2024 MID-ATLANTIC CONFERENCE

12th ANNUAL CURRENT CONCEPTS IN

VASCULAR THERAPIES

2024

Hilton Virginia Beach Oceanfront
Virginia Beach, Virginia

APRIL 18-20

2024 MID-ATLANTIC CONFERENCE

12th ANNUAL CURRENT CONCEPTS IN VASCULAR THERAPIES

2024

Sid Bhende MD

Sentara Vascular Specialists

April 19th 2024

Managing Comorbidities in Atherosclerotic Patients

Disclosures

Outline

1. *WHAT?*

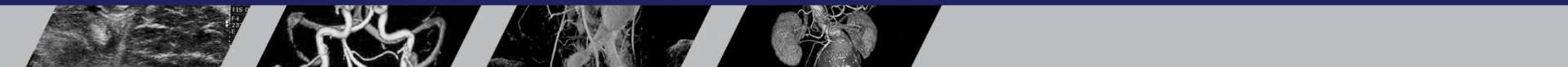
   What comorbidities do atherosclerotic patients develop?

2. *HOW?*

   How do we manage these comorbidities?

3. *WHY?*

   Why do we need to manage these comorbidities?

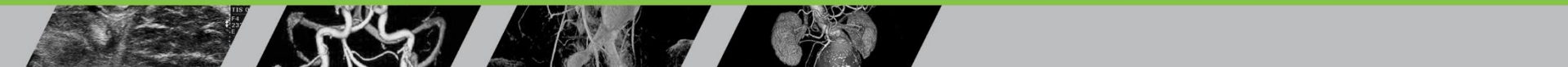


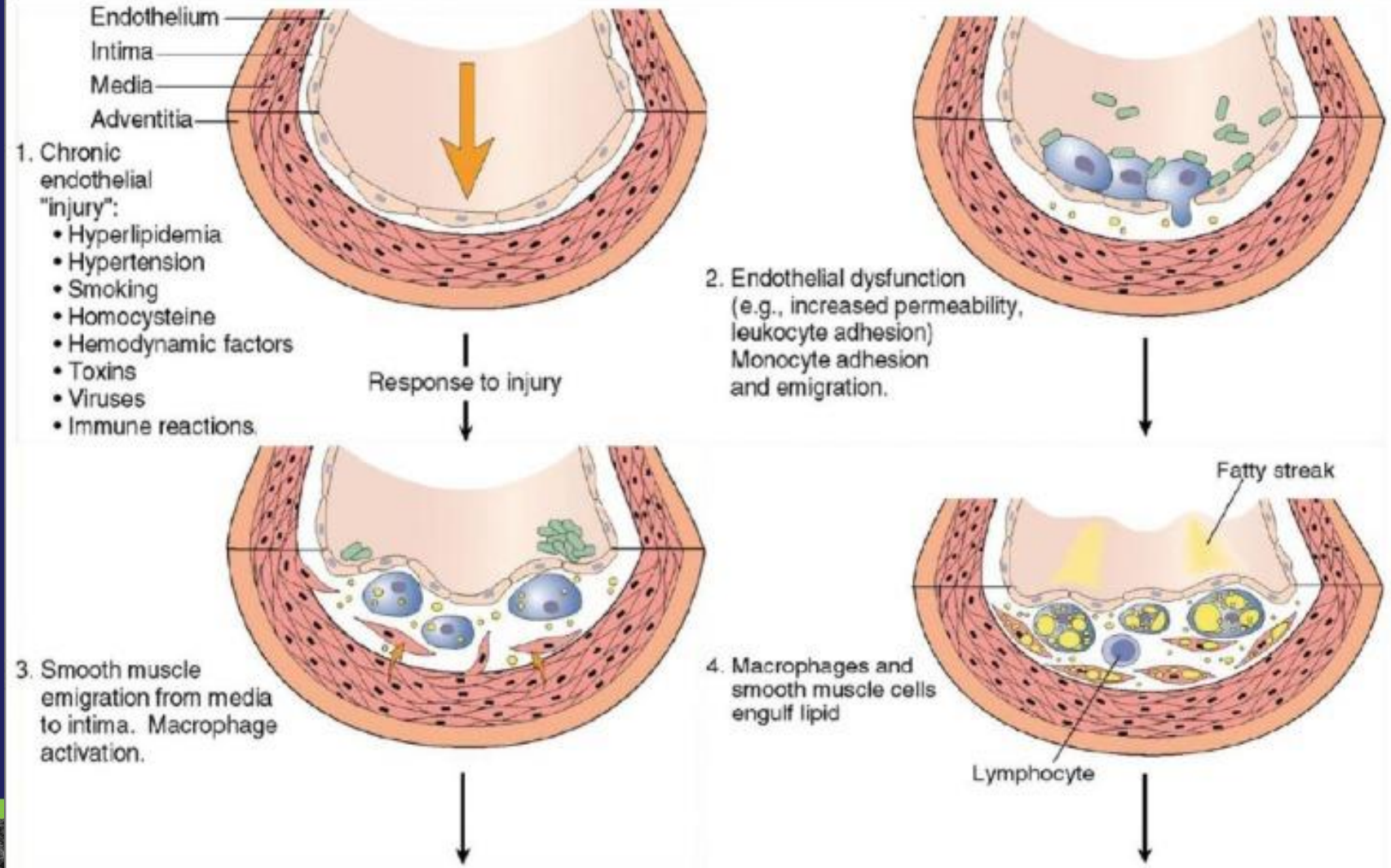
What is Atherosclerosis?

