ACOI 2015 Board Review
Management of Chronic Coronary Syndromes

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What you learn in Vegas, must leave Vegas!!
NO... NO... I SAID I'VE GOT ACUTE ANGINA
NO DISCLOSURES
Chronic Angina Is Prevalent in the United States

- ~10 million Americans have angina pectoris
  - 500,000 new cases are reported annually
- Median angina frequency is ~2 episodes per patient per week
  - > 18 million episodes each week or ~30 episodes each second

New Cases of Stable Angina Per Year (Among Americans ≥ 45 Years of Age)

<table>
<thead>
<tr>
<th>Incidence (Number of New Cases)</th>
<th>Men</th>
<th>Women</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>320,000</td>
<td>180,000</td>
<td>500,000</td>
</tr>
</tbody>
</table>

Pain Symptoms Occur at the End of the Ischemic Cascade

- Biochemical Alterations
- Relaxation
- Contraction
- Diastolic Dysfunction
- Systolic Dysfunction
- ECG Δ
- ST alterations

Adapted from Kern MJ. In: Braunwald's Heart Disease. 7th ed. 2005.
Canadian CV Society Angina Classification

- **Class I**
  Ordinary physical activity does not cause angina, such as walking, climbing stairs.

- **Class II**
  Slight limitation of ordinary activity. Angina on walking or climbing stairs rapidly, walking uphill, walking or stair climbing after meals.

- **Class III**
  Marked limitations of ordinary physical activity. Angina on walking one to two blocks on the level and climbing one flight of stairs.

- **Class IV**
  Inability to carry on any physical activity without discomfort—anginal symptoms at rest.
“The Guidelines”

Circulation. 2007;116:2762-2772
<table>
<thead>
<tr>
<th>CLASS</th>
<th>INDICATION FOR CARDIAC STRESS IMAGING</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Who Are Able to Exercise</td>
</tr>
<tr>
<td></td>
<td>FOR DIAGNOSIS</td>
</tr>
<tr>
<td></td>
<td></td>
</tr>
<tr>
<td>I</td>
<td>1. Exercise myocardial perfusion imaging or exercise echo - intermediate pretest probability of CAD with baseline ECG abnormalities</td>
</tr>
<tr>
<td></td>
<td>a. Preexcitation (Wolff-Parkinson-White) syndrome</td>
</tr>
<tr>
<td></td>
<td>b. &gt;1 mm of ST-segment depression at rest</td>
</tr>
<tr>
<td></td>
<td>2. Exercise myocardial perfusion imaging or exercise echo - prior revascularization (either PCI or CABG)</td>
</tr>
<tr>
<td></td>
<td>3. Adenosine or dipyridamole myocardial perfusion imaging - intermediate pretest probability of CAD &amp; baseline ECG abnormalities:</td>
</tr>
<tr>
<td></td>
<td>a. Electronically paced ventricular rhythm</td>
</tr>
<tr>
<td></td>
<td>b. Left bundle branch block</td>
</tr>
</tbody>
</table>

LEVEL OF EVIDENCE

B

C
ACC/AHA Guideline Criteria for Noninvasive Risk Stratification

### High Risk (>3% Annual Mortality Rate)

1. Severe resting left ventricular dysfunction (LVEF < 0.35)
2. High-risk treadmill score (score ≤ −11)
3. Severe exercise left ventricular dysfunction (exercise LVEF < 0.35)
4. Stress-induced large perfusion defect (particularly if anterior)
5. Stress-induced multiple perfusion defects of moderate size
6. Large, fixed perfusion defect with LV dilation or increased lung uptake
7. Stress-induced moderate perfusion defect with LV dilation or increased lung uptake
8. RWMA (involving more than two segments) developing at low dose of dobutamine or at low heart rate (<120 beats/min)
9. Stress echocardiographic evidence of extensive ischemia
ACC/AHA Guideline Criteria for Noninvasive Risk Stratification

- **Intermediate Risk** (1-3% annual mortality rate)
  1. Mild or moderate LV dysfunction (LVEF = 0.35-0.49)
  2. Intermediate-risk treadmill score (−11 < score < 5)
  3. Stress-induced moderate perfusion defect without LV dilation or increased lung intake
  4. Limited stress echocardiographic ischemia with a wall motion abnormality only at higher doses of dobutamine involving two segments or less

