Hypertension artérielle

ESC European Society of Cardiology

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2018 ESC/ESH Guidelines for the management of arterial hypertension

The Task Force for the management of arterial hypertension of the European Society of Cardiology (ESC) and the European Society of Hypertension (ESH)

Mesure de la pression artérielle

Table 8 Office blood pressure measurement

Patients should be seated comfortably in a quiet environment for 5 min before beginning BP measurements.
Three BP measurements should be recorded, 1–2 min apart, and additional measurements only if the first two readings differ by >10 mmHg. BP is recorded as the average of the last two BP readings.
Additional measurements may have to be performed in patients with unstable BP values due to arrhythmias, such as in patents with AF, in whom manual auscultatory methods should be used as most automated devices have not been validated for BP measurement in patients with AF.[a]
Use a standard bladder cuff (12–13 cm wide and 35 cm long) for most patients, but have larger and smaller cuffs available for larger (arm circumference >32 cm) and thinner arms, respectively.
The cuff should be positioned at the level of the heart, with the back and arm supported to avoid muscle contraction and isometric exercise-dependant increases in BP.
When using auscultatory methods, use phase I and V (sudden reduction/disappearance) Korotkoff sounds to identify SBP and DBP, respectively.
Measure BP in both arms at the first visit to detect possible between-arm differences. Use the arm with the higher value as the reference.
Measure BP 1 min and 3 min after standing from a seated position in all patients at the first measurement to exclude orthostatic hypotension. Lying and standing BP measurements should also be considered in subsequent visits in older people, people with diabetes, and people with other conditions in which orthostatic hypotension may frequently occur.
Record heart rate and use pulse palpation to exclude arrhythmia.

AF = atrial fibrillation; BP = blood pressure; DBP = diastolic blood pressure; SBP = systolic blood pressure.

[a]Most automatic devices are not validated for BP measurement in patients with AF and will record the highest individual systolic pressure wave form rather than an average of several cardiac cycles. This will lead to overestimation of BP.

Table 10 Comparison of ambulatory blood pressure monitoring and home blood pressure monitoring

ABPM	HBPM
Advantages • Can identify white-coat and masked hypertension • Stronger prognostic evidence • Night-time readings • Measurement in real-life settings • Additional prognostic BP phenotypes • Abundant information from a single measurement session, including short-term BP variability	**Advantages** • Can identify white-coat and masked hypertension • Cheap and widely available • Measurement in a home setting, which may be more relaxed than the doctor's office • Patient engagement in BP measurement • Easily repeated and used over longer periods to assess day-to-day BP variability
Disadvantages • Expensive and sometimes limited availability • Can be uncomfortable	**Disadvantages** • Only static BP is available • Potential for measurement error • No nocturnal readings[a]

ABPM = ambulatory blood pressure monitoring; BP = blood pressure; HBPM = home blood pressure monitoring.
[a]Techniques are being developed to enable nocturnal BP measurement with home BP devices.

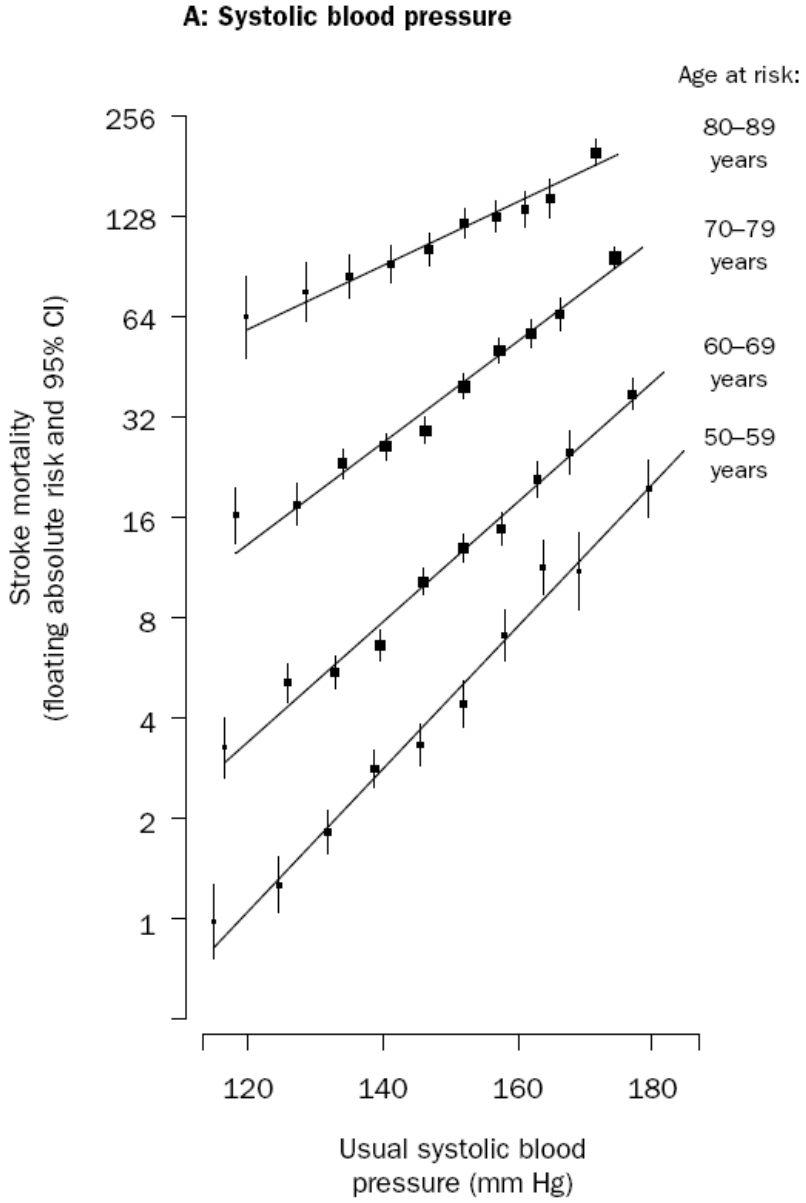
Définition de l’HTA

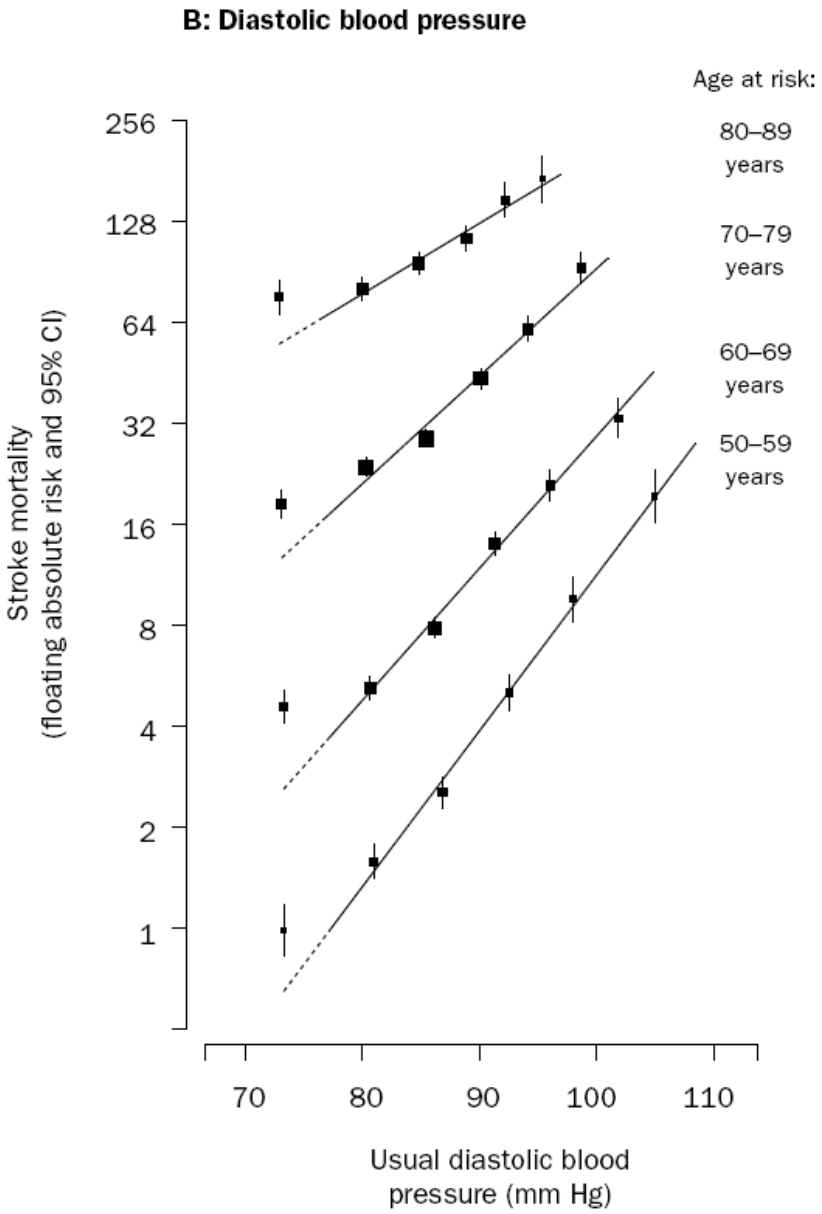
Articles

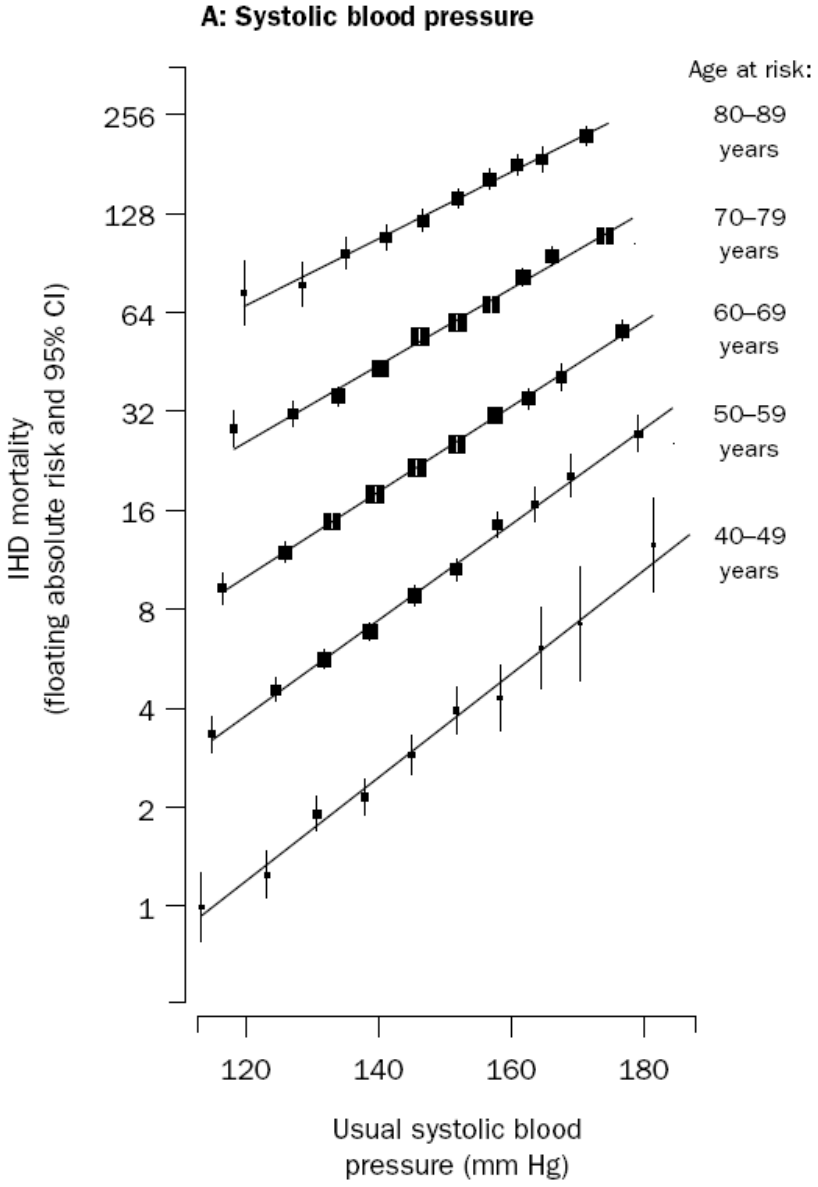
Age-specific relevance of usual blood pressure to vascular mortality: a meta-analysis of individual data for one million adults in 61 prospective studies

