Management of hypertensive Crisis

Dr. Sousan Mahmoudi

Assistant Professor of Cardiology

What is a hypertensive crisis?

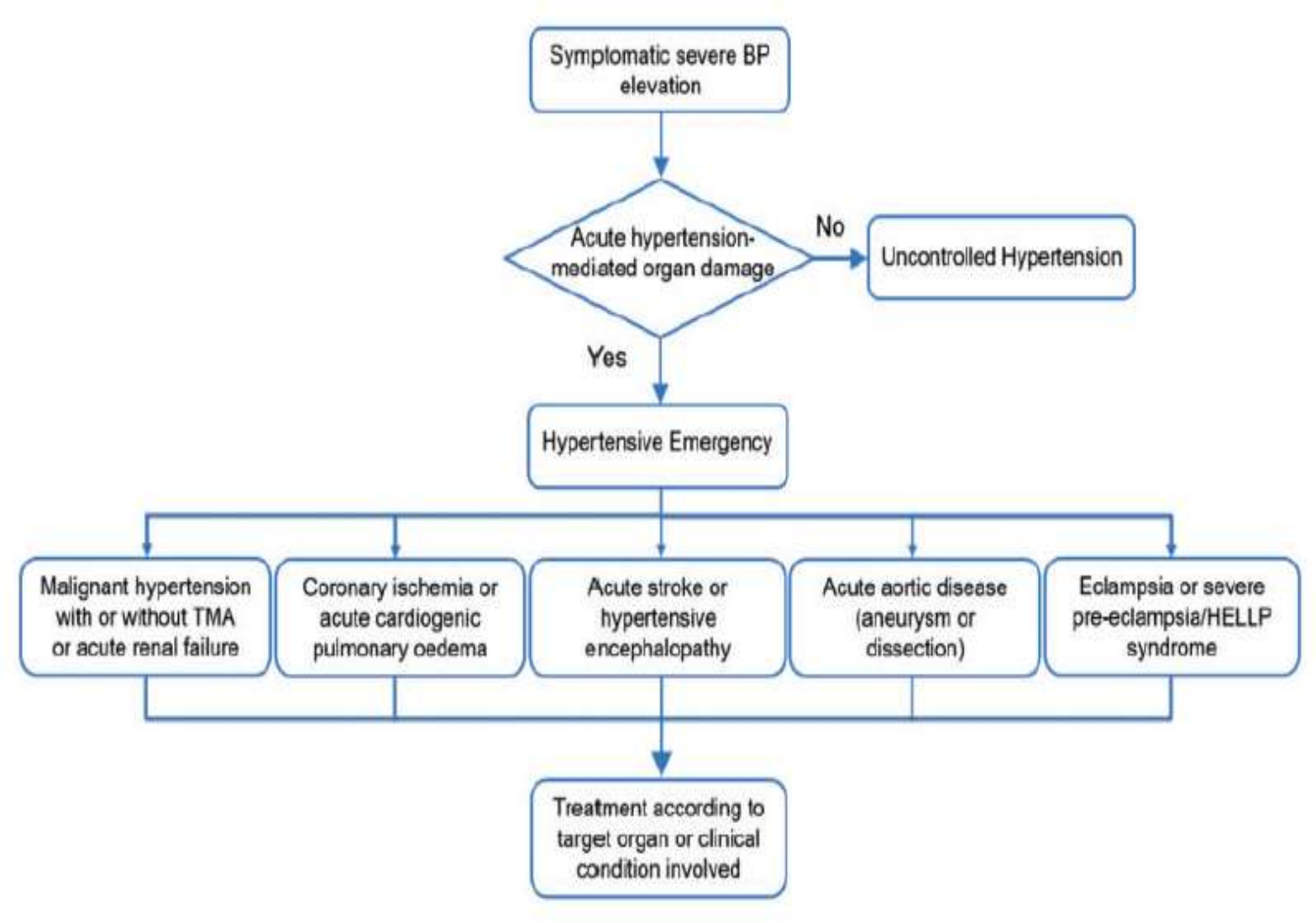
- The term ***Hypertensive Crisis*** generally is inclusive of two different diagnoses, *hypertensive emergency* (HE) and *hypertensive urgency*.
- Distinguishing between the two is important because they require different intensities of therapy.
- **HE** as being "characterized by severe elevations in blood pressure (BP) (>180/120 mm Hg), complicated by evidence of impending or progressive target organ dysfunction."
- **Hypertensive urgency** as "those situations associated with severe elevations in blood pressure without progressive target organ dysfunction."

