

Update on Hypertension

Muhammad Ali Abdool
 Specialist Trainee in Cardiology (North West, UK)
 MBChB (Manchester, UK)
 MRCP (London, UK)
 PgDip Medical Education (Manchester, UK)
 Fellow of Higher Education Academy (UK)
 Honorary Clinical Tutor at the Universities of Manchester and Liverpool
 Member of European Society of Cardiology
 Member of British Cardiovascular Society

Acknowledgments

Thank you to Dr Abdoolhan, Uman and USM for their support and assistance

Disclosures

I have received speaker fees from the following companies:

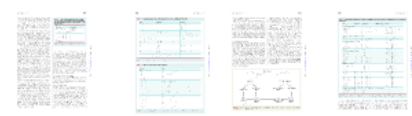
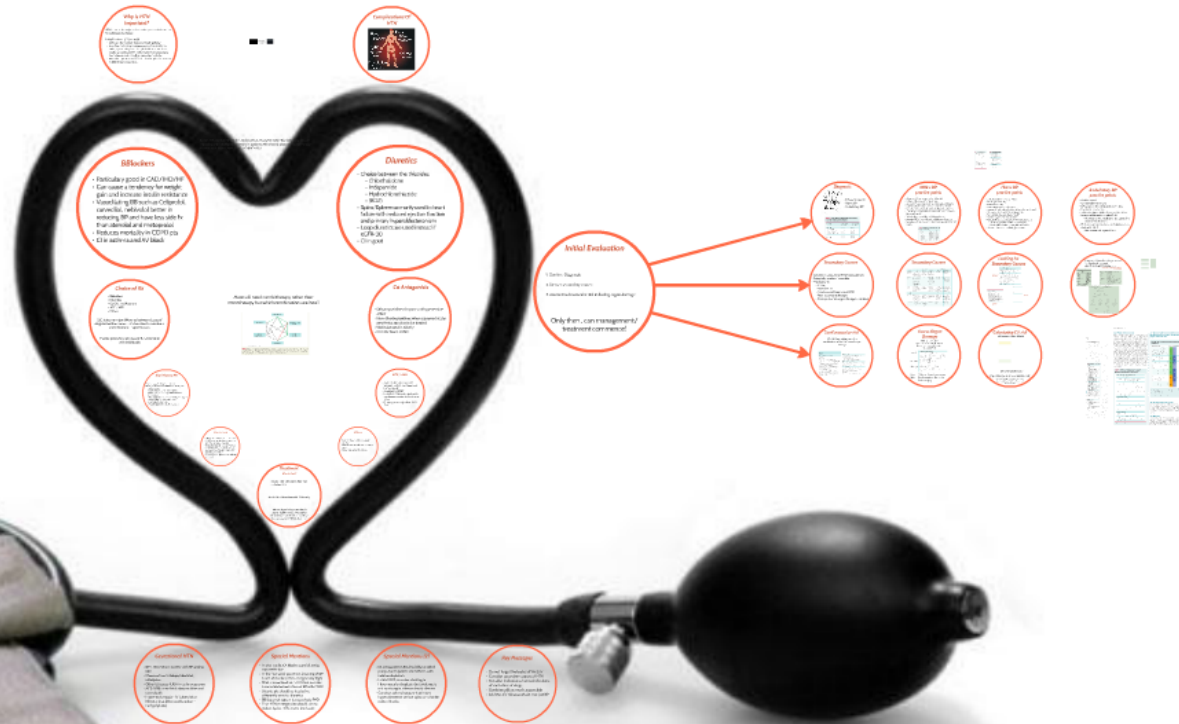
Hoffmann-La Roche, Sanofi, AstraZeneca, Bristol-Myers Squibb, Pfizer, Merck, Janssen, Amgen, Novartis, Boehringer Ingelheim, Merck KGaA, Novartis, Amgen, Bristol-Myers Squibb, Pfizer, Merck, Janssen, Amgen, Novartis, Boehringer Ingelheim, Merck KGaA

Session Aims

- Update on Initial Evaluation of HTN
- Review of Management / Treatment
- Overview of Tx in special conditions

Intro

- My experience and choice of subject
- UK practice based on ESH / ESC Guidelines - 2013
- 53 pages long





Acknowledgments

Thank you to Dr Ackbarckhan, Unicorn
and UOM for their support and
sponsorship

Disclosures

and UOM for their support and
sponsorship

Disclosures

I have received sponsorship from the following
companies:

Medtronic, Boston Scientific, St Jude medical, Bayer,
Abbott, Pfizer, Roche, GSK, Novartis, Astrazeneca,
Merck, Eli Lilly

Session Aims

- Update on Initial Evaluation of HTN
- Review of *Management / Treatment*
- Overview of Tx in special conditions

Intro

- My experience and choice of subject
- UK practice based on ESH / ESC Guidelines - 2013
 - 53 pages long

Why is HTN important?

HTN is one of the major and potentially treatable risk factors for cardiovascular disease

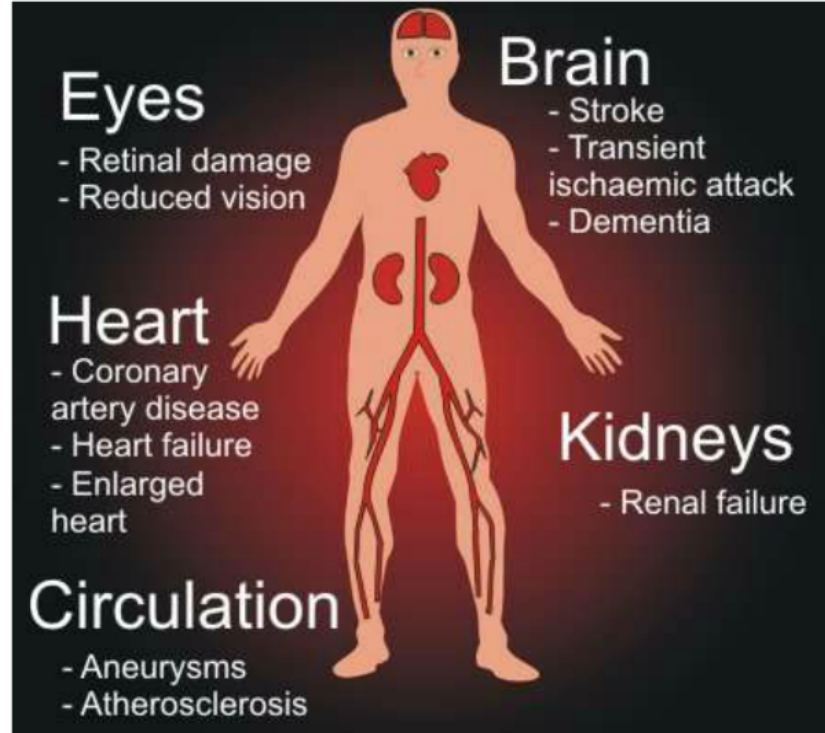
WHO Fact sheet 317 (Jan 2015)

- CVDs are the number 1 cause of death globally
- An estimated 17.5 million people died from CVDs in 2012, representing 31% of all global deaths. Of these deaths, an estimated 7.4 million were due to coronary heart disease and 6.7 million were due to stroke .
- Over three quarters of CVD deaths take place in low- and middle-income countries.

