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Objectives

• Review ACLS management of tachydysrhythmias
• Discuss common narrow complex tachycardias
• Discuss medications used in management of these narrow complex tachycardias
• Indications and technique for cardioversion
• Practice cases
2010 ACLS Guidelines-Management of Symptomatic Arrhythmias

Some important changes

1. Adenosine can now be considered for the diagnosis and treatment of stable undifferentiated wide-complex tachycardia when the rhythm is regular and the QRS waveform is monomorph.

2. IV infusion of chronotropic agents is now recommended as an equally effective alternative to external pacing when atropine is ineffective.

3. Atropine is no longer recommended for routine use in the management of PEA and asystole.
Tachycardia (w/ pulses)

- Assess and support ABC’s
- Give oxygen

1. Symptoms persist

2. Is patient stable?

3. Unstable

Perform immediate synch. cardioversion

4. Stable

- Establish IV access
- Obtain 12 lead EKG
- Is QRS narrow?

5. Narrow

NARROW QRS*

Is rhythm regular?

6. Regular

Give adenosine

Irregular Narrow Complex Tachycardia
* Probable a. fibrillation or a. flutter or MAT
a) Consider consult
b) Control heart rate:
   Diltiazem, B-blockers

7. Does rhythm convert?

8. Converst

If converts, probable re-entry SVT
a) Observe for recurrence
b) Treat with adenosine, diltiazem, B-blockers

9. Does not convert

If no conversion, possible a. flutter, ectopic a. tachycardia, junctional tachycardia
a) Control rate: diltiazem, B-blockers
b) Treat underlying cause
c) Consider consult

10. Other

11. Irregular

If V. tachycardia or uncertain rhythm:
   If V. tachycardia:
   - Amiodarone
   - Control rate: diltiazem, B-blockers
   - Treat underlying cause
   - Consider consult

12. Irregular

WIDE QRS*

Is rhythm regular?

13. Regular

** If A. fibrillation with aberrancy:
   a) See Box 11
** If pre-excited A. fibrillation (AF+WPW):
   a) Avoid adenosine, digoxin, diltiazem, verapamil
   b) Consider amiodarone

** If recurrent polymorphic VT
   a) Call for a consult
** If torsades de pointes:
   a) Give magnesium

During evaluation, treat contributing factors:

- Hypovolemia
- Toxins
- Hypoxia
- Tamponade, cardiac
- Hydrogen ion
- Tension pneumo.
- Hypo/hyperkalemia
- Thrombosis
- Hypoglycemia
- Trauma (hypovolemia)
- Hypothermia

* NOTE: If patient becomes unstable, go to Box 4.
Narrow Complex Tachycardias

Regular or Irregular?

Regular

- Junctional Tachycardia
- SVT: AVNRT and AVRT

Irregular

- Focal Atrial Tachycardia
- Atrial Fibrillation
- MFAT
- Atrial Flutter with variable block
Narrow Complex Tachycardias

- Regular
  - SVT: AVNRT and AVRT
  - Junctional Tachycardia
  - Focal Atrial Tachycardia
- Irregular
  - Atrial Fibrillation
  - MFAT
- Atrial Flutter
- Atrial Flutter with variable block
A-fib and A-flutter

• Considerable overlap in clinical and electrophysiologic features
• Etiologies, workup, and treatment identical to a-fib
Issues in Newly Diagnosed A-fib

• What are the etiologies of atrial fibrillation?
• What workup is required of these patients?
• What are priorities in management?
• What are indications for emergent cardioversion of a-fib of duration >48 hours/unknown duration?
ECG Characteristics of A-fib

• Irregularly irregular ventricular rhythm
• Irregular, wavy pattern in place of p waves, called fibrillatory waves
  – Fibrillatory wave rate is between 350-600/min

Fibrillatory waves appear less than one large box apart = >300/sec

Source Undetermined
Fibrillatory Waves

- May be coarse and look similar to a very irregular flutter, as in this patient with hypothyroidism
Fibrillatory Waves

• May be very fine/unobservable as in this patient
Fine fibrillatory waves
ECG Characteristics of A-flutter

- Regular rate, usually 150bpm or 300bpm
- Sawtooth flutter waves
- AV block
Burden of A-fib

• Affects ~5% of people aged ≥60 years
  ~10% of those aged ≥80 years
• 5%/year stroke rate
• ~30% lifetime risk of stroke
• A-fib increases risk of stroke 5X above baseline