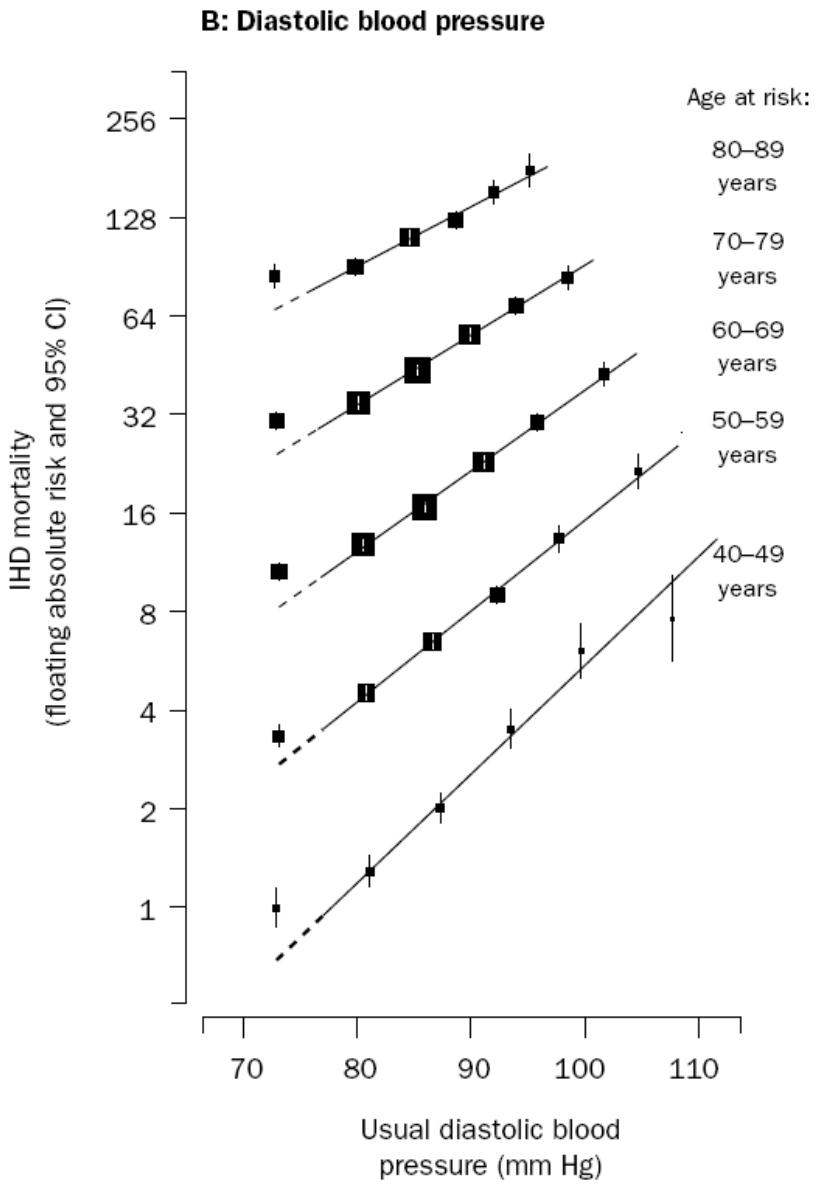
*Prospective Studies Collaboration**

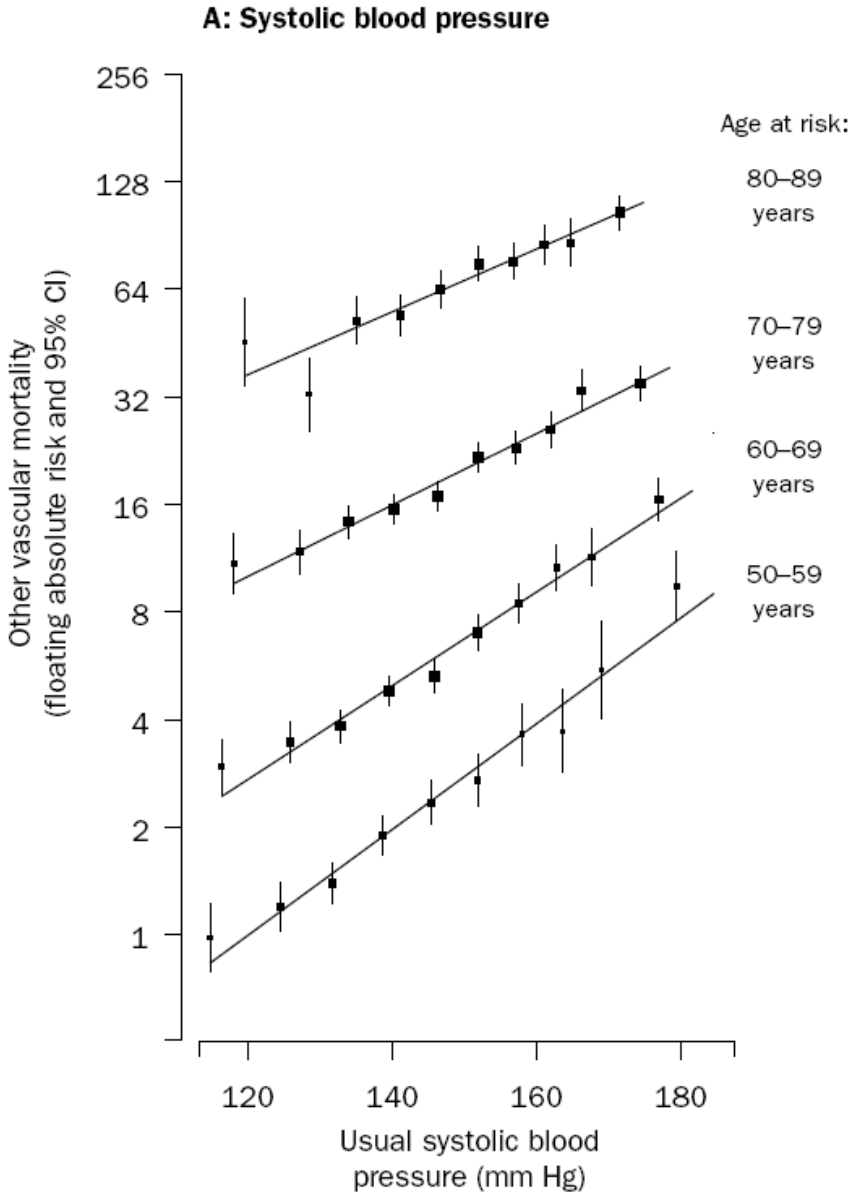
Lancet 2002; **360:** 1903–13

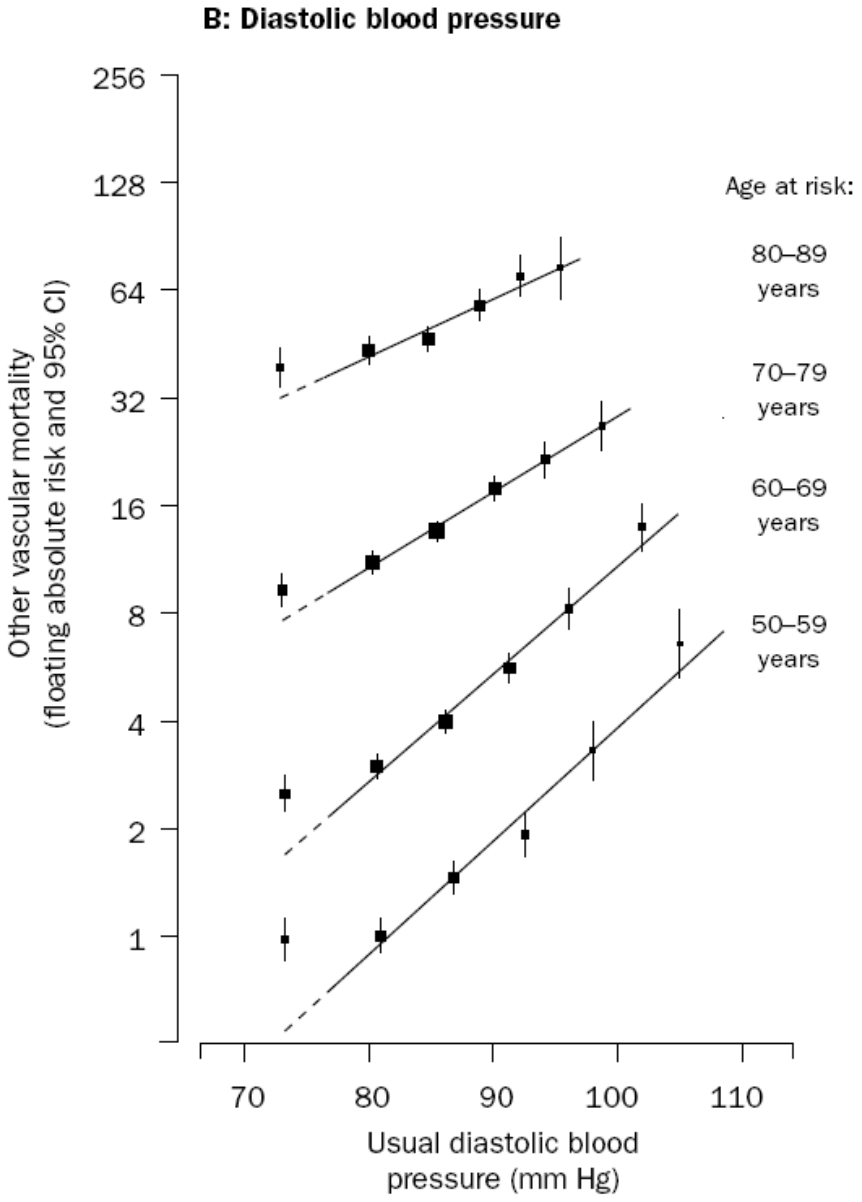

A: Systolic blood pressure
Age at risk:
80–89 years
70–79 years
60–69 years
50–59 years
Stroke mortality
(floating absolute risk and 95% CI)
256
128
64
32
16
8
4
2
1
120
140
160
180
Usual systolic blood pressure (mm Hg)


B: Diastolic blood pressure
Age at risk:
80–89 years
70–79 years
60–69 years
50–59 years
Stroke mortality (floating absolute risk and 95% CI)
256
128
64
32
16
8
4
2
1
70
80
90
100
110
Usual diastolic blood pressure (mm Hg)


A: Systolic blood pressure
Age at risk:
80–89 years
70–79 years
60–69 years
50–59 years
40–49 years
IHD mortality (floating absolute risk and 95% CI)
256
128
64
32
16
8
4
2
1
120
140
160
180
Usual systolic blood pressure (mm Hg)


B: Diastolic blood pressure
IHD mortality
(floating absolute risk and 95% CI)
256
128
64
32
16
8
4
2
1
70
80
90
100
110
Usual diastolic blood
pressure (mm Hg)
Age at risk:
80–89
years
70–79
years
60–69
years
50–59
years
40–49
years


A: Systolic blood pressure
256
128
64
32
16
8
4
2
1
Other vascular mortality
(floating absolute risk and 95% CI)
Age at risk:
80–89
years
70–79
years
60–69
years
50–59
years
120
140
160
180
Usual systolic blood
pressure (mm Hg)


B: Diastolic blood pressure
256
128
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8
4
2
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Other vascular mortality
(floating absolute risk and 95% CI)
Age at risk:
80–89
years
70–79
years
60–69
years
50–59
years
70
80
90
100
110
Usual diastolic blood
pressure (mm Hg)

Interpretation Throughout middle and old age, usual blood pressure is strongly and directly related to vascular (and overall) mortality, without any evidence of a threshold down to at least 115/75 mm Hg.

The NEW ENGLAND JOURNAL of MEDICINE

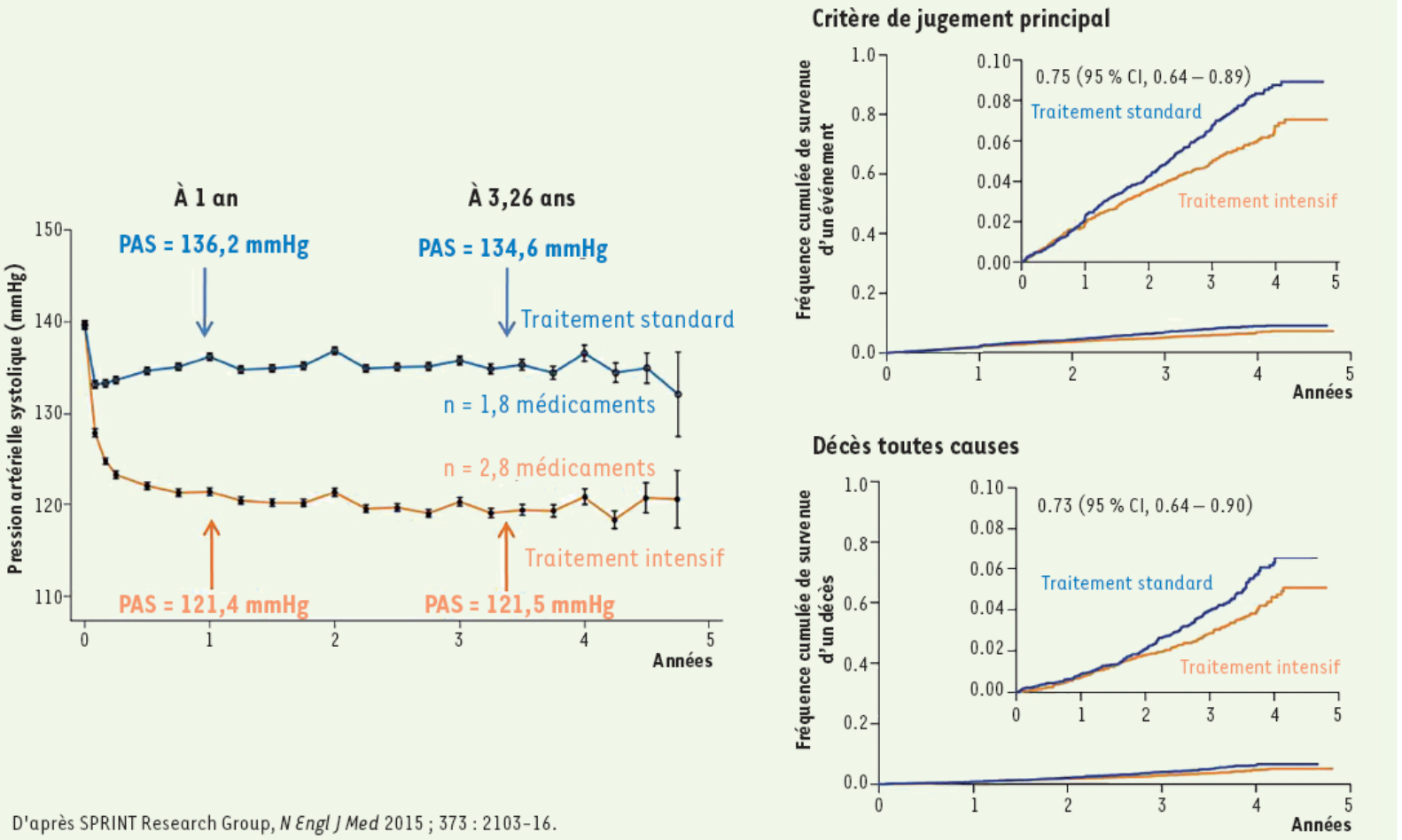
ESTABLISHED IN 1812 NOVEMBER 26, 2015 VOL. 373 NO. 22

A Randomized Trial of Intensive versus Standard Blood-Pressure Control

The SPRINT Research Group*

Essai SPRINT

Dans cet essai randomisé, ouvert, avec analyse en insu (*i.e.* en aveugle) de la randomisation, 9 361 patients hypertendus (PAS clinique ≥ 130 mmHg avec 0 à 4 médicaments antihypertenseurs) de plus de 50 ans, non diabétiques et sans antécédent d'AVC, mais à haut risque cardiovasculaire (maladie cardiovasculaire clinique ou infraclinique, ou insuffisance rénale chronique [DFG, débit de filtration glomérulaire ; MDRD, *modification of diet in renal disease* : 20-60 ml/min/1,73m²], ou un risque cardiovasculaire à 10 ans supérieur à 15 % selon le score de Framingham[3] ou un âge ≥ 75 ans) étaient randomisés soit dans le groupe « traitement standard » (PAS clinique cible < 140 mmHg) soit dans le groupe « traitement intensif » (PAS cible < 120 mmHg). Les



D'après SPRINT Research Group, *N Engl J Med* 2015 ; 373 : 2103-16.

