



HYPERTENSION

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HYPERTENSION

- **INTRODUCTION**
- **EPIDEMIOLOGY**
- **PATHOPHYSIOLOGY**
- **DIAGNOSIS**
- **PATIENT EVALUATION**
- **TREATMENT**



INTRODUCTION

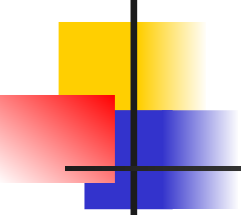
- CVD is currently a world wide epidemic
- Hypertension is a key CVD risk factor
- The communicable diseases epidemic is not yet under control in our country
- We do not have the resources to handle two epidemics
- We need to act quickly to prevent CVD and treat those already affected



Hypertension: definition

- **BP \geq 140/90** is considered as defining term, but hypertension is a **continuum**
- **Cardiovascular risk doubles with every 20/10mmhg rise BP from 115/75mmhg** between age 40 to 70 yrs
- **Isolated systolic/diastolic hypertension** should be treated like any other hypertension, higher figure determines grade
- **White coat hypertension** is also associated with increased cardiovascular risk

Hypertension: definition

- 
- It is an **asymptomatic lifelong** disease **requiring life long treatment**
 - **Progressive** cardiovascular syndrome associated with **target organ damage**
 - It's assoc with increased risk for **stroke, coronary artery disease, heart failure, renal failure, retinopathy** and **peripheral vascular disease**.
 - **Treatment results in reduction** in these complications
 - Major challenges exist in diagnosis and treatment



BP CLASSIFICATION

ESH -ESC category (1)	Systolic BP mmHg	Diastolic BP mmHg	JNC-7 category (2)
Optimal	<120	<80	Normal
Normal	120 – 129	80 – 84	Pre hypertension
High normal	130 – 139	85 – 89	
Grade 1 hypertension (mild)	140 – 159	90 – 99	Stage 1 hypertension
Grade 2 hypertension (moderate)	≥ 160 – 179	≥ 100 – 109	Stage 2 hypertension
Grade 3 hypertension (severe)	180	110	



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Global Burden of Hypertension: Analysis of Worldwide Data

- In 2000, 26.4% (95% CI 26.0-26.8%) of the adult population had hypertension, 26.6% of men & 26.1% of women.
- By 2025, 29.2% (28.8-29.7%) were projected to be hypertensive, 29.0% of men & 29.5% of women.
- In 2000, 972 million adults had hypertension; 333 million (329-336 million) in economically developed countries & 639 million (625-654 million) in economically developing countries.
- By 2025, the number of adults with hypertension was predicted to increase by about 60% to a total of 1.56 billion.

Global Burden of Hypertension: Africa



- In Tanzania, > above 37% in an urban area and more than 26% in a rural population
- Kenya –rural – 33%
- Of all patients with hypertension in Africa, only 1/3 are aware.
- Of those diagnosed only 1/3 are on treatment
- Of those on treatment only 1/3 are controlled



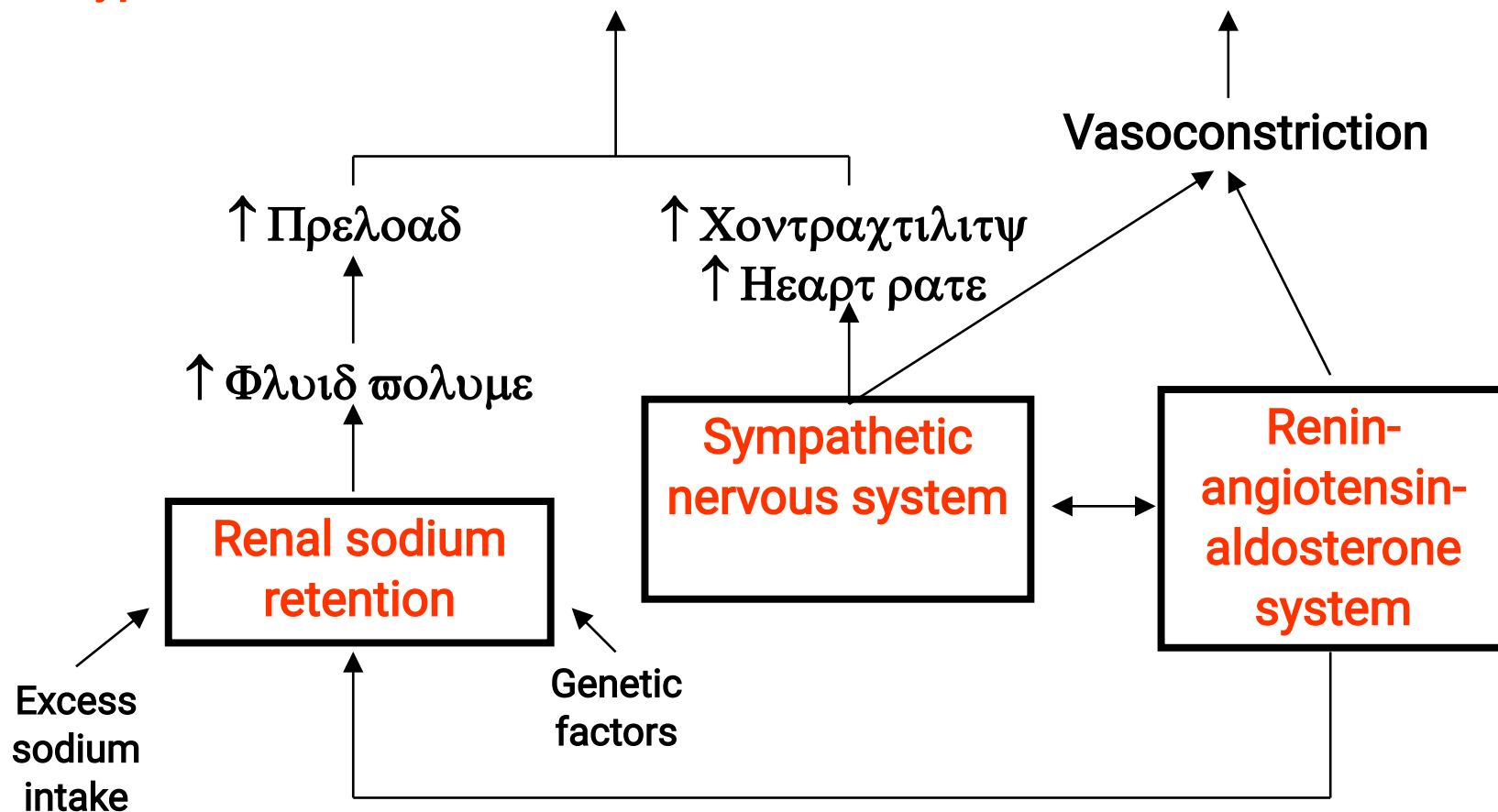
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Control of blood pressure