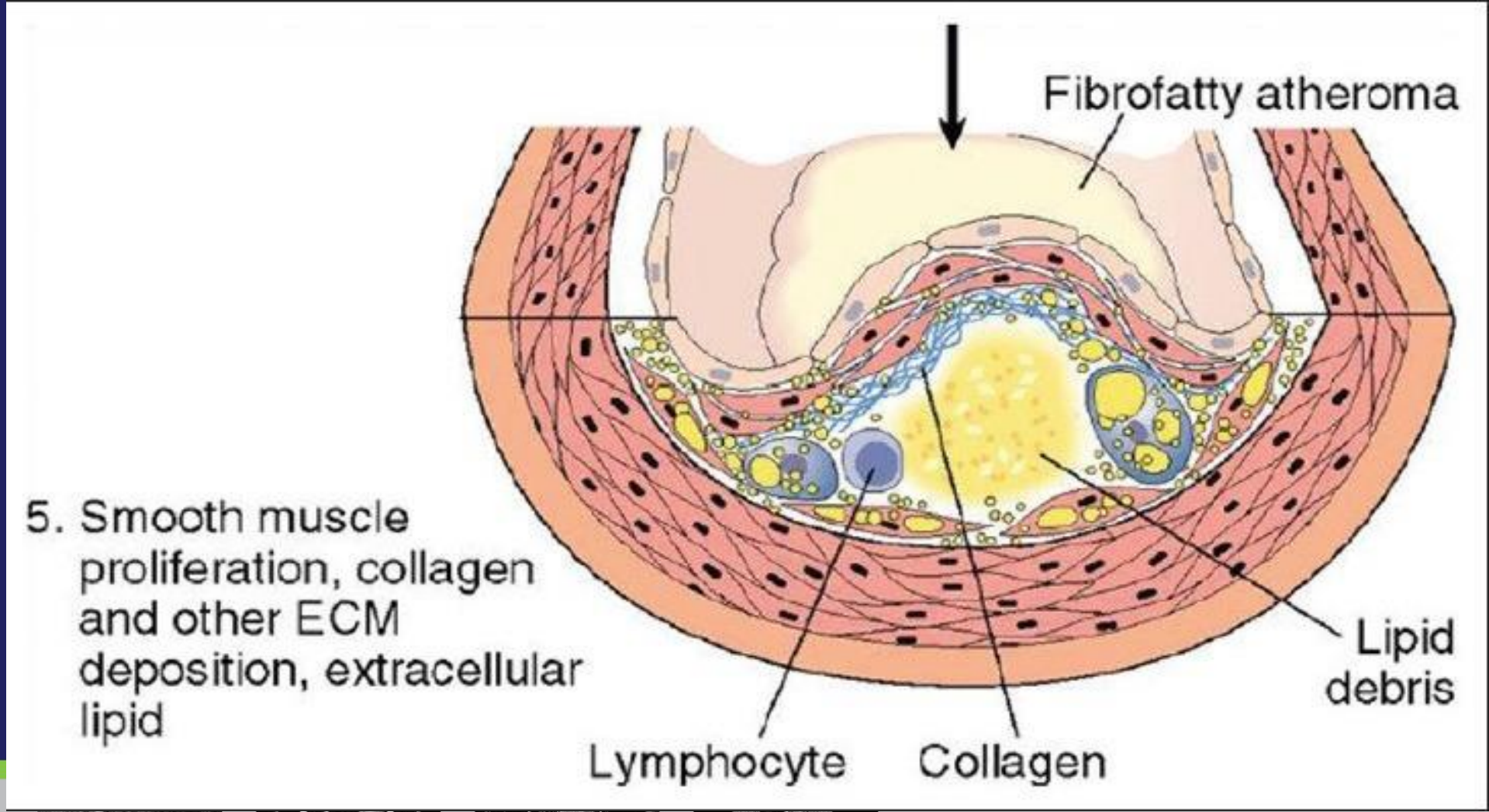
- Atherosclerosis is the process by which plaque builds up in the inner lining of the blood vessel
- Plaque consists of cholesterol, cellular waste calcium, fibrin
- It can affect any artery in the body
- It is an IRREVERSIBLE process

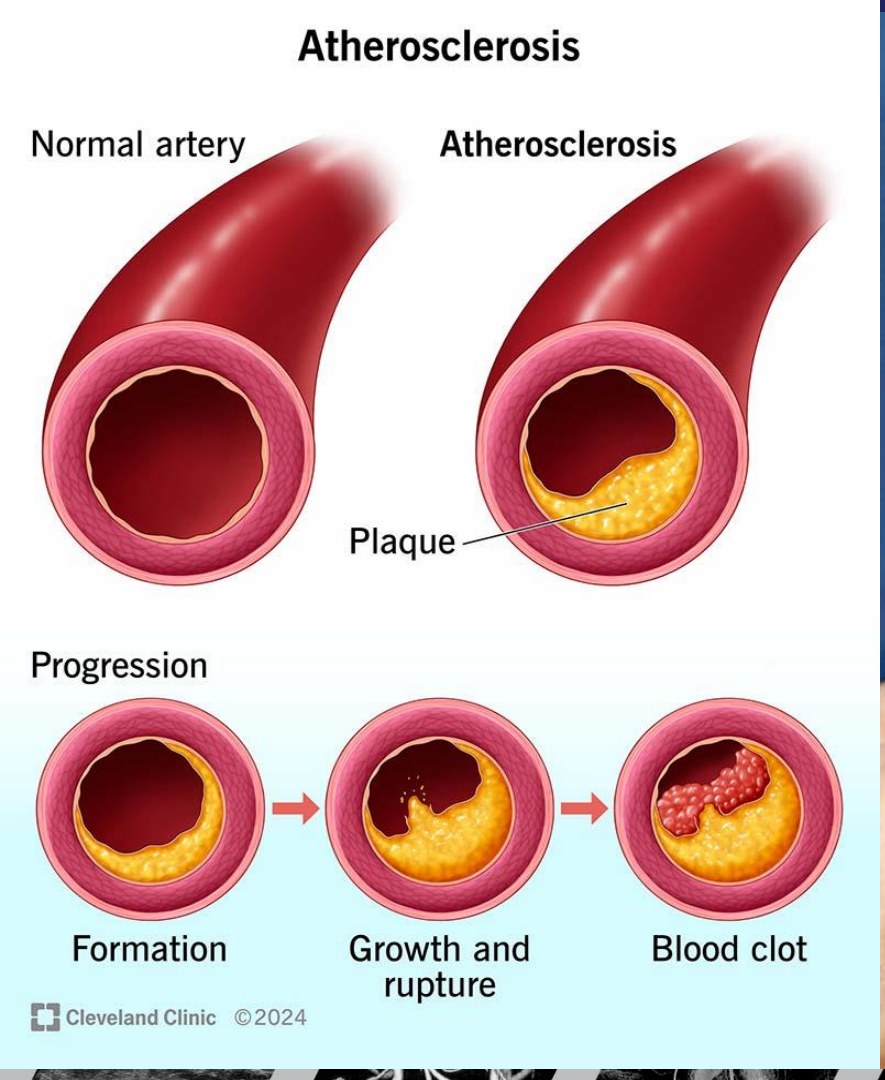
Pathogenesis of Atherosclerosis?

