Management of acute Cardiac Arrhythmias

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Objectives

- Review the etiology and recognition of common arrhythmias.

- Review management of cardiac arrhythmias, with a focus on the relevant recent literature.
Cardiac Action Potential

Phase 0

Phase 1

Phase 2 (Plateau Phase)

Phase 3

Phase 4

(only in pacemaker cells)

Depolarization

mv

R.M.P

Na$^{+}$

Na$^{+}$

Na$^{+}$

Na$^{+}$

Na$^{+}$

Na$^{+}$

m

h

K$^{+}$

K$^{+}$

K$^{+}$

K$^{+}$

K$^{+}$

K$^{+}$

ca$^{++}$

ca$^{++}$

ca$^{++}$

ca$^{++}$

ca$^{++}$

ca$^{++}$

ATPase

Na$^{+}$

Na$^{+}$

Na$^{+}$

Na$^{+}$

Na$^{+}$

Na$^{+}$
Action potential phases
0: Upstroke
1: Early-fast repolarization
2: Plateau
3: Repolarization
4: Diastole
Sinus Rhythm
Implies normal sequence of conduction, originating in the sinus node and proceeding to the ventricles via the AV node and His-Purkinje system.

EKG Characteristics:

- Regular narrow-complex rhythm
- Rate 60-100 bpm
- Each QRS complex is proceeded by a P wave
- P wave is upright in lead II & downgoing in lead aVR
Managing Acute Arrhythmia Steps

- Take a deep breath
  - Things will go a lot slower if you rush or panic

- Make use of those around you
  - Pride can be dangerous

- Keep moving forward
Mechanism of Arrhythmogenesis

1. Disorder of impulse formation.
   a) Automaticity.
   b) Triggered Activity.
      1) Early after depolarization.
      2) Delayed after depolarization.

2. Disorder of impulse conduction.
   a) Block – Reentry.
   b) Reflection.

3. Combined disorder.
Case Study

- A 42 year old man presents to the hospital complaining of weakness and dizziness following an acute chest pain
  - He is pale and diaphoretic appearing but awake
  - Blood pressure 62/30 mm Hg
  - Pulse 40/minute; slightly irregular
  - Physical examination otherwise normal
Acute Management of this patient

A: Thrombolysis
B: Stabilized and treat bradycardia
C: Emergent PCI
D: Cardioversion
E: Refer for permanent Pacemaker implant
Treatment of Acute Bradyarrythmia

Evidence of acutely life-threatening heart failure? E.g., systolic BP < 90 mmHg, heart rate < 40/min, heart failure, bradycardia with ventricular arrhythmia

- Yes
  - 0.5 mg atropine I.V.
  - Adequate response?
    - Yes
      - Repeated administration of atropine I.V. (up to 3 mg) or adrenaline 0.02 – 0.1 mg I.V.; possibly, temporary pacemaker stimulation
    - No
      - Imminent asystole?
        - Asystole already present
        - 2nd-deg. AV block, Mobitz II
        - 3rd-deg. AV block with wide substituted rhythm
          - > 3 sec pauses
        - Yes
          - Temporary pacemaker
        - No
          - Monitoring
PPM after AMI

- The need for TPW after AMI doesn’t automatically indicate a need for PPM.

- Transient conduction disturbances or LAHB are not indications for PPM after AMI.

- PPM is indicated in the presence of advanced AVB (2nd / 3rd degree) whether (persistent) or (transient with associated BBB).
Factors that help determine the need for brady pacemaker include:

- A: Symptoms such as syncope / presyncope
- B: bradyarrhythmia
- C: symptoms correlated to bradyarrhythmia
- D: symptoms not correlated to arrhythmia
- E: none of the above
Factors that help determine the need for brady pacemaker include:

- A: Symptoms such as syncope / presyncope
- B: bradyarrhythmia
- C: symptoms correlated to bradyarrhythmia
- D: symptoms not correlated to arrhythmia
- E: none of the above
ACC/AHA Class I indications for pacing in 3rd (and advanced 2nd) degree AVB

