Original Article

Systemic Hypertension at Emergency Units. The Use of Symptomatic Drugs as Choice for Management

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Objective

Compare the therapeutic response of symptomatic, hypertensive patients to symptomatic medication or anti-hypertensive drugs at the Emergency Unit.

Methods

A randomized, blind clinical trial involving 100 (one hundred) patients assisted at the Cardiology Emergency Unit at Oswaldo Cruz University Hospital (HUOC). All patients reported symptoms associated to systolic pressure (SBP) between 180 and 200 mmHg and/or diastolic pressure (DBP) between 110 and 120 mmHg. Patients were randomized for treatment with symptomatic (dipirone or diazepan) or anti-hipertensive drug (captopril). Those reporting any associated clinical condition and in need of immediate treatment at the Emergency Unit were excluded from the study. Patients reporting no symptoms, and systolic pressure reduced to levels under 180 mmHg and diastolic pressure under 110 mmHg after the 90-minute period were considered as having met discharge criteria.

Results

Mean age of population studied was 54.4 years old, most commonly females. Patients were chronic hypertensive, on irregular pharmacological treatment, with low compliance to non-pharmacologic actions, and classified as overweight and obese grade I. Headache, type D (non-angina) chest pain, and dyspnea were the most frequent complaints. The number of patients treated with symptomatic drug who reached discharge criteria was similar to that of patients treated with anti-hypertensive (p=0.165). No association was found between previous high blood pressure (HBP) diagnosis (p=0.192), pharmacological treatment (p=0.687), and non-pharmacological treatment and discharge criteria.

Conclusion

Blood pressure (BP) was reduced below levels for discharge criteria for a (non-significant) higher rate of patients treated with symptomatic drug, who were turned into asymptomatic after the observation period.

Keywords

hypertension crisis, symptomatic drugs, systemic hypertension, emergency treatment

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In Brazil, 15% to 20% of urban adult population is estimated to be affected by high blood pressure (HBP)[1,2]. Data on hypertension crisis (HC) range from 1% to 27%[3-12]. Most centers do not count on specific standard dignosis procedures for hypertension crisis[13,14]. It is assumed that at quite many circumstances, blood pressure increase during emergency assistance may be misclassified as hypertension crisis[3].

Symptoms have not taken up much relevance in the management of hypertensive patients. Some reports describe situations when treatment is established by taking into consideration hypertension levels only. For patients who report neither evidence of fast deterioration of target organs nor immediate life risk when facing a triggering event – such as pain or emotional stress – blood pressure increase has been called hypertension pseudo-crisis [11-13]. Generally speaking, those are mild to moderate, non-controlled, and/or non-compliant hypertensive patients. That definition makes it clear that blood pressure increase is a result, rather than a cause, of symptom referred by patient. Treating the cause (symptom), in those cases, will result in significant reduction – or even normalization – of blood pressure levels. Resuming chronic management is the most appropriate conduct. There is no evidence that acute blood pressure reduction, in those cases, is benefic[11,15,16]. Therapeutics focusing blood pressure levels as a priority – for those situations – may bring more risks than benefits.

Most individuals who are not under potential life risk, or under target organ acute injury risk, also report non-specific symptoms such as headache, tinnitus, epistaxis, dizziness, dyspnea, palpitations, chest discomfort, numbness, shivering, or even no complaint at all. Many of the symptoms – thought to be caused by blood pressure increase – have been identified as misguiding factors in epidemiological studies. In hypertensive patients under pharmacological management those symptoms are more consistently associated to psychological factors rather than blood pressure levels or treatment being used[17]. Among those symptoms, headache stands out as the most frequent[4,5,14,17,18-22] and the one alledgely most commonly associated to HBP[23]. Both for patients and many doctors, blood pressure increase would be the cause of a headache. It is not always possible to outline the symptoms that are secundary to blood pressure increase. They may at times actually be the cause, and therefore, are prioritized in the therapeutical approach[14]. Headache – especially severe headache – may trigger non-specific sympathetic nervous system activation, resulting in blood pressure increase[23-25].

Literature has not yet provided data on the comparison between an anti-hypertensive management approach – whose therapeutic

target is blood pressure level – and a symptomatic drug-based management approach focusing major complaint, which actually led patient to look for Emergency assistance. While the latter assumes that symptoms may have caused blood pressure increase – and therefore that is the focus for assistance – the former works on the hypothesis that symptoms are caused by blood pressure increase.

