2010 ACCF/AHA/AATS/ACR/ASA/SCA/SCAI/SIR/STS/SVM Guidelines for the Diagnosis and Management of Patients with Thoracic Aortic Disease


Endorsed by the North American Society for Cardiovascular Imaging.
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The full-text guidelines are also available on the following Web sites:
ACC (www.acc.org) and,
AHA (www.americanheart.org)
Special Thanks To

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## Classification of Recommendations

<table>
<thead>
<tr>
<th>Class I</th>
<th>Class IIa</th>
<th>Class IIb</th>
<th>Class III</th>
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</thead>
<tbody>
<tr>
<td>Benefit &gt;&gt;&gt; Risk</td>
<td>Benefit &gt;&gt; Risk</td>
<td>Benefit ≥ Risk</td>
<td>Risk ≥ Benefit</td>
</tr>
<tr>
<td>Procedure/Treatment <strong>SHOULD</strong> be performed/administered</td>
<td><strong>IT IS REASONABLE</strong> to perform procedure/administer treatment</td>
<td>Procedure/Treatment <strong>MAY BE CONSIDERED</strong></td>
<td>Procedure/Treatment <strong>should NOT</strong> be performed/administered <strong>SINCE IT IS NOT HELPFUL AND MAY BE HARMFUL</strong></td>
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**Alternative Phrasing:**

<table>
<thead>
<tr>
<th>should</th>
<th>is reasonable</th>
<th>may/might be considered</th>
<th>is not recommended</th>
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<tbody>
<tr>
<td>is recommended</td>
<td>can be useful/effective/beneficial</td>
<td>may/might be considered usefulness/effectiveness</td>
<td>is not recommended</td>
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<tr>
<td>is indicated</td>
<td>is probably recommended or indicated</td>
<td>is unknown/unclear/uncertain or not well established</td>
<td>is not indicated</td>
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<tr>
<td>is useful/effective/beneficial</td>
<td></td>
<td>may be harmful</td>
<td>should not</td>
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<tr>
<td></td>
<td></td>
<td></td>
<td>is not useful/effective/beneficial</td>
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</table>
## Applying Classification of Recommendations and Level of Evidence

### Level of Evidence:

| Level A: | Data derived from multiple randomized clinical trials or meta-analyses  
Multiple populations evaluated; |
|----------|------------------------------------------------------------------------|
| Level B: | Data derived from a single randomized trial or nonrandomized studies  
Limited populations evaluated |
| Level C: | Only consensus of experts opinion, case studies, or standard of care  
Very limited populations evaluated |

### Class I

*Benefit >>> Risk*

Procedure/Treatment **SHOULD** be performed/administered

### Class Ila

*Benefit >>> Risk*

Additional studies with focused objectives needed

**IT IS REASONABLE** to perform procedure/administer treatment

### Class Ilb

*Benefit ≥ Risk*

Additional studies with broad objectives needed; Additional registry data would be helpful

Procedure/Treatment **MAY BE CONSIDERED**

### Class III

*Risk ≥ Benefit*

No additional studies needed

Procedure/Treatment should **NOT** be performed/administered  
**SINCE IT IS NOT HELPFUL AND MAY BE HARMFUL**
Icons representing the Classification and Evidence Levels for Recommendations
As the writing committee developed this TAD guideline, several critical issues emerged:

- Thoracic aortic diseases (TADs) are usually asymptomatic and not easily detectable until an acute and often catastrophic complication occurs.

- The identification and treatment of stable patients at risk for acute and catastrophic disease presentations (eg, thoracic aortic dissection (AoD) and thoracic aneurysm rupture) prior to such an occurrence are paramount to eliminating the high morbidity and mortality associated with acute presentations.