Source: Halperin JL, AHA 2008
Etiologies of A-fib

- Hyperthyroidism (8.3%)
- Obesity/Metabolic Syndrome
- PE (10-14% of patients)
- Valvular heart disease (16-70%)
- Cardiomyopathy
- Congenital heart disease
- COPD
- OSA

- Hypertension
- Alcohol
- Caffeine
- Medications
- Stimulants
- Cardiac surgery
- Genetic syndromes
Recommended Minimum Workup Newly Diagnosed AF

- ECG
- CXR
- TSH
- Transthoracic Echocardiogram
  - May be deferred to outpatient setting

AHA 2008
Treatment of a-fib/flutter

• Adenosine is both diagnostic and therapeutic
• Electrical cardioversion
  – Safe if done within 48 hours of onset
  – Indicated in any unstable patient regardless of time of onset of a-fib
• Rate Control with AV nodal blocking agent
  – Traditionally use diltiazem or metoprolol
  – Labetalol?
  – Digoxin
Electrical Cardioversion for A-fib

- 24 patients with a-fib <48 hours in PA in whom DC Cardioversion attempted
- Historical rate control group used as comparator
- Median LOS 4 hrs in cardioversion group, 39.3 hrs in rate control group
- Charges of $1598 vs. rate control $4271

Electrical Cardioversion for A-fib

- 33 patients a. fib <48 hrs in Australian ED
- 91% success with biphasic cardioversion
- 7/33 (22%) had recurrence of a. fib at 3 mos.
- Mean LOS in ED 5.6 hours
- 31/33 (97%) of patients satisfied

Electrical Cardioversion

- Common complications
  - Transient asystole (like giving adenosine)
  - Post-cardioversion bradycardia
  - ST segment elevation
- Uncommon complications
  - Converting a-flutter to a-fib
  - Converting a-fib to VF

Ernstl, [Wikimedia Commons](https://commons.wikimedia.org/wiki/File:Electrical_cardioversion.jpg)
Avoiding VF

• Review of 5,155 external cardioversion shocks for a-fib and 1,243 for a-flutter
• All attempted with monophasic devices
• VF in 5 cases
  – All after <100 J shock
  – 2 cases had verified shock during ventricular repolarization

Gallagher et al. Int J Cardiol 2008
Electrical Cardioversion

• Bottom line
  – Very safe procedure
  – Procedural sedation-associated complications were higher (22/388) than those associated with cardioversion (5/388) in one ED-based study

A-fib Classification

PAROXYSMAL
Terminates Spontaneously Within 7 Days

PERSISTENT
Fails to Terminate Spontaneously Within 7 Days

PERMANENT
Lasts Over 1 Year with No or Failed Cardioversion

7 DAYS

1 YEAR

LONE
No Structural Heart Disease
Do all newly dx AF patients need a rule-out?

• One prospective study of 109 patients found 100% negative predictive value for MI if:
  ◦ No ST-segment elevation
  ◦ No ST depression > 2 mm

• Chest pain and ST depression < 2 mm was very common and benign

Do all newly dx AF patients need a rule-out?

- Elevations of troponin due to non-coronary cause occur in substantial proportion of a-fib patients.
- Not helpful in absence of characteristic symptoms and ECG findings

Barasch E, et al. Cardiology 2000
Narrow Complex Tachycardias

Regular or Irregular?

Regular

Irregular

SVT: AVNRT and AVRT

Junctional Tachycardia

Focal Atrial Tachycardia

Atrial Fibrillation

MFAT

Atrial Flutter

Atrial Flutter with variable block
Supraventricular Tachycardia

• Terminology is confusing...
• Two Major Mechanisms
  – AV Nodal Reentrant Tachycardia (AVNRT)
  – AV Reentrant Tachycardia (AVRT)
• Types often indistinguishable on ECG
• Distinctions clinically unimportant in the emergency department
Pathways for AVNRT vs. AVRT
Pathways for AVNRT vs. AVRT

e.g. Wolf-Parkinson-White

Normal electrical pathways
Abnormal electrical pathway in Wolff-Parkinson-White syndrome
Supraventricular Tachycardia

• AV Reentrant Tachycardia (AVRT)
  – 20% of patients with SVT
  – Reentrant circuit involving AV node + accessory pathway (e.g. WPW)
    • Orthodromic conduction in 85% of WPW pts
    • Antidromic conduction
  – P waves more often seen
    • Retrograde
  – Rate usually 169-200 bpm
- Orthodromic conduction
P waves may be buried somewhere in T waves
• Antidromic conductance
Supraventricular Tachycardia