It should be pointed out that, although not common, the anti-hypertensive management practiced at the Emergency Unit may, at times, result in disastrous consequences due to inappropriate blood pressure decrease. Therefore, careful patient monitoring is a must. That, in its turn, involves additional costs for equipment and drugs, as well as operational and human costs. The advantages of complaint-focused therapeutics are not restricted to the reduction of anti-hypertensive undesireble effects or therapy cost only, but also to the possibility of making available the sort of management through which patient and patient´s symptoms are at the very focus of attention, rather than figures that stand for blood pressure levels.

This paper compared the therapeutic response of Emergency Unit symptomatic hypertensive patients to symptomatic or anti-hypertensive drug management.

Methods

This randomized, blind clinical trial studied one hundred (100) patients reporting symptoms associated to blood pressure increase from May, 2001 through to October, 2002, assisted at the Cardiology Emergency Unit at Oswaldo Cruz University Hospital. The study was approved by the hospital Research Ethics Committee. Patients were informed on research guidelines and signed a consent form.

Patients reporting DBP between 110 and 120 mmHg pressure and/or SBP between 180 to 200 mmHg pressure that looked for assistance at the Emergency Unit and reporting the symptoms that follow were included in the study: headache, dyspnea, type D chest pain (non-angina)[26], numbness, palpitations, dizziness and shivering.

Those who included the complaint of dizziness associated to cerebrovascular changes or acute labirynthopathies, chest pain suggesting myocardial ischemia, or acute ischemial evidence in the 12-lead ECG – infra or supra unlevelling of segment ST higher than 1 mm and/or negative, symmetric, increased amplitude of the T wave, palpitations associated to pace changes detected by auscultation or confirmed by ECG (different, not regular sinusal rhythm) or dyspnea resulting from pulmonary congestion, were excluded from the study. Those who reported any other condition requiring Emergency Unit treatement, such as: acute myocardial infarction (AMI); acute lung edema; heart arrythmia; bronchial asthma; chest angina; left ventricular failure etc, were also excluded, since symptoms reported could have been associated to those conditions and some of the drugs used for corresponding management could interfere over blood pressure.

Patients on symptomatic drugs management focusing the complaint that led them to Emergency Unit assistance were included in the group called Symptomatic Group. Patients reporting headache or type D (non-angina) chest pain were then administered oral dipirone (500 mg); patients reporting palpitations, dyspnea, dizziness, shivering or numbness were administered oral diazepan (5 mg). The second group – named Anti-Hypertensive Group – was administered oral captopril – one single, 25 mg dose.

Sample size was calculated as follows:

$$2n=4p(1-p)(Z\alpha+Z\beta)^2/\delta^2$$

where (p) is the ratio for favorable results from standard treatment =90%; (δ) the difference between results ratio among treatment groups =0.15; (α) type I error =0.05; and (β) type II error =0.20. So, sample calculation result was 164 patients (84 in each treatment group).

Alternate randomization process was used, where the first patient assisted was allocated in the anti-hypertensive group; the second, in the symptomatic group, and so on. Patients were the only ones not aware of intervention allocation.

Patient discharge was considered as closing. Patients switching to asymptomatic status, and reporting diastolic pressure reduced to levels under 110 mmHg and diastolic pressure under 180 mmHg after the 90-minute observation period were considered as having reached criteria to be discharged. Those who did not manage to meet such criteria would return to emergency assistance to be conducted by on-duty doctor off study protocol.

Potential misguiding factors were also analyzed, such as: previous diagnosis, pharmacological and non-pharmacological management of HBP.

Patients were assisted by Emergency Unit on-duty doctor at a first moment. Once study inclusion criteria were met, patients were referred to one of the professionals in charge of data collection. Blood pressure was taken again, then through mercury column sphigmomanometer – previously calibrated and research exclusive - to confirm values previously detected. Patient's arm diameter and tensiometer cuff were taken into account. Mean blood pressure (MBP) was measured as follows: MBP=SBP+(2xDBP)/3[13]. Blood pressure (BP) values used in this research were those of the second measure. Patients were then referred to an exclusive room – calm, air conditioned, in the facility next to Emergency Unit – where they were kept all through assistance up to exiting study protocol. Patients could choose to have a family member or accompanying person with. Reassessements were carried out every 30 minutes for a total observation time of ninety (90) minutes. During that time, BP and heart rate (HR) were measured. The improvement of complaints reported at admission, the detection of new symptoms, and possible side effects associated to drug administration were evaluated.

