EKG Conferences – 2016 - 2017

Steven R. Lowenstein, MD, MPH
Critical Issues

- KEY “RULE-OUTS?”
  - When is it not syncope? (TIAs and Seizures – neurologic imposters)
  - What are the clues to serious disease?
- CRITICAL EKG PATTERNS
  - SCG PREDICTORS

Other topics

- LABORATORY TESTS: WHICH ARE USEFUL?
- DECISIONS: WHOM TO REFER OR ADMIT?
  - Hospital-based evaluation (admission or structured ED protocol)
- San Francisco Syncope Rule
Critical EKGs (SCD Predictors)

- (Acute MI)
- Old MI
- Right axis deviation
- Short P-R interval
- QT prolongation
- LVH
  - Hypertrophic cardiomyopathy
  - Aortic Stenosis
- Sinus bradycardia, AV block or fascicular/BBB
  - Sclerodegenerative conduction system disease
- ST-T Δs in V1-V3
  - ARVD
  - Brugada
- ? Benign Early Repolarization ??
THE CONTEXT: Approach to Syncope

1. Shock/Volume ↓
2. Occasional catastrophes
3. Neurologic imposters

CARDIAC
- Structural
- Electrical

NOT-CARDIAC DISEASE
- Orthostatic
- Neurally-Mediated

UNKNOWN
Psychiatric (Psuedo-syncope)
1. RULE-OUT SHOCK, HYPOVOLEMIA

- GI HEMORRHAGE (or other causes of hypovolemia)
- RUPTURED AAA, spleen, cyst, ectopic pregnancy
  Abdominal/back pain + syncope = surgical emergency
- SEPSIS

*Often indicated:*
Orthostatic vital signs
Hematocrit and fecal occult blood
Pregnancy test
Careful examination of abdomen, including U/S
2. Rule out occasional catastrophes

- **SAH**
  - Headache, focal neurologic or cranial nerve findings, persistent confusional state

- **Aortic Dissection**
  - Chest or back pain (sudden, severe; described as ripping, tearing, stabbing)
  - BP/pulse asymmetry; neurologic deficit; abnormal X-ray
  - Hypotension, AI, tamponade

- **Pulmonary embolism**
  - Chest pain, dyspnea; hypoxemia
72 year old man, history of hypertension, presented with 5-6 days of watery diarrhea and a syncopal episode on morning of admission. Also noted shortness of breath.

In ED, HR = 100, BP = 96/68.

Pulse ox = 89% on room air
What is the prevalence of PE in patients with syncope?

Prevalence of Pulmonary Embolism among Patients Hospitalized for Syncope

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Martin H. Prins, M.D., Ph.D., Maurizio Ciammaichella, M.D., Marica Perlati, M.D.,
Nicola Mumoli, M.D., Eugenio Bucherini, M.D., Adriana Visonà, M.D.,
Carlo Bova, M.D., Davide Imberti, M.D., Stefano Campostrini, Ph.D.,
and Sofia Barbar, M.D., for the PESIT Investigators

NEJM: 10/2016

- **Overall prevalence:** 17%
- In patients with “alternative explanations:” 13%
- 25% of PE-proven patients had no clinical manifestations (abnormal VS, SB, CP, hypoxia or clinical signs DVT).
- ECG findings were not reported
- *Now what?* [D-dimer and simplified Wells]
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Seizure: Fit or faint?

- Aura (taste, smell, “funny feeling”)
- Rhythmic muscle jerking
- Post-ictal confusion (often > 5 minutes)
- Lost sphincters
- Lateral tongue trauma
- Transient acidosis
- Other labs (CK, prolactin)
Transient Ischemic Attack-Related Syncope

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Department of Medicine “A” and the Israel and Ione Massada Center for Heart Disease, Beilinson Medical Center, Petah Tikva; the Tel Aviv University Sackler School of Medicine, Tel Aviv, Israel