1. Endothelial dysfunction
2. Formation of lipid layer “fatty streak” within the intima
3. Migration of leukocytes and smooth muscle cells into the vessel wall
4. Foam cell formation
5. Degradation of extracellular matrix




Endothelium
Intima
Media
Adventitia
1. Chronic endothelial "injury":
• Hyperlipidemia
• Hypertension
• Smoking
• Homocysteine
• Hemodynamic factors
• Toxins
• Viruses
• Immune reactions
Response to injury
2. Endothelial dysfunction (e.g., increased permeability, leukocyte adhesion) Monocyte adhesion and emigration.
3. Smooth muscle emigration from media to intima. Macrophage activation.
4. Macrophages and smooth muscle cells engulf lipid
Fatty streak
Lymphocyte


Fibrofatty atheroma
5. Smooth muscle proliferation, collagen and other ECM deposition, extracellular lipid
Lipid debris
Lymphocyte
Collagen


Atherosclerosis
Normal artery
Atherosclerosis
Plaque
Progression
Formation
Growth and rupture
Blood clot
Cleveland Clinic ©2024

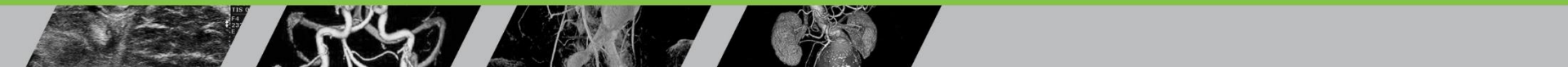

Retina Image Bank

Risk Factors of Atherosclerosis?

1. Hypertension
   - ➢ High pressure causing stress on vessel wall resulting in endothelial dysfunction
2. Diabetes Mellitus
   - ➢ Hyperglycemia causes increased inflammatory response leading to endothelial dysfunction

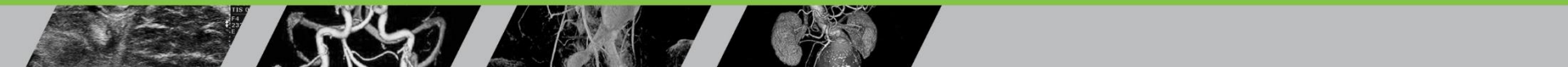
Risk Factors of Atherosclerosis?

3. Dyslipidemia
   - Increased Cholesterol/LDL/Triglycerides in the blood stream will get deposited in the intima causing endothelial dysfunction
4. Smoking/Tobacco use
   - Chemicals and toxins in tobacco cause systemic inflammation leading to endothelial dysfunction



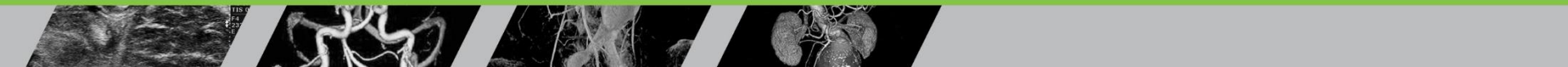
Risk Factors of Atherosclerosis?

5. Age – Male > 45 and Female > 55
   - Increased expression of leukocytes in aged vessels which trigger monocyte migration and inflammation which promotes atherosclerosis
6. Family Hx of Premature CVD is defined as a biologic family member with CVD < 45 yrs
   - Genetic mutations causing increase plasma lipoprotein levels leading to endothelial dysfunction



Risk Factors of Atherosclerosis?

7. Obesity – BMI > 30
   - Increase in sympathetic nerve activation which impairs arterial baroreceptor function leading to hypertension which induces endothelial dysfunction
8. Physical Inactivity
   - Inactivity increases NAPDH oxidase which contributes to endothelial dysfunction




Age and gender
Physical inactivity
Genetics
Diet
Diabetes
Atherosclerosis
Environment pollutants
Hyper cholesterolemia
Smoking
Hypertension
Alcohol

Outline

1. ~~*WHAT?*~~

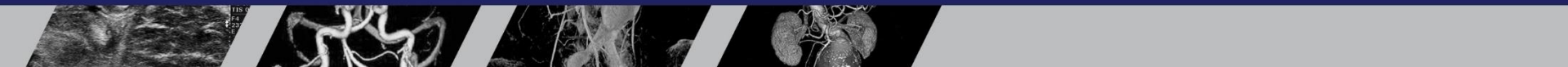
   ~~What comorbidities do atherosclerotic patients develop?~~

2. *HOW?*

   How do we manage these comorbidities?

3. *WHY?*

   Why do we need to manage these comorbidities?

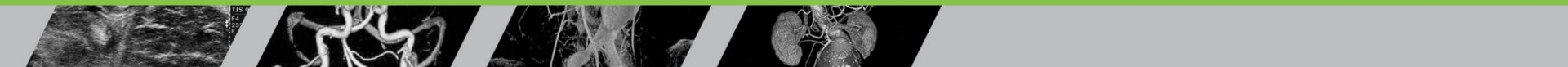


Management of Atherosclerosis?

1. Must be a multispecialty team approach
   - Primary Care Physician
   - Vascular Surgeon
   - Cardiologist
   - Nephrologist
   - Endocrinologist
   - Podiatrist

Management of Atherosclerosis?

1. The goal is to prevent or delay the atherosclerotic process by risk factor modification
   - Modifiable Risk Factors
     - Hypertension, DM, Dyslipidemia, Tobacco Use, Obesity, Physical Inactivity
   - Unmodifiable Risk Factors
     - Family Hx, Age



