Hypertension Quoi de neuf?

PROF BELEN PONTE.

RESPONSABLE DE L'UNITÉ D'HYPERTENSION.

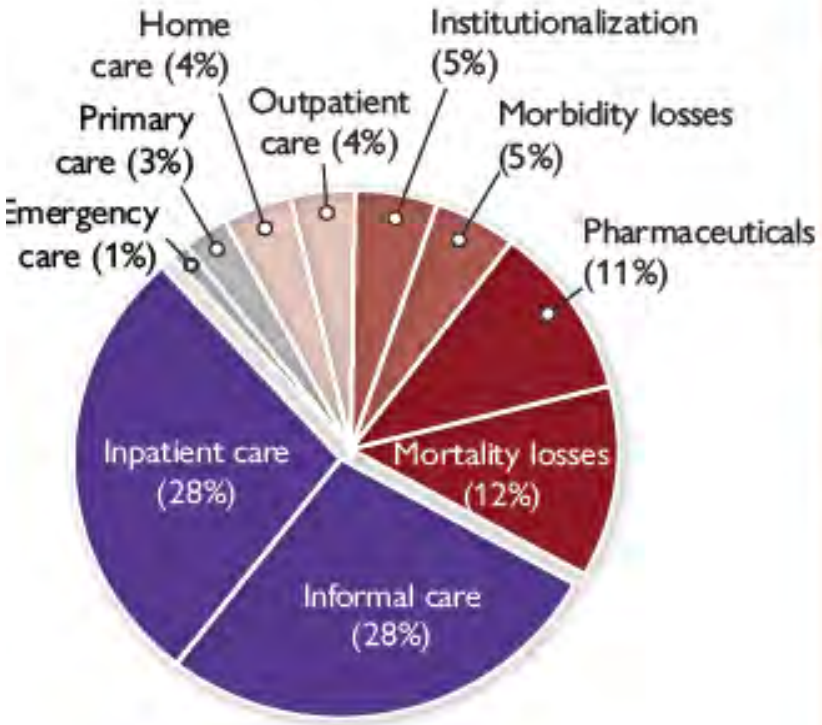
SERVICE DE NEPHROLOGIE ET HYPERTENSION

Hôpitaux Universitaires Genève

SHG SSI SSH

Economic burden of cardiovascular diseases (CVD) in 27 European Union countries is €282 billion annually

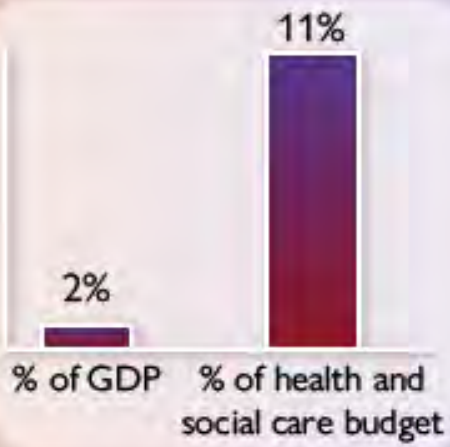
Costs by categories

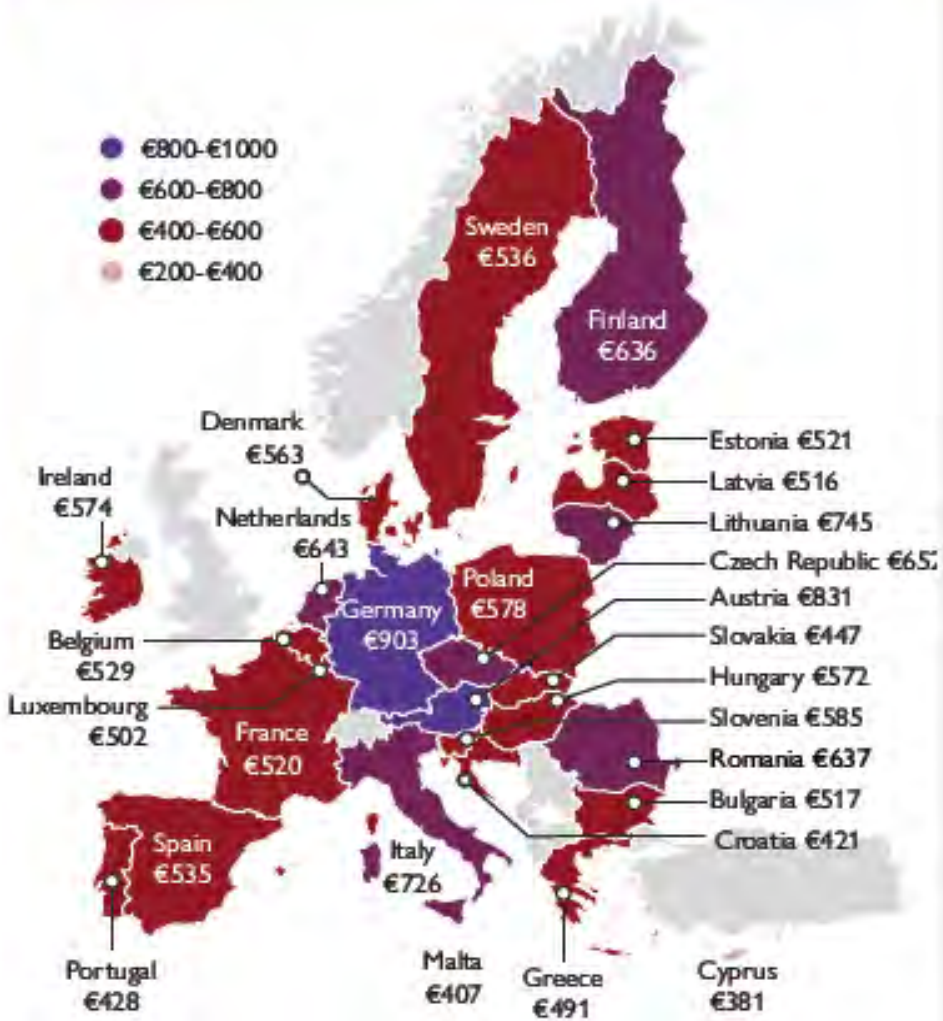


Coronary heart disease: **€77 Billion** (27%)

Cerebrovascular disease: **€76 Billion** (27%)

€630 per capita across EU (ppp-adjusted)

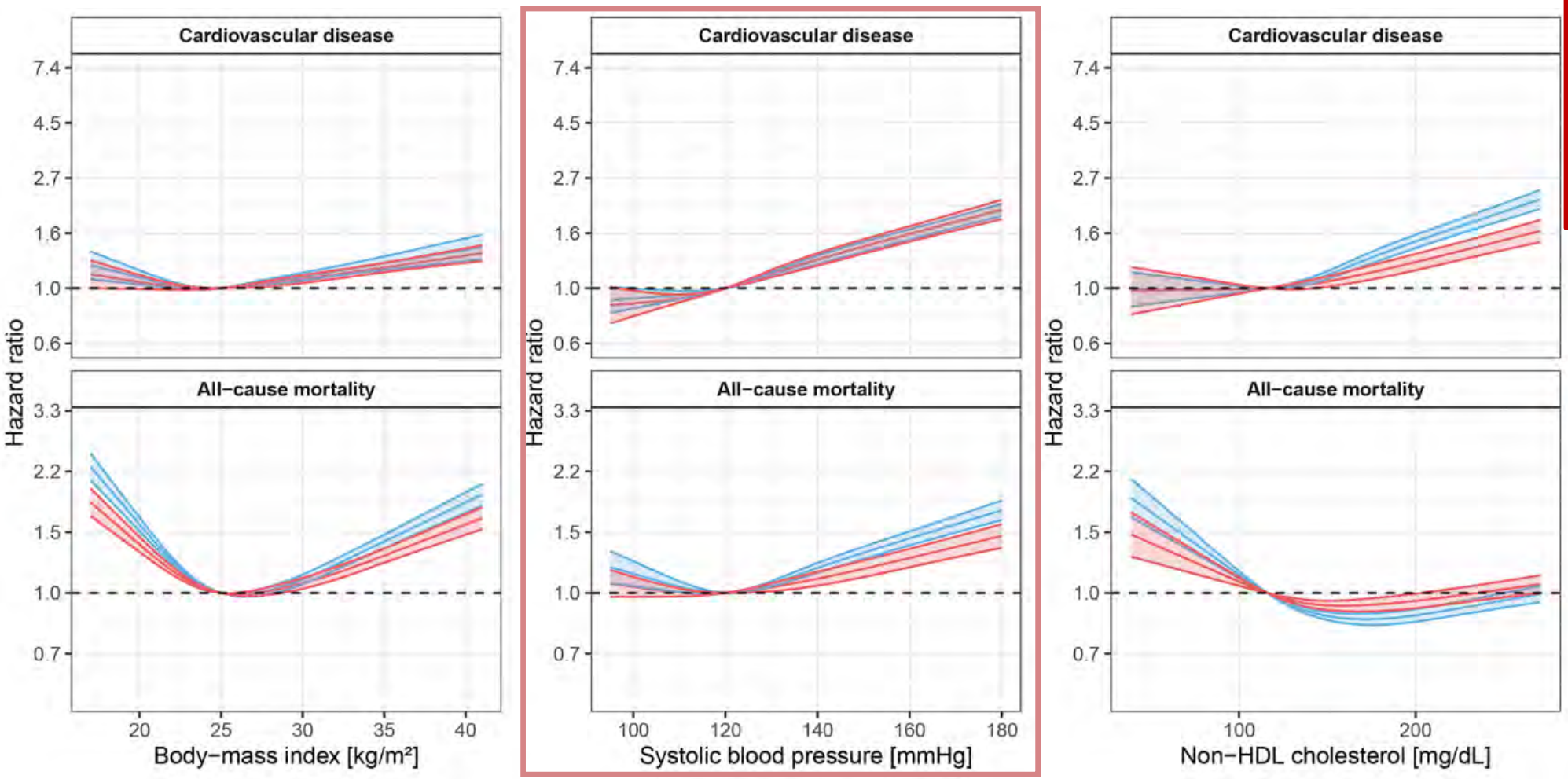


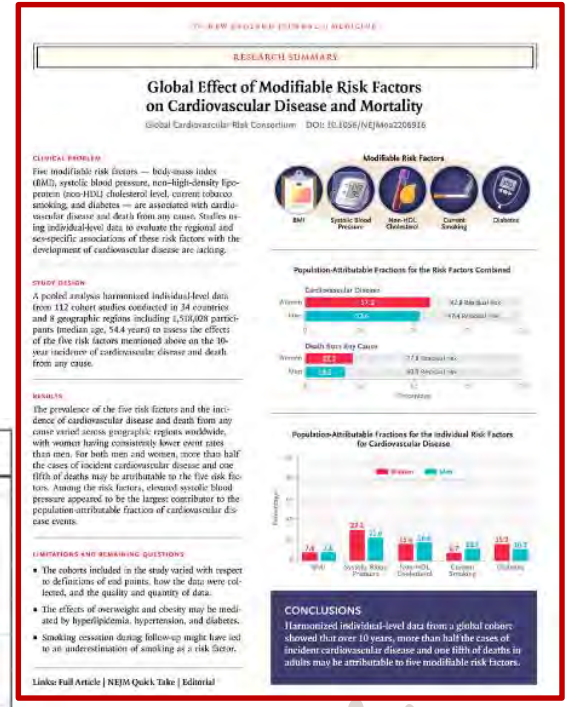


Ramon Luengo-Fernandez .European heart Journal 2023

Global Effect of Modifiable Risk Factors on Cardiovascular Disease and Mortality

112 cohorts, 34 countries; n> 1.5 mio




RESEARCH SUMMARY

Global Effect of Modifiable Risk Factors on Cardiovascular Disease and Mortality

Global Cardiovascular Risk Consortium

Modifiable Risk Factors

Population-Attributable Fractions for the Risk Factors Combined

Population-Attributable Fractions for the Individual Risk Factors for Cardiovascular Disease

CONCLUSIONS

Harmonized individual-level data from a global cohort showed that over 10 years, more than half the cases of incident cardiovascular disease and one fifth of deaths in adults may be attributable to five modifiable risk factors.

Women

Men

PAF CVD SBP:

W 29.3%

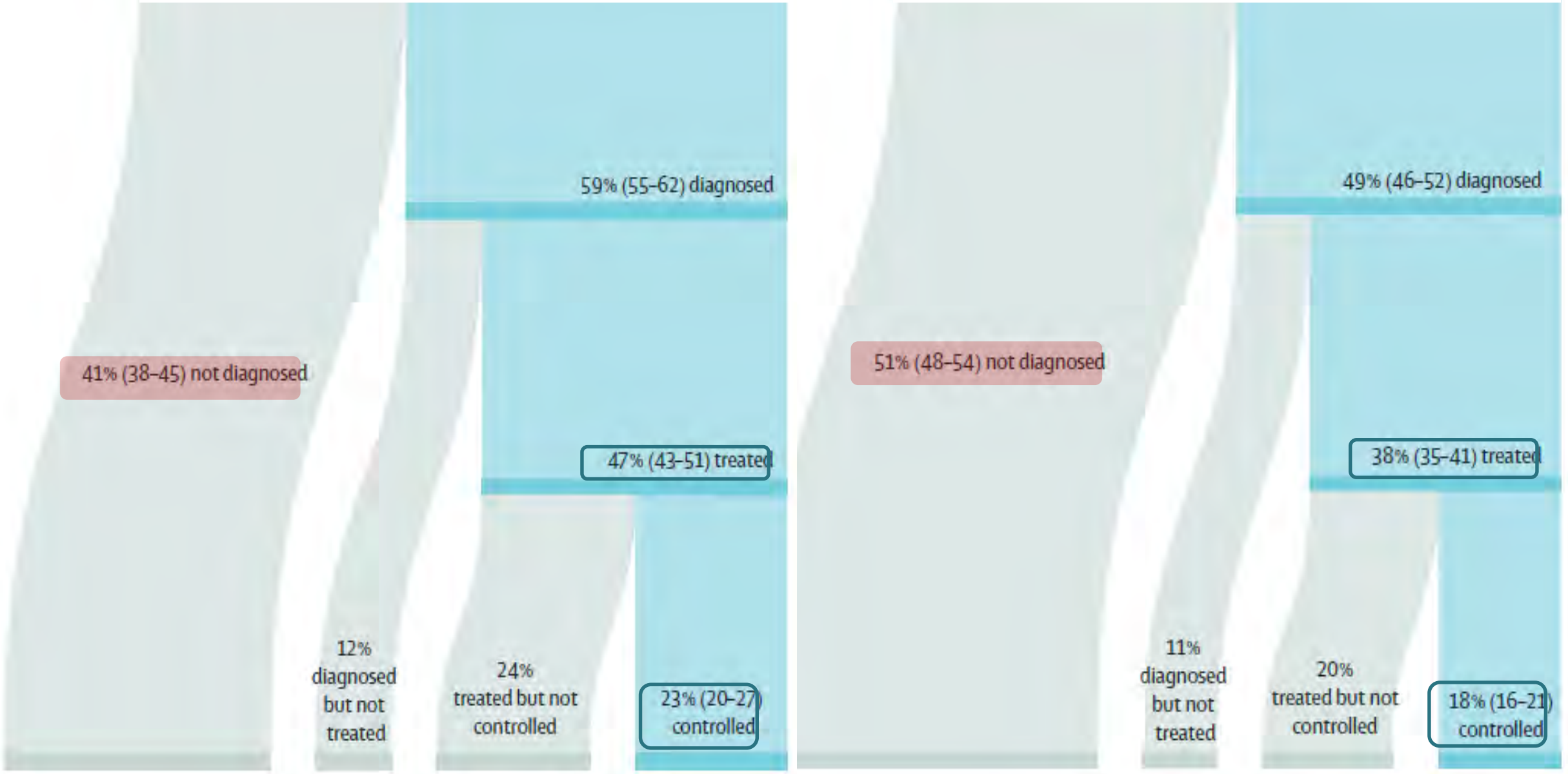
M 21.6%

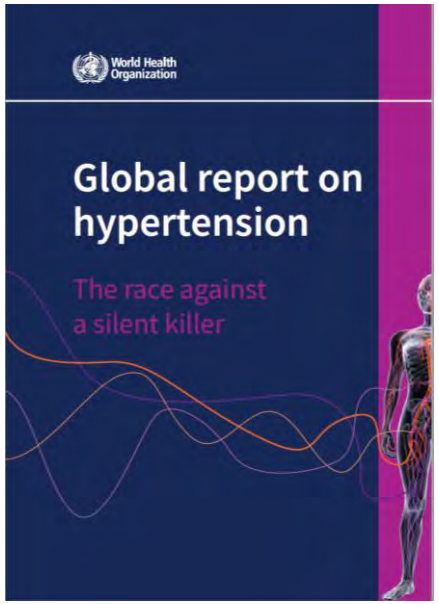
All women with hypertension (world)

Women:32% (30-34), stable

All men with hypertension (world)

Men: 34% (32-37), stable





Switzerland

Hypertension profile

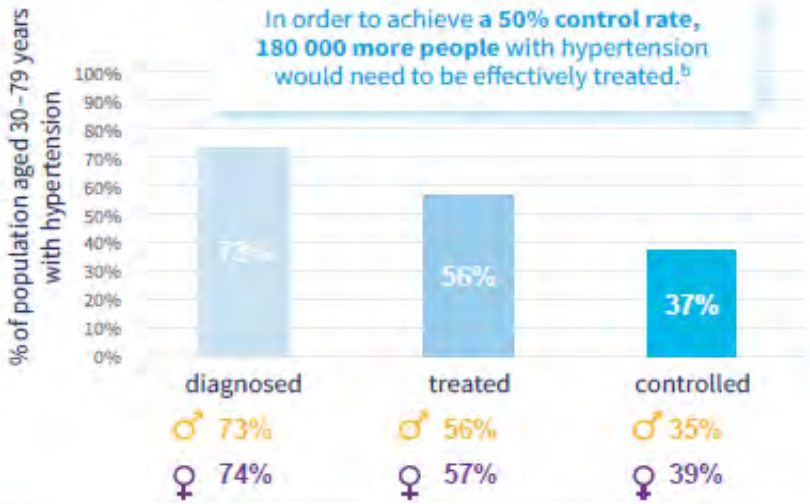
Total population (2019): 8 576 000

Total deaths (2019): 69 100

Age-standardized prevalence of hypertension among adults aged 30–79 years (2019)[a] ♀ 22% ♂ 26% ♀ 18%

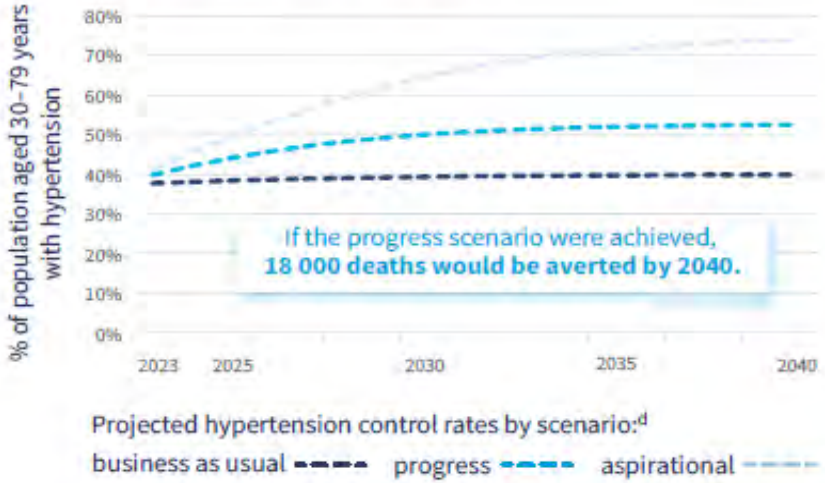
Prevalence of hypertension – global comparison (both sexes)[a]