Blood pressure = Cardiac output x Peripheral resistance

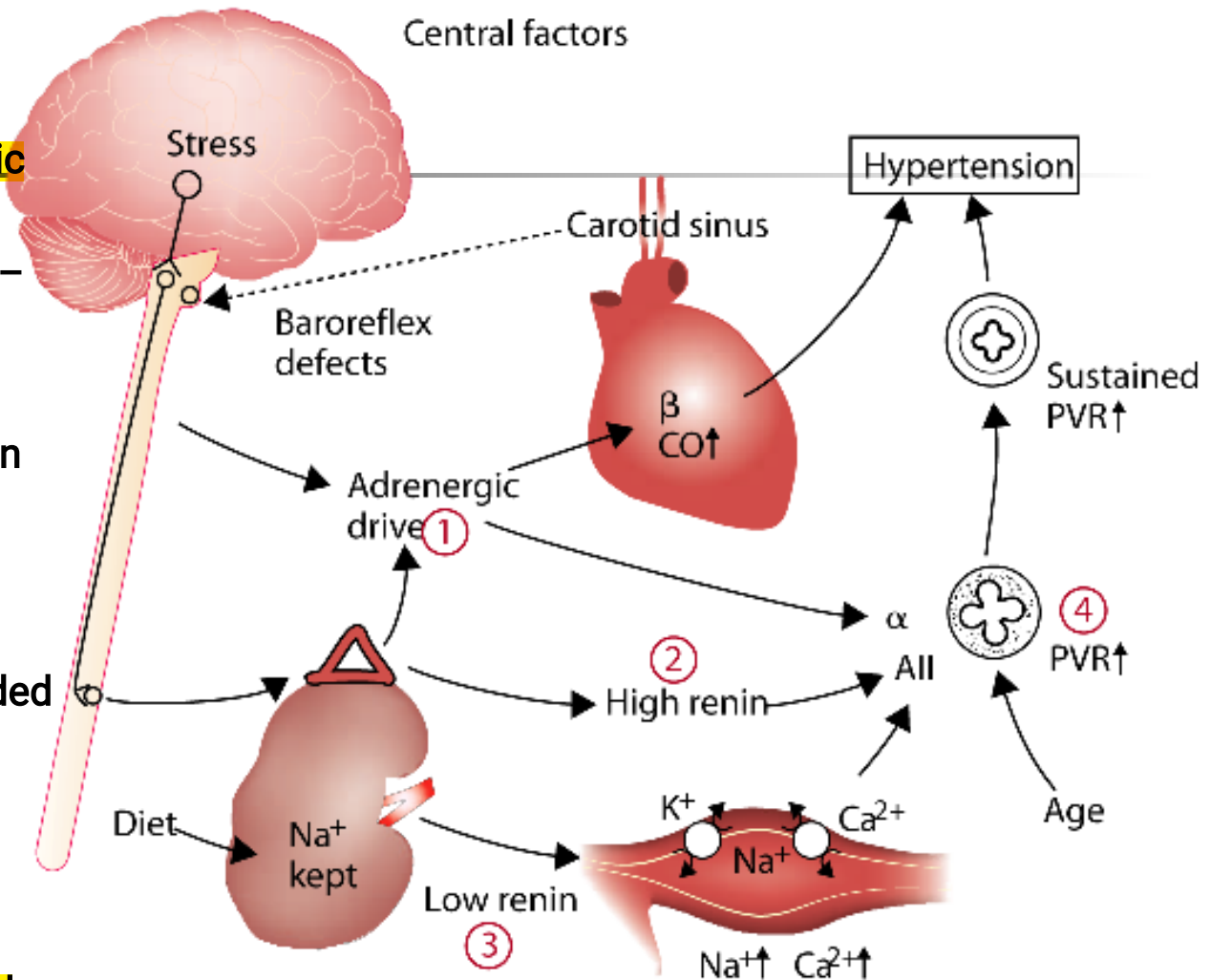
Hypertension = Increased CO and/or Increased PR