Figure 1. ***Pressions artérielles systoliques (PAS) dans le groupe « traitement intensif » (objectif PAS < 120 mmHg) (en orange) et dans le groupe « traitement standard » (objectif PAS < 140 mmHg) (en bleu).*** Le critère de jugement principal est un critère composite associant la survenue d'un infarctus du myocarde, d'un syndrome coronarien aigu, d'un AVC (accident vasculaire cérébral), d'une insufffisance cardiaque ou d'un décès de cause cardiovasculaire. 95% CI : intervalle de confiance à 95%.

Table 2. Primary and Secondary Outcomes and Renal Outcomes.*

Outcome	Intensive Treatment		Standard Treatment		Hazard Ratio (95% CI)	P Value
	no. of patients (%)	*% per year*	*no. of patients (%)*	*% per year*		
All participants	**(N=4678)**		**(N=4683)**			
Primary outcome†	243 (5.2)	1.65	319 (6.8)	2.19	0.75 (0.64–0.89)	<0.001
Secondary outcomes						
Myocardial infarction	97 (2.1)	0.65	116 (2.5)	0.78	0.83 (0.64–1.09)	0.19
Acute coronary syndrome	40 (0.9)	0.27	40 (0.9)	0.27	1.00 (0.64–1.55)	0.99
Stroke	62 (1.3)	0.41	70 (1.5)	0.47	0.89 (0.63–1.25)	0.50
Heart failure	62 (1.3)	0.41	100 (2.1)	0.67	0.62 (0.45–0.84)	0.002
Death from cardiovascular causes	37 (0.8)	0.25	65 (1.4)	0.43	0.57 (0.38–0.85)	0.005
Death from any cause	155 (3.3)	1.03	210 (4.5)	1.40	0.73 (0.60–0.90)	0.003
Primary outcome or death	332 (7.1)	2.25	423 (9.0)	2.90	0.78 (0.67–0.90)	<0.001
Participants with CKD at baseline	**(N=1330)**		**(N=1316)**			
Composite renal outcome‡	14 (1.1)	0.33	15 (1.1)	0.36	0.89 (0.42–1.87)	0.76
≥50% reduction in estimated GFR§	10 (0.8)	0.23	11 (0.8)	0.26	0.87 (0.36–2.07)	0.75
Long-term dialysis	6 (0.5)	0.14	10 (0.8)	0.24	0.57 (0.19–1.54)	0.27
Kidney transplantation	0		0			
Incident albuminuria¶	49/526 (9.3)	3.02	59/500 (11.8)	3.90	0.72 (0.48–1.07)	0.11
Participants without CKD at baseline	**(N=3332)**		**(N=3345)**			
≥30% reduction in estimated GFR to <60 ml/min/1.73 m^2§	127 (3.8)	1.21	37 (1.1)	0.35	3.49 (2.44–5.10)	<0.001
Incident albuminuria¶	110/1769 (6.2)	2.00	135/1831 (7.4)	2.41	0.81 (0.63–1.04)	0.10

† The primary outcome was the first occurrence of myocardial infarction, acute coronary syndrome, stroke, heart failure, or death from cardiovascular causes.

Table 3 Classification of office blood pressure[a] and definitions of hypertension grade[b]

Category	Systolic (mmHg)		Diastolic (mmHg)
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[b]	≥140	and	<90

BP = blood pressure; SBP = systolic blood pressure.
[a]BP category is defined according to seated clinic BP and by the highest level of BP, whether systolic or diastolic.
[b]Isolated systolic hypertension is graded 1, 2, or 3 according to SBP values in the ranges indicated.
The same classification is used for all ages from 16 years.

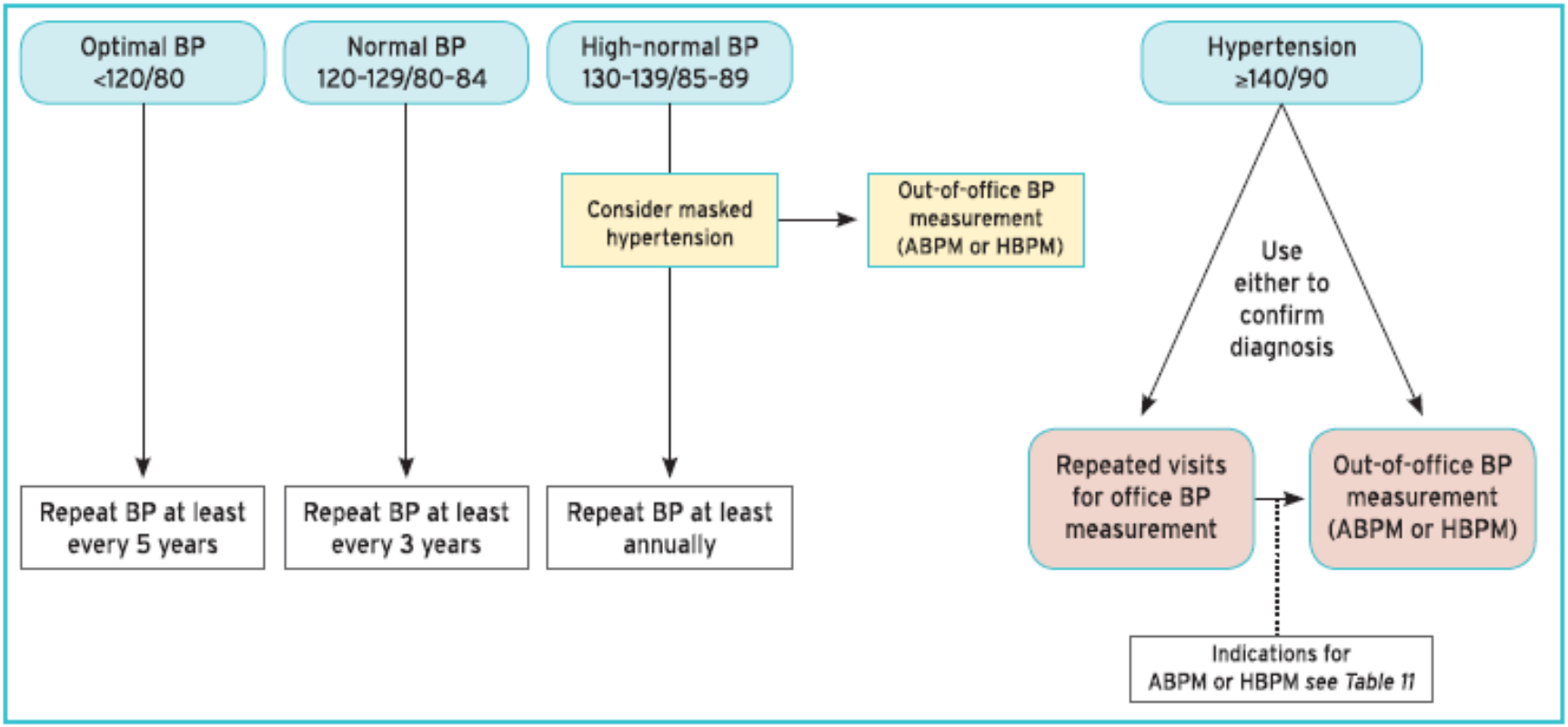


Figure 2 Screening and diagnosis of hypertension. ABPM = ambulatory blood pressure monitoring; BP = blood pressure; HBPM = home blood pressure monitoring.

[a]After detecting a specific BP category on screening, either confirm BP elevation with repeated office BP measurements on repeat visits or arrange use of out-of-office BP to confirm the diagnosis of hypertension.

Table 11 Clinical indications for home blood pressure monitoring or ambulatory blood pressure monitoring

Conditions in which white-coat hypertension is more common, e.g.: • Grade I hypertension on office BP measurement • Marked office BP elevation without HMOD
Conditions in which masked hypertension is more common, e.g.: • High–normal office BP • Normal office BP in individuals with HMOD or at high total CV risk
Postural and post-prandial hypotension in untreated and treated patients
Evaluation of resistant hypertension Evaluation of BP control, especially in treated higher-risk patients Exaggerated BP response to exercise
When there is considerable variability in the office BP
Evaluating symptoms consistent with hypotension during treatment
Specific indications for ABPM rather than HBPM: • Assessment of nocturnal BP values and dipping status (e.g. suspicion of nocturnal hypertension, such as in sleep apnoea, CKD, diabetes, endocrine hypertension, or autonomic dysfunction)

ABPM = ambulatory blood pressure monitoring; BP = blood pressure; CKD = chronic kidney disease; CV = cardiovascular; HBPM = home blood pressure monitoring; HMOD = hypertension-mediated organ damage.

Anamnèse

Table 12 Key information to be collected in personal and family medical history

Risk factors
Family and personal history of hypertension, CVD, stroke, or renal disease
Family and personal history of associated risk factors (e.g. familial hypercholesterolaemia)
Smoking history
Dietary history and salt intake
Alcohol consumption
Lack of physical exercise/sedentary lifestyle
History of erectile dysfunction
Sleep history, snoring, sleep apnoea (information also from partner)
Previous hypertension in pregnancy/pre-eclampsia
History and symptoms of HMOD, CVD, stroke, and renal disease
Brain and eyes: headache, vertigo, syncope, impaired vision, TIA, sensory or motor deficit, stroke, carotid revascularization, cognitive impairment, dementia (in the elderly)
Heart: chest pain, shortness of breath, oedema, myocardial infarction, coronary revascularization, syncope, history of palpitations, arrhythmias (especially AF), heart failure
Kidney: thirst, polyuria, nocturia, haematuria, urinary tract infections
Peripheral arteries: cold extremities, intermittent claudication, pain-free walking distance, pain at rest, peripheral revascularization
Patient or family history of CKD (e.g. polycystic kidney disease)
History of possible secondary hypertension
Young onset of grade 2 or 3 hypertension (<40 years), or sudden development of hypertension or rapidly worsening BP in older patients
History of renal/urinary tract disease
Recreational drug/substance abuse/concurrent therapies: corticosteroids, nasal vasoconstrictor, chemotherapy, yohimbine, liquorice
Repetitive episodes of sweating, headache, anxiety, or palpitations, suggestive of Phaeochromocytoma
History of spontaneous or diuretic-provoked hypokalaemia, episodes of muscle weakness, and tetany (hyperaldosteronism)
Symptoms suggestive of thyroid disease or hyperparathyroidism
History of or current pregnancy and oral contraceptive use
History of sleep apnoea
Antihypertensive Drug Treatment
Current/past antihypertensive medication including effectiveness and intolerance to previous medications
Adherence to therapy

Examen physique

Table 13 Key steps in physical examination

Body habitus
Weight and height measured on a calibrated scale, with calculation of BMI
Waist circumference
Signs of HMOD
Neurological examination and cognitive status
Fundoscopic examination for hypertensive retinopathy
Palpation and auscultation of heart and carotid arteries
Palpation of peripheral arteries
Comparison of BP in both arms (at least once)
Secondary hypertension
Skin inspection: cafe-au-lait patches of neurofibromatosis (phaeochromocytoma)
Kidney palpation for signs of renal enlargement in polycystic kidney disease
Auscultation of heart and renal arteries for murmurs or bruits indicative of aortic coarctation, or renovascular hypertension
Comparison of radial with femoral pulse: to detect radio-femoral delay in aortic coarctation
Signs of Cushing's disease or acromegaly
Signs of thyroid disease

Bilan

Table 14 Routine workup for evaluation of hypertensive patients

Routine laboratory tests
Haemoglobin and/or haematocrit
Fasting blood glucose and glycated HbA_{1c}
Blood lipids: total cholesterol, LDL cholesterol, HDL cholesterol
Blood triglycerides
Blood potassium and sodium
Blood uric acid
Blood creatinine and eGFR
Blood liver function tests
Urine analysis: microscopic examination; urinary protein by dipstick test or, ideally, albumin:creatinine ratio
12-lead ECG

eGFR = estimated glomerular filtration rate; ECG = electrocardiogram; HbA_{1c} = haemoglobin A1c.

