ΕΠΕΙΓΟΥΣΑ ΥΠΕΡΤΑΣΗ

Κωνσταντινίδης Δημήτρης
Καρδιολόγος
Επιστ. Συνεργάτης Μονάδας Υπέρτασης
Ά Πανεπιστημιακή Καρδιολογική Κλινική
ΓΝΑ Ιπποκράτειο
Conflict of interest: NONE
DEFINITIONS

- **Hypertensive crisis** is an acute and severe rise in BP (>180/120mmHg) presenting with highly heterogeneous profiles.

The BP level itself may not be as important as the **rate of elevation**.
OD IN HYPERTENSIVE EMERGENCIES

- Hypertensive encephalopathy
- Cerebral infarction
- Intracranial haemorrhage
- Subarachnoid haemorrhage
- Retinopathy grade 3-4

- Acute LV failure
- ACS

- Aortic dissection

- Acute kidney injury (AKI) (malignant nephrosclerosis)

- Eclampsia

- Microangiopathic hemolytic anemia (schistocytes)
SIZE OF THE PROBLEM - EPIDEMIOLOGY

- 456,259 hospitalizations hypertensive emergency occurred from 2000 to 2007
- In 2025, more than 47 million patients will develop a hypertensive emergency
- 1-2% of hypertensive patients will have an episode of hypertensive crisis in their lifetime

- History of hypertension: 72% hypertensive urgencies
  92% hypertensive emergencies

- Risk factors:
  - (Males)
  - Obesity
  - Hypertensive or Coronary heart disease
  - Large number of antihypertensive drugs
  - Non-adherence

Hellenic Hypertension Excellence Centers

Multicentric study on the management of acute hypertensive events

Date: ..........................  Time: ..........................

Name: ..........................  Age: ..........................  Tel. number: ..........................


   SR  ☐  AF  ☐

Main symptom/organ damage:
   Asymptomatic  ☐
   Headache-dizziness  ☐
   Neurological symptoms  ☐
   Epistaxis  ☐

   Dyspnea / APE  ☐
   Angina / ACS  ☐
   Aortic dissection  ☐
   AKI  ☐

   Other: ..........................

Known HTN:  Yes ☐  No ☐

Antihypertensive treatment:
   ACE inhibitors-AT1  ☐
   Ca++ antagonist  ☐
   b-blocker  ☐
   Diuretic  ☐
   Other ..........................

History of:
   CD  ☐  Stroke  ☐  AF  ☐  DM  ☐

Possible causes of hypertensive crisis:
   Omission of drug therapy during the last 24h  ☐
   Change of drug treatment during the last week  ☐
   Drugs (NSAIDS,.....) ..........................

   Stress  ☐  Intense physical activity  ☐
   Excessive salt intake  ☐

Drug/s administered:
   p.o.  ☐
   iv  ☐
   SL  ☐  im  ☐

   ACE inhibitors-AT1  ☐
   NTL, Nitroprus.  ☐
   NTL (Pensordil)  ☐
   Ca++ antagonist  ☐
   Diuretic  ☐
   Labetalol  ☐
   b-blocker  ☐
   Anxiolytic  ☐
   Phentolamine  ☐
   Other ..........................

Laboratory tests:  

Hospital admission:  Yes ☐  No ☐

Time patient remained in ED: .......................... minutes

BP during exit from hospital: .......................... ..........................
GREEK DATA

• Based on the above mentioned proposal for the registry, we performed a pilot study in the ED of Hippocration General Hospital of Athens.
• The duration of the registry was 3 months (FEB/2017 - APR/2017)

Out of 3634 patients consecutively admitted to the Internal Medicine Section of the ED during the study period,

- 63 (1.17%) had a hypertensive crisis,
- 23 (36.5%) of them had hypertensive emergencies.
## Table 1. Causes of hypertensive crisis