In the meantime, a questionnaire with data collection involving previous diagnosis and family history of HBP, pharmacological and non-pharmacological treatment for HBP and compliance was applied to patients.

For the purpose of this research, compliance to pharmacological treatment of HBP was understood to be represented by individuals on daily medication for high blood pressure who had taken it on day of assistance and within 24 hours preceding assistance. As for non-pharmacological treatment of HBP, study focus was on regular physical exercise. For the purpose of the present study, that was understood to be equal to or more frequent than three times a week, associated to salt-restricted diet, low-calorie diet, and non-smoking habits. Information on primary complaint that led to assistance, as well as data on gender, body weight, height, age and ethnic group were also collected.

Medications were administered while still at Emergency Unit room by researchers themselves. Through the use of auscultatory

technique indirect measure[1,27], Korotkoff[27] phase I was used to determine SBP/phase V was used to determine DBP. All patients had physical examination and 12-lead ECG done, with the purpose of detecting exclusion conditions as described previously.

Data descriptive analysis was carried out through mean and standard deviation for continuous variables, and through frequency and relative frequency (%) for categoric variables. The comparison between variables frequencies in the two groups of patients was carried out through chi-square test or Fisher-Freeman-Halton exact test for categoric variables, and through t Student test when variables were continuous. The comparison of groups treated with symptomatic and anti-hypertensive medication involved point estimates and interval estimates for ratio between discharge rates in the two groups (relative risk). Confidence level for interval estimate was 95%, and significance level for all tests was 5%.

Results

Patient's age range varied from 21 (youngest) to 91 (oldest), with mean and median at 54.4 and 53 years old respectively (SD=13.9 years old). Sixty-six patients were females. As for ethnic groups, 75% were classified as non-whites. Body mass index (BMI) ranged from 18.6 and 44.3 kg/m^2, with mean at 29.0 kg/m^2 (SD=5.3 kg/m^2). Sixty-two patients reported overweight and obesity level grade I. Ninety-one patients had previous hypertension diagnosis. From those, seventy-one (78%) were on anti-hypertensive medication.

Mean values for SBP, DBP and MBP of population under study on admission were 186.4; 107.2 and 133.6 mmHg. No statistically significant differences were reported between blood pressure levels of individuals with or without HBP diagnosis and those on anti-hypertensive drugs or not (table I).

Primary symptom referred to was headache (35%). Precordial pain – classified as type D (non-angina), was reported by 21% of patients under study, followed by dyspnea (16%), and dizziness 14% (fig. 1).

Tables II and III compare data on both treatment groups in regard to demographic and biological, and clinical and therapeutic variables. Only pharmacological treatment compliance reported statistically significant difference between the two groups.

Discharge criteria were met by 58.8% (30/51) of patients treated with anti-hypertensive medication (table IV). Ratio-proportion (RP) for discharge between the symptomatic treatment group and the anti-hypertensive group was 1.31 (95% CI: 0.89 to 1.93). That interval shows that 1, that is, the unit, was a possibility for treatment success ratio-proportion, thus meaning that the assumption that discharge rate was identical in the two treatment groups could not be rejected at 5% level. Result from chi-square test shows that such assumption could not be rejected at any significance level lower than 16.5% (p=0.165).

Previous pharmacological treatment compliance reported statistically significant difference between the two groups (table III), although no statistically significant association was shown between that variable and discharge criteria (p=0,466; table V). Therefore, compliance to pharmacological treatment is not a misguiding factor for the purpose of discharge criteria. Therefore, no re-evaluation of the effect of symptom medication and anti-hypertensive medication to measure pharmacological treatment compliance control was necessary.

At admission, compliant patients reported the following mean values, respectively: SBP, DBP and MBP 187.5; 103.8 and 131.7 mmHg. Non-compliant patients reported mean blood pressure as follows: 189.0 mmHg, 106.4 mmHg and 134.0 mmHg, respectively. No statistically significant difference was shown for BP levels between the two groups of patients (p=0.76; p=0.63 and p=0.56 respectively).

