Primary goals of therapy for patients of ACS

- Reduce the amount of myocardial necrosis that occurs in patients with acute myocardial infarction (AMI), thus preserving left ventricular (LV) function, preventing heart failure, and limiting other cardiovascular complications.
- Prevent major adverse cardiac events (MACE): death, nonfatal MI, and need for urgent revascularization.

Primary goals of therapy for patients of ACS

- Treat acute, life-threatening complications of ACS, such as ventricular fibrillation (VF), pulseless ventricular tachycardia (VT), unstable tachycardias, symptomatic bradycardias, pulmonary edema, cardiogenic shock and mechanical complications of AMI.

Potential delays:

- From onset of symptoms to patient recognition: older age, racial and ethnic minorities, female gender, lower socioeconomic status, and solitary living arrangements.
- During pre-hospital transport: non-classical patient presentations and other confounding diagnostic issues to provider misinterpretation of patient data and inefficient in-hospital system of care.
• During ED evaluation: evaluation of AMI combined with symptoms, EKG, biomarkers, risk factors, and other diagnostic tests. Atypical and unusual symptoms are more common on women, the elderly, and diabetic patients.

• Public education campaigns increase patient awareness and knowledge of the symptoms of ACS, yet have only transient effects on time to presentation.

Public education campaigns increase patient awareness and knowledge of the symptoms of ACS, yet have only transient effects on time to presentation.

ECG Interpretation

• ST-segment elevation or presumed new LBBB is characterized by ST-segment elevation in 2 or more contiguous leads and is classified as ST-segment elevation MI (STEMI).

• Threshold values for ST-segment elevation consistent with STEMI are J-point elevation 0.2 mV (2 mm) in leads V2 and V3 and 0.1 mV (1 mm) in all other leads (men > 40 years old); J-point elevation 0.25 mV (2.5 mm) in leads V2 and V3 and 0.1 mV (1 mm) in all other leads (men < 40 years old); J-point elevation 0.15 mV (2.5 mm) in leads V2 and V3 and 0.1 mV (1 mm) in all other leads (women).

ECG Interpretation

• Ischemic ST-segment depression >0.5 mm (0.05 mV) or dynamic T-wave inversion with pain or discomfort is classified as UA/NSTEMI. Nonpersistent or transient ST-segment elevation <0.5 mm for <20 minutes is also included in this category.

• Threshold values for ST-segment depression consistent with ischemia are J-point depression 0.05 mV (-0.5 mm) in leads V2 and V3 and -0.1 mV (-1 mm) in all other leads (men and women).
ECG Interpretation

- The nondiagnostic ECG with either normal or minimally abnormal (i.e., nonspecific ST-segment or T-wave changes). This ECG is nondiagnostic and inconclusive for ischemia, requiring further risk stratification.
- This classification includes patients with normal ECGs and those with ST-segment deviation of <0.5 mm (0.05 mV) or T-wave inversion of ≤0.2 mV.
- This category of ECG is termed **nondiagnostic**.

Cardiac Biomarkers

- Cardiac troponin is more sensitive than CK-MB.
- Cardiac biomarkers ARE NOT USEFUL in the pre-hospital setting.
- If biomarkers are negative in first 4~6 hours, recheck between 6~12 hours.
- Clinical symptoms + new ECG abnormalities + one biomarker is elevated above the upper limit = MI.
Initial General Therapy for ACS

- Analgesia: Morphine preferred for STEMI (Class I); Class II for UA/NSTEMI.
- Oxygen: no sufficient evidence of using oxygen; harmful if using high-flow oxygen.
- Nitroglycerin: careful with low BP patients; contraindication: hypotension, bradycardia, tachycardia, RV infarction.
- Aspirin

Reperfusion Therapies

- Fibrinolytic therapy: Door-to-needle in 30 minutes.
- Primary PCI: Door-to-balloon inflation in 90 minutes.

**PCI following ROSC after Cardiac Arrest**

- A 12-lead ECG should be performed as soon as possible after ROSC.
- Appropriate treatment of ACS or STEMI, including PCI or fibrinolysis, should be initiated regardless of coma.
- Coma and the use of induced hypothermia are not contraindications or reasons to delay PCI or fibrinolysis.