Modifiable Risk Factors

1. Hypertension

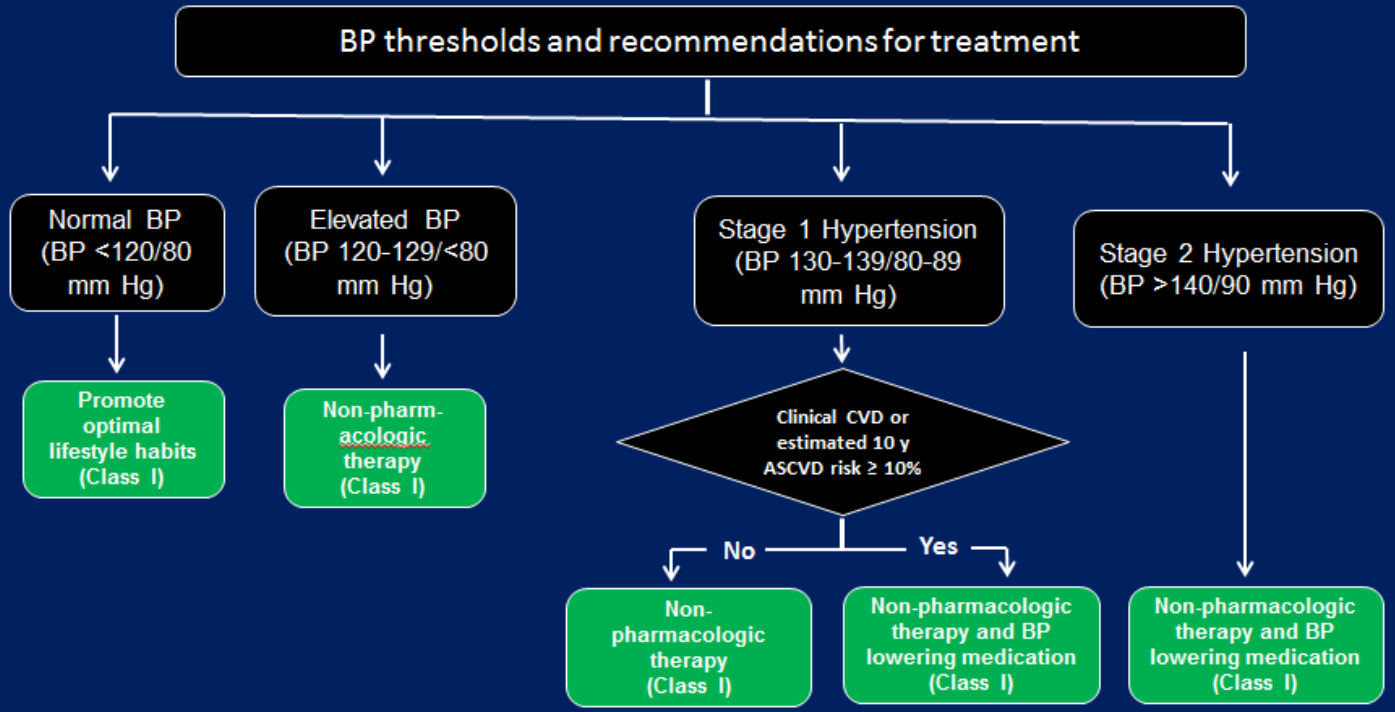
2017 Guideline for the Prevention, Detection, Evaluation and Management of High Blood Pressure in Adults

***BP Classification** (JNC 7 and ACC/AHA Guidelines)*

SBP		DBP	JNC 7	2017 ACC/AHA
<120	and	<80	Normal BP	Normal BP
120–129	and	<80	Prehypertension	Elevated BP
130–139	or	80–89	Prehypertension	Stage 1 hypertension
140–159	or	90-99	Stage 1 hypertension	Stage 2 hypertension
≥160	or	≥100	Stage 2 hypertension	Stage 2 hypertension

- *Blood Pressure should be based on an average of ≥2 careful readings on ≥2 occasions*
- *Adults being treated with antihypertensive medication designated as having hypertension*

BP THRESHOLDS AND RECOMMENDATIONS FOR TREATMENT



Modifiable Risk Factors

1. Hypertension
   - ESH (2023) and ACC (2017) guidelines recommend BP < 130/80 to reduce risk of CV events
   - If BP elevated then utilize diet, exercise and Meds (ACEi, ARBs, CCB, Diuretics)

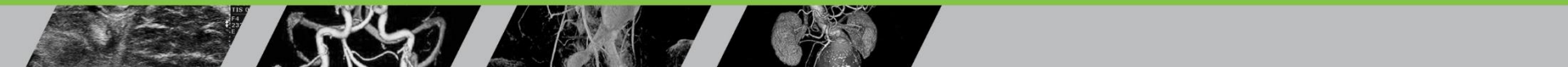
Modifiable Risk Factors

2. Diabetes Mellitus

TABLE 2

DIAGNOSING DIABETES

	A1C (percent)	Fasting Plasma Glucose (mg/dL)	Oral Glucose Tolerance Test (mg/dL)
Diabetes*	6.5 or above	126 or above	200 or above
Prediabetes	5.7 to 6.4	100 to 125	140 to 199
Normal	About 5	99 or below	139 or below



GLUCOSE-CENTRIC ALGORITHM FOR GLYCEMIC CONTROL

LIFESTYLE INTERVENTION

Start or continue metformin if appropriate[1]

INDIVIDUALIZE GLYCEMIC TARGET
A1C ≤6.5% for most persons or 7%-8% if high risk for adverse consequences from hypoglycemia and/or limited life expectancy

	Overweight or Obesity[2]	Hypoglycemia Risk[3]	Access / Cost	Severe Hyperglycemia[4]	Patients may present with >1 scenario
Preferred	GLP-1 RA or GIP/GLP-1 RA or SGLT2i	GLP-1 RA or GIP/GLP-1 RA or SGLT2i	TZD or SU/GLN	Basal Insulin[5] + Prandial Insulin or + GLP-1 RA | GIP/GLP-1 RA[6]	Order of medications suggests hierarchy for selection[7]
Alternatives	DPP-4i[8] or TZD[9]	DPP-4i[8] or TZD	Insulin or DPP-4i[10]	Basal Insulin + other agent(s)	A1C >7.5% start 2 agents, A1C >9.0% or >1.5% above goal start 2-3 agents
Concerns or Not Preferred	Avoid SU/GLN	Avoid SU/GLN	GLP-1 RA | GIP/GLP-1 RA | SGLT2i | COLSVL BRC-QR	Other agents likely ineffective in the setting of glucotoxicity[5]	

Titrate to maximum tolerated dose. If not at glycemic target at ≤3 months, add best available agent not in use[7]
GLP-1 RA | GIP/GLP-1 RA | SGLT2i | TZD | DPP-4i | SU/GLN | COLSVL | BRC-QR | PRAML[11]

IF NOT AT GOAL: CONTINUE TO ALGORITHM FOR ADDING/INTENSIFYING INSULIN

[1]Take with food with dose titration for enhanced tolerance. [2]See also COMPLICATIONS-CENTRIC MODEL FOR THE CARE OF PERSONS WITH OVERWEIGHT/OBESITY and PROFILES OF WEIGHT-LOSS MEDICATIONS table. [3]Evaluate for issues leading to hypoglycemia or hypoglycemia unawareness and manage with patient-centered strategies. [4]If A1C >10% and/or BG ≥300 with symptomatic hyperglycemia, reduce glucose/A1C as promptly and safely as possible. [5]See also ALGORITHM FOR ADDING/INTENSIFYING INSULIN. [6]GLP-1 RA requires titration phase which can delay glycemic control. After glucose toxicity is resolved,consider adding other agents. [7]See also PROFILES OF ANTIHYPERGLYCEMIC MEDICATIONS table. [8]GLP-1 RA and DPP-4i should not be combined. [9]TZD can cause fluid retention but have benefit for NAFLD, CVD prevention, dyslipidemia. [10]Access/Cost are dependent on location of the market. Insulin costs vary widely with devices (e.g., pens versus vials) and formulations (e.g., analogues versus combinations such as 70/30). [11]PRAML is used as an adjunct with prandial insulin.

