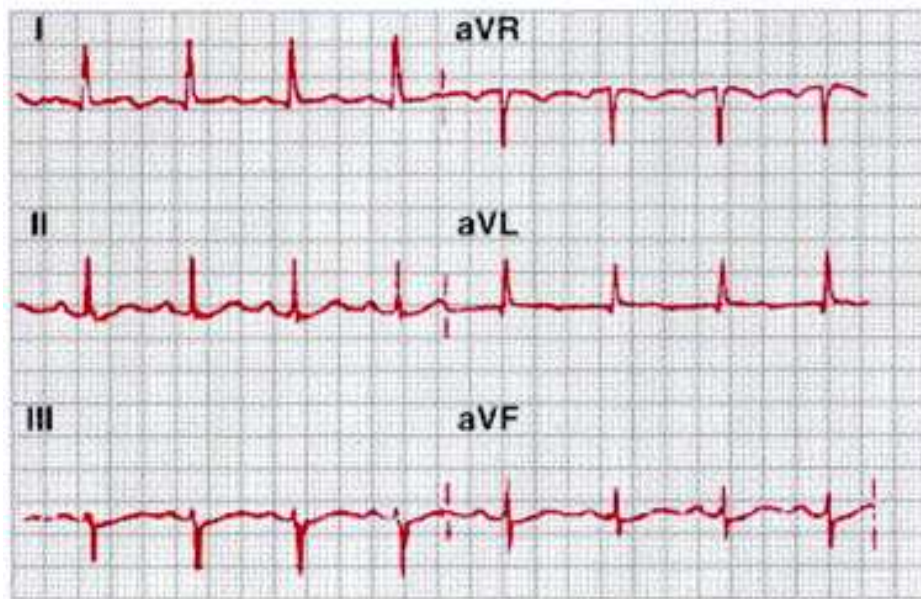
- ▣ Class I---**NEW 2011**
 - Catheter ablation performed in experienced centers* is useful in maintaining sinus rhythm in selected patients with significantly symptomatic, paroxysmal AF who have **failed treatment with an antiarrhythmic drug and have normal or mildly dilated left atria, normal or mildly reduced LV function, and no severe pulmonary disease.** (Level of Evidence: A)

Summary points for atrial fibrillation

- ▣ AF occurs in 10–25% of patients, with hyperthyroidism especially in elderly men
- ▣ AF heart rate control in hyperthyroidism, BB or diltiazem
- ▣ Resting HR below 110
- ▣ Increased mortality after dronedarone therapy for severe heart failure
- ▣ CHADS 2 [cardiac failure, hypertension, age>75, diabetes, stroke (2 points)] (SPAF)

CASE 2

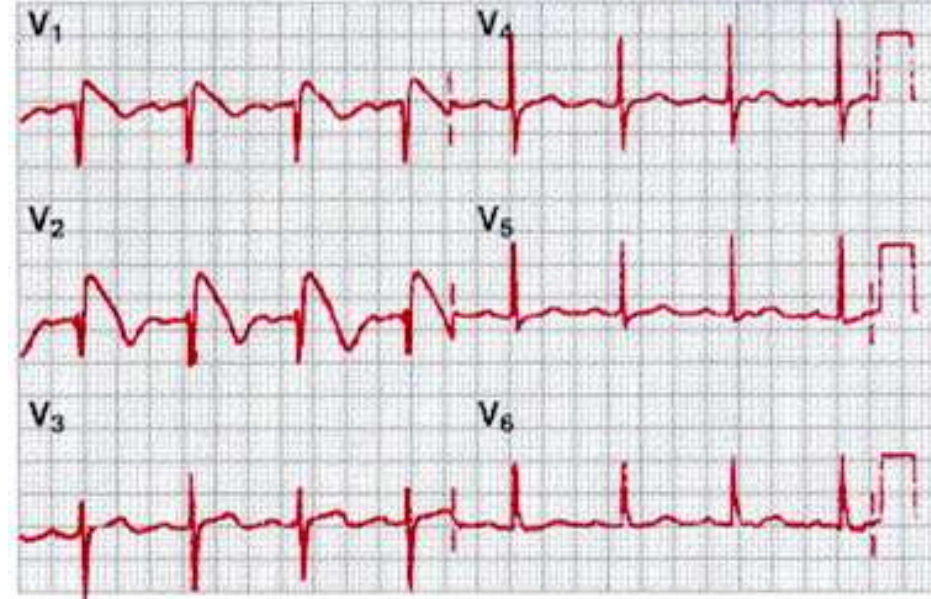
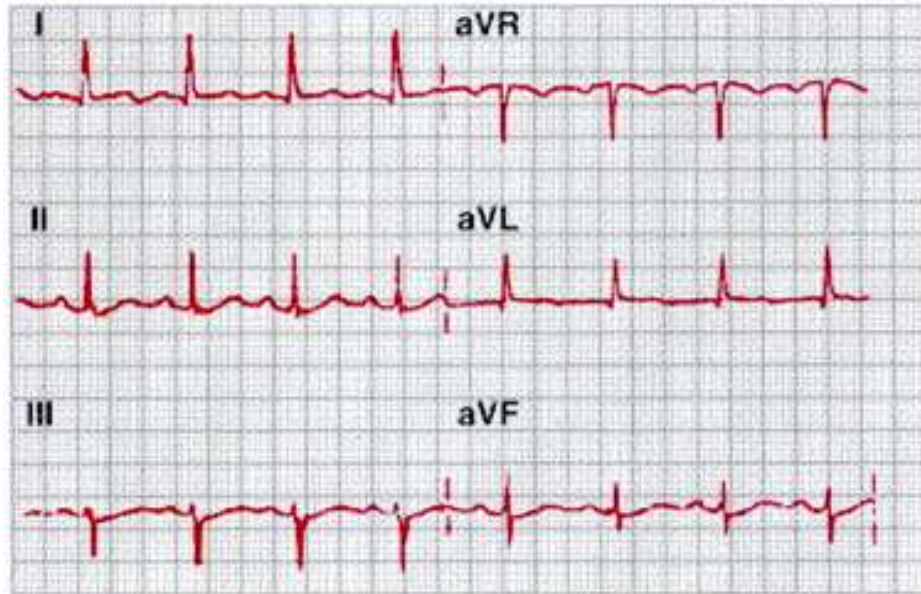
32 y/o male presents to ER after jogging 5 miles and getting CPR. Cardiac enzymes were normal, Lab neg, CXR normal, physical neg. What is your treatment?