<table>
<thead>
<tr>
<th>Category</th>
<th>Causes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Renal</td>
<td>Glomerulonephritis, tubulointerstitial nephritis, end-stage renal disease, renal artery stenosis, haemolytic uremic syndrome, renal vein thrombosis, polyarthritis nodosa, renal cell carcinoma</td>
</tr>
<tr>
<td>Drug or substance withdrawal</td>
<td>Clonidine, beta-blockers, alcohol, sedative-hypnotics</td>
</tr>
<tr>
<td>Medications</td>
<td>Erythropoietin, cyclosporine, monoamine oxidase inhibitors, selective serotonin reuptake inhibitors, tricyclic antidepressants, tyrosine kinase inhibitors, bevacizumab, steroids, pseudoephedrine, anaesthetic agents (malignant hyperthermia)</td>
</tr>
<tr>
<td>Illicit drugs</td>
<td>Cocaine, phencyclidine, sympathomimetics, amphetamines, anabolic steroids</td>
</tr>
<tr>
<td>Endocrine</td>
<td>Pheochromocytoma, Cushing syndrome, primary hyperaldosteronism, renin-secreting tumour, carcinoid syndrome, hyperthyroidism</td>
</tr>
<tr>
<td>Pregnancy-related</td>
<td>Preeclampsia, eclampsia</td>
</tr>
<tr>
<td>Neurologic</td>
<td>Head injury, cerebral infarction, cerebral haemorrhage, brain tumour, spinal cord injury, seizure</td>
</tr>
<tr>
<td>Others</td>
<td>Pain, anxiety, burns, scleroderma, lead intoxication, neck radiation, coarctation of aorta, Guillain–Barre syndrome, acute intermittent porphyria</td>
</tr>
</tbody>
</table>
Acute increase in humoral vasoconstrictors (angiotensin II, norepinephrine) and systemic peripheral resistance

- Increase in mechanical stress on the vascular wall
- Endothelial damage
- Increase in vascular permeability
- Activation of platelets and coagulation cascade
- Fibrin deposition
- Induction of oxidative stress
- Induction of inflammatory cytokines

Vasoconstriction and thrombosis, as a result of vascular damage lead to:

- Hypoperfusion
- End-organ ischemia
- Autoregulatory dysfunction

### CLINICAL PRESENTATION

<table>
<thead>
<tr>
<th>Signs and Symptoms</th>
<th>Hyper. Crises, %</th>
<th>Urgencies, %</th>
<th>Emergencies, %</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Headache</td>
<td>17.0</td>
<td>22.0</td>
<td>3.0</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>Epistaxis</td>
<td>13.0</td>
<td>17.0</td>
<td>0.0</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>Chest pain</td>
<td>13.0</td>
<td>9.0</td>
<td>27.0</td>
<td>&lt;.005</td>
</tr>
<tr>
<td>Dyspnea</td>
<td>12.0</td>
<td>9.0</td>
<td>22.0</td>
<td>&lt;.02</td>
</tr>
<tr>
<td>Faintness</td>
<td>10.0</td>
<td>10.0</td>
<td>10.0</td>
<td>NS</td>
</tr>
<tr>
<td>Psychomotor agitation</td>
<td>7.0</td>
<td>10.0</td>
<td>0.0</td>
<td>&lt;.004</td>
</tr>
<tr>
<td>Neurological deficit</td>
<td>7.0</td>
<td>3.0</td>
<td>21.0</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>Vertigo</td>
<td>6.5</td>
<td>7.0</td>
<td>3.0</td>
<td>NS</td>
</tr>
<tr>
<td>Paresthesia</td>
<td>6.5</td>
<td>6.0</td>
<td>8.0</td>
<td>NS</td>
</tr>
<tr>
<td>Vomitus</td>
<td>2.5</td>
<td>2.0</td>
<td>3.0</td>
<td>NS</td>
</tr>
<tr>
<td>Arrhythmia</td>
<td>1.0</td>
<td>6.0</td>
<td>0.0</td>
<td>&lt;.04</td>
</tr>
<tr>
<td>Other</td>
<td>5.6</td>
<td>2.0</td>
<td>3.0</td>
<td>NS</td>
</tr>
</tbody>
</table>

