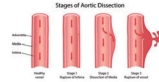
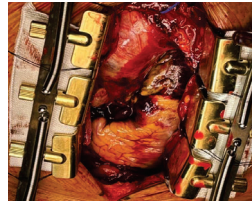
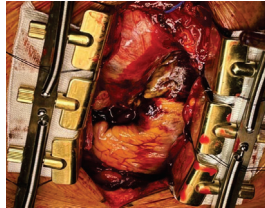


# Aortic Dissections

Understanding The Condition

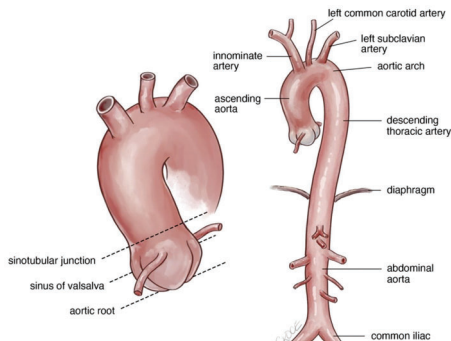
Andres Leal MD.  
Cardiothoracic Surgery  
Presbyterian Hospital Dallas



## Aortic Dissection;

- While rare an acute aortic dissection (AAD) is a catastrophic disorder.
- Aortic dissection is due to the separation of the layers of the aortic wall.
- A tear in the intimal layer results in the progression of the dissection (either proximal or retrograde) chiefly due to the entry of blood in between the intima and media.
- An acute aortic dissection is associated with very high mortality; the majority die even before reaching the emergency department.

## Anatomy of the aorta



## Types of Aortic Dissection

- The Stanford system is more frequently employed. It classifies dissections into two types based on whether ascending or descending part of the aorta involved.
  - Type A involves the ascending aorta, regardless of the site of the primary intimal tear. Type A dissection is defined as a dissection proximal to the brachiocephalic artery.
  - Type B aortic dissection originating distal to the left subclavian artery and involving only descending aorta.
- The DeBakey classification is based upon the site of origin of the dissection.
  - Type 1 originates in the ascending aorta and to at least the aortic arch.
  - Type 2 originates in and is limited to the ascending aorta.
  - Type 3 begins in the descending aorta and extends distally above the diaphragm (type 3a) or below the diaphragm (type 3b).

## Types of Aortic Dissection

Society for Vascular Surgery and Society of Thoracic Surgery  
Reporting Standards for Type B Aortic Dissections

Type	Proximal Extent	Distal Extent
A <sub>D</sub>	0	0
	1	1
	2	2
	3	3
B <sub>PD</sub>	4	4
	5	5
	6	6
	7	7
I <sub>D</sub>	8	8
	9	9
	10	10
	11	11
Unidentified entry tear involving Zone 0	12	12

**Anatomic Reporting of Aortic Dissections are based on:**

- ✓ Location of Entry Tear (A vs B)
- ✓ Proximal & Distal Extent

**EXAMPLES**

**Type A<sub>9</sub>:** Entry tear identified in zone 0 (A), Distal extent in zone 9.

**Type B<sub>4,10</sub>:** Entry tear is identified > zone 0 (B) Proximal extent in zone 4, Distal extent in zone 10.

JVS Journal of Vascular Surgery  
Lombardi et al. J Vasc Surg, March 2020  
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## Etiology

- Hypertension (occurs in 70% of patients with distal Stanford type B AAD)
- An abrupt, transient, severe increase in blood pressure (e.g., strenuous weight lifting and use of sympathomimetic agents such as cocaine, ecstasy, or energy drinks)
- Genetic conditions including Marfan syndrome (In an IRAD review, Marfan syndrome was present in 50% of those under age 40, compared with only 2% of older patients), Ehlers-Danlos syndrome, Turner syndrome, and bicuspid aortic valve, coarctation of the aorta. In patients with Marfan syndrome, cystic medial necrosis is seen in the tissues

## Etiology

- Pre-existing aortic aneurysm
- Atherosclerosis
- Pregnancy and delivery (risk compounded in pregnant women with connective tissue disorders such as Marfan syndrome)
- Family history
- Aortic instrumentation or surgery (coronary artery bypass, aortic or mitral valve replacement, and percutaneous stenting or catheter insertion)
- Inflammatory or infectious diseases that cause vasculitis (syphilis, cocaine use)