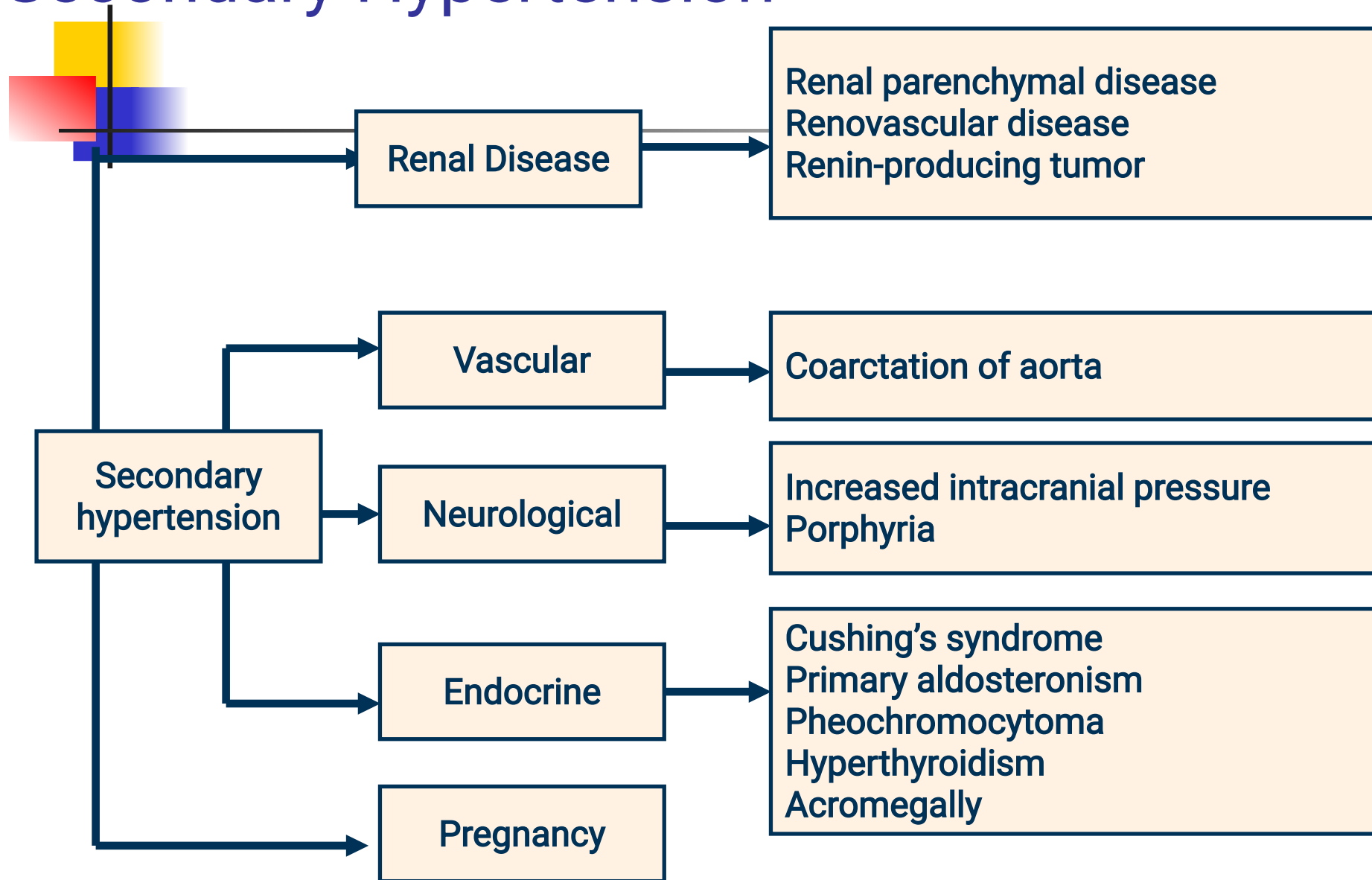
Multifactorial origin of Hypertension

Major mechanisms

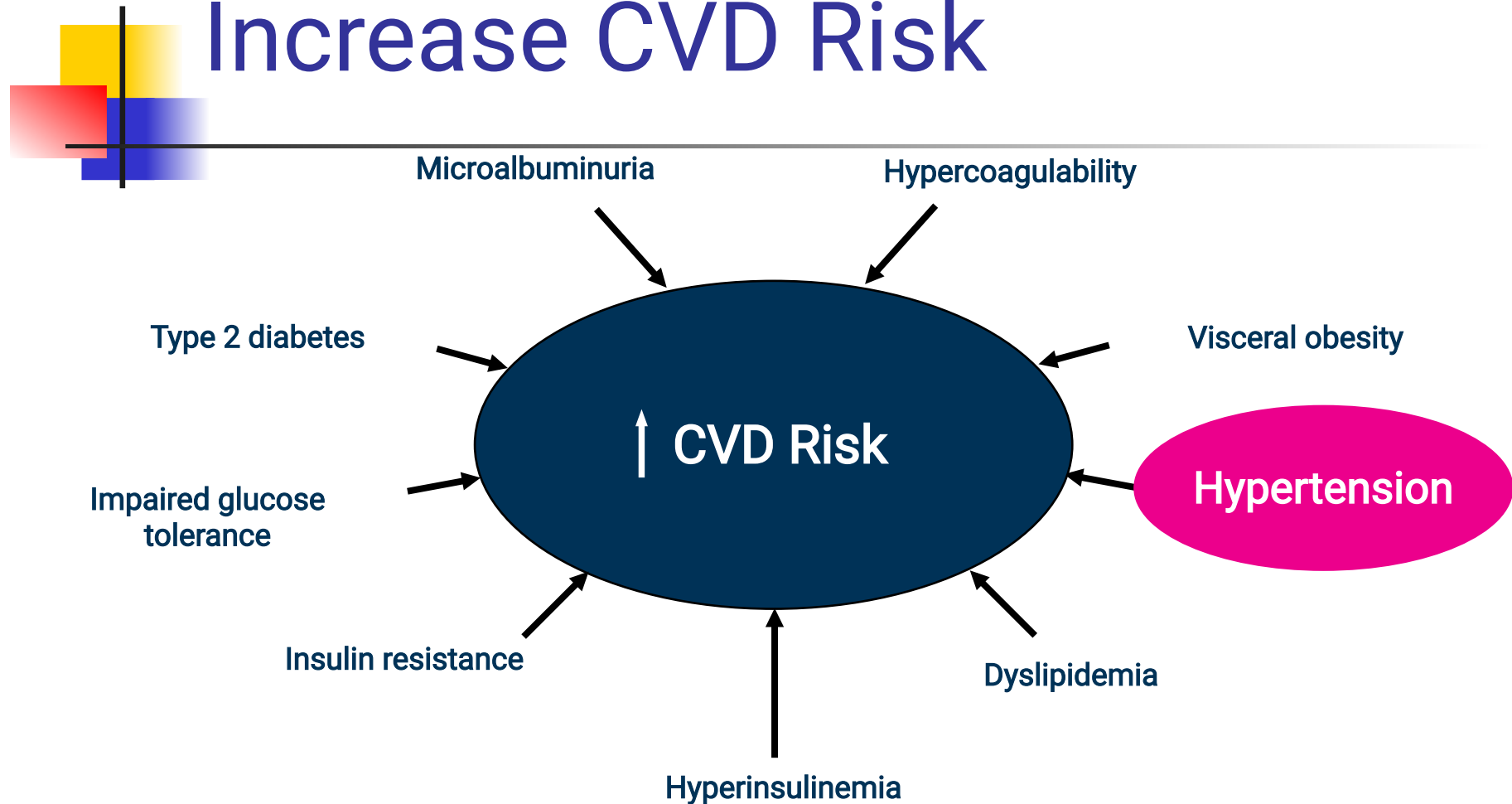
- (1) **increased adrenergic drive**, as often found in young people (aged 30–49 years);
- (2) **high-renin hypertension**, as seen in individuals with **renal dysfunction**;
- (3) **low-renin hypertension**, as recorded in individuals with **inherently raised aldosterone concentrations**;
- (4) **increased peripheral vascular resistance (PVR)**, as seen in elderly patients.