- **Low Risk** (<1% Annual Mortality Rate)
  1. Low-risk treadmill score (score ≥ 5)
  2. Normal or small myocardial perfusion defect at rest or with stress*
  3. Normal stress echocardiographic wall motion or no change of limited resting wall motion abnormalities during stress
## Coronary Angiography for Risk Stratification in Chronic Stable Angina

<table>
<thead>
<tr>
<th>CLASS</th>
<th>INDICATION</th>
<th>LEVEL OF EVIDENCE</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>1. Canadian Cardiovascular Society [CCS] Classes III and IV chronic stable angina despite medical therapy</td>
<td>B</td>
</tr>
<tr>
<td></td>
<td>2. Patients with high-risk criteria on noninvasive testing regardless of anginal severity</td>
<td>B</td>
</tr>
<tr>
<td></td>
<td>3. Patients with angina who have survived sudden cardiac death or serious ventricular arrhythmia</td>
<td>B</td>
</tr>
<tr>
<td></td>
<td>4. Patients with angina and symptoms and signs of CHF</td>
<td>C</td>
</tr>
<tr>
<td></td>
<td>5. Patients with clinical characteristics that indicate a high likelihood of severe CAD</td>
<td>C</td>
</tr>
</tbody>
</table>
# Coronary Angiography for Risk Stratification in Patients with Chronic Stable Angina

<table>
<thead>
<tr>
<th>Ila</th>
<th>1. Patients with significant LV dysfunction (ejection fraction &gt; 0.45), CCS Class I or II angina, and demonstrable ischemia but no high-risk criteria on noninvasive testing</th>
<th>C</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>2. Patients with inadequate prognostic information after noninvasive testing</td>
<td>C</td>
</tr>
<tr>
<td>IIb</td>
<td></td>
<td>C</td>
</tr>
<tr>
<td>III</td>
<td>1. Patients with CCS Class I or II angina who respond to medical therapy and who have no evidence of ischemia on noninvasive testing</td>
<td>C</td>
</tr>
<tr>
<td></td>
<td>2. Patients who prefer to avoid revascularization</td>
<td>C</td>
</tr>
</tbody>
</table>
Tight blocks have usually more healed plaque ruptures

Circ 2001;103:9364
Na⁺/Ca²⁺ overload and ischemia

Myocardial ischemia

↑ Late Na⁺ current

Na⁺ overload

Ca²⁺ overload

↑ Diastolic wall tension (stiffness)

↑ O₂ demand

Intramural small vessel compression (↓ O₂ supply)

Chronic Stable Angina
by the Guidelines

- **Environmental**
  - Stop smoking-1B
  - Physical activity-1B
  - Weight control-1B
  - Chelation therapy-3C
  - Influenza vaccination-1B

- **Vascular/Tissue**
  - Blood pressure-1B
  - RAAS blockade-1A
  - Aldosterone blockade-1A/B

- **Metabolics**
  - Lipids-1B
  - Triglycerides-1B
  - Diabetes-1B
  - Antiplatelets-1A/B

Circulation. 2007;116:2762-2772
Pharmacotherapy for Chronic Stable Angina (class I)

1. **Aspirin** in the absence of contraindications – LOE = A

2. **Beta-blockers** as initial therapy in the absence of contraindications in patients with prior myocardial infarction or without prior myocardial infarction – LOE = A, B

3. **ACE inhibitor** in all patients with CAD who also have diabetes and/or LV systolic dysfunction – LOE = A

4. **LDL-lowering therapy** in patients with documented or suspected CAD and LDL cholesterol > 130 mg/dl, with a target LDL of < 100 mg/dl – LOE = A

5. **Sublingual nitroglycerin** or nitroglycerin spray for the immediate relief of angina – LOE = B

6. **Calcium antagonists** † or long-acting nitrates as initial therapy for reduction of symptoms when beta blockers are contraindicated – LOE = B
Pharmacotherapy for Chronic Stable Angina (class IIa)

1. **Clopidogrel** when aspirin is absolutely contraindicated

2. **Long-acting non-dihydropyridine** calcium antagonists † instead of beta blockers as initial therapy B

3. In patients with documented or suspected CAD and LDL cholesterol 100–129 mg/dl, several therapeutic options are available: B
   - a. **Lifestyle** and/or drug therapies to lower LDL to <100 mg/dl
   - b. **Weight reduction and increased physical activity** in persons with the metabolic syndrome
   - c. Institution of treatment of other lipid or non-lipid risk factors; consider use of **nicotinic acid or fibric acid** for elevated triglycerides or low HDL cholesterol

