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

Ann Emerg Med 2007:49:627-669

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 <u>unlikely</u> if:

BNP < 100 pg/dl or Pro-BNP < 300
(LR - = 0.1)

<u>Key Points on BNP and Pro-BNP:</u>

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.

<u>CPAP:</u>
Improves BP, P, RR
Decreases need to intubate
Possibly" reduces mortality "

(Note other studies have shown 40-50% decrease in need to intubate and 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, <u>but</u> only for moderate to severe pulmonary edema when <u>combined with NTG.</u> (Level B)

BUT…

Be careful with diuretics in HF: Level C
Aggressive therapy with diuretics alone do not decrease need to intubate
Give judiously, 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)

<u>Article's 5 Key Take Home Points</u>

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.
<u>Not</u> 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

Ann of Emerg Med 2007;49:125-136
JACC 2007;49:863-871
Acad EmergMed 2007;14:112-116
J Am Coll Cardiol 2006;48:1919-1928
Circulation 2006;114:2251-2260

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, Hyperthryoidism, 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.

Author	Journal	n	PPV	NPV
Hoffman	Circ 2006	103	61%	100%
Rubenshtein	Circ 2007	58	87%	100%
Hollander	Acad EM 2007	54	80%	100%
Goldstein	JACC 2007	99	87.5%	100%
Gallagher	Ann EM 2007	85	50%	99*-100%

** 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

Diagnostic Studies	(mSv)
PA/Lateral CXR	0.08
Mammogram	0.13
Cardiac Catheterization	4-6
CT Abdomen and Pelvis	7-8
64 Slice MDCT (with/without ECG Pulsing)	**Male 4.8-10 Female 6.8-14**
Tc-99 Rest-Stress MIBI	12

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

Ann Emerg Med 2007:49:431-444

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 <u>non exertional</u> syncope, and no history or signs of cardiac disease, no family history of sudden death, and no comorbities 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?

<u>Four Key High Risk Factors Identified</u>

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

<u>San Francisco Syncope Rule and Validation</u>

Ann Emerg Med 2004;43:224-232
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 <u>NOT</u> 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

Ann Emerg Med
2006;47:448-454

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
- S_1, Q_3, T_3
- Brugada's (V_1, V_2, V_3)
- Voltage of LVH
- Prolonged Q-T

The D_2B Alliance

The Role of Prehospital ECGs

D_2B 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

N Engl J Med 2006;355:2308-2320

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

N Engl J Med 2006;355:2308-2320

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:

EMBED PowerPoint.Slide.8

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

EMBED PowerPoint.Slide.8

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 D_2B from 125 min to 63 min if only using PCI centers

EMBED PowerPoint.Slide.8

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
O_2, ASA, NTG, 12-lead ECG, Prehospital Alert
Rapid Transport to Heart Hosptial
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
D_2B 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, V_2 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, V_5-V_6)
Anterior	(V Leads)
RV	(Deep ST in V_1-V_2 especially with Int AMI)
Post	(V_2: 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

Annals of Emerg Med 2006;47:227-229

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 Strenghts
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

JAMA

1971;215:1454-1460

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.

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_C 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 sub optimal
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. *Annal of Emerg Med 2007;49:314-316.*

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 ≥ 1 mm in 2 or more Limb Leads
ST elevation ≥ 2 mm in 2 or more contiguous Precordial Leads
Any BBB which obscures reading STEMI in patients

Level B:
ST elevation ≥ 1 mm in 2 or more contiguous Precordial Leads
New LBBB
LBBB with ≥ 1 mm ST ↑ in direction of QRS; ≥ 5 mm ST deviation away from positive QRS or ST ↓ ≥ 2 mm in leads V_1-V_3.

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

Note: See NEJM 1996;334:931

SHAPE * MERGEFORMAT

1) ST elevation ≥ 1 mm in same direction as QRS (concordant ST ↑)
2) ST elevation ≥ 5 mm in opposite direction as QRS (discordant ST ↑)
3) ST depression ≥ 2 mm in V_1-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 V_1-V_2-V_3
- R > S in V_2 (or V_3)
- 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 D_2B (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?

Variable	Time in Min
Symptoms ≤ 120 min	94
Symptoms ≥ 120 min	190
Age < 65 yo	71
Age ≥ 65 yo	155
Anterior AMI	115

Non-Anterior AMI	112

2007 ACC/AHA STEMI Reperfusion Guidelines

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

*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 D_2B

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 theses two therapies together… ***but wait there is more:***

Primary Angiplasty vs. Early Routine Post-Fibrinolysis Angiplasty 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

ACC/AHA 2007 Guideline for the Management of Patients With Unstable Angina/ Non-ST-Elevation Myocardial Infarction: Executive Summary.