Pick the best treatment option

- 1. Beta blocker**
- 2. Amiodarone**
- 3. Sotalol**
- 4. AICD**
- 5. Procainamide**

32 y/o male presents to ER after jogging 5 miles and getting CPR Cardiac enzymes were normal, Lab neg, CXR normal, physical neg. What is your treatment?



1. Beta blocker
2. Amiodarone
3. Sotalol
- 4. AICD-answer**
5. Procainamide

Brugada syndrome

- ▣ Distinct form of idiopathic ventricular fibrillation
- ▣ RBBB and ST segment elevation in the anterior precordial leads
- ▣ No evidence of structural heart disease
- ▣ Accounts for 40 to 60 percent of all cases of idiopathic ventricular fibrillation
- ▣ Sudden unexplained nocturnal death syndrome occurring in apparently healthy young Southeast Asians (associated with nightmares sometimes)

Brugada syndrome

- ▣ Loss of the action potential dome in the right ventricular epicardium - Cause of ST elevation
- ▣ VF results from the electrophysiological heterogeneity in the right ventricle
- ▣ Sodium channel blockers can reproduce the EKG findings
 - Mutations in a gene responsible for the sodium channel (SCN5A) has been identified in some families with Brugada syndrome
 - Causing acceleration of sodium channel recovery or in nonfunctional sodium channels
- ▣ Treatment AICD

Right Bundle-Branch Block and ST-Segment Elevation in Leads V₁ Through V₃

A Marker for Sudden Death in Patients Without Demonstrable Structural Heart Disease

Josep Brugada, MD; Ramon Brugada, MD; Pedro Brugada, MD

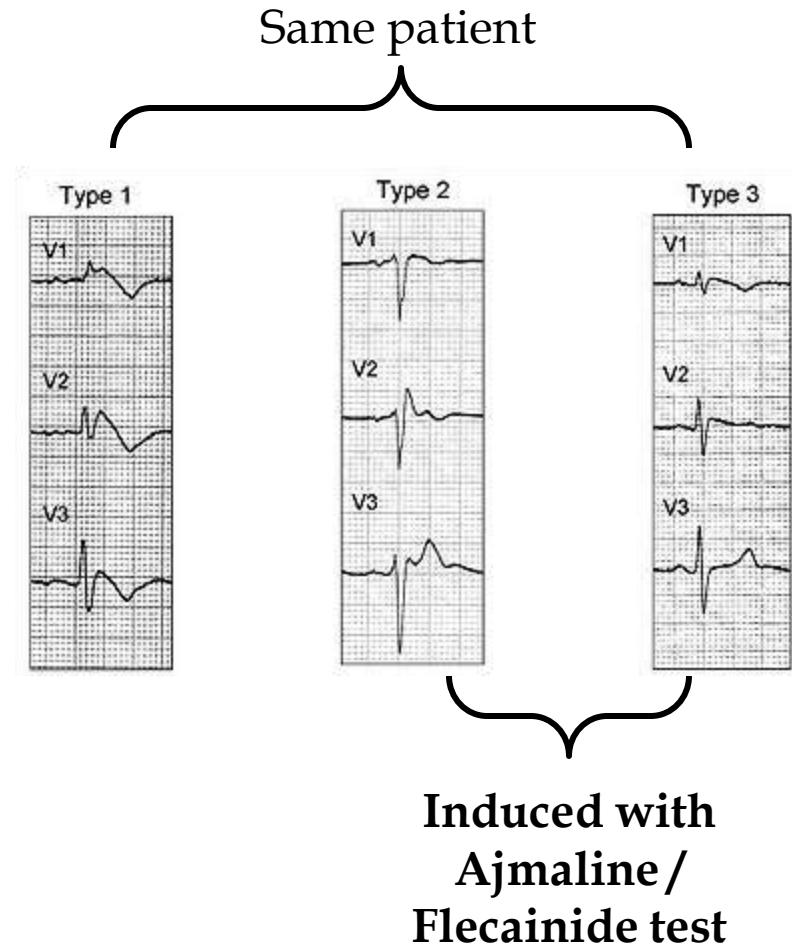
Background—Five years ago, we described a specific ECG pattern of right bundle-branch block and ST-segment elevation in leads V₁ through V₃ associated with sudden death in patients without demonstrable structural heart disease. Information on long-term outcome has become available due to pooled data on a large cohort of patients with this syndrome who are followed at 33 centers worldwide.