Table VI shows the analysis of the association between discharge criteria and each of the following variables: previous Pharmacological treatment of HBP, physical exercise, diet for body weight reduction, low-sodium diet, and active smoking. No statistically significant association was shown between discharge criteria and those variables.

Forty-eight per cent of patients met discharge criteria within 30 minutes under observation, 36.5% within 60 minutes, and 19.2% within total observation time of 90 minutes. Mean time from study inclusion and discharge was 52.5 minutes. In the group treated with captopril mean time was 54.6 minutes, and in the group treated with symptomatic medication it was 51 minutes. There is no statistically significant difference between the two time means (p=0.59).

Symptoms persistance as reported at admission was the primary reason 48% of patients did not reach discharge criteria as established. That was the case for 24 (50%) patients. BP was kept high in 15 (31.3%) patients. In 9 (18.8%) patients neither BP decrease nor symptom improvement was reported. Reasons that prevented discharge criteria to be met in the two treatment groups can be found in Table VII.

Discussion

Lowest BP levels for study inclusion were 110 and 180 mmHg for DBP and SBP, respectively, the reason being that patient medication has most frequently been administered at those levels at Emergency Units. Highest values for DBP and SBP were set at 120 and 200 mmHg, respectively, since it is widely accepted that levels above those significantly increase the risk of acute complications associated to HBP[8].

Choice was made for the administration of an analgesic or an anxyolitic to limit the number of therapeutic agents, as well as for the fact that those drugs are usually used to fight symptoms adopted as criteria for treatment at Emergency Units. Captopril is seen as the safest non-parenteral drug for occurrences when BP increase is associated to target organ acute injury[13,28,29], with faster onset action among all ACE inhibitors[29]. Nifedipine – most commonly used drug for hypertensive crises – has recently been target of severe criticism, and its use has been discouraged[3,8,10,13,19,28,30,31].

Symptoms were limited to those recorded in inclusion criteria, since those were the ones most frequently reported in the pilot study the authors carried out in the past[21], as in other studies, where headache, dizziness, chest pain, palpitations and dyspnea – among others – are pointed out as the most frequent[4,5,14,18,19-21]. Based on results as presented, patients reporting increased BP and other symptoms rather than the ones in the present study would probably benefit from an approach in which symptoms would also be considered when making therapeutic choices.

When target organ is not acutely injured, the recommended approach is to reduce SBP down to 160 to 180 mmHg, and DBP down to 100 to 110 mmHg[8,24,30,32,33]. SBP<180 mmHg

Table I - SBP, DBP and MBP means at hypertensive patients' admission and those on anti-hypertensives being assisted at Cardiology Emergency Service, HUOC; 2002

Variable	Hypertensive, with previous diagnosis			Hypertensive on anti-hypertensives		
	Yes (n=91)	No (n=9)	p	Yes (n=71)	No (n=20)	p
SBP (mean±SD)	186.8±14.6	181.8±8.7	0.15	186.3±15.3	188.7±11.8	0.51
DBP (mean±SD)	108.2±13.6	97.1±10.6	0.016	108.3±13.8	107.6±13.3	0.85
MBP (mean±SD)	134.4±9.9	125.3±7.6	0.018	134.3±10.1	134.7±9.7	0.88

*SD = Standard deviation

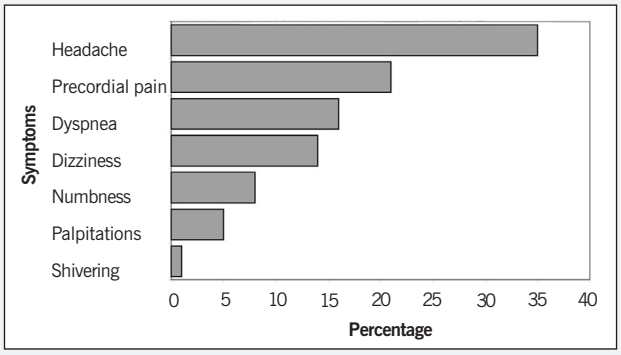

Fig. 1 - Symptoms presented by high blood pressure patients, assisted at the Cardiology Emergency Unit at HUOC; 2002.

and DBP< 110 mmHg levels were taken as discharge criteria - provided the patient were asymptomatic - since under such condition the risk for acute complications is reduced, and the adjustment of chronic use medication – associated to guidance on the relevance of treatment compliance and BP control – seen as the most appropriate approach[11,15].