Algorithm Figure 7-Glucose-Centric Glycemic Control

AACE

Modifiable Risk Factors

3. Dyslipidemia
   - Lipid panel
     - Cholesterol < 200
     - Triglyceride < 150
     - LDL < 100
     - HDL > 40
   - Need to calculate 10 yr ASCVD risk score

Carl U. Lee
45 yrs M DOB: 8/30/1971

ASCVD Risk Calculator

Results | Risk Factors | Recommendations

Sex
Male | Female

Age
45

Race
- White
- African American
- Other

Total Cholesterol (mg/dL)
141

HDL - Cholesterol (mg/dL)
34

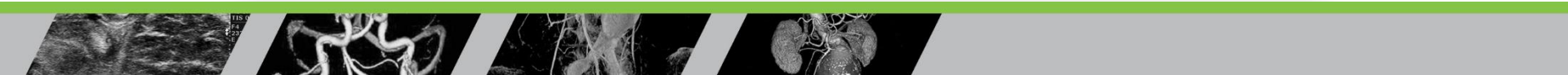
Systolic Blood Pressure
150

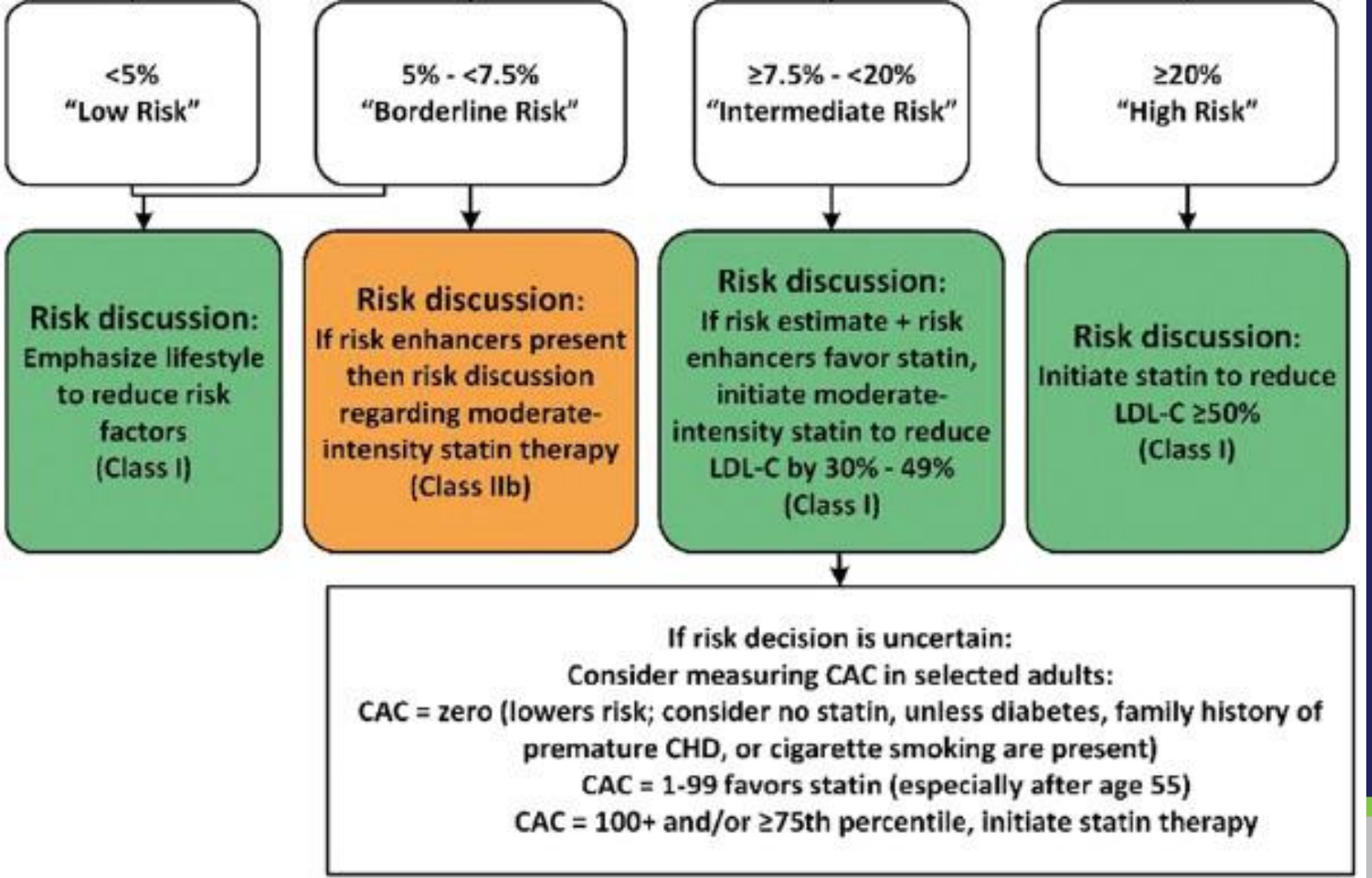
Diabetes
No | Yes

Current Smoking
No | Yes

Treatment for Hypertension
No | Yes

Update Risk Score



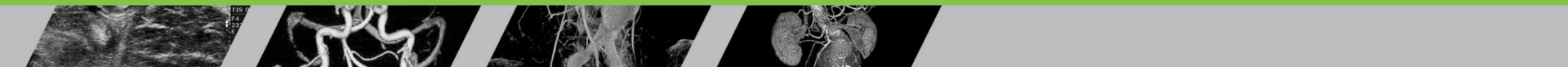

<5%
"Low Risk"
5% - <7.5%
"Borderline Risk"
≥7.5% - <20%
"Intermediate Risk"
≥20%
"High Risk"
Risk discussion:
Emphasize lifestyle to reduce risk factors
(Class I)
Risk discussion:
If risk enhancers present then risk discussion regarding moderate-intensity statin therapy
(Class IIb)
Risk discussion:
If risk estimate + risk enhancers favor statin, initiate moderate-intensity statin to reduce LDL-C by 30% - 49%
(Class I)
Risk discussion:
Initiate statin to reduce LDL-C ≥50%
(Class I)
If risk decision is uncertain:
Consider measuring CAC in selected adults:
CAC = zero (lowers risk; consider no statin, unless diabetes, family history of premature CHD, or cigarette smoking are present)
CAC = 1-99 favors statin (especially after age 55)
CAC = 100+ and/or ≥75th percentile, initiate statin therapy

