Cardiovascular Articles
That Will Change Your Practice

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Practice Standards for Congestive Heart Failure

ACEP Clinical policy: Critical Issues in the Evaluation and Management of Adult Patients Presenting to the Emergency Department with Acute Heart Failure Syndromes

ACEP’s Goal:
This ACEP consensus tries to bring the recent ACC/AHA guidelines of 2005, along with European guidelines (also 2005) into an ED practice and clinical policy.

Article Overview: The following Questions asked, then answered:
Does BNP add value in diagnosing heart failure?
Is there a role for CPAP and BiPAP (NIPPV)?
Vasodilators: NTG, ACE-I, Neseritide?
Should diuretics (Lasix) be used?

Review Points:
Heart failure seen in more than 5 million patients
Yearly mortality is 18.7%
Costs US $27.9 billion per year ($27,900,000,000)
One million admissions per year
Number one discharge diagnosis in patients over 65

ACEP Levels of Evidence:

Level A High degree of certainty
Clinical certainty (Class I evidence or many class II studies)

Level B Moderate Clinical Certainty
(Class II studies or strong consensus of class III studies)

Level C Based on preliminary studies
(inconclusive studies, or even conflicting studies)
ACEP Question 1:

Does BNP or Pro-BNP add to the accuracy of clinical judgment in diagnosing heart failure in the ED?

Note: BNP:
- Produced by cardiac myocytes
- Increased when end diastolic pressure rises (e.g. HF)
- May lag by one hour or more (beware in flash pulmonary edemas)

Consensus Answer: YES.

In ED patients who have acute dyspnea BNP and Pro-BNP add to the accuracy of the clinical judgement in diagnosing CHF (Level B).

Heart failure unlikely if:

BNP < 100 pg/dl or Pro-BNP < 300

(LR - = 0.1)

Key Points on BNP and Pro-BNP:

- BNP not of significant additional diagnostic value if obvious heart failure
- BNP most helpful when unsure of etiology: e.g. Heart Failure versus COPD
- Using BNP may lower costs and days in hospital for admitted patients with SOB
- No clear advantage of BNP versus Pro-BNP

BNP > 500 = Heart Failure; Pro BNP > 1000 = Heart Failure

ACEP Question 2:

Is there a role of NIPPV in ED heart failure patients who have Respiratory Distress?

Note: NIPPV

Provides constant positive end expiratory pressure (PEEP)
CPAP is constant PEEP
BiPAP adds positive inspiratory pressure to the PEEP
Improves oxygenation, decreases work of breathing
Recruits alveoli to improve V/Q match
Improves wedges CO, SV, CI

Consensus Answer: YES.

Use 5-10 mm Hg CPAP in patients with CHF unless the patient is:
- hypotensive or needs to be intubated.

CPAP:
- Improves BP, P, RR
- Decreases need to intubate
- Possibly” reduces mortality “
ACEP Question 3:
Should Vasodilators be used in the ED?
Consensus Answer:
Nitrates: Yes. Level B
Neseritide: No. Level C
ACE-I: Maybe. Level C – watch for hypotension when starting

Further Support for BiPAP and CPAP
The Use of Non Invasive Ventilation in Emergency Department Patients with Acute Cardiogenic Pulmonary Edema: A Systemic Review

1980-2005 search of the literature
Pooled analysis of 11 studies with 494 patients
NIPPV decreased need to intubate by 43%
NIPPV decreased mortality by 61%
Effects of Non Invasive Positive Pressure Ventilation (NIPPV) on Mortality in Patients with Acute Cardiogenic Pulmonary Oedema: a Meta Analysis

23 Studies
Compared:
CPAP vs. Standard Therapy
BiPAP vs. Standard Therapy
CPAP vs. BiPAP
At least 350 patients in each comparison

Results
CPAP and BiPAP both decreased need to intubate by 44-50% respectively
CPAP lowered mortality by 41%: p=0.015
BiPAP lowered mortality but p=NS; did lower by 37%
No significant difference in direct comparison of BiPAP vs CPAP

Take Home Points on BiPAP and CPAP
They work, and work well, in Pulmonary Edema
Go to NIPPV as soon as you have a patient who is not improving with aggressive vasodilatation via NTG supplemented by some Lasix.
There is no proven difference (yet?) between CPAP and BiPAP
Agonal patients and those with AMS/or profound and increasing Hypoxia are probably not NIPPV candidates – go to it sooner, not later.

Nitrates
Many studies have shown effectiveness
Well known by staff in EDs
Improves mortality in heart failure versus inotropes like dobutamine
Was as effective as neseritide in VMAC study which was a “neseritide” study
Be aggressive with dose → titrate ↑ until BP ↓

Neseritide
Neseritide is IV BNP
Venodilator, peripheral arterial vasodilator, coronary vasodilator
Reduces preload and afterload
Not proven more effective than NTG
Early studies suggested dramatic improvements long term;
HOWEVER: may increase death due to renal failure
Not currently an ED drug

ACE-Inhibitors
Blocks renin and angiotensin mediated vasoconstriction
No strong studies in ED heart failure therapy
Beware hypotension
Not a first line heart failure drug for ED MDs

ACEP Question 3: *Should diuretics be used in the ED for heart failure?*
Consensus Answer: **YES.**
Diuretics should be used in ED patients with HF, *but* only for moderate to severe pulmonary edema when combined with NTG. (Level B)

**BUT…**

Be careful with diuretics in HF: Level C
Aggressive therapy with diuretics alone do not decrease need to intubate
Give judiciously, over aggressive use of diuretics can worsen renal function

*Note:*
Lasix alone first raises wedge pressure
Be sure its heart failure before Lasix
Lasix in COPD exacerbation can mean death
May increase risk for AMI in heart failure when compared to NTG
May worsen renal function acutely and in-hospital
↑ Acute Renal Insufficiency = ↑ Morality (up to 3 times)
Heart failure mortality risks: ↑ BUN (43), ↑ Creat (2.7), or ↓ BP (sys 115 or less)

**Article’s 5 Key Take Home Points**
Use BNP/Pro-BNP to help diagnose heart failure
*but only if unsure; or in COPD vs. CHF*

Use CPAP or BIPAP for respiratory distress
*but not if hypotensive, needs intubation, or has AMS*

Use NTG - Be aggressive and titrate upward

Do NOT use neseritide – increases renal failure and probably mortality

Be careful with lasix – use it with NTG only.
*Not useful as monotherapy; dangerous if not congestive heart failure, and it does not decrease need to intubate*

**Coronary Angiography by CT Scan Comes of Age**
*Circulation 2007;115:1762-1768*
For the past few years there has been growing interest in using CT scanning to image the coronary arteries. Older scanners at 4-16 slices required very long breath holds and the image resolution was variable and often sub-optimal. We now, I think, have come of age.