Methods and Results—Data on 63 patients (57 men; mean age, 38±17 years) with the described ECG pattern were analyzed in terms of arrhythmic events and sudden death. Events were analyzed for patients with at least one episode of aborted sudden death or syncope of unknown origin before recognition of the syndrome (symptomatic patients, n=41) and for patients in whom the ECG pattern was recognized by chance or because of screening related to sudden death of a relative (asymptomatic patients, n=22). During a mean follow-up of 34±32 months, an arrhythmic event occurred in 14 symptomatic patients (34%) and 6 asymptomatic patients (27%). An automatic defibrillator was implanted in 35 patients, 15 received pharmacological therapy with β -blockers and/or amiodarone, and 13 did not receive treatment. The incidence of arrhythmic events was similar in all therapy groups (log-rank 0.86); however, total mortality was 0% in the implantable defibrillator group, 26% in the pharmacological group, and 31% in the no therapy group (log-rank 0.0005). All mortality was due to sudden death.

Conclusions—Patients without demonstrable structural heart disease and an ECG pattern of right bundle-branch block and ST-segment elevation in leads V₁ through V₃ are at risk for sudden death. Amiodarone and/or β -blockers do not protect them against sudden death, and an implantable defibrillator seems to be the present treatment of choice. (*Circulation*. 1998;97:457-460.)

Clinical summary Brugada syndrome

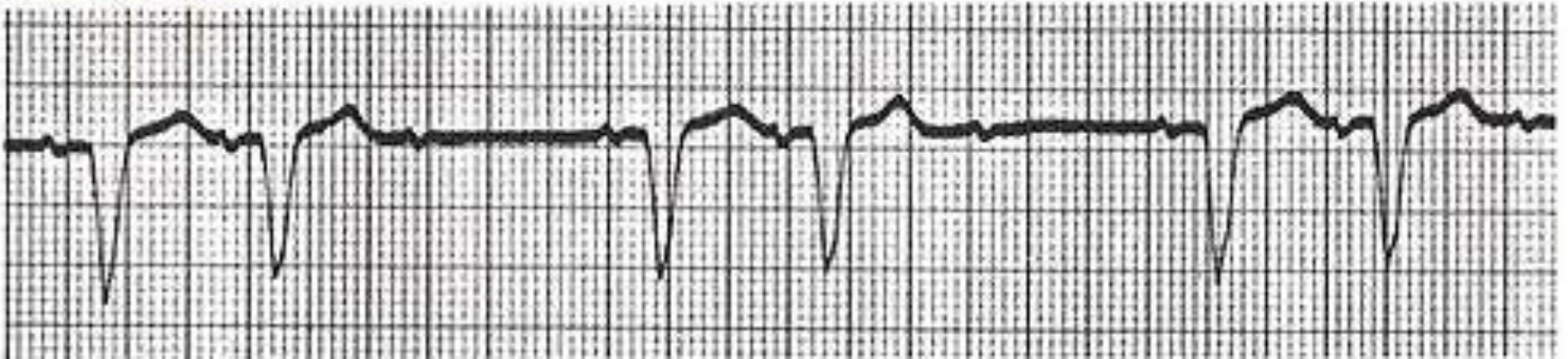
- ▣ Most common cause of sudden death in young men without known underlying cardiac disease
- ▣ According to a recent consensus document, type 1 ST segment elevation either spontaneously present or induced with Ajmaline / Flecainide test is considered diagnostic.
- ▣ AICD