**PCI vs. Fibrinolysis**

- For patients presenting within 12 hours of symptom onset and electrocardiographic findings consistent with STEMI, reperfusion should be initiated ASAP.
- Primary PCI performed at a high-volume center within 90 minutes of first medical contact by an experienced operator that maintains an appropriate status is reasonable, as it improves morbidity and mortality as compared with immediate fibrinolysis (30 minutes door-to-needle).

**PCI vs. Fibrinolysis**

- If PCI cannot be accomplished within 90 minutes of first medical contact, independent of the need for emergent transfer, then fibrinolysis is recommended, assuming the patient lacks contraindications to such therapy.
- For those patients with a contraindication to fibrinolysis, PCI is recommended despite the delay, rather than foregoing reperfusion therapy.
- For those STEMI patients presenting in shock, PCI (or CABG) is the preferred reperfusion treatment. Fibrinolysis should only be considered in consultation with the cardiologist if there is a substantial delay to PCI.
Complicated AMI
• Cardiogenic shock, LV failure, and CHF: PCI preferred as fibrinolysis; use IABP for hemodynamic support.
• RV infarction: Right side ECG; PCI as soon as fibrinolysis; avoid NTG, diuretics, or ACEI due to severe hypotension (↓ cardiac output); treat hypotension with IV bolus.

Adjunctive Therapies for ACS and AMI
• Thienopyridines—Clopidogrel, Prasugrel
• Clopidogrel: ≤75 y/o: 300~600mg loading if ACS is diagnosed; 300mg loading with patient suspect ACS (but no ECG or cardiac biomarkers change) who are allergic to aspirin or major GI intolerance; ≤75 y/o: no strong evidence.
• Prasugrel: (60mg loading dose) reduction in combined event rate with no benefit in mortality compared to clopidogrel but with an overall resultant increase in major bleeding when administered after angiography to patients with NSTEMI undergoing PCI; no direct evidence of using Prasugrel at ED or prehospital settings.

Adjunctive Therapies for ACS and AMI
• Glycoprotein Ilb/IIia Inhibitors: dual platelet inhibitor treatment of patients with planned invasive strategy taking into consideration the ACS risk of the patient and weighing this against the potential bleeding risk.
• β-Adrenergic Receptor Blockers: Contraindications are moderate to severe LV failure and pulmonary edema, bradycardia, hypotension, signs of poor peripheral perfusion, second-degree or third-degree heart block, or reactive airway disease; PO vs. IV (severe HTN or tachyarrhythmias with ACS).

Adjunctive Therapies for ACS and AMI
• Calcium Channel Blockers: no evidence; beta-blockers are better than CCB with AMI patients.
• ACEI & ARB: oral ACEI is recommended within the first 24 hours after onset of symptoms in STEMI patients with pulmonary congestion or LVEF<40%; in the absence of hypotension; oral ACEI can also be useful for all other patients with AMI with or without early reperfusion therapy; IV administration of ACEI is contraindicated in the first 24 hours because of risk of hypotension.

Adjunctive Therapies for ACS and AMI
• HMG Coenzyme A Reductase Inhibitors (Statins): intensive (target LDL values optimally <70mg/dL) statin treatment should be initiated within the first 24 hours after onset of an ACS event in all patients presenting with any form of ACS unless strictly contraindicated.
• Glucose-Insulin-Potassium: no evidence, not helpful.

Adjunctive Therapies for ACS and AMI
• Heparin: UFH vs. LMWH
• UFH: need IV: aPTT; unpredictable response; thrombocytopenia.
• UFH vs. LMWH in UA/ NSTEMI: initial conservative approach—enoxaparin better than UFH; planned invasive approach—enoxaparin or UFH; CRI—UFH; increased bleeding risk—UFH may be considered.
Adjunctive Therapies for ACS and AMI

- UFH vs. LMWH with Fibrinolysis in STEMI: enoxaparin better than UFH (decreased bleeding risk); may use UFH if CRI.
- UFH vs. LWMH with PPCI in STEMI: enoxaparin better than UFH (decreased bleeding risk); need dose adjustment of enoxaparin if CRI.