**Multi-Detector CT Evaluation of Chest Pain: MSCT, MDCT, CTA**
- High Quality Non-invasive coronary Imaging
- 64 Slice allows short breath hold
- Allows for higher quality, more detailed imaging
- Requires administration of IV or PO beta blockade
- Best for ruling out obstructive lesions

**Coronary CT Limitations**
- Radiation with Iodinated contrast
- Reader Expertise (new test)
- Ability to Breath Hold (now just 5-10 sec)
- Need for Beta Blockers (HR below 60-65)
- Increased Coronary Angiography

*10% of Scans Inadequate; 10-20% “Intermediate”*
MDCT Should NOT Be Used If:

- Elevated Biomarker (CK-MB or Trop)
- New ECG Changes
- Hemodynamic Instability, Chest Pain, AFib
- Iodinated Contrast Allergy, Hyperthyroidism, Metaformin Use
- Creatinine is > 1.3 mg/dl (some use 1.5)

A Randomized Controlled Trial of Multi-Slice Coronary Computed Tomography for Evaluation of Acute Chest Pain

*J Am Coll Cardiol 2007; 49:863-871*

**Article Overview**

- 197 Low Risk CP Patients
- Randomized to MDCT vs. Rest-Stress MIBI
- Evaluated, Safety, Accuracy, and Efficiency

**Results**

- 88/99 MSCT discharged
  - No ACS at 6 months; 100% at 6 months NPV
  - 24.1% of MSCT patients required additional testing
  - MSCT significantly shorter ED times than MIBI
    - 3.4 hrs vs. 15.0 hrs

**Computed Tomography Coronary Angiography for Rapid Disposition of Low-risk Emergency Department Patients with Chest Pain Syndromes**

*Acad Emerg Med 2007;14:112-116*

- 54 Low Risk Patients
- 85% (46/54): Negative Scans
- 100% NPV at 30 days
- Eight patients required admission
- 2/8 had totally WNL angiograms
- 2/8 had reversible ischemia

There are now five good studies using 64 slice MDCT in ED chest pain patients. Its benefit is in its negative predictive value (NPV). Thus a test approaching 100% NPV can be used to safely discharge a patient and say “you do not have coronary disease.” Larger series will be appearing so watch for them. Note: a major benefit is that you can see the coronary artery diameters directly, thus truly clean coronaries means no more ischemia work ups for a patient somewhere between 3-5 years (at least) and allows you to refer for GI workup if the CP persists.

<table>
<thead>
<tr>
<th>Author</th>
<th>Journal</th>
<th>n</th>
<th>PPV</th>
<th>NPV</th>
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<tbody>
<tr>
<td>Hoffman</td>
<td>Circ 2006</td>
<td>103</td>
<td>61%</td>
<td>100%</td>
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<tr>
<td>Rubenshtein</td>
<td>Circ 2007</td>
<td>58</td>
<td>87%</td>
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<td>Hollander</td>
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<td>87.5%</td>
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<td>Gallagher</td>
<td>Ann EM 2007</td>
<td>85</td>
<td>50%</td>
<td>99*-100%</td>
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* Note: One “miss” was diagnosed by positive stress test BUT negative MIBI and negative CTA.
**Total Body Radiation Doses**

*Modified from J Nuc Cardiol 2006;13:19-23*

<table>
<thead>
<tr>
<th>Diagnostic Studies</th>
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<tbody>
<tr>
<td>PA/Lateral CXR</td>
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</tr>
<tr>
<td>Mammogram</td>
<td>0.13</td>
</tr>
<tr>
<td>Cardiac Catheterization</td>
<td>4-6</td>
</tr>
<tr>
<td>CT Abdomen and Pelvis</td>
<td>7-8</td>
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<tr>
<td><strong>64 Slice MDCT</strong></td>
<td></td>
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<tr>
<td><em>(with/without ECG Pulsing)</em></td>
<td>Male 4.8-10</td>
</tr>
<tr>
<td></td>
<td>Female 6.8-14</td>
</tr>
<tr>
<td>Tc-99 Rest-Stress MIBI</td>
<td>12</td>
</tr>
</tbody>
</table>

**Estimating Risk of Cancer Associated With Radiation Exposure From 64-Slice Computed Tomography Coronary Angiography**

*JAMA 2007;298(3):317-323*

CTA Dosing:
- 42-91 mSv for lungs
- 50-80 mSv for breast

Increase cancer risk for 20 year old female to 1:143

*Note: This theoretical study uses non gated, wide open field at maximal CT output without breast shields. Be sure your radiologist minimizes field and CT output, is timed with ECG and uses shields in women.*

**My Take**

CTA is faster and better than Rest-Stress MIBI
Less Radiation if done right
Allows rapid R/O and gives images of aorta and lungs
We are not yet at triple rule out… but close
Be sure you have a 64 Slice (or better CT)
Be even more sure you have an experienced reader

**Syncope**

**ACEP Clinical Policy: Critical Issues in the Evaluation and Management of Adult Patients Presenting to the Emergency Department with Syncope**


**Overview:**

This is a key article that provides clinical policies and thus establishes standards we will be held to. This article and the recent San Francisco Syncope Rule studies should help us to admit more accurately and discharge with less anxiety (and less work up too). But: as the studies listed after the ACEP clinical policy show, it pays to keep up with the literature.
**Definition:**
Syncope is a brief loss of consciousness that completely resolves without medical intervention.

