EKG Conferences 2016–2017

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ECG TRACINGS

Don’t-Miss Electrocardiograms for Emergency Physicians

“I do not imagine that electrocardiography is likely to find any very extensive use in the hospital... it can at most be of rare and occasional use.”

Augustus Waller (1919)

Introduction to ECG TRACINGS

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Wide-Complex Tachycardias

• Often considered:
  – A medical emergency
  – Entertaining topic for M&M conferences
  – Challenging diagnostic dilemma
Our Approach to Tachycardias

• Is the patient stable?

• Are the QRS complexes narrow?
  – Most wide, regular tachycardias are VT

• Is the rhythm regular?
  – Most irregularly irregular rhythms are AF
    • Regardless of QRS width or shape
Approach to the patient

- A 58 year-old man presents with continuing chest pressure. He is diaphoretic. BP is 86/68. Pulse Ox= 87%. Rales are heard.
The normal (narrow) QRS Impulse originates above or within AV node
WCT- The differential diagnosis

- Ventricular Tachycardia
- Pre-existing BBB
- Temporary BBB – Tachycardia-related (= SVT with aberrancy)
- Pre-excitation (direct activation) of the ventricle (anti-dromic AVRT)

Toxic-metabolic causes
- Hyperkalemia
- Na-channel blockers

Pacemaker-induced tachycardia

Pre-existing BBB
SVT with aberrancy

- **SVT w/ aberrancy**: QRS complexes resemble standard BBB

- **VT**: the QRS complexes do not resemble classic BBB, because:
  - **Activation is not via normal His-Purkinje conduction system** ...  
    - Instead, activation is through direct ventricular myocardial activation, with by myocyte-to-myocyte transmission
    - Complexes wider, more notched, slurred, abnormal

- The supraventricular impulse is delayed or blocked in one of the bundle branches, resulting in wide QRS
- Wide complexes resemble standard bundle branch block
  - Most often RBBB pattern
- Aberrancy more likely if hyperkalemia or Ic drugs
Approach to the **stable** patient with a WCT
Brugada Algorithm  4-Step algorithm (1991, Circulation)

Original Test Characteristics of Brugada Algorithm:
- Sens: 98.7%
- Spec: 96.5%

1. Absence of an RS complex in all precordial leads?
   - yes → VT SN=.21 SP=1.0
   - no

2. R to S interval > 100ms in one precordial lead?
   - yes → VT SN=.21 SP=1.0
   - no

3. AV dissociation?
   - yes → VT SN=.82 SP=.98
   - no

4. Morphology criteria for VT present both in precordial leads V1-2 and V6?
   - yes
   - no → SVT SN=.965 SP=.987
59 year old man presented in cardiac arrest
VENTRICULAR TACHYCARDIA, rate = 220. No further analysis will be attempted.

-ABNORMAL ECG-

Requested by HANSON Tech ER Room 48
Edited C-HP7

M.D. - 5 JAN 2004 12:32
APPROACH TO THE PATIENT

• After ensuring patient is stable

• After ensuring it is not one of the mimics
  – Torsades, atrial fibrillation, hyperkalemia, TCA or other drug poisoning, STEMI

• **THEN**: VT versus SVT with aberrancy
  – Usually, it is VT
  – Use *high-yield clues* from:
    • History
    • Physical examination ??
    • Rhythm strip
    • 12-lead ECG
Ventricular Tachycardia

• Sustained re-entry VT occurs most commonly in setting of myocardial scar with localized altered conduction properties that develop over time.
  – History of CAD (prior MI, angina, stents)
  – Other structural heart disease
  – Hypertrophic or other cardiomyopathies

• Acute MI/ACS:
  – Complex interaction of ischemia, necrosis, reperfusion, healing, scar formation
  – Commonly causes polymorphic VT
The History: IS IT VT?