- Imaging of the thoracic aorta is the only method to detect thoracic aortic diseases and determine risk for future complications.
  - Radiologic imaging technologies have improved in terms of accuracy of detection of TAD. However, increased use of these technologies increases the potential risk associated with repeated radiation exposure, as well as contrast medium–related toxicity.
  - Imaging for asymptomatic patients at high risk based on history or associated diseases is expensive and not always covered by payers.
A subset of patients with acute AoD are subject to missed or delayed detection of this catastrophic disease state.

- Many present with atypical symptoms and findings, making diagnosis even more difficult.
- Widespread awareness of the varied and complex nature of TAD presentations has been lacking, especially for acute AoD.

There is rapidly accumulating evidence that genetic alterations or mutations predispose some individuals to aortic diseases.

- Identification of the genetic alterations leading to these aortic diseases has potential for early detection among at-risk individuals.
- Biochemical alterations identified in the aortic tissue have the potential to serve as biomarkers for aortic disease.
- Understanding the molecular pathogenesis may lead to targeted therapy to prevent aortic disease.

Medical and gene-based treatments are beginning to show promise for reducing or delaying catastrophic complications of thoracic aortic diseases.
Recommendations for Aortic Imaging Techniques to Determine the Presence and Progression of Thoracic Aortic Disease
Measurements of aortic diameter should be taken at reproducible anatomic landmarks, perpendicular to the axis of blood flow, and reported in a clear and consistent format (see table entitled “Essential Elements of Aortic Imaging Reports”).

For measurements taken by computed tomographic imaging or magnetic resonance imaging, the external diameter should be measured perpendicular to the axis of blood flow. For aortic root measurements, the widest diameter, typically at the mid-sinus level, should be used.
For measurements taken by echocardiography, the internal diameter should be measured perpendicular to the axis of blood flow. For aortic root measurements, the widest diameter, typically at the mid-sinus level, should be used.

Abnormalities of aortic morphology should be recognized and reported separately even when aortic diameters are within normal limits.
The finding of aortic dissection, aneurysm, traumatic injury and/or aortic rupture should be immediately communicated to the referring physician.

Techniques to minimize episodetic and cumulative radiation exposure should be utilized whenever possible.

If clinical information is available, it can be useful to relate aortic diameter to the patient’s age and body size.
Technical Parameters for Computed Tomographic Imaging

Recommended details of CT technique includes the following:

CT angiographic acquisition using intravenous contrast delivered at rate of 3-5 mL/s using a power injector, followed by a saline chaser bolus.

Total contrast volume should be as low as possible (no more than 150 mL)

Recommended technical parameters for image acquisition:

- Slices thickness of 3 mm or less with a reconstruction interval of 50% or smaller than the slice thickness
- Tube rotation of 1 second or less
- 120-140 kVp; mA adjusted to patient size
- ECG gating particularly useful for AoD (Note: prospective triggering has lower radiation exposure than retrospective gating)
- Coverage: thoracic inlet to groin

2- and 3-dimensional reconstructions (e.g., multiplanar and curved multiplanar reformations) and volume rendering may augment interpretation and improve communication of findings, and are likely to play an important role in planning surgical or endovascular treatment approaches.
Essential Elements of Aortic Imaging Reports

The following table outlines specific qualitative and quantitative elements that are important to include in CT and MR reports:

<p>| | |</p>
<table>
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<tbody>
<tr>
<td>1.</td>
<td>The location at which the aorta is abnormal.</td>
</tr>
<tr>
<td>2.</td>
<td>The maximum diameter of any dilatation, measured from the external wall of the aorta, perpendicular to the axis of flow, and the length of the aorta that is abnormal.</td>
</tr>
<tr>
<td>3.</td>
<td>For patients with presumed or documented genetic syndromes at risk for aortic root disease measurements of aortic valve, sinuses of Valsalva, sinotubular junction, and ascending aorta.</td>
</tr>
<tr>
<td>4.</td>
<td>The presence of internal filling defects consistent with thrombus or atheroma.</td>
</tr>
<tr>
<td>5.</td>
<td>The presence of intramural hematoma (IMH), penetrating atherosclerotic ulcer (PAU), and calcification.</td>
</tr>
<tr>
<td>6.</td>
<td>Extension of aortic abnormality into branch vessels, including dissection and aneurysm, and secondary evidence of end-organ injury (e.g., renal or bowel hypoperfusion).</td>
</tr>
<tr>
<td>7.</td>
<td>Evidence of aortic rupture, including periaortic and mediastinal hematoma, pericardial and pleural fluid, and contrast extravasation from the aortic lumen.</td>
</tr>
<tr>
<td>8.</td>
<td>When a prior examination is available, direct image to image comparison to determine if there has been any increase in diameter.</td>
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</tbody>
</table>