**General Facts on Syncope**
1 – 1.5% of ED visits  
6% of hospital admissions  
Many low risk patients admitted unnecessarily  
ED MDs may do too many unnecessary tests  
Good protocols can lower admit rates from almost 60% to 25% – 30%

**Beware of These Five in Syncope**
Subarachnoid Hemorrhage  
Pulmonary Embolus  
Aortic Dissection  
Malignant Arrhythmias  
ACS/STEMI
ACEP Question 1:

*What history and physical exam data help to stratify patients?*

Consensus Answers:

- **Level A**
  - High risk History or physical exam finding of CHF
  - Presence of CHF symptoms identifying high risk patients

- **Level B**
  - Older age, structural heart disease, history of CASHD are risk factors for adverse outcome

- **Level C**
  - Younger patients with non exertional syncope, and no history or signs of cardiac disease, no family history of sudden death, and no comorbidities to be at low risk for an adverse event

Many studies have looked at adverse events, though few have directly done risk stratification.

### What Does the Literature Show?

#### Four Key High Risk Factors Identified

*Ann Emerg Med 1997;29:459-466*

- Abnormal ECG
- History of Ventricular Arrhythmias
- History of CHF
- Age > 45 yo

*Eur Heart J 2003;24:811-819*

- Abnormal ECG
- History of Cardiovascular Disease
- Lack of a Prodrome
- Age > 65 yo

#### San Francisco Syncope Rule and Validation


*Ann Emerg Med 2006;47:448-454*

- Abnormal ECG
- Shortness of Breath
- Systolic BP < 90mm Hg
- HCT < 30
- CHF by history or physical

ACEP Question 2:

*What diagnostic testing should be used to help stratify patients and identify high risk patients?*

Consensus Answers:

- **Level A**
  - 12 Lead ECG should be obtained

- **Level C**
  - Head CT and Cardiac Echo need **NOT** be obtained unless specific findings mandate them

*Note:* 5 ECG findings you should always look for in a syncope patient who does not have ischemic changes or an arrhythmia:
Short P-R interval and delta wave
$S_1$, $Q_3$, $T_3$
Brugada’s (look in leads $V_1$ & $V_2$)
Voltage of LVH (cardiomyopathy, IHSS etc)
Prolonged Q-T

**Required Testing in Syncope** – Be Sure to Document on Chart
Orthostatics – lying then standing
Hematocrit
Serum glucose – fingerstick
ECG monitor
12 Lead

ACEP Question 3:
*Who should be admitted with syncope?*
Consensus Answer:
**Level B**
Admit those with HF
Admit those with structural heart disease
Admit:
  Older age
  Abnormal ECG
  Hct $< 30$
  History of HF
  History of CASHD
  History of Structural Heart Disease

**The San Francisco Syncope Rule**

*Triage systolic BP $< 90$ mm Hg
Complaint of shortness of breath
History of CHF
Non Sinus Rhythm or New ECG Changes
Hct $< 30$

*If any present: $52/290$ complications if none of above present: $1/370$ had adverse 7 day outcome.*

This study found a 98% sensitivity and 56% specificity.

*My Preliminary Take on San Francisco Syncope Rule*

Looks great, looks easy
Would only miss 1-2/100 high risk patients

**But:** Prior articles had included age; this rule does not.
See subsequent articles that say: Don’t Trust the San Francisco Syncope Rule.
External Validation of the San Francisco Syncope Rule

Ann of Emerg Med
2007;49:420-427

Articles Goal: does rule allow us to accurately discharge low risk patients?
Measure: Seven day adverse events

The Study
Single center prospective observational study
Academic, Urban, Level 1 Trauma Center
Two MDs independently did scoring on each patient
Treated and admitted independently of San Francisco Rule
Followed up by telephone or medical records

The Results
477 patients studied; 463 followed up
12% (56/463) had serious adverse event
S.F. Rule only 89% sensitive; 42% specific
Most S.F. “misses” were above age 60 (63, 80, 80, 89, 93)

External Validation of the San Francisco Syncope Rule
in the Australian Context

Prospective Observational Study
Seven day follow up
89 Patients, but mean age of 74yo
Again only 90% sensitivity
Clinical judgment as good as S.F. Rule

Small study, but again 1/10 serious misses.

Older Age Predicts Short-Term, Serious Events After Syncope

Same authors of 2007 Annals paper
Same patients; used 14 day adverse event rate
“Age 60 or older is strongly, is strongly, associated with short-term serious events after an ED visit for syncope.”

Take Home on Syncope

Do not rely on S.F. Rule to discharge
Use S.F. Rule to help admit – especially if “upstairs” says no
Age > 60: Admit; Age >50 be careful admit more often
Be sure you document:
- vital signs and orthostatics
- comment of presence or absence of SOB
- history or physical exam finding of CHF
  (clear lungs?, edema, JVD?)
- Check HCT
- Have looked at PMH, comorbidities

**Considered age: if > 50-60 BE CAREFUL!**

Lastly Check For:
- Short P-R; delta wave
- S₁, Q₃, T₃
- Brugada’s (V₁, V₂, V₃)
- Voltage of LVH
- Prolonged Q-T

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### The D₂B Alliance

**The Role of Prehospital ECGs**

**D₂B Alliance Goal:**

75% of all STEMI patients will receive PCI within 90 minutes of contact with first health care provider.