Summary: The records of 483 patients admitted to the emergency room because of syncope were reviewed. Thirty seven patients (7.7%) were found to suffer from transient ischemic attack- (TIA) related syncope. This group is the subject of this report. Of these patients, 28 (76%) were men (mean age 71 years). Seven patients reported previous syncopeal episodes. Past history revealed a high rate of ischemic heart disease (70%) and hypertension (58%). Concurrent neurologic symptoms, which led to the diagnosis of TIA-related syncope, included mainly vertebrobasilar symptoms: vertigo (in 55% of the patients), ataxia (46%), paraesthesia (41%). Two patients most probably were presenting bilateral carotid artery disease. Various diagnostic tests (including electronecephalography, computed tomography, sonography, and cerebral angiography) were used to exclude other causes of syncope. During follow-up (mean 14.5 months) four patients (11%) had an additional episode of TIA and in three of them syncope reappeared. One patient had a complete stroke. We conclude that TIA is a much more frequent explanation for syncope than has been previously argued. These patients tend to be elderly males with high incidence of ischemic heart disease and hypertension. The concurrent neurologic symptoms, leading to the diagnosis, represent mainly vertebrobasilar territory ischemia.

Key words: syncope, transient ischemic attack, ischemic heart disease

Introduction

Transient loss of consciousness associated with the clinical diagnosis of vertebrobasilar transient ischemic attack (TIA) has been reported in various studies of syncope.1-3 As previous descriptions included only a few cases of TIA-related syncope, the clinical description and evaluation of a large number of patients with TIA-related syncope is required. In our study of 483 patients admitted to the emergency room because of syncope, we identified 37 patients with syncope as the presenting symptom of TIA. The present study had two major goals: to describe the clinical characteristics of a group of patients with TIA-related syncope, and to identify the accompanying symptoms which could indicate the vertebrobasilar (or bilateral carotid) origin of the syncope.

Patients and Methods

Patient Accrual
TIA-RELATED SYNCOPE

- Histories are sometimes vague
  - Funny turns, drop attacks and spells
  - Hard to know if neurologic sx or signs were present
  - Hard to know if true LOC occurred
- Perhaps 5- 7% of patients with syncope have a TIA
  - Elderly patients
  - Associated hypertension, CAD and vascular disease
  - We should ask about posterior circulation symptoms
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Neurally-mediated syncopes

- Neurocardiogenic syncope (Vaso-depressor)
  - Most common reflex syncope
  - Stretch of mechanoreceptors in heart (Neurocardiogenic syncope)
- Carotid Sinus Syncope
- Situational syncope
  - Stretch of receptors in bladder, esophagus, lung, rectum
  - Micturition, swallowing, defecation, cough, laugh

- Common mechanism of fainting
  - Reflexes, however triggered, lead to increased neurologic traffic to brainstem (afferent – IX, glossopharyngeal nerve)
  - Result is bradycardia or vasodilatation or both
Neuro-cardiogenic syncope
*Vaso-vagal* --- the common faint

- Under-filled ventricular chamber:
  - Erect posture, alcohol, dehydration, large meals, vasodilating Rx

- Vigorous contractility:
  - Fright, grief, bleeding, emotions, unpleasant sight, smell, ↑catechols
# Neurocardiogenic syncope

## Classic Clues

- No evidence of heart disease
- Recurrent episodes, often long interval (years) between episodes
- Setting of volume contraction
- Upright posture
- Triggers: fright, discomfort, dehydration
- Prodrome or persistence of abdominal discomfort, warmth, nausea, flushing
- + Tilt table (Passive tilt)
  - +Isoproterenol or NTG

- “Vaso-vagal”
- BENIGN: Long-term risk of death identical to risk-matched patients without syncope
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RULE-OUT HEART DISEASE

MECHANICAL
- Aortic Stenosis
- Hypertrophic Cardiomyopathy
- Pulmonary Hypertension

ELECTRICAL
- CAD-Acute OR old MI
- Sclerodegenerative conduction disease
- Short PR
- Long QT  
  - Electrolytes, Rx, congenital
- Brugada syndrome
- Other muscle disease  
  - LVH, myocarditis, HC, cardiomyopathy, ARVD
HEART DISEASE: Additional history

- **Family History** (SCD, H.C., Long Q-T, ARVD, Brugada)
- **Exertional** (A.S., H.C., PPH, ARVD)
- **Angina/chest pain** (A.S., H.C., PPH, CAD)
- **Recurrent syncope** (in short period, e.g. < 1 yr)
- **Seated/Supine**;
- **No nausea, flushing, blurred vision, sweating** – before or after
- **Palpitations**
- **Fever**
- **Medications** (Produce long QT)
HEART DISEASE: EXAM

