Sensitivity / Specificity for CAD generally defined as >70% stenosis of at least one vessel.

<table>
<thead>
<tr>
<th>Test</th>
<th>Sensitivity</th>
<th>Specificity</th>
</tr>
</thead>
<tbody>
<tr>
<td>Stress EKG</td>
<td>68%</td>
<td>74%</td>
</tr>
<tr>
<td>Stress Echo</td>
<td>86%</td>
<td>77%</td>
</tr>
<tr>
<td>Nuclear Stress Test</td>
<td>91%</td>
<td>80%</td>
</tr>
<tr>
<td>EBCT</td>
<td>93%</td>
<td>89%</td>
</tr>
<tr>
<td>CCTA</td>
<td>93%</td>
<td>96%</td>
</tr>
<tr>
<td>Cardiac Cath</td>
<td>99%</td>
<td>99%</td>
</tr>
</tbody>
</table>

Sensitivity = ability to detect disease when it is present.
Specificity = ability to correctly exclude disease when it is absent.
Predictive value influenced by pre-test prevalence or probability.
Case 1

• 61 year-old man
  – Treated hypertension and hyperlipidemia: well-controlled
  – ECG: left bundle branch block (LBBB)
  – Screening calcium scan (EBCT) done 2009
    • Total score 547 (>75 percentile for age)
    • LM 25, LAD 155, RCA 264, Cx 103
  – Exercise testing
    • Exercise duration 12.5 METS
    • Nuclear stress: “fixed septal defect due to bundle branch block”
    • Echo stress: “normal ejection fraction and wall motion”
  – This year he presented to an ER with chest pain; he ruled out for MI but was taken to cath due to his high calcium score.
    • Cath: 30% proximal RCA; luminal irregularities in the LAD and LCA.
Newly Acquired LBBB
in Community Population
Framingham Study Population

- Eighteen years of observation; 55 new LBBBs in 5,209 people
- Average age of onset 62
- Most LBBBs occurred with HTN, CAD, and cardiac enlargement
- 48% developed CAD or CHF at or after onset
- Within 10 years 50% died from cardiovascular disease
- LBBB contributed independently to increased risk of cardiovascular death

Annals of Internal Medicine 1979; 90(3):303

7,392 Men From a General Population
BBB at Baseline and 28-Year Follow-up
Coronary Death and Sudden Death

HR LBBB all cause mortality 1.84
European Heart Journal 2005; 26:2300-2306
LBBB in Patients with Chronic CAD

- 15,609 with CAD had coronary angiogram and ventriculography
- 522 had BBB
- BBB did not correlate with location of coronary artery stenosis or LV wall abnormality
- 4.9 year follow-up; 2,386 died
- Those with LBBB had a 5X mortality risk
- Those with RBBB had 2X mortality risk
- Cox regression showed LBBB, but not RBBB, is a strong predictor of mortality in this population

J Am Coll Cardiol 1987;10:73-80

Exercise Induced LBBB

- 17,277 exercise tests
- Follow-up 3.7 years
- 70 Exercise induced LBBB
- 25 patients with cardiac events
  - 17 with exercise induced LBBB
  - 8 in control group
- 7 deaths
  - 5 with exercise induced LBBB
  - 2 in control group

J Cardiovasc Electrophysiol 2009; 20:781
Left Anterior Hemiblock and Mortality

Case 1

- 61 year-old man
  - Treated hypertension and hyperlipidemia: well-controlled
  - ECG: left bundle branch block (LBBB)
  - Screening calcium scan (EBCT) done 2009
    - Total score 547 (>75 percentile for age)
    - LM 25, LAD 155, RCA 264, Cx 103
  - Exercise testing
    - Exercise duration 12.5 METS
    - Nuclear stress: “fixed septal defect due to bundle branch block”
    - Echo stress: “normal ejection fraction and wall motion”
  - This year he presented to an ER with chest pain; he ruled out for MI but was taken to cath due to his high calcium score.
    - Cath: 30% proximal RCA; luminal irregularities in the LAD and LCA.
Long-Term Prognosis Associated with Coronary Calcification: Outcomes

Cumulative Survival by Coronary Calcium Score

Cumulative Survival by the Coronary Calcium Extent in the Number of Vascular Territories
Case 1

- 61 year-old man
  - Treated hypertension and hyperlipidemia: well-controlled
  - ECG: left bundle branch block (LBBB)
  - Screening calcium scan (EBCT) done 2009
    - Total score 547 (>75 percentile for age)
    - LM 25, LAD 155, RCA 264, Cx 103
- Exercise testing
  - Exercise duration 12.5 METS
  - Nuclear stress: “fixed septal defect due to bundle branch block”
  - Stress echo: “normal ejection fraction and wall motion”
- This year he presented to an ER with chest pain; he ruled out for MI but was taken to cath due to his high calcium score.
  - Cath: 30% proximal RCA; luminal irregularities in the LAD and LCA.

Duke Treadmill Score

- (Bruce exercise minutes) \text{minus} (5 \times \text{maximal ST segment deviation in mm}) \text{minus} (4 \times \text{exercise angina})
  where
  - 0 = none; 1 = non-limiting; 2 = limiting

- Low risk – score > +5
  - (97% five-year survival)
- Moderate risk – score from -10 to +4
  - (31% have 3-vessel or LM disease with 90% five year survival)
- High Risk – score < -11
  - (74% have 3-vessel or LM disease with 65% five year survival)
For example, a 60-year-old man with a 3-MET capacity has 40% of the age-expected exercise capacity for sedentary men and 30% of that for active men.*

A useful equation to estimate expected METs for an active person:

\[ 18 - (age \times 0.15) = \text{Expected METs} \]

Case 1 expected:

\[ 18 - (61 \times 0.15) = 8.85 \text{ METs} \]

Case 1 achieved = 12.5 METs

* This statement was approved by the American Heart Association Science Advisory and Coordinating Committee in June 2001.