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%) (1).
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 ($p=0.05$)
Primaray outcomes plus bleeding was less with Fondaparinox

(7.3% vs. 9.0%; $p<0.001$)

Take Home

Fondaparinox 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 prophyloxis

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 Fondaparinox

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
Evalutated death, Ami, Urgent Revasc, 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
Bivalarudin 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

Silvers S, Howell J, Kosawsky J, Rokos I, Jadoda A. "Clinical Policy: Critical Issues in the Evaluation and Management of Adult Patients Presenting to the Emergency Department with Acute Heart Failure Syndromes." *Ann Emerg Med 2007;49: 627-669.*

Collins, S. P., L. M. Mielniczuk, et al. "The use of noninvasive ventilation in emergency department patients with acute cardiogenic pulmonary edema: a systematic review." *Ann Emerg Med 2007;48: 260-9, 269 e1-4.*

Peter, J. V., J. L. Moran, et al. "Effect of non-invasive positive pressure ventilation (NIPPV) on mortality in patients with acute cardiogenic pulmonary oedema: a meta-analysis." *Lancet 2006; 367: 1155-63.*

Coronary Angiography

Gallagher M, Ross M, Raff G, Goldstein J, O'Neil W, O'Neail B. "The Diagnostic Accuracy of 64-Slice computed Tomography Coronary Angiography Compared with Stress Nuclear Imagine in Emergency Department Low-Risk Chest Pain Patients." *Ann of Emerg Med 2007;49:125-136.*

Goldstein, J. A., M. J. Gallagher, et al. "A randomized controlled trial of multi-slice coronary computed tomography for evaluation of acute chest pain." *J Am Coll Cardiol 2007; 49: 863-71.*

Hollander, J. E., H. I. Litt, et al. "Computed tomography coronary angiography for rapid disposition of low-risk emergency department patients with chest pain syndromes." *Acad Emerg Med 2007; 14(2): 112-6.*

Rubinshtein R, Halon D, Gaspar T, Jaffe R, Karkabi B, Flugelman M, Koga A, Shapira R, Peled N, Lewis B. "Usefulness of 64-Slice Cardiac Computed Tomographic Angiography for Diagnosing Acute Coronary Syndromes and Predicting Clinical Outcome in Emergency Department Patients With Chest Pain of Uncertain Origin." *Circulation 2007;115:1762-1768.*

Achenbach, S. "Computed tomography coronary angiography." *J Am Coll Cardiol 2006; 48(10): 1919-28.*

Hoffmann, U., J. T. Nagurney, et al. "Coronary multidetector computed tomography in the assessment of patients with acute chest pain." *Circulation 2006; 114: 2251-60.*

Syncope

Cosgriff, T. M., A. M. Kelly, et al. "External validation of the San Francisco Syncope Rule in the Australian context."
CJEM 2007; 9: 157-61.

Huff, J. S., W. W. Decker, et al. "Clinical policy: critical issues in the evaluation and management of adult patients presenting to the emergency department with syncope." *Ann Emerg Med 2007; 49: 431-44.*

Sun, B. C., J. R. Hoffman, et al. "Older age predicts short-term, serious events after syncope." *J Am Geriatr Soc 2007;55: 907-12.*

Sun, B. C., C. M. Mangione, et al. "External validation of the San Francisco Syncope Rule."
Ann Emerg Med 2007; 49: 420-7, 427 e1-4.

Quinn, J., D. McDermott, et al. "Prospective validation of the San Francisco Syncope Rule to predict patients with serious outcomes." *Ann Emerg Med 2006; 47: 448-54.*

Schladenhaufen R, Benenson R, Feilinger S, Pollack M. "Application of San Francisco Syncope Rule to Elderly Emergency Department Patients." *Euro J Emerg Med 2006;13:A7.*

Quinn, J. V., I. G. Stiell, et al. "Derivation of the San Francisco Syncope Rule to predict patients with short-term serious outcomes." *Ann Emerg Med 2004;43: 224-32.*

Colivicchi, F., F. Ammirati, et al. "Development and prospective validation of a risk stratification system for patients with syncope in the emergency department: the OESIL risk score." *Eur Heart J 2003;24: 811-9.*

Martin, T. P., B. H. Hanusa, et al. "Risk stratification of patients with syncope." *Ann Emerg Med 1997; 29: 459-66.*

D2B – Prehospital ECG

"ACC/AHA 2007 Guidelines for the Management of Patients With Unstable Angina/Non ST-Elevation Myocardial Infarction: Executive Summary: A Report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (Writing Committee to Revise the 2002 Guidelines for the Management of Patients With Unstable Angina/Non ST-Elevation Myocardial Infarction): Developed in Collaboration with the American College of Emergency Physicians, the Society for Cardiovascular Angiography and Interventions, and the Society of Thoracic Surgeons: Endorsed by the American Association of Cardiovascular and Pulmonary Rehabilitation and the Society for Academic Emergency Medicine." *Circulation 2007;116: 803-77.*