However in Mtius...
I think we have a
bigger killer!



Complications Of HTN



Initial Evaluation

1. Confirm Diagnosis
2. Detect secondary causes
3. Assess Cardiovascular risk including organ damage

Only then , can management/
treatment commence!

Diagnosis



"I'm going to take your blood pressure, so try to relax and not think about what a high reading might mean for your chances of living a long, healthy life."

- Office/ Clinic BP
- Home BP
- Ambulatory BP

Table 6 Definitions of hypertension by office and out-of-office blood pressure levels

for your chances of living a long, healthy life.”

Table 6 Definitions of hypertension by office and out-of-office blood pressure levels

| Category | Systolic BP (mmHg) | | Diastolic BP (mmHg) |
|-----------------------|--------------------|--------|---------------------|
| Office BP | ≥140 | and/or | ≥90 |
| Ambulatory BP | | | |
| Daytime (or awake) | ≥135 | and/or | ≥85 |
| Nighttime (or asleep) | ≥120 | and/or | ≥70 |
| 24-h | ≥130 | and/or | ≥80 |
| Home BP | ≥135 | and/or | ≥85 |

Office BP practice points

- Allow to sit for 3-5 mins before taking BP
- Patient sitting and cuff at heart level
- At least 2 measurements (more if markedly different)
- Careful with AF - manual measurements are better
- Consider different sized cuffs for different sized arms
- Use disappearance of heart sounds for measurements (Kortkoff I and V)
- BP in both arms and highest BP as reference
- Standing BP at 1 and 3 mins in elderly and diabetics
- Measure resting pulse as this has an independent CV risk profile

Table 3 Definitions and classification of office blood pressure levels (mmHg)^a

| Category | Systolic | | Diastolic |
|----------------------|----------|--------|-----------|
| Optimal | <120 | and | <80 |
| Normal | 120–129 | and/or | 80–84 |
| High normal | 130–139 | and/or | 85–89 |
| Grade 1 hypertension | 140–159 | and/or | 90–99 |
| Grade 2 hypertension | 160–179 | and/or | 100–109 |

Table 3 Definitions and classification of office blood pressure levels (mmHg)^a

| Category | Systolic | | Diastolic |
|--------------------------------|----------|--------|-----------|
| Optimal | <120 | and | <80 |
| Normal | 120–129 | and/or | 80–84 |
| High normal | 130–139 | and/or | 85–89 |
| Grade 1 hypertension | 140–159 | and/or | 90–99 |
| Grade 2 hypertension | 160–179 | and/or | 100–109 |
| Grade 3 hypertension | ≥180 | and/or | ≥110 |
| Isolated systolic hypertension | ≥140 | and | <90 |

Home BP practice points

- Daily measurements for 3-7 days
- Morning and evenings
- Seated for 5 mins
- 2 measurements 1-2 mins apart
- Average (of all readings) is taken after exclusion of Day 1
- Cheaper and extended follow up possible
- More reliable measure than office BP
- Can be as good as Ambulatory BP
- Reveals white coat or masked HTN (13% prev each)
 - White coat is when Office BP > Home BP
 - Masked HTN is when Home BP > Office BP
- No information on true day/night variance

Ambulatory BP practice points

- Gold standard
- On non-dominant hand
- BP variance < 5 mmHG at time of fitting
- No strenuous exercise
- Advise to stop activities during cuff inflation
- Reveals white coat or masked HTN
 - nb. these terms should only be reserved for untreated individuals
- Picks up day/night variance and dipping status (normal is >10%)
 - Non-dippers at higher CV risk



Secondary Causes

- Consider in all patients (5-10% prevalence)
- Potentially curable / reversible
- Mandatory for
 - Young
 - Severe HTN
 - Sudden onset/worsening HTN
 - Poor response to therapy
 - Disproportionate organ damage to duration


Secondary Causes

| Common causes | Clinical indications | | | Diagnostics | |
|---------------------------|---|---|---|--|--|
| | Clinical history | Physical examination | Laboratory investigations | First-line test(s) | Additional/confirmatory test(s) |
| Renal parenchymal disease | History of urinary tract infection or obstruction, haematuria, analgesic abuse; family history of polycystic kidney disease. | Abdominal masses (in case of polycystic kidney disease). | Presence of protein, erythrocytes, or leucocytes in the urine, decreased GFR. | Renal ultrasound | Detailed work-up for kidney disease. |
| Renal artery stenosis | Fibromuscular dysplasia: early onset hypertension (especially in women). Atherosclerotic stenosis: hypertension of abrupt onset, worsening or increasingly difficult to treat; flash pulmonary oedema. | Abdominal bruit | Difference of >1.5 cm in length between the two kidneys (renal ultrasound), rapid deterioration in renal function (spontaneous or in response to RAA blockers). | Renal Duplex Doppler ultrasonography | Magnetic resonance angiography, spiral computed tomography, intra-arterial digital subtraction angiography. |
| Primary aldosteronism | Muscle weakness; family history of early onset hypertension and cerebrovascular events at age <40 years. | Arrhythmias (in case of severe hypokalaemia). | Hypokalaemia (spontaneous or diuretic-induced); incidental discovery of adrenal masses. | Aldosterone–renin ratio under standardized conditions (correction of hypokalaemia and withdrawal of drugs affecting RAA system). | Confirmatory tests (oral sodium loading, saline infusion, fludrocortisone suppression, or captopril test); adrenal CT scan; adrenal vein sampling. |
| Uncommon causes | | | | | |
| Pheochromocytoma | Paroxysmal hypertension or a crisis superimposed to sustained hypertension; headache, sweating, palpitations and pallor; positive family history of pheochromocytoma. | Skin stigmata of neurofibromatosis (café-au-lait spots, neurofibromas). | Incidental discovery of adrenal (or in some cases, extra-adrenal) masses. | Measurement of urinary fractionated metanephrines or plasma-free metanephrines. | CT or MRI of the abdomen and pelvis; I ¹²³ I-labelled meta-iodobenzyl-guanidine scanning; genetic screening for pathogenic mutations. |
| Cushing's syndrome | Rapid weight gain, polyuria, polydipsia, psychological disturbances. | Typical body habitus (central obesity, moon-face, buffalo hump, red striae, hirsutism). | Hyperglycaemia | 24-h urinary cortisol excretion | Dexamethasone-suppression tests |

