HYPERTENSIVE URGENCIES AND EMERGENCIES

DEFINITIONS

- Systolic blood pressure >220 and diastolic >120mmHg.
- Patients with hypertension can be classified into 2 categories based upon their symptoms and the organ systems that are affected at the time of presentation:
- -**HYPERTENSIVE URGENCY**: the BP is a potential risk but has not yet caused acute end-organ damage. These patients require BP control over several days to weeks.

  -**HYPERTENSIVE EMERGENCY (AKA ACCELERATED OR MALIGNANT HYPERTENSION)**: is severe hypertension with acute impairment of an organ system (e.g., central nervous system [CNS], cardiovascular, renal). In these conditions, the blood pressure (BP) should be lowered aggressively over minutes to hours.Presence of papilledema indicates **HYPERTENSIVE ENCEPHALOPATHY** is accelerated or malignant hypertension with CNS signs or symptoms

ETIOLOGY

- **Essential hypertension** : Inadequate blood pressure control and noncompliance are common precipitants
- Renovascular
- Eclampsia/pre-eclampsia
- Acute glomerulonephritis
- Pheochromocytoma
- Anti-hypertensive withdrawal syndromes
- Head injuries and CNS trauma
- Renin-secreting tumors
- Drug-induced hypertension
- Burns
- Vasculitis
- TTP
- Idiopathic hypertension
- Post-op hypertension
- Coarctation of aorta

Take home message

- The commonest cause of hypertensive emergency in 2011 is undiagnosed, untreated, or undertreated essential hypertension

PATHOPHYSIOLOGY

NORMAL AUTOREGULATION

RISE IN BP

↓

ARTERIAL AND ARTERIOLAR CONSTRICTION

↓

Normal flow.(flow=P/r)

AUTOREGULATION FAILURE

RISE IN BP

↓

FAILURE OF VASOCONSTRICTION

↓

ENDOTHELIAL DAMAGE

(due to shear stress on the wall)

PATHOPHYSIOLOGY

- BP=PVR*CO(SV*HR)
- Rate at which MAP rises more important than absolute rise.

Acute rise in BP → Failure of vasoconstriction by autoregulation → Endothelial damage

↓

Depsn. of proteins/ fibrinogen in vessel wall → Activates coagn and inflammation → FIBRINOID NECROSIS

- RAAS plays an important role in initiating and perpetuating BP rise by causing vasoconstriction and fluid retention.

CENTRAL NERVOUS SYSTEM

- CENTRAL NERVOUS SYSTEM: The CNS is affected as the elevated BP overwhelms the normal cerebral autoregulation. Under normal circumstances, with an increase in BP, cerebral arterioles vasoconstrict and cerebral blood flow (CBF) remains constant. During a hypertensive emergency, the elevated BP overwhelms arteriolar control over vasoconstriction and autoregulation of CBF. This results in transudate leak across capillaries and continued arteriolar damage. Subsequent fibrinoid necrosis causes normal autoregulatory mechanisms to fail, leading to clinically apparent papilledema, the sine qua non of malignant hypertension. The end result of loss of autoregulation is hypertensive encephalopathy.

CARDIOVASCULAR SYSTEM

- The cardiovascular system is affected as increased cardiac workload leads to cardiac failure; this is accompanied by pulmonary edema, myocardial ischemia, or myocardial infarction.

RENAL SYSTEM

- The renal system is impaired when high BP leads to arteriosclerosis, fibrinoid necrosis, and an overall impairment of renal protective autoregulation mechanisms. This may manifest as worsening renal function, hematuria, red blood cell (RBC) cast formation, and/or proteinuria.

EPIDEMIOLOGY

- **In the US:** More than 60 million Americans, about 25-30% of the population, have hypertension. Of these individuals, 70% have mild disease, 20% moderate, and 10% severe hypertension (diastolic BP [DBP] >110 mm Hg). Approximately 1-2% develop a hypertensive emergency with end-organ damage.
- **Mortality/Morbidity:** Morbidity and mortality depend on the extent of end-organ damage on presentation and the degree to which BP is controlled subsequently. BP control may prevent progression to end-organ impairment. I yr mortality in untreated pts. >90%.5 yr survival of all presentations is 74%.
- **Race:** African Americans have a higher incidence of hypertensive emergencies than Caucasians.
- **Sex:** Males are at greater risk of hypertensive emergencies than females.
- **Age:**Most commonly in middle-aged people.Peak age:40-50yrs.