Table 15 **Assessment of hypertension-mediated organ damage**

Basic screening tests for HMOD	Indication and interpretation
12-lead ECG	Screen for LVH and other possible cardiac abnormalities, and to document heart rate and cardiac rhythm
Urine albumin:creatinine ratio	To detect elevations in albumin excretion indicative of possible renal disease
Blood creatinine and eGFR	To detect possible renal disease
Fundoscopy	To detect hypertensive retinopathy, especially in patients with grade 2 or 3 hypertension
More detailed screening for HMOD	
Echocardiography	To evaluate cardiac structure and function, when this information will influence treatment decisions
Carotid ultrasound	To determine the presence of carotid plaque or stenosis, particularly in patients with cerebrovascular disease or vascular disease elsewhere
Abdominal ultrasound and Doppler studies	• To evaluate renal size and structure (e.g. scarring) and exclude renal tract obstruction as possible underlying causes of CKD and hypertension • Evaluate abdominal aorta for evidence of aneurysmal dilatation and vascular disease • Examine adrenal glands for evidence of adenoma or phaeochromocytoma (CT or MRI preferred for detailed examination); see section 8.2 regarding screening for secondary hypertension • Renal artery Doppler studies to screen for the presence of renovascular disease, especially in the presence of asymmetric renal size
PWV	An index of aortic stiffness and underlying arteriosclerosis
ABI	Screen for evidence of LEAD
Cognitive function testing	To evaluate cognition in patients with symptoms suggestive of cognitive impairment
Brain imaging	To evaluate the presence of ischaemic or haemorrhagic brain injury, especially in patients with a history of cerebrovascular disease or cognitive decline

ABI = ankle-brachial index; CKD = chronic kidney disease; CT = computed tomography; ECG = electrocardiogram; eGFR = estimated glomerular filtration rate; HMOD = hypertension-mediated organ damage; LEAD = lower extremity artery disease; LVH = left ventricular hypertrophy; MRI = magnetic resonance imaging; PWV = pulse wave velocity.

HTA secondaire

< 5-10 % des cas d'HTA

De plus mauvais pronostic cardiovasculaire par :

- atteinte fréquente des organes cibles
- complications spécifiques à la cause :
  - oedème aigu du poumon en cas d'insuffisance rénale
  - ischémie rénale en cas de sténose des artères rénales
  - arythmies ventriculaires en cas d'hypokaliémie par hyperaldostéronisme
  - …

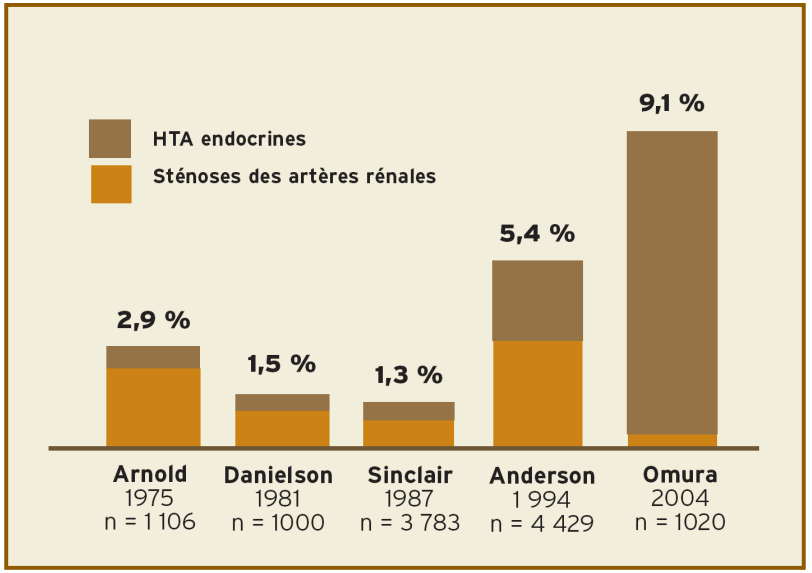


Figure 1 **Prévalence des hypertensions artérielles secondaires dans des centres spécialisés.**

Quand la rechercher ?

Table 25 **Patient characteristics that should raise the suspicion of secondary hypertension**

Characteristic
Younger patients (<40 years) with grade 2 hypertension or onset of any grade of hypertension in childhood
Acute worsening hypertension in patients with previously documented chronically stable normotension
Resistant hypertension (see section 8.1)
Severe (grade 3) hypertension or a hypertension emergency (see section 8.3)
Presence of extensive HMOD
Clinical or biochemical features suggestive of endocrine causes of hypertension or CKD
Clinical features suggestive of obstructive sleep apnoea
Symptoms suggestive of phaeochromocytoma or family history of phaeochromocytoma

CKD = chronic kidney disease; HMOD = hypertension-mediated organ damage.

Table 26 Common causes of secondary hypertension

Cause	Prevalence in hypertensive patients	Suggestive symptoms and signs	Screening Investigations
Obstructive sleep apnoea	5–10%	Snoring; obesity (can be present in non-obese); morning headache; daytime somnolence	Epworth score and ambulatory polygraphy
Renal parenchymal disease	2–10%	Mostly asymptomatic; diabetes; haematuria, proteinuria, nocturia; anaemia, renal mass in adult polycystic CKD	Plasma creatinine and electrolytes, eGFR; urine dipstick for blood and protein, urinary albumin:creatinine ratio; renal ultrasound
Renovascular disease			
Atherosclerotic renovascular disease	1–10%	Older; widespread atherosclerosis (especially PAD); diabetes; smoking; recurrent flash pulmonary oedema; abdominal bruit	Duplex renal artery Doppler or CT angiography or MR angiography
Fibromuscular dysplasia		Younger; more common in women; abdominal bruit	
Endocrine causes			
Primary Aldosteronism	5 - 15%	Mostly asymptomatic; muscle weakness (rare)	Plasma aldosterone and renin, and aldosterone:renin ratio; hypokalaemia (in a minority): note hypokalaemia can depress aldosterone levels
Phaeochromocytoma	<1%	Episodic symptoms (the 5 'Ps'): paroxysmal hypertension, pounding headache, perspiration, palpitations, and pallor; labile BP; BP surges precipitated by drugs (e.g. beta-blockers, metoclopramide, sympathomimetics, opioids, and tricyclic antidepressants)	Plasma or 24 h urinary fractionated metanephrines
Cushing's syndrome	<1%	Moon face, central obesity, skin atrophy, striae and bruising; diabetes; chronic steroid use	24 h urinary-free cortisol
Thyroid disease (hyper- or hypothyroidism)	1 - 2%	Signs and symptom of hyper- or hypothyroidism	Thyroid function tests
Hyperparathyroidism	<1%	Hypercalcaemia, hypophosphataemia	Parathyroid hormone, Ca^{2+}
Other causes			
Coarctation of the aorta	<1%	Usually detected in children or adolescence; different BP (≥20/10 mmHg) between upper–lower extremities and/or between right–left arm and delayed radial-femoral femoral pulsation; low ABI interscapular ejection murmur; rib notching on chest X-ray	Echocardiogram

HTA rénales

- néphropathies chroniques parenchymateuses (3-4 %)
- maladies glomérulaires et vasculaires (vasculites) (y compris à fonction rénale normale)
- polykystose rénale
- insuffisance rénale avancée
- sténose (athéromateuse, fibrodysplasique) de l'artère rénale (HTA rénovasculaire) (1 %)

Examen clinique

- Palpation gros rein
- Souffle vasculaire rénal

Examens complémentaires

- Fonction rénale, protéinurie
- Biopsie rénale
- Échographie rénale
- angioIRM, angioCT

TABLEAU Avantages, inconvénients et limites des différentes techniques d'imagerie utilisées dans le diagnostic des sténoses de l'artère rénale

	Écho-Doppler	TDM multibarettes	IRM
Avantages	Coût faible : 100 € Innocuité Morphologie et fonction Pas de contrindication	Coût moyen : 200 € Morphologie 3D Résolution spatiale haute Étude des surrénales	Produit non néphrotoxique (Gd) Morphologie 3D Non irradiant Calcifications non gênantes
Inconvénients	Opérateur-dépendant Patient-dépendant Limité aux artères et reins	Iode (IRC) Calcifications (degré sténose) Sujet obèse	Coût élevé : 400 € Résolution spatiale < TDM Contre-indications spécifiques Pas d'examen des stents
Limites	Sujet jeune, mince Suivi (stent)	Sujet tout venant Suivi (stent) Cartographie pré-opératoire	Patient ayant une IRC Suivi (non stent) Cartographie pré-opératoire

Gd : gadolinium ; **IRC :** insuffisance rénale chronique ; **IRM :** imagerie par résonance magnétique ; **TDM :** tomodensitométrie.

Coarctation de l'aorte

Examen clinique

- Sujets jeunes
- Absence de pouls fémoraux
- Hypertension artérielle aux membres supérieurs et hypotension aux membres inférieurs
- Souffle à l'endroit de la sténose
- Circulation collatérale thoracique

Examen complémentaire

- angioIRM

HTA endocriniennes

- Hyperaldostéronisme primaire
- Syndrome de Cushing et traitement glucocorticoïde
- Phéochromocytome et paraganglioma
- Dysthyroïdie (HTA systolique)
- Hyperparathyroïdie primaire

Hyperaldostéronisme primaire

- Hyperproduction d'aldostérone soit par un adénome, soit par hyperplasie des glandes surrénales
- A suspecter en cas d'hypokaliémie (< 3,5 mEq/l) avec kaliurie inadaptée (> 30 mEq/24h)
- Élévation du rapport aldostérone plasmatique/activité rénine plasmatique + aldostéronémie ou aldostéronurie augmentée
- Localisation : TDM, cathétérisme veineux sélectif

TABLEAU

Effet des médicaments sur les dosages de rénine et d'aldostérone et délai de sevrage afin d'effectuer les dosages de rénine active et d'aldostérone sans interférence médicamenteuse

	Taux de rénine	Taux d'aldostérone	Effet sur le RAR	Délai de sevrage
Bêtabloquants	↓↓	↓	↑	2 semaines
Diurétiques	↑	↑↑	↓	2 à 4 semaines
Spironolactone Éplérénone	↑	↑↑	↓	6 semaines
IEC	↑	↓	↓	2 semaines
ARA II	↑	↑↑	↓	2 semaines
Inhibiteurs calciques dihydropyridines	↑	→↑	↓	2 semaines
Inhibiteurs de la rénine	↑	↓	↓	6 semaines
AINS	↓↓	↓	↑	2 semaines
Estrogènes de synthèse	↓	→	↑	6 semaines

AINS : anti-inflammatoires non stéroïdiens ; **ARA II** : antagonistes des récepteurs de l'angiotensine 2 ; **IEC** : inhibiteurs de l'enzyme de conversion de l'angiotensine ; **RAR** : rapport aldostérone-rénine.

Syndrome de Cushing

Présentation clinique

- *souvent peu spécifique* :
  - faciès un peu bouffi
  - obésité centrale
  - glycémie limite, petit diabète
  - hypertension artérielle
  - ostéoporose
- *parfois plus typique* :
  - anomalies cutanées : ecchymoses, fragilité, vergetures + amyotrophie des membres
  - dépression, labilité émotionnelle
  - hirsutisme (en faveur cancer surrénalien)
  - hyperpigmentation (en faveur syndrome ectopique)
  - alcalose hypokaliémique (en faveur syndrome ectopique)

Bilan initial

- cortisolurie de 24h (3 récoltes)
- profil cortisolémique (un prélèvement cortisol et ACTH toutes les 4h pendant 24h) : disparition, voire inversion du rythme circadien
- test d'inhibition à la dexaméthasone : significatif si baisse de > 50% cortisolémie

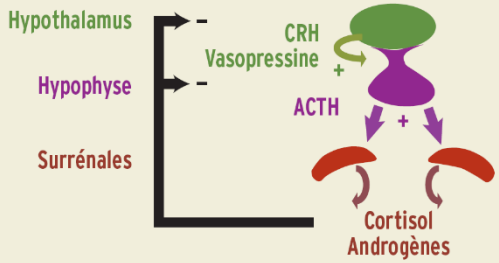
Diagnostic étiologique

- adénome hypophysaire sécrétant de l'ACTH (maladie de Cushing)
- sécrétion ectopique (paranéoplasique) d'ACTH ou CRH (néoplasies)
- syndrome de Cushing ACTH-indépendant : adénome surrénalien, corticosurrénalome, dysplasie micronodulaire, hyperplasie macronodulaire

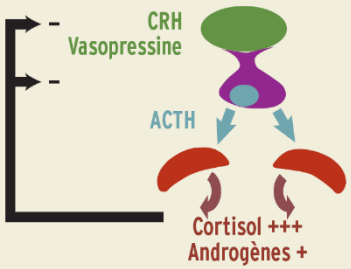
par les tests suivants :

- test léger de Liddle (4 x 0,5 mg/j de dexaméthasone) : chute cortisolémie de > 50 % en faveur origine hypophysaire
- test fort de Liddle (dexaméthasone : 8mg à 23 h ou 2 mg toutes les 6h pendant 48 h) : idem
- test au CRH - minirin (vasopressine) : stimulation