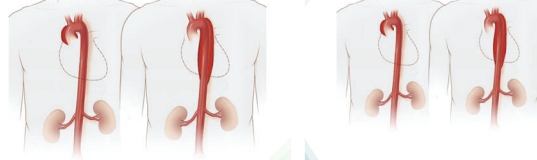


## Epidemiology

- The incidence of aortic dissection is reported to be 5 to 30 cases per 1 million people per year
- Regarding emergency department presentations, three AADs are ultimately diagnosed out of every 1000 patients presenting with acute back, chest, or abdominal pain.
- Age is a risk factor for approximately 75% of aortic dissections occurring in patients who are ages 40 to 70 years, with the majority occurring between the ages of 50 and 65 years.
- However, there are some significant differences between older adult patients and younger patients with dissections involving the ascending aorta. Older patients are significantly more likely to harbor atherosclerosis, prior aortic aneurysm, iatrogenic dissection, or an intramural hematoma.
- Younger patients are much less likely to have a history of hypertension and much more likely to suffer from a connective tissue disorder such as Marfan syndrome. Additionally, AAD is three times more common in men than in women, although women tend to present later and experience worse outcomes.

## Pathophysiology

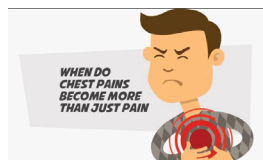
- The aortic wall consists of three layers: the intima, media, and adventitia.
- Constant exposure to high pulsatile pressure and shear stress leads to a weakening of the aortic wall in susceptible patients resulting in an intimal tear.
- Blood flows into the intima-media space, creating a false lumen. Most of these tears take place in the ascending aorta, usually in the right lateral wall where the greatest shear force on the aorta occurs.
- An AAD can propagate antegrade and/or retrograde and depending on the direction the dissection travels, cause branch obstruction that produces ischemia of affected territory (coronary, cerebral, spinal, or visceral), and for proximal type AADs can instigate acute tamponade, aortic regurgitation or aortic rupture.



- In an AAD, the true lumen is lined by the intima whereas the false lumen is within the media. In most cases, the true lumen is smaller than the false lumen. Overtimes, the blood flowing through the false lumen leads to the development of an aneurysm with the potential for rupture. The three common sites for AAD are as follows:
- Nearly 2-2.5 cm above the aortic root (the most common site)
- Just distal to the origin of the left subclavian artery
- In the aortic arch

### Clinical presentation;

- While increasing the probability of AAD when present, classically cited physical findings, such as a discrepancy of blood pressures in the upper extremities, a pulse deficit, or presence of a diastolic murmur, are present in less than 50% of confirmed cases of AAD. Additionally, the presence of chest pain with any neurological finding, the combination of chest and abdominal pain, or chest pain accompanied by limb weakness or paresthesia should alert the clinician to the possibility of AAD.
- The pain of AAD is often sudden in onset, reaches maximal severity quickly, and can be tearing in nature. In about 10% of patients, the AAD is painless, which is more common in Marfan syndrome. The pain can be located in the anterior chest in case of ascending aorta and in the back if the dissection is descending
- Neurological deficits are present in 1/5th of patients. Syncope is also common and may be due to hypovolemia, arrhythmias, MI, or an increased vagal tone. If the dissection is antegrade, it may involve the extremity vessels leading to loss of pulses, paresthesia, and pain.



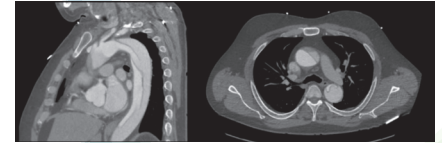
- Hypertension is very common in AAD; if the patient presents with hypotension, then this is a grave sign most likely indicating a rupture. A difference of more than 20mmHg in blood pressure between the arms should raise suspicion of AAD. Other features include:
- Wide pulse pressure
- Aortic insufficiency
- Diastolic murmur
- Muffled heart sounds (suggesting cardiac tamponade)
- Syncope
- Altered mental status
- Loss of peripheral pulses
- Horner syndrome

## Evaluation

- ECG and Chest x-ray;
  - Can help differentiate other possible causes for chest pain but can be misleading. The presence of ECG findings consistent with an acute myocardial infarction occurs in eight percent of cases of AAD. Furthermore, while the widening of the aortic silhouette increases the likelihood of AAD, its absence does not reliably exclude the diagnosis.