HISTORY

- **Focus on circumstances surrounding hypertension & etiology :**

  -Medications:esp. hypertensive drugs/their compliance,illicit drugs

  -Duration of hypertension

  -Duration of current symptoms

  -Date of LMP

  -Other medical problems:prior hypertension,thyrotoxicosis,Cushing's,SLE,renal

- **Focus on complications :**

  -CNS:headaches,blurred vision,wt. loss,nausea,vomiting,weakness,fatigue, confusion and mental status changes.

  -CVS:symptoms of CHF,angina,dissection,SOB

  -Renal:hematuria,oliguria.

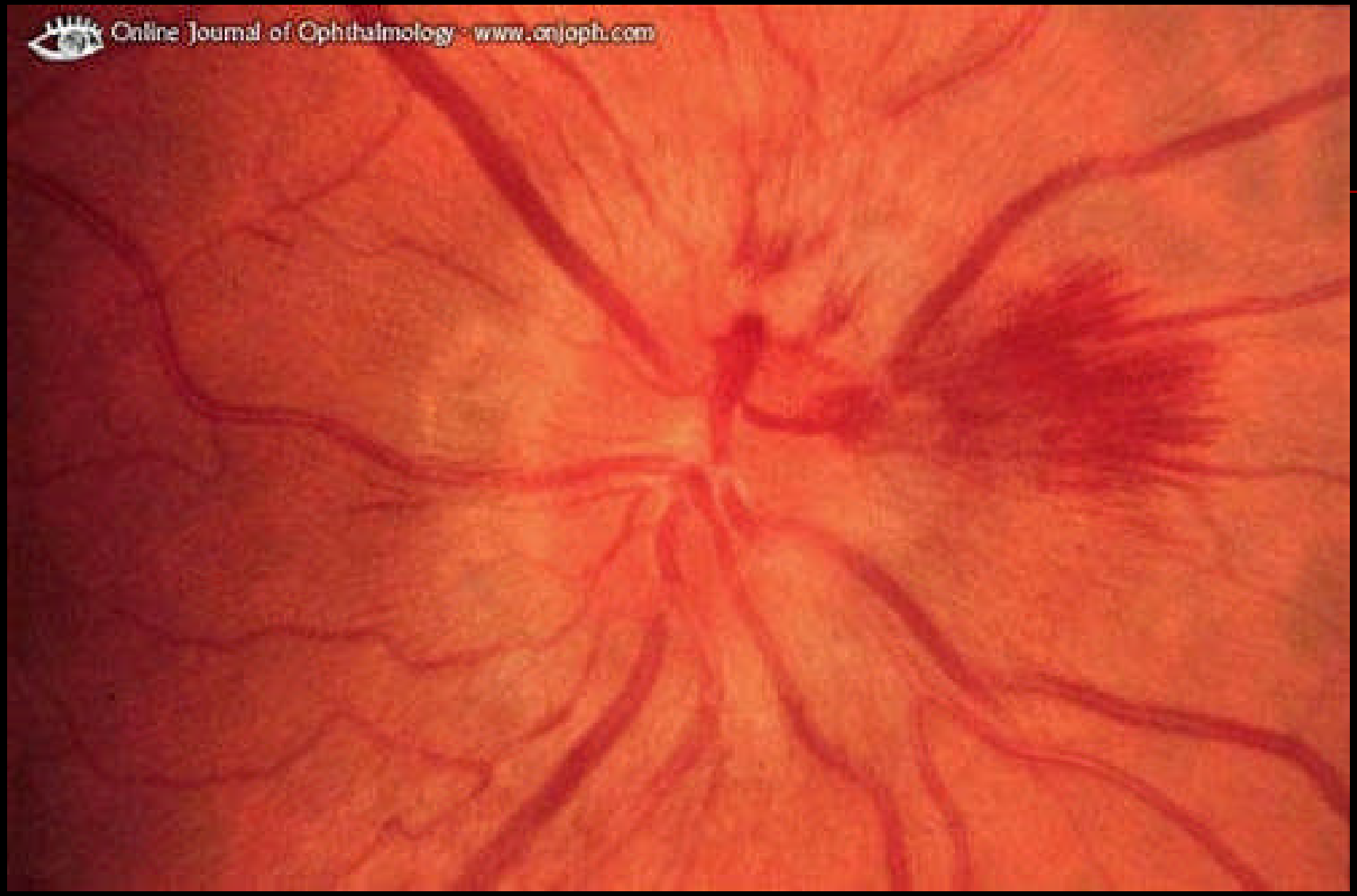
PHYSICAL

- Use an approach based on organ systems to identify signs of end-organ damage

  -CNS: focal neuro deficits,seizures,stupor,coma, papilledema, hemorrhages, exudates, or evidence of closed-angle glaucoma

