COMMUNITY HEALTH CARE ASSOCIATION of New York State

Hypertensive Urgencies and Emergencies

Hypertension Care & Management Webinar Series
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UR MEDICINE

Conflicts of Interest

There are no disclosures

Objectives

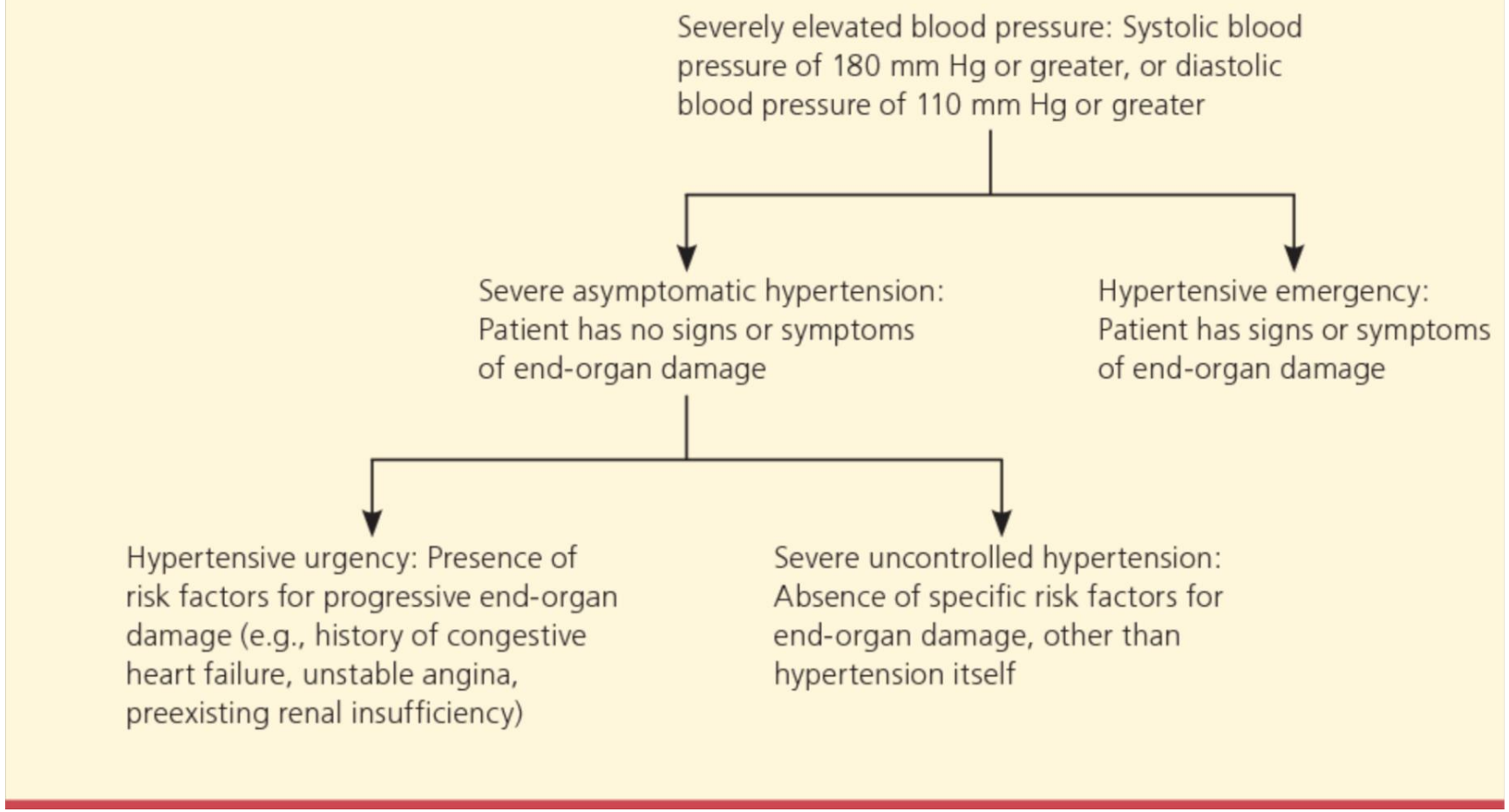
1. Definition of hypertensive urgencies and emergencies
2. Describe epidemiology and disparities in prevalence and control
3. Identify treatment options for patients that present with uncontrolled hypertension in clinic, including symptomatic vs. asymptomatic considerations
4. Explore indications for hospitalization for patients with severe hypertension
5. Review transition of care considerations from the inpatient setting to primary care practice.

Definition

Hypertensive Crises include Hypertensive Emergencies and Hypertensive Urgencies. The term hypertensive crisis is used to describe patients who present with severe BP elevations as follows: Systolic blood pressure and (SBP) greater than 180 mm Hg and Diastolic blood pressure (DBP) greater than 120 mm Hg).

The diagnosis can be further classified as a hypertensive emergency when severe elevation in BP is associated with end-organ damage or hypertensive urgency when severe hypertension occurs without it.

Classification of Severely Elevated Blood Pressure



American family physician,2010 volume={81 4}

Background

Hypertension is an important contributor to global morbidity and mortality.

Despite therapeutic lifestyle and pharmacological measures, a significant proportion of people with hypertension do not reach treatment targets.

Uncontrolled arterial hypertension is a common cause of admittance to the Emergency Departments.

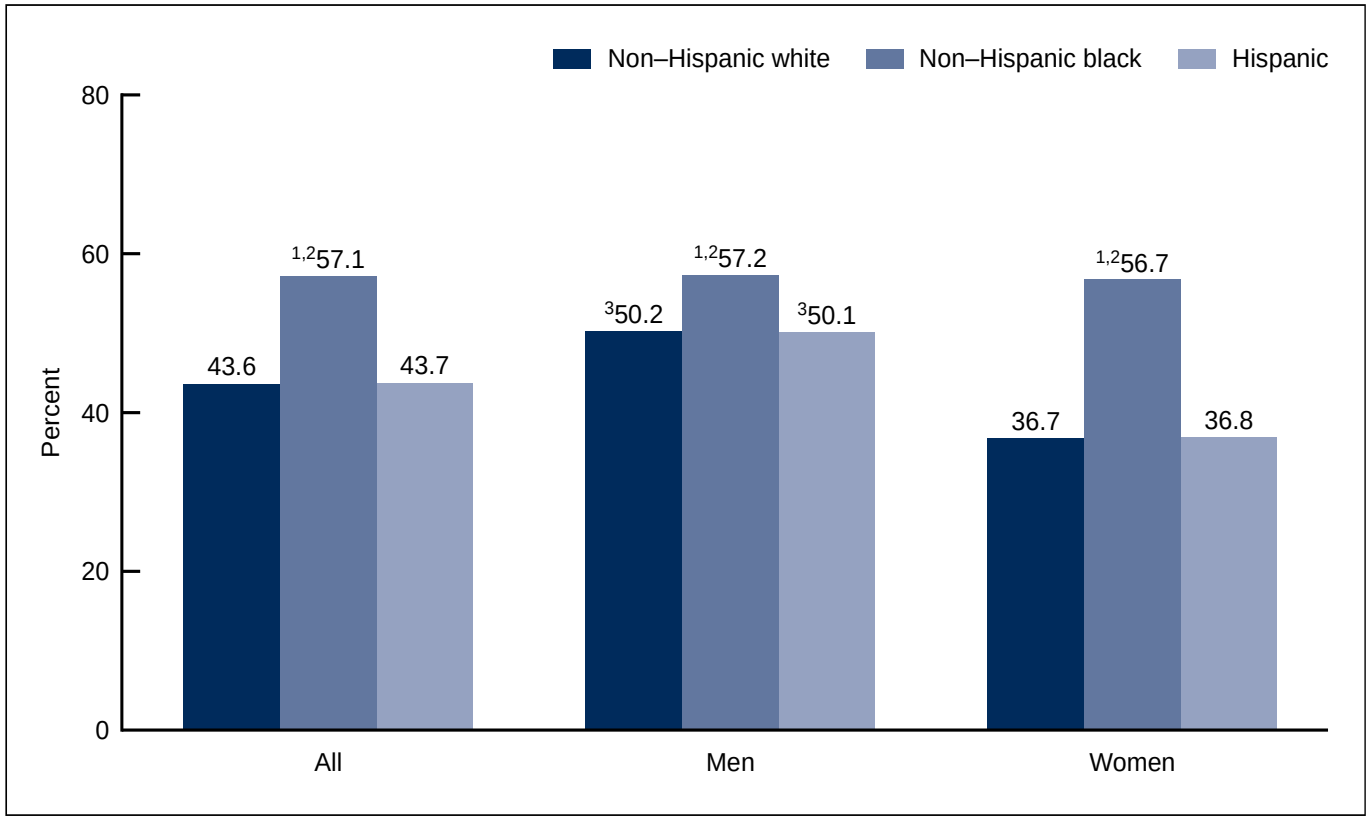
The majority of hypertensive emergencies occur in patients already diagnosed with chronic hypertension.