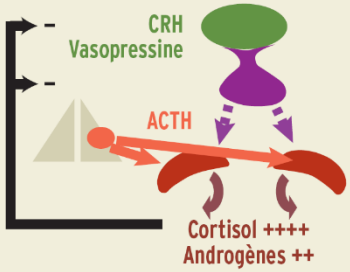
PHYSIOLOGIE



SYNDROME DE CUSHING ACTH-DÉPENDANT

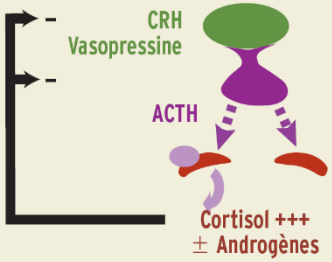


Maladie de Cushing

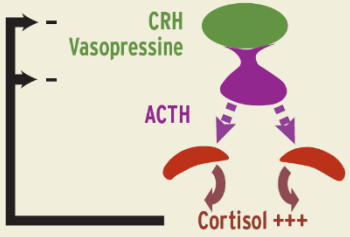


Sécrétion ectopique d'ACTH

SYNDROME DE CUSHING ACTH-INDÉPENDANT



Adénome cortisolique
Corticosurrénalome



Dysplasie micronodulaire pigmentée
Hyperplasie macronodulaire

Localisation

- RMN hypothalamo-hypophysaire (souventt normale)
- RMN surrénale (hyperplasie, parfois nodulaire)
- PET à la méthionine
- cathétérisme des sinus pétreux inférieurs

Phéochromocytomes et paragangliomes

Tableau clinique

- Épisodes paroxystiques d'hypertension artérielle
- Triade classique : céphalées, tachycardie, crises sudorales

Diagnostic

- Dosage des métanéphrines plasmatiques et urinaires
- Localisation : CT ou IRM, scintigraphie au MIBG (méthyl-iodo-benzyl-guanidine)

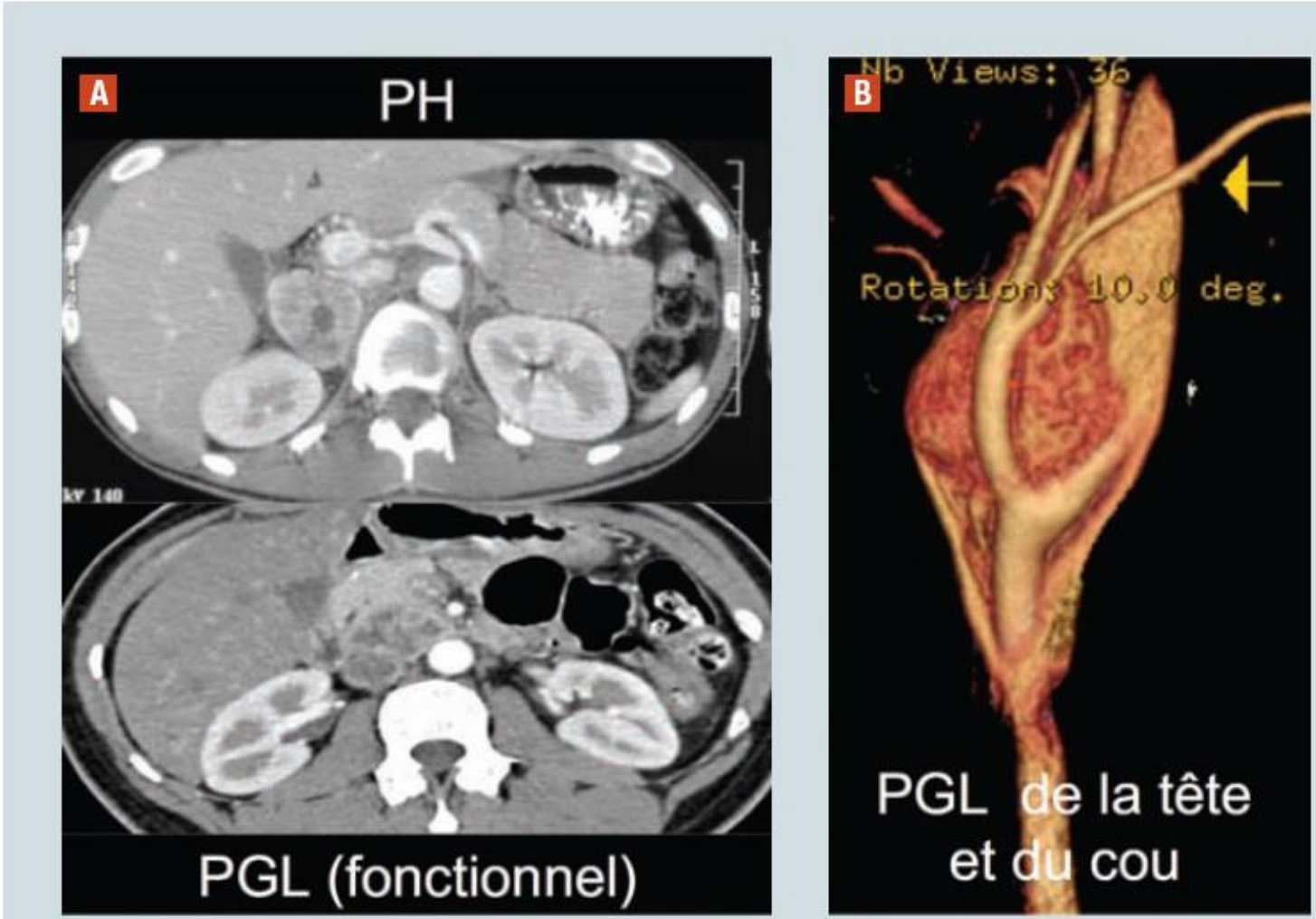


FIGURE 1 **Imagerie du phéochromocytome (PH) et des paragangliomes (PGL).**
A) Tomodensitométrie ; B) Imagerie par résonance magnétique.

TABLEAU

Principales formes génétiques de phéochromocytomes et paragangliomes et modalités de prise en charge

Maladie *Gène de prédisposition*	Premier dépistage	Surveillance
Neurofibromatose type 1 *NF1*	– Examen clinique avec mesure de la pression artérielle et examen cutané – Dosage des métanéphrines totales – Fond d'œil	– Examen clinique avec mesure de la pression artérielle et examen cutané tous les ans – Dosage des métanéphrines totales tous les ans – Fond d'œil tous les ans – TDM (ou IRM) abdominale en cas d'hypertension artérielle ou d'élévation des métanéphrines
Néoplasie endocrinienne de type 2 *RET*	– Examen clinique avec mesure de la pression artérielle – Dosage des métanéphrines totales – TDM (ou IRM) abdominale – Dosage de la calcitonine plasmatique, de la calcémie et de la PTH – Échographie thyroïdienne	– Examen clinique avec mesure de la pression artérielle – Dosage des métanéphrines totales et du calcium tous les ans – Dosage de la calcitonine plasmatique tous les ans (en absence de thyroïdectomie prophylactique) – TDM (ou IRM) abdominale en cas d'hypertension artérielle ou d'élévation des métanéphrines
Maladie de von Hippel-Lindau *VHL*	– Examen clinique avec mesure de la pression artérielle – Dosage des métanéphrines totales – TDM (ou IRM) thoraco-abdomino-pelvienne – Fond d'œil – IRM du système nerveux central et de la moelle	– Examen clinique avec mesure de la pression artérielle – Dosage des métanéphrines totales tous les ans – Fond d'œil tous les ans – TDM (ou IRM) thoraco-abdomino-pelvienne et échographie tous les ans en alternance – IRM du système nerveux central et de la moelle tous les 2 ans
Paragangliome héréditaire *SDHx (SDHA, SDHB, SDHC, SDHD, SDHAF2)*	– Examen clinique avec mesure de la pression artérielle – Dosage des métanéphrines totales – Angio-IRM de la tête et du cou – TDM (ou IRM) thoraco-abdomino-pelvienne – Octréoscan et/ou TEP au 18F-FDG (si mutation *SDHB*) et /ou TEP au 18F-F DOPA (si mutation *SDHD*)	– Examen clinique avec mesure de la pression artérielle – Dosage des métanéphrines totales tous les ans – IRM corps entier tous les 2 ou 3 ans
Phéochromocytome familial *TMEM127* *MAX*	– Examen clinique avec mesure de la pression artérielle – Dosage des métanéphrines totales – Angio-IRM de la tête et du cou – TDM (ou IRM) thoraco-abdomino-pelvienne	– Examen clinique avec mesure de la pression artérielle – Dosage des métanéphrines totales tous les ans – IRM corps entier tous les 2 ou 3 ans

IRM : imagerie par résonance magnétique ; **PTH :** parathormone ; **TDM :** tomodensitométrie ; **TEP :** tomographie par émission de positons.

HTA médicamenteuses et toxiques

- alcool
- stéroïdes (glucocorticoïdes, minéralocorticoïdes, contraception orale…)
- sympathomimétiques (vasconstricteurs nasaux, cocaïne, amphétamines, ecstasy…)
- érythropoïétine
- antiprotéases (indinavir)
- anticalcineurines (ciclosporine, tacrolimus…)
- acide glycérhétinique (réglisse et dérivés)
- antiangiogéniques (bevacizumab, sunitinib…)

TABLEAU 3

Principaux médicaments inducteurs d'hypertension artérielle

- Antidépresseurs : inhibiteurs de la monoamine oxydase (IMAO), tricycliques
- Antihistaminiques
- Antiangiogéniques (inhibiteurs du VEGF)
- Anti-inflammatoires non stéroïdiens classiques et coxibs
- Bêtabloquants (effet rare avec certains d'entre eux)
- Bromocriptine
- Carbamazépine et autres traitements inducteurs du cytochrome P450 (rifampicine, phénytoïne, phénobarbital, millepertuis)
- Ciclosporine
- Contraceptifs oraux contenant de l'éthinylestradiol (effet dose-dépendant plus fréquent si > 50 µg/j)
- Corticothérapie
- Disulfirame (Antabuse)
- Ergotamine
- Érythropoïétine recombinante humaine
- Produits anesthésiants : kétamine, desflurane
- Stéroïdes anabolisants
- Tramadol
- Sympathomimétiques : lévodopa, décongestionnants nasaux (phénylpropanolamine, éphédrine, phényléphrine), anorexigènes (Dexamine), collyre (Visine) : cause rare
- Vasopressine

VEGF : *vascular endothelial growth factor*. D'après la réf. 1.

TABLEAU 4

Principales substances toxiques inductrices d'hypertension artérielle

- Alcool (≥ 30 g/j)
- Amphétamines
- Cocaïne
- Cannabis
- Opioïdes
- Réglisse
- Café à fortes doses (> 4 tasses par jour actuellement remis en question)
- Plomb
- Mercure

D'après la réf. 1

TABLEAU 5

Définition de la consommation à risque d'alcool. Un verre contient 10 g d'alcool pur

Hommes	Femmes
3 verres par jour	2 verres par jour
ou > 21 verres par semaine	ou > 14 verres par semaine
et/ou 5 verres par occasion	et/ou 4 verres par occasion

D'après l'organisation mondiale de la santé.

TABLEAU 2

Principales interactions entre certaines familles thérapeutiques, la pression artérielle et les traitements antihypertenseurs

Famille thérapeutique	Mécanisme d'action	Élévation de la PA	Interaction avec le traitement antihypertenseur
Sympathomimétiques **Exemple : décongestionnants nasaux**	– Récepteur alpha-adrénergique stimulé	Oui	Non
Alcaloïdes de l'ergot de seigle	– Traitement antimigraineux : récepteur sérotoninergique ou récepteur 5 - hydroxytryptamine stimulé – Traitement bronchodilatateur : récepteur bêta-2 stimulé	Oui	Non
Anti-inflammatoires non stéroïdiens	– Rétention hydrosodée – Effet bloquant de la vasodilatation liée aux prostaglandines	Oui	Oui
Contraception avec éthinylestradiol	– Stimulation de la synthèse hépatique de l'angiotensinogène	Oui	Non
Corticostéroïdes	– Rétention hydrosodée – Inhibition de la fonction endothéliale vasodilatatrice – Potentialisation de l'effet des catécholamines	Oui	Oui
Psychotropes : chlorpromazine, tricycliques, IMAO, inhibiteurs de la recapture de la noradrénaline (venlafaxine [Effexor])	– Potentialisation de l'action des catécholamines (inhibition de la recapture de la noradrénaline au niveau des terminaisons nerveuses)	Oui	Oui (antihypertenseurs centraux)
Érythropoïétine recombinante humaine	– Augmentation de la viscosité sanguine – Altération de la fonction endothéliale – Production accrue d'endothéline…	Oui	Non
Ciclosporine	– Altération des fonctions endothéliales (dont la vasodilatation NO-dépendante) – Production accrue d'agents vasopresseurs – Atteinte rénale	Oui	Non
Stéroïdes anabolisants	– Rétention hydrosodée	Oui	Non

IMAO : inhibiteurs de la monoamine oxydase ; NO : oxyde nitrique ; PA : pression artérielle. D'après la réf. 36