Bradley, E. H., J. Herrin, et al. "Strategies for reducing the door-to-balloon time in acute myocardial infarction." *N Engl J Med 2006;355: 2308-20.*

Jacobs A, Antman E, Faxon D, Gregory T, Solis P. "Development of Systems of Care for ST-Elevation Myocardial Infarction Patients: Executive Summary." *Circulation 2006;116:217-230.*

Faxon, David. "Development of Systems of Care for ST-Elevation Myocardial Infarction Patients: Current State of ST-Elevation Myocardial Infarction Care. *Circulation 2006; 116:e29-e32.*

Kalla, K., G. Christ, et al. "Implementation of guidelines improves the standard of care: the Viennese registry on reperfusion strategies in ST-elevation myocardial infarction (Vienna STEMI registry)." *Circulation 2006;113: 2398-405.*

Le May, M. R., R. F. Davies, et al. "Comparison of early mortality of paramedic-diagnosed ST-segment elevation myocardial infarction with immediate transport to a designated primary percutaneous coronary intervention center to that of similar patients transported to the nearest hospital." *Am J Cardiol 2006;98: 1329-33.*

Swor, R., S. Hegerberg, et al. "Prehospital 12-lead ECG: efficacy or effectiveness?" *Prehosp Emerg Care 2006;10: 374-7.*

Verheugt, F. W. "Reperfusion therapy starts in the ambulance." *Circulation 2006;113: 2377-9.*

Sgarbossa E, Pinski S, Barbagelata A, Underwood D, Gates K, Topol E, Califf R, Wagner G. "Electrocardiographic Diagnosis of Evolving Acute Myocardial Infarction in the presence of Left Bundle-Branch Block."
N Engl J Med 1996;334:481-487.

Electrocardiography
Masoudi, F. A., D. J. Magid, et al. "Implications of the failure to identify high-risk electrocardiogram findings for the quality of care of patients with acute myocardial infarction: results of the Emergency Department Quality in Myocardial Infarction (EDQMI) study." *Circulation 2006; 114: 1565-71.*

Arrhythmia Management
Cummins, R. O. and M. F. Hazinski. "The quest for a terminator." *Ann Emerg Med 2006;47: 227-9.*

Marill, K. A., I. S. deSouza, et al. "Amiodarone is poorly effective for the acute termination of ventricular tachycardia." *Ann Emerg Med 2006;47: 217-24.*

Gorgels, A. P., A. van den Dool, et al. "Comparison of procainamide and lidocaine in terminating sustained monomorphic ventricular tachycardia." *Am J Cardiol 1996;78: 43-6.*

Armengol, R. E., J. Graff, et al. "Lack of effectiveness of lidocaine for sustained, wide QRS complex tachycardia." *Ann Emerg Med 1989;18: 254-7.*

Koch-Weser, J. and S. W. Klein. "Procainamide dosage schedules, plasma concentrations, and clinical effects."
JAMA 1971;215: 1454-60.

TASER
Levine, S. D., C. M. Sloane, et al. "Cardiac monitoring of human subjects exposed to the taser(r)." *J Emerg Med 2007;33: 113-7.*

Valentino D, Walter R, Dennis A, Margeta B, Nagy K, Winners J, Bokhari F, Wiley D, Joseph K, Robert R. "TASER Discharges Capture Cardiac Rhythm in a Swine Model." *Acad Emerg Med 2007;14:S 104-a*

Cardiopulmonary Resuscitation
"Cardiopulmonary resuscitation by bystanders with chest compression only (SOS-KANTO): an observational study." *Lancet 2007;369: 920-6.*

Eisenberg, M. S. "Improving Survival From Out-of-Hospital Cardiac Arrest: Back to the Basics." *Ann Emerg Med 2007;49:314-316.*

STEMI and UA
Fernandez-Aviles et al. "Primary Angioplasty vs. Early Routine Post-Fibrinolysis Angioplasty for Acute Myocardial Infarction with ST-Segment Elevations: The Gracia-2 non-inferiority, randomized, controlled trial." *European Heart 2007; 28:949-960.*

William B, O'Rourke R, Teo K, Hartigan P, Maron D, Kostuk W, Knudtson M, Dada M, Casperson P, Harris C, Pharm D, Chaitman B, Shaw L, Gosselin G, Nawaz A, Title L, Gau G, Blaustein A, Booth D,

Bates E, Spertus J, Berman D, Mancini J, Weintraub W. "Optimal Medical Therapy With or Without PCI for Stable Coronary Disease."
New Engl J of Med 2007;356:1503-1516.