Epidemiology

- Approximately 1-2% hypertension patients may develop hypertensive crisis during their lifetime.
- Annual incidence of hypertensive emergencies being 1-2 cases/1,000.000 patients
- Higher rates have been reported in African Americans, low socioeconomic population, in developing countries.
- Incidence in men 2 times higher than in women
- Parallels the distribution of primary hypertension

Current Opinion Cardiol 2006

Age-adjusted prevalence of HTN among adults aged 18 and older, by sex and race and Hispanic origin: United States 2017-18



[1]Significantly different from non-Hispanic white.
[2]Significantly different from Hispanic.
[3]Significantly different from women in the same race and Hispanic-origin group.

Age-adjusted prevalence of HTN was significantly higher among non-Hispanic blacks, than among non-Hispanic white and Hispanic adults

The prevalence of HTN among women is significantly lower in non-Hispanic white and Hispanic adults compared to men

Among non-Hispanic blacks, the prevalence of HTN does not significantly differ by sex

Racial disparities in hypertension prevalence have persisted over time

Hypertensive Crisis

25% of all medical emergencies in the ER present as hypertensive crises

25% of the patients with hypertensive crisis present as Hypertensive Emergencies and 75% as Hypertensive Urgencies

Mortality for HEs is held to be substantial, i.e. about 4%; moreover, the high BP values are instrumental in driving organ damage,

The 1-year death rate associated with hypertensive emergencies is >79%, and the median survival is 10.4 months if the emergency is left untreated.

Etiology

- Noncompliance with antihypertensive medications (especially clonidine withdrawal).
- The use of sympathomimetic drugs (e.g., cocaine, over-the-counter decongestants).
- Nonadherence with CPAP or BiPAP therapy for sleep disordered breathing.
- Volume overload.
- Pain, anxiety, urinary obstruction.
- Other medications (e.g., cyclosporine, tacrolimus, erythropoietin, steroid, NSAIDs, anti-angiogenic drugs).
- Withdrawal (e.g., from alcohol or benzodiazepines).
- Stroke (e.g., ischemic stroke, intracranial hemorrhage, subarachnoid hemorrhage).
- Aortic dissection.
- Preeclampsia.
- Endocrinopathy (e.g., pheochromocytoma, hyperaldosteronism, Cushing's syndrome, hyperthyroidism).
- Renal (scleroderma renal crisis, acute glomerulonephritis, renal artery stenosis).

Symptoms of hypertensive urgencies and emergencies

Hypertensive urgencies	Hypertensive emergencies
Headache 22%	Chest pain 27%
Epistaxis 17%	Dyspnea 22%
Somnolence 9%	Neurologic deficits 21%
Chest pain 9%	Somnolence 10%
Dyspnea 9%	Paraesthesia 8%
Neurologic deficits 7%	Nausea, vomiting, dizziness, headache 12%

Emergency investigations

- Short history and physical examination
- Repeated measurement of BP to confirm severe elevation of BP
- ECG
- Chest X-ray
- Fundoscopy
- Lab: serum electrolytes, creatinine, urea, urinalysis, CBC, troponin and CK-MB if needed
- Abdominal U/S: if dissecting aortic aneurysm is suspected
- Echocardiography if needed
- CT scan of the brain: if neurological deficit is present

Mean arterial pressure (MAP)

Management should be based on MAP

$$MAP = \frac{SBP + 2(DBP)}{3}$$

Range 70-110 mmHg

The average pressure of the arteries during one cardiac cycle.

It is considered to be a better indicator of perfusion to vital organs than systolic blood pressure.

It is vital to have MAP of at least 60 mmHg to provide enough blood to coronary arteries, kidneys and brain.

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ORIGINAL ARTICLE

Management of hypertensive emergencies: a practical approach

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ABSTRACT

Background: Acute increases of high blood pressure values, usually described as 'hypertensive crises', 'hypertensive urgencies' or 'hypertensive emergencies', are common causes of patients' presentation to emergency departments. Owing to the lack of ad hoc randomized clinical trials, current recommendations/suggestions for treatment of these patients are not evidenced-based and, therefore, the management of acute increases of blood pressure values represent a clinical challenge. However, an improved understanding of the underlying pathophysiology has changed radically the approach to management of the patients presenting with these conditions in recent years. Accordingly, it has been proposed to abandon the terms 'hypertensive crises' and 'hypertensive urgencies', and restrict the focus to 'hypertensive emergencies'.

Aims and Methods: Starting from these premises, we aimed at systematically review all available studies (years 2010-2020) to garner information on the current management of hypertensive emergencies, in order to develop a novel symptoms- and evidence-based streamlined algorithm for the assessment and treatment of these patients.

Results and Conclusions: In this educational review we proposed the BARKH-based algorithm for a quick identification of hypertensive emergencies and associated acute organ damage, to allow the patients with hypertensive emergencies to receive immediate treatment in a proper setting.

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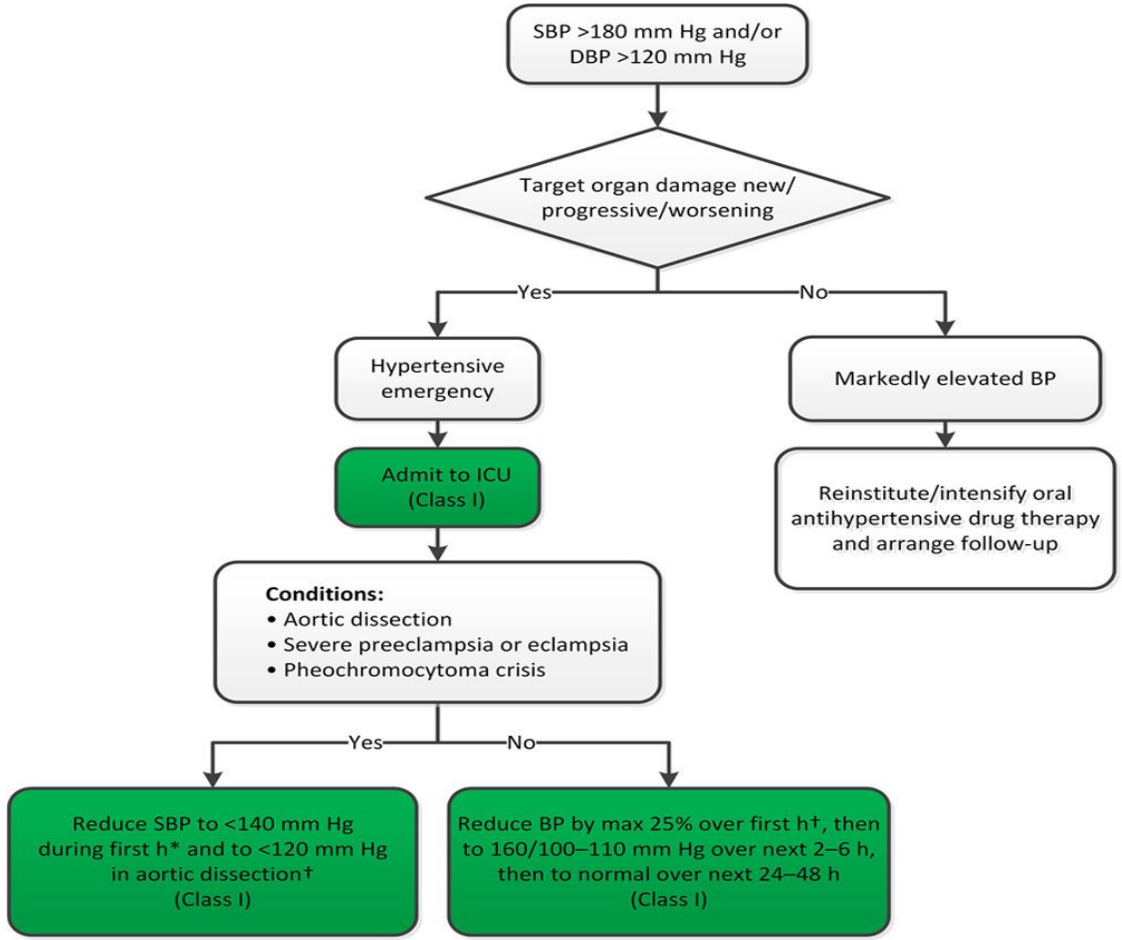
KEYWORDS
Hypertensive emergencies; hypertensive crises; urgencies; organ damage; treatment

Diagnosis and management of a hypertensive crisis



Paul K. Whelton. Hypertension. 2017 A Report of the American College of Cardiology/American Heart Association Task Force on Clinical Practice Guidelines, Volume: 71, Issue: 6, Pages: e13-e115

Hypertensive urgency

- Severely elevated blood pressure (SBP >180 and/or DBP >120 mm Hg) without signs and symptoms of end-organ damage
- It has less serious prognosis than hypertensive emergency in spite of severe hypertension
- Most of the hypertensive crisis presentations (75%) are hypertensive urgencies
- Can be managed as outpatient
- Check medication adherence
- Can be managed with oral medications

Treatment of hypertensive urgencies

This a misnomer because there is no “urgent” need to reduce the blood pressure, specifically:

(1) There is NO need for referral to the emergency department.