What are the causes of hypertensive crisis?

- The most common cause of HE is an abrupt increase in BP in patients with chronic hypertension.
- **Medication noncompliance** is a frequent cause of such changes
- BP control rates for patients diagnosed with hypertension are less than 50%
- Older adults and African Americans are at increased risk of developing an HE
- Other causes of HEs include :
  - Stimulant intoxication (cocaine, methamphetamine, and phencyclidine),
  - Withdrawal syndromes (clonidine, beta-adrenergic blockers),
  - Pheochromocytoma,
  - Physiologic stress in the postoperative period (following cardiothoracic, vascular, or neurosurgical procedures)
  - Adverse drug interactions with monoamine oxidase (MAO) inhibitors

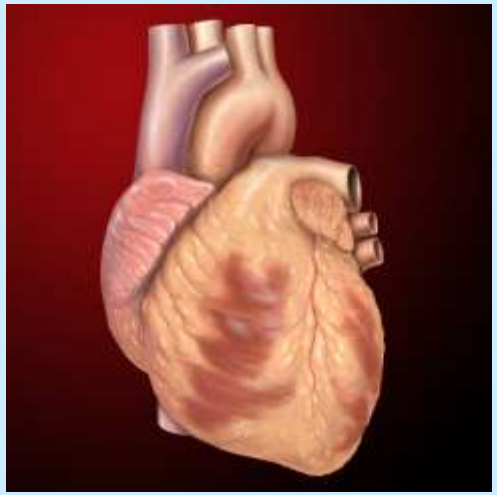
Stratification of hypertensive emergencies according to the condition or target organ involved

Hypertensive emergencies can be classified according to the presence of acute hypertension-mediated target organ damage