*Endorsed by ACC, AHA, ACEP, AAEM, ENA, NAEMSP, ACP, SAEM*

*Circulation* 2007;216:e29-e32
*Circulation* 2007;216:217-230

#### 6 Strategies for Significantly Reducing Door to Balloon Time


- EM MDs activating Cath Lab
- Single Call for activation
- Attending Cardiologist in-House
- Cath Lab ready within 20 minutes
- Real time feedback
- EMS 12 leads ECGs for pre-arrival activation

**EMS 12-leads Allowing Activation**


- Rarely causes false alarms
- Saves at least 15.4 minutes
- Is second most effective practice change
ACC/AHA 2007 Guidelines for the Management of Unstable Angina/Non-STEMI

Circulation 2007;116:803-877

If the 12-lead ECG shows evidence of acute injury or ischemia, it is reasonable that prehospital ACLS providers relay the ECG to a predetermined medical control facility and/or receiving hospital.

It is reasonable that all Prehospital EMS providers perform and evaluate 12-lead ECGs in the field on chest pain patients suspected of ACS to assist in triage decisions. ECGs with validated computer-generated interpretation algorithms are recommended for this purpose.

Prehospital 12-lead ECG Impact on AMI Treatment Time and Mortality

Acad Emerg Med 2006;13:84-89

5 studies evaluated (performed 1990-1997)
Prehospital ECGs added only 1.19 minutes of on scene time
Only one study looked at mortality
Door to Needle Time ↓ by up to 36.1 minutes
   – (22-48 minutes vs. 50-97 minutes)
Prehospital ECGs ↓ mortality by
   – (8.4% vs. 15.6% p = NS)

Evaluated Effectiveness of Prehospital ECGs
Evaluated Impact of Transmitting vs. Bringing to ED

Prehospital Emerg Care 2006;10:374-377

164 STEMIs transported by EMS
56.7% had Prehospital 12-leads
31/164 had ACS Team Activation pre-arrival

Results:

Implementation of Guidelines Improves the Standard of Care

Circulation 2006;113:2398-2405

EMS coordinated with 5 Heart Hospitals
Rotated 24 hr PCI availability
Evaluated frequency of PCI and Lytics
Evaluated Mortality

Comparison of Early Mortality ST Segment Elevation with Immediate Transport to Designated PCI Center to those transported to Nearest Hospital

Am J Cardiol 2006;98:1329-1333

Does EMS Diversion to PCI Centers Affect Outcome?
EMS Bypass of Nearest but Non-PCI Hospitals
108 Consecutive patients vs. 225 Historic Controls
93.5% PCI in study vs. 8.9% Historic
Decreased D₂B from 125 min to 63 min if only using PCI centers
Prehospital ECG’s

- Adds only 1-2 minutes to in-field time
- ECGs High quality equal to hospitals
- Increases early diagnosis of AMI

Reperfusion Therapy Starts in the Ambulance

Circulation 2006;113:2377-2379

EMS must be STEMI ready
- Rapid response to CP patients
- O₂, ASA, NTG, 12-lead ECG, Prehospital Alert
- Rapid Transport to Heart Hospital
- Time to Reconsider:
  - Prehospital lytics, Beta blockers, Plavix, Heparin

Electrocardiography

Implications of the Failure to Identify High-Risk Electrocardiogram Finding for the Quality of Care of Patients with Acute Myocardial Infarction (EDQMI Study)

Background

- EM MDs must be expert in diagnosing AMI
- Time is muscle
- Missed AMI is a missed opportunity to reperfuse
- D₂B goal is 75% of STEMI opened with 90 min

The Study

- Retrospective study from five EDs
- 1,684 AMIs over 2 years
- Evaluated missed acute findings
- ST ↑, ST ↓, T Wave ↓

The Results

- 12% of high risk changes missed!
- Missed findings more common in older patients with history of CHF, less CP
- 8% STEMI/ST ↑ missed
- 18% ST depression missed
14% T Wave inversions missed

**Clinical Results of Missing Acute Changes**

- Patients 21% less likely to get ASA
- 20% less likely to receive Beta Blockade
- 48% less likely to undergo reperfusion
- **Mortality ↑ by 40% (7.9% vs. 4.9%)**

**Recommendations**

**Become expert at reading for Ischemia**

When all done with your ECG read go back and specify:

- Check II, III, F for ST ↑
- Check I, L for ST ↑
- Check V, V₂ for deep depression
- Check each V lead for ST ↑
- Is the QRS newly widened?

**Know all 5 AMI patterns**

- Inferior (II, III, F)
- Lateral (I, L, V₅-V₆)
- Anterior (V Leads)
- RV (Deep ST in V₁-V₂ especially with Int AMI)
- Post (V₂: R>S, ST ↓, T upright)

**5 Ways to Diagnose an AMI**

- ST ↑
- Reciprocal changes ↓
- Q waves
- New ECG changes compared to old tracing
- Evolving ECG changes (repeated in ED/or from EMS)

**Arrhythmia Management**

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

**Background**
Amiodarone is “The Drug” for malignant arrhythmias
ACLS recommended first line for: PVCs, VT, VF and also for PSVT, AFib
“Proven” better in two cardiac arrest trails (ARREST, ALIVE)
Has class I, II, III, IV properties
Classified as Class III, lengthens refractory period
May cause hypotension and myocardial depression

The Study
33 VT patients who got 150mgs Amiodarone
Retrospective study from 4 EDs
Took 10 years to get patients
Evaluated responses

Results
Amiodarone only converted 27% (9/33)
3/33 required emergency cardioversion for hypotension or syncope
1/33 required emergency pacing for asystole

Conclusions on Amiodarone
Not as good as we thought
Much closer to Lidocaine in efficacy
Be careful in older patients
Be prepared to shock VT
Learn about Procainamide

The ACLS Writing Group Response
Dr. Cummings’ and Ms. Hazinski’s editorial reply worth reading
Agree evidence used was mostly from smaller, older studies
Do feel Amiodarone is indicated based on speed of onset
Electrical conversion is most effective
Procainamide, Sotalol and Amidarane all recommended in the 2005 text
Only Amiodarone listed in Algorithm though its not very effective
There is no perfect drug; Amio not very effective; Sotalol is oral;
Procaineamide may induce hypotension and prolong the Q-T
Be ready with sedation and DC Cardioversion
Background Information