Of the **1.5 million** adults aged 30–79 years with hypertension:



Trends in uncontrolled hypertension in adults aged 30–79 years[c]

Hypertension control rate scenarios



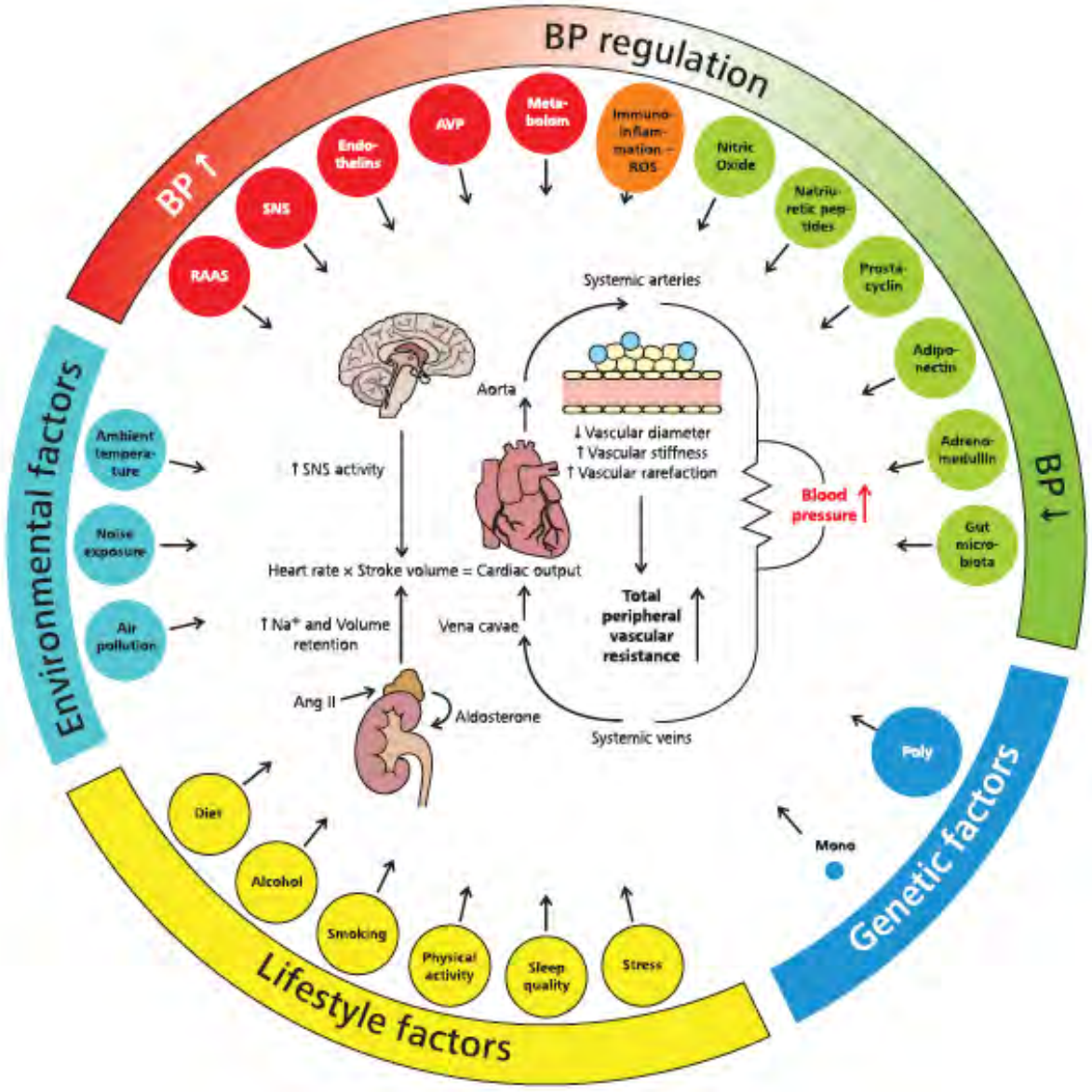
What is new and what has changed?

24 items…

198 pages…

Journal of Hypertension 2023

1. Modified and simplified criteria for evidence grading recommendations
2. Pathophysiological background of primary hypertension
3. Clinical BP measurements by different methods and in different settings and clinical conditions
4. Thorough description of office, ambulatory and home BP measurements and value in different demographic and clinical conditions
5. Upgrading of out-of-office BP measurements in hypertension management
6. New HMOD measurements and their clinical value in hypertension work-up
7. New CV risk factors and update on CV risk assessment
8. Update and comprehensive summary of secondary forms of hypertension
9. Update on lifestyle interventions
10. Update on threshold and targets for antihypertensive drug treatment, including their possible heterogeneity in demographic and clinical subgroups of patients
11. Confirmation of preferred use of RAS blockers, CCBs and Thiazide/Thiazide-like diuretics, and their various combinations for BP-lowering treatment. Inclusion of BBs among the major antihypertensive drugs
12. Update on available combination-based drug treatment strategies, including the quadpill and the polypill
13. Emphasis and update on the diagnosis and management of true resistant hypertension
14. Update on use and position of renal denervation for antihypertensive treatment
15. Impact of hypertension and its treatment on cognitive dysfunction and dementia
16. Management of hypertension in older people according to the frailty and functional level
17. Update on treatment of hypertension in HFrEF and HFpEF
18. New diagnostic approaches to diagnosis and treatment in hypertensive patients with AF
19. Update on treatment in CKD, including kidney transplantation
20. Update and novel treatment approaches to patients with type 2 diabetes
21. Epidemiology, diagnosis and treatment in different BP phenotypes
22. Diagnosis, treatment and follow-up of hypertension in demographic and clinical conditions not or only marginally addressed in previous guidelines:
    a. Children/adolescents and transition to adulthood
    b. Young patients
    c. Sex-related differences
    d. Pregnancy and puerperium
    e. Peripheral artery disease
    f. Aortic aneurism
    g. Valvular heart disease
    h. Treatment of hypertension in acute cerebrovascular diseases
    i. Hypertensive emergencies/urgencies
    j. Perioperative hypertension
    k. Obesity
    l. COVID-19
    m. Chronic inflammatory diseases
    n. Hypertension in oncology
    o. Baroreflex failure and dysautonomia
    p. Glaucoma
23. Detailed recommendations on patients' follow-up strategies, including assessment and minimization of nonadherence and clinical inertia.



Definitions

- Same definitions as in ESH 2018

Definition of BP categories, hypertension grades and stages according to office BP

Recommendations and statements	CoR	LoE
It is recommended that BP is classified as optimal, normal, high normal, or grade 1, 2 or 3 hypertension, according to office BP.	I	C
In addition to grades of hypertension, which are based on BP values, it is recommended to distinguish stage 1, 2, and 3 hypertension. Stage 1: Uncomplicated hypertension without HMOD, diabetes, CVD and without CKD ≥ stage 3. Stage 2: Presence of HMOD, diabetes, or CKD stage 3. Stage 3: Presence of CVD or CKD stage 4 or 5.	I	C

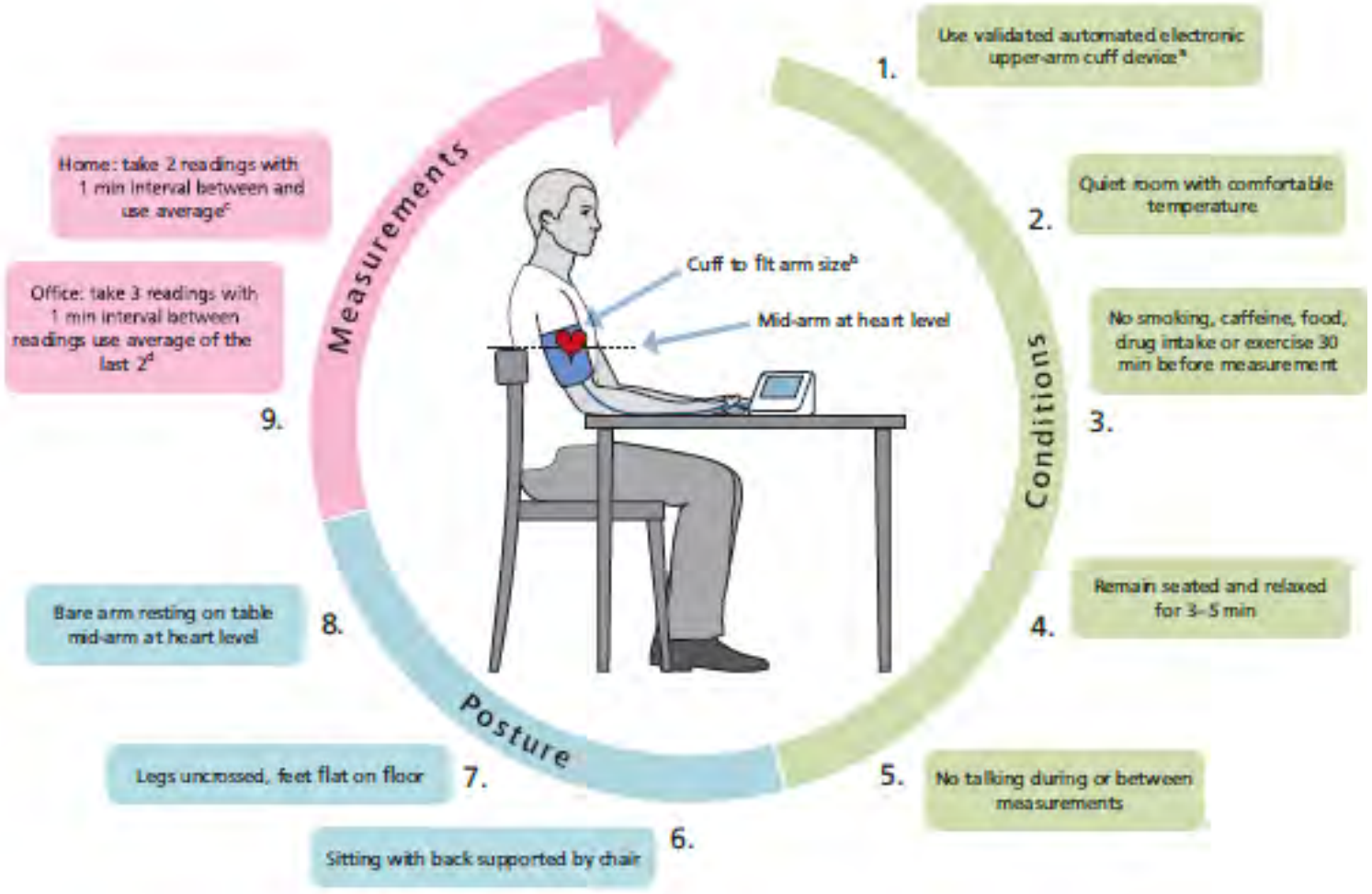
TABLE 1. Classification of office BP and definitions of hypertension grades

Category	Systolic (mmHg)		Diastolic (mmHg)
Optimal	<120	and	<80
Normal	120–129	and	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[a]	≥140	and	<90
Isolated diastolic hypertension[a]	<140	and	≥90

The BP category is defined by the highest level of BP, whether systolic or diastolic.
[a]Isolated systolic or diastolic hypertension is graded 1, 2 or 3 according to SBP and DBP values in the ranges indicated. The same classification is used for adolescents ≥16 years old (see Section 15.1).

! Sur ABPM, définition HTA: (1) jour ≥ 135/85mmHg
(2) nuit ≥ 120/70mmHg → (3) 24h ≥ 130/80mmHg

Mesure and monitoring



Office BP measurements

Recommendations and statements	CoR	LoE
Office BP is recommended for diagnosis of hypertension, because it is the one method by which hypertension-related risk, benefits of antihypertensive treatment, and treatment-related BP thresholds and goals are based.	I	A
Office BP measurements should be performed in standardized conditions, using a standard measurement protocol. Triplicate measurements should be taken and the average of the last two should be referred to as the representative value.	I	C
It is recommended to diagnose hypertension during at least 2 separate office visits (within 4 weeks) unless office BP indicates grade 3 hypertension (≥180/110 mmHg) or patients presents with hypertension related symptoms or there is evidence of HMOD or CVD.	I	C
At the first office visit, BP should be measured in both arms. A consistent between-arm SBP difference >15-20 mmHg suggests atheromatous disease and is associated with increased CV risk. All subsequent measurements should be made on the arm with the highest BP readings.	I	C
Out-of-office BP is a source of multiple BP-related information before and during treatment. It is therefore recommended to obtain additional information on BP values by ABPM or HBPM or both if available.	I	C

2021 European Society of Hypertension practice guidelines for office and out-of-office blood pressure measurement

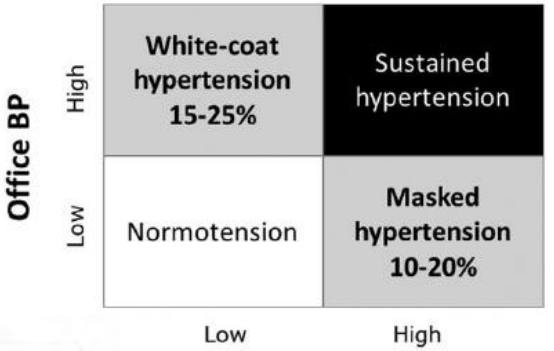


TABLE 4. Interpretation of average OBP (at least 2-3 visits with 2-3 measurements per visit)

	Normal-optimal BP (<130/85 mmHg)	High-normal BP (130–139/85–89 mmHg)	Hypertension Grade 1 (140–159/90–99 mmHg)	Hypertension Grade 2 and 3 (≥160/100 mmHg)
Diagnosis	Normotension highly probable	Consider MH	Consider WCH	Sustained hypertension highly probable
Action	Remeasure after 1 year (6 months in those with other risk factors)	Perform HBPM and/or ABPM. If not available confirm with repeated office visits		Confirm within a few days or weeks[a]. Ideally use HBPM or ABPM

[a]Treat immediately if OBP is very high (e.g. ≥180/110 mmHg) and there is evidence of target organ damage or CVD.

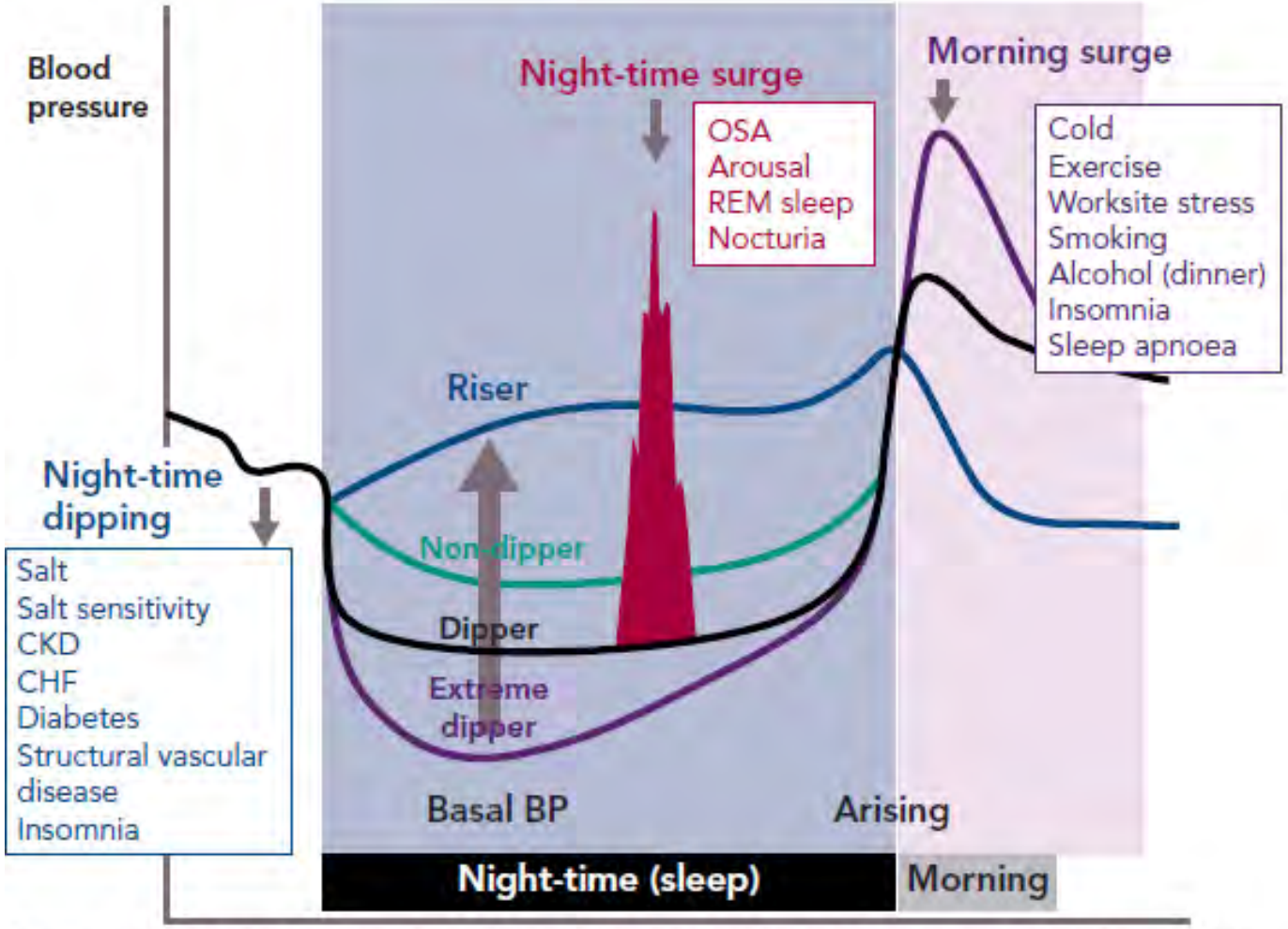
TABLE 12. Clinical utility of office and out-of-office BP measurement methods

Clinical use	Office	Home	24 h ambulatory	Pharmacy
Screening	+++	+	–	++
Initial diagnosis	+	++	+++	–
Treatment titration	+	++	++	–
Follow-up	++	+++	+	+
Main indication	Screening of untreated individuals. Follow-up of treated patients	Long-term follow-up of treated patients (preferred method)	Initial diagnosis (preferred method)	Screening of untreated individuals. Follow-up of treated patients
Hypertension (mmHg)	≥140/90	≥135/85	≥130/80	≥135/85 (?)