Important Recognized Causes of Secondary Hypertension



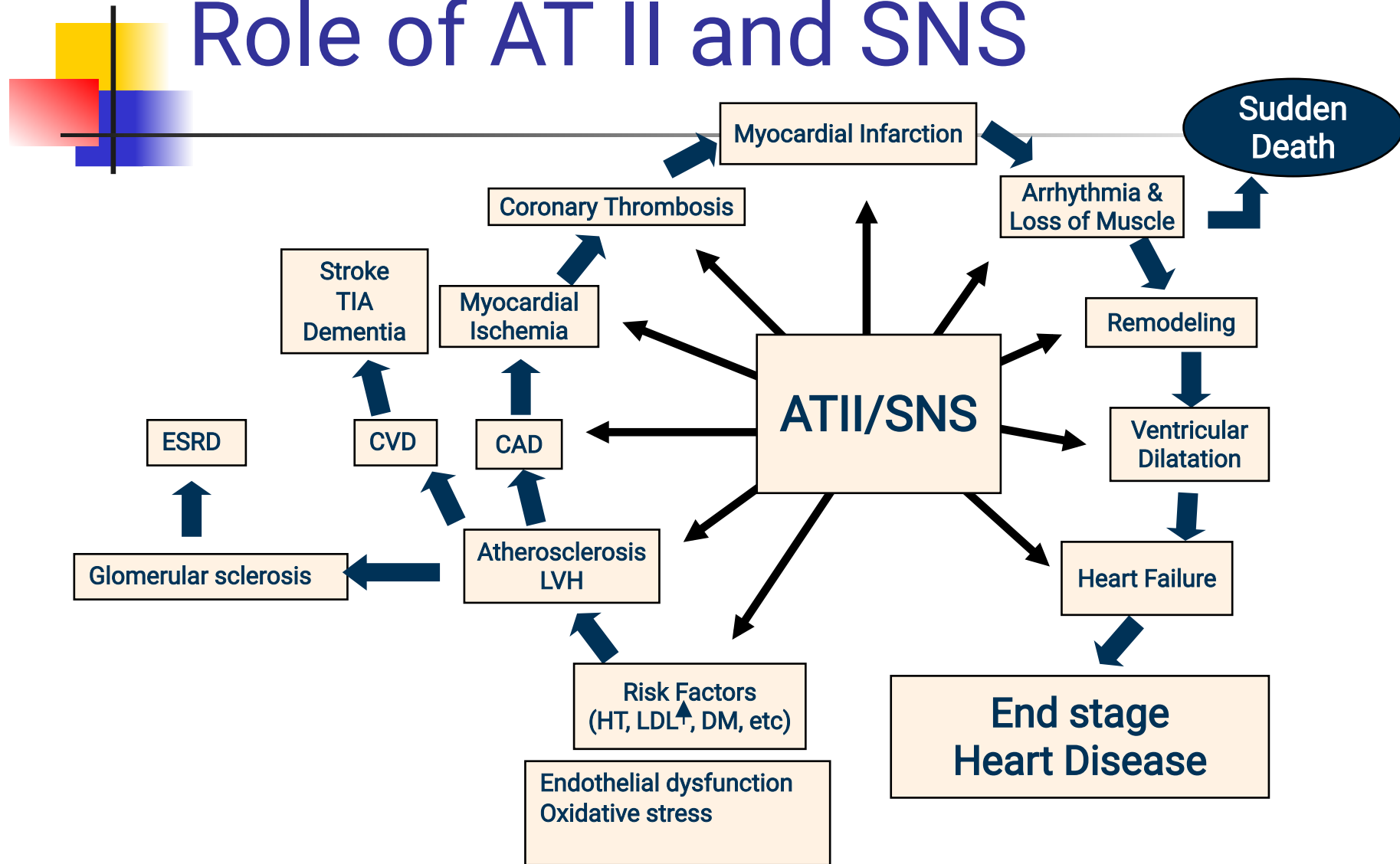
Associated Conditions Increase CVD Risk



The high prevalence of hypertension worldwide has contributed to the present pandemic of cardiovascular diseases (CVD) - responsible for 30% of all deaths worldwide

The cardiovascular continuum

Role of AT II and SNS





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Diagnosis of Arterial Hypertension

Symptoms

— Most patients are asymptomatic

- Diagnosis at routine medical examination
- Diagnosis when stroke or MI
- Hypertension called “Silent Killer”

■ Diagnostic procedures

■ Aim:

- * Establishing **blood pressure levels**
- * Identifying **secondary causes of hypertension**
- * Evaluating the overall cardiovascular risk

■ Procedures:

- * **Repeated blood pressure measurements**
- * Medical history
- * Physical examination
- * Laboratory and instrumental investigations

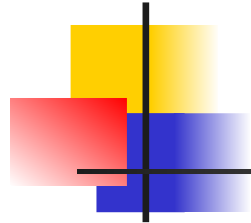
BP measurement




Patient sits for 5 mins -back supported, arm supported
at the level of the heart

- No caffeine/adrenergic stimulants
- No smoking in the preceding 15 mins
- Cuff should encircle and cover two thirds of the length of the arm
- Use a mercury sphygmomanometer or validated electronic BP machine
- Two recordings at each visit at least three sets of readings one week apart
- Deflate at 2mm/hg/second
- Record Korotkoff sound I -SBP, V – DBP

Diagnosis of Arterial Hypertension



- **Medical history** with particular attention to:
 - Hypertension
 - Life style factors – smoking, alcohol, diet, physical exercise, family history etc
 - Diabetes
 - Dyslipidemia
 - Premature coronary heart disease
 - Stroke
 - Renal disease
- **Physical examination**
 - Signs suggesting secondary hypertension obesity height, weight, BMI, WC, HC, HWR
 - Signs of organ damage:
 - * Brain* Retina 
 - * Heart * Peripheral arteries

Diagnosis of Arterial Hypertension



Laboratory and instrumental investigations

■ Routine tests

- Plasma glucose
- Total cholesterol, HDL-cholesterol, fasting serum triglycerides
- Serum creatinine, uric acid
- Serum potassium
- Hemoglobin and hematocrit
- Urinalysis (dipstick, urinary sediment)
- Electrocardiogram

Diagnosis of Arterial Hypertension



Laboratory and instrumental investigations

■ Recommended tests

- Echocardiogram, Carotid ultrasound
- Postprandial plasma glucose (if FBS > 6.1 mmol/l or 110 mg/l)
- C-reactive protein (high sensitivity)
- Microalbuminuria (essential in diabetics)
- 24hr proteinuria (if dipstick positive)
- Funduscopy (in severe hypertension)

Diagnosis of Arterial Hypertension



Factors influencing prognosis

Risk factors for cardiovascular disease used for stratification

- Levels of systolic and diastolic BP
- Men > 55 years; Women > 65 years
- Smoking
- Dyslipidemia
- Family history of PCAD
- ❖ Abdominal obesity (circ) M > 102 cm, W > 88 cm)
- ❖ C-reactive protein > 1 mg/dl
- ❖ Diabetes mellitus
- ❖ Fasting plasma glucose > 7.0 mmol/l
- ❖ Postprandial plasma glucose > 11.0 mmol/l

Diagnosis of Arterial Hypertension



Factors influencing prognosis

Target organ damage (TOD)

- LVH (ECG/ echo)
- Carotid arterial wall thickening or atherosclerotic plaque (US)
- Slight increase in serum creatinine (M 115-133, W 107-124 μ mol/l)
- Microalbuminuria (30-300 mg/24 h; albumin-creatinine ratio M > 2.5, W > 3.5)