Note: This is Table 5 in the full-text version of the TAD Guideline.
Recommendations for Genetic Syndromes Associated with Thoracic Aortic Aneurysms and Dissections
Recommendations for Genetic Syndromes Associated
with Thoracic Aortic Aneurysms and Dissections

An echocardiogram is recommended at the time of diagnosis of Marfan syndrome to determine the aortic root and ascending aortic diameters and 6 months thereafter to determine the rate of enlargement of the aorta.

Annual imaging is recommended for patients with Marfan syndrome if stability of the aortic diameter is documented. If the maximal aortic diameter is 4.5 cm or greater, or if the aortic diameter shows significant growth from baseline, more frequent imaging should be considered.
Recommendations for Genetic Syndromes Associated with Thoracic Aortic Aneurysms and Dissections

Patients with Loeys-Dietz syndrome or a confirmed genetic mutation known to predispose to aortic aneurysms and aortic dissections (\textit{TGFBR1, TGFBR2, FBN1, ACTA2, or MYH11}) should undergo complete aortic imaging at initial diagnosis and 6 months thereafter to establish if enlargement is occurring.

Loeys-Dietz patients should have yearly magnetic resonance imaging from the cerebrovascular circulation to the pelvis.
Patients with Turner syndrome should undergo imaging of the heart and aorta for evidence of bicuspid aortic valve, coarctation of the aorta, or dilatation of the ascending thoracic aorta. If initial imaging is normal and there are no risk factors for aortic dissection, repeat imaging should be performed every 5 to 10 years or if otherwise clinically indicated. If abnormalities exist, annual imaging or follow-up imaging should be done.
Recommendations for Genetic Syndromes Associated with Thoracic Aortic Aneurysms and Dissections

It is reasonable to consider surgical repair of the aorta in all adult patients with Loeys-Dietz syndrome or a confirmed TGFBR1 or TGFBR2 mutation and an aortic diameter of 4.2 cm or greater by transesophageal echocardiogram (internal diameter) or 4.4 to 4.6 cm or greater by computed tomographic imaging and/or magnetic resonance imaging (external diameter).

For women with Marfan syndrome contemplating pregnancy, it is reasonable to prophylactically replace the aortic root and ascending aorta if the diameter exceeds 4.0 cm.
If the maximal cross-sectional area in square centimeters of the ascending aorta or root divided by the patient's height in meters exceeds a ratio of 10, surgical repair is reasonable because shorter patients have dissection at a smaller size and 15% of patients with Marfan syndrome have dissection at a size smaller than 5.0 cm.

In patients with Turner syndrome with additional risk factors, including bicuspid aortic valve, coarctation of the aorta, and/or hypertension, and in patients who attempt to become pregnant or who become pregnant, it may be reasonable to perform imaging of the heart and aorta to help determine the risk of aortic dissection.
Gene Defects Associated With Familial Thoracic Aortic Aneurysm and Dissection