• AV Nodal Reentrant Tachycardia (AVNRT)
  – Most common SVT – 60% of patients
  – Reentrant circuit in AV node
  – P waves not visible 90-95% of time
    • When present retrograde axis (away from inferior leads)
  – Rate ~180-220 bpm
Pathways for AVNRT vs. AVRT
Pathways for AVNRT vs. AVRT

Circuit occurs within the AV node
Re-entrant Pathways

- Re-entry (circus movement)
  - Two pathways for current: one fast, one slow
  - Precipitated by premature beat
  - Immediately begins at maximal rate
  - No beat-to-beat variability
Supraventricular Tachycardia

• Who gets it?
  – Normal people with normal hearts
  – Rheumatic Heart Disease
  – Pericarditis
  – Myocardial Infarction
  – Mitral Valve Prolapse
  – Pre-excitation syndromes (WPW)
Treatment

• If unstable:
  – Electrical Cardioversion (>100 Joules)

• Stable:
  – Vagal maneuvers
  – AV nodal blocking agents
    • Adenosine
    • Beta blockade/CCB
    • Digoxin
Carotid sinus massage

Using the following procedure, success rose from baseline 5% to 30% (n=19):
While lying supine on the bed in a Trendelenberg position, patients forcefully expire into a section of suction tubing and pressure gauge for at least 15 s and at a pressure of at least 40 mm Hg

Carotid sinus massage

• Caution or contraindicated in:
  – Severe carotid stenosis
  – Hx of CVA
Adenosine

• Interacts with A1 receptors on cardiac cells
  – Promotes hyperpolarization of cardiac tissue

• Effects
  – Slowing of sinus rate
  – Increased AV conduction delay
Adenosine

- Rapid bolus injection over 1-2 seconds with NS flush – half life is 20s
- Effects blocked by methylxanthines (aminophylline)
- Effects potentiated by dipyridamole
- Can put heart transplant patients into *permanent* asystole
- Reduce dose through central lines
Adenosine

• May be diagnostic for AVNRT/AVRT
• Often therapeutic for AVNRT/AVRT
• 6 mg followed by 12 mg 2 minutes later if initial dose ineffective
• Warn patients they will may feel flushed, experience chest pain
  – At least 50% report feeling distressed
• Cumulative success of approx 95%
  – Although up to 25% will have early recurrence
Adenosine

• Is it safe to give in cases of WPW?
Adenosine

• Is it safe to give in cases of WPW?
  – It is the preferred treatment for narrow complex tachycardias, including orthodromic WPW (AVRT)
  – 2010 ACLS guidelines recommend its use in undifferentiated, regular, monomorphic wide-complex tachycardia!
AV Nodal Blocking Agents

• Calcium Channel Blockers
  – Non-dihydropyridines act to prolong AV refractory period
  – Diltiazem, Verapamil

• Beta-Blockers
  – Metoprolol, esmolol, propranolol most commonly used
  – Labetalol? Nonselective beta blockade plus alpha-1 blockade
Electrical Cardioversion

• The practicalities
Practice Cases
56 year old female heart racing
Following Adenosine Administration
28 year old female with palpitations
46 year old female lightheaded
60 year old female palpitations
54 year old male with palpitations
Old ECG Obtained
Following Adenosine Administration, A Diagnostic Maneuver was Performed
48 year old male with palpitations
21 year old female with palpitations
Wolff-Parkinson-White Syndrome

• ECG pattern seen in 0.25% of population
  – 1.8% develop syndrome
    • Yearly risk of arrhythmia 1%/patient
• ECG pattern may be intermittent and disappear permanently with age
  – Effects of autonomic tone?
Types of Arrhythmias with WPW

- AVRT: 65%
- Atrial Fibrillation: 30%
- Atrial Flutter: 5%
Risk of Sudden Death

- 0% in patients with ECG pattern who never develop symptoms
- 0.4% annually in patients with symptoms
- A-fib was preceding rhythm in all three deaths out of 162 initially asymptomatic patients followed 5 years

Torsades de pointes
Treatments for WPW with A. fib

• Procainamide if stable
  – Increases refractory period of accessory pathway

• Synchronized electrical cardioversion
  – 200J Biphasic