**Pronestyl, Procainamide, Procaine**

“Our Friend”
Always works (almost always…)
Except if ↑ QT
Know its Strengths
Know its Weaknesses

**Procainamide Mechanism:**

**Slows, Decreases, Lowers, Prolongs**

Slows HR, conduction
Decreases Contractility
Lowers BP
Prolongs Q-T

**Comparison of Procainamide and Lidocaine in**

**Terminating Sustained Monomorphic Ventricular Tachycardia**

*Am J Cardiol 1996;78:43-46*

Procainamide at 100mg/min
Up to 10 mg/kg total
77% conversion rate with Procainamide
1 Episode of Hypotension

**Procainamide Administration**

Loading dose is about 20mg/kg
- But rarely indicated or needed
Usually antiarrhythmic at 250-400 mgs
Recommended Rate is 35 mg/min
- Can go 50-100 mg/min if BP is good
- Consider this for 3-5 minutes
- Then decrease to 35-50 mg/min
- Try not to give more than 500-1000 mgs

**Procainamide Best For**

Wide complex Tachycardia
PSVT vs. VT
Slowing Refractory AFib
Slowing Refractory PSVT
When Drug of Choice Fails

**Procainamide Never**

↑ Q-T
Torsades
Hypo K – Hypo Mag
TCA OD
Hypotensive, Wide QRS, Agonal

Procainamide Dosage Schedule, Plasma Concentrations, and Clinical Effects

Doses of 35-50 mg for 10 min likely to be very effective
Higher dose for longer increased toxicity
Used 100 mg/min for 10 minutes resulted in no major toxicity in 186 patients
Take Home

My Biases and Recommendations

Amiodarone may not work 1/3 – 2/3 times
Consider when Procainamide can be used instead of Amiodarone
Procainamide highly effective, almost always works (70%-90%)
Dose is 35-100 mgs/min based on age and BP
Great agent for VT vs. wide complex PSVT
Great in WPW
Only need 350-500mgs in most patients
Slow drip once 500mgs infused – rarely go to 1000mgs

Beware hypotension or widening QRS
Never use it QT ↑, Torsades etc.

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Are TASERS® Safe: Two Views

Cardiac Monitoring of Human Subjects Exposed to the Taser®

Taser Background
Delivers 50,000 volts
Involuntary muscle contraction
“Incapacitation without Harm”
More than 100,000 safe discharges in the training of police and healthy volunteers

Excited Delirium
Wild, delirious, hot, sweaty
Hypersympathetic state
“Super Human Strength”; running naked
A real syndrome or not?
Blamed as cause of death in Taser use

The Study
105 Subjects Tasered with X-26
Average shock was 3 seconds (0.4 – 5 sec)
Very Benign: HR ↑ by about 15 beats
One PVC; Q-T changes
“Tasers are safe” – few cardiac effects

Note: Authors clearly state “Attempts to calculate the effects of a shock on PR interval, ORS duration, and QT interval were unsuccessful due to technical limitations that prevented accurate interpretation of many tracings.”

This study shows pre and post Taser discharge that there are little if any adverse effects in normal healthy subjects. It does not show what happens during the electric discharge or what happens in “abnormal” individuals – those with underlying heart disease, and/or under the influence of stimulants and/or alcohol.

BUT…

**Taser Discharge Captures Rhythm in a Swine Model (Abstract)**

13 pigs paralyzed and Tasered
Discharges prolonged at 40 seconds
ECG like all prior studies, unreadable
Echocardiographs done simultaneously

**Echo Findings During TASER® Discharge**

Tasers caused immediate HR↑ to 300 bpm
Occurred in ALL animals
Reverted post discharge of Taser
However, 2 episodes VF, 1 VT, 1 death

**“Conclusions” on TASERS® as of September 2007**

Be careful what we say about the safety of Tasers. All information is not in. When used on normal patients, in controlled conditions TASERS are safe. However, what happens during Taser discharge in patients with underlying heart disease, intoxicated patients, patients on proarrythmic medications including those that prolong the QT and/or in agitated patients is not yet fully known.

**Cardiopulmonary Resuscitation**

Cardiopulmonary Resuscitation by Bystanders with Chest Compression Only (SOS-Kanto): an observational study.
Background
CPR outcomes remain dismal
Only AED/PAD programs have significantly improved survival
CPR by MDs, RNs, and the public remains suboptimal
Few bystanders will do mouth to mouth
Teaching CPR is hard, complicated, and time consuming
The Study
Prospective, multicenter, observational study
4,068 adult cardiac arrest patients
Compared:
  - No bystander CPR (72%)
  - Chest compression-only Resuscitation (11%)
  - Full CPR (18%)

The Results
CPR of any type doubled survival (5% vs. 2.2%)
Compression-only improved neurologic outcome by a factor of 2.7 vs. CPR
Mouth to mouth conferred no benefits

In Non Shockable Rhythms
Compression-only – 6.2% survival
Full CPR = 3.1% survival

Conclusions
We need to teach compression-only “CPR” to everyone
Teaching should be quick, easy, and fun
Training should be required in all schools and at all large employers
AED use should be taught at the same time
2 minute video and 5 minutes of practice is all that is needed for most

See also: Improving Survival from Out-of-Hospital Cardiac Arrest: Back to the Basics.

Treating STEMI and UA

Clinical Policy: Indications for Reperfusion Therapy in Emergency Department Patients with Suspected Acute Myocardial Infarction

89 References, great ECGs, detailed review of many studies.

Background
Reviews ECG indications for emergency Lytic therapy
Provides indicatiosn for Lytic Therapy who are at, or will be transferred to a PCI center
Time is muscle
Treat or Transfer?
Only 20-30% of U.S. hospitals have PCI

Lytic Indications (if not going to PCI):
Level A: In Patients who present within 12 hours of symptoms
ST elevation \( \geq 1 \) mm in 2 or more Limb Leads
ST elevation \( \geq 2 \) mm in 2 or more contiguous Precordial Leads
Any BBB which obscures reading STEMI in patients

Level B:
ST elevation \( \geq 1 \) mm in 2 or more contiguous Precordial Leads
New LBBB
LBBB with \( \geq 1 \) mm ST↑ in direction of QRS; \( \geq 5 \) mm ST deviation away from positive QRS or ST ↓ \( \geq 2 \) mm in leads V₁-V₃.