<table>
<thead>
<tr>
<th>Defective Gene Leading to Familial Thoracic Aortic Aneurysms and Dissection</th>
<th>Contribution to Familial Thoracic Aortic Aneurysms and Dissection</th>
<th>Associated Clinical Features</th>
<th>Comments on Aortic Disease</th>
</tr>
</thead>
</table>
| TGFBR2 (transforming growth factor-beta receptor type 2) mutations | 4% | • Thin, translucent skin  
• Arterial or aortic tortuosity  
• Aneurysm of arteries | Multiple aortic dissections documented at aortic diameters <5.0 cm |
| MYH11 (smooth muscle specific beta-myosin heavy chain) mutations | 1% | • Patent ductus arteriosus | Patient with documented dissection at 4.5 cm |
| ACTA2 (actin, alpha 2, smooth muscle aorta) mutations | 14% | • Livedo reticularis  
• Iris flocculi  
• Patent ductus arteriosus  
• Bicuspid aortic valve | Two of 13 patients with documented dissections <5.0 cm |

Note: Table 6 in full-text version of TAD Guidelines
## Genetic Syndromes Associated With Thoracic Aortic Aneurysm and Dissection

<table>
<thead>
<tr>
<th>Genetic Syndrome Genetic</th>
<th>Common Clinical Features</th>
<th>Defect</th>
<th>Diagnostic Test</th>
<th>Comments on Aortic Disease</th>
</tr>
</thead>
<tbody>
<tr>
<td>Marfan syndrome</td>
<td>Skeletal features (see text); Ectopia Lentis; Dural ectasia</td>
<td><em>FBN1</em> mutations*</td>
<td>Ghent diagnostic criteria; DNA for sequencing</td>
<td>Surgical repair when the aorta reaches 5.0 cm unless there is a family history of AoD at &lt;5.0 cm, a rapidly expanding aneurysm or presence or significant aortic valve regurgitation</td>
</tr>
<tr>
<td>Loeys-Dietz syndrome</td>
<td>Bifid uvula or cleft palate; Arterial tortuosity; Hypertelorism; Skeletal features similar to MFS; Craniosynostosis; Aneurysms and dissections of other arteries</td>
<td><em>TGFBR2</em> or <em>TGFBR1</em> mutations</td>
<td>DNA for sequencing</td>
<td>Surgical repair recommended at an aortic diameter of ≥4.2 cm by TEE (internal diameter) or 4.4 to ≥4.6 cm by CT and/or MR (external diameter)</td>
</tr>
<tr>
<td>Ehlers-Danlos syndrome (vascular form)</td>
<td>Thin, translucent skin; Gastrointestinal rupture; Rupture of the gravid uterus; Rupture of medium-sized to large arteries</td>
<td><em>COL3A1</em> mutations</td>
<td>DNA for sequencing; Dermal fibroblasts for analysis of type 3 collagen</td>
<td>Surgical repair is complicated by friable tissues; Noninvasive imaging recommended</td>
</tr>
<tr>
<td>Turner syndrome</td>
<td>Short stature; Primary amenorrhea; Bicuspid aortic valve; Aortic coarctation; Webbed neck, low-set ears, low hairline, broad chest</td>
<td>45,X karyotype</td>
<td>Blood (cells) for karyotype analysis</td>
<td>AoD risk is increased in patients with bicuspid aortic valve, aortic coarctation, hypertension, or pregnancy</td>
</tr>
</tbody>
</table>

* The defective gene at a second locus for MFS is *TGFBR2* but the clinical phenotype as MFS is debated.

AoD = aortic dissection; *COL3A1*, type III collagen; *FBN1*, fibrillin 1; MFS, Marfan syndrome; *TGFBR1*, transforming growth factor-beta receptor type 1; and *TGFBR2*, transforming growth factor beta receptor type 2.

Note: Table 7 in full-text version of TAD Guidelines

* The defective gene at a second locus for MFS is *TGFBR2* but the clinical phenotype as MFS is debated.

AoD = aortic dissection; *COL3A1*, type III collagen; *FBN1*, fibrillin 1; MFS, Marfan syndrome; *TGFBR1*, transforming growth factor-beta receptor type 1; and *TGFBR2*, transforming growth factor beta receptor type 2.