EVALUATION OF TARGET ORGAN DAMAGE

- History (alcohol, drugs, HT)
- Physical examination (neurologic, cardiovascular, renal systems)
- BP measurements:
  - Both arms
  - Sitting and standing
- Electrocardiography (90%*)
- Serum electrolytes, creatinine, complete blood count
- Urinalysis (hematuria or proteinuria)
- Funduscopic examination (13%*)
- Specific examinations
  - Echocardiogram (45%*)
  - Chest radiography (87%*)
  - Cardiac enzymes, D-dimmers
  - Brain CT or MRI (47%*)
  - Chest contrast enhanced CT or transesophageal echocardiography

*Data from the STAT registry
TYPES OF END-ORGAN DAMAGE ASSOCIATED WITH HYPERTENSIVE EMERGENCIES

- Acute heart failure: 36.8%
- Cerebral infarction: 12%
- Hypertensive encephalopathy: 16.3%
- Acute myocardial infarction or unstable angina pectoris: 24.5%
- Intracerebral or subarachnoid hemorrhage: 4.5%
- Eclampsia: 4.5%
- Aortic dissection: 2%

THERAPY HYPERTENSIVE EMERGENCIES

- **Aim:** avoid an acute worsening of organ damage and further long-term complications.
- **Optimal therapy:** varies according to the specific hypertensive emergency.
- **Drugs:** *iv* administration of short-acting and titratable drugs (*Ib*).
- For most hypertensive emergencies, 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.
  - Exceptions to gradual BP lowering:
    - Acute aortic dissection
    - The acute phase of an ischemic stroke
- **Admission ICU**, continuous BP monitoring (arterial line).
- After a suitable period (often 8 to 24 hrs) of BP control at target, oral medications are usually given and the initial intravenous therapy is tapered and discontinued.

*Li JZ, Eagle KA, Vaishnava P. Cardiol Clin 2013; 31:493.*
cerebral edema
clinical syndrome of hypertensive encephalopathy (headache, confusion, nausea, vomiting)

In patients without hypertension, flow is kept constant over a MAP of 60-120mmHg.
In hypertensives, flow is constant over a MAP of 110-180mmHg because of arteriolar thickening.

References:
Strandgaard S, Paulson O. Stroke 1984;15:413–6
## DRUGS – VASODILATORS IV

<table>
<thead>
<tr>
<th>Drug</th>
<th>Dose</th>
<th>Onset of Action</th>
<th>Duration of Action</th>
<th>Adverse Effects</th>
<th>Special Indications</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Nitroprusside</strong></td>
<td>0.25-10 µg/kg/min</td>
<td>Immediate</td>
<td>1-2 min</td>
<td>Nausea, vomiting, muscle twitching, thiocyanate and cyanide toxicity</td>
<td>Not preferred for most hypertensive emergencies</td>
</tr>
<tr>
<td><strong>Nitroglycerin</strong></td>
<td>5-100 µg/min</td>
<td>2-5 min</td>
<td>5-10 min</td>
<td>Headache, vomiting, methemoglobinemia, tolerance</td>
<td>Not preferred but may be useful with coronary ischemia</td>
</tr>
<tr>
<td><strong>Fenoldopam</strong> (dopamine-1 agonist)</td>
<td>0.1-1.6 µg/kg/min</td>
<td>4-5 min</td>
<td>10-15 min</td>
<td>Tachycardia, increased intraocular pressure (glaucoma)</td>
<td>May be indicated for renal insufficiency</td>
</tr>
<tr>
<td><strong>Nicardipine</strong></td>
<td>5-15 mg/h</td>
<td>5-10 min</td>
<td>1-4 h</td>
<td>Headache, nausea, flushing, tachycardia</td>
<td>Most hypertensive emergencies</td>
</tr>
<tr>
<td><strong>Clevidipine</strong></td>
<td>1-21 mg/h maximum</td>
<td>2-4 min</td>
<td>5-15 min</td>
<td>Headache, nausea, flushing, tachycardia, lipid disorders</td>
<td>Most hypertensive emergencies</td>
</tr>
<tr>
<td><strong>Hydralazine</strong></td>
<td>Bolus 5-20 mg</td>
<td>10-20 min</td>
<td>1-4 h</td>
<td>Tachycardia, flushing, headache, vomiting, aggravation of angina</td>
<td>Eclampsia. Not for aortic dissection</td>
</tr>
<tr>
<td><strong>Enalaprilat</strong></td>
<td>Bolus 1.25-5mg every 6h</td>
<td>15 min</td>
<td>12-24 h</td>
<td>Hypotension, renal failure, hyperkalemia</td>
<td></td>
</tr>
<tr>
<td>Drug</td>
<td>Dose</td>
<td>Onset of Action</td>
<td>Duration of Action</td>
<td>Adverse Effects</td>
<td>Special Indications</td>
</tr>
<tr>
<td>-----------------------</td>
<td>-------------------------------------------</td>
<td>-----------------</td>
<td>--------------------</td>
<td>--------------------------------------</td>
<td>------------------------------------------</td>
</tr>
<tr>
<td><strong>Phentolamine</strong></td>
<td>5-15 mg every 5 to 15 min</td>
<td>1-2 min</td>
<td>3-10 min</td>
<td>Tachycardia, flushing, headache</td>
<td>Catecholamine excess</td>
</tr>
<tr>
<td>(nonselective α-adrenergic blocker)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Esmolol</strong></td>
<td>250-500 µg/kg/min for 4 min, then 50-300 µg/kg/min</td>
<td>1-2 min</td>
<td>10-20 min</td>
<td>Hypotension, nausea</td>
<td>Aortic dissection after surgery</td>
</tr>
<tr>
<td>Selective β-blocker</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Labetalol</strong></td>
<td>Bolus 20-80 mg every 10 min. Max 300mg. or 2 mg/min</td>
<td>5-10 min</td>
<td>3-6 h</td>
<td>Vomiting, scalp tingling, burning in throat, dizziness, nausea, heart block, orthostatic hypotension</td>
<td>Most hypertensive emergencies except acute heart failure</td>
</tr>
<tr>
<td>α1 + β-blocker, (β &gt;&gt; α1)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