- It is usually VT (80-85%)
- History of M.I., stent, CHF, angina
- Low ejection fraction
- Cardiomegaly on CXR
- History of abnormal EKG
  - MI
  - BBB, WPW, Long QT
- Pro-arrhythmic medications
- Pacemaker: Pacer-induced tachycardia
- AICD: Likely VT (or pacer-induced tachycardia)
• Young age of patient
• Stable vital signs

*Are these high-yield?*
25 year old man with palpitations and near-syncope while working out at gym. BP=116/96.
Young patient with stable VS

- SVT with aberrancy is more common in young patients
- BUT Don’t be fooled:
  - Positive pred. value of SVT if < 30 years: 70%
  - CAD (anomalous origin of a coronary artery)
  - Hypertrophic cardiomyopathy
  - Dilated cardiomyopathies
  - Arrhythmogenic Right Ventricular Dysplasia
  - RVOT
  - *High-yield*: Ask about a family history of SCD

- Stable vital signs as a clue to SVT?
High-Yield

Rhythm Strip Clues to VT

• Very wide QRS (> .16 seconds)
  – RBBB pattern: > .14 seconds
  – LBBB pattern: > .16 seconds

• AV-Dissociation or VA conduction

• Fusion or capture beats (AV dissociation)

• Rate ??
The rhythm strip
66 y.o. female with weakness and SOB. BP 106/80.
2:1 V-A CONDUCTION
The Physical Examination

• AV dissociation
  – Synchronization of atrial & ventricular contractions is lost
  – Marked variations in left atrial contribution to LV filling + C.O.

• Irregular cannon A-waves
  – Intermittent high-amplitude waves ... reflect periodic
    • Simultaneous atrial and ventricular contraction
    • Right atrium contracts against closed tricuspid valve (retrograde propulsion of blood)

Beat-to-beat fluctuations in:
  – Systolic BP
  – Jugular pulsations
  – Loudness of S₁ and S₂
    (Cacophony of heart sounds)
High-yield

Clues to VT on 12-Lead

• Width of QRS > .16 seconds

• Axis (in “no man’s land”)
  – Northwest quadrant/right shoulder
  – **Key:** Negative in leads I and aVF
    • *Usually will have upright QRS in aVR*
  – Most useful in WCT with RBBB morphology

• Concordance
  – Negative or positive concordance is very suggestive
  – **All 6 precordial leads** are monophasic, same direction

• Patterns
High-yield

12-lead patterns indicating VT

RBBB Pattern
[Upright QRS in V1]

V₁
• Rsr’ with left > right
• Monophasic or biphasic QRS

LBBB Pattern

• r-wave ≥ .04 sec wide
• Delay (≥ 70 msec) onset of r to nadir of S-wave
• Notching, slurring of S
• R-wave to S-wave ≥ 100 msec in any precordial lead (Brugada)

V₆
• Small r, Deep S-Wave
  – R:S ratio < 1 (rS = VT)

• Any Q-wave or QS
Classic RBBB
Male, unknown age. No clinical data available

ECG: Regular WCT with RBBB morphology
High-Yield
12-lead patterns indicating VT

**RBBB Pattern**
- RSr’ with left > right
- Monophasic or biphasic QRS

**V1**
- Small r, Deep S-Wave
  - R:S ratio < 1

**LBBB Pattern (V1)**
- r-wave ≥ 0.04 sec wide
- Delay (> 70 msec) onset of r to nadir of S-wave
- Notching, slurring of S near nadir of S-wave
- R-wave to S-wave ≥ 100 msec in any precordial lead (Brugada)

**V6**
- Any Q-wave or QS
Findings in lead V1 and V2 during LBBB shaped tachycardia pointing to a ventricular origin (see text).