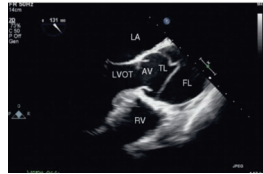
CT scan with contrast is recommended. The spiral CT can quickly detect the location of the intimal tear and help the surgeon plan the procedure. Suggestive of AAD findings include:

- Intimal dissection flap
- Double lumen
- Aortic dilation and hematoma
- Regions of malperfusion
- Contrast leak indicates aortic rupture



Echocardiography is also a useful modality to detect AAD. TEE is the preferred technique that can be done at the bedside and used for intraoperative visualization. The key drawback is the operator experience. However, if the patient is unstable, TEE is the recommended procedure. Other indications for TEE include renal insufficiency and contrast allergy. Findings may include:

- Dissection flap with differential Doppler flow
- True and false lumen in the ascending aorta
- Thrombosis in the false lumen
- Central displacement of intimal calcification
- Pericardial effusion



## Treatment:

- Once the diagnosis of AAD is confirmed or highly suspected, urgent cardiothoracic or vascular surgery consultation should be obtained, regardless of the dissection location. Acute dissections involving the ascending aorta are considered surgical emergencies. The patient should have an arterial line and central venous catheter for monitoring. A foley should be inserted to assess urine output.
- Adequate analgesia (morphine is the preferred analgesic, as it decreases sympathetic output as well)
- Short-acting IV beta-blockers aiming for a heart rate of ~60 bpm (reductions in heart rate and blood pressure reduces aortic wall tension and limit the extent of dissection).
- In patients with contraindications to b-blockade non-dihydropyridine calcium channel blockers can be used for rate control. If the systolic blood pressure remains elevated, nitroprusside can be added to achieve a systolic blood pressure goal of 100 to 120 mmHg (maintain blood pressure in this range as long as there is no compromise of mentation or urine output). Other agents that can be used to lower blood pressure include esmolol, labetalol, or diltiazem.

Surgical therapy for type A AAD involves excision of the intimal tear, obliteration of entry into the false lumen proximally, and reconstitution of the aorta with the interposition of a synthetic vascular graft.