Acute target organ damage

Hypertensive encephalopathy
-Stroke



Acute coronary event
Acute cardiogenic pulmonary oedema

Acute retinopathy

Aortic dissection

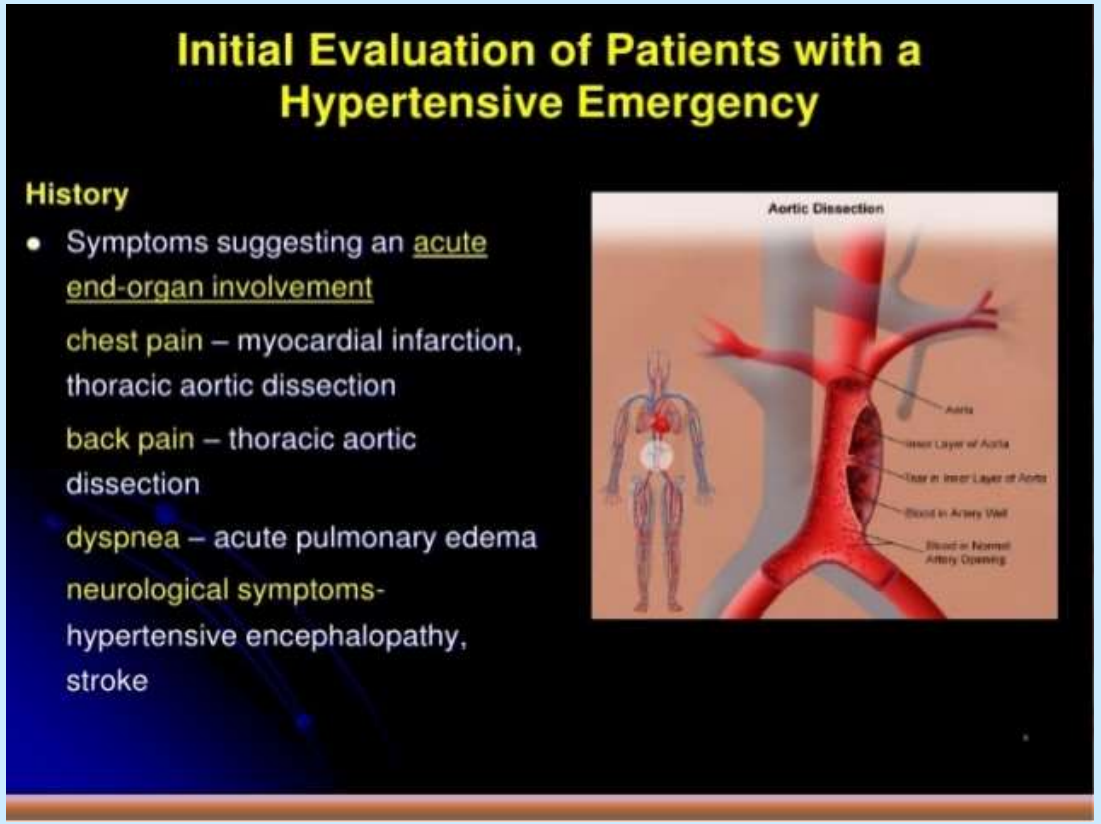
AKI

What historical information should be obtained?

- The medical history-taking should focus on :
  - Emergency symptoms
  - Possible causes : (Non-adherence & Dietary habits)
  - Use of drugs : (Steroids – NSAIDs - Cyclosporine – Sympathomimetics - Cocaine - Anti-angiogenic therapy)
  - secondary causes : (Kidney disease - Renal artery stenosis)
- In patients with pre-existing hypertension :
  - Current antihypertensive treatment
  - Treatment withdrawal
  - Disease duration
  - Previous BP control

What are the common clinical presentations of hypertensive crisis?

- **Typical presentations** include :
  1) Severe headache
  2) Shortness of breath
  3) Epistaxis
  4) Faintness
  5) Severe anxiety
  6) Other frequent(but less specific) symptoms include :
     - ➢ Dizziness : resulting from impaired cerebral autoregulation
     - ➢ Gastrointestinal complaints (abdominal pain, nausea and anorexia)



Clinical presentation

- **Clinical syndromes** typically associated with HE include :
  1) Hypertensive encephalopathy
  2) Intracerebral hemorrhage
  3) Acute myocardial infarction (MI)
  4) Acute heart failure
  5) Pulmonary edema
  6) Unstable angina
  7) Dissecting aortic aneurysm
  8) Preeclampsia/eclampsia

Clinical presentation

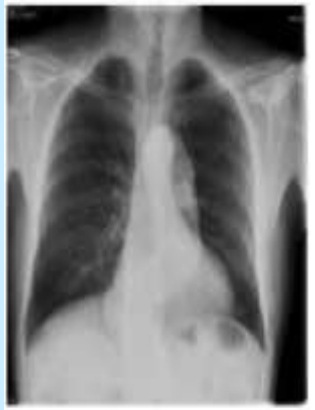
- In patients with hypertensive encephalopathy, the presence of somnolence, lethargy, tonic–clonic seizures, and cortical blindness may precede loss of consciousness
- Focal neurological lesions are rare in hypertensive encephalopathy and should raise the suspicion of intracranial haemorrhage or ischemic stroke

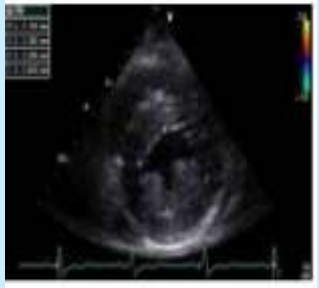
How should the physical examination be focused ?