Level C:
New RBBB
RBBB with similar recommendation listed above for LBBB

Note: See NEJM 1996;334:931

SHAPE /* MERGEFORMAT

1) ST elevation \( \geq 1 \) mm in same direction as QRS (concordant ST↑)
2) ST elevation \( \geq 5 \) mm in opposite direction as QRS (discordant ST↑)
3) ST depression \( \geq 2 \) mm in V₁-V

Five Most Common Causes of Non-MI ST Elevation
- LVH (#1)
- LBBB/Paced
- Early Repolarization
- Ventricular Aneurysm
- Pericarditis

Use of Lytic Therapy for ST ↓
Easy answer “Just Say No”
But Beware Posterior AMI:
- ST ↓ in leads V₁-V₂-V₃
- R > S in V₂ (or V₃)
- T Wave Upright

What are the Indications for Lytic Therapy in Patients Who are at PCI Capable Institutions or Who Will Be Transferred to a PCI Hospital?
Give Lytics if: symptom onset is less than 3 hours from ED presentation AND ED arrival to balloon inflation time is going to be more than 90 minutes (Level B).
Give Lytics up to 6 hours post symptom onset if time to balloon will be greater than 90 minutes post ED arrival (Level C).
Recommendations
We must know most current standards
They keep evolving
Know your hospital’s protocols
Lytics work but cause CNS bleeds
After 2-3 hours of STEMI, the role of Lytics shrink dramatically

Hospital Delays in Reperfusion for ST-Elevation Myocardial Infarction:
Implications When Selecting a Reperfusion Strategy

Evaluated D₂B (DB) vs. Door to Needle (DN) times and benefits.
192,509 patients from 645 NRMI Hospitals
Longer DB-DN times affect mortality
Time is Muscle vs. PCI > Lytic
Both DB-DN times and Patient Characteristics Important

How long can you delay PCI once you are ready to give a Lytic?

<table>
<thead>
<tr>
<th>Variable</th>
<th>Time in Min</th>
</tr>
</thead>
<tbody>
<tr>
<td>Symptoms ≤ 120 min</td>
<td>94</td>
</tr>
<tr>
<td>Symptoms ≥ 120 min</td>
<td>190</td>
</tr>
<tr>
<td>Age &lt; 65 yo</td>
<td>71</td>
</tr>
<tr>
<td>Age ≥ 65 yo</td>
<td>155</td>
</tr>
<tr>
<td>Anterior AMI</td>
<td>115</td>
</tr>
</tbody>
</table>
### 2007 ACC/AHA STEMI Reperfusion Guidelines

<table>
<thead>
<tr>
<th>Balloon Inflation &lt; 90 minutes of ED Arrival</th>
<th>Balloon Inflation &gt; 90 minutes of ED Arrival</th>
</tr>
</thead>
<tbody>
<tr>
<td>(either your hospital or via transfer)</td>
<td>Symptoms &lt; 3 hrs in an Uncomplicated Pt.</td>
</tr>
<tr>
<td>PCI</td>
<td>SX &gt; 3 hours and/or Older &gt; 65-75, IDDM; CHF; CVA; PCI; AMI; ABG; Complicated Pt.</td>
</tr>
<tr>
<td>PCI then Transfer as Needed</td>
<td>PCI</td>
</tr>
</tbody>
</table>

Note: ACC/AHA states, “If symptoms greater than 3 hours, primary PCI is preferred... with a goal of within 90 minutes.” This includes those with CHF, elderly, or in shock.

ACEP states, “Administer fibrinolytic therapy to high risk patients whose STEMI is identified less than 6 hours after symptom onset and expected delay time from initial STEMI identification in the ED until PCI time is greater than 90 minutes.”
AMI Care Lytic vs. Lab vs. Lytic Pre PCI  
ASSENT-4 PCI: Primary vs. TNK-facilitated PCI in Patients with STEMI

**Background**

Lytics can be given sooner  
Yet they only give 54-60% TIMI-3 flow  
PCI works better – 95% TIMI-3 flow  
Yet many hospitals can not provide, or cannot meet a 90 minute D₂B  
Small studies have shown safety and benefit from a lytic “priming dose” pre PCI  
Lytics may begin opening the artery; “stops the clock”, completely opens some

**The Study**

Multi-center, International Randomized Trial  
Planned enrollment of 4,000  
TNK full dose then PCI vs. PCI directly  
All patients got ASA plus 60-70 units per kg UFH IVP  
Clopidogrel if stent

**Results**

Study stopped after 1320 patients  
Increased mortality in TNK-PCI group  
6% AMI with TNK-PCI (p=0.0105)  
Increased strokes and re-infarctions also seen

The Authors Concludes:  
“A strategy of full dose TNK and antithrombotic co-therapy, as used in this study and preceding PCI by 1-3 hours, was associated with more major adverse effects than PCI alone in STEMI and cannot be recommended.”

**My Initial Conclusions on ASSENT-4**

Agree, based on this highly anticipated study, (that I knew would show great benefits), do not even mention these two therapies together… but wait there is more:

Primary Angioplasty vs. Early Routine Post-Fibrinolysis Angioplasty for Acute
Myocardial Infarction with ST-Segment Elevations: 
**The GRACIA-2 non-inferiority, randomized, controlled trial.**

**The Study**
212 STEMI patients TNK-PCI vs. PCI alone  
TNK preceded PCI by 3-12 hours (vs. 1-3 in ASSENT-4)  
Used LMWH (Lovenox) if TNK (vs. UFH in ASSENT-4)  
91% of patients received Plavix or Ticlid (unlike ASSENT-4)

**The Results**
67% of TNK group was TIMI-3 at PCI (vs. 14%)  
Infarct size and LV fraction similar in both groups  
3% Mortality in TNK-PCI vs. 5% PCI only (p=ns)

Author Concludes:  
“To the best of our knowledge, this is the first evidence that the application of a combined lytic-based pharmacological and mechanical reperfusion approach to acute myocardial infarct is feasible and could safely allow a wide window for the definitive repair of the infarct related artery.”