CASE 3

76 y/o white male presents with symptoms of dizziness

Lead V₁

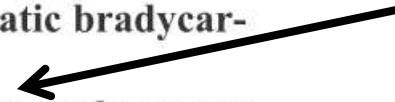
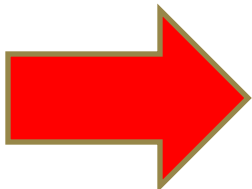
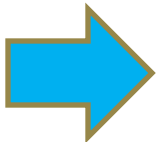


Diagnosis and treatment recommendation by the guidelines

Indications for Permanent Pacing in Acquired Atrioventricular Block in Adults

Class I

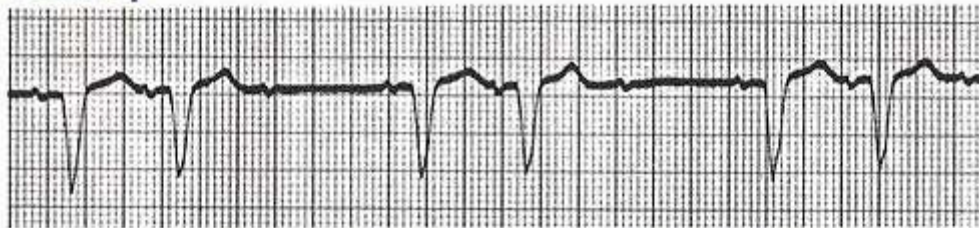
1. Third-degree AV block at any anatomic level associated with any one of the following conditions:
 - a. Bradycardia with symptoms presumed to be due to AV block. (*Level of evidence: C*)
 - b. Arrhythmias and other medical conditions that require drugs that result in symptomatic bradycardia. (*Level of evidence: C*)
 - c. Documented periods of asystole ≥ 3.0 seconds or any escape rate < 40 beats per minute (bpm) in awake, symptom-free patients. (*Level of evidence: B, C*)
 - d. After catheter ablation of the AV junction. (*Level of evidence: B, C*) There are no trials to assess outcome without pacing, and pacing is virtually always planned in this situation unless the operative procedure is AV junction modification.
 - e. Postoperative AV block that is not expected to resolve. (*Level of evidence: C*)
 - f. Neuromuscular diseases with AV block such as myotonic muscular dystrophy, Kearns-Sayre syndrome, Erb's dystrophy (limb-girdle), and peroneal muscular atrophy. (*Level of evidence: B*)
2. Second-degree AV block regardless of type or site of block, with associated symptomatic bradycardia. (*Level of evidence: B*)



CASE 3

76 y/o white male presents with symptoms of dizziness

Lead V₁



2nd degree AV block (type II) with LBBB

LBBB plus Mobitz II 2nd Degree AV Block

Frank Yanowitz, M.D.

CASE 4

- ▣ 45 y/o female has **anterior ST elevation MI** and has ventricular fibrillation requiring cardioversion on hospital day 4
- ▣ Is she a candidate for ICD before discharge?

2012 Guidelines for ST elevation MI

9.5.2. Implantable Cardioverter-Defibrillator Therapy Before Discharge



Class I

1. Implantable cardioverter-defibrillator (ICD) therapy is indicated before discharge in patients who develop sustained VT/VF more than 48 hours after STEMI, provided the arrhythmia is not due to transient or reversible ischemia, reinfarction, or metabolic abnormalities.^{491–493} (*Level of Evidence: B*)

Life-threatening ventricular arrhythmias that occur > 48 hours after STEMI usually are associated with significant LV systolic dysfunction and signify poor prognosis.



Assessment of risk for sudden death post MI

▣ Class I

- 1. Patients with an **initially reduced LVEF** who are possible candidates for ICD therapy should **undergo reevaluation of LVEF 40 or more days after discharge.** (Level of Evidence: B)

The recommended delay to ICD therapy in this setting stems from the results of DINAMIT (Defibrillator in Acute Myocardial Infarction Trial), in which defibrillator implantation 6 to 40 days after MI in patients with EF < 0.35 and impaired cardiac autonomic function was not shown to reduce overall cardiac death risk



Not related to ST elevation MI

Class 1

ICD Guidelines 2008

1. ICD therapy is indicated in patients who are survivors of cardiac arrest due to VF or hemodynamically unstable sustained VT after evaluation to define the cause of the event and to exclude any completely reversible causes. *(Level of Evidence: A)* (16,319–324)
2. ICD therapy is indicated in patients with structural heart disease and spontaneous sustained VT, whether hemodynamically stable or unstable. *(Level of Evidence: B)* (16,319–324)
3. ICD therapy is indicated in patients with syncope of undetermined origin with clinically relevant, hemodynamically significant sustained VT or VF induced at electrophysiological study. *(Level of Evidence: B)* (16,322)
4. ICD therapy is indicated in patients with LVEF less than 35% due to prior MI who are at least 40 days post-MI and are in NYHA functional Class II or III. *(Level of Evidence: A)* (16,333)
5. ICD therapy is indicated in patients with nonischemic dilated cardiomyopathy who have an LVEF less than or equal to 35% and who are in NYHA functional Class II or III. *(Level of Evidence: B)* (4,139–141)
6. ICD therapy is indicated in patients with LV dysfunction due to prior myocardial infarction who are at least 40 days post--myocardial infarction, have an LVEF less than 30%, and are in NYHA functional Class I. *(Level of Evidence: A)* (4,132)
7. ICD therapy is indicated in patients with nonsustained VT due to prior myocardial infarction, LVEF less than 40%, and inducible ventricular fibrillation or sustained VT at electrophysiological study. *(Level of Evidence: B)* (4,131,142)