- Symptomatic bradycardia
- Asymptomatic CHB with asystole > 3 sec or awake HR < 40 bpm
- Before catheter ablation of AVN
- Arrhythmias / medical conditions requiring drugs that result in symptomatic bradycardia
- Neuromuscular disease with AVB
Case 2

- ECG recorded from 55 years old male with history of heart disease in the ED.
- Regular rhythm 120 beats/min
- Wide QRS
- AV dissociation

**dissociated P’s**

**Fusion complexes**
Management of Acute Tachyarrythmia

Acute treatment of tachyarrythmia

Hemodynamically unstable

- possible trial of I.V. antiarrythmic agents

- cardioversion/defibrillation (premedication if possible)
Monomorphic VT

- **Procainamide is back in favour!** For stable monomorphic VT, procainamide is listed as a class IIa intervention with a higher level of evidence than amiodarone (Class IIa, level of evidence B; amio — Class IIa, level of evidence C).

- **IV amiodarone** is most reasonable for patients with sustained monomorphic VT that are hemodynamically unstable despite DC shocks or VT that is persistent/recurrent despite procainamide or other agents.

- **Lignocaine** is considered reasonable and effective when VT is thought to be related to myocardial ischemia or infarction.

- In cases of repetitive monomorphic VT in the setting of ACS, add **beta blockers** to your list of treatment options.
CASE STUDY

- Ms T. B. Y.
- 57 year lady Chinese
- Background
  - Type 2 DM for 5 years
  - Depression
    - Taking Setraline since December 2010
- Fainted 5 times over the past 3 years
  - Preceded by exertion or hunger, →sweating, →dizziness and collapse
  - Spontaneously recovered
- Family history
  - Father SCD age 56 while at work
  - Niece SCD at age of 10
CASE STUDY
What are the differential diagnosis:

1. Ventricular Tachycardia
2. Ventricular Fibrillation
3. SVT with aberrancy
4. Polymorphic Ventricular Tachycardia
5. None of the above
CASE STUDY

How would you treat this patient?

- IV amiodarone
- DC shock 200j
- IV magnesium sulphate
- IV Adenosine
- IV Lidocaine
Management of Acute Tachyarrhythmia

Acute treatment of tachyarrhythmia

Hemodynamically unstable

- possible trial of I.V. antiarrhythmic agents

- cardioversion/defibrillation (premedication if possible)
CASE STUDY

QTc = 650ms
CASE STUDY

QTc = 540ms
Superior vena cava

SA node

Atrium

AV node

Purkinje

Ventricle

Action potential phases
0: Upstroke
1: Early-fast repolarization
2: Plateau
3: Repolarization
4: Diastole

Electrocardiogram (ECG)

0V 200 ms

Overshoot

Resting potential

Vulnerable window
Mechanism of triggered arrhythmias
Torsades de Pointes type of PVT

- TdP is a PVT associated with a prolonged QT interval.
  - Thus drugs that prolong the QT interval (including the sodium channel blocking antiarrhythmics - amiodarone, procainamide, probably lignocaine also) are best avoided.

- Magnesium is the first line drug (unlikely to be effective if the QT interval is normal - i.e. generic PVT).

- Other options include (overdrive) pacing and isoproterenol.

- Immediate withdrawal of any offending drugs or correction of electrolyte abnormalities (hypo-K, hypo-Mg, hypo-Ca) is very important.
Polymorphic VT (PVT)

- DC cardioversion is probably the best, and often the only, choice if the rhythm is persistent.

- For intermittent or recurrent PVT, consider IV Beta Blockers or IV amiodarone (ONLY if the patient has a normal QTs). Procainamide is also probably useful, and Lignocaine can be used especially if in the setting of suspected ACS.

- If ACS is suspected or “can’t be excluded” urgent angiography should be considered.