Exercise Capacity and All-Cause Mortality

Chronotropic Insufficiency

Unable to achieve 85% predicted max HR off beta blockers

Adjusted relative risk of cardiac event 2.2

Heart-Rate Recovery
One Minute after Peak Exercise.

2428 patients without known heart disease

An abnormal value for the recovery of heart rate defined as a reduction of ≤12 beats per minute (bpm) from the heart rate at peak exercise

Circles represent the relative risk of death within six years for each of the quintiles as compared with the fifth quintile which had the lowest risk of death.

Red lines represent the 95 percent confidence interval.
Mortality Risk in Normotensive Individuals with Hypertensive Response to Exercise

6578 asymptomatic individuals (74 percent without hypertension at baseline) who underwent submaximal Bruce treadmill tests and were followed for 20 years.

Among individuals with baseline BP <140/90 mmHg, Bruce stage 2 BP >180/90 compared to ≤180/90 mmHg was associated with a significant increase in risk of cardiovascular death after adjustment for rest BP and other risk factors (adjusted hazard ratio for systolic 1.96, 95% CI 1.40-2.74 and for diastolic 1.48, 95% CI 1.06-2.06).

Weiss S A et al. Circulation 2010;121:2109-2116

Case 1

- 61 year-old man
  - Treated hypertension and hyperlipidemia: well-controlled
  - ECG: left bundle branch block (LBBB)
  - Screening calcium scan (EBCT) done 2009
    - Total score 547 (>75 percentile for age)
    - LM 25, LAD 155, RCA 264, Cx 103
  - Exercise testing
    - Exercise duration 12.5 METS
    - Nuclear stress: “fixed septal defect due to bundle branch block”
    - Echo stress: “normal ejection fraction and wall motion”
  - This year he presented to an ER with chest pain; he ruled out for MI but was taken to cath due to his high calcium score.
    - Cath: 30% proximal RCA; luminal irregularities in the LAD and LCA.
Adjusted for CAD risk factors, the presence of any nonobstructive plaque was associated with higher mortality with the highest risk among those exhibiting nonobstructive CAD in 3 vessels.

Higher mortality for nonobstructive CAD was observed even among patients with low 10-year Framingham risk (3.4%, p=0.0001) as well as those with no traditional, medically treatable CAD risk factors, including diabetes mellitus, hypertension, and dyslipidemia (6.7%, p=0.0001).

Figure 4: Prognosis of Nonobstructive CAD in Low-Risk Patients
Unadjusted significant 3-year Kaplan-Meier survival by the absence of coronary artery disease (CAD) versus presence of nonobstructive CAD in patients with a 10-year Framingham estimated risk <10%.
Case 1: Relative Mortality Risk?

61 year-old man
Treated hypertension and hyperlipidemia: well-controlled
ECG: left bundle branch block (LBBB)
Screening calcium scan (EBCT) done 2009
Total score 547 (>75 percentile for age)
LM 25, LAD 155, RCA 264, Cx 103
Exercise testing
Exercise duration 12.5 METS
Nuclear stress: “fixed septal defect due to bundle branch block”
Echo stress: “normal ejection fraction and wall motion”
This year he presented to an ER with chest pain; he ruled out for MI but was taken to cath due to his high calcium score.
Cath: 30% proximal RCA; luminal irregularities in the LAD and LCA.

Relative Mortality
• 100 %
• 150 %
• 200 %
• 250 %
• 300 %
• 300 %+

Case 2

47-year-old male dentist and marathon runner with history of childhood asthma and atrial fibrillation since 1999. Drinks alcohol socially once or twice weekly

Atrial Fibrillation History:
March 1998: first episode AF, spontaneously converted
September 2007: initiated warfarin and atenolol followed by cardioversion
February 2009: atrial fibrillation walking up stairs; reinitiated warfarin and atenolol followed by cardioversion
March 2009: pulmonary vein isolation: six-week follow up with only one brief episode of AF, nothing sustained. Six and twelve-month follow up: No atrial fibrillation
Projected Number of Adults With AF in the US: 1995 to 2050

Time since *First* Pulmonary Vein Isolation (PVI)

Long-Term Results of Catheter Ablation in Paroxysmal Atrial Fibrillation
Lessons From a 5-Year Follow-Up
Stroke in AF

• Stroke in AF is often severe and results in long-term disability or death. Approximately every fifth stroke is due to AF; furthermore, undiagnosed ‘silent AF’ is a likely cause of some ‘cryptogenic’ strokes. Paroxysmal AF carries the same stroke risk as permanent or persistent AF.

• Cognitive dysfunction, including vascular dementia, may be related to AF. Small observational studies suggest that asymptomatic embolic events may contribute to cognitive dysfunction in AF patients in the absence of an overt stroke.
Case 2: Relative Mortality Risk?