- Cardiovascular disease; CVA/TIA
- Heart disease: AMI, angina, CABG, CHF
- Renal disease: diabetic nephropathy
- Peripheral vascular disease
- Advanced retinopathy: haemorrhages or exudates, papilloedema

Cardiovascular Risk Stratification to Quantify Prognosis

	Normal SBP 120-129 or DBP 80-84	High normal SBP 130-139 or DBP 85-89	Grade 1 SBP 140-159 or DBP 90-99	Grade 2 SBP 160-179 or DBP 100-109	Grade 3 SBP \geq 180 or DBP \geq 110
No other Risk Factors (RF)	Average risk	Average risk	Low added risk	Moderate added risk	High added risk
1 or 2 Additional RF	Low added risk	Low added risk	Moderate added risk	Moderate added risk	Very high added risk
3 or more RF, or Met synd or TOD	Moderate added risk	High added risk	High added risk	High added risk	Very high added risk
Established CV or Renal disease	High added risk	Very high added risk	Very high added risk	Very high added risk	Very high added risk

Absolute **added** 10 year risk of cardiovascular disease (Framingham):

< 15%
 15-20%
 20-30%
 > 30%

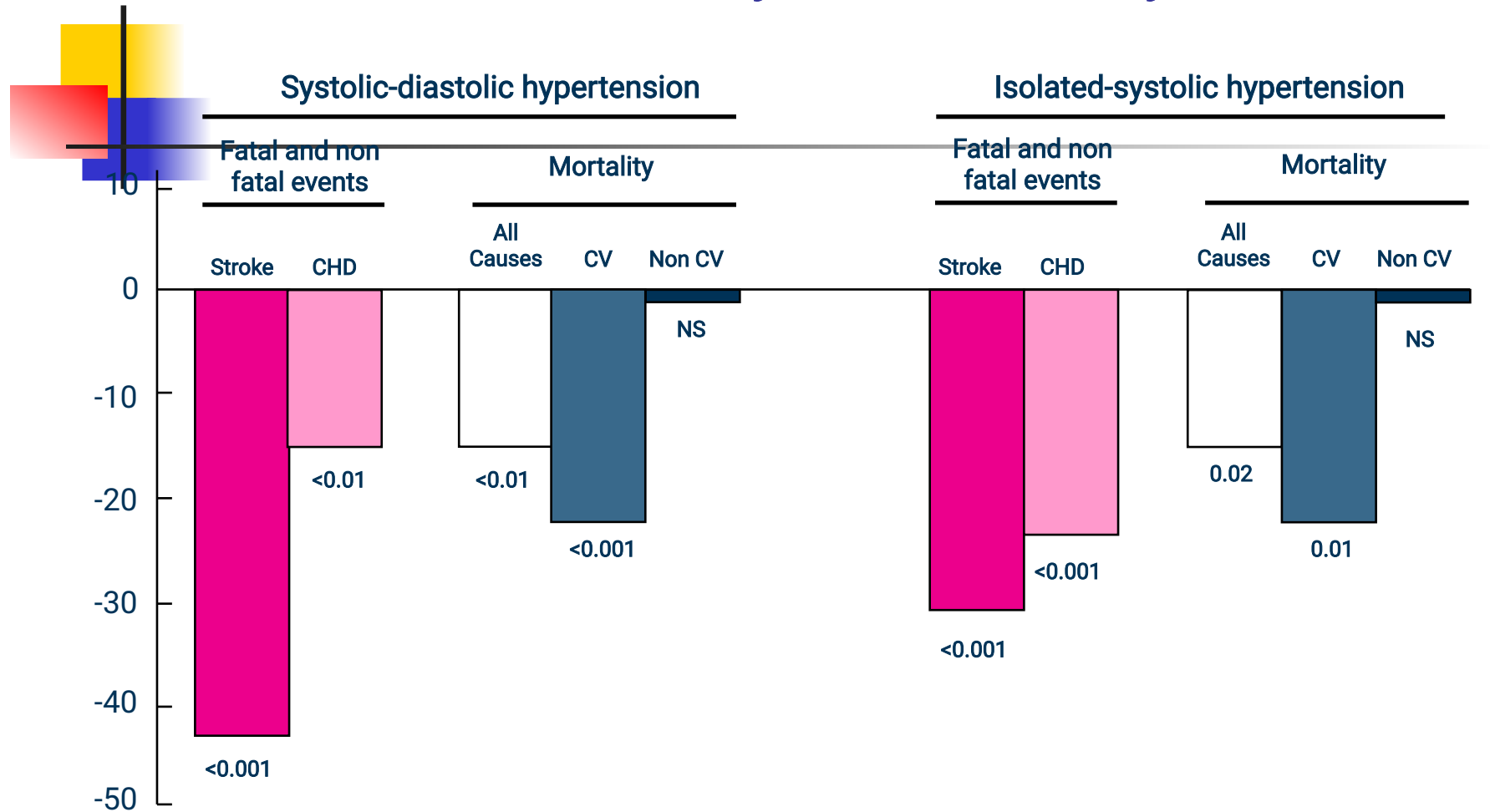
TOD = Target Organ Dammage



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Effective Blood Pressure Control Reduces Cardiovascular Morbidity and Mortality



Event reduction in patients on active antihypertensive treatment versus placebo or no treatment.
 CHD: coronary heart disease; CV: cardiovascular

BP Reductions as Little as 2 mm Hg Reduce Risk of CV Events by Up to 10%

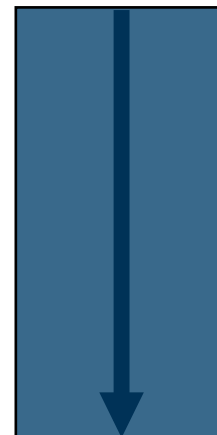
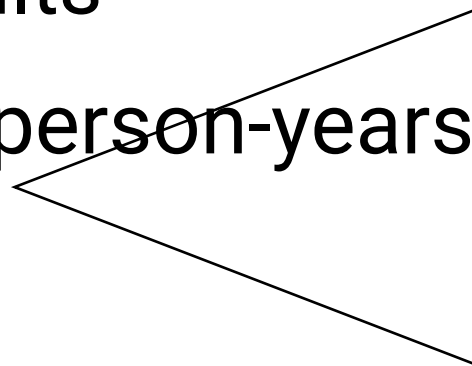
- Meta-analysis of 61 prospective, observational studies