- Magnesium is not likely to be effective in these patients (normal QT patients).
Case 3

- Young patient presenting with sudden onset palpitations. ECG recorded in the ED.
AV nodal re-entrant tachycardia

- 60%+ of SVT, F > M
- Acute termination with adenosine
- Antiarrhythmic prophylaxis
  - Beta-blockade
  - Verapamil
  - Flecainide
- Curable with radiofrequency ablation

- Success rates 95%
- Risk of AV node damage 1-2%
- Therefore usually when medical therapy failed or unacceptable
<table>
<thead>
<tr>
<th>Hemodynamic stability?</th>
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<tbody>
<tr>
<td>Systolic BP &lt; 90 mmHg, impairment of consciousness, chest pain, heart failure</td>
</tr>
<tr>
<td>No</td>
</tr>
<tr>
<td>Cardioversion, resuscitation if necessary</td>
</tr>
<tr>
<td>Yes</td>
</tr>
<tr>
<td>QRS complex narrow (=120 msec) or wide (&gt;120 msec)</td>
</tr>
<tr>
<td>Narrow</td>
</tr>
<tr>
<td>Regular?</td>
</tr>
<tr>
<td>No</td>
</tr>
<tr>
<td>&quot;Atrial fibrillation&quot;</td>
</tr>
<tr>
<td>Initially, rate control; if necessary, electrical or pharmacological cardioversion (amiodarone, class IC antiarrhythmic agents*1)</td>
</tr>
<tr>
<td>Yes</td>
</tr>
<tr>
<td>Supraventricular tachycardia</td>
</tr>
<tr>
<td>Vagal maneuvers (including Valsalva maneuver, carotid massage)</td>
</tr>
<tr>
<td>If ineffective: adenosine (6–18 mg I.V.)</td>
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<tr>
<td>Alternatively: calcium antagonists (verapamil, diltiazem)</td>
</tr>
<tr>
<td>Beta-blockers</td>
</tr>
<tr>
<td>If necessary, class IC antiarrhythmic agents*1</td>
</tr>
<tr>
<td>Wide</td>
</tr>
<tr>
<td>Regular?</td>
</tr>
<tr>
<td>Yes</td>
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<tr>
<td>Ventricular tachycardia or tachycardia of unknown origin</td>
</tr>
<tr>
<td>Amiodarone 150–300 mg I.V., then possibly 900 mg/24 hr Alternatively, ajmaline 25–50 mg I.V.*1</td>
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<tr>
<td>Definite supraventricular tachycardia with fascicular block</td>
</tr>
<tr>
<td>Adenosine 6–18 mg I.V.; if necessary, class IC antiarrhythmic agents*1</td>
</tr>
<tr>
<td>No</td>
</tr>
<tr>
<td>&quot;Atrial fibrillation&quot; with fascicular block: Treat like atrial fibrillation with narrow QRS complex</td>
</tr>
<tr>
<td>&quot;Atrial fibrillation&quot; with accessory conduction: Amiodarone; if necessary, class IC antiarrhythmic agents*1</td>
</tr>
<tr>
<td>Polymorphic ventricular tachycardia without QT prolongation: Normalize electrolytes Myocardial ischemia? Medication overdose? Treatment: amiodarone I.V.; if necessary, lidocaine I.V., beta-blockers</td>
</tr>
<tr>
<td>&quot;Torsades de pointes&quot; in long-QT syndrome: Magnesium I.V., lidocaine I.V.; if necessary, adrenaline/orciprenaline I.V., temporary pacemaker stimulation</td>
</tr>
</tbody>
</table>
A 28 y.o. medical officer comes to your clinic describing an incident of “fluttering” in her chest that began this morning after an overnight call. She has no relevant past medical history and takes no medications.

Her pulse is 136 and regular.

She drank a full 2-liter bottle of Mountain Dew this AM before rounds to “wake up.”