Fesmire, F. M., W. J. Brady, et al. "Clinical policy: Indications for Reperfusion Therapy in Emergency Department Patients with Suspected Acute Myocardial Infarction. American College of Emergency Physicians Clinical Policies Subcommittee (Writing Committee) on Reperfusion Therapy in Emergency Department Patients with Suspected Acute Myocardial Infarction." *Ann Emerg Med 2006;48:358-83.*

Fernandez-Aviles et al. "Primary Angioplasty vs. Early Routine Post-Fibrinolysis Angioplasty for Acute Myocardial Infarction with ST-Segment Elevations: The Gracia-2 non-inferiority, randomized, controlled trial." *European Heart 2007; 28:949-960.*

"Primary Versus Tenecteplase-Facilitated Percutaneous Coronary Intervention in Patients with ST-Segment Elevation Acute Myocardial Infarction (ASSENT-4 PCI): Randomized Trial." *Lancet 2006;367: 569-78.*

"Primary versus tenecteplase-facilitated percutaneous coronary intervention in patients with ST-segment elevation acute myocardial infarction (ASSENT-4 PCI): randomized trial." *Lancet 2006;367: 569-78.*

Pinto, D. S., A. J. Kirtane, et al. "Hospital delays in reperfusion for ST-elevation myocardial infarction: implications when selecting a reperfusion strategy." *Circulation 2006;114: 2019-25.*

Patti, G., G. Colonna, et al. "Randomized trial of high loading dose of clopidogrel for reduction of periprocedural myocardial infarction in patients undergoing coronary intervention: results from the ARMYDA-2 (Antiplatelet therapy for Reduction of MYocardial Damage during Angioplasty) study." *Circulation 2005; 111: 2099-106.*

Kandzari, D. E., P. B. Berger, et al. "Influence of treatment duration with a 600-mg dose of clopidogrel before percutaneous coronary revascularization." *J Am Coll Cardiol 204; 44: 2133-6.*

Stone G. W. et al. "Bivalirudin for Patients with Acute Coronary Syndromes." *N Engl J Med 2006;355: 2203-2216.*

Unstable Angina and Non-ST Elevation AMI

"ACC/AHA 2007 Guidelines for the Management of Patients With Unstable Angina/Non ST-Elevation Myocardial Infarction: Executive Summary: A Report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (Writing Committee to Revise the 2002 Guidelines for the Management of Patients With Unstable Angina/Non ST-Elevation Myocardial Infarction): Developed in Collaboration with the American College of Emergency Physicians, the Society for Cardiovascular Angiography and Interventions, and the Society of Thoracic Surgeons: Endorsed by the American Association of Cardiovascular and Pulmonary Rehabilitation and the Society for Academic Emergency Medicine." *Circulation 2007;116: 803-77.*

Fesmire F, Decker W, Diercks D, Ghaemmaghami C, Nazarian D, Brady W, Hahn S, Jagoda S. "Clinical Policy: Critical Issues in the Evaluation and Management of Adult Patients with Non-ST-Segment Elevation Acute Coronary Syndromes." *Ann of Emerg Med 2006;48:270-301.*

Stone G. W. et al. "Bivalirudin for Patients with Acute Coronary Syndromes." *N Engl J Med 2006;355: 2203-2216.*

Antman, E. M., D. A. Morrow, et al. "Enoxaparin Versus Unfractionated Heparin with Fibrinolysis for ST-Elevation Myocardial Infarction." *N Engl J Med 2006; 354: 1477-88.*

Yusuf, S., S. R. Mehta, et al. "Effects of fondaparinux on mortality and reinfarction in patients with acute ST-segment elevation myocardial infarction: the OASIS-6 randomized trial." *JAMA 2006; 295: 1519-30.*

Yusuf, S, Mehta S, Chrolavicius S, Afzal R, Pogue J, Granger C, Budaj A, Peters R, Bassand j, Wallentin L, Joyner C, Fox K. "Comparison of Fondaparinux and Enoxaparin in Acute Coronary Syndromes (OASIS-5)."
N Engl J Med 2006;354:1464-1476.

PAGE

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BiPAP is Level C

Annals Emerg Med 2006;48:260-269

Lancet 2006;367:1155-1163

Can J Emerg Med 2007;9:157-161

J Am Geriatr Soc 2007;55:907-912

Your Gestalt and Fear Need to Be Used!

Circulation 2006; 114: 1565-1571

One ECG Begets Another

Annals of Emerg Med 2006; 47: 217-224

Journal of Emerg Med 2007; 33:113-117

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.

Annals of Emerg Med 2006;48:358-383

Lancet 2006; 367: 569-578

Circulation 2007; 116:803-877

European Heart 2007; 28: 949-960

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

EMBED PowerPoint.Slide.8

Circulation 2006;114:2019-2025

Circulation 2006;114:2019-2025

Heart failure likely if:
BNP > 500 pg/dl or Pro-BNP > 1000
(LR + = 6)

1

2

3

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).