Traitement

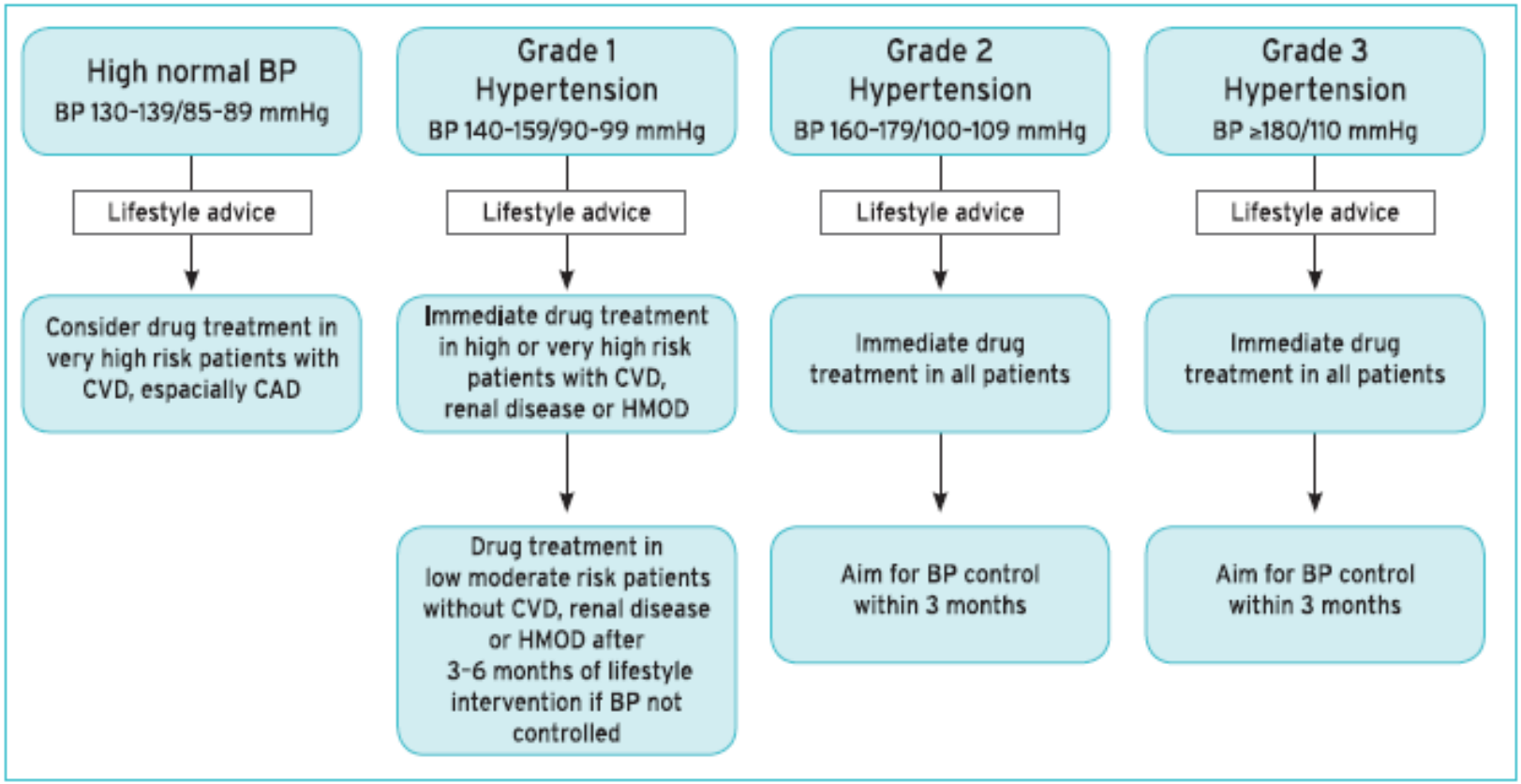

High normal BP
BP 130-139/85-89 mmHg
Lifestyle advice
Consider drug treatment in very high risk patients with CVD, espacially CAD
Grade 1 Hypertension
BP 140-159/90-99 mmHg
Lifestyle advice
Immediate drug treatment in high or very high risk patients with CVD, renal disease or HMOD
Drug treatment in low moderate risk patients without CVD, renal disease or HMOD after 3-6 months of lifestyle intervention if BP not controlled
Grade 2 Hypertension
BP 160-179/100-109 mmHg
Lifestyle advice
Immediate drug treatment in all patients
Aim for BP control within 3 months
Grade 3 Hypertension
BP ≥180/110 mmHg
Lifestyle advice
Immediate drug treatment in all patients
Aim for BP control within 3 months
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Table 19 Summary of office blood pressure thresholds for treatment

Age group	Office SBP treatment threshold (mmHg)					Office DBP treatment threshold (mmHg)
	Hypertension	+ Diabetes	+ CKD	+ CAD	+ Stroke/TIA	
18-65 years	≥140	≥140	≥140	≥140[a]	≥140[a]	≥90
65-79 years	≥140	≥140	≥140	≥140[a]	≥140[a]	≥90
≥80 years	≥160	≥160	≥160	≥160	≥160	≥90
Office DBP treatment threshold (mmHg)	≥90	≥90	≥90	≥90	≥90	

ESC/ESH 2018

BP = blood pressure; CAD = coronary artery disease; CKD = chronic kidney disease; DBP = diastolic blood pressure; SBP = systolic blood pressure; TIA = transient ischaemic attack.

[a]Treatment may be considered in these very high-risk patients with high–normal SBP (i.e. SBP 130–140 mmHg).

Office BP treatment targets in hypertensive patients

Recommendations	Class[a]	Level[b]
It is recommended that the first objective of treatment should be to lower BP to <140/90 mmHg in all patients and, provided that the treatment is well tolerated, treated BP values should be targeted to 130/80 mmHg or lower in most patients.[2,8]	I	A
In patients <65 years receiving BP-lowering drugs, it is recommended that SBP should be lowered to a BP range of 120–129 mmHg in most patients.[c 2,215,229]	I	A
In older patients (aged ≥65 years) receiving BP-lowering drugs:		
• It is recommended that SBP should be targeted to a BP range of 130–139 mmHg.[2,235,244]	I	A
• Close monitoring of adverse effects is recommended.	I	C
• These BP targets are recommended for patients at any level of CV risk and in patients with and without established CVD.[2,8]	I	A
A DBP target of <80 mmHg should be considered for all hypertensive patients, independent of the level of risk and comorbidities.[2,26,235]	IIa	B

Lifestyle interventions for patients with hypertension or high-normal BP

Recommendations	Class[a]	Level[b]
Salt restriction to <5 g per day is recommended.[248,250,255,258]	I	A
It is recommended to restrict alcohol consumption to: • Less than 14 units per week for men. • Less than 8 units per week for women.[35]	I	A
It is recommended to avoid binge drinking.	III	C
Increased consumption of vegetables, fresh fruits, fish, nuts, and unsaturated fatty acids (olive oil); low consumption of red meat; and consumption of low-fat dairy products are recommended.[262,265]	I	A
Body-weight control is indicated to avoid obesity (BMI >30 kg/m^2 or waist circumference >102 cm in men and >88 cm in women), as is aiming at healthy BMI (about 20–25 kg/m^2) and waist circumference values (<94 cm in men and <80 cm in women) to reduce BP and CV risk.[262,271,273,290]	I	A
Regular aerobic exercise (e.g. at least 30 min of moderate dynamic exercise on 5–7 days per week) is recommended.[262,278,279]	I	A
Smoking cessation, supportive care, and referral to smoking cessation programs are recommended.[286,288,291]	I	B

Drug treatment strategy for hypertension

Recommendations	Class[a]	Level[b]
Among all antihypertensive drugs, ACE inhibitors, ARBs, beta-blockers, CCBs, and diuretics (thiazides and thiazide-like drugs such as chlorthalidone and indapamide) have demonstrated effective reduction of BP and CV events in RCTs, and thus are indicated as the basis of antihypertensive treatment strategies.[2]	I	A
Combination treatment is recommended for most hypertensive patients as initial therapy. Preferred combinations should comprise a RAS blocker (either an ACE inhibitor or an ARB) with a CCB or diuretic. Other combinations of the five major classes can be used.[233,318,327,329,341–345]	I	A
It is recommended that beta-blockers are combined with any of the other major drug classes when there are specific clinical situations, e.g. angina, post-myocardial infarction, heart failure, or heart rate control.[300,341]	I	A
It is recommended to initiate an antihypertensive treatment with a two-drug combination, preferably in an SPC. Exceptions are frail older patients and those at low risk and with grade 1 hypertension (particularly if SBP is <150 mmHg).[342,346,351]	I	B
It is recommended that if BP is not controlled[c] with a two-drug combination, treatment should be increased to a three-drug combination, usually a RAS blocker with a CCB and a thiazide/thiazide-like diuretic, preferably as an SPC.[349,350]	I	A
It is recommended that if BP is not controlled[c] with a three-drug combination, treatment should be increased by the addition of spironolactone or, if not tolerated, other diuretics such as amiloride or higher doses of other diuretics, a beta-blocker, or an alpha-blocker.[310]	I	B
The combination of two RAS blockers is not recommended.[291,298,299]	III	A

ACE = angiotensin-converting enzyme; ARB = angiotensin receptor blocker; BP = blood pressure; CCB = calcium channel blocker; CV = cardiovascular; RAS = renin-angiotensin system; RCT = randomized controlled trial; SBP = systolic blood pressure; SPC = single-pill combination.

[a]Class of recommendation.

[b]Level of evidence.

[c]Adherence should be checked.

Table 20 **Compelling and possible contraindications to the use of specific antihypertensive drugs**

Drug	Contraindications	
	Compelling	Possible
Diuretics (thiazides/thiazide-like, e.g. chlorthalidone and indapamide)	• Gout	• Metabolic syndrome • Glucose intolerance • Pregnancy • Hypercalcaemia • Hypokalaemia
Beta-blockers	• Asthma • Any high-grade sinoatrial or atrioventricular block • Bradycardia (heart rate <60 beats per min)	• Metabolic syndrome • Glucose intolerance • Athletes and physically active patients
Calcium antagonists (dihydropyridines)		• Tachyarrhythmia • Heart failure (HFrEF, class III or IV) • Pre-existing severe leg oedema
Calcium antagonists (verapamil, diltiazem)	• Any high-grade sinoatrial or atrioventricular block • Severe LV dysfunction (LV ejection fraction <40%) • Bradycardia (heart rate <60 beats per min)	• Constipation
ACE inhibitors	• Pregnancy • Previous angioneurotic oedema • Hyperkalaemia (potassium >5.5 mmol/L) • Bilateral renal artery stenosis	• Women of child-bearing potential without reliable contraception
ARBs	• Pregnancy • Hyperkalaemia (potassium >5.5 mmol/L) • Bilateral renal artery stenosis	• Women of child-bearing potential without reliable contraception

ACE = angiotensin-converting enzyme; ARB = angiotensin receptor blocker; HFrEF = heart failure with reduced ejection fraction; LV = left ventricular.

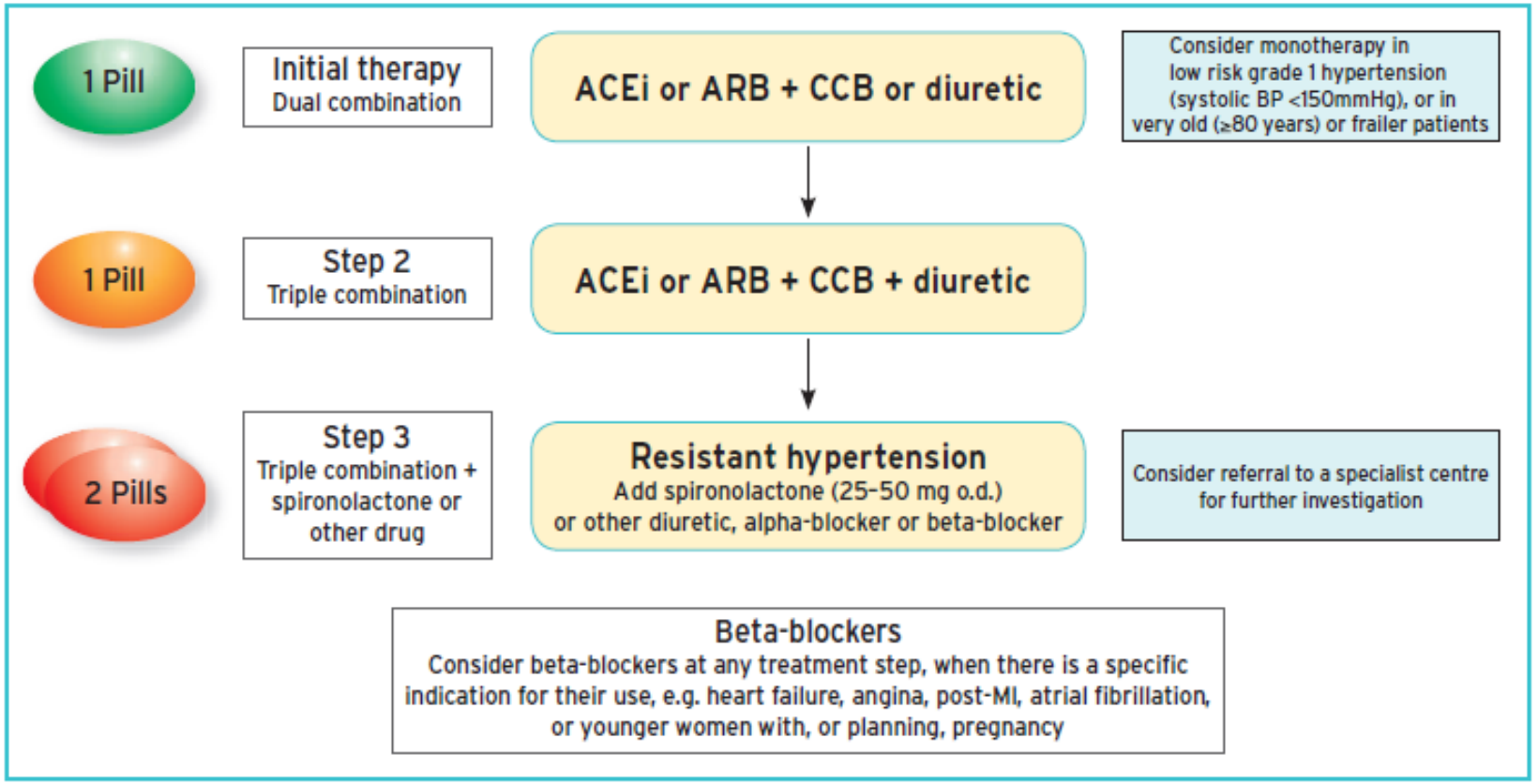

Figure 4 Core drug treatment strategy for uncomplicated hypertension. The core algorithm is also appropriate for most patients with HMOD, cerebrovascular disease, diabetes, or PAD. ACEi = angiotensin-converting enzyme inhibitor; ARB = angiotensin receptor blocker; CCB = calcium channel blocker; HMOD = hypertension-mediated organ damage; MI = myocardial infarction; o.d. = omni die (every day); PAD = peripheral artery disease.