Problems related to sample size are not uncommon when equivalence studies are analyzed in literature[34]. Wide samples are known to be required for minor differences between two treatments to be detected[35]. The present study had the objective to compare the equivalence between symptomatic and anti-hypertensive treatments while reducing BP levels and turning patients into asymptomatic. The study was based on a 100-patient sample. Although study sample was smaller than the one used for calculation, choice was made to interrupt data collection based on results found.

Based on sample used, a 13.9% difference was detected between the individuals that reached discharge criteria in the symptomatic medication group and in the captopril group. Such difference was not statistically significant, since δ value (difference between results ratio in treatment groups) previously adopted was 15%. The comparison between an anti-hypertensive drug and other drugs that do not include that property led authors to assume the possibility of some difference between treatments used that would be favorable to the group administered standard treatment as usually carried out at Emergency Units - captopril. The reason for data collection interruption at a given number of patients was based on the fact that the difference found was favorable to the group treated with symptomatic medication. The present study did not use Ethics Committees (EC). The reason was that study closings under study were not commonly the responsibility of this sort of committees, as for instance: fatal and non-fatal infarction, death, etc. However, based on results found, the recommendation for early interruption might have been made by such committees, should they have been resorted to.

The likeability of a bias – since researcher was not "blind" in the data collection stage – was minimized by strict compliance to study protocol in regard to drug administration regimen, as well as by the fact that the researcher would never have no information on the kind of patient that would look for assistance at an Emergency Unit, or on patient's clinical condition or time of visit.

Mean age (54.4 years old) found for population under study is consistent with most data found in literature, where age range is 50 to 54 years of age[5,20,21,33,36].

BP control evaluation studies have shown that women seem to be more aware of their condition. Therefore, thee are more treatment compliant[17,37]. Women have also been found to report a wider range of complaints when data on chronic, hypertensive patients lifestyle is being assessed[38]. Higher prevalence of women in data collected may be partially explained by those very reasons. Literature data on HBP prevalence do not agree in regard to gender. Some studies report prevalence for females and males; others report similar prevalence for both after 60 years of age, or discreet prevalence among women[4,18,23,36,39,40].

Literature reports HBP prevalence among black population, although in Brazil the ratio between black and white population was shown to be lower than the one reported for the United

Tabale II - Demographics and biological data on both treatment groups at admission HUOC; 2002

Variable	Treatment		p
	Symptomatic medication (n=51)	Anti-hypertensive medication (n=49)	
Age in years (mean±SD*)	52.6±14.0	56.3±13.7	0.18
Gender			0.78
Males	18 (35.3%)	16 (32.7%)	
Females		33 (64.7%)	33 (67.3%)
Race			0.42
White	11 (21.6%)	14 (28.6%)	
Non-white		40 (78.4%)	35 (71.4%)
BMI in Kg/m^2 (mean±SD)	28.9±5.4	29.0±5.4	0.94

*SD = mean±SD.

Table III - Clinical and therapeutic data for both treatment groups at admission, HUOC; 2002

Variable	Treatment		P
	Symptomatic medication (n=51)	Anti-hypertensive medication (n=49)	
HBP Diagnosis			0.77
Yes	46 (90.2%)	45 (91.8%)	
No		5 (9.8%)	4 (8.2%)
Time for diagnosis (in months) (mean±SD*)	97.6±98.8	131.0±111.0	0.097
SBP (mean±SD*)	184.2±13.9	188.6±14.3	0.12
DBP (mean±SD*)	106.7±16.5	107.6±10.0	0.75
MBP (mean±SD*)	132.6±11.7	134.6±8.1	0.31
Pharmacological treatment for HBP			0.65
Yes	35 (76.1%)	36 (80.0%)	
No		11 (23.9%)	9 (20.0%)
Compliance with pharmacological treatment			0.008
Yes	13 (28.3%)	25 (55.6%)	
No		33 (71.7%)	20 (44.4%)
Compliance with non-pharmacological treatment			1.00
Yes	1 (2.0%)	1 (2.0%)	
No	50 (98.0%)	48 (98.0%)	
Symptoms			0.293
Precordial pain	13 (25.5%)	8 (16.3%)	
Headache	13 (25.5%)	22 (45.0%)	
Dyspnea	10 (19.7%)	6 (12.2%)	
Dizziness	7 (13.7%)	7 (14.3%)	
Numbness			
Palpitations	4 (7.8%)	4 (8.2%)	
Shivering	4 (7.8%)	1 (2.0%)	
	0 (-)	1 (2.0%)	
Exercise			0.170
Yes	7 (13.7%)	12 (24.5%)	
No		44 (86.3%)	37 (75.5%)
Weight losing diet			0.539
Sim	8 (15.7%)	10 (20.4%)	
Não	43 (84.3%)	39 (79.6%)	
Low-sodium diet			0.097
Yes	33 (64.7%)	39 (79.6%)	
No		18 (35.3%)	10 (20.4%)
Active smoking			0.526
Yes	12 (23.5%)	9 (18.4%)	
No		39 (76.5 5)	40 (81.6%)