1) Physical examination should start with recording the BP in both arms with an appropriately sized BP cuff and at the lower limb to detect pressure differences caused by aortic dissection
2) Direct ophthalmoscopy should be performed with attention to evaluating for papilledema and hypertensive exudates.
3) A brief, focused neurologic examination to assess mental status and the presence or absence of focal neurologic deficits should be performed.
4) The cardiopulmonary examination should focus on signs of pulmonary edema and aortic dissection, such as rales, elevated
5) Repeated measurements should be performed over time, since in a significant proportion of patients, the BP will fall considerably

Diagnostic workup for patients with a suspected hypertension emergency

Common tests for all potential causes
Fundoscopy is a critical part of the diagnostic workup
12-lead ECG
Haemoglobin, platelet count, fibrinogen
Creatinine, eGFR, electrolytes, LDH, haptoglobin
Urine albumin:creatinine ratio, urine microscopy for red cells, leucocytes, casts
Pregnancy test in women of child-bearing age





Specific tests by indication
Troponin, CK-MB (in suspected cardiac involvement, e.g. acute chest pain or acute heart failure) and NT-proBNP
Chest X-ray (fluid overload)
Echocardiography (aortic dissection, heart failure, or ischaemia)
CT angiography of thorax and/or abdomen in suspected acute aortic disease (e.g. aortic dissection)
CT or MRI brain (nervous system involvement)
Renal ultrasound (renal impairment or suspected renal artery stenosis)
Urine drug screen (suspected methamphetamine or cocaine use)

Acute management of hypertensive emergencies

- In patients with a hypertensive emergency immediate BP reduction is indicated to limit extension or promote regression of acute hypertension-mediated organ damage.
- The type of target organ damage is the principal determinant of the choice of treatment, target BP, and timeframe by which BP should be lowered.
- A typical example of a hypertensive emergency is the coexistence of very high BP values (often >200/ 120mmHg) with advanced retinopathy, acute renal failure, and/or thrombotic microangiopthy (TMA)

Evidence of Acute Ongoing Target Organ Damage

YES

NO

Evidence of Acute Ongoing Target Organ Damage

Hypertensive Emergency

General Goals:

- ✓Stop progression of Target Organ Damage
- ✓Avoid organ hypoperfusion during treatment

Points of emphasis:

- Parenteral therapy should be initiated immediately
- Further diagnostic testing should not delay Rx
- ICU admission & intra-arterial BP monitoring is preferred

Hypertensive Urgency

- ❑ Requires initiating, reinitiating, modifying, or **titrating oral therapy**
- ❑ **Does not require** ICU or hospital admission

Hypertensive Urgency

- Patients without acute hypertension-mediated organ damage :
  1) Treated with oral BP-lowering medication or adaptation of their current BP-lowering medication
  2) Rapid BP lowering is not recommended, as this can lead to cardiovascular complications
  3) Therapeutic goal is controlled BP reduction to safer levels without risk of hypotension
  4) Hence, short acting nifedipine should not be used given the rapid BP falls
  5) Oral BP lowering drugs : captopril, labetalol, and nifedipine

Hypertensive Emergency

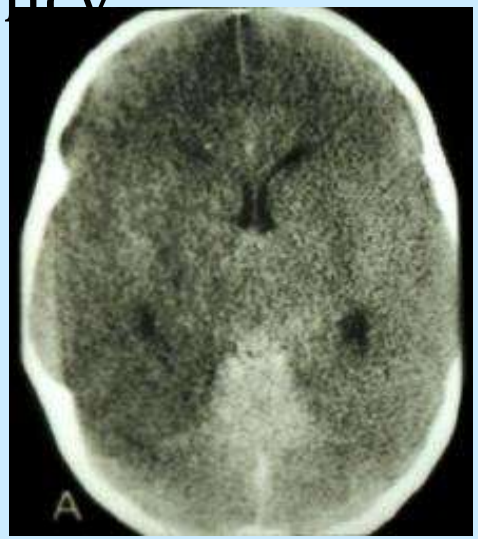
- Treatment in patients with a hypertensive emergency is driven by the type of hypertensive organ damage.
- Acute hypertension-mediated organ damage includes :
  1) Stroke (ischaemic or haemorrhagic)
  2) Acute hypertensive microangiopathy and encephalopathy
  3) Cardiogenic pulmonary oedema
  4) Coronary ischaemia
  5) Acute aortic disease
- The treatment goal is to prevent or limit further hypertensive damage by a controlled BP reduction
- In most cases, this can be best achieved by intravenous medication in a clinical area with facilities for close haemodynamic monitoring.