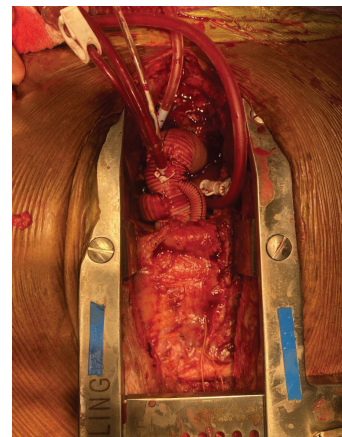
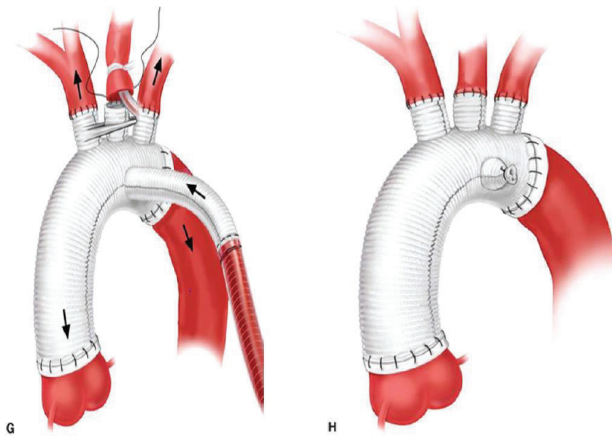
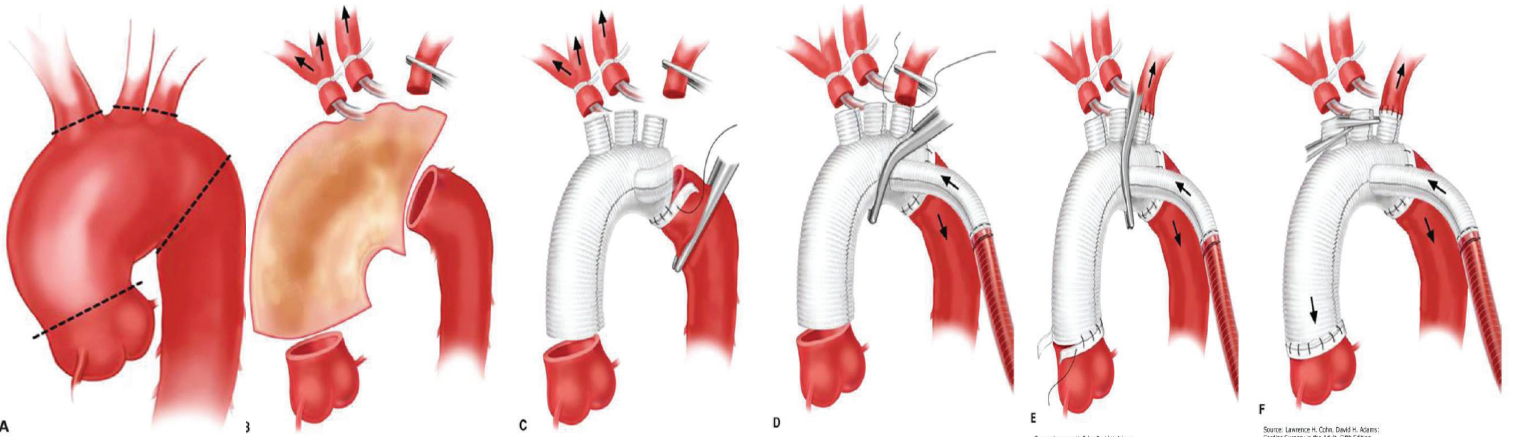
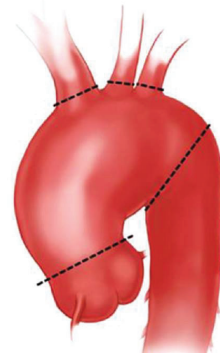


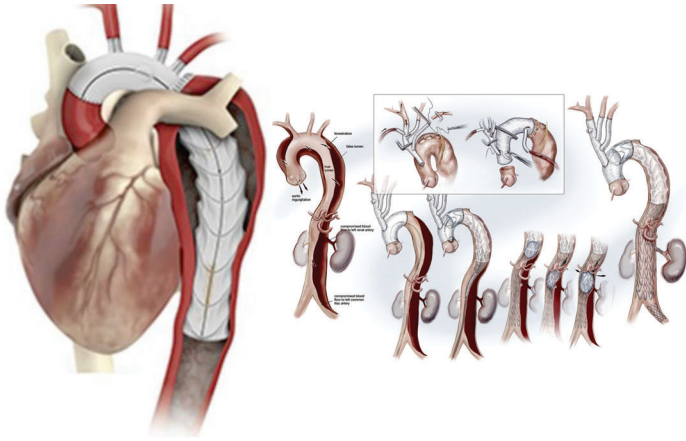
- Valve sparing aortic root reimplantation using Dacron graft with Premade Sinus of Valsalva segment.

# AORTIC ARCH REPLACEMENT



- Composite root replacement with mechanical prosthesis





Surgical intervention for type B AAD tends to be reserved for patients who have a complicated course. Endovascular stent-grafting (TEVAR) has been employed as a less invasive alternative to surgery, primarily for patients with complicated type B dissections

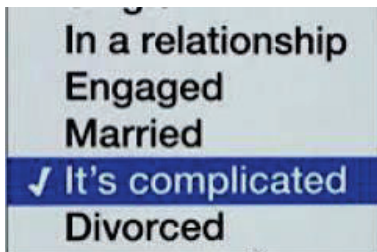


## Current Therapies for Aortic Dissection

Gerardo Gonzalez-Guardiola, MD  
Assistant Professor  
Department of Vascular & Endovascular Surgery  
University of Texas Southwestern