(2) There is NO need for hospital admission.

(3) There is *definitely* NO need for ICU admission!

General guidelines - immediate therapy:

- Gradual lowering of elevated BP within 24 hours to avoid the development of serious complications
- Controlled lowering of elevated BP (reduction of mean arterial pressure by 25%, aim DBP 100 - 110 mm Hg) to avoid sudden drop of BP and cerebral hypoperfusion - outpatient treatment is possible.

Hypertensive Urgency, Therapy

- There is lack of compelling evidence to recommend one antihypertensive agent or class of agents over another in treating hypertensive urgencies
- Certain agents, however, should be avoided
  - Data suggests that sublingual nifedipine and sublingual captopril have the potential to lower BP in a rapid and uncontrolled fashion, and may result in morbidity from cardiac and cerebral ischemia
- Goal is to reduce BP to ≤160/≤100 mmHg but the mean arterial pressure should not be lowered by more than 25-30% over the first several hours.
- Many hypertensive urgencies occur in known hypertensives who are inadequately treated
  - For these individuals, it may make sense to restart or increase the dose of a current Rx
  - On the other hand, if a Rx's side effects are causing non-compliance it would be prudent to offer a Rx with more tolerable profile
  - Addition of diuretic and dietary sodium restriction if etiology his high Na intake
- In other situations, it may be useful to add an additional agent to a patient's regimen for additional BP lowering
- Be sure to remember that medication non-compliance can be a result of a drug's cost (financial side-effects) as well as its true physical side-effects
- Certain Rx's, though effective, may be less desirable to use in hypertensive urgencies
  - Clonidine is an effective antihypertensive, however, risks of rebound HTN may outweigh its benefits when used in an urgent care situation where follow-up and patient compliance cannot be guaranteed
  - Other antihypertensives such as amlodipine and HCTZ can take days to affect BP

Hypertensive emergency

- According to Joint National Committee on hypertension report
- Severely elevated BP (systolic >180 mm Hg and/or diastolic >120) with signs and
- symptoms of acute end organ damage
- Life-threatening (mortality 20 % if untreated) requires immediate but careful intervention
- Requires hospitalization
- Requires parenteral medications

Hypertensive emergencies

Cerebrovascular
- Hypertensive encephalopathy
- Ischemic stroke
- Intracerebral/subarachnoid hemorrhage

Cardiac
- Acute aortic dissection
- Acute left ventricular failure
- Acute or impending MI
- After CABG

Renal
- Acute renal failure
- Acute glomerulonephritis
- Tubulointerstitial nephritis
- Renal crises from collagen vascular disease
- Post-kidney transplant
- Microangiopathic hemolytic anemia

Excessive circulating catecholamines
- Pheochromocytoma crisis
- Food/drug interactions with MAOIs
- Sympathomimetic drug use (e.g., cocaine, amphetamines)
- Rebound HTN after sudden cessation of antihypertensive Rx (clonidine)

Obstetric
- Severe pre-eclampsia/eclampsia
- HELLP syndrome

Surgical
- Severe HTN in patients requiring immediate surgery
- Postoperative HTN
- Postoperative bleeding from vascular suture lines

Severe body burns

Severe epistaxis

- Ischemic stroke: 24%
- Pulmonary edema: 22% 3.
- Hypertensive encephalopathy: 16%
- Acute heart failure: 14%
- Acute myocardial infarction: 12%
- Intracerebral hemorrhage: 5%
- Eclampsia (SBP > 170, DBP > 110 mm Hg): 5%
- Dissecting aortic aneurysm: 2%
- Subarachnoid hemorrhage
- Acute renal failure

Pathogenesis

Causes of Blood Pressure Spikes

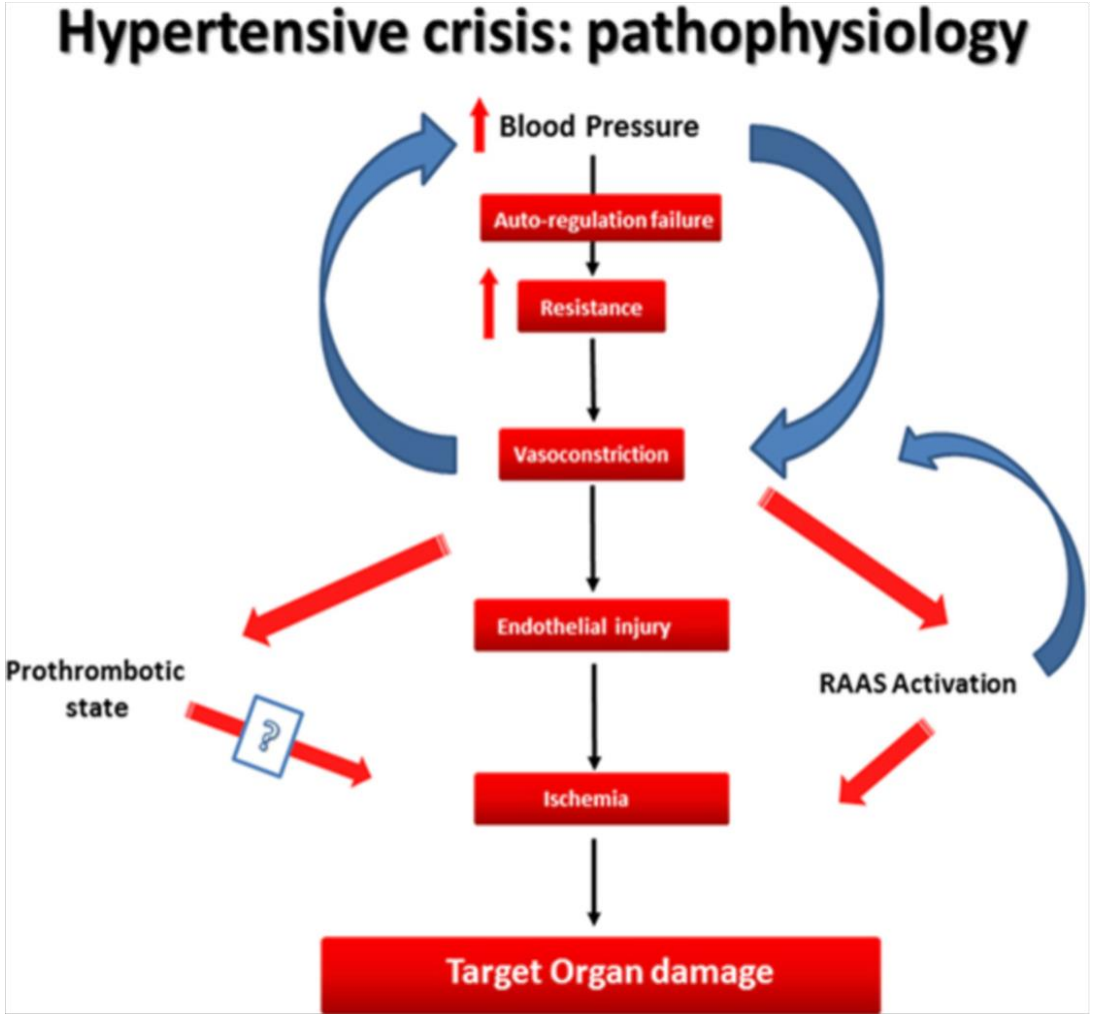
- Most common is essential HTN (90%) – under-treatment, non-compliance, and anti-hypertensive withdrawal (especially clonidine). Most common in older patients.

- Less common cause is secondary to underlying pathological condition - renovascular HTN, renal parenchymal disease, collagen vascular disease, drug use/withdrawal, tyramine crisis, preeclampsia/eclampsia, pheochromocytoma, acute glomerulonephritis, head injury, cerebrovascular disease, renin or aldosterone secreting tumor, vasculitis. Most common in young Caucasian patients.