47-year-old male dentist and marathon runner with history of childhood asthma and atrial fibrillation since 1999. Drinks alcohol socially once or twice weekly

Atrial Fibrillation History:
- March 1998: first episode of AF, spontaneously converted
- September 2007: initiated warfarin and atenolol followed by cardioversion
- February 2009: atrial fibrillation walking up stairs; reinitiated warfarin and atenolol followed by cardioversion
- March 2009: pulmonary vein isolation: six-week follow up with only one brief episode of AF, nothing sustained. Six and twelve-month follow up: No atrial fibrillation

Relative Mortality
- 100%
- 150%
- 200%
- 250%
- 300%
- 300%+

Case 3

75-year-old male ex-smoker with exemplary cardiovascular risk factors evaluated in 2006 (age 69) for dyspnea and a positive stress test

2006 SPECT: Symptom-limited stress ECG showed 3 mm downsloping ST depression at 8.2 METs.

Resting EF 55%; stress EF 60%; TID ratio 1.2; 4% reversible defect RCA distribution

CT Angiogram: Ca++ score 598 (82% tile). Prox LAD 25-49%; 1st diag. 30% ostial; prox. RCA 1-24%; mid-RCA 25-49%, and ostial PDA 1-24%

2010 SPECT: Symptom-limited stress to 7.7 METs with 3mm ST depression. New 12% reversible defect RCA distribution. EF 58%

2010 CTA: calcium score 770 (87th percentile). LAD score 495. RCA 192
Myocardial Perfusion Imaging (MPI)
SPECT, Radionuclide Scan, Nuclear Scanning

Indications for Pharmacologic Stress
- Unable to exercise
  - Aortic stenosis
  - LBBB
  - Pacemaker
  - Recent MI
- Severe HTN

Vasodilators (“stealers”)
- Adenosine and analogs
- Dipyridamole
- Inotrope/Chronotrope
- Dobutamine

Imaging agents
- Thallium-201 (K+)
- Tc-99m sestamibi (Ca++)
  (e.g., Cardiolite)
- Technitium tetrofosmin
  (e.g., Myoview)
- Technitium teboroxime
  (e.g., Cardiotec)
Transient Ischemic Dilation Ratio (TID)
Average ventricular size after stress compared with rest

JACC 2003; 42:1818-1825

TID ratios > 1.22 with exercise or > 1.36 with pharmacologic stress suggest extensive CAD, even in presence of normal MPI.

(71% sensitivity and 95% specificity)


Table 1. Distribution of the Study Population 1 by Quartiles of TID Ratio

<table>
<thead>
<tr>
<th>Subgroup</th>
<th>Number of Patients</th>
<th>Mean ± SD of TID Ratio</th>
<th>Range of TID Ratio (Minimum-Maximum)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1st quartile</td>
<td>365</td>
<td>0.93 ± 0.06</td>
<td>0.80-0.99</td>
</tr>
<tr>
<td>2nd quartile</td>
<td>400</td>
<td>1.02 ± 0.02</td>
<td>1.00-1.07</td>
</tr>
<tr>
<td>3rd quartile</td>
<td>409</td>
<td>1.13 ± 0.04</td>
<td>1.08-1.20</td>
</tr>
<tr>
<td>4th quartile</td>
<td>390</td>
<td>1.33 ± 0.14*</td>
<td>1.21-1.39</td>
</tr>
</tbody>
</table>

* p < 0.001 across the groups.
TID = transient ischemic dilation.

Case 3

75-year-old male ex-smoker with exemplary cardiovascular risk factors evaluated in 2006 (age 69) for dyspnea and a positive stress test

2006 SPECT: Symptom-limited stress ECG showed 3 mm downsloping ST depression at 8.2 METs.

Resting EF 55%; stress EF 60%; TID ratio 1.2; 4% reversible defect RCA distribution

CT Angiogram: Ca++ score 598 (82nd percentile). Prox LAD 25-49%; 1st diag. 30% ostial; prox. RCA 1-24%; mid-RCA 25-49%, and ostial PDA 1-24%

2010 SPECT : Symptom-limited stress to 7.7 METs with 3mm ST depression. New 12% reversible defect RCA distribution. EF 58%

2010 CTA: calcium score 770 (87th percentile). LAD score 495. RCA, 192
Silent Lesions (<50%)

Relationship Between Calcium Score and the Severity of Coronary Artery Stenosis

Slide 35

Newer Anatomic Imaging

Calcium score

Rapid x-ray of the heart allowing for the detection of calcium buildup in the coronaries. Density x brightness=Agaston score

CCTA

3D reconstruction of the heart and blood vessels developed from data acquired by a CT technology that allows for increased spatial and temporal resolution.

Slide 36
CTA Will Replace Diagnostic Invasive Angiography

- Accurate
- Prognostic value

<table>
<thead>
<tr>
<th></th>
<th>Sensitivity</th>
<th>Specificity</th>
<th>Negative Protective Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Per segment</td>
<td>91%</td>
<td>96%</td>
<td>98%</td>
</tr>
<tr>
<td>Per patient</td>
<td>96%</td>
<td>90%</td>
<td>99%</td>
</tr>
</tbody>
</table>

486 suspected acute coronary syndrome patients in ER:
84% discharged home after normal CT
30 days: no events
1 year: no MI

Ann Emerg Med 2009

Case 3 Relative Mortality Risk?

75-year-old male ex-smoker with exemplary cardiovascular risk factors evaluated in 2006 (age 69) for dyspnea and a positive stress test.