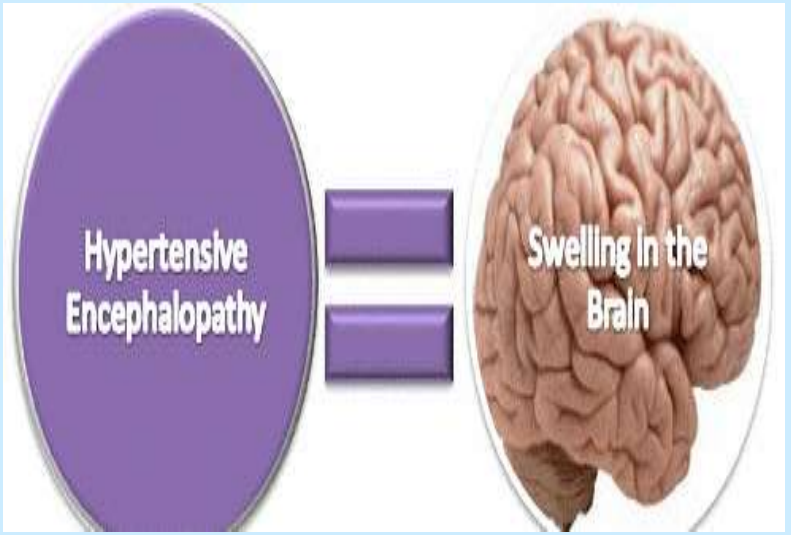
Malignant hypertension

- Large reductions in BP (exceeding a >50% decrease in mean arterial pressure) have been associated with ischaemic stroke and death
- If BP is very high (>220/ 120mmHg) or BP-lowering therapy is indicated for another reason (e.g. acute coronary event, acute heart failure, aortic dissection), it is probably safe to lower mean arterial pressure by 15% in the first 24 h
- Sodium nitroprusside, labetalol, nicardipine, and urapidil all appear to be safe and effective for the treatment of malignant hypertension.
- Fenoldopam , a short acting selective dopamine-1 agonist, and clevidipine , an ultra-short acting calcium-channel blocker for intravenous use, have been used for the treatment of patients with severe hypertension but are not widely available.
- Alternatively, oral administration of ACE-inhibitors is currently used by some teams, but must be started at a very low dose to prevent sudden decreases in BP.
- Because patients are often volume depleted as a result of pressure natriuresis , intravenous saline infusion can be used to correct precipitous BP falls if

Hypertensive encephalopathy

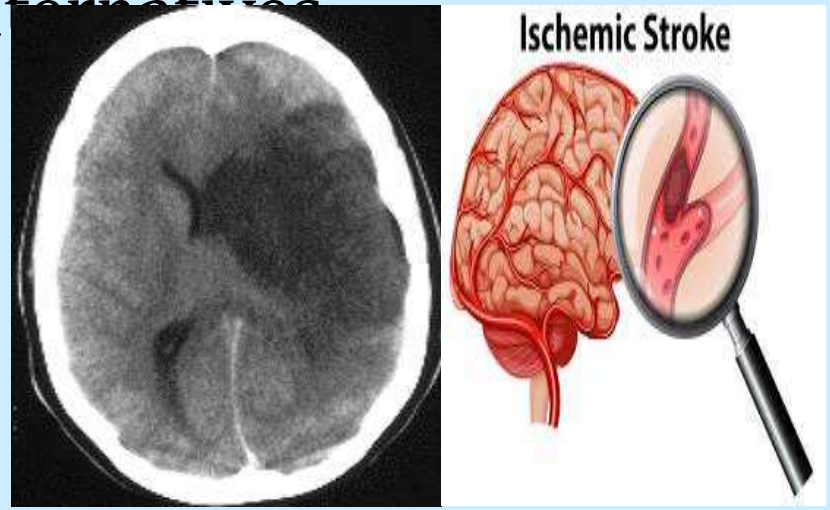
- In patients with hypertensive encephalopathy labetalol may be preferred as it leaves cerebral blood flow relatively intact for a given BP reduction compared with nitroprusside, and does not increase intracranial pressure.
- Nitroprusside and nicardipine can alternatively be used for this type of emergency.