Pathophysiology

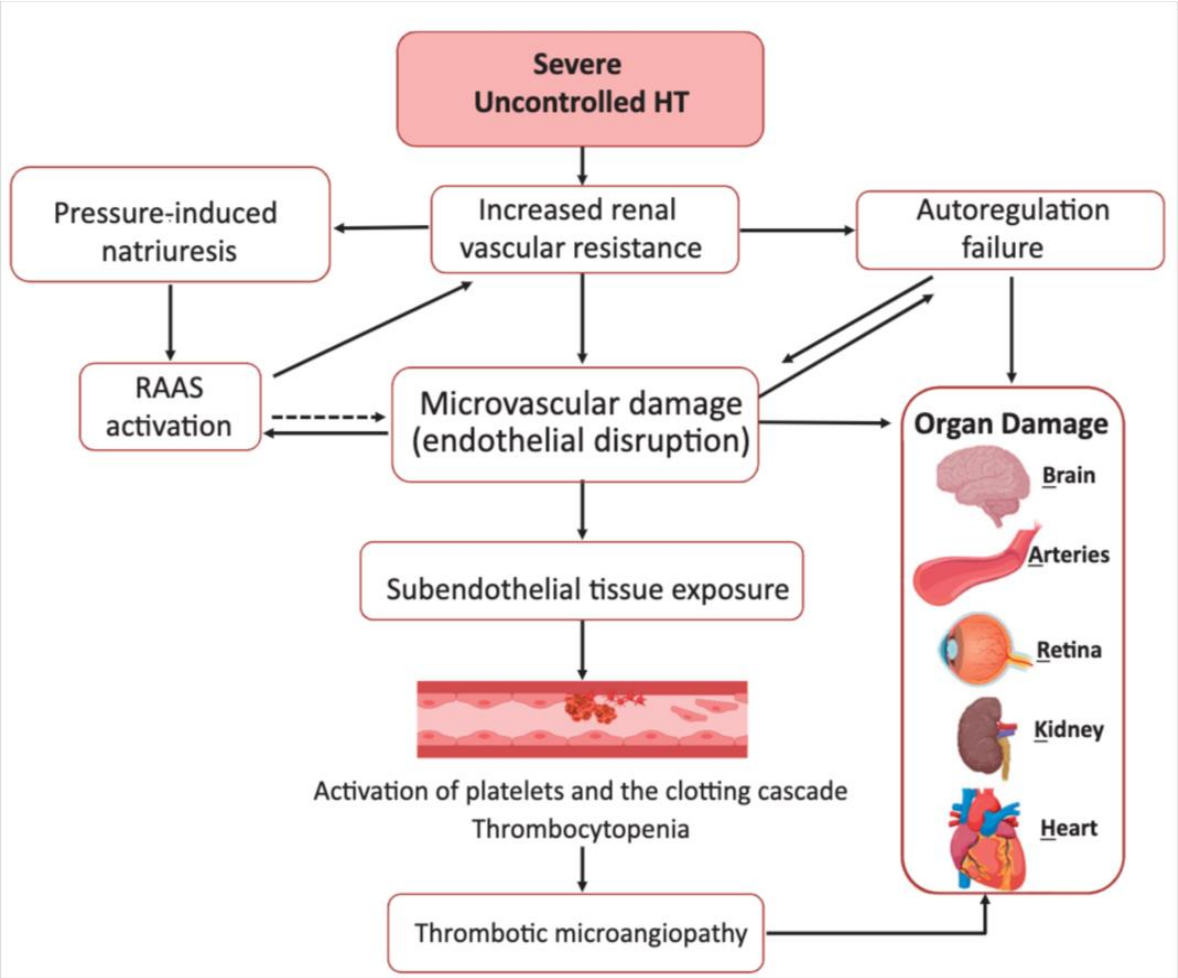
The pathophysiology of hypertensive emergency is not fully understood but following abnormalities are involved in the development of hypertensive emergency:

1. Generalized vasoconstriction
2. Cerebral edema
3. Spasms of cerebral vessels
4. Activation of Renin-Angiotensin-System
5. Activation of Sympathico-Adrenal-System



Mechanisms that lead to loss of autoregulation and injury to microcirculation in severe uncontrolled HTN

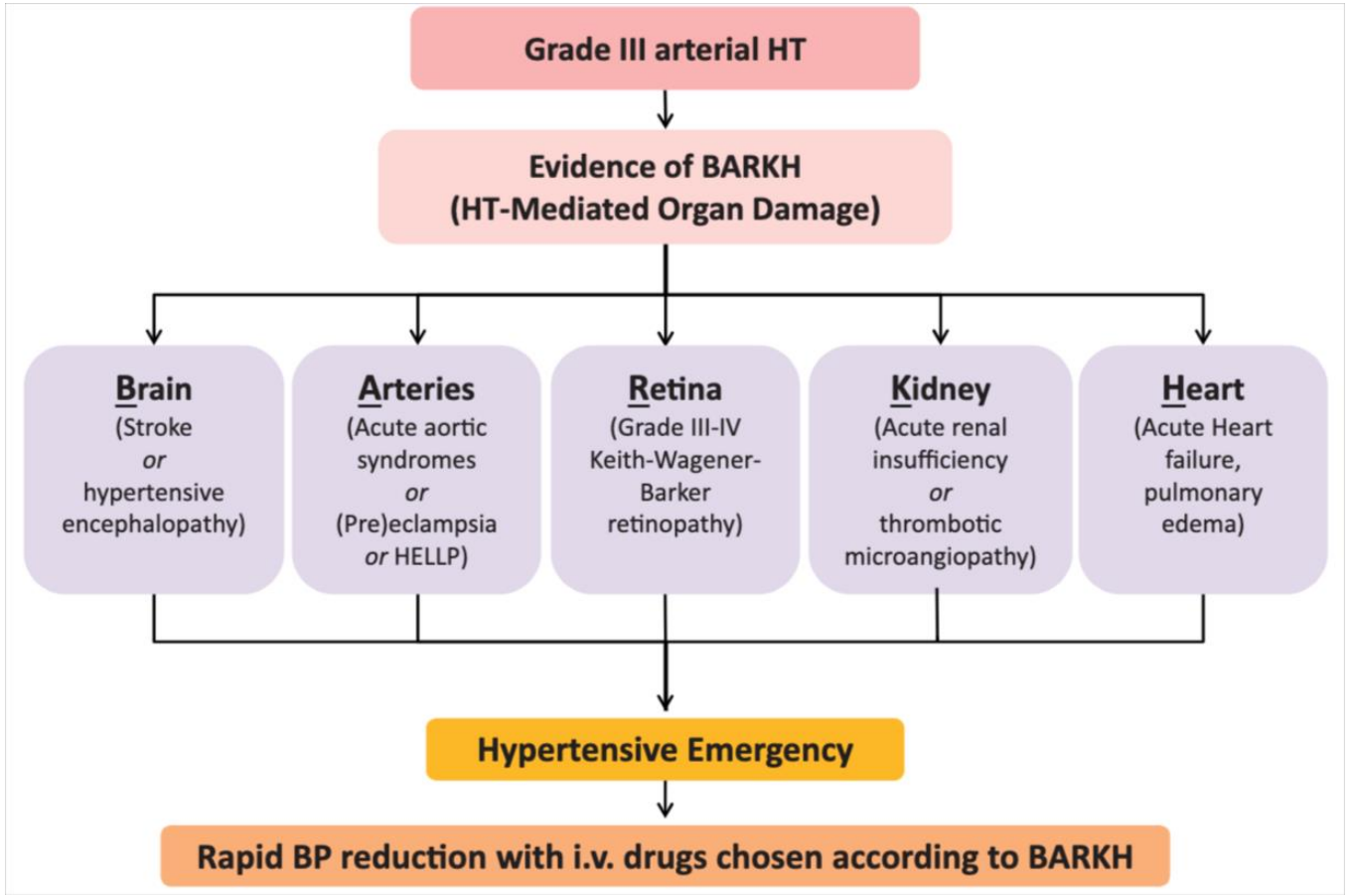
The speed and severity of BP elevation are the main factors driving the onset of a Hypertensive Emergency.



A sudden increase in the vascular resistance induces natriuresis that activates the renin–angiotensin-aldosterone system (RAAS), thus causing oxidative stress, increasing BP and augmenting the microvascular damage (endothelial dysfunction and damage).
Disruption of the endothelial lining, causing exposure of the sub-endothelial tissues to blood, triggers platelet aggregation and the clotting cascade leading to thrombotic microangiopathy.
(Modified from van den Bornet al).