- 1 million adults

- 12.7 million person-years

2 mm Hg

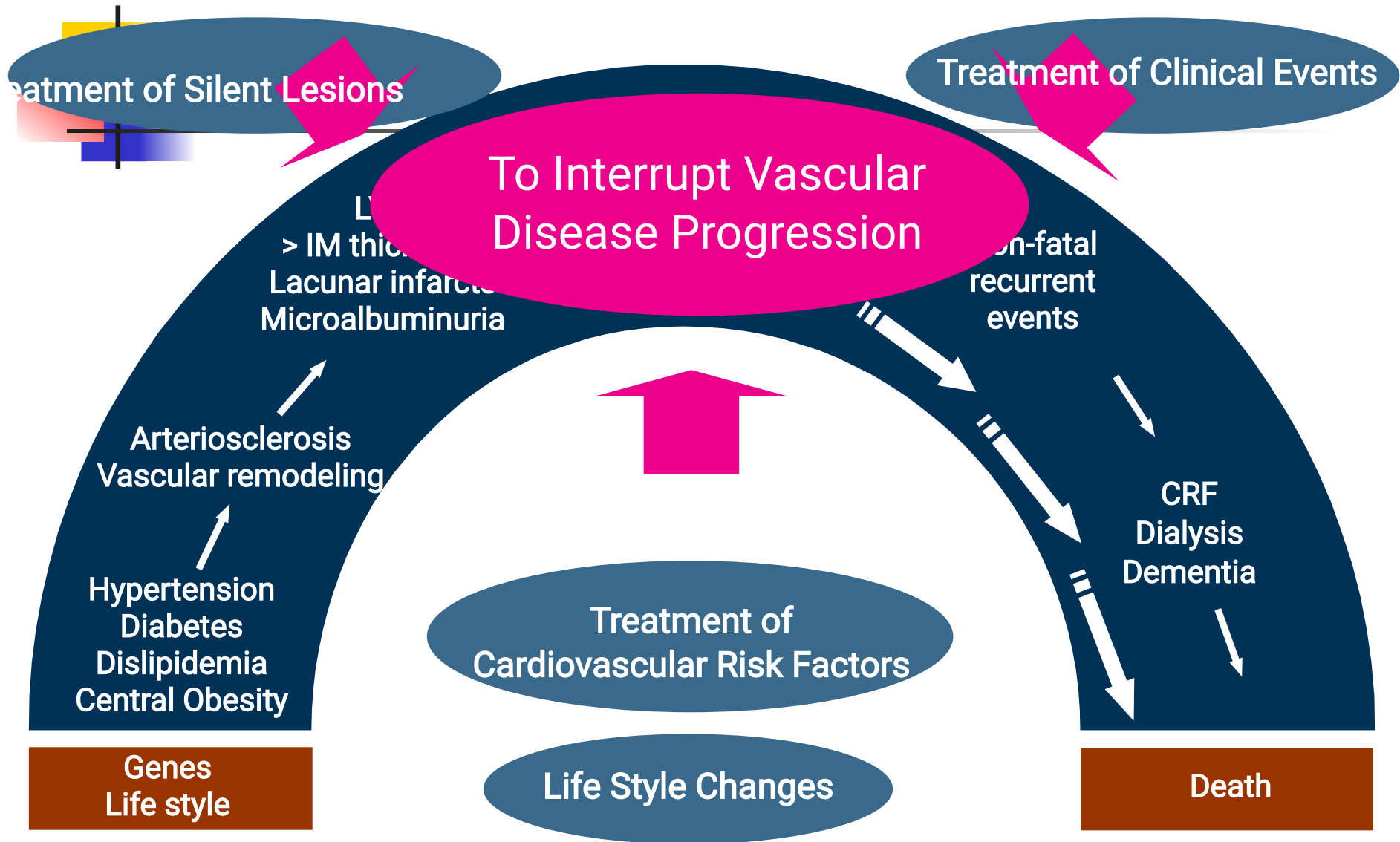
decrease in mean
SBP



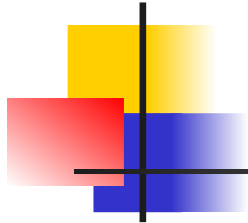
7% reduction in
risk of ischemic
heart disease
mortality

10% reduction in
risk of stroke
mortality

Natural History of Cardiovascular Disease



Goals of Treatment



- Maximum decrease in long-term total CV risk
- Lower level of systolic and diastolic BP per se
- **Treat all reversible risk factors and associated conditions**
- **Target BP:**
 - At least < **140/90 mmHg** in all HT patients
 - < **130/80 mmHg** in diabetics and other high risk patients eg renal dysfunction, CVA, proteinuria, post AMI
 - **Difficult to achieve in the elderly**
- **Start anti hypertensive** before cardiovascular complications develop

Lifestyle Changes

Purpose: lower BP, control other risk factors, reduce number of drugs required to reach target

❖ *This is for all and should be normal way of life in all to prevent hypertension*

- Smoking cessation
- Weight reduction
- Reduction of excessive alcohol intake
- Physical exercise
- Reduction of salt intake
- Increase in fruit and vegetable intake
- Decrease in saturated and total fat intake

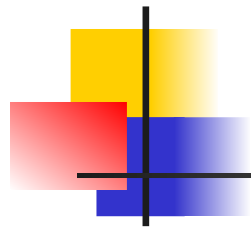


Initiation of antihypertensive treatment

Blood pressure (mmHg)					
Other risk factors and disease history	Normal SBP 120-129 or DBP 80-84	High normal SBP 130-139 or DBP 85-89	Grade 1 SBP 140-159 or DBP 90-99	Grade 2 SBP 160-179 or DBP 100-109	Grade 3 SBP \geq 180 or DBP \geq 110
No other Risk Factors (RF)	No BP intervention	No BP intervention	Lifestyle changes for several months, then drug treatment	Lifestyle changes for several wks then drug treatment	Immediate drug treatment and lifestyle changes
1- 2 Additional RF	Lifestyle changes	Lifestyle changes	Lifestyle changes for several wks then drug treatment	Lifestyle changes for several wks, then drug treatment	Immediate drug treatment and lifestyle changes
3 or more RF, or Diabetes or TOD	Lifestyle changes + drug treatment if DM	Drug treatment and lifestyle changes	Drug treatment and lifestyle changes	Drug treatment and lifestyle changes	Immediate drug treatment and lifestyle changes
Established CV or Renal disease	Drug treatment and lifestyle changes	Immediate drug treatment and lifestyle changes	Immediate drug treatment and lifestyle changes	Immediate drug treatment and lifestyle changes	Immediate drug treatment and lifestyle changes

TOD, target organ damage; SBP, systolic blood pressure; DBP, diastolic blood pressure.