Whereas:

“Swift, smooth downstroke of S-wave indicates SVT more likely”

A: > 30 ms FAVOURS VT
B: NOTCHING, SLURRING FAVOURS VT
C: > 70 ms FAVOURS VT

Wellens H J Heart 2001;86:579-585
Figure 12. Helpful characteristics in lead V₁ of a VT with left bundle branch block shape (A) versus SVT with left bundle branch block (B). VT is characterized by initial positivity of more than 30 msec, an interval between the beginning of QRS to the nadir of the S wave of more than 70 msec, and slurring or notching of the downstroke of the S wave.
79 year-old female with dizziness

When compared with ECG of 16-JAN-2002 07:23, LBBB HAS DEVELOPED PROBABLE DUE TO CONTINUED RAPID RATE 25mm/s 10mm/mV 40Hz 005E 12SL 78 CID: 28
Referred by: B GROVES Reviewed and Interpreted by: GILBERT BLOUNT M.D.
Vent. rate 170 BPM
PR interval *
QRS duration 128 ms
QT/QTc 308/518 ms
P-R-T axes * -46 124
01-OCT-1922 (79 yr)
Female Caucasian
Loc:28
66 y.o. man attending a funeral, when he had an episode of “syncope vs. seizure.” Mild chest pain & SOB. Alert and comfortable in the ED; skin warm & dry. BP=108/68.
Stable vs. Unstable
Patient in the *gray area*

- **Strategies**
  - Assess patient, vital signs at two points in time
  - Brief trial of medication is reasonable
  - Use only one agent (avoid pro-arrhythmia + other adverse effects)
  - Reassess frequently --- cardiovert any time patient becomes unstable
  - Most “conservative” approach: Cardioversion
Adenosine for WCT?

- Treatment algorithms are straightforward

**ACLS Provider Manual (ACLS-PM):** Immediate synchronized cardioversion is indicated for the patient with *Unstable Tachycardia*. Most such patients will be in VT – but on occasion a patient in SVT may also be hemodynamically unstable. But **IF** the patient with a *monomorphic* (= similar-QRS morphology) WCT (*Wide-Complex Tachycardia*) is *stable* — ACLS-PM states *(on pg 129)* that: “Recent evidence suggests IV adenosine is relatively safe for both treatment and diagnosis”. ACLS-PM goes on to state, “IV antiarrhythmic drugs (procainamide-amiodarone-sotalol) may be effective …”. Therefore – ACLS-PM suggests the following approach for Stable Regular WCT:

  - **Polymorphic WCT** — should be immediately defibrillated *(varying QRS morphology precludes being able to use synchronized cardioversion)*.

  - **Monomorphic WCT** — Consider adenosine early in the process. Consider antiarrhythmic drugs *(See Issue #2)*. Consider expert consultation *(if available)*.
Adenosine for wide-complex tachycardia: Efficacy and safety*

Keith A. Marill, MD; Sigrid Wolfram, MD; Ian S. deSouza, MD; Daniel K. Nishijima, MD; Darren Kay, MD; Gary S. Setnik, MD; Thomas O. Stair, MD; Patrick T. Ellinor, MD, PhD

**Objectives:** To determine whether adenosine is useful and safe as a diagnostic and therapeutic agent for patients with undifferentiated wide QRS complex tachycardia. The etiology of sustained monomorphic wide QRS complex tachycardia is often uncertain acutely.

**Design:** A retrospective observational study.

**Setting:** Treatment associated with emergency visits at nine urban hospitals.

**Patients:** Consecutive patients treated with adenosine for regular wide QRS complex tachycardia between 1991 and 2006.

**Interventions:** Treatment with adenosine infusion.

**Measurements and Main Results:** Measured outcomes included rhythm response to adenosine, if any, and all adverse effects. A positive response was defined as an observed change in rhythm including temporary atrioventricular conduction block or tachycardia termination. A primary adverse event was defined as emergent electrical or medical therapy instituted in response to an adverse adenosine effect. A rhythm diagnosis was made in each case. The characteristics of adenosine administration as a test for a supraventricular as opposed to ventricular tachycardia were determined, and the adverse event rates were calculated. A total of 197 patients were included: 104 (90%) of 116 (95% confidence interval, 83%–95%) and two (2%) of 81 (95% confidence interval, 0.3%–1.6%) supraventricular tachycardia and ventricular tachycardia patients demonstrated a response to adenosine, respectively. The odds of supraventricular tachycardia increased by a factor of 36 (95% confidence interval, 9–143) after a positive response to adenosine. The odds of ventricular tachycardia increased by a factor of 9 (95% confidence interval, 6–16) when there was no response to adenosine. The rate of primary adverse events for patients with supraventricular tachycardia and ventricular tachycardia was 0 (0%) of 116 (95% confidence interval, 0%–3%) and 0 (0%) of 81 (95% confidence interval, 0%–4%), respectively.