- AORTIC STENOSIS
- HYPERTROPHIC CARDIOMYOPATHY
- ? WPW (short PR interval)
Heart Disease: EKG

- "Identifies the cause of syncope in just 5%"
- Every current clinical guideline (ACC, ACEP)
  - All syncope patients must have an ECG
  - Safe, rapid and inexpensive
  - Usually does not document "the event."
- May point to Underlying arrhythmogenic substrate for syncope or sudden cardiac death
  - ACC/AHA Task force on Clinical Practice Guidelines
  - ACEP
Don’t-miss EKGs in Syncope

- **MI - OLD:** (VT - Strong association)
- **MI - Acute:** VT, Heart block, LV failure
- **Sinus bradycardia, BBB or slow AF:** Clues to sclerodegenerative conduction system disease, advanced heart block
- **Short P-R:** WPW- Syncope due to AF
Don’t-miss EKGs in Syncope

- **Long \(Q-T\)**: Torsades de Pointes
- **RAD**: Pulmonary hypertension, pulmonary embolism
- **RV \(ST-T\) \(\Delta\)’s, accentuated \(J\)-waves**: Brugada, ARV Dysplasia
- **LVH, strain, \(ST-T\) \(\Delta\)’s**: H.C., A.S., other structural heart disease
  - LVH of any cause \(\uparrow\) risk of SCD in syncope
- “Benign” Early Repolarization (?)
Syncope in the Elderly

- Higher likelihood of cardiac disease
- Higher morbidity because of the falls
- An important cause is conduction system disease
- Especially prone to orthostasis (drugs, meals)
- High rate of micturition syncope

**Synergistic syncope:** Anemia, vascular disease, drugs, altered reflexes, unstable gait, deconditioning

- Typically, older age is a criterion for admission in clinical decision rules
  - AHA, ACEP: Age > 60 years
The yield of the ECG in finding a cause is low (less than 5%), but the test is noninvasive and relatively inexpensive and can occasionally pick up potentially life-threatening conditions such as pre-excitation syndromes, prolonged QT syndromes, or Brugada syndrome in otherwise healthy-appearing young adults.
SUDDEN DEATH IN YOUNG PEOPLE

- HYPERTROPHIC CARDIOMYOPATHY (#1)
- Q-T PROLONGATION,
- PRE-EXCITATION
- ARVD, BRUGADA
- AORTIC STENOSIS
- ACUTE MI, CAD,
  - Anomalous coronary arteries, tunneled, kinked, intramyocardial bridging
- MYOCARDITIS, CARDIOMYOPATHY
- CEREBRAL HEMORRHAGE, ASTHMA, TRAUMA
SUDDEN DEATH IN YOUNG PEOPLE

PREMONITORY SYMPTOMS*

- Syncope (23%) – Especially exertional
- Fever (16%)
- Chest pain (11%)
- Palpitations (7%)
- Family History (16%)

*Chest. 1988;93:345
Syncope in young people...

- Even if healthy, athletic

MAY BE TOMORROW’S SUDDEN CARDIAC DEATH
SYNCOPE CASES

DON’T-MISS EKGs

• Diagnosis
• Disposition
38 year old man with syncope
Four months later, this 38 y.o. man presented with 3 hours palpitations and near-syncope. At triage, BP = 115/70, then 100/80.
**University Hospital Emergency Dept.**

- **Date:** 4/13/92
- **Time:** 10:30
- **Assessment:** Syncope
- **Next Steps?**

### Medical History

- **Past History:**

### Current Medication

- **Allergies:**

### Mode of Arrival

- **Ambulance:**

### Vital Signs

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<th>B/P</th>
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<td>9:30</td>
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### Nurses Note

- **Time:** 10:30 to #11, see nurses note

### Physician Note


- **PM:** Drank 2 pint ETCH

- **1 PT:**

- **SUB:**

- **INJ:**

### TETANUS DATE

- **LMP:**

### Other

- **Visual Acuity OD:**
- **Spun Hct.:**

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**NEXT STEPS?**
Serum Mg = 0.4