4. **ACE inhibitor** in patients with CAD or other vascular disease
Pharmacotherapy for Chronic Stable Angina (IIb or III)

- **IIb** (weak supportive evidence)
  - Low-intensity anticoagulation with warfarin in addition to aspirin
  - May be useful in aneurysmal CAD

- **III** (not indicated)
  1. Dipyridamole
  2. Chelation therapy
Vascular / Tissue Modification

- **Blood pressure-1B**
  - Lifestyle (low salt, weight control and exercise)
  - Moderate etoh & vegetables
  - BP <140 / 90 by JNC VIII-HCTZ
  - Diabetes & CKDx 130 / 80
  - HT with CAD—BB &/or ACEI

- **RAAS blockade-1A**
  - EF<40 ACEI
  - Mild/moderate risk & normal EF-2B

- **Aldosterone blockade-1A/B**
  - After MI (normal kid function & K+)
  - Patients already on BB & ACEI
  - EF<40 with HF or diabetes
Myocardial ischemia: Sites of action of anti-ischemic medication

Development of ischemia
- ↑ O₂ Demand
- Heart rate
- Blood pressure
- Preload
- Contractility
- ↓ O₂ Supply

Consequences of ischemia
- Ca²⁺ overload
- Electrical instability
- Myocardial dysfunction (↓ systolic function, ↑ diastolic stiffness)

Traditional anti-ischemic medications:
- β-blockers
- Nitrates
- Ca²⁺ blockers

Ranolazine

Courtesy of PH Stone, MD and BR Chaitman, MD. 2006.
Diabetic Cardiomyopathy: Dual Benefit of Ranolazine

Ranolazine

Late INa

High Glucose

Hypothesis

* Hypothesis: Glucose increases pCaMKII, which increases late INa

* Nishio et al JMCC 52 (2012) 1103–1111
* Luo and Anderson et al JCI, 2013
* Mourouzis et al Unpublished data 2013
TERISA Trial

- Evaluation of Ranolazine in Patients with Type 2 Diabetes Mellitus and Chronic Stable Angina. Results from the TERISA randomized clinical trial.

### Baseline Characteristics by Study Group

<table>
<thead>
<tr>
<th></th>
<th>Ranolazine n=462</th>
<th>Placebo n=465</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Antianginal medications</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>on 1 (%)</td>
<td>56.1</td>
<td>55.7</td>
</tr>
<tr>
<td>on 2 (%)</td>
<td>43.9</td>
<td>44.3</td>
</tr>
<tr>
<td><strong>Beta blockers (%)</strong></td>
<td>90.5</td>
<td>89.9</td>
</tr>
<tr>
<td><strong>Calcium channel blockers (%)</strong></td>
<td>26.8</td>
<td>30.8</td>
</tr>
<tr>
<td><strong>Long acting nitrates (%)</strong></td>
<td>34.8</td>
<td>32.5</td>
</tr>
<tr>
<td><strong>Statins (%)</strong></td>
<td>82.5</td>
<td>82.4</td>
</tr>
<tr>
<td><strong>Antiplatelet agents (%)</strong></td>
<td>89.8</td>
<td>86.5</td>
</tr>
<tr>
<td><strong>ACE-I/ARBs (%)</strong></td>
<td>88.1</td>
<td>87.5</td>
</tr>
<tr>
<td><strong>Diary compliance - median % (IQR)</strong></td>
<td>98 (95-98)</td>
<td>98 (95-98)</td>
</tr>
</tbody>
</table>
# Primary Endpoint

<table>
<thead>
<tr>
<th></th>
<th>Ranolazine (n=462)</th>
<th>Placebo (n=465)</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Least squares mean (95% CI)</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Angina frequency, baseline (#/wk)</strong></td>
<td>6.6 (6.3-7.0)</td>
<td>6.8 (6.4-7.2)</td>
<td>0.54</td>
</tr>
<tr>
<td><strong>Angina frequency, on treatment (#/wk)</strong></td>
<td>3.8 (3.6-4.1)</td>
<td>4.3 (4.0-4.5)</td>
<td>0.008</td>
</tr>
</tbody>
</table>
Weekly Angina Frequency by Study Group