Stergiou et al. Journal of Hypertension 2021

Sur profil tensionnel: PA nocturne

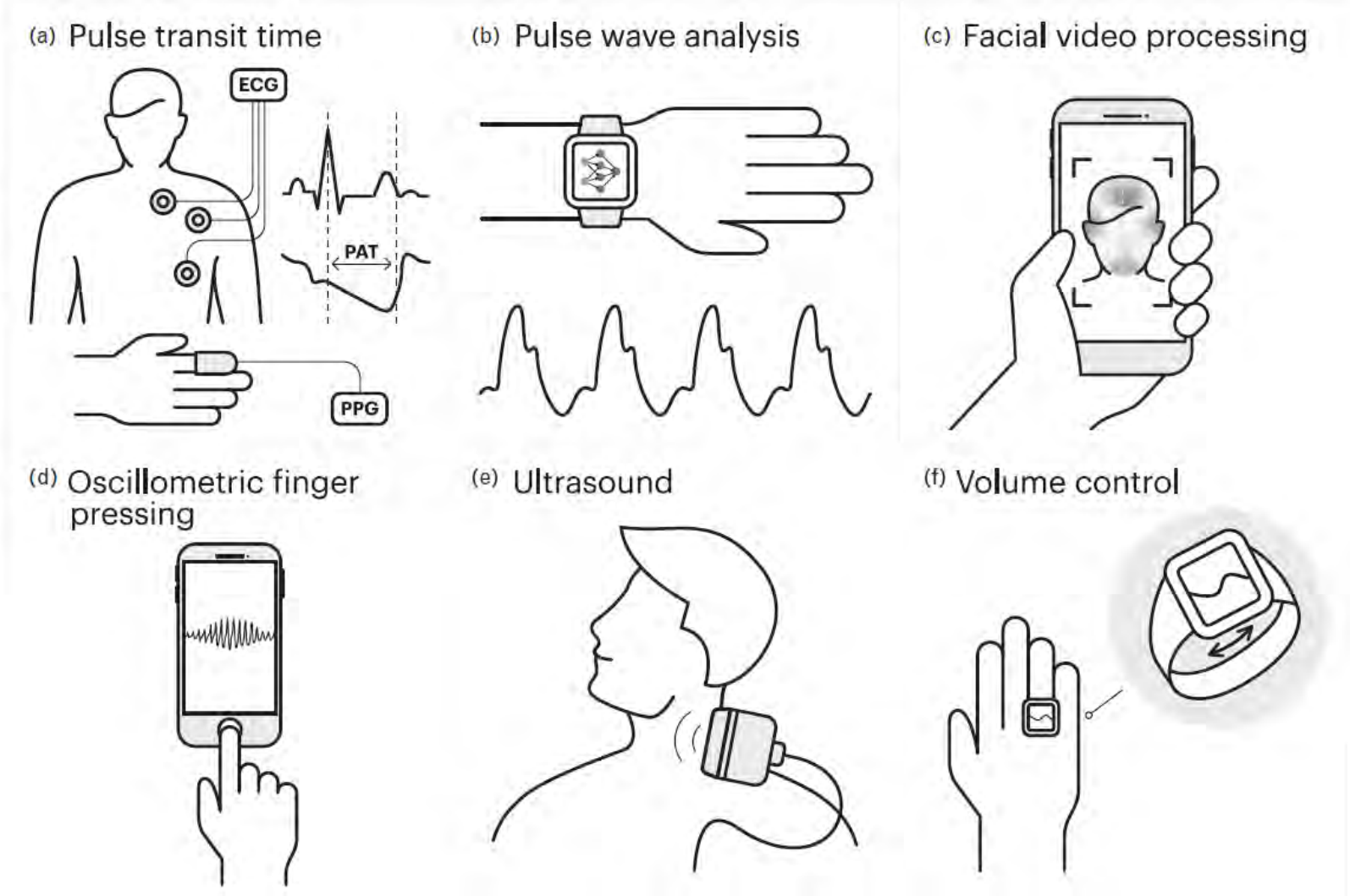
Nocturnal HT
SBP≥120 ± DBP ≥70
Normal dip 10-20%

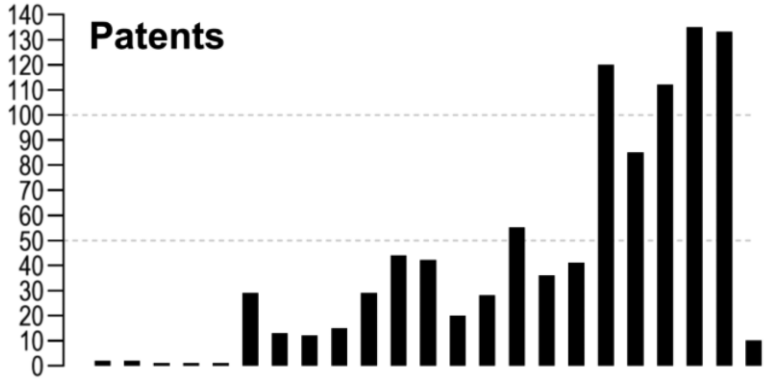


CHF = chronic heart failure; CKD = chronic kidney disease; OSA = obstructive sleep apnoea; REM = rapid eye movement. Source: Kario et al. 2018.[8] Reproduced with permission from Wolters Kluwer Health.

Cappucio F. ECRJ 2020

Cuffless blood pressure measuring devices: review and statement by the European Society of Hypertension Working Group on Blood Pressure Monitoring and Cardiovascular Variability





Journal of Hypertension 2022

Cuffless devices: validation

	IEEE Standard 1708–2014 [18] & 1708a–2019 [19]	ISO Standard 81060-3 : 2022 [12]	ESH Recommendations 2023 (current document)
Device type	Cuffless wearable BP devices	Cuffless continuous BP devices	Cuffless intermittent BP devices (9 types – cf.)
D. Before recalibration	Yes	Yes	Yes, for some device types[a]
Test and reference BP measurement	Simultaneous or sequential	Simultaneous	Sequential (24-h BP simultaneous)
Pass requirements (BP difference)	≤7 mmHg (mean absolute difference)	≤6 ± 10 mmHg (mean ± SD of difference)	≤5 ± 8 mmHg (mean ± SD of difference)

Journal of Hypertension 2023

Workup HT: CV risk + Target Organs

TABLE 2. Factors that influence CV risk in patients with hypertension

Parameter for risk stratification, which are included in SCORE2 and SCORE2-OP
- Sex (men >women)
- Age
- Level of SBP[a]
- Smoking – current or past history
- Non-HDL cholesterol

Established and suggested novel factors
- Family or parental history of early onset hypertension
- Personal history of malignant hypertension
- Family history of premature CVD (men aged <55 years; women aged <65 years)
- Heart rate (resting values >80 bpm)
- Low birth weight
- Sedentary lifestyle
- Overweight or Obesity
- Diabetes
- Uric acid
- Lp(a)
- Adverse outcomes of pregnancy (recurrent pregnancy loss, preterm delivery, hypertensive disorders, gestational diabetes)
- Early-onset menopause
- Frailty
- Psychosocial and socioeconomic factors
- Migration
- Environmental exposure to air pollution or noise

Additional clinical conditions or comorbidities
- True resistant hypertension
- Sleep disorders (including OSA)
- COPD
- Gout
- Chronic inflammatory diseases
- Nonalcoholic fatty liver disease (NASH)
- Chronic infections (including long COVID-19)
- Migraine
- Depressive syndromes
- Erectile dysfunction

Hypertension-mediated organ damage (HMOD)
- Increased large artery stiffness:
- Pulse pressure (in older people) ≥60 mmHg
- Carotid–femoral PWV >10 m/s (if available)
- Presence of non-hemodynamically significant atheromatous plaque (stenosis) on imaging
- ECG LVH (Sokolow–Lyon index >35 mm, or R in aVL ≥11 mm; Cornell voltage-duration product (+6 mm in women) >2440 mm*ms, or Cornell voltage >
  in men or >20 mm in women)
- Echocardiographic LVH (LV mass index: men >50 g/m$^{2.7}$; women >47 g/m$^{2.7}$ (m = height in meters); indexation for BSA may be used in normal-weight pa
  >115 g/m^2 in men and >95/m^2 in women
- Moderate increase of albuminuria 30–300 mg/24 h or elevated ACR (preferably in morning spot urine) 30–300 mg/g
- CKD stage 3 with eGFR 30–59 ml/min/1.73 m^2
- Ankle–brachial index <0.9
- Advanced retinopathy: hemorrhages or exudates, papilledema

Established cardiovascular and kidney disease
- Cerebrovascular disease: ischemic stroke, cerebral hemorrhage, TIA
- Coronary artery disease: myocardial infarction, angina, myocardial revascularization
- Presence of hemodynamically significant atheromatous plaque (stenosis) on imaging
- Heart failure, including heart failure with preserved ejection fraction
- Peripheral artery disease
- Atrial fibrillation
- Severe albuminuria > 300 mg/24 h or ACR (preferably in morning urine) >300 mg/g
- CKD stage 4 and 5, eGFR < 30 mL/min/1.73 m^2

Hypertension disease staging	Other risk factors, HMOD, CVD or CKD	BP (mmHg) grading			
		High-normal SBP 130–139 DBP 85–89	Grade 1 SBP 140–159 DBP 90–99	Grade 2 SBP 160–179 DBP 100–109	Grade 3 SBP ≥ 180 DBP ≥ 110
Stage 1	No other risk factors[a]	Low risk	Low risk	Moderate risk	High risk
	1 or 2 risk factors	Low risk	Moderate risk	Moderate to high risk	High risk
	≥3 risk factors	Low to moderate risk	Moderate to high risk	High risk	High risk
Stage 2	HMOD, CKD grade 3, or diabetes mellitus	Moderate to high risk	High risk	High risk	Very high risk
Stage 3	Established CVD or CKD grade ≥4	Very high risk	Very high risk	Very high risk	Very high risk

	<50 years	60–69 years	≥70 years
	<2.5%	<5%	<7.5%
	2.5 to <7.5%	5 to <10%	7.5 to <15%
	≥7.5%	≥10%	≥15%

Complementary risk estimation in Stage 1 with SCORE2/SCOR2-OP

CV risk assessment

ESC European Society of Cardiology

The ESC · Congresses & Events · Journals · Guidelines

European Society of Cardiology · Education · Practice Tools & Support · CVD Prevention Toolbox

Practice Tools & Support

- CVD Prevention Toolbox
- EACVI Imaging Toolboxes
- ACVC Clinical Decision-Making Toolkit
- Pocket Guidelines App
- Guidelines Summary Cards
- ACS Trials Comparison Tool
- Atrial Fibrillation Patient Website
- Heart Failure Patient Website
- ESC Science In Your Language
- ESC Prevention of CVD Programme

SCORE2 and SCORE2-OP

Risk assessment models to estimate the 10-year risk of cardiovascular disease in Europe.

SCORE2 and SCORE2-OP

Two new algorithms, SCORE2 and SCORE2-OP (older persons), were published in June 2021:

SCORE2

SCORE2 risk prediction algorithms: new models to estimate 10-year risk of cardiovascular disease in Europe

SCORE2 working group and ESC cardiovascular risk collaboration

European Heart Journal, ehab309, https://doi.org/10.1093/eurheartj/ehab309

SCORE2-OP

SCORE2-OP risk prediction algorithms: estimating incident cardiovascular event risk in older persons in four geographical risk regions

SCORE2-OP working group and ESC cardiovascular risk collaboration

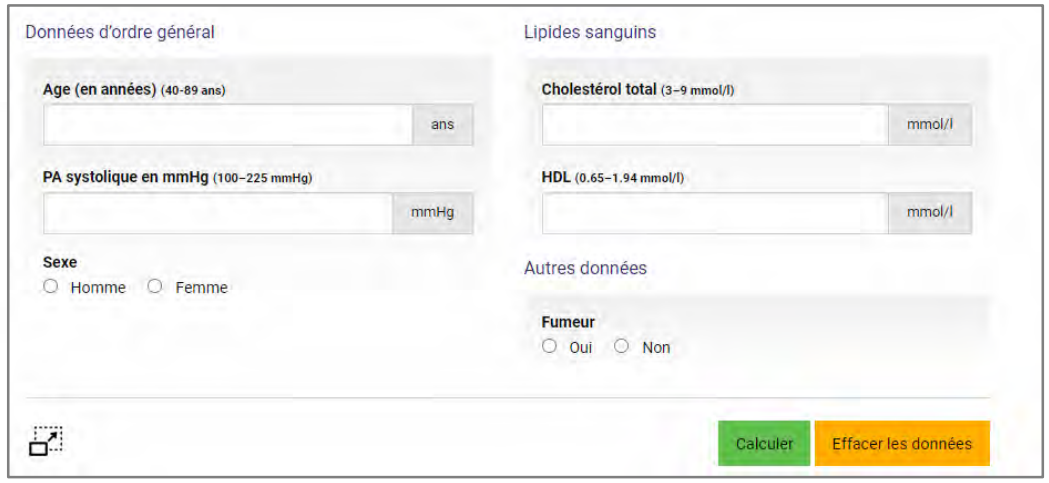
European Heart Journal, ehab312, https://doi.org/10.1093/eurheartj/ehab312

SCORE2 and SCORE2-OP interactive version

Access HeartScore, the interactive tool based on the SCORE2 and SCORE2-OP algorithms.

Risk assessment in hypertension with SCORE2 and SCORE2-OP

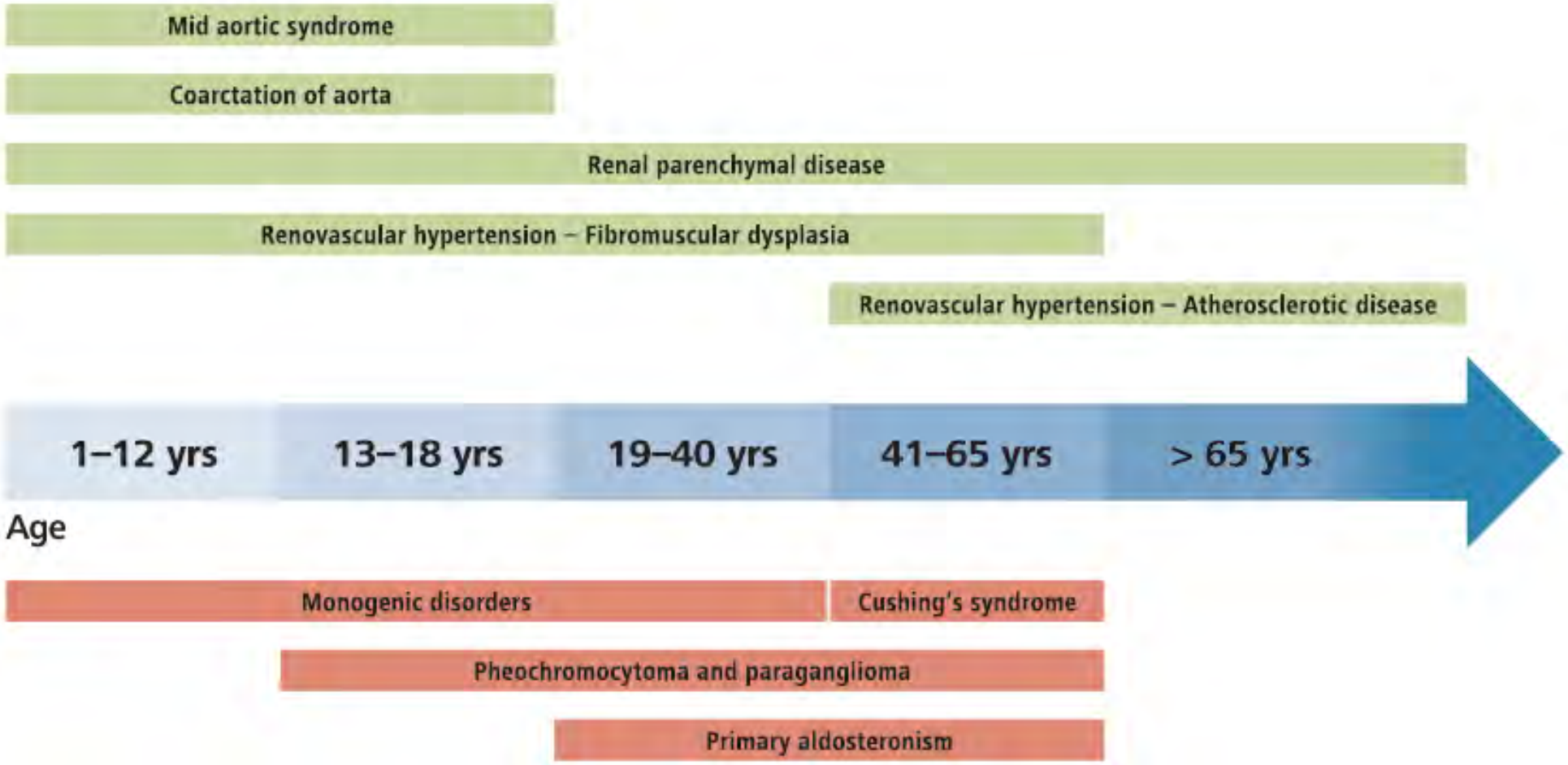
Recommendations and statements	CoR	LoE
CV risk assessment with the SCORE2 and SCOR2-OP system is recommended for hypertensive patients who are not already at high or very high risk due to established CVD or CKD, long-lasting or complicated diabetes, severe HMOD (e.g. LVH) or a markedly elevated single risk factor (e.g. cholesterol, albuminuria).	I	B



Secondary HT

TABLE 13. Patient characteristics that should raise the suspicion of secondary hypertension

Younger patients (<40 years) with grade 2 or 3 hypertension or hypertension of any grade in childhood
Sudden onset of hypertension in individuals with previously documented normotension
Acute worsening of BP control in patients with previously well controlled by treatment
True resistant hypertension hypertension
Hypertensive emergency
Severe (grade 3) or malignant hypertension
Severe and/or extensive HMOD, particularly if disproportionate for the duration and severity of the BP elevation
Clinical or biochemical features suggestive of endocrine causes of hypertension
Clinical features suggestive of atherosclerotic renovascular disease or fibromuscular dysplasia
Clinical features suggestive of obstructive sleep apnea
Severe hypertension in pregnancy (>160/110 mmHg) or acute worsening of BP control in pregnant women with preexisting hypertension



Prevalence: 6–20%[a]

Suggestive symptoms, signs and findings

Resistant hypertension
Grade 2 or 3 hypertension
Hypokalemia/Potassium in the low-normal range
Atrial fibrillation
OSA
Adrenal incidentaloma[b]
Family history of PA/early stroke

1st choice screening test[c]

