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En la revista *Journal of Hypertension* se publico la [Guía de Práctica Clínica para el tratamiento de la hipertensión arterial de la Sociedad Europea de Hipertensión \(ESH\)](#), avalada por la Asociación Renal Europea (ERA) y la Sociedad Internacional de Hipertensión (ISH), que consta de 22 secciones que sobre temas relacionados con el diagnóstico y el tratamiento de la hipertensión.

Los cambios más relevantes con respecto a la guía previa de 2018 son:

- Mayor importancia de las mediciones de presión arterial fuera de la consulta.
- Utilización de los suplementos de potasio como recomendación relacionada con el estilo de vida.
- Consejos más explícitos sobre el inicio de tratamiento con medicamentos betabloqueantes.
- Consideraciones sobre la denervación renal, en adición o como alternativa al aumento de la medicación, en pacientes con hipertensión resistente no controlada.
- Enfoque simplificado del tratamiento con antihipertensivos en pacientes con insuficiencia cardíaca.
- Recomendaciones para el tratamiento de pacientes con enfermedad renal crónica, incluyendo el uso de los inhibidores selectivos del transportador renal de glucosa (iSGLT-2 o gliflozinas) y la finerenona, que es un antagonista mineralocorticoide no esteroideo.
- Terapia antihipertensiva para prevenir el deterioro cognitivo y la progresión a demencia.

Además, se hacen pequeñas modificaciones en recomendaciones sobre la medición, clasificación, intervenciones en el estilo de vida, comienzo y elección del tratamiento con medicamentos antihipertensivos, uso de combinaciones a dosis fijas en una sola forma farmacéutica y aumento de la dosis en adultos con control inadecuado.

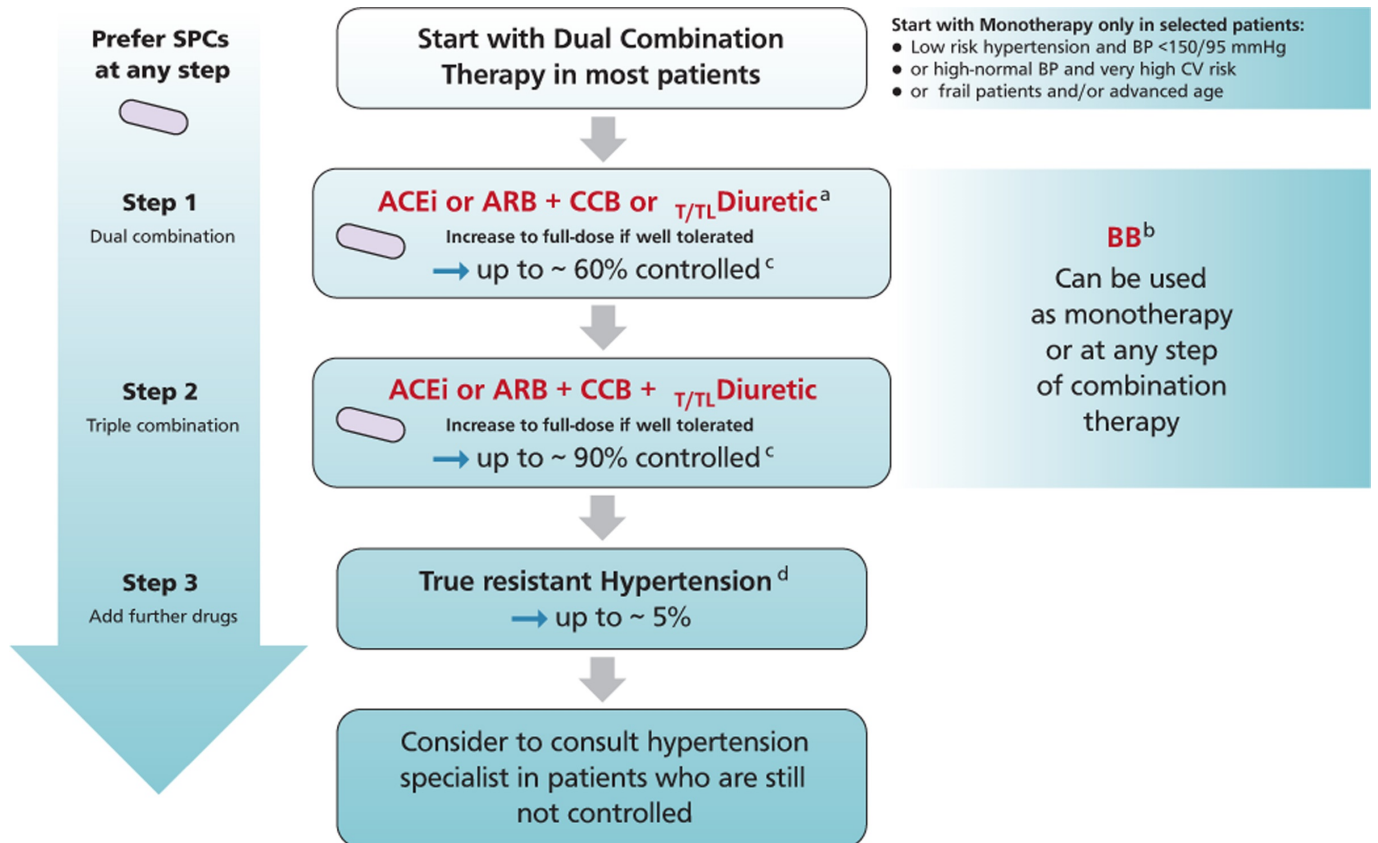
Relacionado con este tema se puede consultar el BTA del 2020 sobre [tratamiento de la hipertensión arterial: nuevas guías](#), que incluye consideraciones sobre la guía europea anterior a esta actualización.