Recommendations for Cardiac Resynchronization Therapy in Patients With Severe Systolic Heart Failure

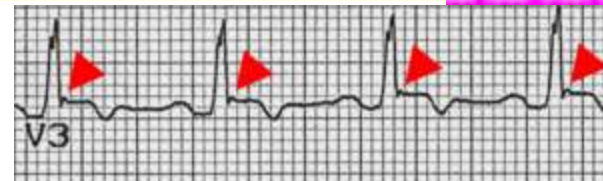
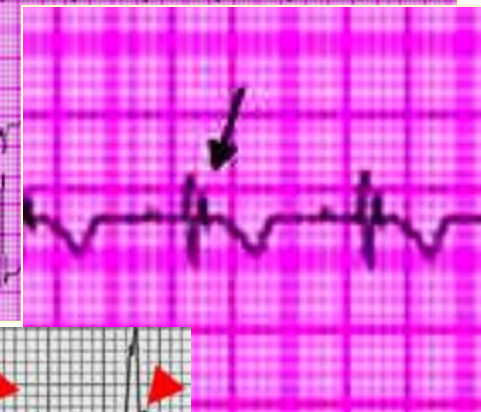
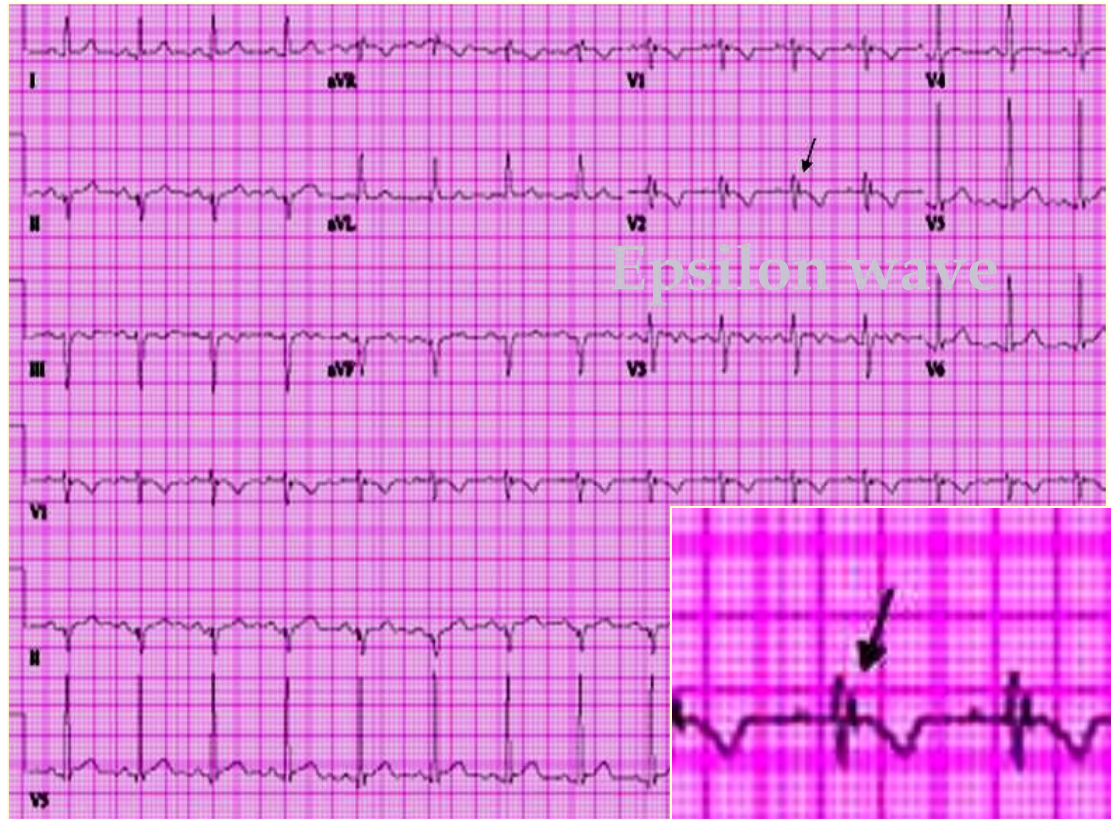
Class I