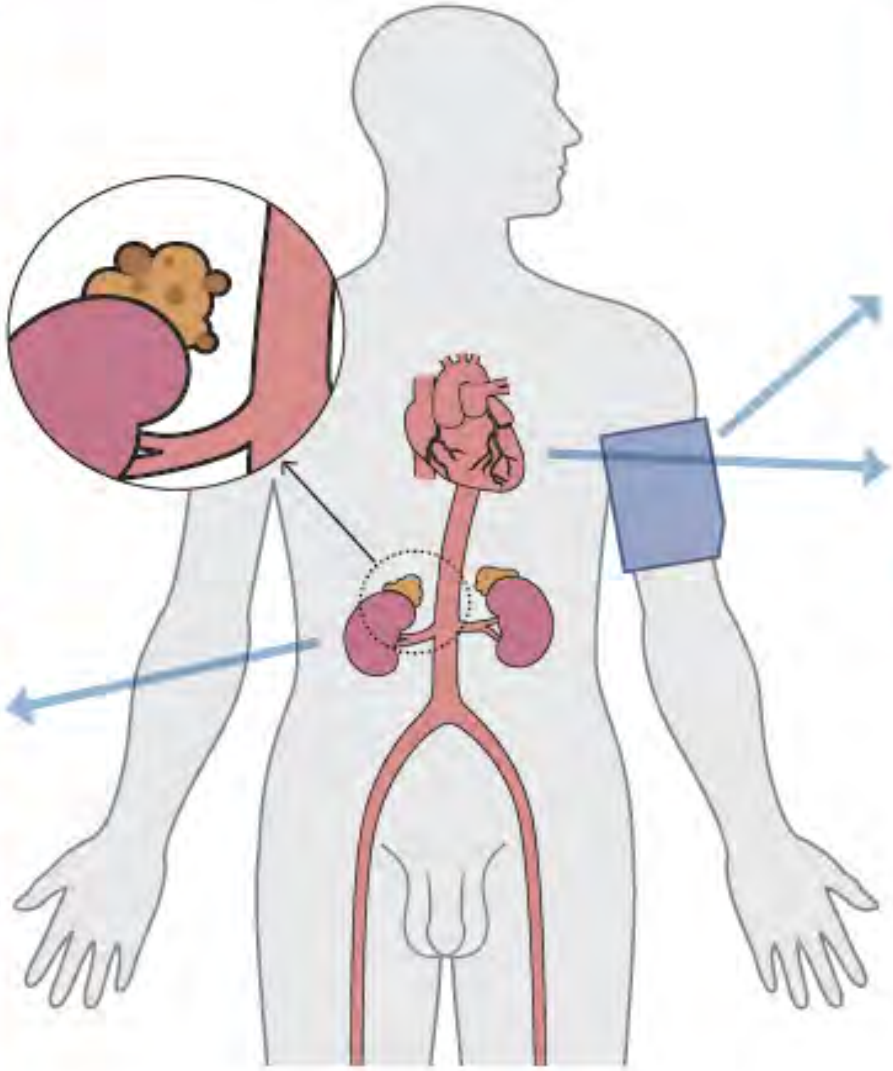
Plasma aldosterone to renin ratio (ARR)

Further work-up[d]

CT scanning
IV saline infusion test (SIT)
Fludrocortisone suppression test (FST)
Oral sodium loading test (SLT)
Captopril challenge test (CCT)
Adrenal vein sampling
Genetic testing in selected cases[e]

Treatment

Surgical treatment (laparoscopic adrenalectomy) – unilateral PA
Medical treatment – bilateral adrenal disease[f]



Cardiovascular phenotype

24 ABPM – true resistant hypertension, frequent non-dipping

- LVH
- Decreased diastolic function
- Myocardial fibrosis (MRI)

Increased CV Risk and mortality

Primary Hyperaldosteronism

Tests activité renine/aldo on neutral treatment: doxazocine, diltiazem (non DHP calcium blocker)

Plasmatic renine activity
+ aldostérone
Morning 8h, fasting

+ Aldosteronuria 24h
(N <=10ug/j)

Médications	Aldostérone	Rénine	RAR	Délai d'interruption (en semaines)
Diurétiques thiazidiques	→↑	↑↑	↓ (FN)	2
Diurétiques de l'anse	↑	↑↑	↓ (FN)	2
Antag. Rc minéralocortic.	↑	↑↑	↓(FN)	6
IEC et Sartan	↓	↑↑	↓ (FN)	2
Inhibiteur de la rénine	↓	↑ si RD ↓ si ARP	↑ (FN) ↑ (FP)	6
β-bloqueur	↓	↓↓	↑ (FP)	2
Agoniste alpha2 central (clonidine)	↓	↓↓	↑ (FP?)	2 (idéalement)
α1bloquant,non DHP doxazosin, moxonidine	→	→	→	
Anti-calciques	↓ (DHP)	↑ (DHP)	↓ (DHP)	2 (DHP)

(A) Atherosclerotic renovascular disease

Prevalence: 6–14%[a]

Suggestive symptoms, signs and findings

Resistant hypertension
Flash pulmonary edema
Rapidly declining kidney function
Acute renal function degradation on ACEi or ARB
Generalized atherosclerosis[b]

1st choice screening test

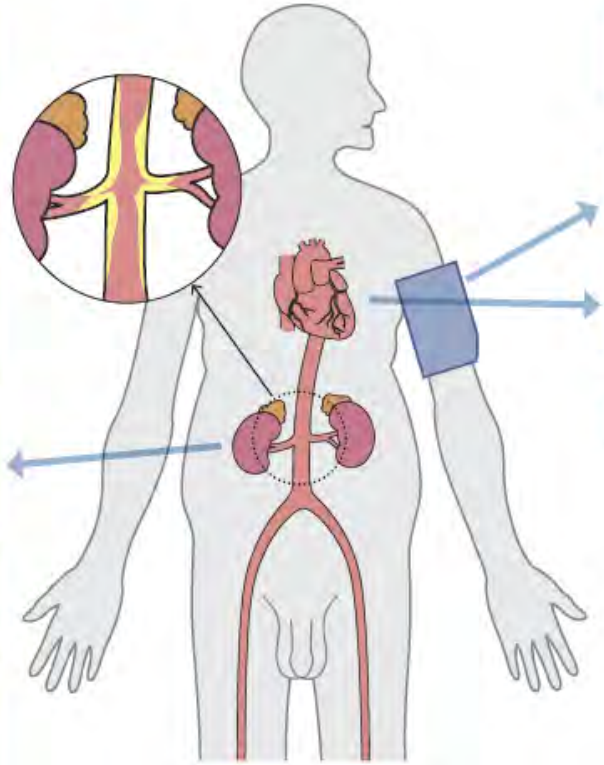
Renal artery duplex ultrasound; otherwise angio-CT or angio-MR

Further work-up

Angio-CT or angio-MR
Invasive catheter angiography

Treatment[c,d]

Antihypertensive treatment
Strict control of CV risk factors
Revascularization (selected cases)

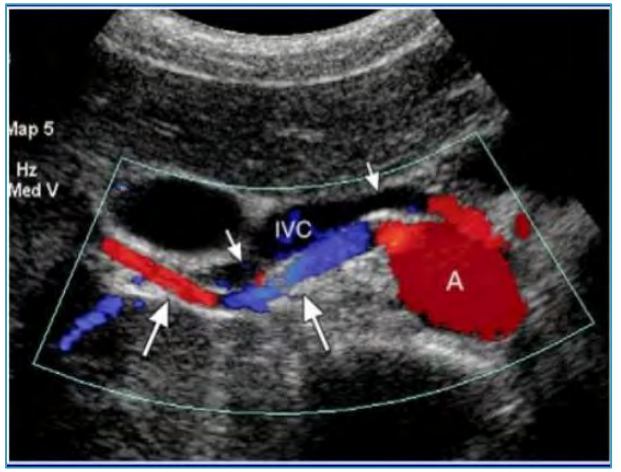


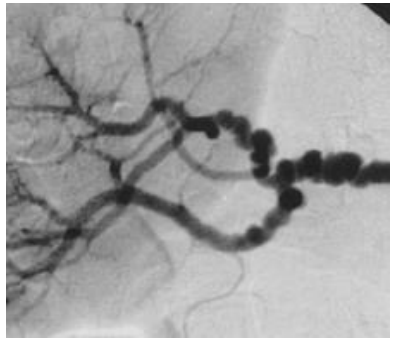
Cardiovascular phenotype

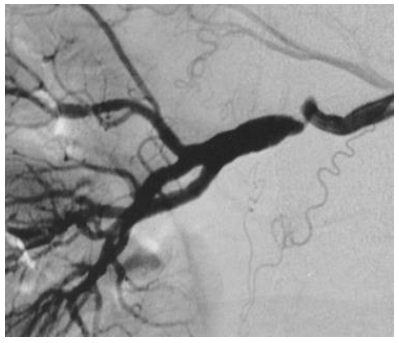
24h ABPM – resistant hypertension, frequent non-dipping

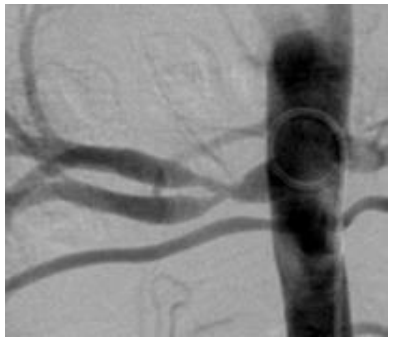
- LVH
- Decreased diastolic function
- Decreased systolic function

Increased CV Risk and mortality









(B) Fibromuscular Dysplasia

Prevalence: <1 to 6%[a]

Suggestive symptoms, signs and findings

Early-onset/ severe hypertension
Migraine
Pulsatile tinnitus

1st choice screening test[b]

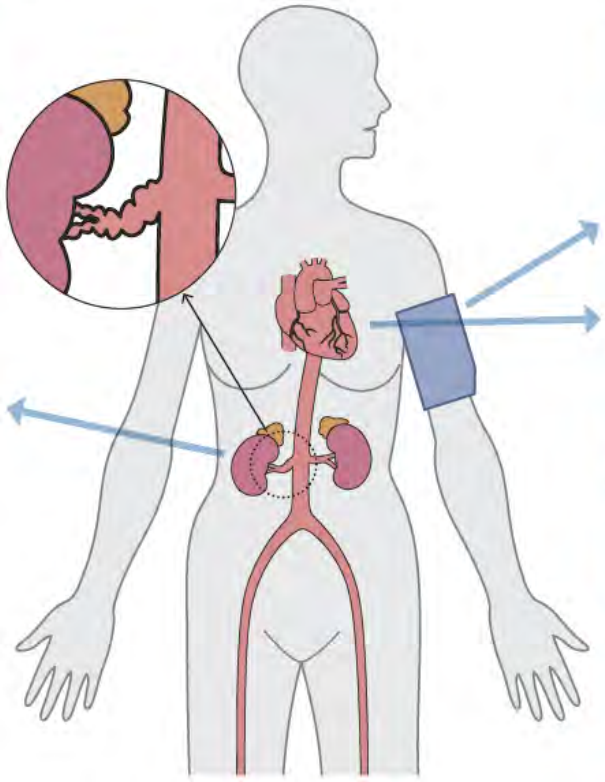
Renal artery duplex ultrasound; otherwise angio-CT or angio-MR

Treatment

Antihypertensive treatment
Angioplasty without stenting[c,d]

Follow-up

- Whole body angio-CT or angio-MR at diagnosis[e]
- Indefinite follow-up



Cardiovascular phenotype

24h ABPM – early onset or resistant hypertension

Frequent in patients with Spontaneous Coronary Artery Dissection (SCAD)

May affect all medium sized arteries (most frequent: renal and cervical arteries)

Often associated with arterial dissections and aneurysms

Cardiovascular phenotype:
From asymptomatic to resistant hypertension, stroke, renal, mesenteric or myocardial infarction

(E) Cushing's syndrome

Prevalence: 2–5%[a]

Suggestive symptoms and signs

Resistant hypertension
Easy bruising, facial plethora, ‚moon' face, skin thinning
Proximal myopathy
Weight gain with centripetal distribution of body fat
Diabetes mellitus

1st choice screening test[a]

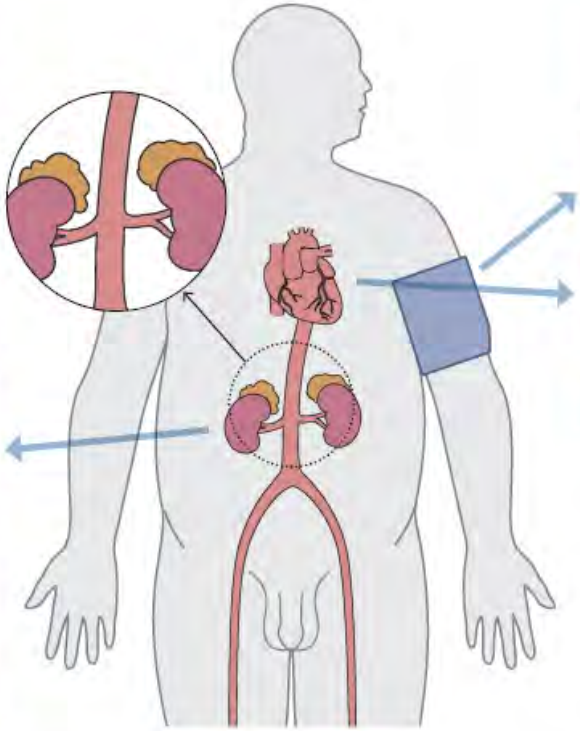
Overnight 1 mg dexamethasone suppression test
24-h urinary free cortisol
Late-night salivary cortisol

Further work-up

Morning plasma ACTH
ACTH stimulation by CRH or desmopressin
CT

Treatment

Medical – normalization of cortisol levels
Surgical – first line treatment for Cushing's disease , ectopic Cushing's syndrome and ACTH-independent hypercortisolism



Cardiovascular phenotype

24h ABPM – frequent non-dipping
Short-term BP variability

- LVH
- Decreased systolic function
- Decreased diastolic function

Increased CV Risk and mortality

(D) Pheochromocytoma and paraganglioma

Prevalence: <1%[a]

Suggestive symptoms and signs[b]

- paroxysmal symptoms (such as headache, sweating, palpitation, increased HR)
- large BP variation
- CV manifestations (e.g. MI, arrhythmias, Takotsubo cardiomyopathy)

1st choice screening test

Plasma or urinary free metanephrines

Further work-up

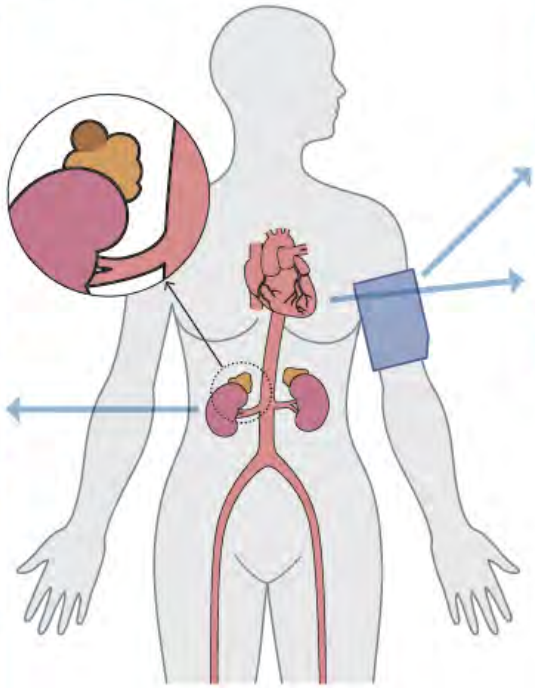
Contrast enchanced CT or MRI
Functional imaging
Genetic testing[c]

Treatment[d]

Surgical resection
(Pheochromocytoma: minimally invasive laparoscopic adrenalectomy)

Follow-up[e]

In most cases > 10 yrs



Cardiovascular phenotype

24h ABPM – frequent non-dipping

- LVH
- Decreased systolic function
- Myocardial fibrosis (MRI)