Run In Phase

Placebo

Ranolazine

Weekly Angina Frequency

Study Week

Treatment Phase

p=0.008

Weekly Angina Frequency

Placebo

Ranolazine
# Key Secondary Endpoint

<table>
<thead>
<tr>
<th>SL NTG doses, baseline – (#/wk)</th>
<th>Ranolazine n=462</th>
<th>Placebo n=465</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Least squares mean (95% CI)</td>
<td>4.1 (3.7-4.6)</td>
<td>4.5 (4.1-5.0)</td>
<td>0.27</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>SL NTG doses, on treatment (#/wk)</th>
<th>Ranolazine n=462</th>
<th>Placebo n=465</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Least squares mean (95% CI)</td>
<td>1.7 (1.6-1.9)</td>
<td>2.1 (1.9-2.3)</td>
<td>0.003</td>
</tr>
</tbody>
</table>
Exploratory Analysis – HbA1c

- **HbA1c >6**: Ranolazine better, Incidence Density Ratio (IDR) = 0.7
- **HbA1c ≤ 6**: Placebo better, IDR = 1.2
- **HbA1c >6.5**: Ranolazine better, IDR = 0.8
- **HbA1c ≤ 6.5**: Placebo better, IDR = 1.1
- **HbA1c >7**: Ranolazine better, IDR = 0.9
- **HbA1c ≤ 7**: Placebo better, IDR = 1
- **HbA1c >7.5**: Ranolazine better, IDR = 0.9
- **HbA1c ≤ 7.5**: Placebo better, IDR = 1
- **HbA1c >8**: Ranolazine better, IDR = 0.8
- **HbA1c ≤ 8**: Placebo better, IDR = 1

Significance levels:
- **p for interaction**: 0.046 (HbA1c >6), 0.047 (HbA1c ≤ 6), 0.022 (HbA1c >7), 0.041 (HbA1c >7.5), 0.038 (HbA1c >8).
## Safety and Tolerability

<table>
<thead>
<tr>
<th>Serious Adverse Events</th>
<th>Ranolazine n=470</th>
<th>Placebo n=474</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Serious adverse event</td>
<td>16 (3.4)</td>
<td>20 (4.2)</td>
<td>0.51</td>
</tr>
<tr>
<td>Death</td>
<td>3 (0.6)</td>
<td>2 (0.4)</td>
<td>0.69</td>
</tr>
<tr>
<td>Nonfatal myocardial infarction</td>
<td>1 (0.2)</td>
<td>3 (0.6)</td>
<td>0.62</td>
</tr>
<tr>
<td>Stroke/transient ischemic attack</td>
<td>1 (0.2)</td>
<td>4 (0.8)</td>
<td>0.37</td>
</tr>
<tr>
<td>Unstable angina or coronary revascularization</td>
<td>6 (1.3)</td>
<td>7 (1.5)</td>
<td>0.79</td>
</tr>
</tbody>
</table>

### Notable non-serious adverse events

<table>
<thead>
<tr>
<th>Adverse Event</th>
<th>Ranolazine n=470</th>
<th>Placebo n=474</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dizziness</td>
<td>17 (3.6)</td>
<td>6 (1.3)</td>
<td>0.019</td>
</tr>
<tr>
<td>Nausea</td>
<td>17 (3.6)</td>
<td>2 (0.4)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Headache</td>
<td>7 (1.5)</td>
<td>9 (1.9)</td>
<td>0.63</td>
</tr>
<tr>
<td>Constipation</td>
<td>8 (1.7)</td>
<td>2 (0.4)</td>
<td>0.063</td>
</tr>
<tr>
<td>Hypoglycemia</td>
<td>3 (0.6)</td>
<td>0 (0.0)</td>
<td>0.12</td>
</tr>
</tbody>
</table>

### Any Adverse Event

<table>
<thead>
<tr>
<th></th>
<th>Ranolazine n=470</th>
<th>Placebo n=474</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Any Adverse Event</td>
<td>126 (26.8)</td>
<td>105 (22.2)</td>
<td>0.096</td>
</tr>
</tbody>
</table>
Does moderate-severe ischemia need blood?