Secondary causes

| | Clinical indications | | | Diagnostics | |
|---------------------------|---|--|---|--|--|
| Common causes | Clinical history | Physical examination | Laboratory investigations | First-line test(s) | Additional/ confirmatory test(s) |
| Renal parenchymal disease | History of urinary tract infection or obstruction, haematuria, analgesic abuse; family history of polycystic kidney disease. | Abdominal masses (in case of polycystic kidney disease). | Presence of protein, erythrocytes, or leucocytes in the urine, decreased GFR. | Renal ultrasound | Detailed work-up for kidney disease. |
| Renal artery stenosis | Fibromuscular dysplasia: early onset hypertension (especially in women). Atherosclerotic stenosis: hypertension of abrupt onset, worsening or increasingly difficult to treat; flash pulmonary oedema. | Abdominal bruit | Difference of >1.5 cm in length between the two kidneys (renal ultrasound), rapid deterioration in renal function (spontaneous or in response to RAA blockers). | Renal Duplex Doppler ultrasonography | Magnetic resonance angiography, spiral computed tomography, intra-arterial digital subtraction angiography. |
| Primary aldosteronism | Muscle weakness; family history of early onset hypertension and cerebrovascular events at age <40 years. | Arrhythmias (in case of severe hypokalaemia). | Hypokalaemia (spontaneous or diuretic-induced); incidental discovery of adrenal masses. | Aldosterone–renin ratio under standardized conditions (correction of hypokalaemia and withdrawal of drugs affecting RAA system). | Confirmatory tests (oral sodium loading, saline infusion, fludrocortisone suppression, or captopril test); adrenal CT scan; adrenal vein sampling. |
| Uncommon causes | | | | | |
| Pheochromocytoma | Paroxysmal hypertension or a crisis superimposed | Skin stigmata of neurofibromatosis | Incidental discovery of adrenal (or in some | Measurement of urinary fractionated | CT or MRI of the abdomen and pelvis; |

| | | | | | |
|------------------------|---|---|---|--|--|
| Primary aldosteronism | Muscle weakness; family history of early onset hypertension and cerebrovascular events at age <40 years. | Arrhythmias (in case of severe hypokalaemia). | Hypokalaemia (spontaneous or diuretic-induced); incidental discovery of adrenal masses. | Aldosterone–renin ratio under standardized conditions (correction of hypokalaemia and withdrawal of drugs affecting RAA system). | Confirmatory tests (oral sodium loading, saline infusion, fludrocortisone suppression, or captopril test); adrenal CT scan; adrenal vein sampling. |
| Uncommon causes | | | | | |
| Pheochromocytoma | Paroxysmal hypertension or a crisis superimposed to sustained hypertension; headache, sweating, palpitations and pallor; positive family history of pheochromocytoma. | Skin stigmata of neurofibromatosis (café-au-lait spots, neurofibromas). | Incidental discovery of adrenal (or in some cases, extra-adrenal) masses. | Measurement of urinary fractionated metanephrines or plasma-free metanephrines. | CT or MRI of the abdomen and pelvis; ¹²³ I-labelled meta-iodobenzyl-guanidine scanning; genetic screening for pathogenic mutations |
| Cushing's syndrome | Rapid weight gain, polyuria, polydipsia, psychological disturbances. | Typical body habitus (central obesity, moon-face, buffalo hump, red striae, hirsutism). | Hyperglycaemia | 24-h urinary cortisol excretion | Dexamethasone-suppression tests |



Looking for Secondary Causes

History

Secondary hypertension

- a) Family history of CKD (polycystic kidney).
- b) History of renal disease, urinary tract infection, haematuria, analgesic abuse (parenchymal renal disease).
- c) Drug/substance intake, e.g. oral contraceptives, liquorice, carbenoxolone, vasoconstrictive nasal drops, cocaine, amphetamines, gluco- and mineralocorticosteroids, non-steroidal anti-inflammatory drugs, erythropoietin, cyclosporine.
- d) Repetitive episodes of sweating, headache, anxiety, palpitations (pheochromocytoma).
- e) Episodes of muscle weakness and tetany (hyperaldosteronism).
- f) Symptoms suggestive of thyroid disease.

Signs suggesting secondary hypertension

- Features of Cushing syndrome.
- Skin stigmata of neurofibromatosis (pheochromocytoma).
- Palpation of enlarged kidneys (polycystic kidney).
- Auscultation of abdominal murmurs (renovascular hypertension).

Routine tests

- Haemoglobin and/or haematocrit.
- Fasting plasma glucose.
- Serum total cholesterol, low-density lipoprotein cholesterol, high-density lipoprotein cholesterol.
- Fasting serum triglycerides.
- Serum potassium and sodium.
- Serum uric acid.
- Serum creatinine (with estimation of GFR).
- Urine analysis: microscopic examination; urinary protein by dipstick test; test for microalbuminuria.
- 12-lead ECG.

Additional tests, based on history, physical examination, and findings from routine laboratory tests

- Haemoglobin A_{1c} (if fasting plasma glucose is ≥ 5.6 mmol/L (102 mg/dL) or previous diagnosis of diabetes).
- Catecholamine metabolites if dipstick test is microalbuminuria.

Tests

(hyperaldosteronism).

f) Symptoms suggestive of thyroid dise

Signs suggesting secondary hypertension

- Features of Cushing syndrome.
- Skin stigmata of neurofibromatosis (pheochromocytoma).
- Palpation of enlarged kidneys (polycystic kidney).
- Auscultation of abdominal murmurs (renovascular hypertension).
- Auscultation of precordial or chest murmurs (aortic coarctation; aortic disease; upper extremity artery disease).
- Diminished and delayed femoral pulses and reduced femoral blood pressure compared to simultaneous arm BP (aortic coarctation; aortic disease; lower extremity artery disease).
- Left–right arm BP difference (aortic coarctation; subclavian artery stenosis).