Increased CV Risk and mortality

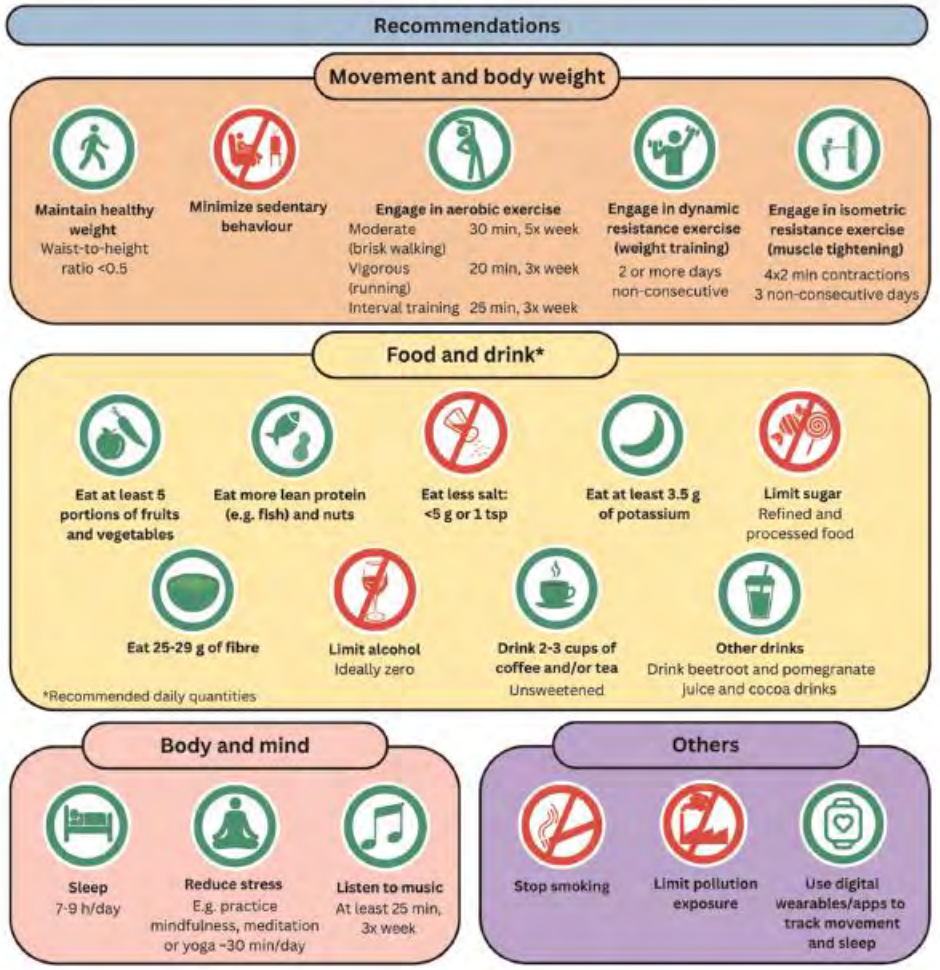
Hypertension and drugs

NSAIDs	Inhibition of COX-1 and 2 decreasing PG I2 and E2 synthesis with subsequent reduction in urinary Na excretion and an increased systemic vascular resistance.	Mild, dose-dependent increase in BP. Increased risk with age, preexisting hypertension, salt-sensitive patients, patients with renovascular hypertension.
Paracetamol (acetaminophen)	Presumably via inhibition of cydooxygenases and reduced production of prostaglandins.	Increased relative risk of 1.34 of hypertension with almost daily paracetamol use.
Estrogens and progestins	Increased renin synthesis (by estrogens) leading to RAS activation and subsequent Na^+ and water retention.	Mild, sustained increase in BP (6/3 mmHg increase with high doses of estrogen >50 μg of estrogen and 1–4 μg progestin) but can be severe, common in premenopausal women, cause hypertension in 5% of women.
Glucocorticoids	Enhanced Na^+ reabsorption and fluid retention via stimulation of mineralocorticoid receptors. Increased systemic vascular resistance due to upregulation of AT1 receptors on vascular smooth muscle cells.	Dose-dependent, low doses have less effect on BP, more common in older patients, or with a family history of primary hypertension.
Calcineurin inhibitors	Reduced NO production, ET-1 overproduction, systemic and renal vasoconstriction, renal Na^+ retention.	Dose-dependent, mild-to-moderate increase in BP. Severe hypertension has been reported. Increased risk with preexisting hypertension, elevated creatinine levels and maintenance therapy with corticosteroids. See Section 20.8.2
Antidepressants SNRIs	Increased noradrenaline release causing adrenergic activation and increased SNS activity.	Dose-dependent, mild (2/1 mmHg) increase in BP.
Nasal decongestants	Vasoconstriction due to stimulation of alpha-1 receptors on vascular smooth muscles.	Dose-dependent, sustained increase in BP.
Erythropoietin-stimulating agents	Increased thromboxane, reduced prostacyclin levels and activation of the local RAS. Increased ET-1 production, decreased NO synthesis with subsequent vasoconstriction.	Dose-dependent, mild increase in BP, increased risk with preexisting hypertension, or when the initial hematocrit level is low. See Section 20.8.2
Stimulants - Modafinil - Amphetamines - Methylphenidate	 Block noradrenaline or dopamine reuptake. Promote release of catecholamines	
VEGF inhibitors	Decreased NO production via VEGFR-2 antagonism and stimulation of ET-1 receptors promoting vasoconstriction.	A class effect. The incidence of hypertension is dose-related, risk is increased by preexisting hypertension, old age and overweight. See Section 20.8.2.
Substances of abuse - MDMA - PCP - Methamphetamine - Cocaine - Alcohol	Increased release and inhibited reuptake of monoamine neurotransmitters with subsequent SNS activation. Increased CNS catecholamine release with decreased neuronal uptake. Cocaine induces acute sympathomimetic effects and chronic HMOD, i.e. an increase in arterial wall stiffness. Alcohol increases SNS and RAS activity.	Cocaine induces both acute and chronic increases in BP. Alcohol causes a dose-dependent, sustained increase in BP.
Herbal products - Licorice - Ephedra - St. John's wort - Yohimbine - Ginseng (high doses) - Ma huang	Chronic excessive liquorice use mimics hyperaldosteronism by stimulating the mineralocorticoid receptor and inhibiting cortisol metabolism. Ephedra activates the alpha-1 receptor increasing SNS activity.	Licorice: Dose-dependent, sustained increase in BP characterized by hypokalemia, metabolic alkalosis and suppressed plasma renin activity and aldosterone levels Yohimbine causes acute, dose-dependent increase in BP.
Diet pills - Sibutramine - Phenylpropanolamine	Increased levels of norepinephrine with subsequent activation of noradrenergic transmission	Mild increase in BP.

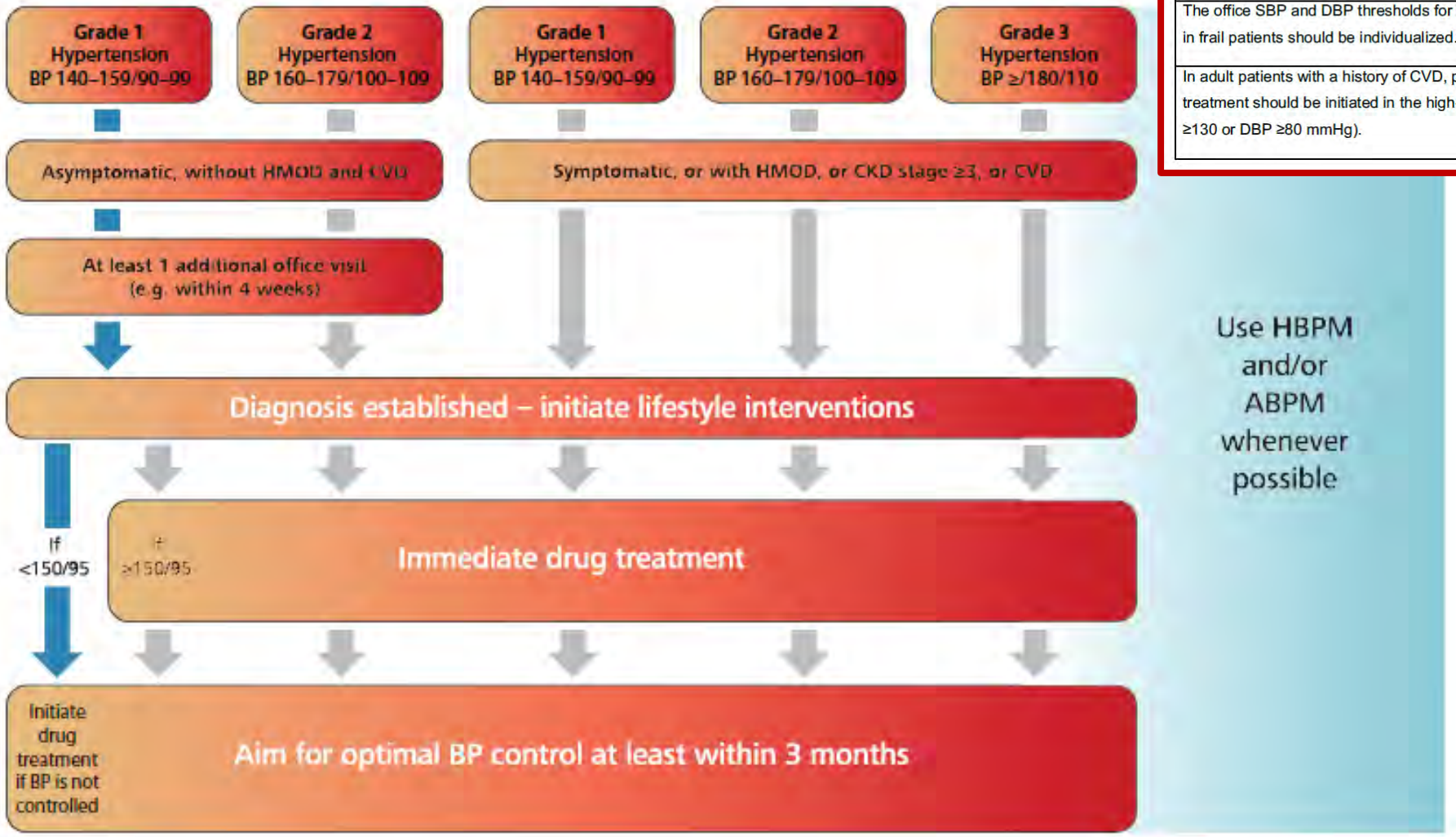
Treatments

Treatments (1) Health life style

Recommendations and statements	CoR	LoE
In adults with elevated BP who are overweight or obese, weight reduction is recommended to reduce BP and improve CV outcomes.	I	A
Preferred dietary products include vegetables, fruits, beans, nuts, seeds, vegetable oils, and fish and poultry among meat products. Fatty meats, full-fat dairy, sugar, sweetened beverages, and sweets should be limited. Overall, a healthy dietary pattern including more plant-based and less animal-based food is recommended.	I	B
In adults with hypertension consuming a high sodium diet (most Europeans), salt substitutes replacing part of the NaCl with KCl is recommended to reduce BP and the risk for CVD.	I	A
Dietary salt (NaCl) restriction is recommended for adults with elevated BP to reduce BP. Salt (NaCl) restriction to < 5 g (~2g sodium) per day is recommended.	I	B
Increased potassium consumption, preferably via dietary modification, is recommended for adults with elevated BP, except for patients with advanced CKD.	I	B



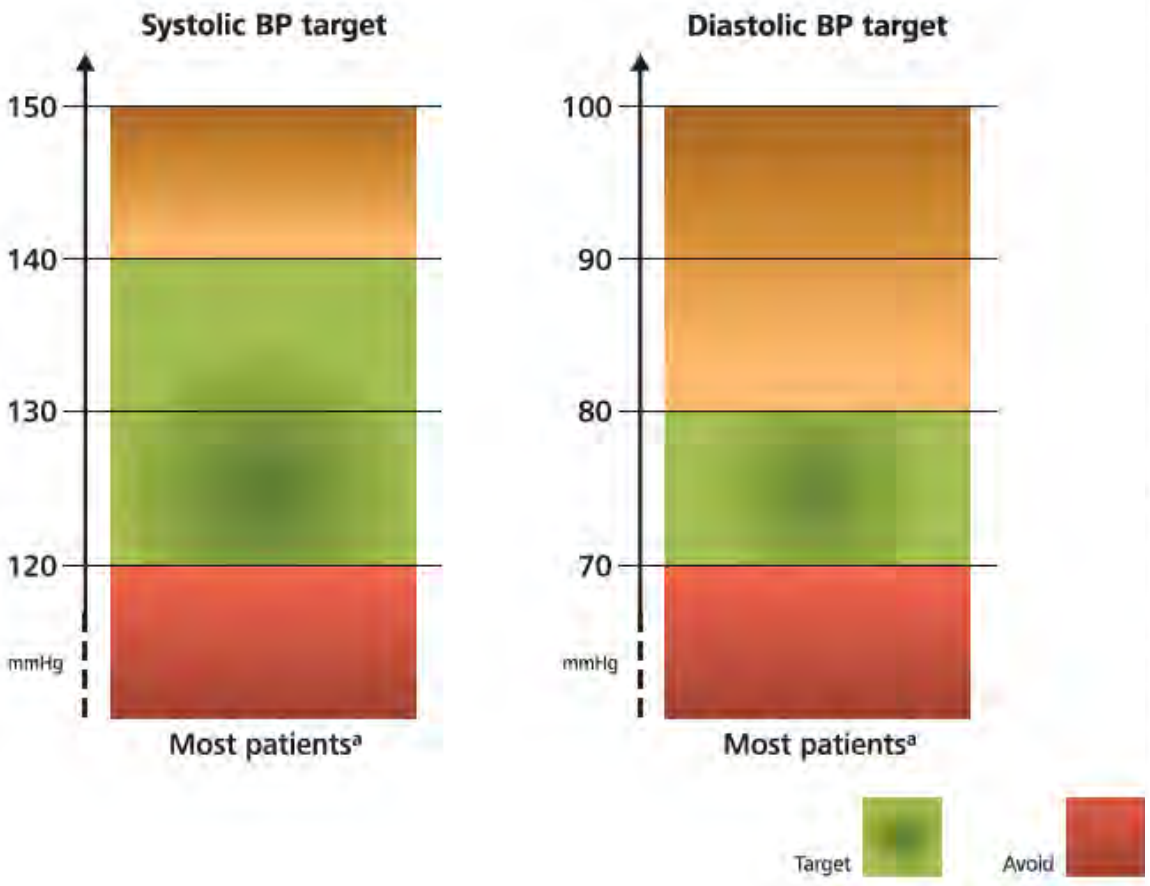
Treatments(2) Initiation



BP thresholds for drug treatment initiation

Recommendations and statements	CoR	LoE
In patients 18 to 79 years, the recommended office threshold for initiation of drug treatment is 140 mmHg for SBP and/or 90 mmHg for DBP.	I	A
In patients ≥80 years, the recommended office SBP threshold for initiation of drug treatment is 160 mmHg.	I	B
However, in patients ≥80 years a lower SBP threshold in the range 140 – 159 mmHg may be considered.	II	C
The office SBP and DBP thresholds for initiation of drug treatment in frail patients should be individualized.	I	C
In adult patients with a history of CVD, predominantly CAD, drug treatment should be initiated in the high-normal BP range (SBP ≥130 or DBP ≥80 mmHg).	I	A

Target…

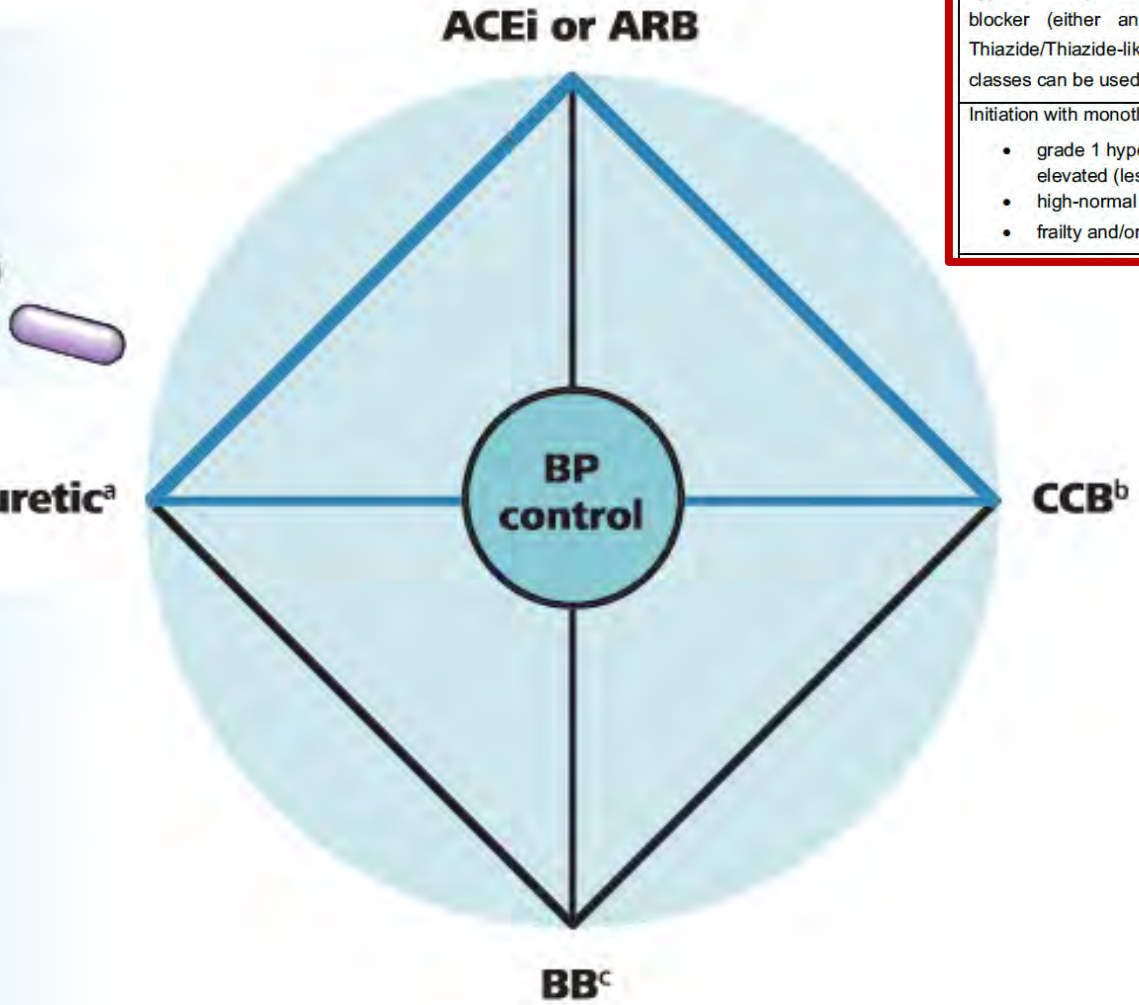


Recommendations and statements	CoR	LoE
Patients 18 to 64 years old		
The goal is to lower office BP to <130/80mmHg.	I	A
Patients 65 to 79 years old		
The primary goal of treatment is to lower BP to <140/80mmHg.	I	A
However, lowering BP to below 130/80mmHg can be considered if treatment is well tolerated.	II	B
Patients 65 to 79 years old with ISH		
The primary goal of treatment is to lower SBP in the 140 to 150 mmHg range.	I	A
However, a reduction of office SBP in the 130 to 139 mmHg range should be considered if well tolerated, albeit cautiously if DBP is already below 70 mmHg.	I	B
Patients ≥80 years old		
Office SBP should be lowered to a SBP in the 140 to 150 mmHg range.	I	A
However, reduction of office SBP between 130 to 139 mmHg may be considered if well tolerated, albeit cautiously if DBP is already below 70 mmHg.	II	B
Additional safety recommendations		
In frail patients, the treatment target for office SBP and DBP should be individualized.	I	C
Do not aim to target office SBP below 120 mmHg or DBP below 70 mmHg during drug treatment.	III	C
However, in patients with low office DBP, i.e. below 70 mmHg, SBP should be still lowered, albeit cautiously, if on-treatment SBP is still well above target values.	II	C
Reduction of treatment can be consider in patient aged 80 years or older with a low SBP (< 120 mmHg) or in the presence of severe orthostatic hypotension or a high frailty level.	II	C

Drugs

Prescribing patterns:

- Start with dual combination therapy in most patients
- Uptitrate to maximum well tolerated doses and to triple therapy if needed
- **Once daily (preferred in the morning)**
- **Add further drugs if needed**
- **Preferred use of SPCs at any step**



Additional drug classes

General antihypertensive therapy:

- Steroidal MRA
- Loop Diuretic
- Alpha-1 Blocker
- Centrally acting agent
- Vasodilator

Special comorbidities:

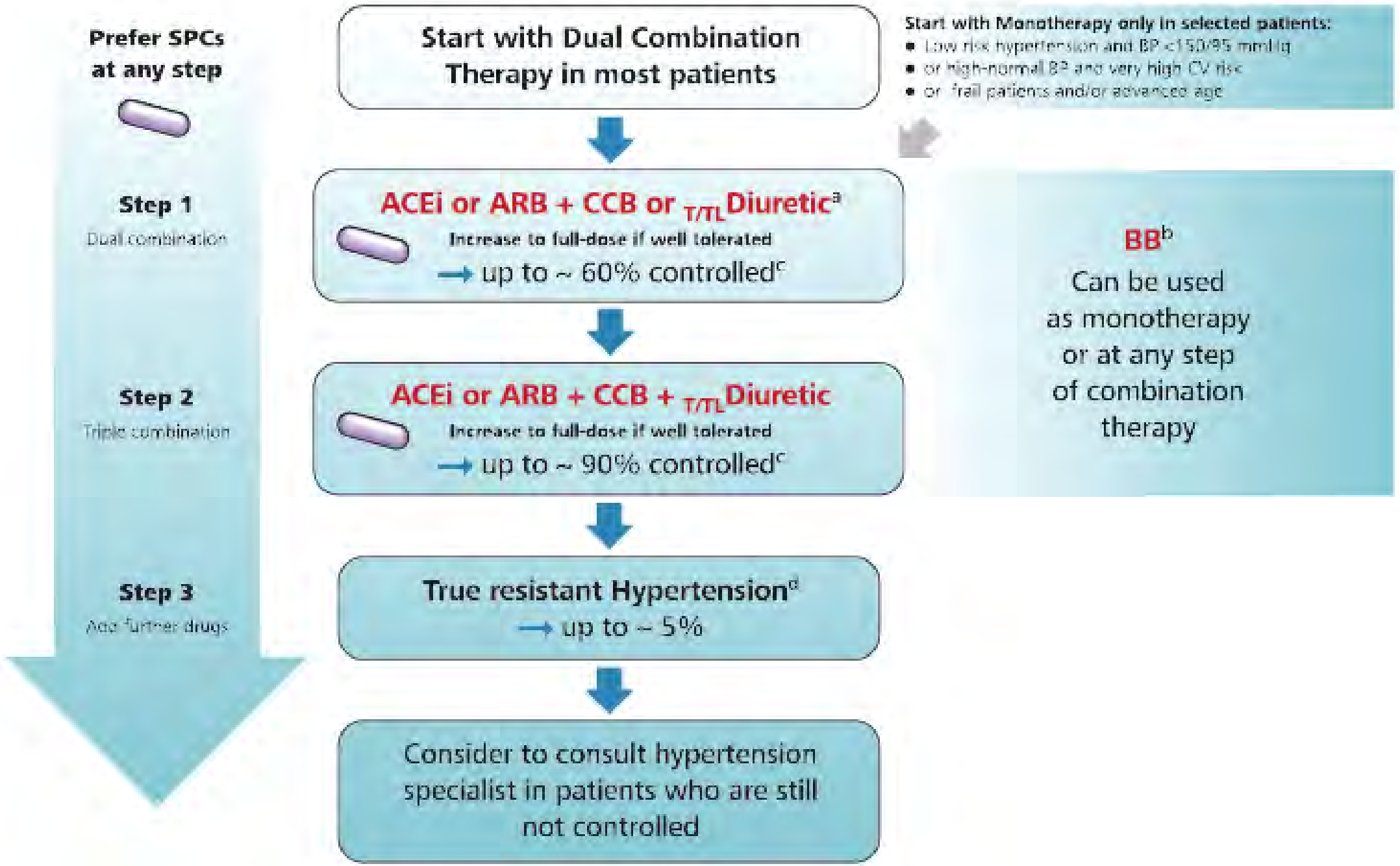
- ARNi
- SGLT2i
- Non-Steroidal MRA

Recommendations and statements	CoR	LoE
BP lowering should be prioritized over the selection of specific antihypertensive drug classes because treatment benefit largely originates from BP reduction.	I	A
Five major drug classes including ACEis, ARBs, BBs, CCBs, and Thiazide/Thiazide-like diuretics have effectively reduced BP and CV events in RCTs. These drugs and their combinations are recommended as the basis of antihypertensive treatment strategies.	I	A
Initiation of therapy with a two-drug combination is recommended for most hypertensive patients. Preferred combinations should comprise a RAS blocker (either an ACE inhibitor or an ARB) with a CCB or Thiazide/Thiazide-like diuretic. Other combinations of the five major drug classes can be used.	I	A
Initiation with monotherapy should be considered in patients with: • grade 1 hypertension and low-risk if BP is only marginally elevated (less than 150 mmHg SBP and 95 mmHg DBP) • high-normal BP and very high CV risk, • frailty and/or and advance age.	I	C

TABLE 16. Selected diseases and conditions for the use of BBs in patients with hypertension [591]

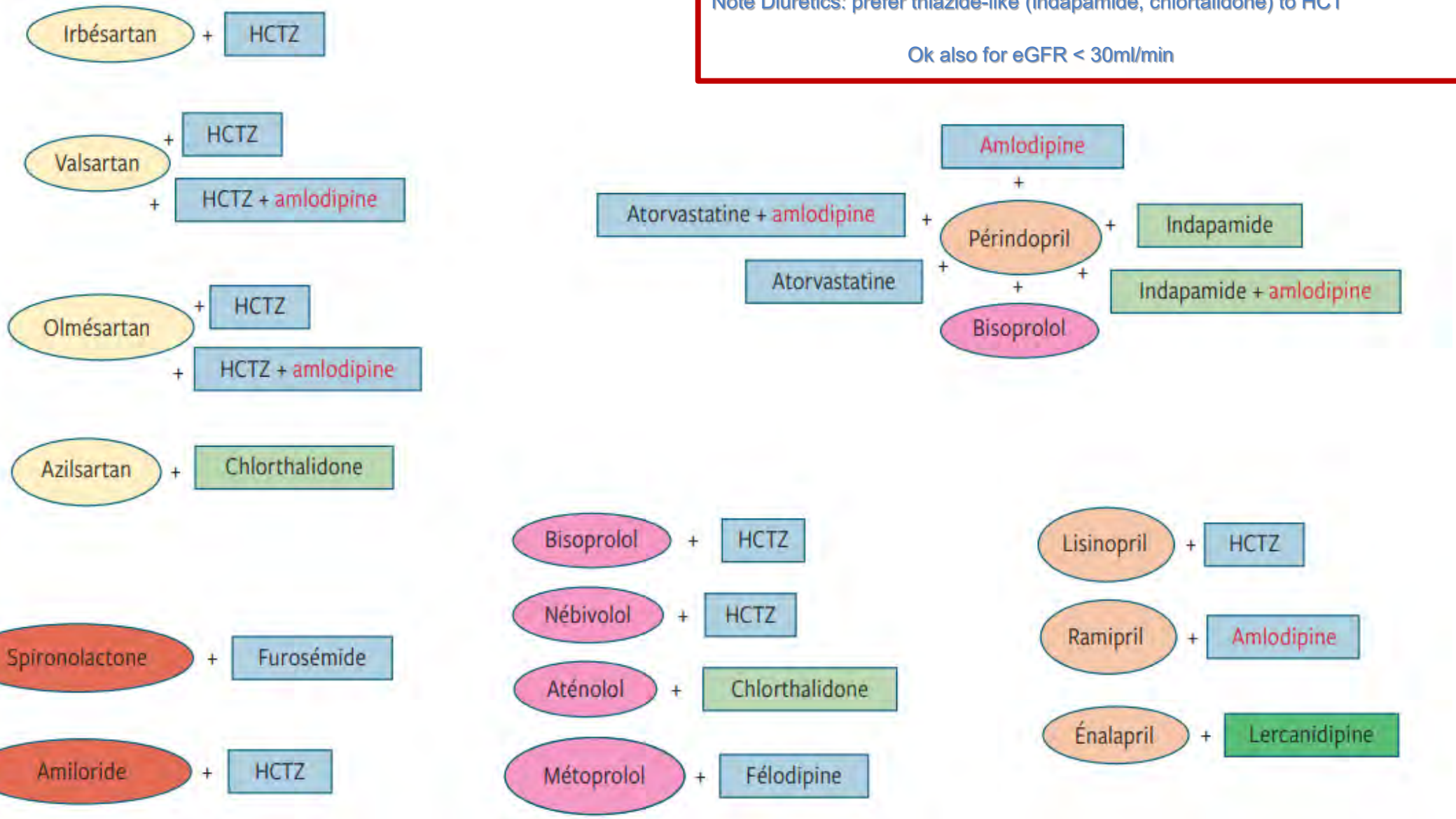
Selected indications with guideline directed medical therapy for BBs
Chronic coronary syndromes, antiischemic therapy
Postmyocardial infarction: arrhythmias, angina, known incomplete re-vascularization, HF
Acute coronary syndrome
HFrEF and HFpEF if coronary disease (ischemia), arrhythmias and tachycardia
Atrial fibrillation: prevention, rhythm control, heart rate control
Women with child-bearing potential/planning pregnancy
Hypertension disorders in pregnancy
Selected other conditions in which therapy with BBs can be favourable
Hypertension with elevated resting heart rate >80 bpm
Emergency, urgency and parenteral administration
Perioperative hypertension
Major noncardiac surgery
Excessive pressor response to exercise and stress
Hyperkinetic heart syndrome
Postural orthostatic tachycardia syndrome
Orthostatic hypertension
OSA
Peripheral arterial disease with claudication ?
COPD
Portal hypertension, cirrhosis-related esophageal varices and recurrent variceal bleeding
Glaucoma
Thyrotoxicosis, hyperthyroidism
Hyperparathyroidism in uremia ?
Migraine headache
Essential tremor
Performance anxiety and anxiety disorders
Psychiatric disorders (posttraumatic stress)

BB rehabilitation : really?


Prefer SPCs at any step
Step 1
Dual combination
Step 2
Triple combination
Step 3
Add further drugs
Start with Dual Combination Therapy in most patients
Start with Monotherapy only in selected patients:
• Low risk hypertension and BP <150/95 mmHg
• or high-normal BP and very high CV risk
• or frail patients and/or advanced age
ACEi or ARB + CCB or T/TL Diuretic[a]
Increase to full-dose if well tolerated
→ up to ~ 60% controlled[c]
ACEi or ARB + CCB + T/TL Diuretic
Increase to full-dose if well tolerated
→ up to ~ 90% controlled[c]
True resistant Hypertension[d]
→ up to ~ 5%
Consider to consult hypertension specialist in patients who are still not controlled
BB[b]
Can be used as monotherapy or at any step of combination therapy

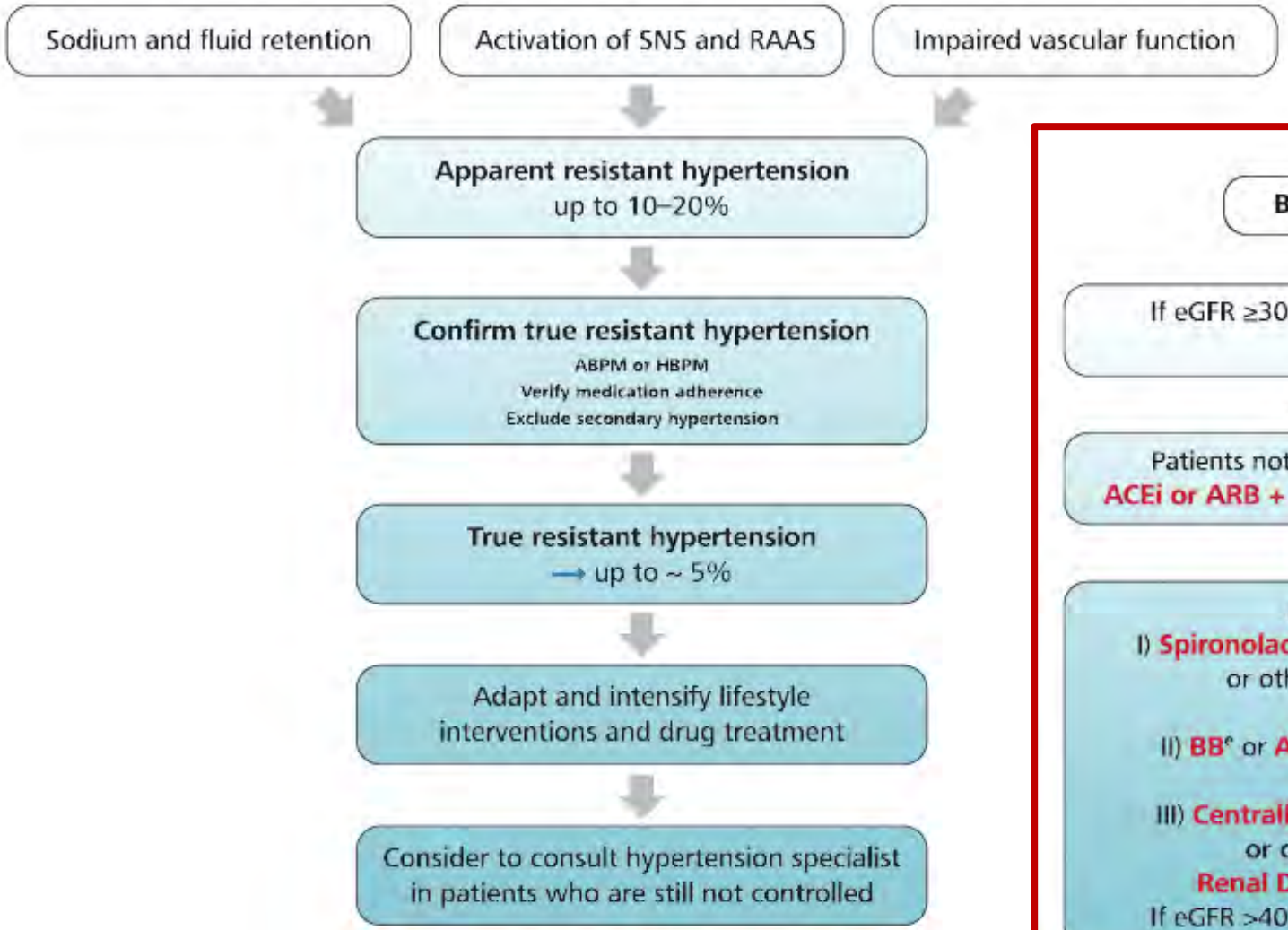
Note Diuretics: prefer thiazide-like (indapamide, chlortalidone) to HCT

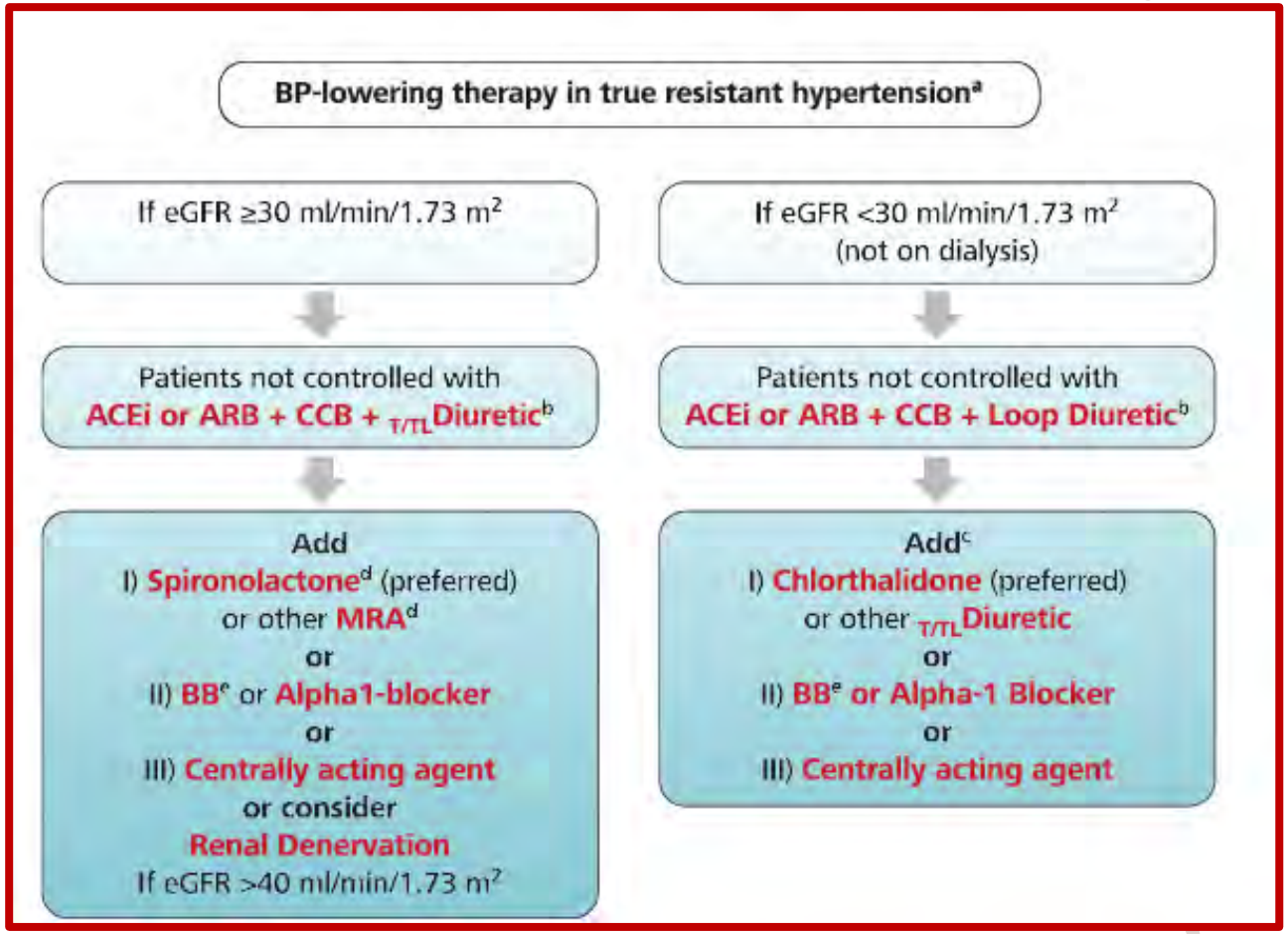
Ok also for eGFR < 30ml/min



Combinations e.g (Sandoz E. RMS 2023)

Resistant HT - Strategies





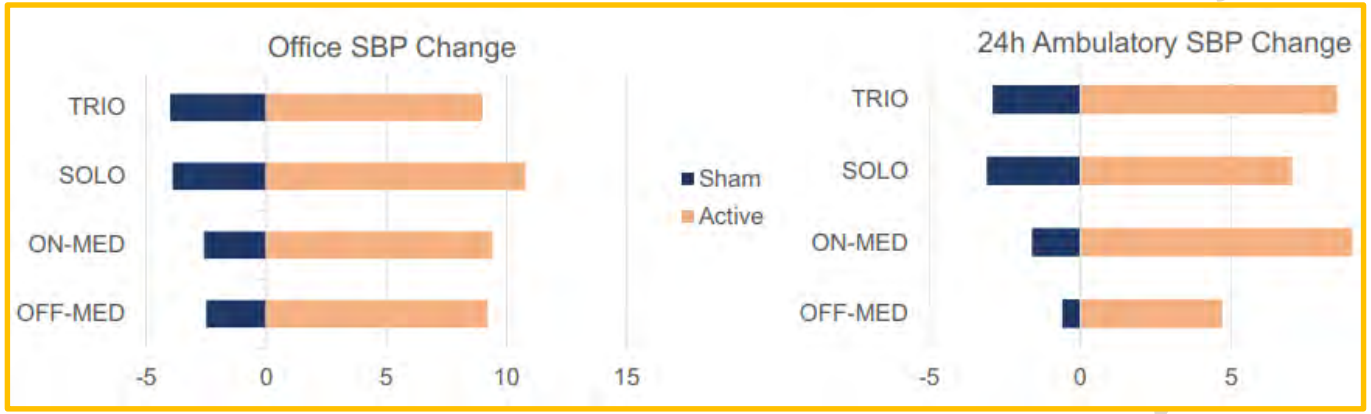
Renal Denervation

Recommendations and statements	CoR	LoE
RDN can be considered as a treatment option in patients with an eGFR >40 ml/min/1.73m^2 who have uncontrolled BP despite the use of antihypertensive drug combination therapy, or if drug treatment elicits serious side effects and poor quality of life.	II	B
RDN can be considered as an additional treatment option in patients with true resistant hypertension if eGFR is >40 ml/min/1.73m^2.	II	B
Selection of patients to whom RDN is offered should be done in a shared decision-making process after objective and complete patient's information.	I	C
RDN should only be performed in experienced specialized centers to guarantee appropriate selection of eligible patients and completeness of the denervation procedure.	I	C

Table 1. Completed Trials of Renal Denervation[24-27,41-44]

Study	Method	N	Active:Sham	Inclusion	Primary Outcome	Results
Trials excluding patients with reduced eGFR[a]						
SPYRAL HTN-OFF MED Pivotal[25]	RFA	331	1:1	Office BP 150-179/ ≥90 mm Hg on no BP meds	24-h ABPM SBP at 3 mo	RDN: ↓5 mm Hg; sham: ↓1 mm Hg
RADIANCE-HTN SOLO[24]	US	146	1:1	Office BP 140-180/ 90-110 mm Hg on no BP meds	Daytime ABPM SBP at 2 mo	RDN: ↓7 mm Hg; sham: ↓2 mm Hg
SPYRAL HTN-ON MED Pilot[27]	RFA	80	1:1	Office BP 150-179/ ≥90 mm Hg on 1-3 stable BP meds	24-h ABPM SBP at 6 mo	RDN: ↓9 mm Hg; sham: ↓2 mm Hg
RADIANCE-HTN TRIO[26]	US	136	1:1	Office BP ≥140/ ≥90 mm Hg on 3 stable BP meds	Daytime ABPM SBP at 2 mo	RDN: ↓8 mm Hg; sham: ↓3 mm Hg
Studies in patients with CKD						
Ott et al[41]	RFA	27	No sham	CKD 3-4 with resistant HTN (ESH/ESC definition)	Nephroprotection	eGFR slope improved at 1 y post intervention
Kiuchi and Chen[42]	RFA	108	No sham	CKD with or without controlled HTN	Nephroprotection	CKD with uncontrolled HTN had better eGFR outcome vs CKD with controlled HTN at 6 mo
Hering et al[43]	RFA	46	No sham	eGFR ≤60 mL/min/ 1.73 m^2	Nephroprotection	eGFR stabilized over 12-24 mo follow-up
Ott et al[44]	RFA	6	No sham	HD and 24 h ABPM ≥135/85 mm Hg on 3 meds	Δ24-h ABPM	24-h ABPM: ↓20/15 mm Hg at 6 mo

Abbreviations: ABPM, ambulatory blood pressure measurement; CKD, chronic kidney disease; eGFR, estimated glomerular filtration rate; ESH/ESC, European Society of Hypertension/European Society of Cardiology; HD, hemodialysis; HTN, hypertension; RDN, renal denervation; RFA, radiofrequency ablation; SBP, systolic blood pressure; US, ultrasound.
[a]Defined variously as eGFR <40-45 mL/min/1.73 m^2.