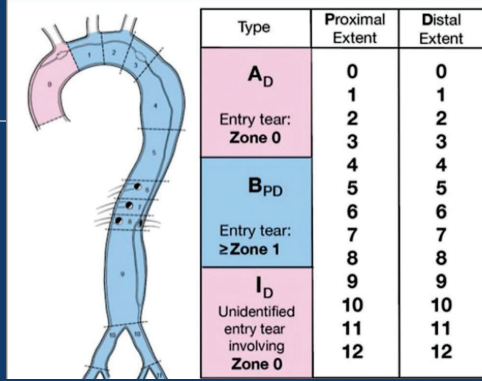


## Perspective on Dissections



- Acute aortic dissection is the most common catastrophic event affecting the aorta
- Mortality of acute dissection (type A and B) left untreated will exceed 22.7% within 6 hours, 50% within 24 hours, and 68% within the first week.
- Follow-up mortality after type B dissection exceeds the follow-up mortality seen after acute type A aortic dissection and exceeds the cumulative incidence of mortality in other diseases such as coronary artery disease, moderate chronic obstructive pulmonary disease, and stage II colon cancer.

Uncomplicated type B dissection has a 30d mortality rate of 10% and 5yr mortality 48%.



Anatomic reporting of aortic dissection based on the SVS/STS reporting standards. Lombardi et al

TABLE 1. SVS/STS CHRONICITY CLASSIFICATION FOR AORTIC DISSECTION

Chronicity Classification	Time From Symptom Onset
Hyperacute	< 24 h
Acute	1-14 d
Subacute	15-90 d
Chronic	> 90 d

Abbreviations: STS, Society for Thoracic Surgery; SVS, Society of Vascular Surgery. Reprinted from Journal of Vascular Surgery, 71/3, Lombardi JV, Hughes GC, Appoo JJ, et al, Society for Vascular Surgery (SVS) and Society of Thoracic Surgeons (STS) reporting standards for type B aortic dissections, 723-747, 2020, with permission from Elsevier.

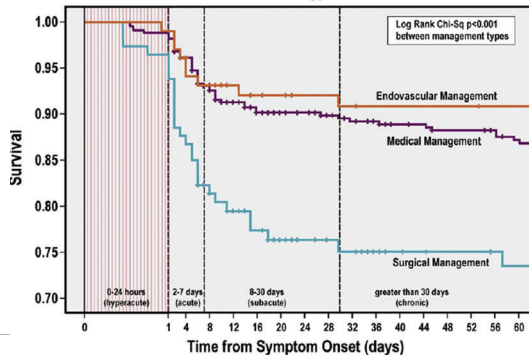
### Management of Acute Type B Aortic Dissection

- Timely diagnosis : CTA Aorta
- **TEMPORIZING** prompt control of blood pressure
- Early recognition of the malperfusion syndrome ( pulses/abdominal/neurological examination, serial creatinine, lactate)
- **Early re-imaging**
- A complication-specific approach via surgical and endovascular options to treat malperfusion syndromes remains the standard of care.

### Management of Acute Type B Aortic Dissection

- The management approach for acute type B aortic dissections has changed over the last decade.
- Contributions from multicenter collaborations such as The **International Registry of Acute Aortic Dissection (IRAD)**, the **Investigation of Stent Grafts in Aortic Dissection (INSTEAD)** trial, and large single institution series have **cast doubt on the ability of medical therapy alone to effectively treat acute type B aortic dissections.**
- **STABLE Trial**

Kaplan-Meier Survival Curve  
Dissection Type: B



### ATBD ≠ Medical Management

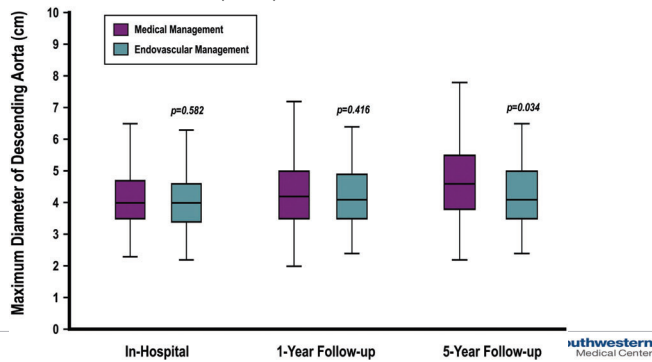
#### Criteria for High Risk TBAD

- Total Aortic Diameter > 4.5 cm
- Rupture/Impending Rupture
- Malperfusion Syndromes
- Early Aortic Expansion