Exam

pheochromocytoma).

muscle weakness and tetany
(hypocalcaemia).

suggestive of thyroid disease.

| Routine tests |
|--|
| • Haemoglobin and/or haematocrit. |
| • Fasting plasma glucose. |
| • Serum total cholesterol, low-density lipoprotein cholesterol, high-density lipoprotein cholesterol. |
| • Fasting serum triglycerides. |
| • Serum potassium and sodium. |
| • Serum uric acid. |
| • Serum creatinine (with estimation of GFR). |
| • Urine analysis: microscopic examination; urinary protein by dipstick test; test for microalbuminuria. |
| • 12-lead ECG. |
| Additional tests, based on history, physical examination, and findings from routine laboratory tests |
| • Haemoglobin A _{1c} (if fasting plasma glucose is >5.6 mmol/L (102 mg/dL) or previous diagnosis of diabetes). |
| • Quantitative proteinuria (if dipstick test is positive); urinary potassium and sodium concentration and their ratio. |
| • Home and 24-h ambulatory BP monitoring. |
| • Echocardiogram. |
| • Holter monitoring in case of arrhythmias. |
| • Exercise testing. |
| • Carotid ultrasound. |
| • Peripheral artery/abdominal ultrasound. |
| • Pulse wave velocity. |
| • Ankle-brachial index. |
| • Fundoscopy. |
| Extended evaluation (mostly domain of the specialist) |
| • Further search for cerebral, cardiac, renal, and vascular damage, mandatory in resistant and complicated hypertension. |
| • Search for secondary hypertension when suggested by history, physical examination, or routine and additional tests. |

Tests



Diagnosis of Secondary Hypertension: An Age-Based Approach

ANTHONY J. VIERA, MD, MPH, and DANA M. NEUTZE, MD, PhD

University of North Carolina at Chapel Hill School of Medicine, Chapel Hill, North Carolina

American Family Physician Volume 82, Number 12 ·

December 15, 2010

of Secondary

th
use *Most common etiologies†*

Renal parenchymal disease
Coarctation of the aorta

Table 2. Select Drugs That May Elevate Blood Pressure

| <i>Drug class</i> | <i>Common examples</i> |
|-------------------|------------------------|
|-------------------|------------------------|

Basic Approach

A, MD, MPH, and DANA M. NEUTZE, MD, PhD

Carolina at Chapel Hill School of Medicine, Chapel Hill, North Carolina

American Family Physician Volume 82, Number 12 ·

December 15, 2010

Table 2. Select Drugs That May Elevate Blood Pressure

| <i>Drug class</i> | <i>Common examples</i> |
|--------------------------------|---|
| Estrogen | Oral contraceptives |
| Herbal | Ephedra, ginseng, ma huang |
| Illicit | Amphetamines, cocaine |
| Nonsteroidal anti-inflammatory | Cyclooxygenase-2 inhibitors, ibuprofen, naproxen (Naprosyn) |
| Psychiatric | Buspirone (Buspar), carbamazepine (Tegretol), clozapine (Clozaril), fluoxetine (Prozac), lithium, tricyclic antidepressants |
| Steroid | Methylprednisolone (Depo-Medrol), prednisone |
| Sympathomimetic | Decongestants, diet pills |

Approach for Suspected Secondary Hypertension

Check accuracy of blood pressure measurements,
rule out diet- and drug-related causes



Table 3. Most Common Causes of Secondary Hypertension by Age*

| <i>Age groups</i> | <i>Percentage of hypertension with an underlying cause</i> | <i>Most common etiologies†</i> |
|-------------------------------------|--|---|
| Children (birth to 12 years) | 70 to 85 | Renal parenchymal disease Coarctation of the aorta |
| Adolescents (12 to 18 years) | 10 to 15 | Renal parenchymal disease Coarctation of the aorta |
| Young adults (19 to 39 years) | 5 | Thyroid dysfunction Fibromuscular dysplasia Renal parenchymal disease |
| Middle-aged adults (40 to 64 years) | 8 to 12 | Aldosteronism Thyroid dysfunction Obstructive sleep apnea Cushing syndrome Pheochromocytoma |
| Older adults (65 years and older) | 17 | Atherosclerotic renal artery stenosis Renal failure Hypothyroidism |

*—Excluding dietary and drug causes and the risk factor of obesity.

†—Listed in approximate order of frequency within groups.

Information from references 2, 3, and 30 through 34.

der adults
55 years and
lder)

17

Obstructive sleep apnea
Cushing syndrome
Pheochromocytoma
Atherosclerotic renal artery
stenosis
Renal failure
Hypothyroidism

Psychiatric

Buspirone (Buspar), carbamazepine (Tegretol),
clozapine (Clozaril), fluoxetine (Prozac), lithium,
tricyclic antidepressants

Steroid

Methylprednisolone (Depo-Medrol), prednisone

Sympathomimetic

Decongestants, diet pills

-Excluding dietary and drug causes and the risk factor of obesity.
Listed in approximate order of frequency within groups.

Information from references 2, 3, and 30 through 34.

Evaluation for Suspected Secondary Hypertension

Check accuracy of blood pressure measurements,
rule out diet- and drug-related causes

History, physical examination, laboratory testing

Clinical clues

See Table 1

No clinical clues, but secondary hypertension
remains a concern (e.g., in a child, rapid onset or
acceleration of hypertension, resistant hypertension)

Child/adolescent

Young adult

Middle-aged adult

Older adult

Urinalysis*
Urine culture
Renal ultrasonography

MRI with gadolinium
contrast media or
CT renal artery†
TSH*

Renin and
aldosterone levels
TSH*

MRI with gadolinium
contrast media or
CT renal artery†
TSH*
Urinalysis*

Echocardiography

Polysomnography
(sleep study)

24-hour urinary cortisol

24-hour urinary fractionated metanephrines

Cardiovascular risk

CV risk, hence prognosis, is a combination of risk factors and organ damage

Risk factors

Male sex

Age (men ≥ 55 years; women ≥ 65 years)

Smoking

Dyslipidaemia

Total cholesterol > 4.9 mmol/L (190 mg/dL) and/or

Diabetes mellitus

Fasting plasma glucose ≥ 7.0 mmol/L (126 mg/dL) on two repeated measurements, and/or

HbA_{1c} $> 7\%$ (53 mmol/mol), and/or

Post-load plasma glucose ≥ 11.0 mmol/L (198 mg/dL)

combination of risk factors and organ damage

Risk factors

Male sex

Age (men ≥ 55 years; women ≥ 65 years)