Note: Table 7 in full-text version of TAD Guidelines
Guidelines for Thoracic Aortic Disease

Recommendations for Familial Thoracic Aortic Aneurysm and Dissections
Aortic imaging is recommended for first degree relatives of patients with thoracic aortic aneurysm and/or dissection to identify those with asymptomatic disease.

If the mutant gene (FBN1, TGFBR1, TGFBR2, COL3A1, ACTA2, MYH11) associated with aortic aneurysm and/or dissection is identified in a patient, first degree relatives should undergo counseling and testing. Then, only the relatives with the genetic mutation should undergo aortic imaging.
If one or more first degree relatives of a patient with known thoracic aortic aneurysm and/or dissection are found to have thoracic aortic dilatation, aneurysm, or dissection, then imaging of second-degree relatives is reasonable.

Sequencing of the ACTA2 gene is reasonable in patients with a family history of thoracic aortic aneurysms and/or dissections to determine if ACTA2 mutations are responsible for the inherited predisposition.
Sequencing of other genes known to cause familial thoracic aortic aneurysms and/or dissection (TGFBR1, TGFBR2, MYH11) may be considered in patients with a family history and clinical features associated with mutations in these genes.

If one or more first degree relatives of a patient with known thoracic aortic aneurysm and/or dissection are found to have thoracic aortic dilatation, aneurysm, or dissection, then referral to a geneticist may be considered.
Guidelines for Thoracic Aortic Disease

Recommendations for Bicuspid Aortic Valve and Associated Congenital Variants in Adults
First-degree relatives of patients with a bicuspid aortic valve, premature onset of thoracic aortic disease with minimal risk factors, and/or a familial form of thoracic aortic aneurysm and dissection should be evaluated for the presence of a bicuspid aortic valve and asymptomatic thoracic aortic disease.

All patients with a bicuspid aortic valve should have both the aortic root and ascending thoracic aorta evaluated for evidence of aortic dilatation.
Guidelines for Thoracic Aortic Disease

Recommendations for Takayasu Arteritis and Giant Cell Arteritis
Initial therapy for active Takayasu arteritis and active giant cell arteritis should be corticosteroids at a high dose (prednisone 40 to 60 mg daily at initiation or its equivalent) to reduce the active inflammatory state.

The success of treatment of patients with Takayasu arteritis and giant cell arteritis should be periodically evaluated to determine disease activity by repeated physical examination and either an erythrocyte sedimentation rate or C-reactive protein level.

Elective revascularization of patients with Takayasu arteritis and giant cell arteritis should be delayed until the acute inflammatory state is treated and quiescent.
The initial evaluation of Takayasu arteritis or giant cell arteritis should include thoracic aorta and branch vessel computed tomographic imaging or magnetic resonance imaging to investigate the possibility of aneurysm or occlusive disease in these vessels.

It is reasonable to treat patients with Takayasu arteritis receiving corticosteroids with an additional anti-inflammatory agent if there is evidence of progression of vascular disease, recurrence of constitutional symptoms, or re-elevation of inflammatory marker.
Guidelines for Thoracic Aortic Disease

Recommendations for Initial Evaluation and Management of Acute Thoracic Aortic Disease
Providers should routinely evaluate any patient presenting with complaints that may represent acute thoracic aortic dissection to establish a pretest risk of disease that can then be used to guide diagnostic decisions. This process should include specific questions about:

- medical history,
- family history, and
- pain features.

This process should also include a focused examination to identify findings that are associated with aortic dissection (outlined in the next 3 slides).
Estimation of Pretest Risk of Thoracic Aortic Dissection

High Risk Conditions

- Marfan Syndrome
- Connective tissue disease*
- Family history of aortic disease
- Known aortic valve disease
- Recent aortic manipulation (surgical or catheter-based)
- Known thoracic aortic aneurysm
- Genetic conditions that predispose to AoD†

* Loeys-Dietz syndrome, vascular Ehlers-Danlos syndrome, Turner syndrome, or other connective tissue disease.