Towsend. AJKD review 2022

«New» antihypertensive drugs

Drug class	Drug(s)/compound name(s)	Target site(s); route	Stage*
RAAS			
1. Angiotensin (1-7) analogues	AVE0991, HP-β-CD/Ang1-7	Mas receptor; oral	Preclinical
2 Angiotensin II vaccines	CYT006-AngQB, AGMG0201	Circulation; SC, IM	Phase IIa
3 Aldosterone synthase inhibitors	LCI699, LY3045697, RO6836191	Adrenal cortex; oral	Phase II
Other enzymes/receptors			
1. Dopamine β-hydroxylase inhibitors	Etamicastat, zamicastat	Adrenal medulla; oral	Phase I
2 Neprilysin inhibitors	LHW090	Kidney; oral	Phase II
3 Aminopeptidase A inhibitors	Firibastat, NI956	Brain; oral	Phase III
4 Endothelin receptor antagonists	Aprocitentan, atrasentan	Blood vessels; oral	Phase III
5 Angiotensin receptor-neprilysin inhibitors	Sacubitril/valsartan	AT2R, kidney; oral	Phase IV
6 SGLT2 inhibitor	Gliflozins	Kidney; oral	Phase IV

Ordered by stage of development. AT2R indicates angiotensin II type 2 receptor; RAAS, renin–angiotensin–aldosterone system; and SGLT2, sodium glucose co-transporter 2.

* Highest stage of development for compound(s) in the class.

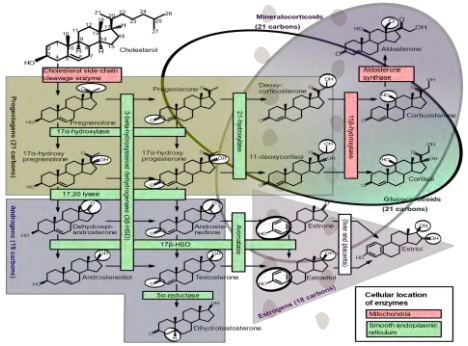
Journal of the American Heart Association
Volume 11, Issue 20, 18 October 2022
https://doi.org/10.1161/JAHA.122.027694

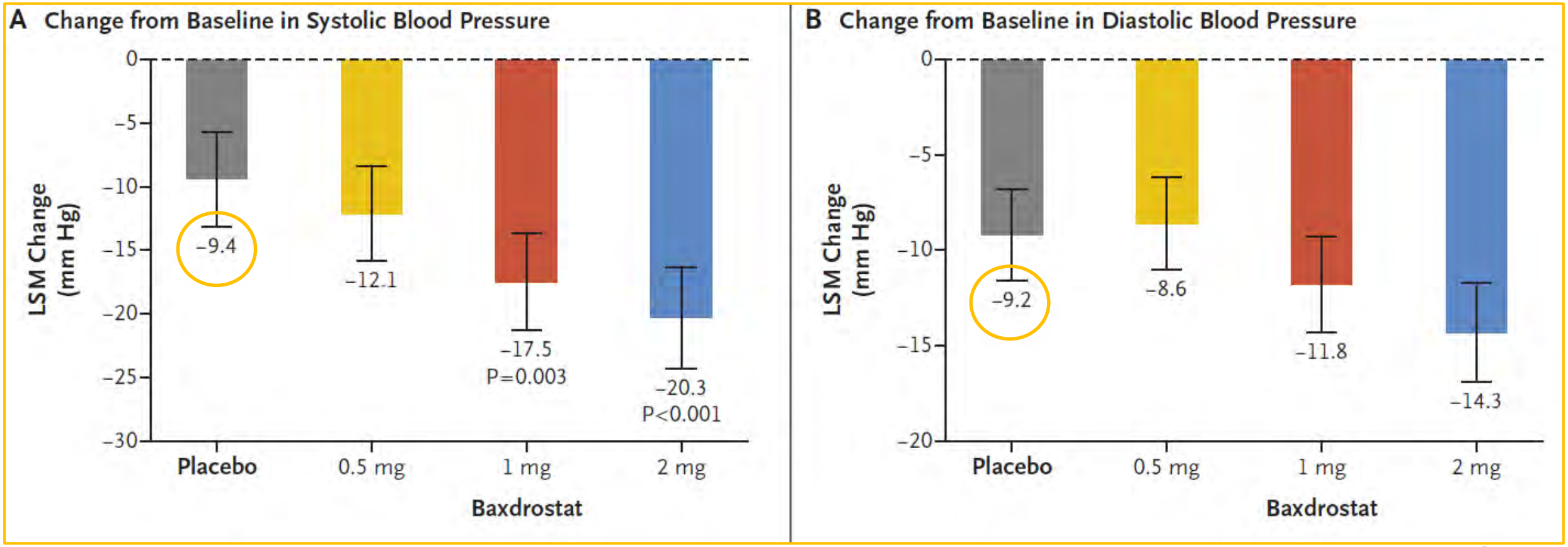
The NEW ENGLAND JOURNAL of MEDICINE

ESTABLISHED IN 1812 FEBRUARY 2, 2023 VOL. 388 NO. 5

Phase 2 Trial of Baxdrostat for Treatment-Resistant Hypertension

Aldosterone synthasis inhibition





THE NEW ENGLAND JOURNAL of MEDICINE

RESEARCH SUMMARY

Zilebesiran, an RNA Interference Therapeutic Agent for Hypertension

Desai AS et al. DOI: 10.1056/NEJMoa2208391

CLINICAL PROBLEM

Nearly half of patients with hypertension do not reach guideline-recommended blood-pressure targets. Zilebesiran is an investigational RNA interference therapeutic agent that inhibits the production of angiotensinogen, the precursor of angiotensin, which plays a key role in the pathogenesis of hypertension.

CLINICAL TRIAL

Design: A four-part, multicenter, phase 1 study assessed the safety and blood-pressure–lowering effects of zilebesiran in adults ≤65 years of age with treated or untreated hypertension.

Intervention: 107 patients were enrolled. In Part A, patients were randomly assigned to a single subcutaneous dose of zilebesiran (at one of seven doses ranging from 10 to 800 mg) or placebo. In Part B, zilebesiran (800 mg) or placebo was administered under low- and high-salt dietary conditions, and in Part E, irbesartan was added to zilebesiran (800 mg). (Part C was removed during a protocol amendment, and Part D is ongoing.) The primary end point was the frequency of adverse events.

RESULTS

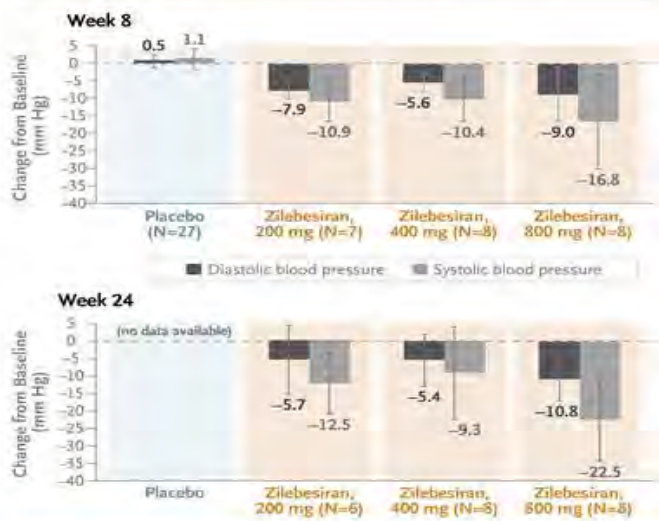
Safety: Overall, adverse events were not more frequent with zilebesiran than with placebo. Five zilebesiran recipients had mild, transient injection-site reactions. No patient received interventions for hypotension, hyperkalemia, or worsening of renal function.

Efficacy: In Part A, single doses of zilebesiran of ≥200 mg were associated with dose-dependent decreases in blood pressure that were apparent by week 8 and were sustained for up to 24 weeks. In Part B, a high-salt diet appeared to attenuate the blood-pressure–lowering effects of zilebesiran. In Part E, irbesartan appeared to enhance the effects of zilebesiran.

LIMITATIONS AND REMAINING QUESTIONS

- The efficacy end points were exploratory.
- The study was too small and short to fully assess safety.
- Whether zilebesiran has the teratogenic effects of other renin–angiotensin system inhibitors is unknown.

Links: Full Article | NEJM Quick Take | Science behind the Study



CONCLUSIONS

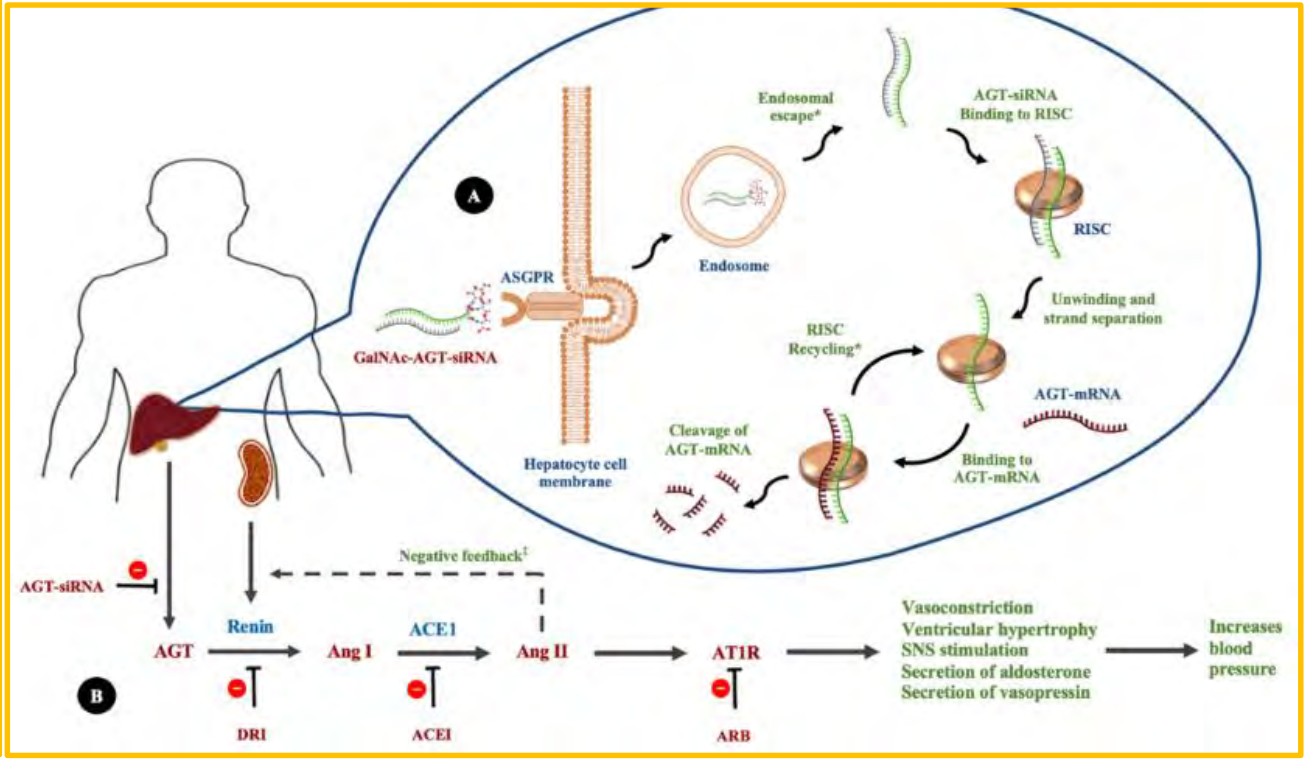
In patients with hypertension, the investigational RNA interference therapeutic agent zilebesiran was associated with mild injection-site reactions and led to dose-dependent decreases in blood pressure that were sustained at 24 weeks of follow-up.

Journal of the American Heart Association
Volume 11, Issue 20, 18 October 2022
https://doi.org/10.1161/JAHA.122.027694

VIEWPOINTS

Small Interfering RNA Therapeutics in Hypertension: A Viewpoint on Vasopressor and Vasopressor-Sparing Strategies for Counteracting Blood Pressure Lowering by Angiotensinogen–Targeting Small Interfering RNA

Priyanga Ranasinghe, MD, PhD ; Melisande L. Addison, MBBS, PhD ; David J. Webb, MD, DSc



Different Populations

Older patients (>80 ans): strategies…

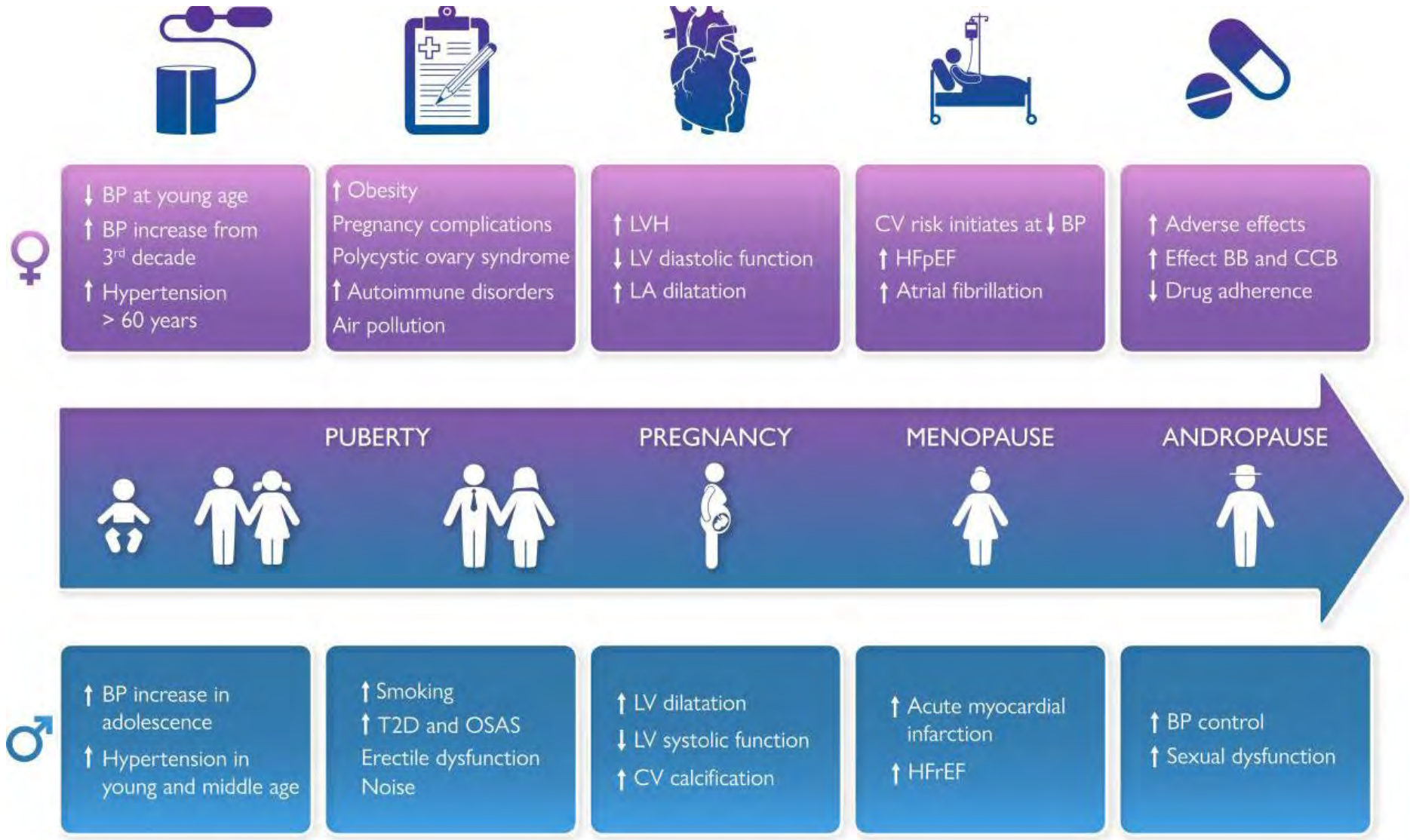
TABLE 21. Adapting BP-lowering strategies in patients older than 80 years according to their functional/autonomy status (adapted from [38])