Hypertensive emergencies (HE's) and associated organ damage

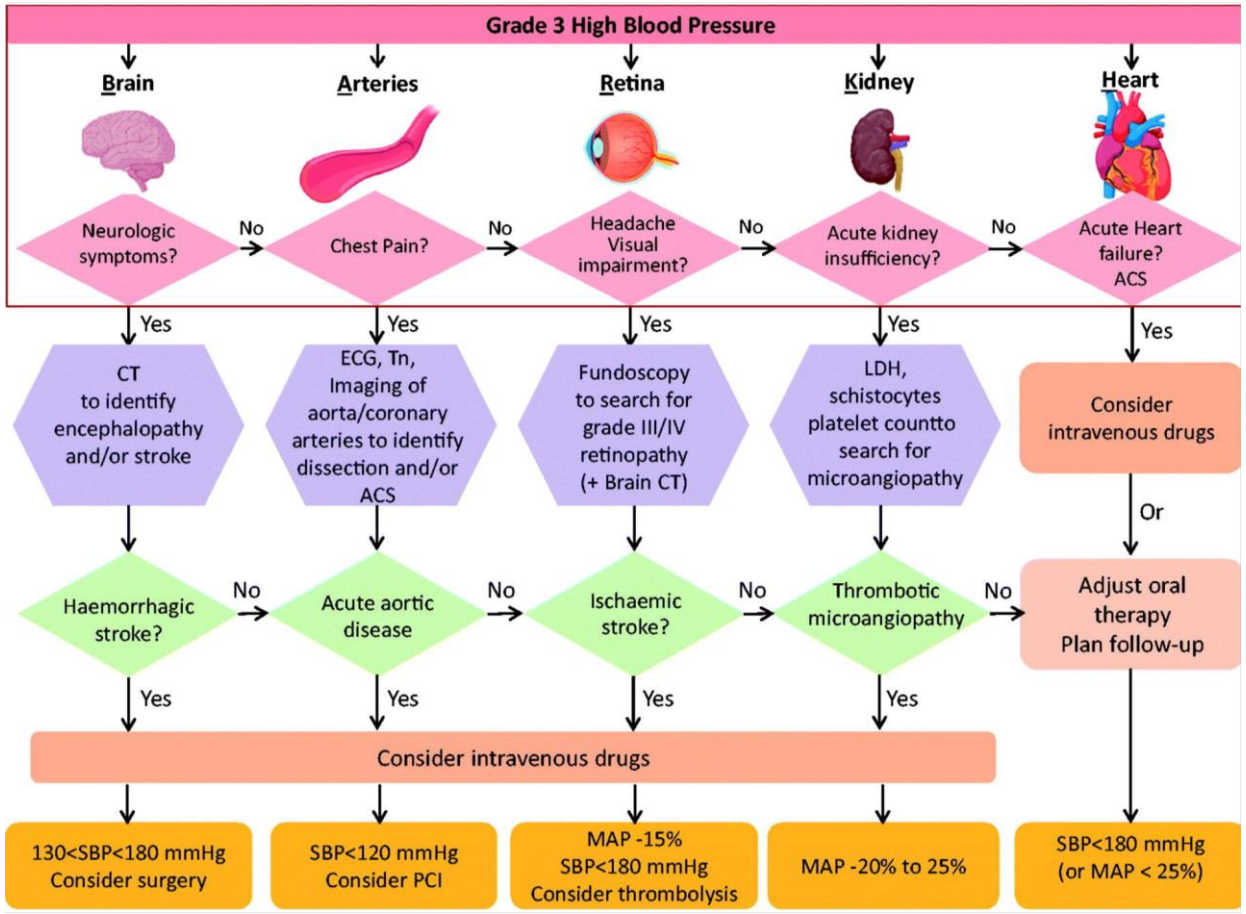
Simplified brain, arteries, retina, kidney and/or heart (BARKH)-based algorithm for a quick identification of the hypertensive emergencies (HEs) and the associated acute organ damage.



If BARKH involvement is detected, the reduction of BP values should be undertaken with i.v. treatment; in any other case, an oral treatment is recommended. BP: blood pressure; HT: hypertension; HELLP: haemolysis elevated liver enzymes low platelets.

Blood pressure 2021, vol. 30; No4: 208-19

Management of hypertensive emergencies (end-organ damage): a practical approach

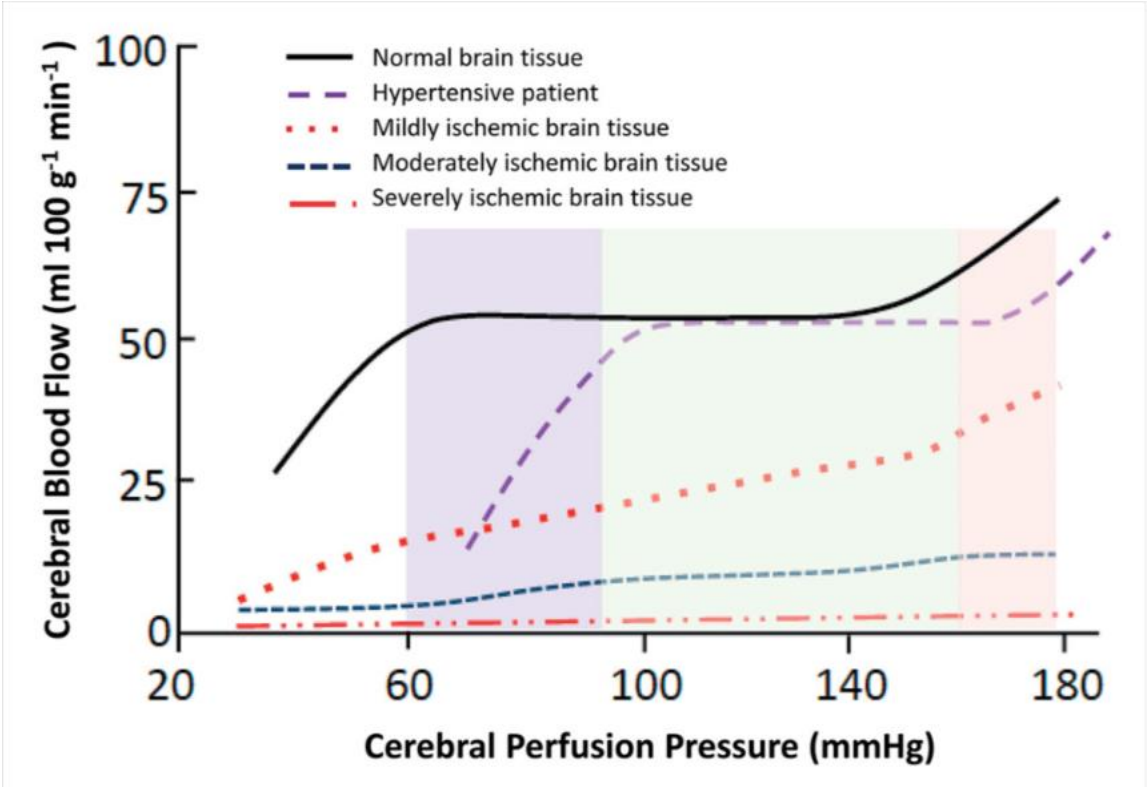


The BARKH approach to identification and treatment of HEs, a simplified symptom-based hierarchical algorithm to assist physicians in the rapid evaluation of patients presenting with suspected HEs.

Management of hypertensive emergencies: a practical approach, Blood Pressure, 30:4, 208-219

Cerebral autoregulation of blood flow in normotensive subjects(continuous line) and in hypertensive patients (dotted lines)with and without ischaemic brain damage.

Cerebral blood flow is physiologically maintained at a constant level with mean arterial pressure between 70 and 90 mmHg, below which it dramatically drops. In hypertensive patients (violet dotted line) the autoregulation range of BP is shifted to right towards higher values, between 110 and 150 mmHg, and is narrowed (central shaded green area). At lower and higher cerebral perfusion pressure levels, a fall or an abrupt pressure rise can induce ischaemia (left shaded violet area)or oedema(right shaded pink area). After an ischaemic injury, blood flow blunts proportionally to the injury severity(red and blue dotted lines).