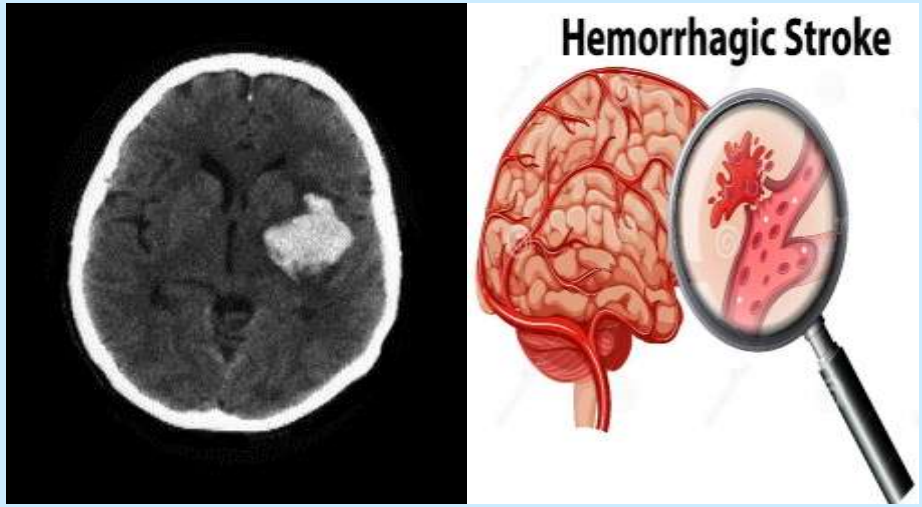




Acute ischaemic and haemorrhagic stroke

- For acute ischaemic stroke and an indication for thrombolytic therapy, lowering BP to <185mmHg systolic and 110mmHg diastolic is recommended before thrombolysis is given.
- In case acute reduction in BP is desired, labetalol is the drug of choice with nicardipine and sodium nitroprusside being useful alternatives





Acute coronary artery events

- In case severe hypertension is associated with acute coronary syndrome (cardiac ischaemia or myocardial infarction), afterload needs to be reduced without an increase in heart rate in order to decrease myocardial oxygen demand without jeopardizing diastolic filling time.
- Both nitroglycerine and labetalol have been used to lower BP in patients with an acute coronary event.
- Additional beta-blockade may be indicated for patients receiving nitroglycerine, especially if tachycardia is present

Acute cardiogenic pulmonary oedema

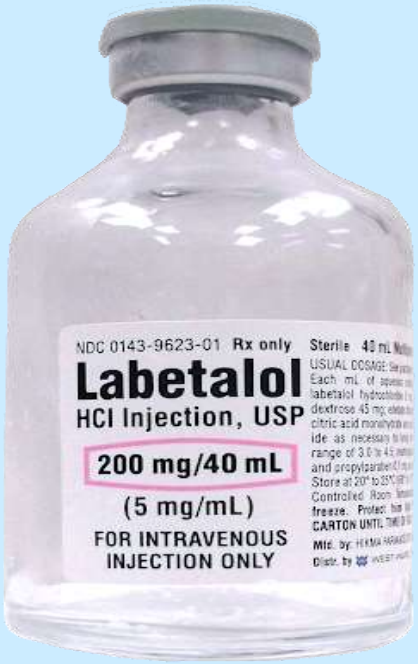
0.3–10 µg/kg/min i.v. infusion, increase by 0.5 µg/kg/min every 5 min until goal BP

- In patients with acute pulmonary oedema caused by hypertensive heart failure, both nitroglycerine and sodium nitroprusside can be used as they will optimize preload and decrease afterload
- Nitroprusside is the drug of choice as it will acutely lower ventricular pre- and afterload
- Nitroglycerine may be a good alternative, although high doses (>200mg/min) may be required to achieve the desired BP-lowering effect
- Concomitant administration of loop diuretics decreases volume overload and helps to further lower BP