*SD = Standard deviation.

States[36]. Although the focus was not to subdivide patients into different ethnic groups - due to the difficulties that would be posed from Brazil's high miscinegation – prevalence showed to be among patients classified as non-white (75%).

Brazil data point out that obesity prevalence has increased in all socioeconomic layers, both for males and females. Obesity prevalence among hypertensive is considerably higher when compared to normotensives[41]. Having that in mind, average BMI (29 kg/m^2), as well as ratio of individuals classified as overweight and obese grade I in the population under study, seems to be in agreement with data referred in the literature[37].

As referred to by literature, most patients (91%) were aware of previous HBP diagnosis[4,8,11,21,42].

DBP and MBP were shown to be significantly higher among individuals with previous HBP diagnosis. Patients who are aware of their diagnosis, of their BP levels and of HBP complications (retinopathy and papiledema) have shown to more commonly report symptoms such as headache when their BP is increased[22,25,43]. That reinforces the relevance of such awareness when symptoms surface. The assumption that HBP diagnosis could influence BP additional increase at the moment of any discomfort that would have led to the Emergency Unit may explain such data. On the other hand, it is well known that blood pressure level control is still far from desired status among hypertensive patients[9,37,38,44-47], which in itself may partially contribute to explain study findings.

A significant number of hypertensive patients under study was

Table IV - Study patients following treatment groups and discharge criteria, HUOC; 2002

Treatment	Discharge criteria		Total
	Yes	No	
Symptomatic medication	30 (58.8%)	21 (41.2%)	51 (100.0%)
Anti-hypertensive medication	22 (44.9%)	27 (55.1%)	49 (100.0%)
Total	52 (52.0%)	48 (48.0%)	100 (100.0%)

p = 0,165.

Table V - Study patients following compliance with previous pharmacological treatment and discharge criteria, HUOC; 2002

Compliance with pharmacological treatment	Discharge criteria		Total
	Yes	No	
Yes	23 (60.5%)	15 (39.5%)	38 (100.0%)
No	28 (52.8%)	25 (47.2%)	53 (100.0%)
Total	51 (56.0%)	40 (44.0%)	91 (100.0%)

p = 0.466.

Table VI - Association between discharge criteria and some clinical and therapeutic variables - patients assisted at the Cardiology Emergency Unit - HUOC; 2002

Variable	Discharge criteria		p
	Yes	No	
HBP Diagnosis			0.192
Yes	51 (56.0%)	40 (44.0%)	
No	3 (33.3%)	6 (66.7%)	
Medication-based treatment			0.687
Yes	39 (54.9%)	32 (45.1%)	
No	12 (60.0%)	8 (40.0%)	
Physical exercise			0.374
Yes	11 (57.9%)	8 (42.1%)	
No	41 (50.6%)	40 (49.4%)	
Weight losing diet			0.707
Yes	9 (50.0%)	9 (50.0%)	
No	43 (52.4%)	39 (47.6%)	
Low-sodium diet			0.844
Yes	37 (51.4%)	35 (48.6%)	
No	15 (53.6%)	13 (46.4%)	
Active smoking			0.596
Yes	12 (57.1%)	9 (42.9%)	
No	40 (56.6%)	39 (49.4%)	

Table VII - Patients who were not discharged following reason for no-discharge and treatment received, HUOC; 2002

Reason for no-discharge	Treatment		Total
	Anti-hypertensive medication	15 (100.0%)	
High blood pressure	6 (40.0%)	24 (100.0%)	
Persistence of symptoms	11 (45.8%)	9 (100.0%)	
High blood pressure and Persistence of symptoms	4 (44.4%)	5 (55.6%)	
Total	21 (43.7%)	27 (56.3%)	48 (100.0%)

χ^2: p=0.937.

on pharmacological treatment (78%). Literature has also reported that most hypertensive patients assisted at Emergency Units with HC diagnosis are chronic hypertensive on anti-hypertensive therapy, although poorly controlled[8,21]. Prior do admission BP levels did not differ among hypertensives on regular anti-hypertensive drugs and those off drugs. Again, improper control of BP in hypertensive population - either for lack of dose adjustment or due to treatment compliance problems - may explain the lack of difference.