Optimal Medical Therapy Vs. Revascularization Vs. Both
Stable CAD: PCI vs Conservative Medical Management

Meta-analysis of 11 randomized trials; N = 2,950

<table>
<thead>
<tr>
<th>Event</th>
<th>Favors PCI</th>
<th>Favors Medical Management</th>
</tr>
</thead>
<tbody>
<tr>
<td>Death</td>
<td>0.68</td>
<td>P</td>
</tr>
<tr>
<td>Cardiac death or MI</td>
<td>0.28</td>
<td>0.12</td>
</tr>
<tr>
<td>Nonfatal MI</td>
<td>0.12</td>
<td>0.82</td>
</tr>
<tr>
<td>CABG</td>
<td>0.82</td>
<td>0.34</td>
</tr>
<tr>
<td>PCI</td>
<td>0.34</td>
<td>0.82</td>
</tr>
</tbody>
</table>

Risk ratio (95% CI)

Optimal Medical Therapy with or without PCI for Stable Coronary Disease: COURAGE

- Stable coronary artery disease with stenosis of at least 70% in at least one proximal epicardial coronary artery and objective evidence of myocardial ischemia
  - N=1149 PCI + optimal medical therapy
  - N=1138 optimal medical therapy alone
  - F/U 2.5 to 7.0 years (median, 4.6)

Primary outcome (NS)
Death from any cause and nonfatal MI
19.0% - PCI group
18.5% - Medical only
○ Hazard ratio 1.05; 95% confidence interval [CI], 0.87 to 1.27; P = 0.62)

33% crossed over to PCI

Levels at end of study
LDL-70
HDL-42
TRG-125
BP 122/70

N Engl J Med March 27, 2007;356:000
Survival Free of Death from Any Cause and Myocardial Infarction

Optimal Medical Therapy (OMT)

Hazard ratio: 1.05
95% CI (0.87-1.27)
P = 0.62

Number at Risk

<table>
<thead>
<tr>
<th></th>
<th>Years</th>
<th>Medical Therapy</th>
<th>PCI</th>
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</thead>
<tbody>
<tr>
<td>0</td>
<td></td>
<td>1138</td>
<td>1149</td>
</tr>
<tr>
<td>1</td>
<td></td>
<td>1017</td>
<td>1013</td>
</tr>
<tr>
<td>2</td>
<td></td>
<td>959</td>
<td>952</td>
</tr>
<tr>
<td>3</td>
<td></td>
<td>834</td>
<td>833</td>
</tr>
<tr>
<td>4</td>
<td></td>
<td>638</td>
<td>637</td>
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<tr>
<td>5</td>
<td></td>
<td>408</td>
<td>417</td>
</tr>
<tr>
<td>6</td>
<td></td>
<td>192</td>
<td>200</td>
</tr>
<tr>
<td>7</td>
<td></td>
<td>30</td>
<td>35</td>
</tr>
</tbody>
</table>
Fractional Flow Reserve–Guided PCI versus Medical Therapy in Stable Coronary Disease

Bernard De Bruyne, M.D., Ph.D., Nico H.J. Pijls, M.D., Ph.D., Bindu Kalesan, M.P.H., Emanuele Barbato, M.D., Ph.D., Pim A.L. Tonino, M.D., Ph.D., Zsolt Piroth, M.D., Nikola Jagic, M.D., Sven Mobius-Winkler, M.D., Gilles Rioufol, M.D., Ph.D., Nils Witt, M.D., Ph.D., Petr Kala, M.D., Philip MacCarthy, M.D., Thomas Engström, M.D., Keith G. Oldroyd, M.D., Kreton Mavromatis, M.D., Ganesh Manoharan, M.D., Peter Verlee, M.D., Ole Frobert, M.D., Nick Curzen, B.M., Ph.D., Jane B. Johnson, R.N., M.Sc., Peter Jüni, M.D., and William F. Fearon, M.D., for the FAME 2 Trial Investigators*
Primary End Point

Composite of

- all cause death
- myocardial infarction
- unplanned hospitalization with urgent revascularization
FAME 2: FFR-Guided PCI versus Medical Therapy in Stable CAD

Flow Chart

Stable CAD patients scheduled for 1, 2 or 3 vessel DES-PCI
N = 1220

FFR in all target lesions

Randomized Trial

At least 1 stenosis with FFR ≤ 0.80 (n=888)

Randomization 1:1

PCI + MT

MT

73%

Registry

When all FFR > 0.80 (n=332)