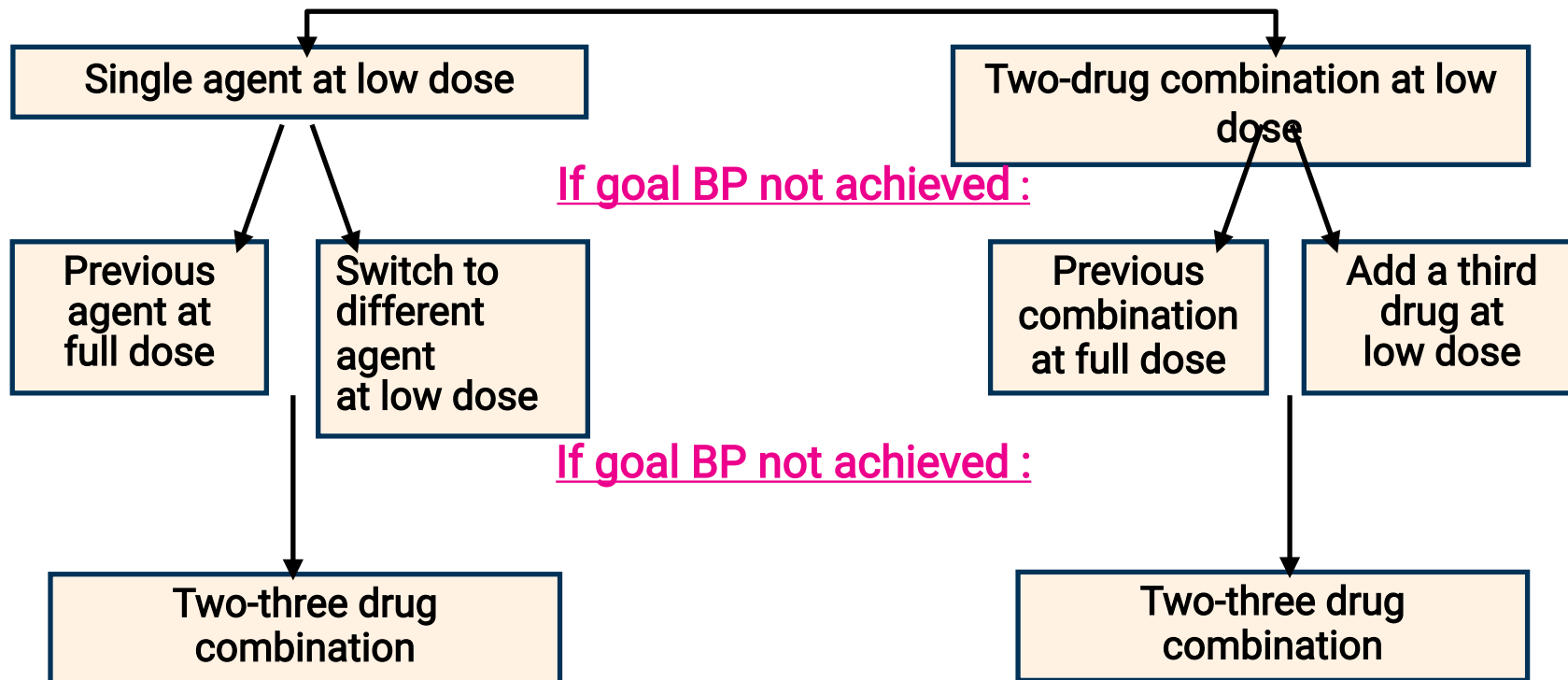
Algorithm for Pharmacological Treatment of Hypertension



Consider :

Blood pressure level before treatment
Absence or presence of TOD and risk factors

Choose between :



If goal BP not achieved :

If goal BP not achieved :



Recommendations by ADA, ISHIB, and NKF consistent with JNC 7

- Drug therapy is recommended for *all* patients with hypertension (SBP/DBP >140/90 mmHg)
- BP goals
 - <140/90 mmHg
 - <130/80 mmHg for patients with diabetes mellitus
 - <125/75mmHg CRF + proteinuria >1g/day
- *Multiple drug therapy* with 2 or more agents at adequate doses (thiazide diuretic, ACE inhibitor, ARB, beta-blocker, CCB) is usually required to achieve BP targets
- ISHIB guidelines: *consider initiating* treatment with 2 drugs if BP is $\geq 15/10$ mmHg above goal

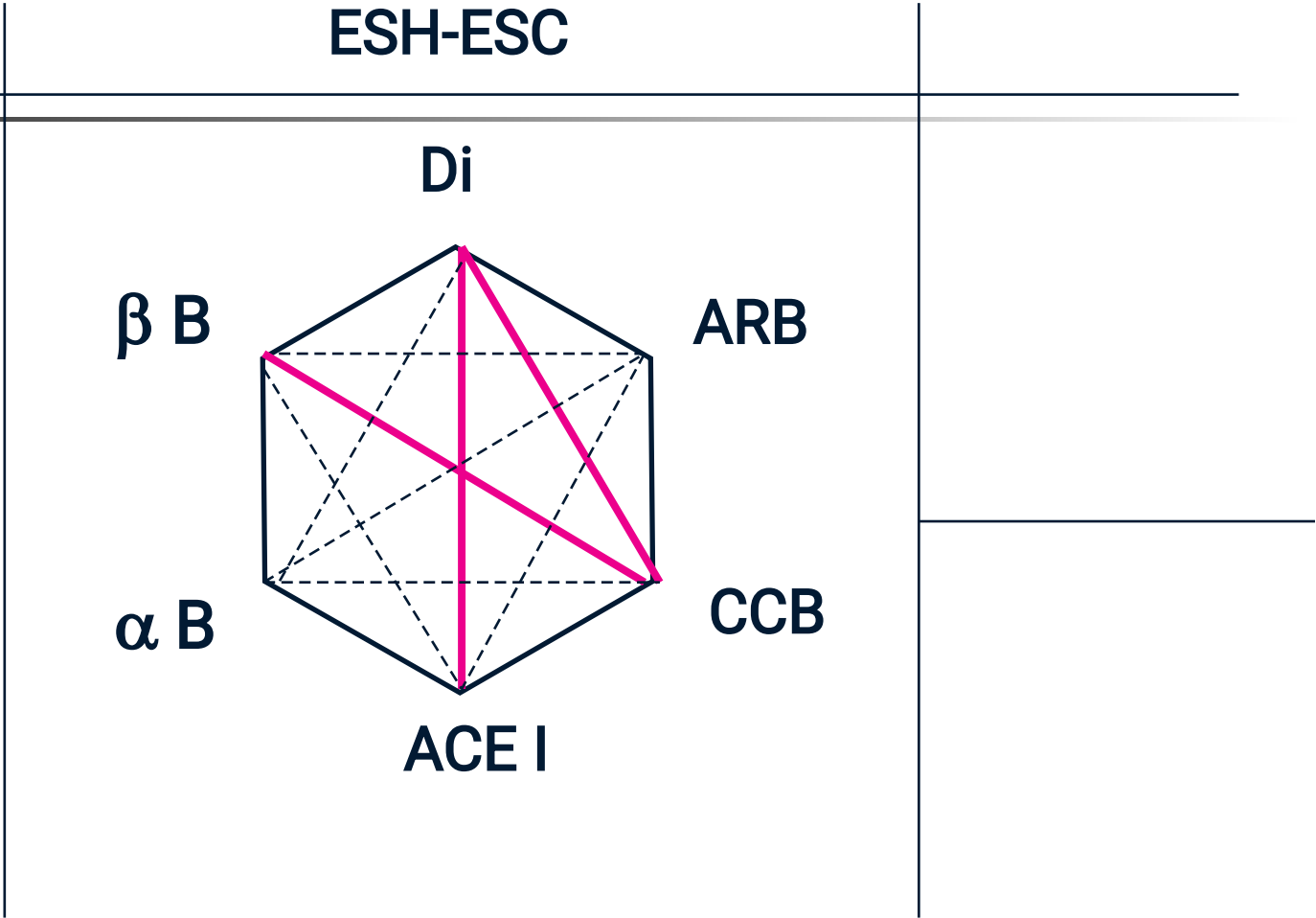
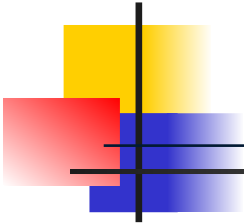
Compelling drug indications