Characteristics	Group 1 Fit	Group 2 Slowed but autonomous for most activities	Group 3 Severely dependent
Diagnosis	-ADL (Katz) ≥5/6 and -absence of clinically significant dementia (MMSE≤20/30) and -routine walking activities	-Profile between Groups 1 and 3	-ADL (Katz) ≤2/6 or -severe dementia (MMSE ≤10/30) or chronic bedridden or -end of life
Therapeutic strategy	- Initiate drug treatment if office SBP ≥160 mmHg - However, in most cases initiation of treatment in the 140 to 159 mmHg range maybe considered - Office SBP should be lowered in the 140 to 150 mmHg range - However, reduction of office SBP between 130 to 139 mmHg may be considered if well tolerated, albeit cautiously if DBP is already below 70 mmHg - Consider to start with monotherapy	- Initiate drug treatment if office SBP ≥160mmHg - However, a lower office SBP threshold in the 140 to 159 mmHg range may be considered - Office SBP should be lowered in the 140 to 150mmHg range - However, reduction of office SBP between 130 to 139mmHg may be considered if well tolerated, albeit cautiously if office DBP is already below 70 mmHg - Consider to start with monotherapy and titrate antihypertensive medication cautiously - Consider treatment reduction if office SBP is very low (<120 mmHg) or in patients with orthostatic hypotension - Make a more detailed evaluation of the functional status: SPPB (mobility), handgrip (muscular force), mini-GDS scale (depression), and MNA-SF (nutritional status)	- Priorize therapeutic strategies according to comorbidities and polypharmacy issues - Consider treatment if office SBP ≥160 mmHg - Target treatment of office SBP to a range of 140–150mmHg - Reduce treatment if office SBP is very low (<120 mmHg) or in patients with orthostatic hypotension - Correct other factors and medications decreasing BP

Recommendations and statements	CoR	LoE
Patients 65 to 79 years old		
The recommended office threshold for initiation of drug treatment is 140/90 mmHg.	I	A
The primary goal of treatment is to lower BP to <140/80mmHg.	I	A
However, lowering BP to below 130/80mmHg can be considered if treatment is well tolerated.	I	B
Patients 65 to 79 years old with ISH		
The primary goal of treatment is to lower SBP in the 140 to 150 mmHg range.	I	A
However, a reduction of office SBP in the 130 to 139 mmHg range should be considered if well tolerated, albeit cautiously if DBP is already below 70 mmHg.	I	B
In dedicated RCTs in older patients with ISH, CCBs and Thiazide/Thiazide-like diuretics have been mainly used. However, all other major drug classes can be used, because of the frequent co-existence of compelling indications and the need of combination therapy to control SBP.	I	A
Initiation of treatment with a two-drug combination is also recommended in most older patients with ISH, who are not frail.	I	C
Patients ≥80 years old		
The recommended office SBP threshold for initiation of drug treatment is 160 mmHg.	I	B
However, a lower SBP threshold in the 140 to 159 mmHg range may be considered.	II	C
Office SBP should be lowered in the 140 to 150 mmHg range.	I	A
However, reduction of office SBP between 130 to 139 mmHg may be considered if well tolerated, albeit cautiously if DBP is already below 70 mmHg.	II	B

Additional recommendations[a]		
In frail patients, initiation of drug treatment and the treatment target for office SBP and DBP should be individualized.	I	C
Initiation with monotherapy should be considered in patients with frailty and/or advanced age.	I	C
Do not aim to target office SBP below 120 mmHg or DBP below 70 mmHg during drug treatment.	III	C
However, in patients with low office DBP, i.e. below 70 mmHg, SBP should be still lowered, albeit cautiously, if on-treatment SBP is still well above target values	II	C
Reduction of treatment can be considered in patients age 80 years or older with a low SBP (<120mmHg) or in the presence of severe orthostatic hypotension or a high frailty level.	II	C
Withdrawal of BP-lowering drug treatment on the basis of age, even when patients attain an age of ≥ 80 years, is not recommended, if treatment is well tolerated.	III	B
In older patients, treatment may start with lower doses and uptitration should be slower.	II	C
The search for orthostatic hypotension in old patients should be systematic, even in the absence of symptoms. Back titration or discontinuation of BP lowering drugs should be considered in patients with orthostatic hypotension.	I	C
In old patients with hypertension there should always be an assessment of functional/autonomy status including cognitive function.	I	C
In patients with reduced functional/autonomy status and/or dementia treatment should be individualized.	I	C

Gender and Hypertension



Women

- Pregnancy: 10% HTA

TABLE 22. Classification of hypertensive disorders in pregnancy

A. Preexisting (chronic) hypertension
Hypertension either preceding pregnancy or developing before 20 weeks gestation, usually persisting for more than 42 days postpartum, and may be associated with proteinuria.
1. Primary hypertension
2. Secondary hypertension
3. White-coat hypertension
4. Masked hypertension

B. Gestational hypertension
Hypertension develops after 20 weeks gestation and usually resolves within 42 days postpartum.
Transient gestational hypertension
- Usually detected in the clinic but then settles with repeated BP measurements taken over several hours, it is associated with a 40% risk of developing true gestational hypertension or preeclampsia in the remainder of the pregnancy, thus requiring careful follow-up.

Preeclampsia is gestational hypertension accompanied by one or more of the following new-onset conditions at or after 20 weeks gestation:
- Proteinuria (urinary albumin excretion in a 24h urine sample >0.3g/day or UACR in a random spot urine sample >30mg/mmol (0.3 mg/mg)
- Other maternal organ dysfunction
- Acute kidney injury (serum creatinine ≥90 μmol/l; 1 mg/dL)
- Liver involvement (elevated ALT or AST >40 IU/l; 0.67 >μkat/l with or without right upper quadrant or epigastric abdominal pain)
- Neurological complications (e.g. edampsia, altered mental status, blindness, stroke, clonus, severe headaches, persistent visual scotomata)
- Hematological complications (platelet count <150000/μl, DIC, hemolysis)
- Uteroplacental dysfunction (fetal growth restriction, abnormal umbilical artery Doppler waveform analysis, or stillbirth)

C. Preexisting hypertension + superimposed preeclampsia
Preexisting hypertension associated with any of the above maternal organ dysfunctions consistent with preedampsia or a further increase in BP with new-onset proteinuria

D. Antenatally unclassifiable hypertension
When BP is first recorded after 20 weeks gestation, and hypertension is diagnosed, reassessment is necessary at or after 42 days postpartum. If hypertension resolves, it should be reclassified as gestational hypertension, whereas if hypertension persists, it should be reclassified as preexisting hypertension.

Hypertension management in pregnancy

RESEARCH SUMMARY

Treatment for Mild Chronic Hypertension during Pregnancy

Tita AT et al. DOI: 10.1056/NEJMoa2201295

CLINICAL PROBLEM

Chronic hypertension during pregnancy increases risk of poor pregnancy and birth outcomes. Although pharmacologic antihypertensive therapy is standard treatment for severe hypertension during pregnancy, its benefits and safety are unclear for mild chronic hypertension in pregnant women.

CLINICAL TRIAL

Design: A U.S. multicenter, open-label, randomized, controlled trial assessed whether treatment of mild chronic hypertension in pregnant women, as compared with no treatment, would reduce adverse pregnancy outcomes without harming fetal growth.

Intervention: 2408 women with a known or new diagnosis of mild chronic hypertension and a singleton fetus at <23 weeks' gestation were randomly assigned to receive either active treatment with antihypertensive medications approved for pregnancy or standard treatment — i.e., no treatment, unless systolic blood pressure was ≥160 mm Hg or diastolic blood pressure was ≥105 mm Hg. The primary outcome was a composite of preeclampsia with severe features, medically indicated preterm birth at <35 weeks, placental abruption, fetal death, or neonatal death.

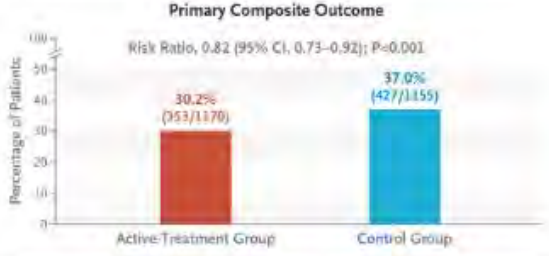
RESULTS

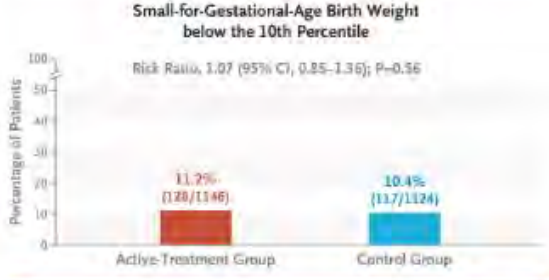
Efficacy: Active treatment of mild chronic hypertension reduced the frequency of primary outcome events.

Safety: The percentage of infants who were small for gestational age (<10th percentile) was similar in the active-treatment and control groups.

LIMITATIONS AND REMAINING QUESTIONS

- Patients were aware of their treatment group.
- There was a high ratio of women screened to women enrolled (12:1).
- The study was not powered to assess treatment effects across subgroups.





CONCLUSIONS

Treating mild chronic hypertension in pregnancy reduced adverse pregnancy outcomes without impairing fetal growth.

Recommendations and statements	CoR	LoE
In women with hypertensive disorders in pregnancy, initiation or intensification of drug treatment is recommended when SBP is ≥ 140 mmHg and/or DBP ≥ 90 mmHg.	I	C
In women with pre-existing hypertension (with or without superimposed pre-eclampsia), BP should be lowered to a target below 140/90 mmHg.	I	A
In women with gestational hypertension (with or without pre-eclampsia), BP should be lowered to a target below 140/90 mmHg.	I	C
In women with hypertensive disorders in pregnancy, too marked BP-lowering should be avoided. On-treatment DBP <80 mmHg is not recommended.	III	C
Labetalol[a] and α-methyl-DOPA are the first choice BP-lowering agents for hypertensive disorders in pregnancy unless contraindicated.	I	B
Extended-release nifedipine is recommended as an alternative BP-lowering agent during pregnancy.	I	B
Up-titration of monotherapy may precede any combination drug treatment.	II	C
Combination drug treatment between labetalol, extended-release nifedipine, or α-methyldopa may be reasonable to achieve the desirable BP target after the failure of up-titrated monotherapy.	II	C
ACE inhibitors, ARBs, or direct renin inhibitors are not recommended during pregnancy.	III	C
Aspirin (100-150 mg, at bedtime, weeks 11-35) should be administered in pregnant women at high or moderate risk of pre-eclampsia.	I	A
Severe hypertension (≥160/110 mmHg) in a pregnant woman requires prompt hospital admission.	I	C
In pre-eclampsia with severe features, magnesium sulfate should be administered immediately.	I	C

CONCLUSIONS

- HT frequent wordwide and high burden in health care costs
- Increasing mainly in countries of Low-Middle Income
- Persisting problem of:

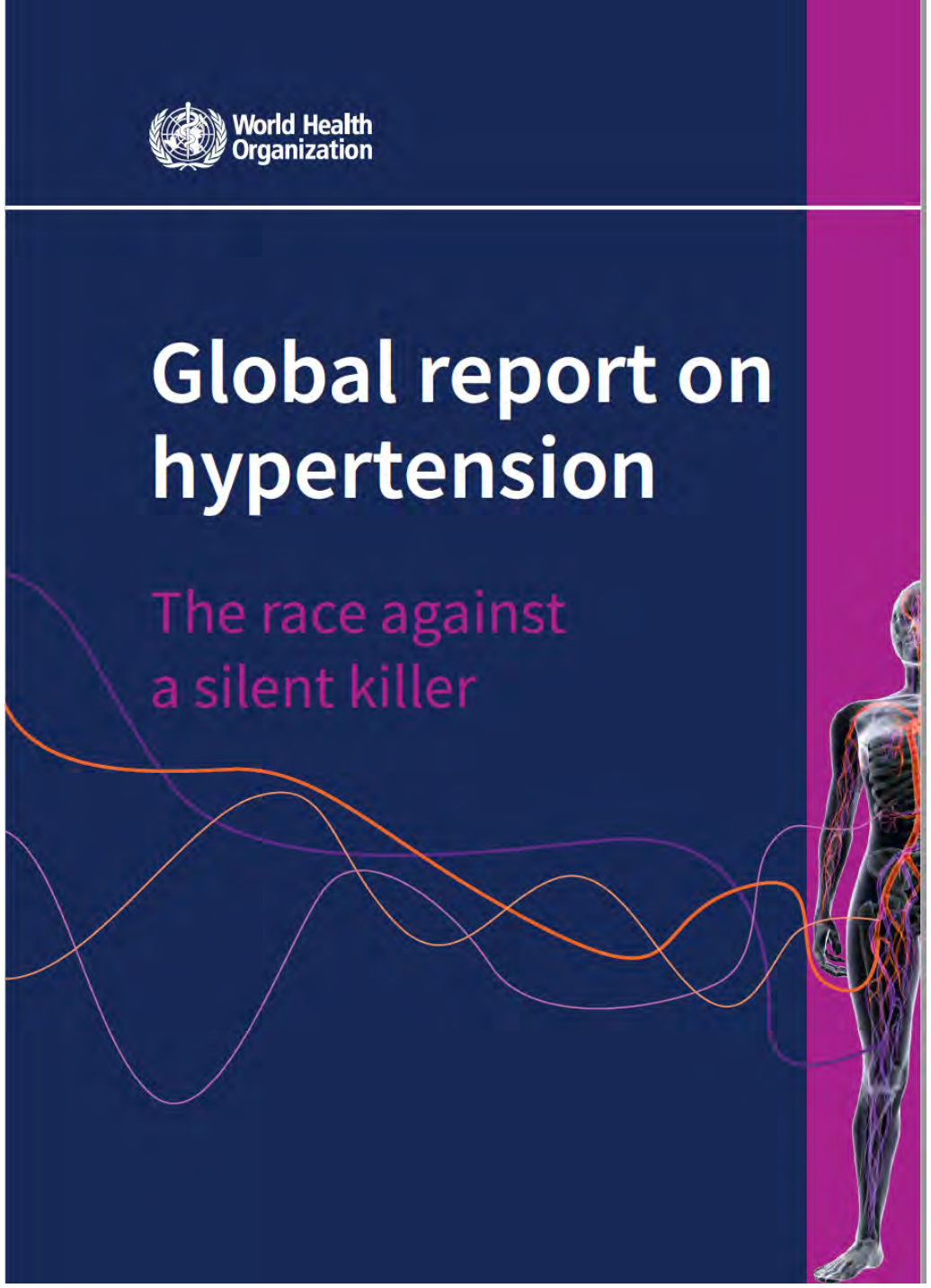
  1) underdiagnosed 2) undertreated 3) undercontrolled
- New guidelines provide evidences for diagnosis, targets, treatments
- Importance of combinations
- New treatments under the radar….
- Guidelines also considered specific populations: older, pregnancy,

Not only start the treatment

Diagnosis	Initiation Phase	Short-term Follow-Up	Long-term Follow-Up
• Patient/family history • Physical examination • Office BP measurement – Supplemented by ABPM/ HBPM if feasible • Basic/extended[a] lab tests • ECG • Assessment of CV risk and HMOD • Initation of: – Lifestyle interventions and – Drug therapy	• During the first 3 months repeated visits with BP measurements, including virtual visits • Verify lifestyle factors and adherence • Selected lab tests, ECG if necessary • Adjust drug treatment if necessary • Aim for BP control within 3 months	• In patients not difficult to control and with low-risk repeat visit after 1 year • Difficult to control patients or high risk patients repeat visit <1 year • Check-up program: – History including HBPM data, including lifestyle and adherence – Physical examination if necessary – Office BP measurement – Basic/extended[a] lab test – ECG – Re-evaluation of CV risk and HMOD – Adjust drug treatment if necessary	• Patients not difficult to control and with low-risk, annual follow-up with basic check-up program or extended check-up (e.g. HMOD re-evalution) every ≥3 years • Individualized and more frequent follow-up in patients with difficult to control BP or at high risk or with already treated secondary hypertension

Encourage use of Home BP monitoring and telehealth technologies to improve care

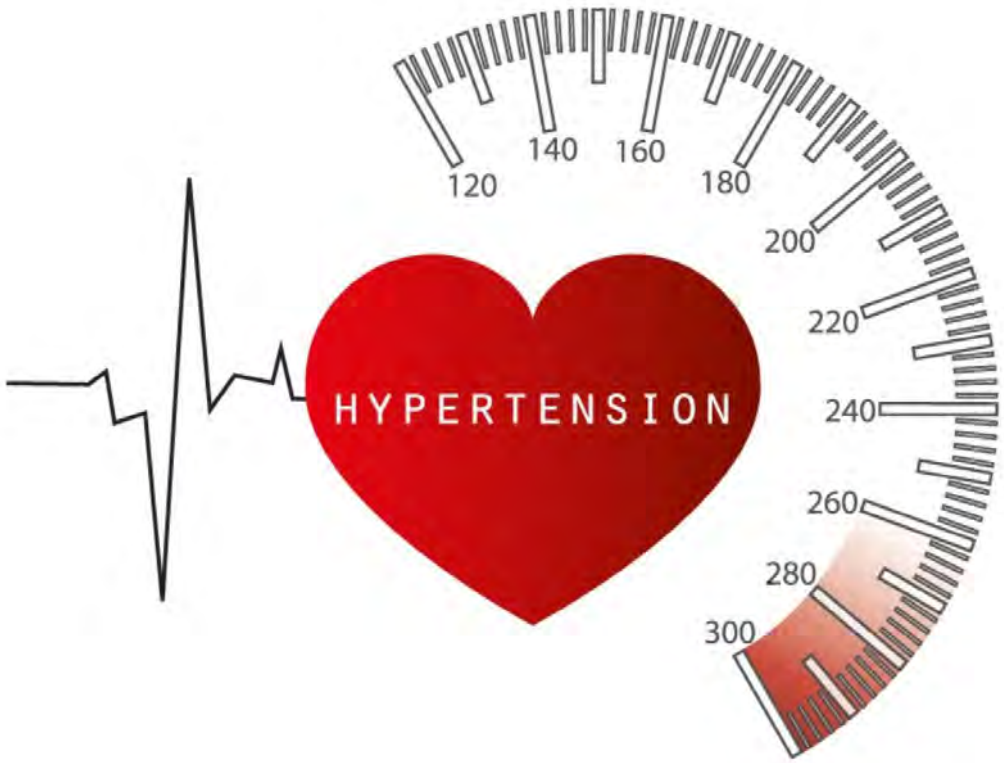
First 3 months	First year	After first year
Aim for optimal BP control	Maintain optimal BP control	Maintain optimal BP control



Targets…

1.	A one third relative reduction in the overall mortality from cardiovascular diseases, cancer, diabetes, or chronic respiratory diseases by 2030[a]
2.	A 20% relative reduction in the harmful use of alcohol by 2030[b]
3.	A 15% relative reduction in prevalence of insufficient physical activity by 2030[a]
4.	A 30% relative reduction in mean population intake of salt/sodium[c]
5.	A 30% relative reduction in prevalence of current tobacco use[c]
6.	A 25% relative reduction in the prevalence of raised blood pressure or to contain the prevalence of raised blood pressure[c]
7.	Halt the rise in diabetes and obesity[c]
8.	At least 50% of eligible people (aged 40 years and older with a 10-year cardiovascular risk ≥20%) including those with CVD to receive drug therapy and counselling (including glycaemic control) to prevent heart attacks and strokes[c]
9.	An 80% availability of the affordable basic technologies and essential medicines, including generics, required to treat major noncommunicable diseases in both public and private facilities[c]

THANK YOU



HYPERTENSION WORLDWIDE

Worldwide, 1 in 3 adults has high blood pressure—a condition that leads to heart attack and stroke.

Everyone can take **five concrete steps** to help prevent high blood pressure:

- Healthy diet
- Physical activity
- Avoiding tobacco
- Avoiding harmful use of alcohol
- Managing stress in healthy ways

CDC