ECG...
Wolff-Parkinson-White Syndrome
Tachyarrhythmia - Stable

Atrial Fibrillation with preexcitation

AV nodal blockers

Give:

Procainamide

Ibutilide
Case 5

- A 75 y.o. man with long-standing hypertension diagnosed 30 years ago, comes to your office complaining of fatigue and a sense of his “heart pounding” for the past day. He has never had this feeling before.
- His pulse is rapid and irregularly, irregular
- EKG...
Case 5

- Diagnosis: Atrial Fibrillation
CASE STUDY

How would you treat this patient?

- IV amiodarone
- DC shock 200j
- IV magnesium sulphate
- IV Adenosine
- IV Lidocaine
Case 5: After IV Amiodarone

Diagnosis: Atrial Flutter (2:1)
Hemodynamic stability?
Systolic BP < 90 mmHg, impairment of consciousness, chest pain, heart failure

No → Cardioversion, resuscitation if necessary

Yes →

QRS complex narrow (=120 msec) or wide (>120 msec)

Narrow

Regular?

No → "Atrial fibrillation"
- Initially, rate control; if necessary, electrical or pharmacological cardioversion (amiodarone, class IC antiarrhythmic agents*)

Yes → Supraventricular tachycardia
- Vagal maneuvers (including Valsalva maneuver, carotid massage)
  - If ineffective: adenosine (6–18 mg I.V.)
  - Alternatively: calcium antagonists (verapamil, diltiazem)
  - Beta-blockers
  - If necessary, class IC antiarrhythmic agents*

Wide

Regular?

Yes → Ventricular tachycardia or tachycardia of unknown origin
- Amiodarone 150–300 mg I.V., then possibly 900 mg/24 hr
  - Alternatively, ajmaline 25–50 mg I.V.*¹
- Definite supraventricular tachycardia with fascicular block
- Adenosine 6–18 mg I.V.; if necessary, class IC antiarrhythmic agents*¹

No → "Atrial fibrillation" with fascicular block:
- Treat like atrial fibrillation with narrow QRS complex

"Atrial fibrillation" with accessory conduction:
- Amiodarone; if necessary, class IC antiarrhythmic agents*¹
- Polymorphic ventricular tachycardia without QT prolongation:
  - Normalize electrolytes
  - Myocardial ischemia?
  - Medication overdose?
  - Treatment: amiodarone I.V.; if necessary, lidocaine I.V., beta-blockers

"Torsades de pointes" in long-QT syndrome:
- Magnesium I.V., lidocaine I.V.; if necessary, adrenaline/orciprenaline I.V., temporary pacemaker stimulation
AF Treatment Strategy

Atrial fibrillation → Record 12-lead ECG → Presentation (EHRA score, Associated disease, Initial assessment)

Anticoagulation issues → Assess TE Risk → Oral anticoagulant (Aspirin, None)

Rate and rhythm control → AF type Symptoms → Rate control (± Rhythm control, Antiarrhythmic drugs, Ablation)

Treatment of underlying disease ‘Upstream’ therapy

Consider referral → ACEs/ARBs Statins/PUFAs Others
Estimation of stroke risk in AF using CHADS$_2$

<table>
<thead>
<tr>
<th>CHADS$_2$ criteria</th>
<th>Score</th>
</tr>
</thead>
<tbody>
<tr>
<td>Congestive heart failure</td>
<td>1</td>
</tr>
<tr>
<td>Hypertension</td>
<td>1</td>
</tr>
<tr>
<td>Age &gt;75 yrs</td>
<td>1</td>
</tr>
<tr>
<td>Diabetes mellitus</td>
<td>1</td>
</tr>
<tr>
<td>Stroke/transient ischaemic attack</td>
<td>2</td>
</tr>
</tbody>
</table>

Data from Gage BF et al. JAMA 2001;285:2864–70
What is happening in the real world?

AF population

<table>
<thead>
<tr>
<th>Low risk 25%</th>
<th>Moderate/high risk 75%</th>
</tr>
</thead>
</table>

Optimal antithrombotic treatment

ASA/no treatment

VKA

Limited VKA use due to contra-indications

Not eligible for VKA 35%

Eligible for VKA 65%

Limited VKA use due to logistical problems and underestimation benefit-risk ratio

Not receiving VKA 50%

Receiving VKA 50%

What really happens!