2006 SPECT: Symptom-limited stress ECG showed 3 mm downsloping ST depression at 8.2 METs.

Resting EF 55%; stress EF 60%; TID ratio 1.2; 4% reversible defect RCA distribution

CT Angiogram: Ca++ score 598 (82 %tile). Prox LAD 25-49%; 1st diag. 30% ostial; prox. RCA 1-24%; mid-RCA 25-49%, and ostial PDA 1-24%

2010 SPECT : Symptom-limited stress to 7.7 METs with 3mm ST depression. New 12% reversible defect RCA distribution. EF 58%

2010 CTA: calcium score 770 (87th percentile). LAD score 495. RCA, 192

Relative Mortality

- 100 %
- 150 %
- 200 %
- 250 %
- 300 %
- 300 %+
Case 4

62-year-old male investment advisor with history of an "abnormal ECG" since 1988 (age 38) and echocardiographic evidence of an MI in 1996 (age 46). Five feet eight inches (BMI 25) BP 132/75 with an exemplary risk factor profile. Good medical records depict excellent care and good health otherwise.

March 2011 SPECT 8.0 METs (2006: 10 METs):
- ECG: positive 2mm ST depression five minutes into exercise resolving two minutes into recovery
- SPECT: moderate to severe fixed perfusion defect anterior wall, post stress ejection fraction 37% (post stress EF 2006 51%)
- Echocardiogram: LVID 6.0, LA 3.6, Septum 1.0, PW 1.2, resting EF 50%, and mild anteroseptal hypokinesis
- ECG: QS V1-V3; IVCD (QRS 0.14)

June 2012 SPECT (10 METs): ECG and SPECT unchanged except stress EF back to 51%
Thrombus with identification of a) White Cell b) platelets c) Fibrin fibers d) Red Cell

Cholesterol crystals rupture biological membranes and human plaques during acute cardiovascular events—a novel insight into plaque rupture by scanning electron microscopy.

Abele GS et al. Scanning 2006;28:3-10.

Identification of crystal like structure* – potentially cholesterol plaque debris
## Left Ventricular Ejection Fraction (EDV-ESV)/EDV

### Modality
- M-mode echocardiography
- 2-D echocardiography
- 3-D echocardiography
- MRI
- CT
- Nuclear Cardiac Imaging
  - SPECT
  - MUGA (RGV, RNA RNCA ERNA)

### Sources of Error
- Gating and rhythm abnormalities
- Identifying the endocardial border
- Detecting end-systole and end-diastole
- Software algorithm variations
- Geometric assumptions
- Image planes
- Regional wall motion variations
- Acoustic windows
- Anatomic variations

- Modified Quinones (planar)
- Modified Simpson (biplane method of disks)*

*Recommended by the American Society of Echocardiography
*Most reliable non-invasive methods

### Comparing LVEF by Echo, MPI (SPECT), and MRI

52 patients with chronic stable heart failure

- Echo M-Mode cube method $39 \pm 16\%$
- Echo M-Mode Teichholz method $29 \pm 15\%$
- Echo 2-D Simpsons Biplane $31 \pm 10\%$
- Radionuclide Ventriculography $24 \pm 9\%$
- Cardiovascular MR $30 \pm 11\%

Eur Heart J 2000; 21, 1387-1396
Case 4

62-year-old male investment advisor with history of an abnormal ECG since 1988 (age 38) and echocardiographic evidence of an MI in 1996 (age 46). 5’8” 167 lbs (BMI 25) BP 132/75 with an exemplary risk factor profile. Good medical records depict excellent care and good health otherwise.

March 2011 SPECT 8.0 METs (2006: 10 METs):
ECG: positive 2mm ST depression five minutes into exercise resolving two minutes into recovery
SPECT: moderate to severe fixed perfusion defect anterior wall, post stress ejection fraction 37% (post stress EF 2006 51%)
Echocardiogram : LVID 6.0, LA 3.6, Septum 1.0, PW 1.2, resting EF 50%, and mild anteroseptal hypokinesis
ECG: QS V1-V3; IVCD (QRS 0.14)

June 2012 SPECT (10 METs): ECG and SPECT unchanged except stress EF back to 51%
Non-BBB IVCD

10,899 subjects *general* population 30 years QRS > 0.11

<table>
<thead>
<tr>
<th></th>
<th>HR All-Cause Death</th>
<th>HR Cardiac Death</th>
<th>HR Arrhythmic Death</th>
</tr>
</thead>
<tbody>
<tr>
<td>QRS &gt; 0.11</td>
<td>1.48</td>
<td>1.94</td>
<td>2.14</td>
</tr>
<tr>
<td>QRS &gt; 0.11</td>
<td>2.01</td>
<td>2.53</td>
<td>3.11</td>
</tr>
</tbody>
</table>

without BBB Pattern

Case 4: Relative Mortality Risk?

62-year-old male investment advisor with history of an abnormal ECG since 1988 (age 38) and echocardiographic evidence of an MI in 1996 (age 46).

5’8” 167 lbs (BMI 25) BP 132/75 with an exemplary risk factor profile. Good medical records depict excellent care and good health otherwise.

March 2011 SPECT 8.0 METs (2006: 10 METs):

- ECG: positive 2mm ST depression five minutes into exercise resolving two minutes into recovery
- SPECT: moderate to severe fixed perfusion defect anterior wall, post stress ejection fraction 37% (post stress EF 2006 51%)
- Echocardiogram: LVID 6.0, LA 3.6, Septum 1.0, PW 1.2, resting EF 50%, and mild anteroseptal hypokinesis
- ECG: QS V1-V3; IVCD (QRS 0.14)

June 2012 SPECT (10 METs): ECG and SPECT unchanged except stress EF back to 51%

Relative Mortality

- 100 %
- 150 %
- 200 %
- 250 %
- 300 %
- 300 %+
Case 5

64-year-old physically active dentist. Due to palpitations he had an echocardiogram in December 2004 that revealed a bicuspid aortic valve, aortic root 3.8 cm, moderate LVH and mild AI.