1. For patients who have LVEF less than or equal to 35%, a QRS duration greater than or equal to 0.12 seconds, and sinus rhythm, CRT with or without an ICD is indicated for the treatment of NYHA functional Class III or ambulatory Class IV heart failure symptoms with optimal recommended medical therapy. (*Level of Evidence: A*)^{222,224,225,231}

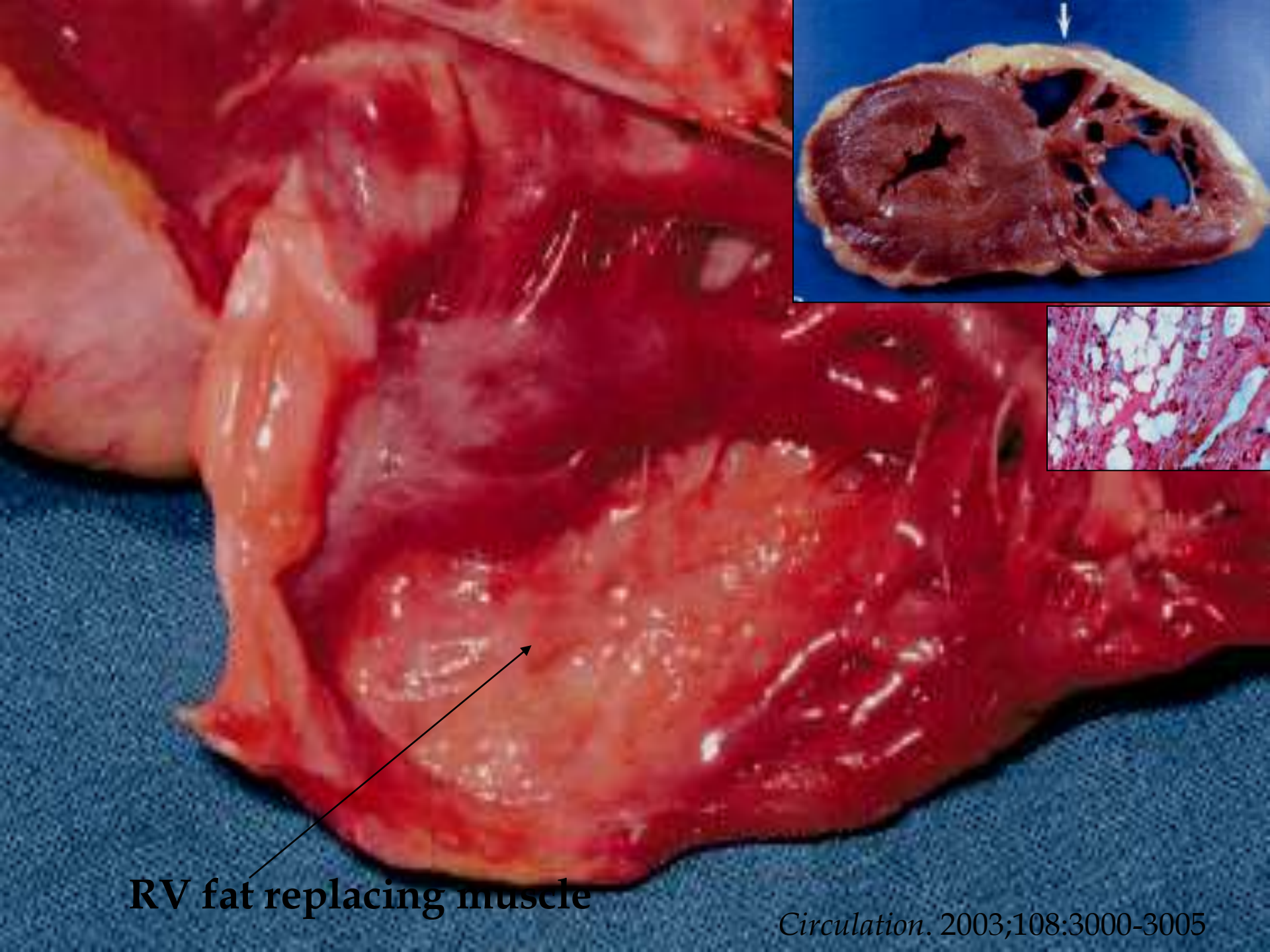


Arrhythmogenic right ventricular dysplasia

- ▣ Cardiomyopathy-childhood-appear normal hearts
- ▣ Diffuse or localized RV dilatation
- ▣ Presence of fatty tissue predisposing to ventricular tachycardia and sudden cardiac death
- ▣ VT that generally has a **left bundle branch block** contour (since the tachycardia arises in the right ventricle) & right-axis deviation and T waves inverted over the right precordial leads
- ▣ A terminal notch in the QRS (called an **epsilon wave**) may be present as a result of **slowed intraventricular conduction** (50% of patients)