Summary

- Early detect, early contact EMS, early CAB support by EMS, early ECG classification, early transport.
- PCI is preferred than fibrinolysis.
- Door-to-balloon: 90 minutes.
- Door-to-needle: 30 minutes.

The “8D’s” of Stroke Care

- Detection: Rapid recognition of stroke symptoms.
- Dispatch: Early activation and dispatch of emergency medical services (EMS) system by calling 911.
- Delivery: Rapid EMS identification, management, and transport.
- Door: Appropriate triage to stroke center.
- Data: Rapid triage, evaluation, and management within the emergency department (ED).
- Decision: Stroke expertise and therapy selection.
- Drug: Fibrinolytic therapy, intra-arterial strategies.
- Disposition: Rapid admission to stroke unit, critical-care unit.

Stroke Recognition and EMS care

- Stroke warning signs
- 119 and EMS Dispatch
- Stroke assessment tools: CPSS & LAPSS
Table 1. The Cincinnati Prehospital Stroke Scale

- Facial droop (patient drops teeth or smile)
- Normal — both sides of face move equally
- Abnormal — one side of face does not move as well as the other side
- Arm drift (patient closes eyes and holds both arms straight out for 10 seconds)
- Normal — both arms move the same or both arms do not move at all
- Abnormal — one arm does not move or one arm drifts compared with the other
- Abnormal speech (have the patient say “you can’t teach an old dog new tricks”)  
- Normal — patient uses correct words with no slurring
- Abnormal — patient slurs words, uses the wrong words, or is unable to speak

Interpretation: If any 1 of these 3 signs is abnormal, the probability of a stroke is 70%.

Table 2. Algorithm for Antithrombotic Therapy in Acute Ischemic Stroke Patients Who Are Potential Candidates for Acute Reparative Therapy

- Patient ultimately eligible for acute reparative therapy except that blood pressure is >185/105 mm Hg
- Labetalol 10-20 mg IV over 1-2 minutes, may repeat x 2-3, or
- Nitroprusside 5 mg/hr, titrate up to 2.5 mg/hr every 15-30 minutes, maximum 15 mg/hr, when desired blood pressure reached, lower to 3 mg/hr, or
- Other agents (hydralazine, enalaprilat, etc.) may be considered when appropriate
- Blood pressure is not maintained: Stroke Patients Who Are Not Potential Candidates for Acute Reparative Therapy

- Management of blood pressure
- Consider lowering blood pressure in patients with acute ischemic stroke if systolic blood pressure >185 mm Hg or diastolic blood pressure >105 mm Hg
- Consider blood pressure reduction as indicated for other coexistent organ system injury
- Initial BP 100-129 mm Hg
- Labetalol 10 mg IV bolus
- Nitroprusside 5 mg/hr, then 3-5 mg/hr
- Acute myocardial infarction
- CONSENSUS trial
- Acute diastolic stroke
- If blood pressure not contralateral, a reasonable target is to lower blood pressure by 15% to 25% within the first day

Table 3. Approach to Antithrombotic Therapy in Acute Ischemic Stroke Patients Who Are Potential Candidates for Acute Reparative Therapy

- Patient remains candidate for fibrinolytic therapy
- Review risks/benefits with patient and family
- Give tPA
- No anticoagulants or antiplatelet treatment for 24 hours

- Begin post-tPA stroke pathway
- Approach the patient
  - BP protocol (Tables 2 and 3)
- For neurologic evaluation
- Emergent admission to stroke unit or intensive care unit

- Candidate

- Not a Candidate
General Stroke Care

- **Blood pressure management**: keep adequate perfusion to maintain euvoelma.
- **Glycemic control**: no direct evidence that improves clinical outcome; keep F/S ≤ 185 mg/dL.
- **Temperature control**: treat fever ≥ 37.5°C; no evidence of hypothermia therapy.
- **Dysphagia screening**: to prevent aspiration pneumonia.
- **Others**: Airway, O2, nutrition; seizure vs. anticonvulsants; ICP.

Summary

- The ultimate goal of stroke care:
- To minimize ongoing injury.
- Emergently recanalize acute vascular occlusions.
- Begin secondary measures to maximize functional recovery.

Thanks for your attention!