A continuación, y a modo de ejemplo, se reproducen la figura 12 sobre estrategia general para bajar la presión arterial en pacientes con hipertensión; y las tablas: 15, 20 y 25 sobre: contraindicaciones y condiciones que requieren un uso cauteloso de fármacos para reducir la presión arterial; medicamentos y otras sustancias que pueden aumentar la presión sanguínea; y, tipos de medicamentos, dosis y

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características para el tratamiento de emergencia de la hipertensión, respectivamente.

Figure 12. General bP lowering strategy in patients with Hypertension



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TABLE 15. Compelling contraindications and conditions requiring cautious use of BP-lowering drugs

Drug class	Contraindications	Cautious use
ACEi	<ul style="list-style-type: none"> • Pregnancy • Women planning pregnancy • Previous angioneurotic edema • Severe hyperkalemia (e.g. potassium >5.5 mmol/l) • Bilateral renal artery stenosis or stenosis in solitary (functional) kidney 	<ul style="list-style-type: none"> • Women of child-bearing potential without reliable contraception
ARB	<ul style="list-style-type: none"> • Pregnancy • Women planning pregnancy • Severe hyperkalemia (e.g. potassium >5.5 mmol/l) • Bilateral renal artery stenosis or stenosis in solitary (functional) kidney 	<ul style="list-style-type: none"> • Women of child-bearing potential without reliable contraception
Beta-blocker	<ul style="list-style-type: none"> • Severe asthma • Any high-grade sino-atrial or atrioventricular block • Bradycardia (e.g. heart rate <60bpm) 	<ul style="list-style-type: none"> • Asthma • Glucose intolerance • Athletes and physically active patients
DHP-CCB		<ul style="list-style-type: none"> • Tachyarrhythmia • Heart failure (HFrEF, class III or IV) • Preexisting severe leg edema
Non-DHP-CCB (verapamil, diltiazem)	<ul style="list-style-type: none"> • Any high-grade sino-atrial or AV block • Severe LV dysfunction (LV EF <40%), HFrEF • Bradycardia (e.g. heart rate <60bpm) • Co-medications susceptible to significant drug interactions mediated by P-gp or CYP3A4 	<ul style="list-style-type: none"> • Constipation
Thiazide/Thiazide-like diuretics	<ul style="list-style-type: none"> • Hyponatremia • CKD due to obstructive uropathy • Sulfonamide allergies 	<ul style="list-style-type: none"> • Gout • Glucose intolerance • Pregnancy • Hypercalcemia • Hypokalemia • Cancer patients with bone metastasis
MRA	<ul style="list-style-type: none"> • Severe hyperkalemia (e.g. potassium >5.5 mmol/l) • eGFR <30 ml/min/1.73 m² 	<ul style="list-style-type: none"> • Co-medications susceptible to significant drug interactions mediated by P-gp or CYP3A4 for eplerenone