QT_c > 600 msec
40 YEAR OLD FEMALE WITH 4 SYNCOPAL EPISODES IN PAST YEAR
Arrive “alert & oriented”
- Post-ictal confusion?
- No ECG OR cardiac exam
Neurology Follow-up

- Examination normal
- EEG – Negative
- Head CT - negative
Follow-up exam

- **IV/VI late peaking systolic murmur, radiated to neck**
- **Marked precordial thrill**
- Carotid upstroke: 1+, delayed
- **Echo**: Peak aortic gradient = 104 mm Hg; severe AS + AI
**TIVERSITY HOSPITAL - EMERGENCY DEPT.**

**FOLLOW UP TIME 12:22**

**Past History:**
- Hypertension
- Diabetes

**Allergies:**
- Penicillin
- Ibuprofen

**Past Medication:**
- Benadryl

**TETANUS DATE:**

**THERAPEUTIC:**
- Midazolam
- Flexeril
- Cardizem

**PHYSICIAN NOTE:**

- Arrived at ER 12:30. Room 8, no complaints. Current gain 0.5 lbs. Skin warm, dry, to touch. Good ECG. O2 flow. Leuvenstein study 0.5 of patient.

- Current status: dexmedetomidine 1.5 mcg/kg/hr. 10 pm. Angina 0.6 syncope, episodes over last 3 days. Passed out 11 pm. Tinnitus 2:30 am, 7:30 am.

- History of no complaints for past 14 months. No syncope.

- Recheck EKG: 9/10, 12/13, 16/16.

- Chest x-ray: normal.

- Discharged from Unit 11/15/10 at 3:45 PM. No TIA.

- No neuromuscular tone to No TIA.
“Don’t miss” EKG: “Old MI”

- The presence of pathologic q-waves on the EKG, indicating prior transmural MI, is strongly associated with ventricular tachycardia as a cause of syncope

  - Harrison’s Medicine
More don’t miss EKGs in syncope
42 yo female had one syncopal episode, while walking. One day of nausea and vomiting. History of chronic methadone, alcohol in past.
ECG *not obtained initially*, until patient had a cardiac arrest back in treatment bed in ED

- $\text{QT}_c = 685$
- $\text{Mg}^{++} = 1.6$
- ?Other meds
33 yo man with moderate asthma exacerbation, *syncope*. ECG obtained due to tachycardia.
Brugada Syndrome

- Gene mutation (coding structure/function of Na-channels)
  - Often + family history of sudden cardiac death
  - Brugada pattern may be transient, provoked by sodium channel blocking agents (procainamide, TCA, cocaine)

- Arrhythmogenic substrate is usually RVOT

- If borderline, Brugada pattern often unmasked if V1-V2 leads recorded 1-2 interspaces higher (or by procainamide challenge)
**DISTINCTIVE ECG FEATURES**

- High takeoff of ST-segment (elevated J-point) with rapid descent of ST into inverted T-wave
- Persistent saddle-back ST-segment elevation (w/ J-point elevation)
- **Hallmark of Brugada:**
  - ST-elevations/J-point elevations in V1-V3
  - Can look like STEMI or RBBB
  - Patients are at risk for polymorphic VT and SCD
  - Mean age at death: 41 years

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**Type 1 Brugada Pattern**

**Type 2 Brugada Pattern**

**Type 3 Brugada Pattern**
A 22 year-old man was brought by EMS after a single syncopal episode. History of chronic anxiety, PTSD, depression, and reported frequent bouts of chest pain and palpitations.
A 27 year-old man presented after a bout of dizziness and near-syncope.
ARVD

- Hereditary; 3 x more common in men; especially Greece, Italy
- + Family history of SCD
- Young, healthy patients: 2\textsuperscript{nd} most common cause of SCD in patients < 35 years, after HOCM
- Right ventricular fibro-fatty cardiomyopathy
- Clinical course: Syncope due to RV VT, SCD, Right ventricular heart failure
- EKG: Right-sided ST-T changes
  - T-inversions (like “juvenile”)
  - Epsilon wave (late potentials) at end of QRS
  - Sometimes incomplete RBBB
  - RV PVCs and VT
- Dx: Echo and MRI
25 year-old man had a syncopal episode at the airport. No chest pain or dyspnea. A “cardiac alert” was called by paramedics because of the ST-elevations on the pre-hospital ECG.
Long-Term Outcome Associated with Early Repolarization on Electrocardiography


ABSTRACT

BACKGROUND

Early repolarization, which is characterized by an elevation of the QRS-ST junction (J point) in leads other than V1 through V3 on 12-lead electrocardiography, has been associated with vulnerability to ventricular fibrillation, but little is known about the prognostic significance of this pattern in the general population.