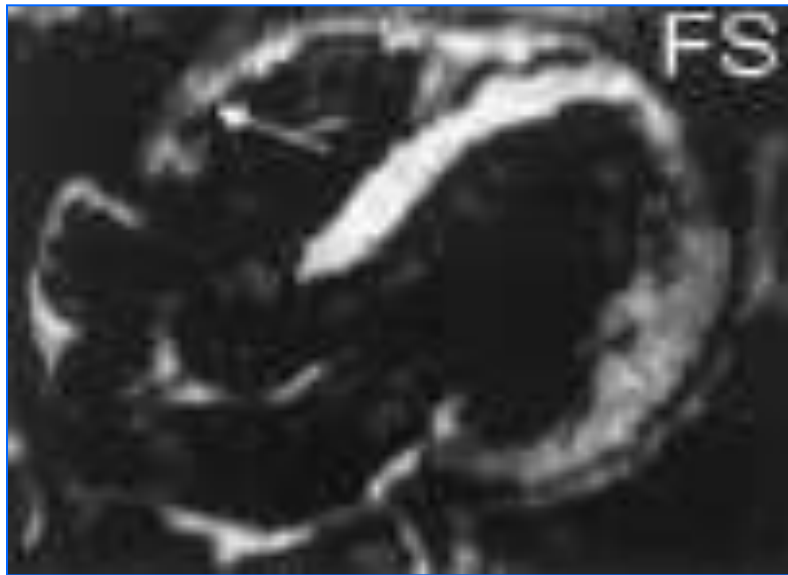


This is described as a terminal notch in the QRS complex. It is due to slowed intraventricular conduction

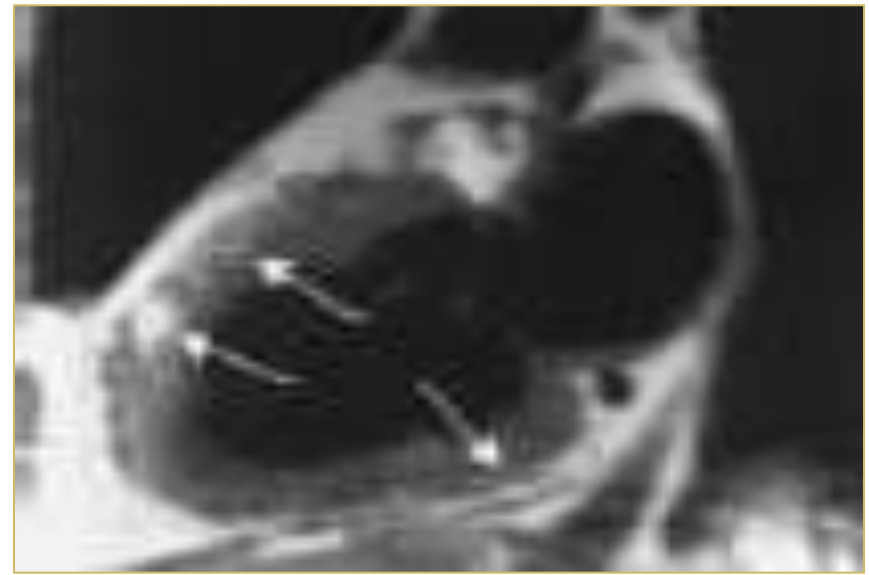


RV fat replacing muscle

Left Ventricular Involvement in Arrhythmogenic Right Ventricular Cardiomyopathy



**MRI showing RV compatible with
fat infiltration**



**Fat saturation over the
anterior and inferior
myocardium**

Arrhythmogenic right ventricular dysplasia/cardiomyopathy diagnostic criteria

Group	Major criteria	Minor criteria
Structural or functional RV abnormality	Severe RV dilation and reduction of RV ejection fraction with little or no LV involvement Localized RV aneurysm Severe segmental dilation of the right ventricle	Mild global RV dilation and/or ejection fraction reduction with normal LV Mild segmental dilation of the right ventricle Regional RV hypokinesia
Tissue characterization	Infiltration of RV myocardium by fibrofatty replacement tissue	No criteria listed
Electrocardiogram depolarization/conduction abnormality	Epsilon waves or localized prolongation (>110 ms) of the QRS complex in right precordial leads (V1–V3)	Late potentials on signal-averaged electrocardiogram
Electrocardiogram repolarization abnormality	No criteria listed	Inverted T waves in electrocardiogram leads V1–V3, aged >12 years, without RBBB
Arrhythmias	No criteria listed	LBBB-type ventricular tachycardia (sustained or nonsustained) Frequent premature ventricular contractions (>1,000 per 24 h)
Family history	Family history of ARVD/C confirmed on autopsy or surgery	Family history of ARVD/C clinically and independently diagnosed Familial history of premature sudden death (<35 years) owing to suspected ARVD/C

Abbreviations: ARVD/C, arrhythmogenic right ventricular dysplasia/cardiomyopathy; LBBB, left bundle branch block; LV, left ventricular; RBBB, right bundle branch block; RV, right ventricular. Permission obtained from the BMJ Publishing Group © McKenna W *et al.* (1994) 71: 215–218.