  -CVS:JVD,lung auscultaion for crackles,peripheral edema,extra heart sounds, equal and symmetric BP and pulses bilaterally.

  -Check for abdominal masses and bruits.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DIFFERENTIALS

Acute Coronary Syndrome
Aortic dissection
CHF,pulmonary edema
Acute Coronary Syndrome
Aneurysm, Abdominal
Anxiety
Congestive Heart Failure and Pulmonary Edema
Cushing Syndrome
Delirium Tremens
Encephalitis
Glomerulonephritis, Acute
Headache, Cluster
Headache, Migraine
Headache, Tension
Hyperthyroidism, Thyroid Storm, and Graves Disease
Myocardial Infarction
Pregnancy, Eclampsia
Pregnancy, Preeclampsia
Stroke, Hemorrhagic
Stroke, Ischemic
Subarachnoid Hemorrhage
Systemic Lupus Erythematosus

Differential(contd.)

- Others:

-Steroid use
-Use of over-the-counter or recreational sympathomimetic drugs
-Pheochromocytoma
-Acute vasculitis
-Serotonin syndrome
-Other CNS pathology
-Coarctation of the aorta

Work-up

- CBC,Chem 8
- Urinanalysis:hematuria,proteinuria,RBCs,RBC casts.
- Toxicology,pregnancy,endocrine causes.
- Imaging:Chest X-ray,Head CT,Chest CT,aortic angiogram
- EKG,cardiac enzymes

TREATMENT

- Weigh benefits of decreasing BP against risks of decreasing end-organ perfusion. Important steps include:

  -Appropriately evaluating patients with an elevated BP

  -Correctly classifying the hypertension

  -Determining aggressiveness of therapy

  An important point to remember in the management of the patient with any degree of BP elevation is to "treat the patient and not the number."

Treatment

- Initial considerations: Place patient who is not in distress in a quiet room and reevaluate after an initial interview. In one study, 27% of patients with an initial DBP >130 mm Hg had their DBP fall below critical levels after relaxation without specific treatment.
- Consider the context of the elevated BP (eg, severe pain)
- Screen for end-organ damage- Patients with end-organ damage usually require admission and rapid lowering of BP using iv meds.Suggested meds depend on the end-organ system damaged.
- Patients without evidence of end-organ effects may be discharged with follow–up.It is a misconception that a patient should not be discharged from the ER with elevated BP.Giving oral meds such as nifedipine to rapidly lower BP may be dangerous as the BP may have been elevated for sometime and there may be organ hypoperfusion.Acute control has not improved long term mortality and morbidity rates.

INITIAL STEPS

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DRUGS

- Once the diagnosis of hypertension is made and end-organ damage confirmed,the BP should be lowered by about 25% of the mean arterial pressure.
- There are 2 main classes of drugs:

  -Vasodilators

  -Adrenergic inhibitors

VASODILATORS

DRUG	DOSAGE	ONSET/DUR	ADV.EFFE
Nitroprusside	0.25-10mcg/kg/min	Instant/1-2min.	Thiocyanate,cyanide poisoning
Nitroglycerine	5-100mcg/min	1-5min/3-5min	Flushing,headache,methemoglobin
Nicardipine	5-15mg/hr	5-10min/1-4hr	Tachycardia,flushing.avoid-heart failure
Hydralazine	10-20mg	5-15min/3-8hr	Flushing,tachy,avoid-A.diss,MI
Enalapril	10-40mg IM,1.25-5MG1Vq6hr	20-30min/6hr	Hypotension,renal failure,hyperkalemia
Fenoldopam	0.1-0.3mcg/kg/min	5min/10-15min	Flushing,headache,tachy