Smoking

Dyslipidaemia

Total cholesterol >4.9 mmol/L (190 mg/dL), and/or

Low-density lipoprotein cholesterol >3.0 mmol/L (115 mg/dL), and/or

High-density lipoprotein cholesterol: men <1.0 mmol/L (40 mg/dL), women <1.2 mmol/L (46 mg/dL), and/or

Triglycerides >1.7 mmol/L (150 mg/dL)

Fasting plasma glucose 5.6–6.9 mmol/L (102–125 mg/dL)

Abnormal glucose tolerance test

Obesity [BMI ≥ 30 kg/m² (height²)]

Abdominal obesity (waist circumference: men ≥ 102 cm; women ≥ 88 cm) (in Caucasians)

Family history of premature CVD (men aged <55 years; women aged <65 years)

Diabetes mellitus

Fasting plasma glucose ≥ 7.0 mmol/L (126 mg/dL) on two repeated measurements, and/or

HbA_{1c} $>7\%$ (53 mmol/mol), and/or

Post-load plasma glucose >11.0 mmol/L (198 mg/dL)

Established CV or renal disease

Cerebrovascular disease: ischaemic stroke; cerebral haemorrhage; transient ischaemic attack

CHD: myocardial infarction; angina; myocardial revascularization with PCI or CABG

Heart failure, including heart failure with preserved EF

Symptomatic lower extremities peripheral artery disease

CKD with eGFR <30 mL/min/1.73m² (BSA); proteinuria (>300 mg/24 h).

Advanced retinopathy: haemorrhages or exudates, papilloedema

Assess Organ Damage

OD is an intermediate stage in the continuum of vascular disease, and hence a determinant of overall CV risk

Heart

Asymptomatic organ damage

Pulse pressure (in the elderly) ≥ 60 mmHg

Electrocardiographic LVH (Sokolow–Lyon index > 3.5 mV;
RaVL > 1.1 mV; Cornell voltage duration product > 244 mV*ms) or

OD is an intermediate stage in the continuum of vascular disease, and hence a determinant of overall CV risk

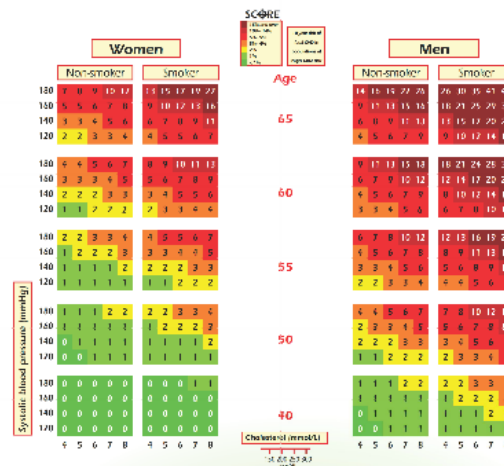
| | Asymptomatic organ damage |
|----------|--|
| Heart | Pulse pressure (in the elderly) ≥ 60 mmHg Electrocardiographic LVH (Sokolow–Lyon index >3.5 mV; RaVL >1.1 mV; Cornell voltage duration product >244 mV*ms), or Echocardiographic LVH [LVM index: men >115 g/m ² ; women >95 g/m ² (BSA)] ^a |
| Arteries | Carotid wall thickening (IMT >0.9 mm) or plaque Carotid–femoral PWV >10 m/s Ankle-brachial index <0.9 |
| Kidneys | CKD with eGFR 30–60 mL/min/1.73 m ² (BSA) Microalbuminuria (30–300 mg/24 h), or albumin–creatinine ratio (30–300 mg/g; 3.4–34 mg/mmol) (preferentially on morning spot urine) |
| Eyes | Retinopathy on fundoscopy |
| Brain | Cerebrovascular disease on brain imaging |

Calculating CV risk

ESC recommends SCORE

SCORE - European High Risk Chart

10 year risk of fatal CVD in high risk regions of Europe by gender, age, systolic blood pressure, total cholesterol and smoking status



How do I use the SCORE chart to assess CVD risk in my patients?

1. Use the chart to assess the 10-year risk of fatal CVD in high risk regions of Europe by gender, age, systolic blood pressure, total cholesterol and smoking status. The chart is divided into two main sections: Women and Men. Each section is further divided into Non-smoker and Smoker. The risk is stratified by age (40, 50, 55, 60, 65) and systolic blood pressure (120, 140, 160, 180 mmHg). The risk is further stratified by total cholesterol (4, 5, 6, 7, 8 mmol/L). Risk levels are color-coded: Green (Low risk, <1%), Yellow (Low-moderate risk, 1-5%), Orange (Moderate risk, 5-10%), and Red (High risk, >10%).

2. Use the chart to assess the 10-year risk of fatal CVD in high risk regions of Europe by gender, age, systolic blood pressure, total cholesterol and smoking status. The chart is divided into two main sections: Women and Men. Each section is further divided into Non-smoker and Smoker. The risk is stratified by age (40, 50, 55, 60, 65) and systolic blood pressure (120, 140, 160, 180 mmHg). The risk is further stratified by total cholesterol (4, 5, 6, 7, 8 mmol/L). Risk levels are color-coded: Green (Low risk, <1%), Yellow (Low-moderate risk, 1-5%), Orange (Moderate risk, 5-10%), and Red (High risk, >10%).