**My Conclusions as of September 2007**
Lytics right before PCI not indicated  
**But if delay will be greater than 2-3 hours, Lytics pre PCI may “stop the clock”**  
May have broad applicability to rural centers  
Note: use of LMWH and Plavix in subsequent studies may alter how we think of lytic pre PCI.

**The 2007-2008 Guidelines for Unstable Angina and Non-ST Elevation AMI**


Newest Guidelines  
370 References; all major studies reviewed  
Endorsed by:  
ACC, AHA, AFP, ACEP, ACP, SAEM

**Has Four Classes of Recommendations:**

**Class I** Recommended by multiple trials or meta analysis
Class IIa  Recommended but some conflicting evidence
Class IIb  Recommended but efficacy less well established
Class III  Not recommended; could be harmful

And Has Three Levels of Evidence:
A Level  Multiple populations (3-5) studied
B Level  Limited populations (2-3) studied
C Level  Very limited population (1-2) studied

Specific Recommendations with a Focus on Changes and New Recommendations

EMS
All EMS should do 12 leads with computer assisted readings if available. (IIa).
Send ECG to ED if ACS detected. (IIa).

ED Evaluation
Repeat ECG if high risk patients Q 15-30 min. (I)
Troponin is preferred biomarker and should be measured in all patients. (I)
Repeat biomarkers at 8 hours if first assay done within 6 hours of CP onset. (I)
A 2 hour Delta CK-MB in conjunction with a Delta Troponin may be useful. (IIb)

ED Care if ACS Not Yet continued:
If ED evaluation is negative, a stress test to provoke ischemia should be done in the ED or shortly after discharge. (I)
If outpatient testing planned: ASA, NTG, and/or Beta Blockers should be prescribed. (I)
CTA is “reasonable” instead of a stress test in low or intermediate probability ACS. (IIa)

For ACS in ED and Hospital
Oral Beta Blockers within 24 hours unless contraindicated. (I)
Oral ACE-Inhibitor within 24 hours if signs of CHF or low EF (<40%) (I).
IV Beta Blockers if hypertensive, and BB not contraindicated. (IIa)
Clopidogrel if ASA allergic. (I)
Clopidogrel loading dose if invasive therapy planned or may choose 2b-3a antagonist. (I)
Clopidogrel loading dose if conservative therapy planned. (I)
If UA/Non-STEMI, add antiplatelet therapy ASAP. (I)
UFH, Enoxoparin, Bivalirudin, and Fondaparinux all acceptable if invasive therapy planned. (I)
Enoxoparin, Fondaparinux preferred over UFH if conservative therapy. (II)
Fondaparinux preferred if high risk of bleeding, and no PCI planned. (I)

Note: morphine for NTG refractory pain is now IIa down from class I due to CRUSADE
data on increased mortality.

**What is the Right Loading Dose of Clopidogrel**

New 2007 ACC/AHA guidelines state loading does of “at least 300mgs.”
Standard loading dose of 300 mgs takes about 6 hours to maximally inhibit patients.
Be aware two studies show 600-900mgs work in about 2 hours

**AHA for ACS 2b – 3a Inhibitor Use:**

Decrease platelet activity by about 80%
Early studies highly positive but were: preclopidogrel and pre newer anticoagulants.
2007 guidelines decrease emphasis on 2b-3a use
In lower risk patients either 2b-3a or clopidogral in combination with a heparin or heparinoid now recommended.
In high risk patients, troponin positive, going to PCI, 2b-3a use recommended at a IIb level, But can be started in PCI-Lab

**Role is focused now on PCI patients, and much less emphasis on early in-ED initiation**

Abciximab: only if PCI or PCI within 24 hours
Tirofiban: PCI and medical only patients with ACS
Eptifibatide: PCI and medical-only patients with ACS

**Take Home for 2b-3a Inhibitors**

Role continues to decline in ED
Will most likely be replaced by newer agents

**AHA/ACS**

**Who Goes to PCI:** “Invasive” vs. “Conservative”

Invasive:
- Positive Troponin
- Hemodynamics instability
- Dynamic ST-T wave changes
- Known CAD/Prior PCI with high risk history
- Positive Imaging Study

*Note: In stable patients, well controlled on medical management, PCI does not confer a long term benefit when compared to aggressive medical management.*

*COURAGE Trial: N Eng J Med*

2007;356:1503-1516

Know COURAGE’S conclusion – Its says stable patients… don’t have someone say that an ED chest pain patient can now go home as their pain is gone and “the literature says aggressive management with PCI doesn’t help”… it sure does for unstable patients with
vulnerable plaque.

**LMWH, Fondaparinux, and Bivalirudin for 2007**

**Enoxaparin versus Unfractionated Heparin with Fibrinolysis for ST-Elevation Myocardial Infarction (EXTACT-TIMI 25)**

**The Study**

Prior studies have shown LMWH superior to UFH (ASSENT-3)
Directly compared LMWH to UFH in 20,506 patients
Lytics included SK, TPA, RPA, TNK
UFH at 60 u/kg bolus; 12 u/kg an hour
LMWH 30 mg IV bolus; 1 mg/kg Q12h

**The Results**

LMWH: 9.9 Death or AMI at 30d
UFH: 12.0 Death or AMI at 30d
LMWH 17% superior (p<0.001)
23% better if subsequent PCI
Note: more bleeding, but not ICH with LMWH (2.1% vs. 1.4%)

**Take Home**

If you use Lytic, use LMWH.
Many centers are very cautious about IV loading; especially in the elderly.
**LMWH has more anti Xa vs. anti IIa than UFH.**