MT

27%

50% randomly assigned to FU

Follow-up after 1, 6 months, 1, 2, 3, 4, and 5 years
**Primary Outcomes**

**Cumulative incidence (%)**

- **PCI+MT vs. MT:** HR 0.32 (0.19-0.53); p<0.001
- **PCI+MT vs. Registry:** HR 1.29 (0.49-3.39); p=0.61
- **MT vs. Registry:** HR 4.32 (1.75-10.7); p<0.001

**No. at risk**

<table>
<thead>
<tr>
<th></th>
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<th>3</th>
<th>4</th>
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<th>9</th>
<th>10</th>
<th>11</th>
<th>12</th>
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<tbody>
<tr>
<td><strong>MT</strong></td>
<td>441</td>
<td>414</td>
<td>370</td>
<td>322</td>
<td>283</td>
<td>253</td>
<td>220</td>
<td>192</td>
<td>162</td>
<td>127</td>
<td>100</td>
<td>70</td>
<td>37</td>
</tr>
<tr>
<td><strong>PCI+MT</strong></td>
<td>447</td>
<td>414</td>
<td>388</td>
<td>351</td>
<td>308</td>
<td>277</td>
<td>243</td>
<td>212</td>
<td>175</td>
<td>155</td>
<td>117</td>
<td>92</td>
<td>53</td>
</tr>
<tr>
<td><strong>Registry</strong></td>
<td>166</td>
<td>156</td>
<td>145</td>
<td>133</td>
<td>117</td>
<td>106</td>
<td>93</td>
<td>74</td>
<td>64</td>
<td>52</td>
<td>41</td>
<td>25</td>
<td>13</td>
</tr>
</tbody>
</table>
PCI+MT vs. MT: HR 0.13 (0.06-0.30); p<0.001
PCI+MT vs. Registry: HR 0.63 (0.19-2.03); p=0.43
MT vs. Registry: HR 4.65 (1.72-12.62); p=0.009
Figure Legend:

Spectrum of IHD
Guidelines relevant to the spectrum of IHD are in parentheses. CABG indicates coronary artery bypass graft; CV, cardiovascular; ECG, electrocardiogram; IHD, ischemic heart disease; PCI, percutaneous coronary intervention; SCD, sudden cardiac death; SIHD, stable ischemic heart disease; STEMI, ST-elevation myocardial infarction; UA, unstable angina; UA/NSTEMI, unstable angina/non–ST-elevation myocardial infarction; and VA, ventricular arrhythmia.
Morality predictor by risk factor

<table>
<thead>
<tr>
<th>Risk Factor</th>
<th>Score Contribution</th>
<th>Individual's Score</th>
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<td>Comorbidity</td>
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<td>Angina score</td>
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<td>Class III</td>
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<td>Duration of symptoms</td>
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<td>≥6 months</td>
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<tr>
<td>&lt;6 months</td>
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<td>Abnormal ventricular function</td>
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<td>ST depression or T wave inversion on resting electrocardiogram</td>
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Euro Heart Score Sheet to Calculate Risk Score for Patients Presenting With Stable Angina (Derived From 3,779 Patients With Newly Diagnosed SIHD)
Annual Risk of 3 vessel CAD
Based on 5 variables

Figure Legend:
Nomogram Showing the Probability of Severe (3-Vessel or Left Main) Coronary Disease Based on a 5-Point Score. One point is awarded for each of the following variables: male sex, typical angina, history and electrocardiographic evidence of MI, and diabetes mellitus and use of insulin. Each curve shows the probability of severe coronary disease as a function of age.
SYNTAX Score and MACE

Cumulative Incidence of MACE in Patients With 3-Vessel CAD Based on SYNTAX Score at 3-Year Follow-Up in the SYNTAX Trial Treated With Either CABG (Blue) or PCI (Gold)

CABG indicates coronary artery bypass graft; CAD, coronary artery disease; MACE, major adverse cardiovascular event; PCI, percutaneous coronary intervention; and SYNTAX, Synergy between Percutaneous Coronary Intervention with TAXUS and Cardiac Surgery.

Genetics and Environment Trump Intervention

- 4 take home messages
- Family History is paramount
- Control your environment …drugs / surgery are not match for uncontrolled environment
- Vascular / tissue – blood pressure very important…… Minimize wall stress
- Metabolics – nutrients of vascular life…needs clean fuel for healthy endothelium
- Nitric oxide is life

Acta Physiol 2009, 196, 193–222