† Patients with mutations in genes known to predispose to thoracic aortic aneurysms and dissection, such as FBN1, TGFBR1, TGFBR2, ACTA2, and MYH11.
High Risk Pain Features

Chest, back, or abdominal pain features described as pain that:

• is abrupt or instantaneous in onset.
• is severe in intensity.
• has a ripping, tearing, stabbing, or sharp quality.
Estimation of Pretest Risk of Thoracic Aortic Dissection

High Risk Examination Features

• Pulse deficit
• Systolic BP limb differential > 20mm Hg
• Focal neurologic deficit
• Murmur of aortic regurgitation (new or not known to be old and in conjunction with pain)
Recommendations for Estimation of Pretest Risk of Thoracic Aortic Dissection

Patients presenting with sudden onset of severe chest, back, and/or abdominal pain, particularly those less than 40 years of age, should be questioned about a history and examined for physical features of Marfan syndrome, Loeys-Dietz syndrome, vascular Ehlers-Danlos syndrome, Turner syndrome, or other connective tissue disorder associated with thoracic aortic disease.
Patients presenting with sudden onset of severe chest, back, and/or abdominal pain should be questioned about a history of aortic pathology in immediate family members as there is a strong familial component to acute thoracic aortic disease.

Patients presenting with sudden onset of severe chest, back, and/or abdominal pain should be questioned about recent aortic manipulation (surgical or catheter-based) or a known history of aortic valvular disease, as these factors predispose to acute aortic dissection.
In patients with suspected or confirmed aortic dissection who have experienced a syncopal episode, a focused examination should be performed to identify associated neurologic injury or the presence of pericardial tamponade.

All patients presenting with acute neurologic complaints should be questioned about the presence of chest, back, and/or abdominal pain and checked for peripheral pulse deficits as patients with dissection-related neurologic pathology are less likely to report thoracic pain than the typical aortic dissection patient.
Patients with no high-risk features of TAD present are considered at low risk for TAD. The following clinical steps are recommended for low-risk TAD patients:

- Proceed with diagnostic evaluation as clinically indicated by presentation.
- Alternative diagnosis identified?
  - Yes: Initiate appropriate Therapy.
  - No: Unexplained hypotension or widened mediastinum on CXR?
    - Yes: Expedited aortic imaging
      - TEE (preferred if clinically unstable)
      - CT scan (image entire aorta: chest to pelvis)
      - MR (image entire aorta: chest to pelvis)
    - No: Consider aortic imaging study for TAD based on clinical scenario (particularly in patients with advanced age, risk factors for aortic disease, or syncope)
Risk-based Diagnostic Evaluation: Patients with Intermediate Risk of TAD

The following steps for patients with *intermediate risk* of TAD should be followed when any single high-risk feature is present.

1. **EKG consistent with STEMI?**
   - Yes → Likely primary ACS. In absence of other perfusion deficits, strongly consider immediate coronary re-perfusion therapy. If PTCA performed, is culprit lesion identified?
   - No → **CXR with clear Alternate diagnosis?**
     - Yes → Initiate appropriate therapy.
     - No → **History and physical exam strongly suggestive of specific alternate diagnosis?**
       - Yes → Alternate diagnosis confirmed by further testing?
       - No → Expedited aortic imaging
         - TEE (preferred if clinically unstable)
         - CT scan (image entire aorta: chest to pelvis)
         - MR (image entire aorta: chest to pelvis)
Patients at *high-risk* for TAD are those that present with at least 2 high-risk features (outlined in more detail in the following slides).

The recommended course of action for high-risk TAD patients is to seek immediate surgical consultation and arrange for expedited aortic imaging.