ADRENERGIC INHIBITORS

DRUG	DOSAGE	ONSET/DUR	ADV.EFF
Labetalol (a+b blocker)	20-80mgiv bolus every 10 min,2mg.min iv infusion	5-10min/3-6hrs	Heart block,ortho hypotension.avoid-heart failure,asthma
Esmolol (b-1 selective blocker)	200-500 mcg/kg/min for 4min,then 150-300mcg/kg/min	1-2min/10-20min	Hypotension,avoid-heart failure,asthma
Phentolamine (a1 blocker)	5-15mg iv	1-2min/3-10min	Tachycardia,flushing ,headache

ORAL DRUGS

DRUG	DOSAGE	ONSET/DURATION	ADV. EFF.
CAPTOPRIL (ACE inhibitor)	6.25-25MG q 6hrs.	15-30min/6 hrs.	Hypotension in high renin states
CLONIDINE (a2 agonist-centrally acting)	0.1-0.2 mg hrly, Upto max 0.8mg in 24hrs.	30-60min/6-12hrs.	Sedation,bradycardia,dry mouth
LABETALOL	100-200mg q 12hrs	30-120min/8-12hrs	Heart failure,heart block,bronchospasm

RAPID BP REDUCTION

- Acute myocardial ischemia:IV NTG,b-blockers,ACE inhibitors.
- CHF with pulmonary edema:iv NTG,furosemide,morphine
- Acute aortic dissection:iv nitroprusside+b-blockers or iv trimethaphan+b-blockers.
- Hypertensive encephalopathy or sub-arachnoid hemorrhage:iv nitroprusside,labetalol or nimodipine.
- MAO-tyramine interactions with acute hypertension:iv phentolamine.

SPECIFIC TREATMENT

- **Hypertensive Encephalopathy**: Goal is to reduce MAP by not>25% or DBP tp100mmHg in the first hour.Nitroprussi(widely used in past)is a powerful arteriloar dilator,so a rise in ICP may occur.**Labetalol,fenoldopam** used more now.
- **Intracerebral Hemorrhage**: CPP=MAP-ICP.As ICP rises,MAP must rise for perfusion but this raises risk of bleeding from small arteries and arterioles.A prosp. Obs. study in 1997 did not confirm these concerns but it was obscured by early use of anti-hypertensives.Cerebral autoregulation curve in chronic hypertensives may be altered,making them less likely to tolerate aggressive lowering of BP.MAP guidelines:decrease when MAP>130 or SBP>220.**Labetalol,esmolol** agents of choice.
- **SAH**: **Nimodipine** decreases vasospasm that occurs due to chemical irritation of arteries by blood.Not recommended routinely due to high incidence of hypotension.Cognitive status is a guide.Labetalol,esmolol agents of choice.
- **Acute Ischemic Stroke**: High BP can cause hemorrhagic transformation of infarct ,cerebral edema.But,if CPP is low,ischemic penumbra may occur.CPP beyond obstn is low.Distal vessels become dilated with ,loss of autoregulation.A decilne to pre-stroke values in 4 days has been documented often..A Cochrane review examining 65 RCTs with 11,500 pts. Concluded that insufficient data exists to evaluate BP lowering post-stroke.AHA guidelines:BP be reduced only if SBP>220 or DBP>120mmHg.(unless end-organ damage is due to BP).**Labetalol,nitroprusside**-agents of choice.For thrombolysis,BP<185/110.

40 year old European male
Presents to ED with 3 day history headaches and visual blurring
History Was briefly on blood pressure medication 7 years ago.
Never went for follow-up

O/E
BP 240/140
Fundoscopy – haemorrages and exudates
No other abnormal findings

ECG – LVH and strain pattern
CXR – LV+, unfolded aorta, clear lung fields
Creatinine 120 umol/l
Urine Dipstick – protein 1+

Hypertensive urgency or emergency?
Admit or discharge?
Oral or IV treatment?

GP calls you about:
70 year old European female
New to his practice
History of hypertension, on metoprolol 23.75mg daily
On routine check has BP 220/100 – rechecked by nurse after period of rest 210/95
Exam otherwise OK
Fundoscopy – silver wiring and AV nipping
ECG – electrical LVH
Urine Dipstick – trace protein

Hypertensive urgency or emergency?
Requires hospital admission?
Oral or IV treatment?