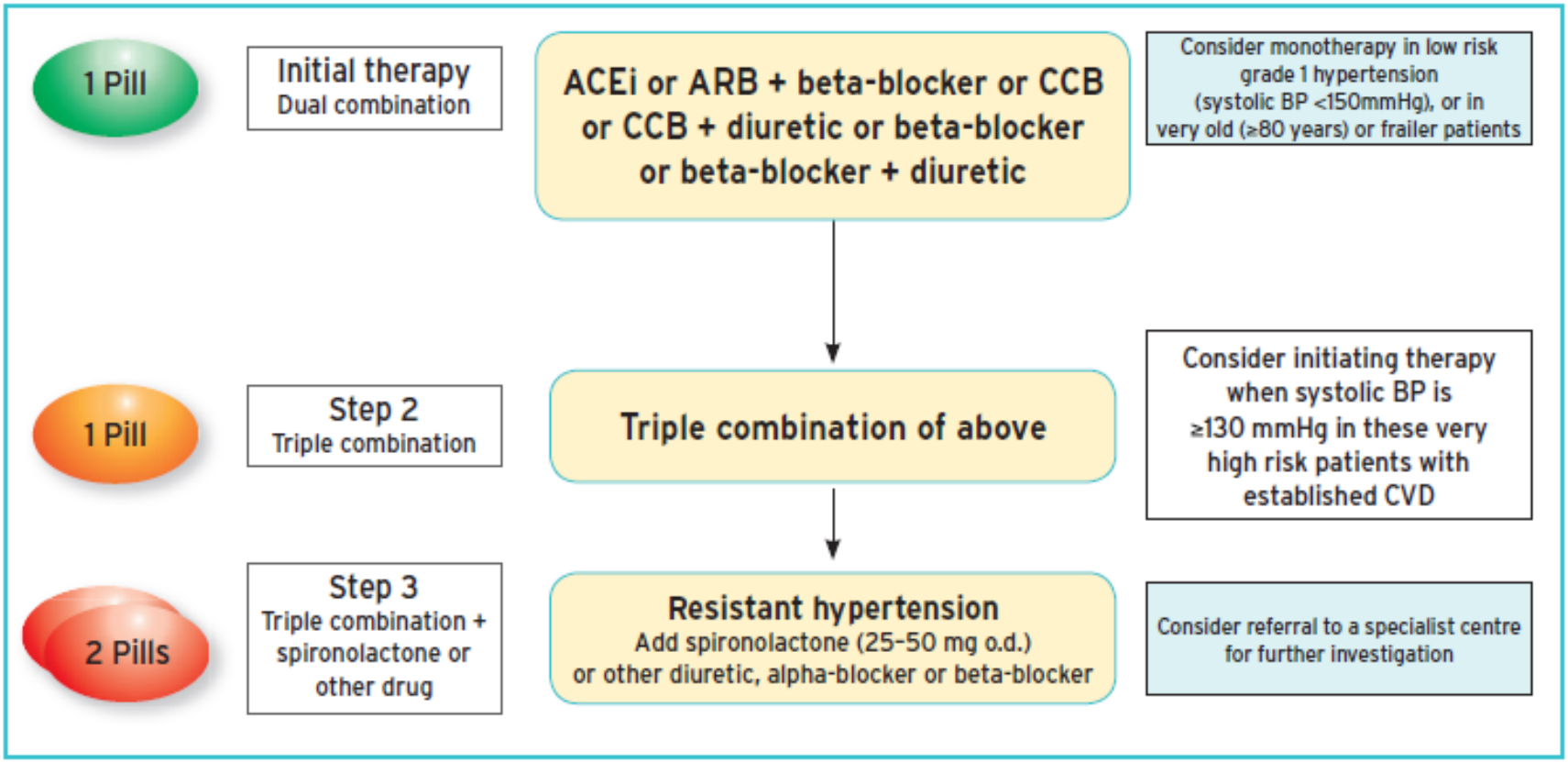

Figure 5 Drug treatment strategy for hypertension and coronary artery disease. ACEi = angiotensin-converting enzyme inhibitor; ARB = angiotensin receptor blocker; BP = blood pressure; CCB = calcium channel blocker; CVD = cardiovascular disease; o.d. = omni die (every day).

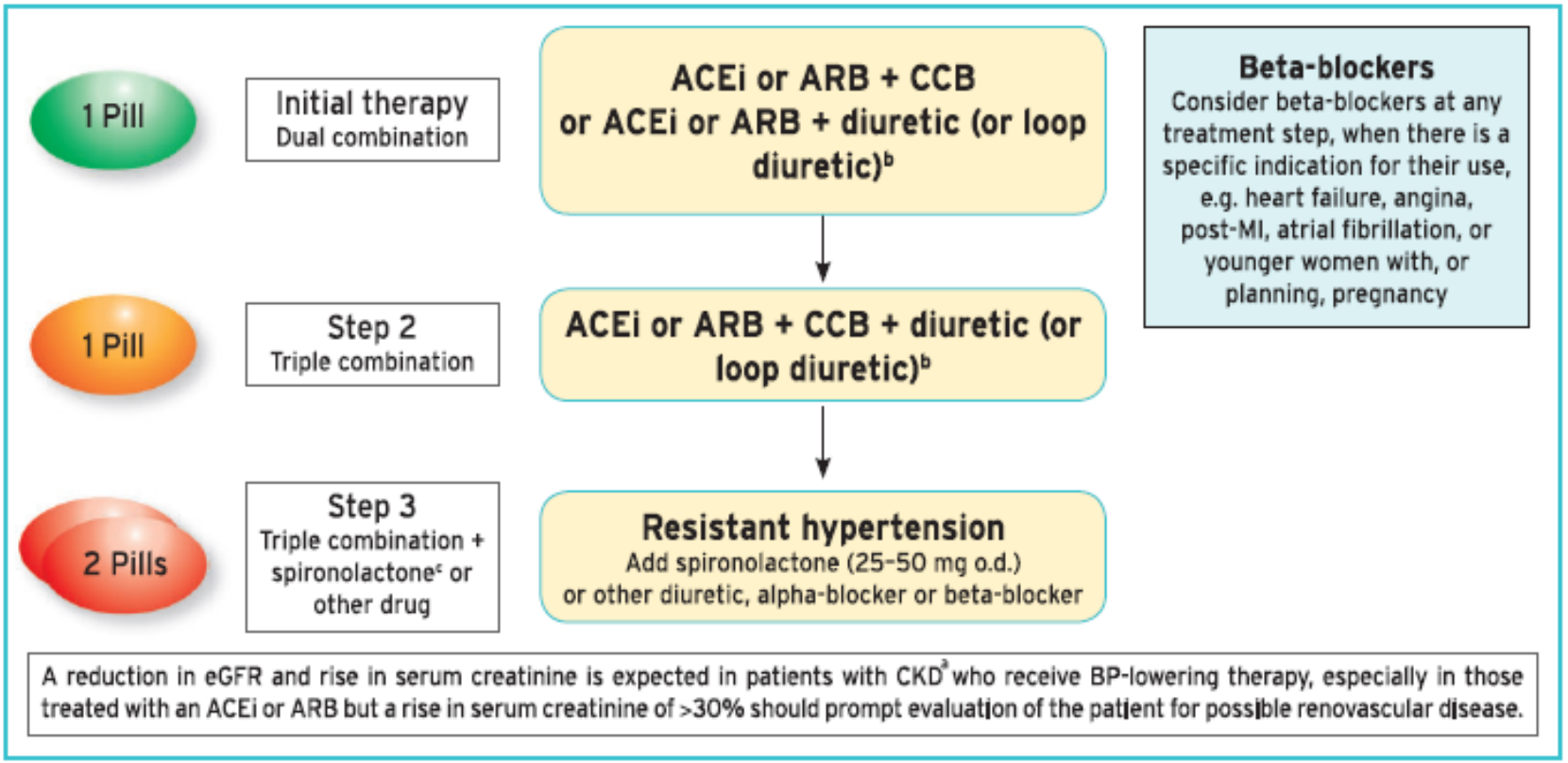

Figure 6 Drug treatment strategy for hypertension and chronic kidney disease. ACEi = angiotensin-converting enzyme inhibitor; ARB = angiotensin receptor blocker; BP = blood pressure; CCB = calcium channel blocker; CKD = chronic kidney disease; eGFR = estimated glomerular filtration rate; MI = myocardial infarction; o.d. = omni die (every day).

[a]CKD is defined as an eGFR <60 mL/min/1.72 m^2 with or without proteinuria.

[b]Use loop diuretics when eGFR is <30 mL/min/1.72 m^2, because thiazide/thiazide-like diuretics are much less effective/ineffective when eGFR is reduced to this level.

[c]Caution: risk of hyperkalaemia with spironolactone, especially when eGFR is <45 mL/min/1.72 m^2 or baseline K^+ ≥4.5 mmol/L.

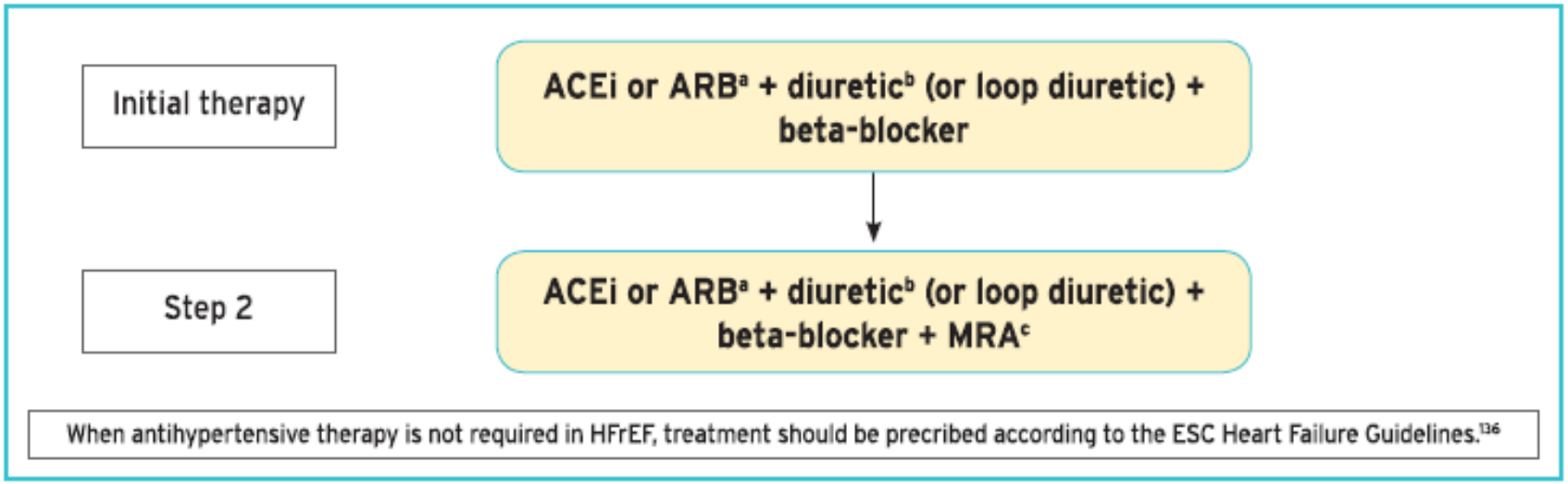

Figure 7 Drug treatment strategy for hypertension and hear failure with reduced ejection fraction. Do not use non-dihydropyridine CCBs (e.g. verapamil or diltiazem). ACEi = angiotensin-converting enzyme inhibitor; ARB = angiotensin receptor blocker; CCB = calcium channel blocker; ESC = European Society of Cardiology; HFrEF = heart failure with reduced ejection fraction; MRA = mineralocorticoid receptor antagonist.
[a]Consider an angiotensin receptor/neprilysin inhibitor instead of ACEi or ARB per ESC Heart Failure Guidelines.[136]
[b]Diuretic refers to thiazide/thiazide-like diuretic. Consider a loop diuretic as an alternative in patients with oedema.
[c]MRA (spironolactone or eplerenone).