**Conclusions:** Adenosine is useful and safe as a diagnostic and therapeutic agent for patients with regular wide QRS complex tachycardia. (Crit Care Med 2009; 37:2512–2518)

**Key Words:** tachycardia, ventricular; tachycardia, supraventricular; adenosine; anti-arrhythmia agents; diagnostic techniques; cardiovascular; adverse effects
Adenosine for diagnosis of wide QRS tachycardia: Rapid infusion for an easier conclusion*

In this issue of Critical Care Medicine, Marill and colleagues (1) describe the diagnostic use of adenosine in patients presenting with wide QRS tachycardia. The results are unequivocally valuable to critical care clinicians. The study emphasizes adenosine’s utility in distinguishing supraventricular from ventricular tachycardia (VT) and reports that its short half-life (estimated as low as 1.5 and certainly <10 secs) makes it difficult to blunder during drug administration (2).

Drury and Szent-Györgyi first described cardiovascular actions of adenine nucleotides and nucleosides in 1929 (3, 4). Subsequently, adenosine receptors have been characterized into four subtypes: A1, A2A, A2B, and A3. Interest in adenosine’s antiarrhythmic potential was (re)kindled by the observation that adenosine accumulates protein G1 (2). IAdo is present in atria, sinoatrial and atrioventricular (AV) nodes, but not in ventricular myocardium (5).

Most of adenosine’s direct electrophysiologic effects result from hyperpolarization (↑ negativity) of resting membrane potentials by activation of IA (2). This alone indirectly deactivates inward calcium currents that dominate in sinoatrial and AV nodes. Other direct effects include inhibition of the pacemaker current (I1) in sinoatrial and AV nodes and minimal direct inhibition of inward calcium current (ICa) (5).

A rapid intravenous bolus of adenosine results (within 10–30 secs) in brief (<10 secs) sinus bradycardia (hyperpolarization plus inhibition of I1) followed by sinus tachycardia (10–45 secs). Sinus tachycardia is likely due to direct stimulation of chamarceptors in the parietal structural heart disease) does not respond to adenosine (5). Two forms of catecholamine-mediated “triggered” VT (caused by intracellular calcium overload mediated by increased intracellular cyclic adenosine monophosphate) are adenosine sensitive (9, 10). Adenosine’s antiadrenergic (indirect) effect in ventricular myocytes results from adenyl cyclase inhibition (2). These VTs occur most frequently in the right ventricular outflow tract in patients without (or with minimal) structural heart disease (11). Diagnostic confusion may occur when frequent ventricular ectopy from this region results in reversible myocardial dysfunction analogous to tachycardia-mediated cardiomyopathy (12).

Although adenosine results in facial flushing, chest discomfort, and dyspnea in 20% to 50% of patients, its short half-life
In contrast to all of the historical factors, physical examination and ECG findings, non-response to adenosine was the only finding that is both highly sensitive and specific for VT ... This is because adenosine acts rapidly and briefly to slow conduction in the AV node, and VT generally does not involve or require AV nodal conduction.
Adenosine for WCT

• No reason to give adenosine if the diagnosis is clearly VT (history, ECG)
  – Exception: Suspicion of RVOT VT

• Cautions
  – If rhythm is irregular, pre-excited AF is likely (Do not use adenosine)
  – Brief vasodilatory effects (can worsen hypotension in patients with decompensated VT)

• Safe and useful if the diagnosis is uncertain
  – ONLY if regular and monomorphous (NOT polymorphous VT or AF)
  – Only 1 case report of destabilization of VT to VF
  – The test dose is 6-12 mg
  – Highly likely to terminate SVT involving AV node
  – May convert some forms of adenosine-responsive VT
  – In VT may induce retrograde VA conduction block
25 year old man with palpitations and near-syncope while working out at gym. Using Redline (fat burner, testosterone preparations, CoQ10, other supplements.
BP=116/96