Modified from Blumenfeld and Laragh

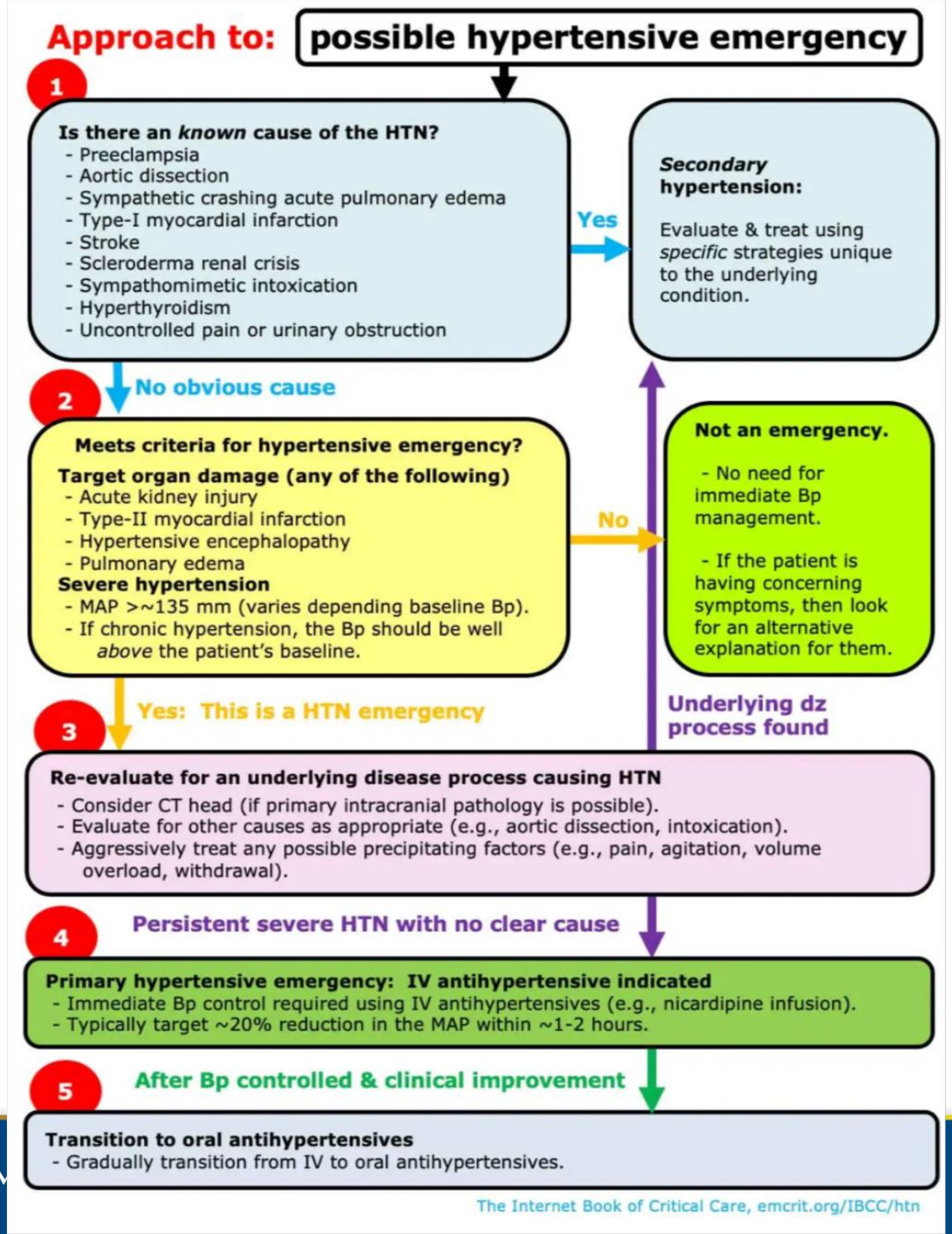
Medical Treatment

The medical treatment of hypertensive emergencies depends on the clinical presentation - No treatment should be started without clinical evaluation.

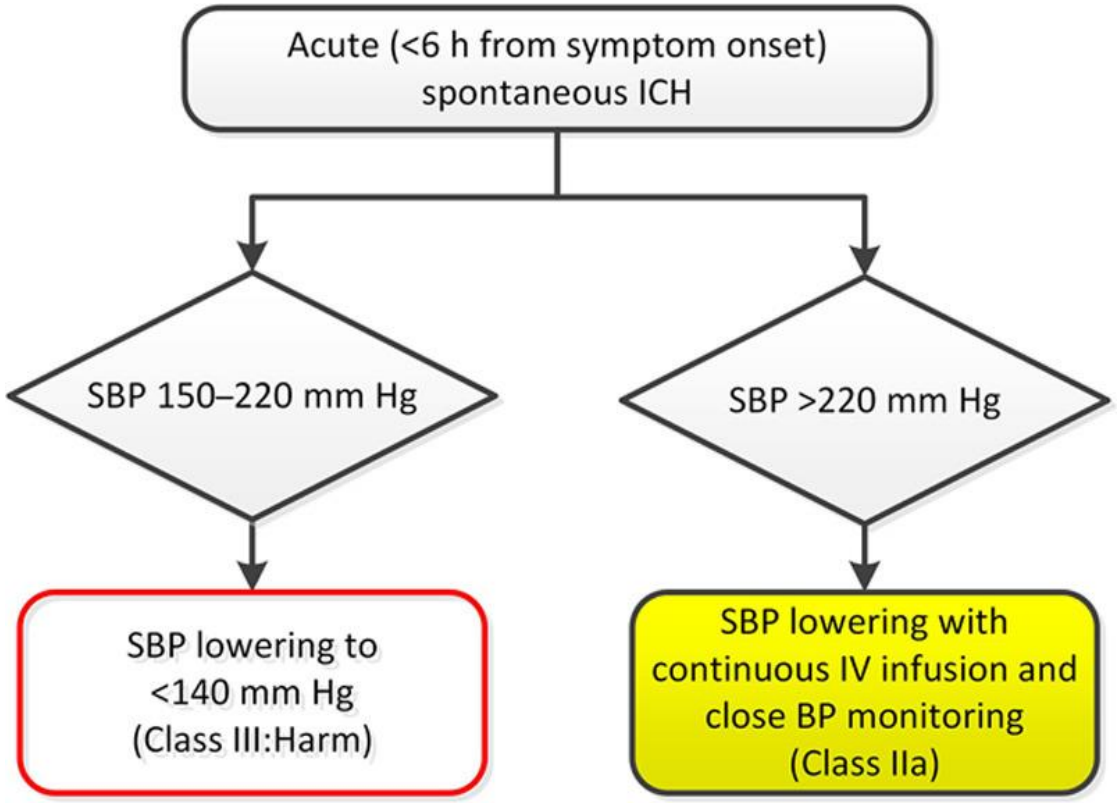
However, in selected cases immediate treatment is needed before diagnostic tests are completed or before laboratory results are available!

The clinical presentation and rapidity of BP rises are more relevant than the actual level of BP treat the patient and not the number!

Clinical evaluation should be performed without delay.

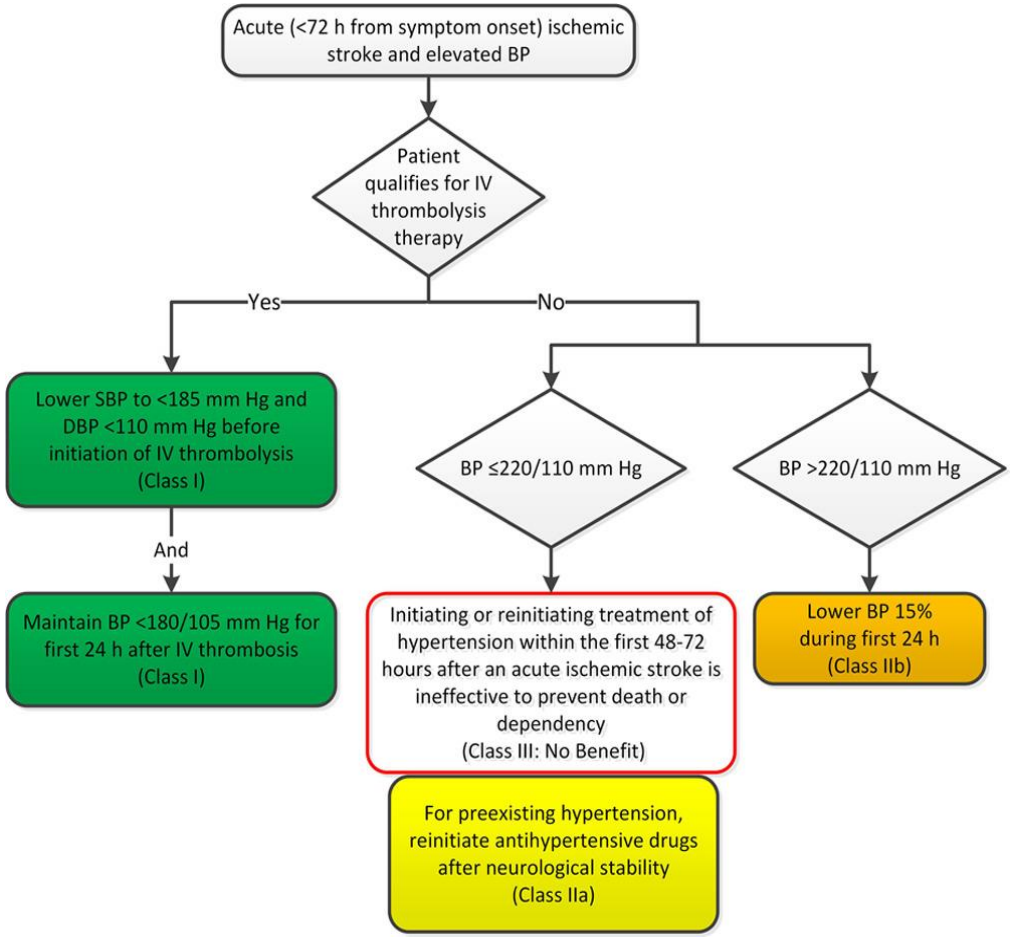

Approach to: possible hypertensive emergency
1
Is there an known cause of the HTN?
- Preeclampsia
- Aortic dissection
- Sympathetic crashing acute pulmonary edema
- Type-I myocardial infarction
- Stroke
- Scleroderma renal crisis
- Sympathomimetic intoxication
- Hyperthyroidism
- Uncontrolled pain or urinary obstruction
Yes
Secondary hypertension:
Evaluate & treat using specific strategies unique to the underlying condition.
No obvious cause
2
Meets criteria for hypertensive emergency?
Target organ damage (any of the following)
- Acute kidney injury
- Type-II myocardial infarction
- Hypertensive encephalopathy
- Pulmonary edema
Severe hypertension
- MAP >~135 mm (varies depending baseline Bp).
- If chronic hypertension, the Bp should be well above the patient's baseline.
No
Not an emergency.
- No need for immediate Bp management.
- If the patient is having concerning symptoms, then look for an alternative explanation for them.
Yes: This is a HTN emergency
3
Underlying dz process found
Re-evaluate for an underlying disease process causing HTN
- Consider CT head (if primary intracranial pathology is possible).
- Evaluate for other causes as appropriate (e.g., aortic dissection, intoxication).
- Aggressively treat any possible precipitating factors (e.g., pain, agitation, volume overload, withdrawal).
Persistent severe HTN with no clear cause
4
Primary hypertensive emergency: IV antihypertensive indicated
- Immediate Bp control required using IV antihypertensives (e.g., nicardipine infusion).
- Typically target ~20% reduction in the MAP within ~1-2 hours.
After Bp controlled & clinical improvement
5
Transition to oral antihypertensives
- Gradually transition from IV to oral antihypertensives.
The Internet Book of Critical Care, emcrit.org/IBCC/htn