Aspirin / no treatment 75%

VKA 25%
Classification of Anti-Arrhythmic Drugs

Class I: \( \text{Na}^+ \) channel blockers

Class II: Beta blockers

Class III: \( \text{K}^+ \) channel blockers

Class IV: \( \text{Ca}^{++} \) channel blockers

Phase 0

Pacemaker potential

Phase 1

(Plateau Phase)

Phase 2

Phase 3

Phase 4

R.M.P
## Classification of Antiarrhythmic Drugs based on Drug Action

<table>
<thead>
<tr>
<th>CLASS</th>
<th>ACTION</th>
<th>DRUGS</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>I.</strong></td>
<td>Sodium Channel Blockers</td>
<td><strong>Quinidine, Procainamide, Disopyramide</strong></td>
</tr>
<tr>
<td>1A.</td>
<td>Moderate phase 0 depression and slowed conduction (2+); prolong repolarization</td>
<td></td>
</tr>
<tr>
<td>1B.</td>
<td>Minimal phase 0 depression and slow conduction (0-1+); shorten repolarization</td>
<td><strong>Lidocaine</strong></td>
</tr>
<tr>
<td>1C.</td>
<td>Marked phase 0 depression and slow conduction (4+); little effect on repolarization</td>
<td><strong>Flecainide</strong></td>
</tr>
<tr>
<td><strong>II.</strong></td>
<td>Beta-Adrenergic Blockers</td>
<td>Propranolol, esmolol</td>
</tr>
<tr>
<td><strong>III.</strong></td>
<td>K⁺ Channel Blockers (prolong repolarization)</td>
<td>Amiodarone, Sotalol, Ibutilide</td>
</tr>
<tr>
<td><strong>IV.</strong></td>
<td>Calcium Channel Blockade</td>
<td>Verapamil, Diltiazem</td>
</tr>
<tr>
<td>Agent</td>
<td>Acute treatment</td>
<td>Recurrence prevention</td>
</tr>
<tr>
<td>----------------</td>
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</tr>
<tr>
<td><strong>IA</strong></td>
<td></td>
<td></td>
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<tr>
<td>Ajmaline</td>
<td>25–50 mg I.V.</td>
<td>Up to 300 mg I.V./12hr</td>
</tr>
<tr>
<td>Disopyramide</td>
<td>2 mg/kg BW I.V.</td>
<td>400–600 mg po qd</td>
</tr>
<tr>
<td><strong>IB</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Lidocaine</td>
<td>1.5–2 mg/kg BW I.V. (maximum 4 g/24hr)</td>
<td>60–120 mg/h I.V.</td>
</tr>
<tr>
<td><strong>IC</strong></td>
<td></td>
<td></td>
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<tr>
<td>Flecainide</td>
<td>1–2 mg/kg BW I.V.</td>
<td>50–100 mg po bid</td>
</tr>
<tr>
<td>Propafenone</td>
<td>1–2 mg/kg BW I.V.</td>
<td>450–900 mg po qd</td>
</tr>
<tr>
<td><strong>II Beta-blockers (selected)</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Metoprolol</td>
<td>5–10 mg I.V. (up to 20 mg)</td>
<td>50–200 mg po qd</td>
</tr>
<tr>
<td>Esmolol</td>
<td>0.5 mg/kg BW I.V. over 2–3 minutes I.V.</td>
<td>0.1–0.2 mg/kg BW per minute I.V.</td>
</tr>
<tr>
<td><strong>III</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Sotalol</td>
<td>20 mg I.V. over 5 minutes (up to 1.5 mg/kg BW)</td>
<td>80–160 mg x 2 po tid</td>
</tr>
<tr>
<td>Amiodarone</td>
<td>5 mg/kg BW I.V. (up to 450 mg)</td>
<td>I.V. or po loadings dose or p.o. 0.6–1.0 g qd for 7–10 days; maintenance dose: 100–400 mg po qd</td>
</tr>
<tr>
<td><strong>IV Calcium antagonists (selected)</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Verapamil</td>
<td>5–10 mg I.V.</td>
<td>80–120 mg po tid</td>
</tr>
<tr>
<td>Diltilzem</td>
<td>0.3 mg/kg BW I.V.</td>
<td>0.2–1 mg/min I.V. 60 mg po tid</td>
</tr>
<tr>
<td><strong>Miscellaneous</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Adenosine</td>
<td>3–12 mg I.V.</td>
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</table>
Amiodarone