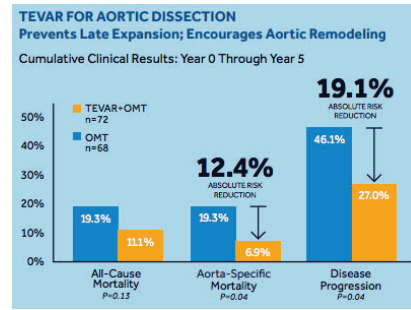
#### Criteria for Mild/Moderate RISK TBAD

- Uncontrolled Hypertension
- Ongoing Pain
- Large Entry Size (>15mm)
- False Lumen Size (> 22 mm)

Survival After Endovascular Therapy in Patients With Type B Aortic Dissection: A Report From the International Registry of Acute Aortic Dissection (IRAD)



The Investigation of Stent Grafts in Aortic Dissection (INSTEAD) Trial 5-Year Long-Term Results

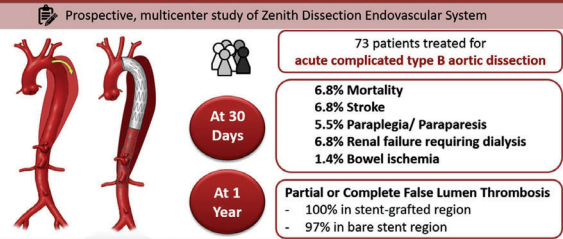


Crossover rate of 20% to the TEVAR + OMT Arm @ 2 yrs

Summary

- Providing the **correct therapy** to the **correct patient** at the **correct time** remains the central questions for escalating beyond optimal medical therapy
- All dissections should be considered as **complicated aortic dissection**
- The role for TEVAR of **low risk Type B Dissection** warrants further investigation but intervention within several months of disease onset might promote positive aortic remodeling and decrease long term mortality/complications

STABLE II Clinical Trial On Endovascular Treatment Of Acute Complicated Type B Aortic Dissection With Composite Device Design



Lombardi et al. *J Vasc Surg*, April 2020  
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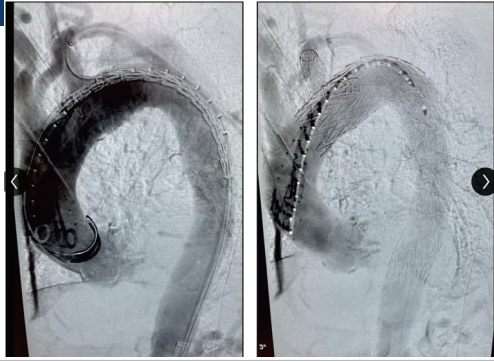
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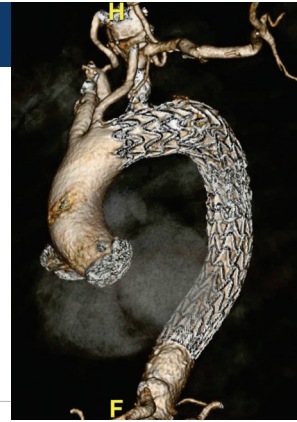


55 Y M with expanding aneurysmal degeneration of Type B Dissection

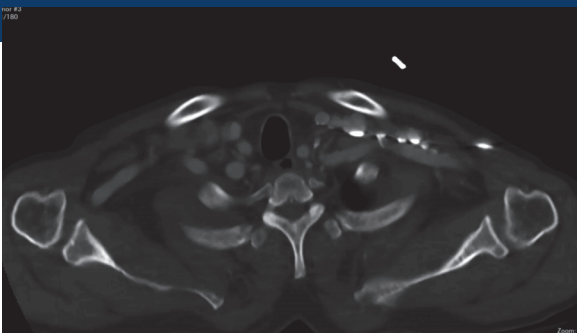




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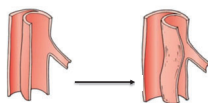
Questions



Pathogenesis of Malperfusion Syndromes

- End organ ischemia secondary to aortic branch compromise
- True lumen collapses and false lumen expands

• Dynamic Obstruction



• Static Obstruction