Management of hypertension in patients with acute ICH



A Report of the American College of Cardiology/American Heart Association Task Force on Clinical Practice Guidelines, Volume: 71, Issue: 6, Pages: e13-e115. Paul K. Whelton. Hypertension. 2017

Management of hypertension in patients with acute ischemic stroke



Paul K. Whelton. Hypertension. 2017 Task Force on Clinical Practice Guidelines, Volume: 71, Issue: 6, Pages: e13-e115.

Treatment of hypertensive emergencies

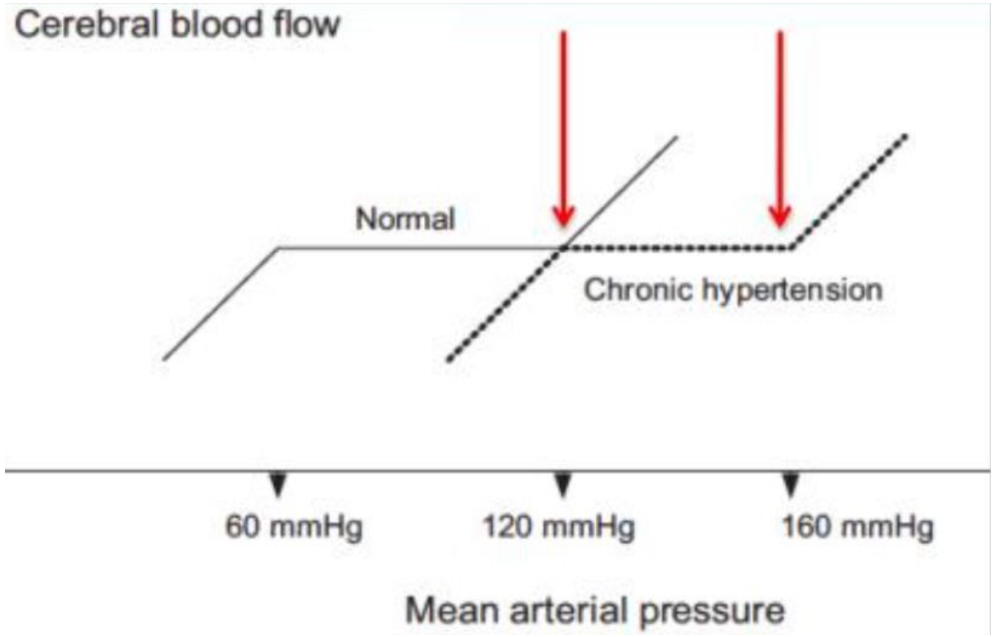
General guidelines: - immediate therapy: prompt lowering of elevated BP within 30 min to avoid and limit the risk of serious complications (cerebral hypoperfusion, perivascular edema, increased ICP)

a. Controlled lowering of elevated BP to a safer noncritical level but not to normal range (reduction of mean arterial pressure by 25% in the first hour, aim SBP 160-170, aim DBP 100-110 mm Hg) by 2-6 hours to avoid sudden drop of BP and reduction of cerebral perfusion and development of brain infarction.
   Subsequent gradual normalization in 24-48 hours.

b. Close observation and in-patient treatment is required.

How low should you go?

- 25% reduction in MAP within 1st hour
- Target ~ 160/100 mmHg by 2-6 hours



Better answer:

- It really depends on clinical condition
  - Less aggressive with ischemic stroke
  - More aggressive with hemorrhagic stroke, acute pulmonary edema, and aortic dissection

Marik and Varon; Critical care 2003; 7: 374-84

First line and alternative treatments for hypertensive emergencies

Condition	Time line and target BP	1st line treatment	Alternative
TMA or acute renal failure	Several hours, MAP −20% to −25%	Labetalol Nicardipine	Nitroprusside Urapidil
Hypertensive encephalopathy	Immediate, MAP −20% to −25%	Labetalol Nicardipine	Nitroprusside
Acute ischaemic stroke and systolic BP >220mmHg or diastolic BP >120mmHg	1 h, MAP −15%	Labetalol Nicardipine	Nitroprusside
Acute ischaemic stroke with indication for thrombolytic therapy and systolic BP >185mmHg or diastolic BP >110mmHg	1 h, MAP −15%	Labetalol Nicardipine	Nitroprusside
Acute haemorrhagic stroke and systolic BP >180mmHg	Immediate, systolic 130 < BP <180 mmHg	Labetalol Nicardipine	Urapidil
Acute coronary event	Immediate, systolic BP <140 mmHg	Nitroglycerine Labetalol	Urapidil
Acute cardiogenic pulmonary oedema	Immediate, systolic BP <140 mmHg	Nitroprusside or Nitroglycerine (with loop diuretic)	Urapidil (with loop diuretic)
Acute aortic disease	Immediate, systolic BP <120 mmHg and heart rate <60 b.p.m.	Esmolol and Nitroprusside or Nitroglycerine or Nicardipine	Labetalol or Metoprolol
Eclampsia and severe pre-eclampsia/HELLP	Immediate, systolic BP <160 mmHg and diastolic BP <105 mmHg	Labetalol or Nicardipine and Magnesium sulphate	

BP: blood pressure; HELLP: haemolysis, elevated liver enzymes and low platelets; TMA: thrombotic microangiopathy. Modified from van den Born et al. [1].

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Medications

Agent	Dosing range	Onset	Duration
Vasodilators			
Hydralazine	IV Bolus: 10-20 mg IM : 10-40 mg q30 min PRN	IV: 10 mins IM: 20 mins	IV: 1-4 hours IM: 2-6 hours
Nitroglycerine	IV 5-200 mcg/min Titrate by 5-25 mcg/min q5-10 mins	2-5 mins	5-10 mins
Sodium nitroprusside	IV 0.25-10 mcg/kg/min Titrate by 0.1-0.2 mcg/min q5min	seconds	1-2 mins
CCB			
Clevidipine	IV 1-6 mg/hr Titrate by 1-2 mg/hr q90s; max 32 mg/hr	1-4 min	5-15 min
Nicardipine	IV 5-15 mg/hr Titrate by 2.5 mg/hr q5-10 min	5-10 min	2-6 hours

Agent	Dosing range	Onset	Duration
Beta Blockers			
Esmolol	IV 25-300 mcg/kg/min Titrate by 25 mcg/kg/min q3-5 mins	1-2 mins	10-20 mins
Labetalol	IV bolus: 20 mg; may repeat doses of 20-80 mg q5-10 mins PRN IV 0.5-10 mg/min Titrate by 1-2 mg/min q2hr.	2-5 min; peak 5-15 mins	2-6 hr Upto 18 hr
Metoprolol	IV bolus: 5-15 mg q5-15 min PRN	5-20 min	2-6 hr
ACEI			
Enalaprilat	IV bolus: 1.25 mg q6hr Titrate no more than q12-24 hr: Max dose : 5 mg q6hr	15-30 min	12-24 hr
a-Antagonist			
Phentolamine	IV bolus: 1-5 mg PRN; Max 15 mg	Seconds	15 min

Table 1. Drugs that can be used intravenously for treatment of hypertensive emergencies (HEs).