**Expedited aortic imaging**

- TEE (preferred if clinically unstable)
- CT scan (image entire aorta: chest to pelvis)
- MR (image entire aorta: chest to pelvis)
Risk Factors for Development of Thoracic Aortic Dissection

Conditions Associated With Increased Aortic Wall Stress

- Hypertension, particularly if uncontrolled
- Pheochromocytoma
- Cocaine or other stimulant use
- Weight lifting or other Valsalva maneuver
- Trauma
- Deceleration or torsional injury (eg, motor vehicle crash, fall)
- Coarctation of the aorta

Note: Information on this slide is adapted from Table 9 in full-text version of TAD Guidelines
Conditions Associated With Aortic Media Abnormalities

Genetic

- Marfan syndrome
- Ehlers-Danlos syndrome, vascular form
- Bicuspid aortic valve (including prior aortic valve replacement)
- Turner syndrome
- Loeys-Dietz syndrome
- Familial thoracic aortic aneurysm and dissection syndrome

Note: Information on this slide is adapted from Table 9 in full-text version of TAD Guidelines
Risk Factors for Development of Thoracic Aortic Dissection (continued)

Conditions Associated With Aortic Media Abnormalities (continued)

Inflammatory vasculitides
- Takayasu arteritis
- Giant cell arteritis
- Behçet arteritis

Other
- Pregnancy
- Autosomal dominant polycystic kidney disease
- Chronic corticosteroid or immunosuppression agent administration
- Infections involving the aortic wall either from bacteremia or extension of adjacent infection

Note: Information on this slide is adapted from Table 9 in full-text version of TAD Guidelines
Aortic Dissection Classification: DeBakey and Stanford Classifications

Note: Figure 20 in full-text version of TAD Guidelines. Reprinted with permission from The Cleveland Clinic Foundation.
An electrocardiogram should be obtained on all patients who present with symptoms that may represent acute thoracic aortic dissection.

- Given the relative infrequency of dissection-related coronary artery occlusion, the presence of ST-segment elevation suggestive of myocardial infarction should be treated as a primary cardiac event without delay for definitive aortic imaging unless the patient is at high risk for aortic dissection.
The role of chest x-ray in the evaluation of possible thoracic aortic disease should be directed by the patient’s pretest risk of disease as follows.

- **Intermediate risk**: Chest x-ray should be performed on all intermediate-risk patients, as it may establish a clear alternate diagnosis that will obviate the need for definitive aortic imaging.

- **Low risk**: Chest x-ray should be performed on all low-risk patients, as it may either establish an alternative diagnosis or demonstrate findings that are suggestive of thoracic aortic disease, indicating the need for urgent definitive aortic imaging.
Urgent and definitive imaging of the aorta using transesophageal echocardiogram, computed tomographic imaging, or magnetic resonance imaging is recommended to identify or exclude thoracic aortic dissection in patients at high risk for the disease by initial screening.

A negative chest x-ray should not delay definitive aortic imaging in patients determined to be high risk for aortic dissection by initial screening.
Selection of a specific imaging modality to identify or exclude aortic dissection should be based on patient variables and institutional capabilities, including immediate availability.

If a high clinical suspicion exists for acute aortic dissection but initial aortic imaging is negative, a second imaging study should be obtained.
Initial management of thoracic aortic dissection should be directed at decreasing aortic wall stress by controlling heart rate and blood pressure as follows:

**a.** In the absence of contraindications, intravenous beta blockade should be initiated and titrated to a target heart rate of 60 beats per minute or less.

**b.** In patients with clear contraindications to beta blockade, nondihydropyridine calcium channel–blocking agents should be used as an alternative for rate control.
c. If systolic blood pressures remain greater than 120mm Hg after adequate heart rate control has been obtained, then angiotensin-converting enzyme inhibitors and/or other vasodilators should be administered intravenously to further reduce blood pressure that maintains adequate end-organ perfusion.

d. Beta blockers should be used cautiously in the setting of acute aortic regurgitation because they will block the compensatory tachycardia.