He went to different cardiologist in March 2006. Again an echocardiogram showed a bicuspid aortic valve, an aortic root of 4.4 cm, mild AI, and an EF of 55-60%.

In March 2008 his echo showed his aortic root at 4.2 cm, his left atrial dimension of 3.5 cm, a LVID of 6.1, an EF of 57%, and moderate AI. A CT of the chest to r/o a thoracic aneurysm showed the aortic root at 4.7.

Bicuspid Aortic Valve

• Male > female (2:1)
• Usually picked up between age 40 and 60
• Associated with congenital aortic and proximal coronary artery problems (Marfan’s; Ehlers Danlos)
• About half are associated with widened and expanding aortic root and ascending aorta due to (cystic medial degeneration).
• Increased risk of aortic aneurysm and dissection (5-9 x)
• Nearly all require surgery during lifetime
Bicuspid Aortic Valve and Cystic Medial Degeneration

Retrospective Look at Aortic Valve Replacements

Nearly all bicuspid valves require surgery during lifetime

Aortic valve replacements
• 7% replaced before age 50 with 2/3 bicuspid
• 40% between age 50 and 70 with 2/3 bicuspid
• 50%+ after age 70 with 40% bicuspid valves

• Risk rises with aortic insufficiency, aortic stenosis, and enlarged aortic root, especially > 45mm.
Case 5: Relative Mortality Risk

64-year-old physically active dentist. Due to palpitations he had an echocardiogram in December 2004 that revealed a bicuspid aortic valve, aortic root 3.8 cm, moderate LVH and mild AI.

He went to different cardiologist in March 2006. Again an echocardiogram showed a bicuspid aortic valve, an aortic root of 4.4 cm, mild AI, and an EF of 55-60%.

In March 2008 his echo showed his aortic root at 4.2 cm, his left atrial dimension of 3.5 cm, a LVID of 6.1, an EF of 57%, and moderate AI. A CT of the chest to r/o a thoracic aneurysm showed the aortic root at 4.7 cm.

Relative Mortality
- 100 %
- 150 %
- 200 %
- 250 %
- 300 %
- 300 %+

Case 6

75-year-old retired school teacher with controlled hypertension. As part of the physical exam she had an echocardiogram that led to a chest CT.

Chest CT December 2009
Aortic root 4.1 cm
Ascending aorta is prominent measuring 3.9 cm in the mid-ascending segment.
Proximal Aortic Arch 3.9 cm
Descending thoracic aorta 2.7 cm
Same retired 75-year-old school teacher two years later:

Chest CT December 2011:
Study described as “unchanged” from December 2009
Aortic root 4.2 cm (was 4.1 cm 2009)
Ascending aorta 4.1 cm in the mid-ascending segment (was 3.9 cm in 2009)
Proximal Aortic Arch 4.2 cm (was 3.9 cm in 2009)
Descending thoracic aorta 3.2 cm (was 2.7 cm in 2009)
No evidence of dissection.

Five-year Risk of Rupture
Thoracic Aneurysm

The 5-year risk of rupture as a function of aneurysm size at recognition was:

- 0% for aneurysms less than 4 cm in diameter
- 16% (95% CI, 4%-28%) for those 4 to 5.9 cm, and
- 31% (95% CI, 5%-56%) for aneurysms 6 cm or more.

JAMA 1998 280;22:1926
Normal Diameter and Upper Limit of Ascending and Descending Aorta Related to Age

Our 75-year-old
2009 to 2011
Aortic root 4.1 – 4.2
Ascending 3.9 – 4.1
Descending 2.7 – 3.2

Average Growth Rate
Ascending thoracic: 0.07 cm/yr
Descending thoracic: 0.19 cm/yr

Mean aortic diameters (in cm) at various levels measured by helical CT in 70 adults.

Average Growth Rate
Ascending thoracic: 0.07 cm/yr
Descending thoracic: 0.19 cm/yr

Male
Aortic sinus: 3.63 to 3.91 cm
Ascending aorta: 2.86 cm
Mid-descending aorta: 2.39 to 2.98 cm
At diaphragm: 2.43 to 2.69 cm

Female
Aortic root: 3.5 to 3.72 cm
Ascending aorta: 2.86 cm
Mid-descending aorta: 2.45 to 2.64 cm
At diaphragm: 2.40 to 2.44 cm

WRITING GROUP MEMBERS et al. Circulation 2010;121:e266-e369
Case 6: Relative Mortality Risk?

75-year-old retired school teacher with controlled hypertension. As part of the physical exam she had an echocardiogram that led to a chest CT.

**Chest CT December 2009**
- Aortic root: 4.1 cm
- Ascending aorta is prominent measuring 3.9 cm in the mid-ascending segment.
- Proximal Aortic Arch: 3.9 cm
- Descending thoracic aorta: 2.7 cm

**Chest CT December 2011:**
Study described as “unchanged” from December 2009
- Aortic root: 4.2 cm (was 4.1 cm in 2009)
- Ascending aorta: 4.1 cm in the mid-ascending segment (was 3.9 cm in 2009)
- Proximal Aortic Arch: 4.2 cm (was 3.9 cm in 2009)
- Descending thoracic aorta: 3.2 cm (was 2.7 cm in 2009)
No evidence of dissection.