- A diuretic may be needed after other antihypertensives are used, because reactive renal Na⁺ retention usually accompanies a fall in pressure and may blunt the efficacy of non-diuretic agents.
THE STAT REGISTRY (STUDYING THE TREATMENT OF ACUTE HYPERTENSION)

- Number of antihypertensive drugs for BP control:
  - 36% were treated with 1 intravenous drug,
  - 37% received 2,
  - 26% received at least 3.

- Drugs used:
  - labetalol 32%,
  - metoprolol 17%,
  - nitroglycerin 15%,
  - hydralazine 15%

- Those initially treated with nicardipine were the most likely to be managed with a single intravenous agent

- 3.4% had iatrogenic hypotension requiring treatment with fluids and/or vasopressor drugs

- The median duration of iv antihypertensive therapy was 6.5 hrs.

NEUROLOGIC EMERGENCIES

• **Hypertensive encephalopathy**: reduce the MAP 25% over 8hrs.
  - Prefer: **Labetalol, Nicardipine, Esmolol**. Avoid: nitroprusside and hydralazine.

• **Acute ischemic stroke**:
  - Initiate treatment if BP is >220/120mmHg, reduction of 15% during the first 24h after onset of stroke (II-B) (or when required by comorbid conditions I-C).
  - If fibrinolytic therapy (I-B) or intra-arterial therapy is planned (IIa-B), initiate treatment if BP >185/110mmHg. BP should be maintained <180/105 mm Hg for 24h after alteplase.
  - Prefer: **Labetalol, Nicardipine** and **Clevidipine**

• **Acute intracerebral hemorrhage** (INTERACT-2, ATACH-2):
  - Increased ICP: maintain MAP <130mmHg (or SBP <180mmHg) for the first 24 hrs
  - Without increased ICP: maintain MAP <110mmHg (or SBP <160mmHg) for the first 24 hrs.
  - Prefer: **Labetalol, Nicardipine, Esmolol**. Avoid: nitroprusside and hydralazine.

CARDIOVASCULAR EMERGENCIES

• **Aortic dissection**: rapid reduction (10-20min) of SBP <100-120mmHg (caution if signs of end-organ hypoperfusion are present).
  - Prefer: **b-blockers** (esmolol, labetalol), **vasodilators** (nicardipine, nitroprusside), narcotic **analgesics** (morphine sulfate).
  - Avoid b-blockers if there is aortic valvular regurgitation or suspected cardiac tamponade.