- Been first-line drug for the treatment of stable ventricular tachycardia (VT) in recent years.
- Reason for preference: repeated demonstration that Lignocaine (prev drug of choice) is effective in terminating < 25% of cases of VT; another was that Procainamide is very slow to work.

1) **Amiodarone Is Poorly Effective for the Acute Termination of Ventricular Tachycardia**


- Set out to assess how effect Amiodarone is at terminating VF
33 VT patients

5 required electrical treatment within 20 minutes of initiation of Amiodarone (due to presyncope, hypotension, or other adverse effects which seemed related to the amiodarone).

Only 8 (29%) of remaining 28 pts successfully converted.

18 of the 33 patients (55%) required electrical therapy (overdrive pacing, cardioversion or defibrillation) because of worsening symptoms or failure to respond.

5 (18%) of pts that did not respond to amiodarone did respond to another antidysrhythmic.

Direct current cardioversion with sedation is still the most effective means of terminating VT and should be the preferred treatment in the emergency setting.

Amiodarone is probably not as effective as previously thought; be wary of side effects & have electricity on hand!
Cardioversion
Arrhythmic Complications of Electrical Cardioversion: Relationship to Shock Energy


Guidelines for electrical cardioversion (ECV) of patients with atrial dysrhythmias have recommended starting with low energy (e.g. 50J-100J) and increasing in increments if the initial shocks fail. This recommendation is based on fears that high-energy shocks might induce myocardial damage or induce ventricular fibrillation.

- Authors proposed that higher energy levels may be safe and more effective.
For biphasic defibrillators, the Guidelines for ECV of AFib (1) suggest starting at 200 J, which is equivalent to monophasic shocks of 360 J. Since the authors caution against using monophasic shocks < 200J, they correspondingly caution against using biphasic shocks of < 100J.

In the discussion, the authors make an interesting point, stating that “It has been shown (2) that the initial use of a higher energy setting reduces the number of shocks required to effect [successful] cardioversion and in many cases [actually] reduces the total energy delivered.” So it appears that higher energy levels are more effective and associated with slightly fewer complications.


ACC/AHA/ESC Guidelines for Management of Patients With Ventricular Arrhythmias and the Prevention of Sudden Cardiac Death
— Executive Summary

(MANY AUTHORS) CIRCULATION 2006;114:1088-1132.
C. Sustained Monomorphic Ventricular Tachycardia

Recommendations

Class I
1. Wide-QRS tachycardia should be presumed to be VT if the diagnosis is unclear. (Level of Evidence: C)
2. Direct current cardioversion with appropriate sedation is recommended at any point in the treatment cascade in patients with suspected sustained monomorphic VT with hemodynamic compromise. (Level of Evidence: C)

Class IIa
1. Intravenous (IV) procainamide (or ajmaline in some European countries) is reasonable for initial treatment of patients with stable sustained monomorphic VT. (Level of Evidence: B)
2. IV amiodarone is reasonable in patients with sustained monomorphic VT that is hemodynamically unstable, that is refractory to conversion with countershock, or recurrent despite procainamide or other agents. (Level of Evidence: C)
3. Transvenous catheter pace termination can be useful to treat patients with sustained monomorphic VT that is refractory to cardioversion or is frequently recurrent despite antiarrhythmic medication. (Level of Evidence: C)

Class III
IV lidocaine might be reasonable for the initial treatment of patients with stable sustained monomorphic VT specifically associated with acute myocardial ischemia or infarction. (Level of Evidence: C)