Case 7

65-year-old mechanical engineer with statin-treated hyperlipidemia and well-controlled hypertension. Height 72” and weight 215 pounds. BMI 28.5 BSA 2.175 m²

In **May 2010** due to non-specific abdominal pain he had an abdominal ultrasound that showed an abdominal aorta of **4.3 cm**

(a) Scenario
- **May 2012** abdominal ultrasound reveals AAA **4.3 cm**

(b) Scenario
- Same as (a) except that AAA size is **4.6 cm**
Abdominal Aortic Aneurysm

- A focal dilatation with at least a 50% increase over the normal diameter, 3 cm for the abdominal aorta.

- Highly correlated with atherosclerosis, endothelial dysfunction, and the inflammatory mediators that mediate endothelial dysfunction.

- Rupture risk is related to diameter, rate of expansion, and gender.

Cardiovascular Risk Factors Aggravate Aortic Aneurysms

Aortic aneurysms are highly correlated with atherosclerosis and aggravated by factors that accelerate atherosclerosis, especially tobacco smoking.

Prevalence of abdominal aortic aneurysm 4.0 cm or larger in men by age and smoking history

Lederle, F. A. Ann Intern Med 2003;139:516-522
Annual Risk of Rupture of Abdominal Aortic Aneurysm

- < 4.0 cm; zero risk
- 4.0 to 4.9 cm: 0.5 to 5%
- 5.0 to 5.9 cm: 3-15%
- 6.0 to 6.9 cm: 10-20%
- 7.0 to 7.9 cm: 20 to 40%
- > 8.0 cm: 30 to 50%

Abdominal Aneurysm

Growth Rates by Diameter

- 2.8 to 3.9 cm diameter: average growth rate 0.19 cm per year
- 4.0 to 4.5 cm diameter: average growth rate 0.27 cm per year
- 4.6 to 8.5 cm diameter: average growth rate 0.35 cm per year

Growth rates are variable (some don’t grow), so serial measurements are necessary.

Growth is more rapid in smokers
Risk of Rupture Relative to Body Surface Area ("Indexed Risk")

<table>
<thead>
<tr>
<th>Aortic size (cm)</th>
<th>3.5</th>
<th>4.0</th>
<th>4.5</th>
<th>5.0</th>
<th>5.5</th>
<th>6.0</th>
<th>6.5</th>
<th>7.0</th>
<th>7.5</th>
<th>8.0</th>
</tr>
</thead>
<tbody>
<tr>
<td>1.30</td>
<td>2.69</td>
<td>3.00</td>
<td>3.40</td>
<td>3.85</td>
<td>4.23</td>
<td>4.62</td>
<td>5.00</td>
<td>5.38</td>
<td>5.77</td>
<td>6.15</td>
</tr>
<tr>
<td>1.40</td>
<td>2.60</td>
<td>2.86</td>
<td>3.21</td>
<td>3.57</td>
<td>3.90</td>
<td>4.24</td>
<td>4.66</td>
<td>5.00</td>
<td>5.38</td>
<td>5.77</td>
</tr>
<tr>
<td>1.50</td>
<td>2.33</td>
<td>2.67</td>
<td>3.00</td>
<td>3.33</td>
<td>3.67</td>
<td>4.00</td>
<td>4.33</td>
<td>4.67</td>
<td>5.00</td>
<td>5.33</td>
</tr>
<tr>
<td>1.60</td>
<td>2.19</td>
<td>2.50</td>
<td>2.80</td>
<td>3.13</td>
<td>3.44</td>
<td>3.75</td>
<td>4.06</td>
<td>4.38</td>
<td>4.69</td>
<td>5.00</td>
</tr>
<tr>
<td>1.70</td>
<td>2.05</td>
<td>2.35</td>
<td>2.65</td>
<td>2.94</td>
<td>3.24</td>
<td>3.53</td>
<td>3.82</td>
<td>4.12</td>
<td>4.41</td>
<td>4.71</td>
</tr>
<tr>
<td>1.80</td>
<td>1.94</td>
<td>2.22</td>
<td>2.50</td>
<td>2.79</td>
<td>3.06</td>
<td>3.35</td>
<td>3.64</td>
<td>3.93</td>
<td>4.21</td>
<td>4.44</td>
</tr>
<tr>
<td>1.90</td>
<td>1.84</td>
<td>2.11</td>
<td>2.37</td>
<td>2.63</td>
<td>2.89</td>
<td>3.16</td>
<td>3.42</td>
<td>3.65</td>
<td>3.95</td>
<td>4.22</td>
</tr>
<tr>
<td>2.00</td>
<td>1.75</td>
<td>2.00</td>
<td>2.25</td>
<td>2.50</td>
<td>2.75</td>
<td>3.00</td>
<td>3.25</td>
<td>3.50</td>
<td>3.75</td>
<td>4.00</td>
</tr>
<tr>
<td>2.10</td>
<td>1.67</td>
<td>1.90</td>
<td>2.14</td>
<td>2.38</td>
<td>2.62</td>
<td>2.86</td>
<td>3.10</td>
<td>3.33</td>
<td>3.57</td>
<td>3.80</td>
</tr>
<tr>
<td>2.20</td>
<td>1.59</td>
<td>1.82</td>
<td>2.05</td>
<td>2.27</td>
<td>2.50</td>
<td>2.72</td>
<td>2.95</td>
<td>3.18</td>
<td>3.41</td>
<td>3.64</td>
</tr>
<tr>
<td>2.30</td>
<td>1.52</td>
<td>1.74</td>
<td>1.96</td>
<td>2.17</td>
<td>2.39</td>
<td>2.61</td>
<td>2.83</td>
<td>3.04</td>
<td>3.26</td>
<td>3.48</td>
</tr>
<tr>
<td>2.40</td>
<td>1.46</td>
<td>1.67</td>
<td>1.88</td>
<td>2.08</td>
<td>2.29</td>
<td>2.50</td>
<td>2.71</td>
<td>2.92</td>
<td>3.13</td>
<td>3.33</td>
</tr>
<tr>
<td>2.50</td>
<td>1.40</td>
<td>1.60</td>
<td>1.80</td>
<td>2.00</td>
<td>2.20</td>
<td>2.40</td>
<td>2.60</td>
<td>2.80</td>
<td>3.00</td>
<td>3.20</td>
</tr>
</tbody>
</table>