The most prevalent symptom in the present study – headache – has also been reported by many studies in the literature[4,5,14,18,19-22]. However, recent publications have tried to dissociate the symptom as being secondary to BP increase[23,25,43]. In the opinion of some authors, the association – already quite established - between headache and hypertensive encephalopathy and feochromocitome – must not be taken as evidence that headache is usually caused by simultaneous BP increase in mild to moderate HBP patients[25]. Other symptoms – such as chest pain, numbness, shivering, palpitations, and dizziness – have been described in literature as associated both to anxiety and HC conditions. Research with non-treated hypertensive patients has shown that increased frequency of subjective symptoms has been associated to BP increase. Such findings have also been reported by treated hypertensive patients, for whom DBP reduction was shown to be associated to increased well-being[38].

Both treatment groups reported similar characteristics when compared – as recommended when a clinical trial is carried out – except for previously established pharmacological treatment. However, results were not influenced by compliance, which is to say, no association was reported between compliance and discharge criteria. Patients who look for assistance at na Emergency Unit with increased BP symptoms may have skipped medication on previous day or on that very day. Medication interruption certainly interferes in BP control. Therefore, individual treatment approach is key, since resuming treatment is the best decision in such cases. The same cannot be said for hypertensives who regularly use medication, or for those without previously diagnosed hypertension. For that very reason, the definition adopted by the authors for pharmacological treatment compliance, while trying to find the explanation that leads increased BP patient to assistance,

was the use of anti-hypertensive – in the last 48 hours of Emergency Unit assistance as well. Therefore, only 53.5% of patients were classified as treatment compliant. Among those, a higher number was found in the groups assigned to be administered captopril (55.6%). No statistically significant difference was found for BP levels at admission, though, for individuals considered as compliant and non-compliant. Treatment compliance does not necessarily result in BP level control, as pointed out earlier. Other factors may be involved, such as proper dosing regimen, with the purpose of maximizing response and clinical control.

Mean of individuals reaching discharge criteria – in the symptomatic medication group – was 58.8%. In the captopril group, mean was 44.9%. As mean differences were not statistically significant (p=0.16), equivalence of effect could be assumed for the treatments. Efficacy ratio in regard to BP reduction – as reported in literature – for captopril are heterogeneous as a result of criteria adopted to define treatment success, ranging from 42% to 90%[48-55]. It is important to point out that most of the trials were carried out to evaluate the efficacy of captopril for the chronic treatment of HBP (outpatient units), in addition to no data availability on symptoms for discharge criteria. Data available are on BP levels only. Mansur and collaborators.[5] have shown that SBP and DBP percentual reduction with diazepan to be between de 10.1 and 7.7%, respectively, when comparing that drug to nifedipine, propranolol and nifedipine and propranolol combination. In the pilot study carried out by the authors[21], no statistically significant difference was reported for BP after 45 minutes either when symptomatic and anti-hypertensive drugs were administered, or in the percentage of patients that met discharge criteria. In both studies, discharge criteria did not include improvements of symptoms reported at admission.

When analyzing assistance to increased BP patients at the Emergency Unit it was found that anti-hypertensive management was the preference approach when DBP level was above 110 mmHg, even for asymptomatic patients. The same was not true for patients reporting increased DBP, although under 110 mmHg. For such cases, concern was lower towards BP levels, and closer attention was given to symptoms in regard to treatment[20]. No progress was found for the population under study as compared to admission at first re-evaluation point in time – 71.7% of patients[20]. Another retrospective study[14] showed that increased BP patients had distinctive treatment when assisted at the Cardiology Outpatient Unit or at the Emergency Unit. It was also found that BP levels were the parameter used for prescription at the Emergency Unit, whereas no patient assisted at the Outpatient Unit was administered any medication. That led the authors to the conclusion that a significant number of the medical community is unaware of HC and its implications.