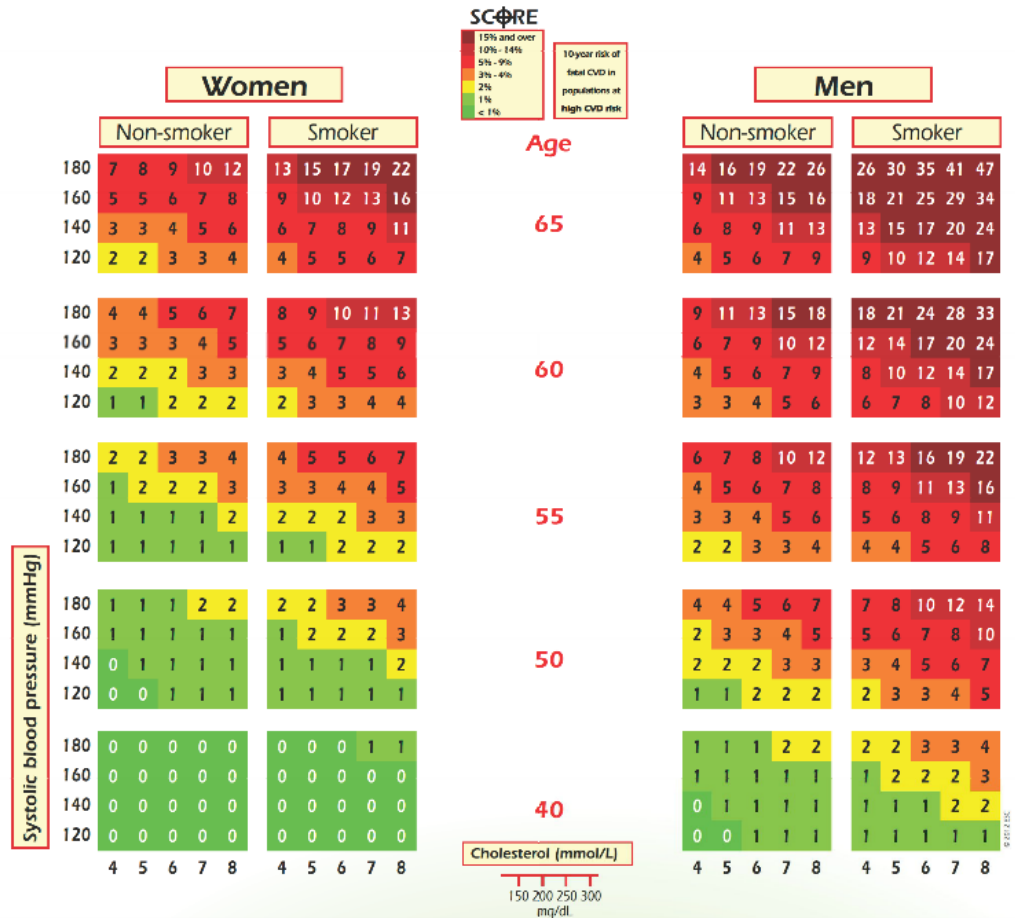
UK (and I) prefer JBS 3

<http://www.jbs3risk.com/JBS3Risk.swf>

Address CV Risk with appropriate interventions

SCORE - European High Risk Chart

10 year risk of fatal CVD in high risk regions of Europe by gender, age, systolic blood pressure, total cholesterol and smoking status



How do I use the SCORE charts to assess CVD risk in asymptomatic persons?

1. Use the **low risk charts** in Andorra, Austria, Belgium*, Cyprus, Denmark, Finland, France, Germany, Greece*, Iceland, Ireland, Israel, Luxembourg, Malta, Monaco, The Netherlands*, Norway, Portugal, San Marino, Slovenia, Spain*, Sweden*, Switzerland and the United Kingdom.

Use the **high risk charts** in other European countries. Of these, some are at very high risk and the charts may underestimate risk in these. These include Armenia, Azerbaijan, Belarus, Bulgaria, Georgia, Kazakhstan, Kyrgyzstan, Latvia, Lithuania, Macedonia FYR, Moldova, Russia, Ukraine and Uzbekistan.

Relative Risk Charts

Note that a low total cardiovascular risk in a young person may conceal a high relative risk; this may be explained to the person by using the relative risk chart. As the person ages, a high relative risk will translate into a high total risk. More intensive lifestyle advice will be needed in such persons. This chart refers to relative risk, not percentage risk, so that a person in the top right corner is at 12 times higher risk than a person in the bottom left corner.

Another approach to explaining risk to younger persons is to use cardiovascular risk age. For example, in the high risk chart, a 40 year old male hypertensive smoker has a risk of 4%, which is the same as a 65 year old with no risk factors, so that his risk age is 65. This can be reduced by reducing his risk factors.

Risk estimation using SCORE: Qualifiers

- The charts should be used in the light of the clinician's knowledge and judgement, especially with regard to local conditions.
- As with all risk estimation systems, risk will be over-estimated in countries with a falling CVD mortality rate, and under-estimated if it is rising.
- At any given age, risk appears lower for women than men. However, inspection of the charts shows that their risk is merely deferred by 10 years, with a 60 year old woman resembling a 50 year old man in terms of risk.
- Risk may be higher than indicated in the chart in:
 - Sedentary or obese subjects, especially those with central obesity
 - Those with a strong family history of premature CVD
 - Socially deprived individuals and those from some ethnic minorities

Cyprus, Denmark, Finland, France, Germany, Greece, Iceland, Ireland, Israel, Italy, Luxembourg, Malta, Monaco, The Netherlands*, Norway, Portugal, San Marino, Slovenia, Spain*, Sweden*, Switzerland and the United Kingdom.

Use the **high risk charts** in other European countries. Of these, some are at very high risk and the charts may underestimate risk in these. These include Armenia, Azerbaijan, Belarus, Bulgaria, Georgia, Kazakhstan, Kyrgyzstan, Latvia, Lithuania, Macedonia FYR, Moldova, Russia, Ukraine and Uzbekistan.



How do I use the SCORE charts to assess CVD risk in asymptomatic persons?

1. Use the **low risk charts** in: Andorra, Austria, Belgium*, Cyprus, Denmark, Finland, France, Germany, Greece*, Iceland, Ireland, Israel, Italy, Luxembourg, Malta, Monaco, The Netherlands*, Norway, Portugal, San Marino, Slovenia, Spain*, Sweden*, Switzerland and the United Kingdom.

Use the **high risk charts** in other European countries. Of these, some are at very high risk and the charts may underestimate risk in these. These include Armenia, Azerbaijan, Belarus, Bulgaria, Georgia, Kazakhstan, Kyrgyzstan, Latvia, Lithuania, Macedonia, FYR, Moldova, Russia, Ukraine and Uzbekistan.

Relative Risk Charts

Note that a low total cardiovascular risk in a young person may conceal a high relative risk; this may be explained to the person by using the relative risk chart. As the person ages, a high relative risk will translate into a high total risk. More intensive lifestyle advice will be needed in such persons. This chart refers to relative risk, not percentage risk, so that a person in the top right corner is at 12 times higher risk than a person in the bottom left corner.

Another approach to explaining risk to younger persons is to use cardiovascular risk age. For example, in the high risk chart, a 40 year old male hypertensive smoker has a risk of 4%, which is the same as a 65 year old with no risk factors, so that his risk age is 65. This can be reduced by reducing his risk factors.