Calcium channel blockers such as verapamil and diltiazem should not be used in patients to terminate wide-QRS-complex tachycardia of unknown origin, especially in patients with a history of myocardial dysfunction. (Level of Evidence: C)

D. Repetitive Monomorphic Ventricular Tachycardia

Recommendations

Class IIa
IV amiodarone, beta blockers, and IV procainamide (or sotalol or ajmaline in Europe) can be useful for treating repetitive monomorphic VT in the context of CHD and Idiopathic VT. (Level of Evidence: C)

E. Polymorphic Ventricular Tachycardia

Recommendations

Class I
1. Direct current cardioversion with appropriate sedation as necessary is recommended for patients with sustained polymorphic VT with hemodynamic compromise and is reasonable at any point in the treatment cascade. (Level of Evidence: B)
2. IV beta blockers are useful for patients with recurrent polymorphic VT, especially if ischemia is suspected or cannot be excluded. *(Level of Evidence: B)*

3. IV loading with amiodarone is useful for patients with recurrent polymorphic VT in the absence of abnormal repolarization related to congenital or acquired QT syndrome. *(Level of Evidence: C)*

4. Urgent angiography with a view to revascularization should be considered for patients with polymorphic VT when myocardial ischemia cannot be excluded. *(Level of Evidence: C)*

**Class Iib**

IV lidocaine may be reasonable for treatment of polymorphic VT specifically associated with acute myocardial ischemia or infarction. *(Level of Evidence: C)*

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**F. Torsades de Pointes**

**Recommendations**

**Class I**

1. Withdrawal of any offending drugs and correction of electrolyte abnormalities are recommended in patients presenting with torsades de pointes. *(Level of Evidence: A)*

**Class Iib**

1. Potassium repletion to 4.5 to 5 mM/L may be considered for patients who present with torsades de pointes. *(Level of Evidence: B)*

2. IV lidocaine or oral mexiletine may be considered in patients who present with LQTS and torsades de pointes. *(Level of Evidence: C)*

2. Acute and long-term pacing is recommended for patients presenting with torsades de pointes due to heart block and symptomatic bradycardia. *(Level of Evidence: A)*

**Class Iia**

1. Management with IV magnesium sulfate is reasonable for patients who present with long QT syndrome (LQTS) and few episodes of torsades de pointes. Magnesium is not likely to be effective in patients with a normal QT interval. *(Level of Evidence: B)*

2. Acute and long-term pacing is reasonable for patients who present with recurrent pause-dependent torsades de pointes. *(Level of Evidence: B)*

3. Beta blockade combined with pacing is reasonable acute therapy for patients who present with torsades de pointes and sinus bradycardia. *(Level of Evidence: C)*

4. Isoproterenol is reasonable as temporary treatment in acute patients who present with recurrent pause-dependent torsades de pointes who do not have congenital LQTS. *(Level of Evidence: B)*
G. Incessant Ventricular Tachycardia

Recommendations

Class I  Revascularization and beta blockade followed by IV antiarrhythmic drugs such as procaainamide or amiodarone are recommended for patients with recurrent or Incessant polymorphic VT due to acute myocardial ischemia. (Level of Evidence: C)

Class IIa  IV amiodarone or procaainamide followed by VT ablation can be effective in the management of patients with frequently recurring or Incessant monomorphic VT. (Level of Evidence: B)

Class IIb  1. IV amiodarone and IV beta blockers separately or together may be reasonable in patients with VT storm. (Level of Evidence: C)
2. Overdrive pacing or general anesthesia may be considered for patients with frequently recurring or Incessant VT. (Level of Evidence: C)
3. Spinal cord modulation may be considered for some patients with frequently recurring or Incessant VT. (Level of Evidence: C)
QUESTION

YOU HAVE THE RIGHT TO REMAIN SILENT...!

INSURANCE INDUSTRY

GOP PATIENTS BILL OF RIGHTS

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THE CHARLOTTE OBSERVER