- In ED denied SOB, chest pain
- Given adenosine (6, 12, 12, 18) with no effect
- Given IV verapamil without effect
- Cardioverted synchronized 150 J to normal sinus rhythm
Adenosine-responsive VT

• A minority of patients with VT (?10%)

• Best known is RVOT VT
  – Often young, healthy patients
  – Catecholamine-facilitated; often w/ exercise
  – No (or minimal) structural heart disease
    • Syncope, hemodynamic compromise, SCD are rare

• ECG:
  – LBBB morphology
  – Forces directed inferiorly and rightward (superior origin of the impulses)
    • Axis ~ - 90 degrees; tall, upright r-waves in II, III, aVF

• Often responds to adenosine, CSM or BBs
REVIEW TRACINGS
Vent. rate: 214 BPM
PR interval: * ms
QRS duration: 212 ms
QT/QTc: 324/611 ms
F-R-T axes: * 141 -65

Ventricular tachycardia
Right bundle branch block
Lateral infarct, age undetermined
T wave abnormality, consider inferior ischemia Digitalis effect
Abnormal ECG
No previous ECGs available

Technician: BREANNE LYONS
Test ind: ARYTHMIA

Referred by:
Reviewed and Interpreted by:
53 year-old man at detox facility for 5 days; had a seizure and then a respiratory arrest. In field, rapid HR up to 160/min with weak pulse, unable to obtain BP. Given shocks, lidocaine, bretyllium. History of psychiatric disease.
Rate 130  Tachycardia with unusual P axis, rate 130... P axis not -30 to 120, rate >= 100
PR 210  Multiple atrial premature complexes... Short R-R intervals, normal ORSD
QRS 189  Left anterior fascicular block and... QRS axis -45 deg., QRS > 120 mS
QT 471  Nonspecific intraventricular conduction
tQT 693  Delay
P -89  Right atrial enlargement
--Axis--
ST segment elevation
P > 0.25 mV
ST > 0.20 mV

Lead(s) V4, V5, V6 were not used for morphology analysis

- ABNORMAL ECG -

Unconfirmed diagnosis.
72 yo man brought by EMS after passing out in his car. BP 90/70. On arrival had no complaints. Alert but cool and slightly diaphoretic. On lasix.

• Initially treated with lidocaine (no effect)
• Patient requested “shock”
- Extremely wide
- Lead II:
  - 1:1 VA conduction with inverted p-waves
Clinical Rules of Behavior

• Cardiovert the unstable patient
• If *gray area*: Repeat VS at 2 points in time
  – *Trial of amiodarone (single drug)*
  – *Cardiovert at any point patient is unstable*
• Notice if irregular (pre-excited AF or polymorphic VT)
• Consider metabolic causes (hyperkalemia, TCA or other drug poisoning, especially if slower)
• *If regular*, play the odds (presume it is VT) and use *high-yield rhythm strip and 12-lead ECG clues*
• Don’t trust the computer
• Don’t rely on age or vital signs to make diagnosis
• Do not use verapamil or beta blockers
Treatment Algorithms: Overview

- **Stable patient with mono-morphic VT:**
  - Amiodarone (150 mg over 10 min);
  - Alternative: procainamide 20 mg/min
  - Correct K+, Mg++
  - Treat CHF, ischemia (“yes” to troponin)
  - Adenosine (6-12 mg rapid IV push)

- **Unstable mono-morphic VT – with pulses**
  - Synchronized shock (100 J biphasic) + **sedation**

- **Pulseless VT/VF:** Defibrillation shock (maximal energy, usually 200-300-360 (escalating biphasic)

- **Polymorphic VT, including torsades - Unstable):**
  - Defibrillation
  - Consider **acute ischemia/STEMI** (if not due to congenital or drug-induced long QT)