• **Acute coronary syndrome**: maintain BP <160/100mmHg. Reduce the BP by 20-30% of baseline.
  - Prefer: **b-blockers, Nitroglycerin**.
  - BP is >180/110mmHg relative contraindication of thrombolysis.

• **Acute heart failure**: treat with **vasodilators** (in addition to diuretics) for a SBP >140mmHg.
  - Prefer: **nitroglycerin, Enalaprilat**.

SYMPATHOMIMETIC HYPERTENSIVE CRISES
COCAINE TOXICITY / PHEOCHROMOCYTOMA

- **Hyperadrenergic states** can be produced:
  - use of drugs: cocaine, amphetamines….
  - withdrawal of short-acting antihypertensive agents: clonidine, beta blockers
  - Pheochromocytoma

- **Prefer**: Phentolamine, Diazepam and Nitroglycerin / Nitroprusside

- Avoid b-blocker before administering phentolamine.

- **Cocaine toxicity**: Usually hypertension and tachycardia is responsive to iv benzodiazepines. Phentolamine are the preferred agents for cocaine-associated acute coronary syndromes.

- **Pheochromocytoma**: Only after phentolamine, b-blockers can be added for BP and HR control.

WHAT HAPPENED TO SODIUM NITROPRUSSIDE?

- Sodium nitroprusside recommended for all hypertensive emergencies except eclampsia
  
  Mansoor and Friedman. Heart Disease 2002; 4:358

- Sodium nitroprusside recommended for
  - acute aortic dissection
  - acute pulmonary edema with systolic dysfunction

  Marik and Varon. Chest 2007; 131:1949

- Disadvantages of sodium nitroprusside
  I. Decreases cerebral blood flow and increases intracranial pressure
  II. “Coronary steal” in patients with coronary disease
  III. Risk of cyanide toxicity

- Use when other agents not effective
  - Monitor thiocyanate levels
  - Avoid in renal or hepatic dysfunction
HYPERTENSIVE URGENCIES

- Usually in patients non-adherent with their chronic antihypertensive drug regimen and their low-sodium diet or inadequate treatment.
- Exclude acute, ongoing, target-organ damage
- In 32% of adults with severe asymptomatic hypertension, 30 min of rest in a quiet room reduce BP ≥20/10mmHg.
- The best therapeutic approach is the **oral** administration of antihypertensive drugs aimed to lower BP gradually over 24–48h.
- Usually used agents:
  - Oral furosemide (if volume overloaded) 20mg (higher if CKD)
  - Oral clonidine 0.2mg
  - Oral captopril 6.25-12.5mg
- Hospitalization admission is **not** indicated, managed on an ambulatory basis.
- Pre-discharge BP value is not associated with immediate serious adverse events and does not affect short-term BP control.

In 6 yrs, of 2,199,019 unique patient office visits, 59,836 (4.6%) met the definition of hypertensive urgency (>180/110mmHg).

Mean age was 63.1 yrs; 57.7% were women; and 76.0% were white.

Mean SBP 182.5 mmHg; and mean DBP 96.4 mmHg

<table>
<thead>
<tr>
<th>Outcome</th>
<th>No. (%) of Patients</th>
<th>P Value</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Referred to Hospital</td>
<td>Sent Home</td>
</tr>
<tr>
<td></td>
<td>(n = 426)</td>
<td>(n = 58,109)</td>
</tr>
<tr>
<td>MACE&lt;sup&gt;c&lt;/sup&gt;</td>
<td></td>
<td></td>
</tr>
<tr>
<td>7 d</td>
<td>2 (0.5)</td>
<td>61 (0.1)</td>
</tr>
<tr>
<td>8-30 d</td>
<td>2 (0.5)</td>
<td>119 (0.2)</td>
</tr>
<tr>
<td>1-6 mo</td>
<td>4 (0.9)</td>
<td>492 (0.8)</td>
</tr>
<tr>
<td>Uncontrolled hypertension</td>
<td></td>
<td></td>
</tr>
<tr>
<td>1 mo&lt;sup&gt;d&lt;/sup&gt;</td>
<td>349 (81.9)</td>
<td>49,320 (84.9)</td>
</tr>
<tr>
<td>6 mo&lt;sup&gt;e&lt;/sup&gt;</td>
<td>213 (66.6)</td>
<td>24,819 (60.2)</td>
</tr>
<tr>
<td>All-cause hospital admission</td>
<td></td>
<td></td>
</tr>
<tr>
<td>7 d</td>
<td>35 (8.2)</td>
<td>2,311 (4.0)</td>
</tr>
<tr>
<td>8-30 d</td>
<td>48 (11.3)</td>
<td>3,897 (6.7)</td>
</tr>
</tbody>
</table>
FOLLOW-UP