Risk estimation using SCORE: Qualifiers

- The charts should be used in the light of the clinician's knowledge and judgement, especially with regard to local conditions.
- As with all risk estimation systems, risk will be overestimated in countries with a falling CVD mortality rate, and underestimated if it is rising.
- At any given age, risk appears lower for women than men. However, inspection of the charts shows that their risk is merely deferred by 10 years, with a 50 year old woman resembling a 60 year old man in terms of risk.
- Risk may be higher than indicated in the chart in:
 - Sedentary or obese subjects, especially those with central obesity
 - Those with a strong family history of premature CVD
 - Socially deprived individuals and those from some ethnic minorities

UK (and I) prefer JBS 3

<http://www.jbs3risk.com/JBS3Risk.swf>

Address CV Risk with appropriate interventions such as antiplatelets, antidiabetics and lipid lowering therapy



Treatment

Who to treat?

- Grade 1 with OD and/or High Risk
- All Grade 2/3

Grade 1 low risk without OD - F/up only

Grade 1 low risk without OD - F/up only

Isolated Systolic Hypertension in young, healthy males, f/up rather than treat as no indication that they develop essential HTN in the future

How to treat

- SBP goal of <140 for almost all patients
- DBP goal of <90 in almost all patients with goal of <85 for diabetics
- In elderly, relax SBP goal to 140-150
- In elderly > 80, not in good physical and mental health, check tolerability and relax goals further
- Try to use combi pills when steady state achieved

Non Pharma Mx

Can be as good as drug monotherapy

Lifestyle measures with evidence or reducing BP

- salt restriction
- moderation of alcohol consumption
- high consumption of vegetables and fruits
- low-fat diet
- fibre, whole grains and plant protein, soy milk, fish
- weight reduction and maintenance
- regular physical exercise
- MOST importantly - smoking cessation



Choice of Rx

- Bblockers
- Diuretics
- Ca Channel Blockers
- ACE / ARB
- Others

ESC states no major difference between classes of drugs but outlines some limitations/contraindications and indications in specific cases

- Ca Channel Blockers
- ACE / ARB
- Others

ESC states no major difference between classes of drugs but outlines some limitations/contraindications and indications in specific cases

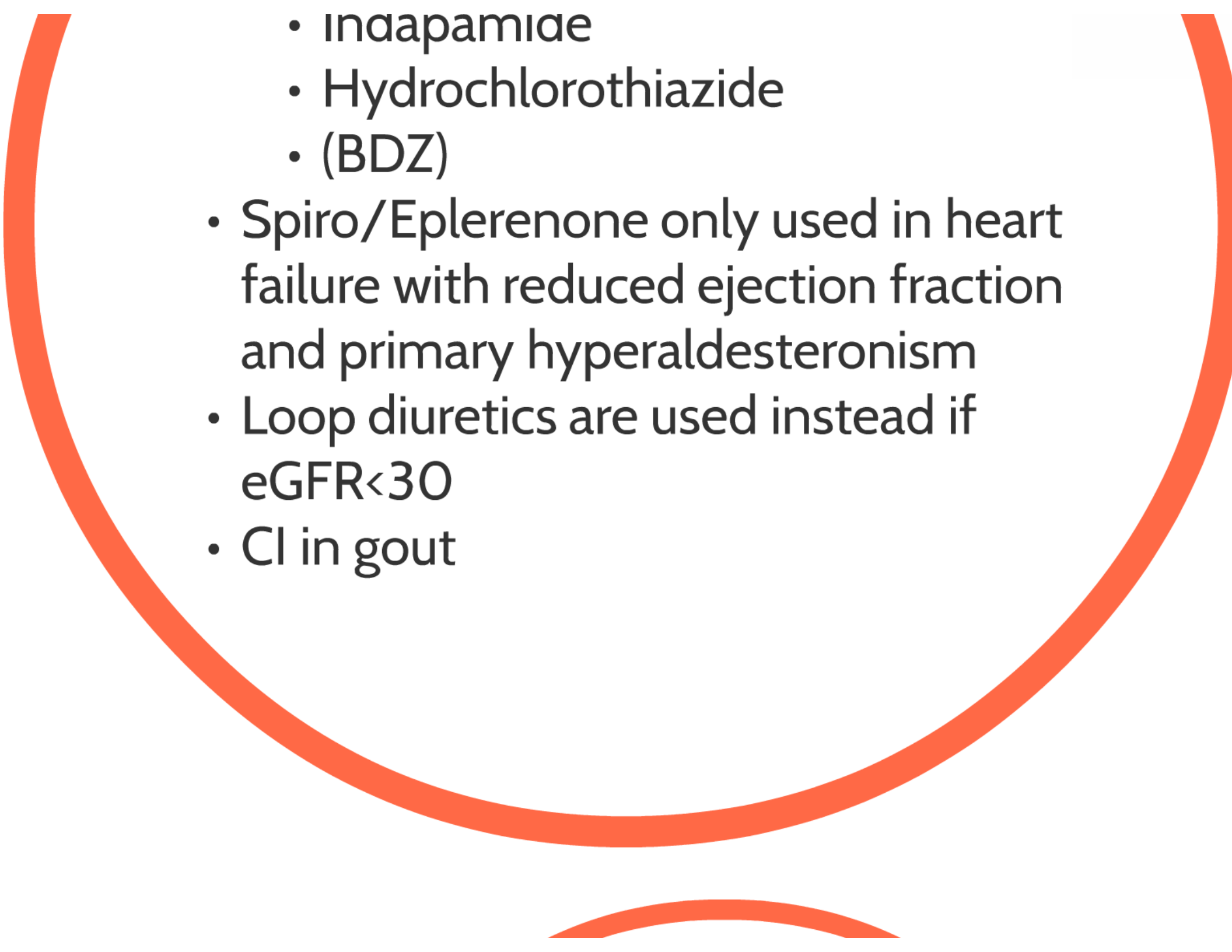
Practice points for each class of Rx will be dealt with individually

***BB**lockers*

- Particular good in CAD/IHD/HF
- Can cause a tendency for weight gain and increase insulin resistance
- Vasodilating BB such as Celiprolol, carvedilol, nebivolol better in reducing BP and have less side fx than atenolol and metoprolol
- Reduces mortality in COPD pts
- CI in asthma and AV block

Diuretics

- Choice between the thiazides
 - Chlorthalidone
 - Indapamide
 - Hydrochlorothiazide
 - (BDZ)
- Spiro/Eplerenone only used in heart failure with reduced ejection fraction and primary hyperaldosteronism

- 
- Indapamide
 - Hydrochlorothiazide
 - (BDZ)
 - Spiro/Eplerenone only used in heart failure with reduced ejection fraction and primary hyperaldosteronism
 - Loop diuretics are used instead if $eGFR < 30$
 - CI in gout