Acute aortic disease

- In patients with acute aortic disease (dissection or rupture) systolic BP and heart rate need to be immediately reduced to 120mmHg or lower and 60 b.p.m. or less to reduce aortic wall stress and disease progression.
- Beta-blockers are therefore considered first line treatment.
- Esmolol can be used together with ultra-short acting vasodilating agents such as nitroprusside
- Alternatively, bolus injections of metoprolol or labetalol can be used with the possible

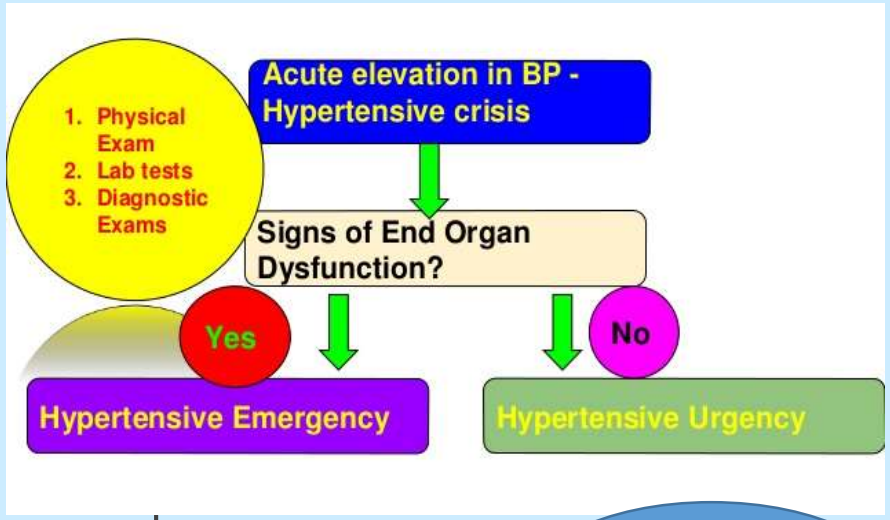
Eclampsia and severe pre-eclampsia



0.25–0.5 mg/kg i.v. bolus; 2–4 mg/min infusion until goal BP is reached

- In patients with eclampsia or severe pre-eclampsia , BP-lowering therapy is given next to intravenous magnesium Sulfate and delivery needs to be considered after the maternal condition has stabilized.
- The consensus is to lower systolic and diastolic BP <160/105 mmHg to prevent acute hypertensive complications in the mother.
- Both labetalol and nicardipine have shown to be safe and effective for the treatment of severe pre-eclampsia if intravenous BP-lowering therapy is necessary.
- In both cases monitoring of foetal heart rate is necessary.
- To prevent foetal bradycardia the cumulative dose of labetalol should not exceed 800 mg/24 h.
- Treatment with hydralazine is not widely available anymore and not recommended as it has been associated with adverse perinatal outcomes
- Treatment with nitroprusside is contraindicated because it carries the risk of foetal cyanide toxicity.

Diagnosis and Management of a Hypertensive Crisis



Hypertensive emergency

Admit to ICU (Class I)

Conditions:
- Aortic dissection
- Severe preeclampsia or eclampsia
- Pheochromocytoma crisis

Aortic dissection

Pheo crisis

eclampsia

Yes

No

Reduce SBP to <140 mm Hg during first h* and to <120 mm Hg in aortic dissection† (Class I)

Reduce BP by max 25% over first h†, then to 160/100–110 mm Hg over next 2–6 h, then to normal over next 24–48 h (Class I)

COR	LOE	Recommendations for Hypertensive Crises and Emergencies
I	B-NR	In adults with a hypertensive emergency, admission to an intensive care unit is recommended for continuous monitoring of BP and target organ damage and for parenteral administration of an appropriate agent.
I	C-EO	For adults with a compelling condition (i.e., aortic dissection, severe preeclampsia or eclampsia, or pheochromocytoma crisis), SBP should be reduced to less than 140 mm Hg during the first hour and to less than 120 mm Hg in aortic dissection.
I	C-EO	For adults without a compelling condition, SBP should be reduced by no more than 25% within the first hour; then, if stable, to 160/100 mm Hg within the next 2 to 6 hours; and then cautiously to normal during the following 24 to 48 hours.