• Secondary causes of hypertension are more common in patients who have a hypertensive crisis compared with other hypertensive populations.

• Thus, patients with a hypertensive emergency should be evaluated for common forms of secondary hypertension and, if there are suggestive clinical clues, less common causes of secondary hypertension.

• Patients should be reminded that adherence to long-term antihypertensive drug therapy can reduce the risk of recurrent hospitalization for hypertensive emergencies.
CONCLUSIONS

BP > 180 and/or 120 mmHg
(after repeated measurement at rest)

Symptoms and signs suggesting end-organ damage
- Physical examination including fundoscopy
- Creatinine, electrolytes
- Blood count
- BNP, cardiac troponin and biomarkers
- Urine analysis (for proteinuria, haematuria and metanephrines)
- Renin, aldosterone and catecholamines (if secondary hypertension suspected)
- Other (according to clinical presentation)

ECG
- Chest x-ray
- Transthoracic echocardiogram
- Brain CT (if neurological alterations)
- Thoracic contrast CT (if aortic dissection suspected)

Prompt treatment with IV drugs according to clinical picture and hospital admission

Absence of symptoms and signs suggesting end-organ damage
- Physical examination
- Repeat BP measurements

BP still elevated
- Creatinine, electrolytes
- Blood count
- Urine analysis
- ECG
- Fundoscopy

Drug treatment (oral administration)
- Short observation
- Referral to «hypertension clinic» for close follow-up and further investigations

BP normal or decreased
- Referral to GP for follow-up and treatment changes

TREAT THE PATIENT AND NOT THE NUMBER

THANK YOU

13 April 1945
THE CATIS RANDOMIZED CLINICAL TRIAL

CONCLUSIONS

- Patients presenting to emergency departments with severe hypertension deserve prompt triage to establish the presence of a hypertensive emergency or urgency.

<table>
<thead>
<tr>
<th>Variable</th>
<th>Emergencies</th>
<th>Urgencies</th>
</tr>
</thead>
<tbody>
<tr>
<td>Symptoms</td>
<td>Yes</td>
<td>No or minimal</td>
</tr>
<tr>
<td>Acute BP increase</td>
<td>Yes</td>
<td>Yes</td>
</tr>
<tr>
<td>Acute target organ damage</td>
<td>Yes</td>
<td>No</td>
</tr>
<tr>
<td>BP reduction rate</td>
<td>Minutes to hours</td>
<td>Hours to days</td>
</tr>
<tr>
<td>Evaluation for secondary hypertension</td>
<td>Yes</td>
<td>Yes</td>
</tr>
</tbody>
</table>

- The approach in the hypertensive crisis is not well established.
- In hypertensive emergencies use of parenteral antihypertensive drugs to prevent progression of target organ damage and admission to an ICU with continuous blood pressure monitoring.
- Most patients with hypertensive urgencies can be managed on an ambulatory basis with initiation or adjustment of appropriate oral antihypertensive therapy.
REAL LIFE  (PSEUDO-HYPERTENSIVE CRISIS)

• May represent the consequence of acute anxiety, panic attacks, painful syndromes, venous epistaxis or alcohol withdrawal; the treatment of these conditions is associated with a concomitant reduction of BP.

• Elderly patients often use these BP measurements as a way to explain their symptoms.

• Almost 10% of patients presenting to ERs complained of high BP, despite the fact that only 7.5% are classified as hypertensive emergencies.

• Anxiolytics in the treatment of excessive hypertension are very effective. When compared to captopril, treatment with diazepam or alprazolam showed to be equally effective in lowering BP.

Tandeter H. Medical Hypotheses 88 (2016) 35–37