Modifiable Risk Factors

3. Dyslipidemia
   - If statin intolerance (inability to achieve therapeutic targets on maximum statin dosage or muscle related symptoms) then consider PCSK9 inhibitors (Repatha & Praluent)

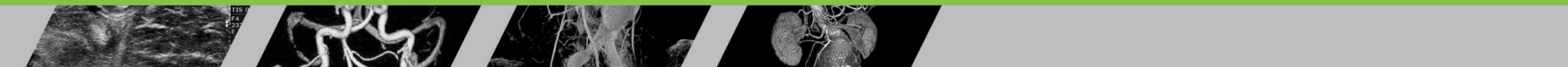
Modifiable Risk Factors

3. Dyslipidemia
   - Do you initiate statin therapy for patients who have normal lipid panel and evidence of atherosclerosis?
   - Absolutely! There is ample evidence that patients with evidence of atherosclerosis (asymptomatic or symptomatic) should be on statin therapy to reduce CV risk



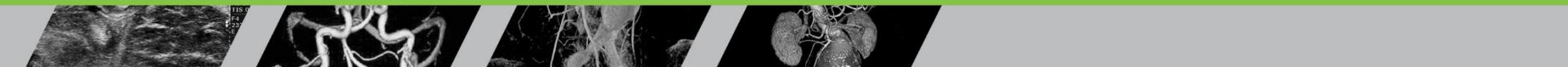
Modifiable Risk Factors

4. Tobacco Use
   - Must quit!
   - Smoking cessation has shown to decrease the progression of atherosclerosis
   - Will quitting smoking reverse atherosclerosis?
     - No, however there is lower risk of PAD, CAD and stroke within 5 yrs of smoking cessation



Modifiable Risk Factors

4. Tobacco Use
   - What about vaping?
     - E-cigarettes are associated with several noxious compounds - nicotine, propylene glycol, particulate matter, heavy metals, and flavorings, that lead to atherosclerosis progression

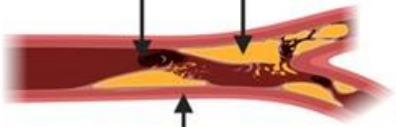


Cigarette

Nicotine
Carbon monoxide
Benzene
VOCs
Tar
Metals

Atherosclerosis

Thrombosis

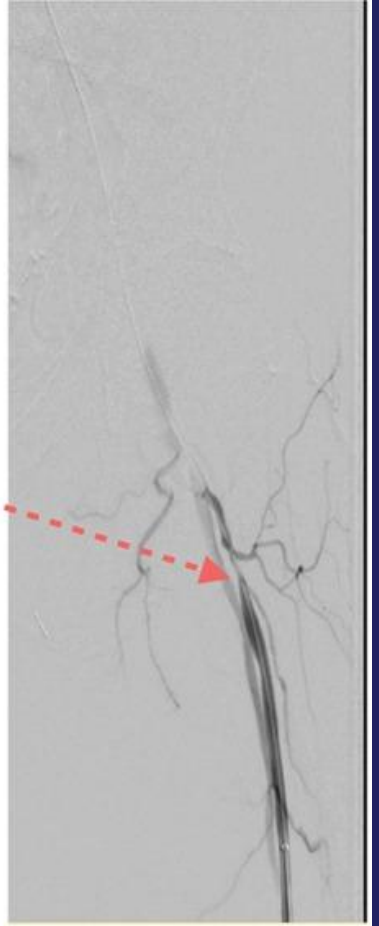


Endothelial Injury

?

Nicotine
Glycerol
Flavouring
Propylene glycol
Ultrafine particles
Heavy metals
VOCs

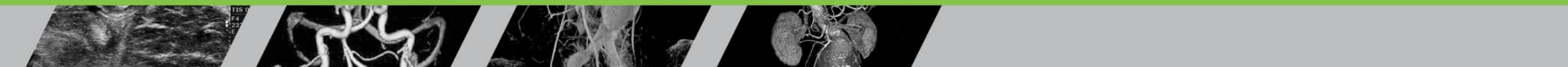
Vape (e-Cigarette)



Angiogram with stenotic lesion (red arrow)