Drug	Onset of action	Duration of action	Dose	EMA/FDA Approval	Contraindications	Reflex tachycardia	Adverse effects
Labetalol	5–10 min	3–6 h	0.25–1.0 mg/kg; 2–4 mg/min until goal BP is reached, thereafter 5–20 mg/h	EMA & FDA: severe hypertension	3rd-degree AV block, decompensated heart failure, asthma, bradycardia Known sensitivity	No	Broncho-constriction, foetal bradycardia
Urapidil	3–5 min	4–6 h	12.5–25 mg as bolus injection; 5–40 mg/h as continuous infusion	EMA: HEs/severe hypertension FDA: HEs not mentioned	Liver/kidney failure (relative) Aortic coarctation, A-V shunts (fistulas) Known sensitivity	Yes	Headache
Nitroprusside	Immediate	1–2 min	0.3–0.5 mcg/kg/min, increase by 0.5 mcg/kg/min every 5 min	EMA: HEs FDA: immediate reduction of BP in hypertensive HEs	Erectile dysfunction medications within the past 24 h. Increased intracranial pressure Liver/kidney failure (relative) Known sensitivity	Yes	Cyanide intoxication
Nitroglycerine	Immediate	3–5 min	5–200 mcg/min, 5 mcg/min increase every 5 min	EMA: hypertensive crisis FDA: treatment of peri-operative hypertension and induction of intraoperative hypotension.	Erectile dysfunction medications within the past 24 h Increased intracranial pressure Known sensitivity	Yes	Headache, reflex tachycardia
Fenoldopam	5–15 min	30–60 min	0.1 mcg/kg/min, increase every 15 min (0.05–0.1 mcg/kg/min increments) until goal BP is reached	EMA: HEs FDA: Short-term (up to 48 h) management of HEs.	Caution in glaucoma Known sensitivity	Yes	Hypokalaemia
Clevidipine	2–3 min	5–15 min	2 mg/h, increase every 2 min with 2 mg/h until BP goal	EMA: quick BP reduction during the perioperative period FDA: Reduction of BP when oral therapy is not feasible or desiderable	Known sensitivity	Yes	Headache
Nicardipine	5–15 min	30–40 min	5–15 mg/h as continuous infusion, starting dose 5 mg/h, increase every 5–15 min with 2.5 mg until goal BP, thereafter decrease to 3 mg/h	EMA: HEs and control of high BP after surgery. Use of i.v. nicardipine for other indications is not recommended. FDA: short-term treatment of hypertension when oral therapy is not feasible	Liver failure Known sensitivity	Yes	Headache
Phentolamine	1–2 min	10–30 min	1–5 mg bolus injections OR 1–40 mg/h as continuous infusion	EMA: HEs not mentioned FDA: prevention or control of episodes that occur in pheochromocytoma as a result of stress or manipulation during preoperative preparation and excision.	Evidence of coronary artery disease Known sensitivity	No	Bradycardia, myocardial infarction

BP: blood pressure; EMA: European Medicines Agency; FDA: Food Drug Administration. Modified from van den Born et al. [1].

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Post discharge evaluation for secondary hypertension

- Timely follow up post discharge to adjust medications.
- History, physical exam, basic labs
- Workup for potentially reversible causes or secondary causes
- 24-hour urine collection should be obtained on the patient's usual diet for determination of sodium excretion, creatinine clearance, and aldosterone excretion
- If primary aldosteronism is suspected (HTN with hypokalemia and metabolic alkalosis) – paired morning measurement of the plasma aldosterone concentration (PAC) and plasma renin activity (PRA) to determine whether the patient has an elevated or high-normal PAC, suppressed PRA, and elevated PAC/PRA ratio
- Most patient should undergo non-invasive evaluation for renal artery stenosis (RAS). Clinical clues to renovascular disease:
  - HTN before 30 yo (especially w/o FHx) or recent onset of significant HTN after 55 yo
  - Abdominal bruit, particularly if it continues into diastole and is lateralized
  - Recurrent (flash) pulmonary edema
  - Renal failure of uncertain etiology, especially with normal urinary sediment
  - Coexisting diffuse atherosclerotic vascular disease, especially in heavy smokers
  - Acute renal failure precipitated by anti-HTN therapy, especially ACEi/ARB
  - Noninvasive screening tests: captopril-enhanced radionuclide renal scan, duplex doppler flow studies, or MRA
  - Definitive Dx is with renal angiogram (15)
- If pheochromocytoma is suspected (episodic HTN, HA, sweating, tachycardia) – 24-hour urine fractionated metanephrines and catecholamines
- If Cushing's syndrome is suspected (weight gain, plethora, round face, hirsutism, dorsal fat pad) – late-night salivary cortisol (two measurements), 24-hour urinary free cortisol excretion (two measurements), or the overnight dexamethasone suppression test

Factors to consider at outpatient follow up of hospitalization for hypertensive crisis

Agents that can interfere with blood pressure control

Non-narcotic analgesics (NSAIDs, selective COX-2 inhibitors, ASA)
Sympathomimetic agents (decongestants, diet pills)
Stimulants (methylphenidate, dexmethylphenidate, dextroamphetamine, amphetamine, modafinil)
Illicit drugs (cocaine, methamphetamine)
EtOH
Oral contraceptives
Glucocorticoids, mineralocorticoids
Cyclosporine, tacrolimus
Atypical antipsychotics (clozapine, olanzapine)
EPO
VEGF inhibitors
Natural licorice
Herbal compounds (ephedra or ma huang)

Consider Secondary causes of resistant HTN

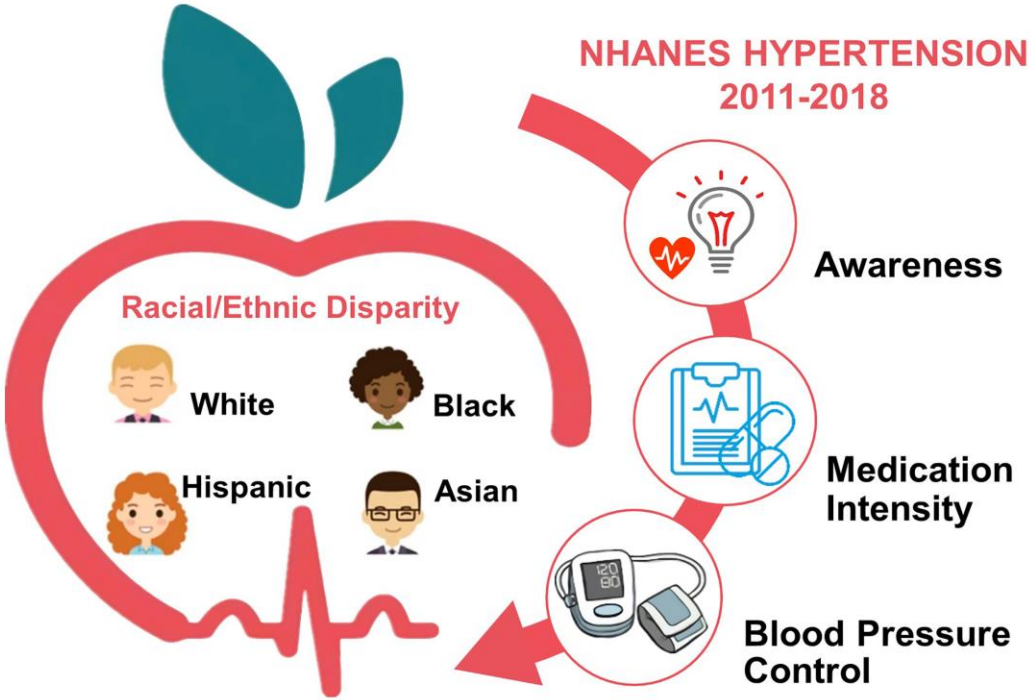
Common

- OSA
- Renal parenchymal disease
- Primary aldosteronism
- Renal artery stenosis
- Medications

Uncommon

Pheochromocytoma
Cushing's disease
Hyperparathyroidism
Aortic coarctation
Intracranial tumor

Trends in Racial Disparities in Hypertension



Main findings

- Despite receiving more antihypertensive medications, Black people have poorer hypertension control compared with White people.
- The poorer hypertension control among Asian and Hispanic people is associated with their lower hypertension awareness and treatment compared with White people

There are racial disparities in awareness, diagnosis and treatment of HTN, which have to be improved.

Yuan Lu. Hypertension. National Trends in Racial and Ethnic Disparities in Antihypertensive Medication Use and Blood Pressure Control Among Adults With Hypertension, 2011–2018, Volume: 79, Issue: 1, Pages: 207-217

Conclusions

- HTN remains among the most important public health issues, leading to markedly increased risk of cardiovascular, renal, and neurological disease
- Though we have an ever-expanding list of pharmaceuticals at our disposal for the treatment of high BP, HTN remains underdiagnosed and undertreated
- Hypertensive crises - most commonly result of exacerbation of essential HTN
- To decrease the incidence, morbidity, and cost of these events, the importance of adequately identifying and managing essential HTN by PCPs cannot be over emphasized.
- True hypertensive emergencies are just that: emergencies. General agreement exists in their management.
- In contrast, hypertensive urgencies do not represent such an imminent risk and their management remains quite varied
  - It is possible that hypertensive urgencies tend to be treated with too much enthusiasm and an unnecessary sense of impending crisis
    - This results in long ED visits and costly hospital admissions for a problem that could likely be managed in an outpatient setting (or even prevented in the first place)
  - It would be valuable to examine the course, treatment, and outcomes of hypertensive urgencies to reach treatment consensus on the "big numbers" of hypertensive urgencies should be treated in this era of evidence-based medicine