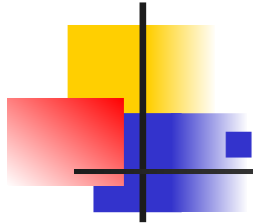
In case of Sub clinical organ damage

- LVH – ARB, ACE, CCB
- Microalbuminuria/ renal dysfunction – ARB, ACE
- Heart failure – ACE, BB, ARB, diuretics, aldosterone inhibitors
- Metabolic syndrome – ARB, ACE, CCB
- ISH (elderly) – diuretics, CCB
- Pregnancy – BB, CCB, alpha methyldopa

Recommended combinations



Control is often poor



■ Poor compliance

- Denial
- Inadequate patient education
- Inadequate life style change
- Cost
- Side effects
- Competing alternative therapies
- Fatigue
- Poly pharmacy
- Poor drug combinations
- Sub optimal dosing

Lifestyle Changes are not easy to implement and sustain

CANINE CONSTITUTIONAL



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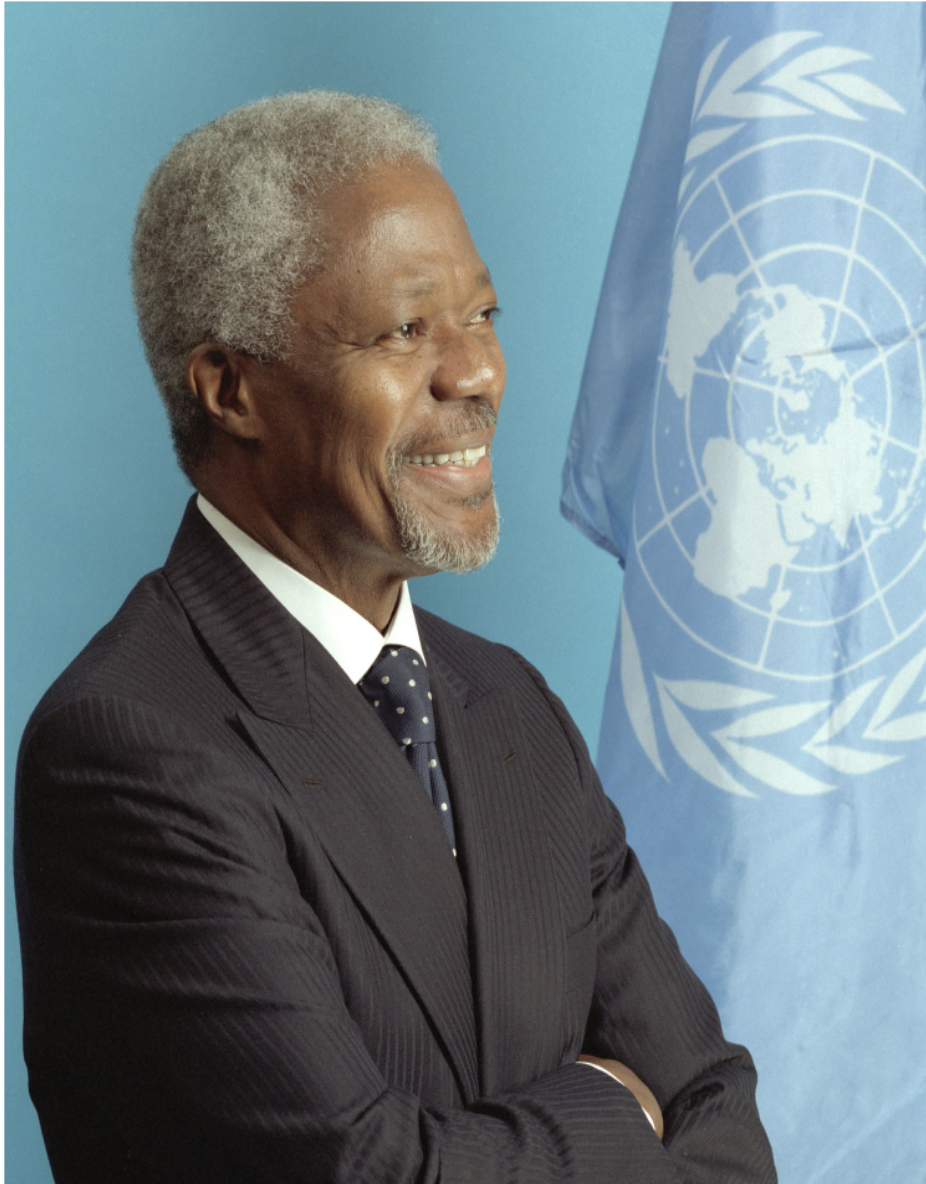


HK resident smoking through SARS mask

Physical (In)Activity



Towards a Culture of Prevention



“Today no one disputes that prevention is better and cheaper than reacting to crises after the fact. And yet our political and organizational cultures and practices remain oriented far more towards reaction than prevention.”

Kofi Annan, September 9, 1999