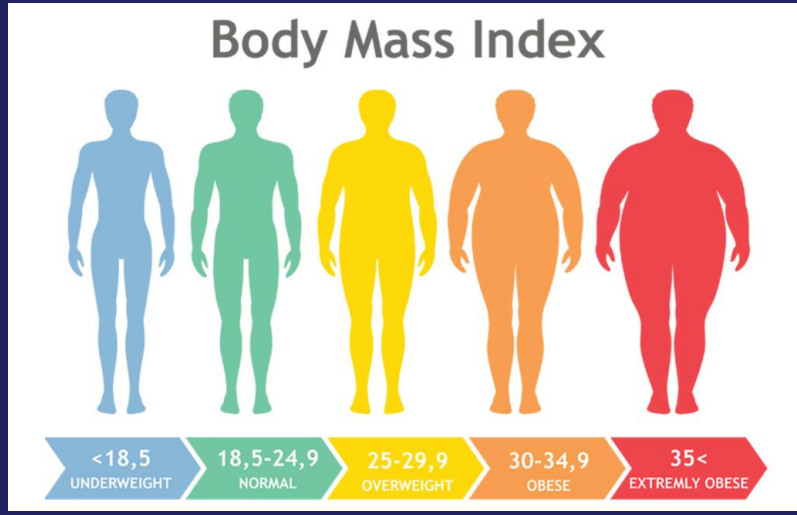
Modifiable Risk Factors

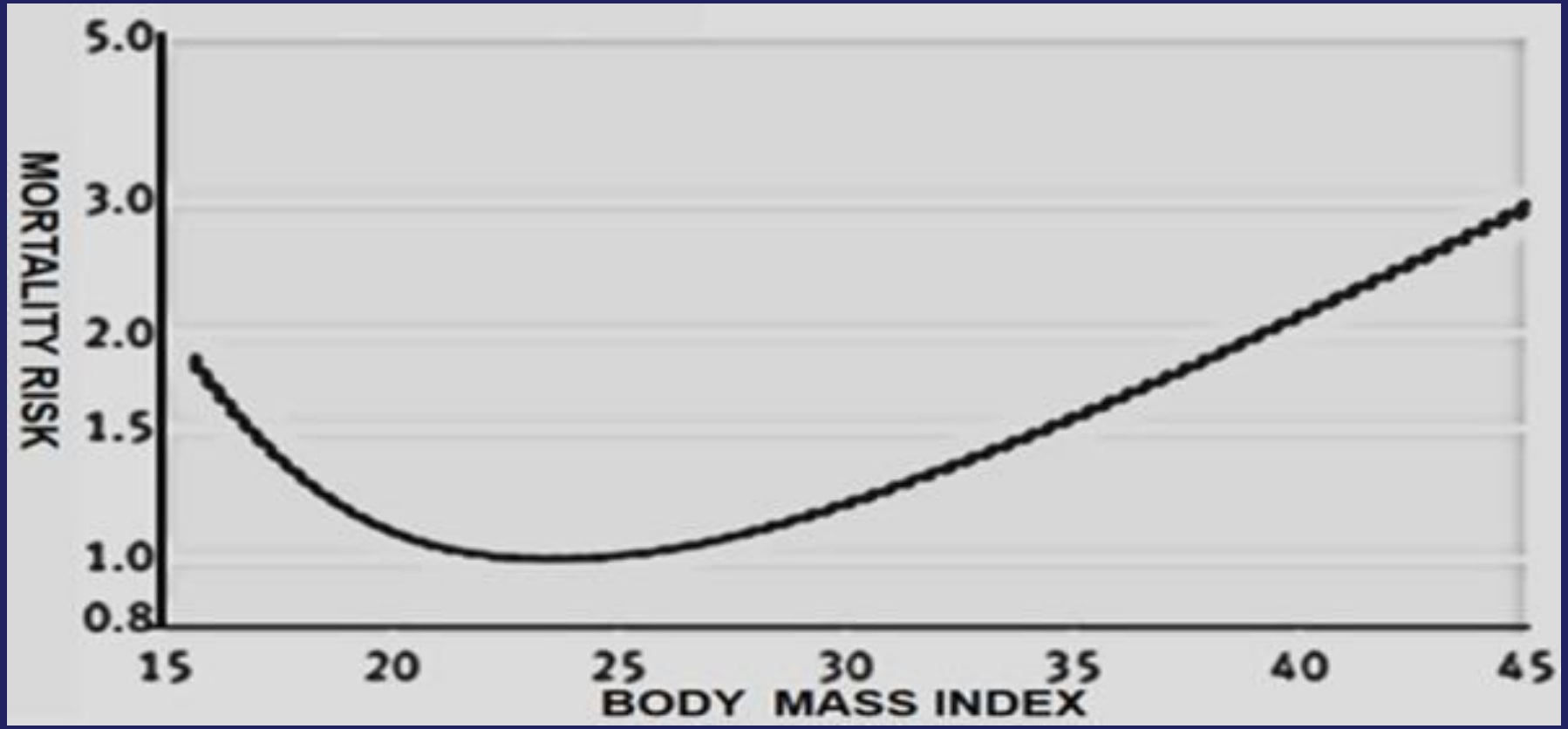
4. Tobacco Use
   - What about smoking marijuana?
     - No convincing evidence yet that smoking marijuana causes atherosclerosis or any CV mortality



Modifiable Risk Factors

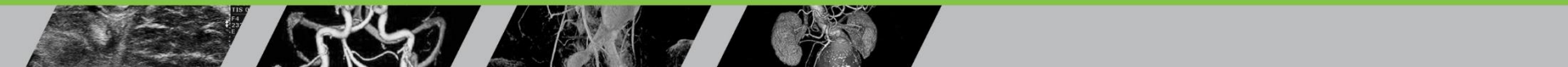
5. Obesity
   - 5 categories based on BMI
     - Underweight - BMI < 18.5
     - Normal weight – BMI 18.5-24.9
     - Overweight – BMI 25-29.9
     - Obesity – BMI 30-39.9
     - Morbid Obesity – BMI > 40




MORTALITY RISK
5.0
3.0
2.0
1.5
1.0
0.8
15
20
25
30
35
40
45
BODY MASS INDEX

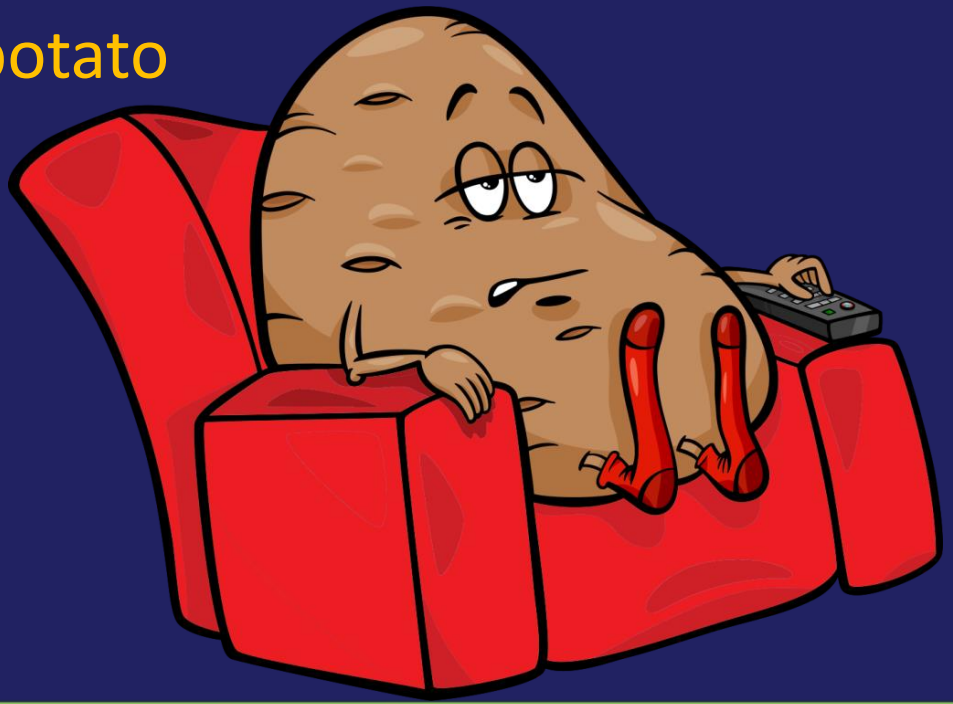
Modifiable Risk Factors

5. Obesity
   - Will weight loss help reduce atherosclerosis?
     - No but you will see decrease in BP, reduction in lipid panel, decrease in BS or complete resolution of DM