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TABLE 20. Medications and other substances that may increase BP

Medication/substance	Proposed mechanism	Comments
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 cyclooxygenases 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		Caffeine may cause persistent BP effects with regular consumption.
- Modafinil - Amphetamines - Methylphenidate	Block noradrenaline or dopamine reuptake. Promote release of catecholamines	Genetic polymorphisms may affect BP response.
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		Cocaine induces acute but not chronic increase in BP.
- MDMA - PCP - Methamphetamine	Increased release and inhibited reuptake of monoamine neurotransmitters with subsequent SNS activation. Increased CNS catecholamine release with decreased neuronal uptake.	Alcohol causes a dose-dependent, sustained increase in BP independent from obesity or salt intake.
- Cocaine	Cocaine induces an increase in arterial wall stiffness and atherosclerosis.	
- Alcohol	Alcohol increases SNS and RAS activity.	
Herbal products		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.
- 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.	
Diet pills		Mild increase in BP.
- Sibutramine - Phenylpropranolamine	Increased levels of norepinephrine with subsequent activation of noradrenergic transmission	

SNRI, serotonin–noradrenaline reuptake inhibitors.

Table 25. Drug types, dose and characteristics for treatment of hypertension emergencies

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Drug	Onset of action	Duration of action	Dose	Contraindications	Adverse effects
Esmolol	1 min	10–30 min	0.5–1 mg/kg i.v. bolus; 50–300 µg/kg/min i.v. infusion	Second-degree or third-degree AV block, systolic heart failure, asthma, bradycardia	Bradycardia
Metoprolol	1–2 min	5–8 h	2.5–5 mg i.v. bolus over 2 min; may repeat every 5 min to a maximum dose of 15 mg	Second-degree or third-degree AV block, systolic heart failure, asthma, bradycardia	Bradycardia
Labetalol ^a	5–10 min	3–6 h	10–20 mg i.v. bolus in 1 min; incremental doses ≥20 mg may be administered i.v. at 10 min intervals (max 80 mg) or 1–3 mg/min i.v. infusion until goal BP is reached	Second-degree or third-degree AV block; systolic heart failure, asthma, bradycardia	Bronchoconstriction, fetal bradycardia
Fenoldopam	5–15 min	30–60 min	0.1–0.3 µg/kg/min i.v. infusion, increase every 15 min with 0.1 µg/kg/min increments until goal BP is reached	Cautions in glaucoma	
Clevidipine	2 min	10 min	1–2 mg/h i.v. infusion, increase every 2 min with 2 mg/h until goal BP, then titrate by smaller increments every 5–10 min		Headache, reflex tachycardia
Nicardipine	5–15 min	4–6 h	5–15 mg/h i.v. infusion, starting dose 5 mg/h, increase every 15–30 min with 2.5 mg until goal BP, maximum 15 mg/h	Liver failure	Headache, reflex tachycardia
Nitroglycerine	1–5 min	5–10 min	5–200 µg/min i.v. infusion, 5 µg/min increase every 5 min		Headache, reflex tachycardia
Nitroprusside	Immediate	1–3 min	0.3–0.5 µg/kg/min i.v. infusion, increase by 0.5 µg/kg/min every 5 min until goal BP (maximum dose 10 µg/kg/min)	Liver/kidney failure (relative)	Cyanide intoxication
Enalaprilat	5–15 min	4–6 h	0.62–1.25 mg i.v. bolus given over 5 min every 6 h	History of angioedema	
Urapidil	3–5 min	4–6 h	12.5–25 mg i.v. bolus; 5–40 mg/h as continuous infusion		
Clonidine	30 min	4–6 h	0.2–0.5 µg/kg/min i.v.		Sedation, rebound hypertension
Phentolamine	1–2 min	10–30 min	1–5 mg i.v. bolus or continuous i.v. infusion at a rate of 0.5–20 µg/kg/min		Tachyarrhythmia, chest pain

^aNot available in several countries.