Abdominal Aortic Rupture Rates

Risk of rupture of an abdominal aortic aneurysm (AAA) over time according to the first measurement of aneurysm diameter in 1,745 men and 464 women. The risk of rupture increases independently in aneurysms larger than 5.5 cm in diameter.

Abdominal Aortic Rupture Rates

Risk of rupture of an abdominal aortic aneurysm (AAA) over time according to the first measurement of aneurysm diameter in 1,745 men and 464 women. The risk of rupture increases independently in aneurysms larger than 5.5 cm in diameter.

Endovascular Repair of AAA

At present, EVR accounts for > 60% of all repairs

Operative mortality associated with EVR only 1/3 that of open repair

Early benefit of EVR lost in the longer term

After 4 years, aneurysm-related mortality significantly higher in EVR

Risk extends out at least 8 years Re-intervention after EVR remains a substantial risk after 4 years

Case 7: Relative Mortality Risk?

65-year-old mechanical engineer with statin-treated hyperlipidemia and well-controlled hypertension. In May 2010 due to non-specific abdominal pain he had an abdominal ultrasound that showed an abdominal aorta of 4.3 cm

(a) Scenario
May 2012 abdominal ultrasound reveals AAA 4.3 cm

(b) Scenario
Same as (a) except that AAA size is 4.6 cm

Relative Mortality

• 100 %
• 150 %
• 200 %
• 250 %
• 300 %
• 300 %+

N ENGL J MED Vol 358, January 31, 2008
45-year old male

Background:

Family History of premature CAD with MI & death in 50s: paternal grandfather, maternal grandfather, maternal aunt, maternal uncle.

Treated and well controlled hyperlipidemia. Borderline normal BP, 5'9" 179#, asymptomatic and runs for exercise.

Had EBCT and “positive” calcium score, but we do not have results. This was followed by a CCTA for “coronary artery disease” on 7/6/09

ETT 7/20/09: 12:00, 13 METS, HR to 172 and reported as EKG negative and normal Echocardiogram response to exercise.
Case 8 . . . Cont.

Same 45-year-old male

CTA 2009:

There is diffuse calcified plaque throughout the proximal and mid-LAD with two potential areas of flow limiting stenosis.

Mild calcified plaque is present in the proximal and mid-left circumflex artery as well as the proximal and mid RCA without significant coronary artery stenosis.

Silent Lesions (< 50%)

The burden of angiographic disease detected by CTA provides both independent and incremental value in predicting all-cause mortality.

JACC 2008
Silent Lesions (< 50%)

24,775 patients suspected CAD; age 57 +/- 13 years 54% male
Follow up: 2.3 +/- 1.1 years; 404 deaths

CONFIRM JACC 2011

Case 8: Relative Mortality Risk?

45 y/o with family history of premature CAD with MI & death in 50s: paternal grandfather, maternal grandfather, maternal aunt, maternal uncle.

Treated and well controlled hyperlipidemia. Borderline normal BP, 5'9" 179#, asymptomatic and runs for exercise.

Had EBCT and "positive" calcium score, but we do not have results. This was followed by a CCTA for "coronary artery disease" on 7/6/09

ETT 7/20/09: 12:00, 13 METS, HR to 172 and reported as EKG negative and normal Echocardiogram response to exercise.

CTA 2009: diffuse calcified plaque throughout the proximal and mid-LAD with two potential areas of flow limiting stenosis.

Mild calcified plaque is present in the proximal and mid-left circumflex artery as well as the proximal and mid RCA without significant coronary artery stenosis.
60-year-old female with hypertension and chronic microalbuminuria with negative renal workup. Also sleep apnea surgery

EBCT February 2008 Zero
Echocardiogram 2008 normal

Cooper Clinic stress test February 2008: negative; no PVCs (report)

Jan 2011 stress test: rsr' pattern in V1-2; negative for ischemia no PVCs (Balke Protocol)

Dec 2011 - Stress test done as part of insurance physical
Case 9

And... The Medical Director Worst Nightmare

“Benign” Arrhythmias that kill

<table>
<thead>
<tr>
<th>Type of VT</th>
<th>QRS morphology/axis</th>
<th>Pharmacotherapy sensitivity</th>
<th>Treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td>RVOT VT/monomorphic extrasystoles</td>
<td>LBBB/ inferior axis</td>
<td>Adenosine, B-blocker, verapamil (or diltiazem) B-Blocker, verapamil</td>
<td>RF ablation</td>
</tr>
<tr>
<td>LVOT VT</td>
<td>S wave in lead I, R-wave transition in V1 or V2</td>
<td>Adenosine, B-blocker, verapamil (or diltiazem) B-Blocker, verapamil</td>
<td>RF ablation</td>
</tr>
<tr>
<td>Fascicular VT</td>
<td>RBBB/ left superior axis (left posterior fascicle); RBBB right inferior axis (left anterior fascicle)</td>
<td>Verapamil</td>
<td>RF ablation</td>
</tr>
</tbody>
</table>
Case 9: Relative Mortality Risk?