2 major criteria, or 1 major and 2 minor criteria, or 4 minor criteria, with each criterion coming from a different group.

Awad et al *Nature Clin Pract* MAY 2008;5:258

EXTRA

Recommendations for Permanent Pacing After the Acute Phase of Myocardial Infarction

Class III

1. Permanent ventricular pacing is not indicated for transient AV block in the absence of intraventricular conduction defects. (*Level of Evidence: B*)¹²⁶
2. Permanent ventricular pacing is not indicated for transient AV block in the presence of isolated left anterior fascicular block. (*Level of Evidence: B*)¹²⁸
3. Permanent ventricular pacing is not indicated for new bundle-branch block or fascicular block in the absence of AV block. (*Level of Evidence: B*)^{66,126}
4. Permanent ventricular pacing is not indicated for persistent asymptomatic first-degree AV block in the presence of bundle-branch or fascicular block. (*Level of Evidence: B*)¹²⁶

Circulation. 2008;117:e350-e408



Recommendations for Permanent Pacing in Chronic Bifascicular Block

Class I

1. Permanent pacemaker implantation is indicated for advanced second-degree AV block or intermittent third-degree AV block. (Level of Evidence: B)
2. Permanent pacemaker implantation is indicated for type II second-degree AV block. (Level of Evidence: B)
3. Permanent pacemaker implantation is indicated for alternating bundle-branch block. (Level of Evidence: C)



2.1.5. Hypersensitive Carotid Sinus Syndrome and Neurocardiogenic Syncope

- ▣ Syncope or presyncope resulting from an extreme reflex response to carotid sinus stimulation. There are 2 components of the reflex:
 - Cardioinhibitory, which results from increased parasympathetic tone and is manifested by slowing of the sinus rate or prolongation of the PR interval and advanced AV block, alone or in combination.
 - Vasodepressor, which is secondary to a reduction in sympathetic activity that results in loss of vascular tone and hypotension. This effect is independent of heart rate changes.

Circulation. 2008;117:e350-e408



Recommendations for Permanent Pacing After the Acute Phase of Myocardial Infarction*

Class I

1. Permanent ventricular pacing is indicated for persistent second-degree AV block in the His-Purkinje system with alternating bundle-branch block or third-degree AV block within or below the His-Purkinje system after ST-segment elevation MI. (*Level of Evidence: B*)^{79,126–129,131}
2. Permanent ventricular pacing is indicated for transient advanced second- or third-degree infranodal AV block and associated bundle-branch block. If the site of block is uncertain, an electrophysiological study may be necessary. (*Level of Evidence: B*)^{126,127}
3. Permanent ventricular pacing is indicated for persistent and symptomatic second- or third-degree AV block. (*Level of Evidence: C*)

Class IIb

1. Permanent ventricular pacing may be considered for persistent second- or third-degree AV block at the AV node level, even in the absence of symptoms. (*Level of Evidence: B*)⁵⁸

Class III

1. Permanent ventricular pacing is not indicated for transient AV block in the absence of intraventricular conduction defects. (*Level of Evidence: B*)¹²⁶
2. Permanent ventricular pacing is not indicated for transient AV block in the presence of isolated left anterior fascicular block. (*Level of Evidence: B*)¹²⁸
3. Permanent ventricular pacing is not indicated for new bundle-branch block or fascicular block in the absence of AV block. (*Level of Evidence: B*)^{66,126}
4. Permanent ventricular pacing is not indicated for persistent asymptomatic first-degree AV block in the presence of bundle-branch or fascicular block. (*Level of Evidence: B*)¹²⁶

Congenital QT Prolongation

- ▣ Diagnostic Criteria:
 - Asymptomatic patient, $QTc > 470$ msec
 - OR: Male with $QTc > 440$ or female with $QTc > 460$ PLUS:
 - ▣ Stress-related syncope
 - ▣ Torsade de pointes
 - ▣ Family history of early (<35yo) SCD
 - These criteria are neither totally sensitive or specific

Romano-Ward (autosomal dominant) Jervell-Lange-Nielsen (autosomal recessive-no hearing)

- ▣ Beta-Blockers have proven effective in preventing syncope in 75-80% of LQTS patients.
- ▣ However, despite full dose beta-blockers, 20-25% of patients continue to have syncopal episodes and remain at a high risk for sudden cardiac death.
- ▣ For those unresponsive patients, high thoracic left sympathectomy have been used. Recently, an international prospective study provided evidence that left cardiac sympathetic denervation is a very effective therapy.
- ▣ AICD is now becoming more commonly used, especially if arrest
- ▣ Q-T interval in excess of 440msec, familial in 85% of cases