**Comparison of Fondaparinux and Enoxaparin in Acute Coronary Syndromes (OASIS-5)**

**The Study**

Direct Comparison of Fondaparinox to LMWH
20,078 patients with ACS for about 6 days
Fondaparinox 2.5mg vs. Lovenox 1mg/kg BID
Evaluated Death, AMI, refractory ischemia at 9 days
Six month follow up

**The Results**

Primary outcomes the same (5.8% vs. 5.7%)
Less deaths with Fondaparinox at 30 and 180 days (p=0.05)
Primary outcomes plus bleeding was less with Fondaparinox
(7.3% vs. 9.0%; p<0.001)

**Take Home**

Fondaparinux may be superior to LMWH.
Causes less bleeding, resulting in improved survival.
Should know it’s a direct Xa inhibitor, unlike LMWH which affects IIa also.
Effects of Fondaparinux on Mortality and Reinfarction in Patients With Acute ST-Segment Elevation Myocardial Infarction (OASIS-6)

**Background**
Fondaparinux is a factor Xa inhibitor
Has track record in DVT prophylaxis

**The Study**
12,092 STEMI patients
UFH vs. Fondaparinux (2.5 mg QD)

**The Results**
Fondaparinux: 9.7% Death or AMI at 30d (31% better)
UFH: 11.2% Death or AMI
No increased bleeding with Fondaparinux

**Take Home**
Study’s importance is the no bleeding increase as compared to ↑ risk with LMWH

Bivalirudin for Patients with Acute Coronary Syndromes (ACUITY)

**Background**
Bivalirudin is a direct-acting antithrombin
Has shown promise vs. UFH + 2b-3a in PCI patients

**The Study**
13,819 ACS patients who would get PCI
UFH or LMWH + 2b-3a vs. BiV + 2b-3a vs. BiV alone
Evaluated death, Ami, Urgent Revase, and Bleeding

**The Results**
Bivalirudin alone as compared to BiV with a 2b-3a, or compared to UFH with a 2b-3a was just as good.
30d Combined End Points: LMWH or UFH + 2b-3a vs. Bivalirudin + 2b-3a vs. Bivalirudin alone 11.7% vs. 11.8% vs. 10.1%
Less bleeding was seen with Bivalirudin alone (p<0.001)
3.0% vs. 5.7%

**Take Home Points**
Bivalirudin will be used more
Do not need to add 2b-3a (at least pre PCI in ACS)
Less bleeding with equal efficacy
Bivalirudin is a direct thrombin antagonist
ACEP Clinical Policy: Critical Issues in the Evolution and Management of Adult Patients with NON-ST-Segment Elevation Acute Coronary Syndromes

ACEP Question 1:
Are serial ECGs useful during the ED Evaluation of patients with suspected acute coronary syndromes?
Consensus Answer: YES.
Repeat ECG during ED evaluation
No recommendation on timing: 30-60 min after initial ECG is reasonable (B)
Likelihood of finding new changes based on risk status (B)

ACEP Question 2:
Is there a preferred regimen of serum markers testing in the ED for the exclusion on NON-STEMI AMI?
Consensus Answer:
Do not use markers to rule out unstable angina (A)
A negative CK-MB or Troponin 8-12 hours AFTER symptom onset can rule out NON-STEMI (B)
A negative Delta CK-Mass plus delta troponin mass may be used in patients presenting under 8 hours (B)
A negative myoglobin plus negative ck-mg or troponin at baseline and 90 minutes may also be used (B)

ACEP Question 3:
What are the indications for ED administration of glycoprotein IIb/IIIa inhibitors with Non-STEMI ACS?
Consensus Answer:
Consider administration prior to PCI if early intervention
Positive Troponin or
Ischemic ST segment depression (Level C)
Consider IIb/IIIa administration if no intervention planned (C)

ACEP Question 4:
What are the indications for ED Administration of Clopidogrel in patients with NON-STEMI ACS?
Consensus Answer: (Level B)

Administer a loading does of Clopidogrel in a patients with
Positive Troponin or
Ischemic ST depression

Patients not going to PCI or those going to PCI but not high risk for urgent bypass surgery.

**Optimal timing of dose (in ED vs. Lab) can not be determined; standard loading dose takes 6 hours for greatest benefit**
References:

**Congestive Heart Failure**


**Coronary Angiography**


**Syncope**


**D2B – Prehospital ECG**


Electrocardiography

Arrhythmia Management


TASER


Cardiopulmonary Resuscitation


STEMI and UA

Bates E, Spertus J, Berman D, Mancini J, Weintraub W. “Optimal Medical Therapy With or Without PCI for Stable Coronary Disease.”


Unstable Angina and Non-ST Elevation AMI


BiPAP is Level C

Annals Emerg Med 2006;48:260-269
Lancet 2006;367:1155-1163

Your Gestalt and Fear Need to Be Used!

Circulation 2006; 114: 1565-1571

One ECG Begets Another


Tasers have been associated with more than 200 death in patients in custody or with ED – Excited Delirium

Acad Emerg Med 2007; 14: S 104
Lancet 2007; 369: 920-926

Compression only Cardiac Resuscitation for first 4-12 minutes may be equal to, or superior to, full CPR.
LBBB with Three Criteria for STEMI

ISAR-REACT JACC 2004; 44:2133-2136
ARMYDA-2 Circulation 2005; 111:2099-2106

New Eng J Med 2006;354:1477-1488
JAMA 2006;295:1519-1530
New Eng J Med 2006;355:2203-2216
Circulation 2006;114:2019-2025

Heart failure likely if:
BNP > 500 pg/dl or Pro-BNP > 1000
(LR + = 6)
Age must be used too.

Posterior AMI

\[ V_2 \]

*New Eng J Med* 2006;354:1464-1476

*Annal of Emerg Med* 2006;48:270-301

115 Refs; many studies abstracted

ACEP Clinical Policy

"There is insufficient information at this time to make any recommendations in regards to the exact location or timing for initiation of glycoprotein IIb/IIa inhibitor therapy (i.e. ED vs. in-hospital)."