78 year old hypertensive European man admitted with left hemiparesis. CT head normal. Diagnosis ischaemic stroke

BP 200/110

How should he be managed over the next 24 hours?

Is he a candidate for stroke thrombolysis?

50 year old Maori man admitted with left hemiparesis
CT reveals right intracerebral haemorrage ~ 25cc
BP 230/130

How should he be managed over the next 24 hours?

A 55 year old man with a history of hypertension is admitted with sudden onset severe chest pain radiating through to the interscapular region

BP 250/140

CT scan reveals Type 2 thoracic aortic dissection

How and where should he be managed?

A 60 year old diabetic female is admitted with unstable angina. Usual meds Inhibace Plus and metformin

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BP 220/110 even after opioid pain relief, sinus rhythm 90BPM

ECG – non-specific T wave changes only

Troponins normal

How should her blood pressure be managed?

40 year old European male
Presents to ED with 3 day history headaches and visual blurring
History Was briefly on blood pressure medication 7 years ago.
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Hypertensive urgency or emergency?
Admit or discharge?
Oral or IV treatment

Hypertensive emergency

Admit

Trial of oral meds OK initially:

Aim to get BP tp ~ 180/110 within a few hours

Captopril 25mg + Amlodipine 5mg po
Can repeat after 3-4 hours

GP calls you about:
70 year old European female
New to his practice
History of hypertension, on metoprolol 23.75mg daily
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Exam otherwise OK
Fundoscopy – silver wiring and AV nipping
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Hypertensive urgency or emergency?
Requires hospital admission?
Oral or IV treatment?

Hypertensive urgency
Can be managed in community
Bring BP under control in days rather than weeks
Check basic labs + investigate for 2’ causes

Rx

Lisinopril 10mg + Amlodipine 5mg stat

See again in 1-2 days

78 year old hypertensive European man admitted with left hemiparesis. CT head normal. Diagnosis ischaemic stroke

BP 200/110

How should he be managed over the next 24 hours?

Should he be considered for thrombolysis?

In ischaemic stroke thrombolysis C/I if SBP > 185 or DBP > 110

If thrombolysis planned, treat with IV labetolol

If thrombolysis not planned, unless compelling indication, withold BP treatment unless SBP > 220 or DBP > 120

If treating – limit reduction in BP to ~ 15% in 24 hours

Restart usual oral BP meds after a couple of days

50 year old Maori man admitted with left hemiparesis
CT reveals right intracerebral haemorrage ~ 25cc
BP 230/130

How should he be managed over the next 24 hours?

For intracerebral haemorrage, SBP > 180mmHg or MAP> 130mmHg warrants immediate BP lowering

Optimally obtain intracranial pressure monitoring and adjust BP lowering to maintain cerebrtal perfusion pressure 60-80mmHg

Labetolol infusion

Nitroprusside infusion

A 55 year old man with a history of hypertension is admitted with sudden onset severe chest pain radiating through to the interscapular region

BP 250/140

CT scan reveals Type 2 thoracic aortic dissection

How and where should he be managed?

In aortic dissection aim to lower BP ASP to lowest tolerated level (100-110 systolic)

Rx of choice is IV nitroprisside infusion in combination with beta blocker (to prevent reflex tachycardia as BP is lowered)
– IV esmolol or propranolol

IV labetolol is a useful alternative (boluses or infusion)

A 60 year old diabetic female is admitted with unstable angina. Usual meds Inhibace Plus and metformin

---

BP 220/110 even after opioid pain relief, sinus rhythm 90BPM

ECG – non-specific T wave changes only

Troponins normal

How should her blood pressure be managed?

IV GTN Infusion + IV or oral beta blocker

You are called to the operating theatre by an anaesthetist to see a 30 year old woman: As he was anaesthetising her for a tubal ligation she developed severe hypertension, a tachycardia, and short runs of VT. The operation was cancelled and he woke her up again.

The patient is tachycardic, sweaty, and complains of a headache. BP is 250/150

How should she be managed?

This is an adrenergic crisis and she likely has a phaeochromocytoma

Important to establish alpha blockade before adding beta blocker (unopposed alpha receptor stimulation in presence of catecholamine excess may worsen BP)

IV phentolamine infusion followed by oral or IV beta blocker.

When settled change to oral phenoxybenzamime and oral beta blocker