METHODS

We assessed the prevalence and prognostic significance of early repolarization on 12-lead electrocardiography in a community-based general population of 10,864 middle-aged subjects (mean ±SE age, 42±8 years). The primary endpoint was death from cardiac causes, and secondary end points were death from any cause and death from arrhythmia during a mean follow-up of 36±11 years. Early repolarization was stratified according to the degree of J-point elevation (≥0.1 mV or >0.2 mV) in either inferior or lateral leads.

RESULTS

The early-repolarization pattern of 0.1 mV or more was present in 650 subjects (5.8%); 384 (5.5%) in inferior leads and 262 (2.4%) in lateral leads, with elevations in both leads in 16 subjects (0.1%). J-point elevation of at least 0.1 mV in inferior leads was associated with an increased risk of death from cardiac causes (adjusted relative risk, 1.28; 95% confidence interval [CI], 1.04 to 1.59; P=0.03); 36 subjects (0.3%) with J-point elevation of more than 0.2 mV in inferior leads had a markedly elevated risk of death from cardiac causes (adjusted relative risk, 2.98; 95% CI, 1.85 to 4.92; P=0.001) and from arrhythmia (adjusted relative risk, 2.92; 95% CI, 1.45 to 5.89; P=0.001). Other electrocardiographic risk markers, such as a prolonged QT interval corrected for heart rate (P=0.03) and left ventricular hypertrophy (P=0.004), were weaker predictors of the primary endpoint.

CONCLUSIONS

An early-repolarization pattern in the inferior leads of a standard electrocardiogram is associated with an increased risk of death from cardiac causes in middle-aged subjects.
J-Wave syndromes expert consensus conference report: Emerging concepts and gaps in knowledge

Endorsed by the Asia Pacific Heart Rhythm Society (APHRS), the European Heart Rhythm Association (EHRA), the Heart Rhythm Society (HRS), and the Latin American Society of Cardiac Pacing and Electrophysiology (Sociedad Latinoamericana de Estimulación Cardíaca y Electrofisiología [SOLAECE])
“Benign” Early Repolarization

- Common (~ 10% of population), but not always benign
- Elevated risk of polymorphic VT, SCD
- Newer term: Early Repolarization Pattern (ERP)
- Higher-risk ERP ECGs:
  - Involvement of inferior leads as well as lateral precordial leads
  - Prominent J-point elevation (> 2 mm)
  - ST-elevations that are horizontal or descending (rather than upsloping)
Early repolarization pattern

- ERP and Brugada overlap in genetics, arrhythmogenicity, ECG patterns, response to meds
- Even newer term for ERP and Brugada Syndrome: J-wave syndromes
  - J-wave: Notch or slur at end of QRS complex
- Accentuation of J-wave is associated with life-threatening ventricular arrhythmias
Recommendations for my patient – 25 yo man, syncope at airport?

- Incidental finding of ERP in asymptomatic patient should not be considered a ‘high risk’ situation.
- Benign in vast majority of cases
- Consider consultation if:
  - Unheralded syncope suggestive of arrhythmic event (e.g., recumbent, at rest, no sx); AND
  - *Family history of SCD or unexplained cardiac arrest*
  - Possibly high-risk ERP (high J-point, inferior leads, descending ST-segment elevations)
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Other topics

- Indications for hospital-based structured evaluation
- Use of the laboratory
- San Francisco and other clinical decision rules
  - Dismiss those with 7-day or 30-day adverse event outcomes
Most important lesson

The key factor in the investigation of syncope is the presence (or absence) of structural heart disease or an abnormal EKG ...

because these can warn of sudden cardiac death
ADMIT (Hospital-based evaluation)