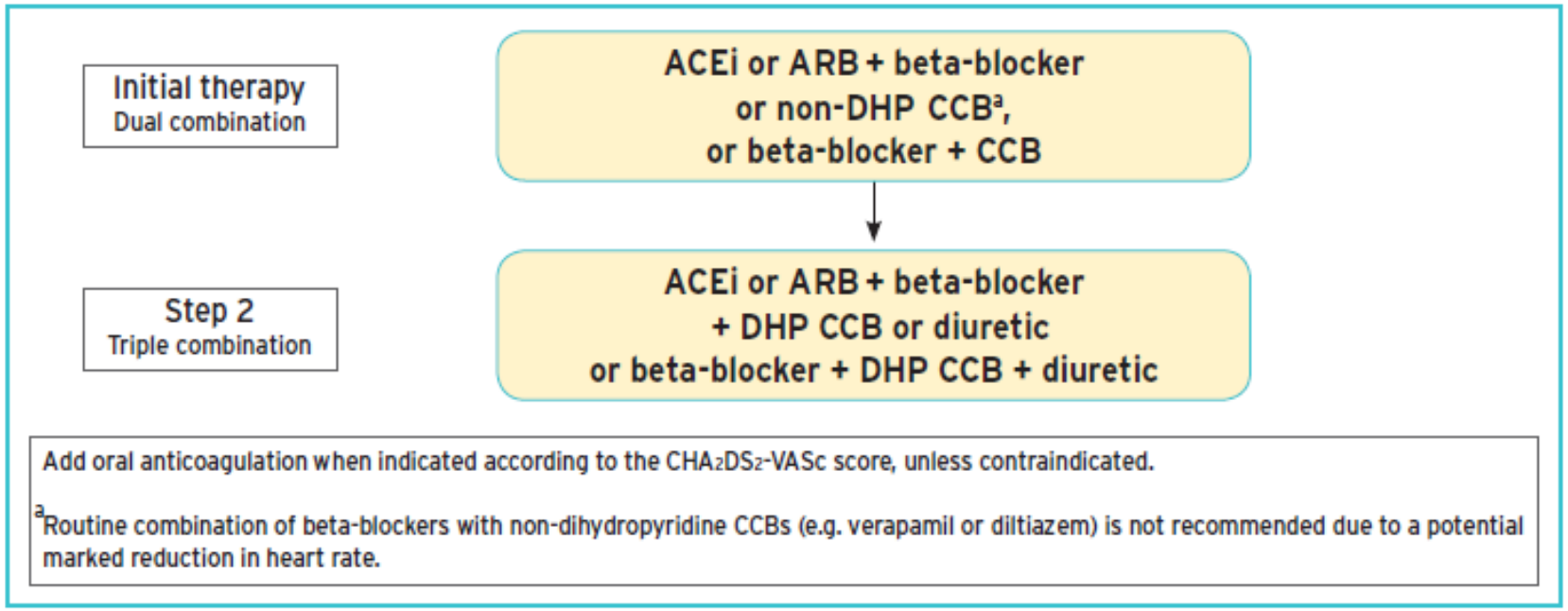

Figure 8 Drug treatment strategy for hypertension and atrial fibrillation. ACEi = angiotensin-converting enzyme inhibitor; AF = atrial fibrillation; ARB = angiotensin receptor blocker; CCB = calcium channel blocker; CHA_2DS_2-VASc = CHA_2DS_2-VASc = Cardiac failure, Hypertension, Age ≥75 (Doubled), Diabetes, Stroke (Doubled) – Vascular disease, Age 65–74 and Sex category (Female); DHP = dihydropyridine. [a]Non-DHP CCB (non-DHP CCB, e.g. verapamil or diltiazem).

Table 23 Office blood pressure treatment target range

Age group	Office SBP treatment target ranges (mmHg)					Office DBP treatment target range (mmHg)
	Hypertension	+ Diabetes	+ CKD	+ CAD	+ Stroke[a]/TIA	
18 - 65 years	**Target to 130** *or lower if tolerated* Not <120	**Target to 130** *or lower if tolerated* Not <120	**Target to <140 to 130** *if tolerated*	**Target to 130** *or lower if tolerated* Not <120	**Target to 130** *or lower if tolerated* Not <120	70–79
65 - 79 years[b]	**Target to 130-139** *if tolerated*	**Target to 130-139** *if tolerated*	**Target to 130-139** *if tolerated*	**Target to 130-139** *if tolerated*	**Target to 130-139** *if tolerated*	70–79
≥80 years[b]	**Target to 130-139** *if tolerated*	**Target to 130-139** *if tolerated*	**Target to 130-139** *if tolerated*	**Target to 130-139** *if tolerated*	**Target to 130-139** *if tolerated*	70–79
Office DBP treatment target range (mmHg)	70–79	70–79	70–79	70–79	70–79	

CAD = coronary artery disease; CKD = chronic kidney disease (includes diabetic and non-diabetic CKD); DBP = diastolic blood pressure; SBP = systolic blood pressure; TIA = transient ischaemic attack.

[a]Refers to patients with previous stroke and does not refer to blood pressure targets immediately after acute stroke.

[b]Treatment decisions and blood pressure targets may need to be modified in older patients who are frail and independent.

Bénéfice escompté

Les méta-analyses de ces essais comparant bêtabloqueur et diurétique au placebo, voire en les associant, ont montré une réduction du risque d'accident vasculaire cérébral de 42 % et d'insuffisance coronarienne de 14 %. Ce bénéfice a été confirmé chez le sujet âgé, avec une réduction de 35 % du risque d'accident vasculaire cérébral et de 15 % du risque d'insuffisance coronarienne.

Crise hypertensive

Définition

élévation sévère de la pression artérielle (grade 3)

- urgence hypertensive : en présence d'atteinte des organes cibles
- crise hypertensive : en l'absence de cette atteinte

Il n'y a pas de raison d'instaurer un traitement en urgence devant une HTA moins sévère

TABLEAU 1 URGENCES HYPERTENSIVES

- Syndrome coronaire aigu
- Syndrome aortique aigu : dissection, hématome ou fissuration d'un anévrisme aortique
- Insuffisance cardiaque gauche dont l'œdème aigu du poumon
- Encéphalopathie hypertensive
- Accident vasculaire cérébral ischémique (constitué ou transitoire) ou hémorragique
- Hémorragie sous-arachnoïdienne
- Hémorragie aiguë d'origine artérielle
- HTA postopératoire
- Éclampsie
- Insuffisance rénale aiguë ou rapidement progressive
- Crise de phéochromocytome
- HTA liée à la prise de substances récréatives (amphétamines, LSD, cocaïne ou ecstasy)
- HTA maligne

Principales étiologies

- arrêt brutal ou sevrage du traitement antihypertenseur chez un hypertendu chronique
- hypertension rénovasculaire et néphropathies (atteintes parenchymateuses, GNA)
- médicaments (sympathomimétiques, antidépresseurs tricycliques, cyclosporine A, corticoïdes, AINS, érythropoïétine)
- phéochromocytome
- hyperhydratation salée
- vasculite
- tumeur sécrétant de la rénine (cancer du rein, lymphomes)
- micro-angiopathie thrombotique
- syndrome de Cushing

Traitement de la crise hypertensive

- Survient chez le patient asymptomatique
- Repos au calme avec contrôle de la pression artérielle très régulièrement: souvent baisse spontanée en 1 heure
- Rechercher un facteur déclenchant à corriger : douleur; hypoxémie, hypercapnie, acidose; hypervolémie; frissons; globe vésical ...
- Eviter toute chute brutale de la pression artérielle (diminution de 25 à 30% dans les premières heures, diminution sur 1 à 2j)
- Traitement antihypertenseur par voie orale éventuellement diurétique thiazidique ou IEC
- En cas d'AVC chez un hypertendu : attendre 48-72 h avant reprise du traitement antihypertenseur

Traitement de l'urgence hypertensive

- en cas d'atteinte d'organes cibles : admettre à l'USI et monitorer la PA
- objectif : réduction de 25 % de la PAM puis graduellement se rapprocher d'une PA de 160/110 mm Hg
- traitement de choix : **nicardipine** : 1 mg i.v. directe par minute, renouvelable jusqu'à 10 mg, puis perfusion de 0,5 à 5 mg/h, en glucosé 5 %, éventuellement jusqu'à 15 mg/h par paliers de 15 min et de 0,5 mg/h (p.o. 3 x 20 mg/j)
- alternative : **labétalol** – α et β bloquant – 20 mg i.v. en 1 min; puis 40 à 80 mg toutes les 10 min i.v. jusqu'à contrôle ou DT 300 mg. Puis perfusion de 30 à 120 mg/h (p.o. 2 x100 à 200 mg/j)
- alternative: **Urapidil** α1-bloquant et action centrale

Table 31 Hypertensive emergencies requiring immediate blood pressure lowering with intravenous drug therapy

Clinical presentation	Timeline and target for BP reduction	First-line treatment	Alternative
Malignant hypertension with or without acute renal failure	Several hours Reduce MAP by 20–25%	Labetalol Nicardipine	Nitroprusside Urapidil
Hypertensive encephalopathy	Immediately reduce MAP by 20–25%	Labetalol, nicardipine	Nitroprusside
Acute coronary event	Immediately reduce SBP to <140 mmHg	Nitroglycerine, labetalol	Urapidil
Acute cardiogenic pulmonary oedema	Immediately reduce SBP to <140 mmHg	Nitroprusside or nitroglycerine (with loop diuretic)	Urapidil (with loop diuretic)
Acute aortic dissection	Immediately reduce SBP to <120 mmHg AND heart rate to <60 bpm	Esmolol and nitroprusside or nitroglycerine or nicardipine	Labetalol OR metoprolol
Eclampsia and severe pre-eclampsia/HELLP	Immediately reduce SBP to <160 mmHg AND DBP to <105 mmHg	Labetalol or nicardipine and magnesium sulfate	Consider delivery

BP = blood pressure; bpm = beats per min; DBP = diastolic blood pressure; HELLP = haemolysis, elevated liver enzymes, and low platelets; i.v. = intravenous; MAP = mean arterial pressure; SBP = systolic blood pressure.

phéochromocytome : labétalol ou phentolamine

Table 32 Drug types, doses, and characteristics for treatment of hypertension emergencies

Drug	Onset of action	Duration of action	Dose	Contraindications	Adverse effects
Esmolol	1–2 min	10–30 min	0.5–1 mg/kg as i.v. bolus; 50–300 µg/kg/min as i.v. infusion	Second or third-degree AV block, systolic heart failure, asthma, bradycardia	Bradycardia
Metoprolol	1–2 min	5–8 h	2.5–5mg i.v. bolus over 2 minutes - may be repreated every 5 minutes to a maximum dose of 15mg	Second or third-degree AV block, systolic heart failure, asthma, bradycardia	Bradycardia
Labetalol	5–10 min	3–6 h	0.25–0.5 mg/kg i.v. bolus; 2–4 mg/min infusion until goal BP is reached, thereafter 5–20 mg/h	Second or third-degree AV block; systolic heart failure, asthma, bradycardia	Bronchoconstriction, foetal bradycardia
Fenoldopam	5–15 min	30–60 min	0.1 µg/kg/min i.v. infusion, increase every 15 min with 0.05 - 0.1 µg/kg/min increments until goal BP is reached	Caution in glaucoma	
Clevidipine	2–3 min	5–15 min	2 mg/h i.v. infusion, increase every 2 min with 2 mg/h until goal BP		Headache, reflex tachycardia
Nicardipine	5–15 min	30–40 min	5–15 mg/h i.v. infusion, starting dose 5 mg/h, increase every 15–30 min with 2.5 mg until goal BP, thereafter decrease to 3 mg/h	Liver failure	Headache, reflex tachycardia
Nitroglycerine	1–5 min	3–5 min	5–200 µg/min i.v. infusion, 5 µg/min increase every 5 min		Headache, reflex tachycardia
Nitroprusside	Immediate	1–2 min	0.3–10 µg/kg/min i.v. infusion, increase by 0.5 µg/kg/min every 5 min until goal BP	Liver/kidney failure (relative)	Cyanide intoxication
Enalaprilat	5–15 min	4–6 h	0.625–1.25 mg i.v. bolus	History of angioedema	
Urapidil	3–5 min	4–6 h	12.5–25 mg as bolus injection; 5–40 mg/h as continuous infusion		
Clonidine	30 min	4–6 h	150–300 µg i.v. bolus over 5–10 min		Sedation, rebound hypertension
Phentolamine	1–2 min	10–30 min	0.5–1 mg/kg i.v. bolus OR 50–300 µg/kg/min as i.v. infusion		Tachyarrhythmias, chest pain

Therapeutic strategies in hypertensive patients with acute stroke and cerebrovascular disease

Recommendations	Class[a]	Level[b]
In patients with acute intracerebral haemorrhage: • Immediate BP lowering is not recommended for patients with SBP <220 mmHg.[509–513]	III	A
• In patients with SBP ≥220 mmHg, careful acute BP lowering with i.v. therapy to <180 mmHg should be considered.[509–513]	IIa	B
In acute ischaemic stroke, routine BP lowering with antihypertensive therapy is not recommended,[516,517] with the exceptions:	III	A
• In patients with acute ischaemic stroke who are eligible for i.v. thrombolysis, BP should be carefully lowered and maintained at <180/105 mmHg for at least the first 24 h after thrombolysis.[514,515]	IIa	B
• In patients with markedly elevated BP who do not receive fibrinolysis, drug therapy may be considered, based on clinical judgement, to reduce BP by 15% during the first 24 h after the stroke onset.	IIb	C
In hypertensive patients with an acute cerebrovascular event, antihypertensive treatment is recommended: • Immediately for TIA.[526]	I	A
• After several days in ischaemic stroke.[526]	I	A
In all hypertensive patients with ischaemic stroke or TIA, an SBP target range of 120–130 mmHg should be considered.[244,524,526]	IIa	B
The recommended antihypertensive drug treatment strategy for stroke prevention is a RAS blocker plus a CCB or a thiazide-like diuretic.[338]	I	A

BP = blood pressure; CCB = calcium channel blocker; i.v. = intravenous; RAS = renin–angiotensin system; SBP = systolic blood pressure; TIA = transient ischaemic attack.
[a]Class of recommendation.
[b]Level of evidence.