Modifiable Risk Factors

6. Physical Inactivity
   - Don’t be a couch potato



Modifiable Risk Factors

6. Physical Inactivity
   - Healthy adults (18-65) should participate in moderate intensity aerobic activity for 30min/day x 5 days or vigorous aerobic activity for 20min/day x 3 days
   - Need to maintain muscle strength for at least 2 days/week

How much activity do I need?

Moderate-intensity aerobic activity

Anything that gets your heart beating faster counts.

AND

Muscle-strengthening activity

Do activities that make your muscles work harder than usual.

Tight on time this week? **Start with just 5 minutes.** It all adds up!

Outline

1. ~~*WHAT?*~~

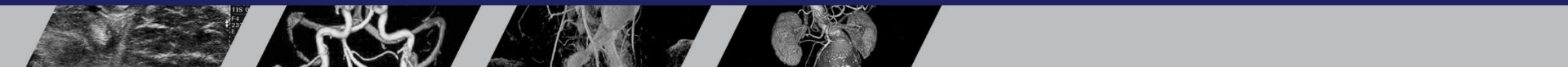
~~What comorbidities do atherosclerotic patients develop?~~

2. ~~*HOW?*~~

~~How do we manage these comorbidities?~~

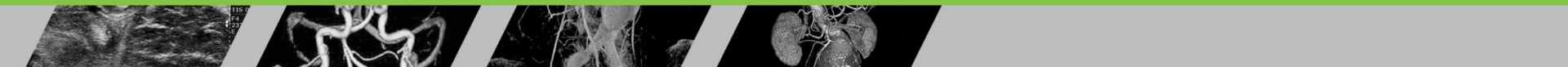
3. *WHY?*

Why do we need to manage these comorbidities?



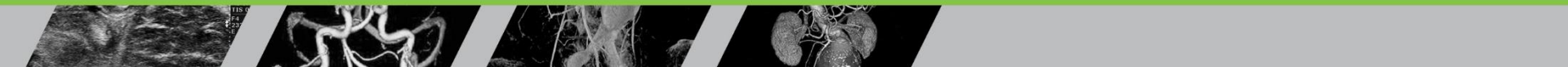
Comorbidities that develop with Atherosclerosis?

- Coronary Artery Disease (MI, HF, Arrythmia)
  - Leading cause of mortality in USA causing 610,000 deaths annually
  - 3rd leading cause of mortality worldwide causing 17.8 million deaths annually
  - Healthcare cost for CAD > 200 billion dollar annually in USA
  - CAD is preventable



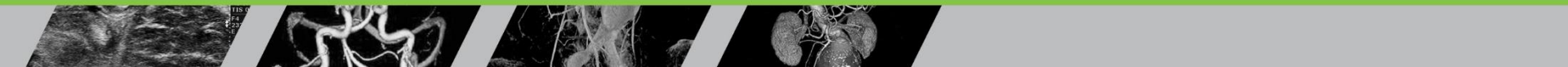
Comorbidities that can develop with Atherosclerosis?

- Carotid Artery Disease (CVA)
  - Stroke – 85% are ischemic, 15% hemorrhagic
  - 15-20% of ischemic strokes come from atherosclerosis of the extra-cranial carotid artery



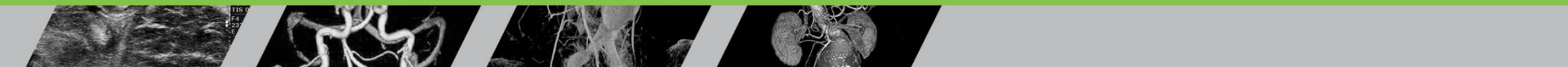
Comorbidities that can develop with Atherosclerosis?

- Peripheral Arterial Disease (Claudication, Rest pain, Ulceration, Gangrene, Amputation)
  - PAD affects over 8 million people in USA
  - PAD affects over 200 million people worldwide
  - 50% of PAD patients are asymptomatic
  - Claudicants – 1% per year risk of amputation
  - CLI – 30% per year risk of amputation



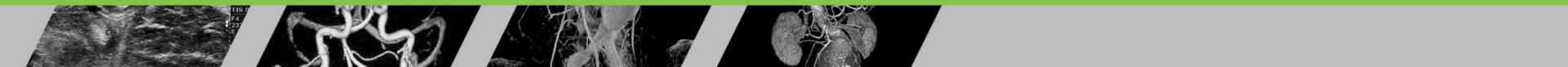
Comorbidities that can develop with Atherosclerosis?

- Aneurysm Formation
  - Thoracic, Aortic, Femoral, Popliteal, Mesenteric, Cerebral
- Mesenteric Angina/Ischemia
- Chronic Kidney Disease/Renal artery stenosis
- Erectile Dysfunction



Summary

- Atherosclerosis
  - Induced by endothelial dysfunction
  - Irreversible process
  - Team Approach
  - Risk factors – HTN, DM, Dyslipidemia, Tobacco Use, Obesity, Physical inactivity, Age, Genetics
  - Comorbidities associated with atherosclerosis are – CAD, Carotid disease, PAD, Aneurysm formation, Mesenteric ischemia, CKD




Thank You
Merci
Obrigado
Dank Je
Salamat
Dyakuyu
Arigato
Asante
Juspaxar
Kiitos
Grazie
Spasibo
Mamana
Multumesc
Maake
Terma Kasih
Chokrane
Matur Nuwun
Raibh Maith Agat
Mochchakkeram
Dankon
Matondo
Obrigado
Ua Tsaug Rau Koj
Welalin
Meri
Kia Ora
Nirringrazzjak
Cam on ban
Vinaka

CHEERS