- Heart disease, any type
- Abnormal EKG
- Abnormal cardiac exam
- CHF (↓ LV ejection fract.)
  - (BNP > 300: ? New biomarker)
- Hx: MI, CHF, PVC’s, VT
- Age > 60
- Vascular disease
- SOB or abnormal 02 sat

Suggestive histories
- Effort-related
- Family history of SCD
- Recurrent
- Recumbent
- Chest pain
- Palpitations
- Injury resulted
- Sx not suggestive of reflex-mediated syncope

ACP Clinical Efficacy Project; ACEP Clinical Policy; ACC/AHA
Published Clinical Decision Rules
The laboratory in syncope

**Broad-panel testing**

- Low-yield
- Unproven and not-recommended
- Testing based on history, examination, suspected causes
- Tests that may correlate with cardiac causes:
  - High-sensitivity troponins
  - Brain natriuretic peptide

**Recommended testing**

- **Hematocrit**: All Patients (+ orthostatics)
- **Pulse oximetry**: All Patients
- **EKG**: All patients, young and old
- **Electrolytes**: Only if
  - Seizure suspected
  - Vomiting or diarrhea
  - Suspect low Mg, K, suggestive EKG
- **Glucose**: Only if diabetes or seizures
Use of the Laboratory

- **Chest x-ray**: Helpful if cardiac disease suspected: Enlarged heart, CHF, etc
- **Echocardiogram**: Very useful:
  - Evidence of heart disease
  - Exertional syncope (before ETT)
- **Brain CAT Scan**: Seldom helpful, unless seizures or TIA likely or the neurologic examination is abnormal
Carotid sinus syndrome

- In patients with *carotid sinus hypersensitivity*
- Pre-syncope or syncope may be precipitated by maneuver that stimulates carotid sinus
  - Turning head, looking up, tight collars or neck-ties
- Carotid sinus massage --- + for CS hypersensitivity if:
  - Cardio-inhibition (> 3 second pause) – increased parasympathetic tone
  - Vasodepression (> 50 mm Hg BP fall) – sympathetic withdrawal
Failure to Validate the San Francisco Syncope Rule in an Independent Emergency Department Population

Adrienne Birnbaum, MD, MS
David Esses, MD
Polly Bljur, PhD
Andrew Wollowitz, MD
E. John Gallagher, MD

Study objective: We conduct a prospective independent validation of the San Francisco Syncope Rule to identify emergency department (ED) syncope patients with short-term serious outcomes.

Methods: This was a prospective observational cohort study of adult patients presenting to a university hospital ED with acute syncope or near syncope. Patients meeting inclusion criteria as defined in the San Francisco Syncope Rule derivation were evaluated for 5 previously derived predictor variables: abnormal ECG result, shortness of breath, hematocrit level less than 30%, urine systolic blood pressure less than 90 mm Hg, and history of congestive heart failure. Hospital admission occurred at the discretion of the emergency physician, independent of the decision rule. Follow-up occurred through contact with the inpatient attending physician for admitted patients and by telephone contact with patients not hospitalized or those hospitalized and discharged before day 7. Predetermined outcome measures as defined by the San Francisco Syncope Rule were death, myocardial infarction, arrhythmia, pulmonary embolism, stroke, subarachnoid hemorrhage, significant hemorrhage, or any condition causing or likely to cause a return ED visit and hospitalization for a related event.

Results: Complete predictor and follow-up data were available for 713 of 743 (96%) enrolled patients. Sixty-one of 713 (9%) patients met predetermined criteria for serious outcome. Sixteen of 61 (26%; 95% confidence interval [CI] 16% to 39%) patients with a serious outcome were not identified as high risk by the rule. Rule performance to predict serious outcomes was sensitivity 74% (95% CI 61% to 84%), specificity 57% (95% CI 53% to 61%); negative likelihood ratio 0.5 (95% CI 0.3 to 0.7) and positive likelihood ratio 1.7 (95% CI 1.4 to 2.0).
San Francisco Syncope Rule

- 2008 External Validation Study (713 ED patients)
  - Identified just 74% of patients with “serious outcomes”
  - Most of the missed outcomes were arrhythmias
- Serious outcomes measured *at 7 days*
- Other validation studies: outcomes at 30 days