Ca Antagonists

- Dihydropyridines for pure antihypertensive effect
- Non-dihydropyridines when supraventricular arrhythmias are also to be treated
- Well tolerated in elderly
- CI in AV block, LVSD

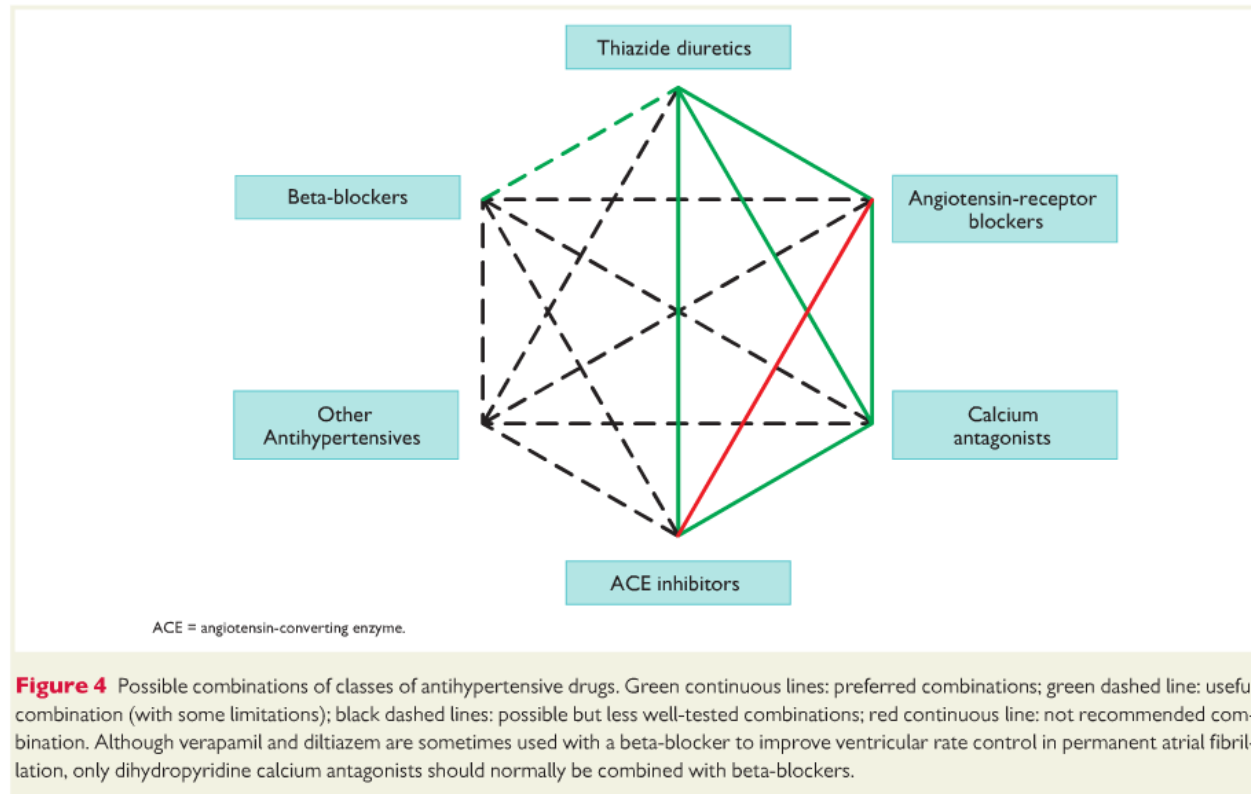
ACE / ARB

- Most widely used as very well tolerated, useful in most cases and has few side-fx
- Mandatory in HFREF
- Useful in CKD to reduce proteinuria and slow progression of chronic renal failure
- CI in pregnancy, angioedema, Bilat RAS

Others

- Renin inhibitor - aliskiren is under scrutiny
- Doxazosin as 3rd/4th line in resistant cases
- Moxonidine as last line therapy

Most will need combi therapy rather than monotherapy but which combinations are best?



Gestational HTN

- BP > 150/95 or 140/90 with RF and/or OD
- Choice of methyldopa, labetalol, nifedipine
- Other BB cause IUGR in early pregnancy
- ACE/ARB cause fetal abnormalities and even death
- In pre-eclampsia - IV Labetalol or Nitrates should be used (caution - tachyphylaxis)

Special Mentions

- In afro-carrib, CA Blockers and diuretics most effective
- In the first week post CVA, lowering of BP is not advisable (unless dangerously high)
- OSA is associated with HTN but no trials have validated reduction of BP with CPAP
- Dialysis pts should be treated no differently save for diuretics
- BB does not appear to exacerbate PAD
- Tx of HTN emergencies should aim to reduce bp by <25% every few hours

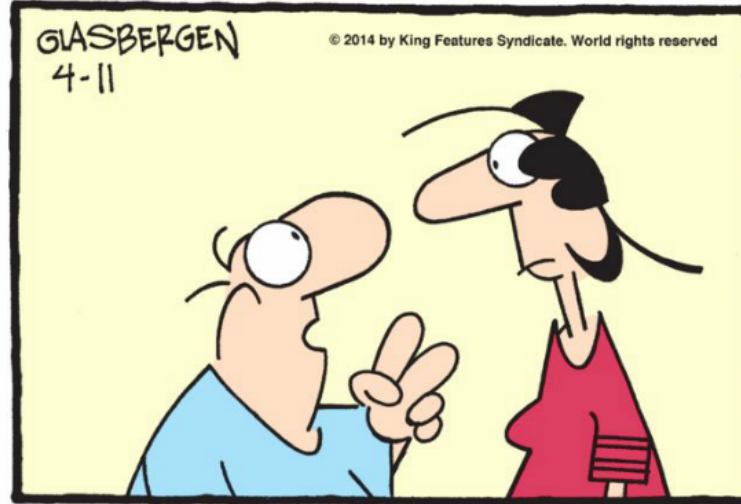
Special Mentions (2)

- Diuretics/ACE/ARB should be avoided preop due to potentiated effects with volume depletion
- In bilat RAS, consider stenting in Fibromuscular dysplasia but evidence is not so strong in atherosclerotic disease
- Consider adrenal surgery in primary hyperaldosteronism but spiro can also be quite effective

Key Messages

- Do not forget the basics of Hx & Ix
- Consider secondary causes of HTN
- Consider indications/contraindications of each class of drugs
- Combine pills as much as possible
- Address CV risk as a whole, not just BP

*Thank you and questions
(Only ones I can answer
please)*



“Marriage is two hearts beating as one.
That might be what’s causing my
high blood pressure.”