60-year-old female with hypertension and chronic microalbuminuria with negative renal workup. Also sleep apnea surgery

EBCT February 2008 Zero
Echocardiogram 2008 normal

Cooper Clinic stress test February 2008: negative; no PVCs (report)

Jan 2011 stress test: rsr' pattern in V1-2; negative for ischemia no PVCs (Balke Protocol)

Dec 2011 - Stress test done as part of insurance physical

Relative Mortality
- 100 %
- 150 %
- 200 %
- 250 %
- 300 %
- 300 %+

Case 10

61 year-old male, 5'6" 272 pounds, with stable angina. Diabetes ten years treated with insulin (HbA1c between 7.5 – 8%). In 2007 right common femoral artery stented for PAD.

2010 : Angina with positive stress test
New Cath:

RCA: 100% obstructed
LAD: proximal stent is patent
First diagonal: moderate ostial obstruction
Circumflex: proximal stent patent
OM1 50 %; OM2 99%

"The angina may be possibly due to his chronic total occlusion – insufficient collateral's to RCA". Treated with long acting nitrates
December 2011: leg ulceration (Severe PAD?)

APS: last office visit March 2012 (one page)
“The patient has recent recurrence of chest pain”

SPECT Study – 7 minutes modified Bruce protocol (METS ?)
Resting EF 45% - no significant wall motion abnormality
No evidence of exercise induced ischemia

ARTS II: Event free survival
At one year, there was no difference in event-free survival between the ARTS II SES group and the ARTS I CABG group. However, the ARTS II group showed significantly higher rates of survival free from cardiac death, MI, and reintervention than the ARTS I bare metal stent group. The groups were not significantly different in the primary endpoint of survival free from MACCE.

p = <0.001
p = 0.003
p = 0.46
ARTS II: MACCE at one year

Overall MACCE at 1 year

- At 1 year, there was no difference in the incidence of MACCE between the ARTS II SES group and the ARTS I CABG group.

- The ARTS I bare metal stent group was associated with a significantly higher rate of 1-year MACCE compared to the other groups.

PTCA Fallacy #1

Fallacy
“Doc said I had a 90% blockage. Thank goodness he fixed it in time and saved my life”

Facts
- There is no such thing as a 90% stenosis
- Even if there were, in most cases PTCA is not a life-saving intervention

The COURAGE Paradigm
PTCA Fallacy #2

Fallacy

“Mr. Jones had severe 2-vessel disease but really didn’t want a bypass operation, so I stented both vessels”

Facts

• PTCA is often the first step on the road to CABG
• If a patient really wants to avoid CABG at all costs, medical therapy is the way to go

The COURAGE Paradigm

---

PTCA Fallacy #3

Fallacy

“For most patients with multi-vessel disease, PTCA can provide comparable long-term survival benefits and quality of life as bypass surgery”

Facts

• The randomized trials of PCI vs. CABG have included only highly selected patients
• Observational data still suggest improved survival with CABG in severe multivessel disease

The COURAGE Paradigm
PTCA Fallacy #4

Fallacy
An asymptomatic patient with an abnormal ETT is at high risk for short term complications

“Thankfully, doc ordered that screening stress test when I turned 50 and that other nice doctor did an angioplasty the next day. I could have had a heart attack!”

Facts
• The benefit of revascularization in asymptomatic patients (even our high-risk diabetic patients) is likely restricted to those with high risk stress test findings.
• Risk of periprocedural MI approximates annual risk in some cases

Sorraja P. Circulation. 2005; 112: I311

PTCA Fallacy #5

Fallacy
Stable Angina means the patient needs revascularization to prevent a heart attack

“Thankfully, doc sent me to the hospital for those chest pains and that other nice doctor did an angioplasty the next day. I could have had a heart attack!”

Facts
• PTCA reduces symptoms in chronic angina and may increase the risk of MI, or needing a CABG

Sorraja P. Circulation. 2005; 112: I311
**Case 10: Relative Mortality Risk?**

61 year-old male, 5’6” 272 pounds, with stable angina. Diabetes ten years treated with insulin (HbA1c between 7.5 – 8%). In 2007 right common femoral artery stented for PAD.

<table>
<thead>
<tr>
<th>Relative Mortality</th>
<th>100 %</th>
<th>150 %</th>
<th>200 %</th>
<th>250 %</th>
<th>300 %</th>
<th>300 %+</th>
</tr>
</thead>
</table>

**2010:** Angina with positive stress test  
**New Cath:**  
- RCA: 100% obstructed  
- LAD: proximal stent is patent  
- First diagonal: moderate ostial obstruction  
- Circumflex: proximal stent patent  
- OM1 50%; OM2 99%

“The angina may be possibly due to his chronic total occlusion – insufficient collateral’s to RCA”. Treated with long acting nitrates

**December 2011:** leg ulceration (Severe PAD?)

APS: last office visit March 2012 (one page) “The patient has recent recurrence of chest pain”

**SPECT Study – 7 minutes modified Bruce protocol (METS ?)**  
Resting EF 45% - no significant wall motion abnormality  
No evidence of exercise induced ischemia