The present study did not aim – as mentioned earlier – at comparing the efficacy of treatments used to reduce BP levels in regard to levels reached by each group at the end of the observation period, since an anti-hypertensive drug was compared to other drugs without those properties. As for pressure levels, the objective was – for both groups – to reduce levels under those considered as posing risk to patients. Those results suggest that the shift in focus – from figures, isolatedly, to improvement of symptoms associated to BP levels, which, although high, are off risk range - may produce satisfactory results with additional benefits, as pointed out earlier.

The likelihood or biased results – due to the fact that a higher number of individuals classified as compliant were randomized to the captopril group – might be based on the assumption that being compliant to previsously established pharmacological treatment, the absence of response to treatment administered at Emergency Unit results from the fact that those individuals are more severely hypertensive. However, the comparison between BP levels at admission in compliant and non-compliant individuals does not support such assumption, and therefore discards the likelihood of a bias. No statistically significant difference was found among individuals who were either compliant or non-compliant to chronic treatment of HBP either, in regard to meeting discharge criteria.

As for reaching discharge criteria, a similar number of individuals was reported for individuals with and without previous hypertension. The same was true for patients on or off Pharmacological treatment of HBP. Those data favor the argument that although information regarding previous HBP diagnosis, treatment and compliance are important – and therefore one should also take the opportunity for Emergency Unit assistance to emphasize the need for BP proper control – they do not seem to play a key role as determinants of BP increase at the Emergency Unit scenario. The analysis of the reasons and symptoms that lead patients to look for Emergency Unit assistance may provide more relevant information in regard to approach design.

Likewise, the lack of an association between non-pharmacological procedures when treating hypertension and fulfilling the discharge criteria may be justified. As referred to earlier, all those cofactors under analysis are recognizedly important in the evaluation and assessment of chronic treatment of HBP Considering those are measures that influence BP control in the mid and long term, they are of little impact on management at Emergency Units.

Most patients were discharged within 30 minutes of observation. Mean staying time at Emergency Unit was 52.5 minutes. No time difference was reported between the two groups. Such data suggest that, in both groups, in addition to the placebo effect, resting time at a calm setting out of the emergency room may explain higher incidence of discharge within 30 minutes, since captopril action onset varies from 15 to 30 minutes[3,13,56], and peak plasma concentration for the drugs used in the symptomatic group, from 30 minutes to 2 hours[17].

Among patients who were not discharged 50% kept the same symptoms as reported at admission, although BP had been lowered below pre-established levels. The number of patients reporting such status – having been treated with captopril – was similar to that of those who were administered symptomatic medication. The severity of symptoms and severity reduction level after treatment were not objects of the present study. Only the presence or absence of symptoms was investigated. Let us assume that a patient assisted at an Emergency Unit with a complaint of headache – he(she) considers severe – associated to BP increase received analgesic as treatment. At the re-assessment stage BP level is lower and headache has improved - now referred to as mild. The benefit of treatment administered is undeniable, even if symptoms have not ben fully eliminated. A detailed assessment protocol of such complaints at admission and progress after treatment – despite subjectivity in such assessment – may succeed in detecting some difference in favor of the group treated with symptomatic medication.

Among the individuals turned into asymptomatic, though, BP was kept increased. No statistically significant difference was reported between the groups. Here, group is identified where the diagnosis of pseudo hypertensive crisis is less likely, and a complaint-focused approach - although having succeeded in turning the patient asymptomatic - did not reduce BP levels. For those patients, a more detailed assessment at the outpatient unit, trying to identify the possible pathophysiologic mechanisms involved in BP increase, may give better guidance to the management of hypertension.

No difference was reported between the individuals who were administered anti-hypertensive or symptomatic medication for those who did not report improvement of symptoms or BP level reduction. The explanation provided could be applied to individual analysis of each patient who was not discharged in the latter group.

Results from the present study point towards the need for higher attention to symptoms referred by high BP patients assisted at Emergency Units. In addition to giving more humane assistance to patients, such attention will produce satisfactory, objective results. That implies making the medical community aware of the difference between hypertensive crises and those occurrences when BP increase could result from patients' symptoms.

Future clinical trials may disclose that this approach for hypertensive patient treatment may also reduce assistance costs and the side effets resulting from hypertensive administration, as well as improve doctor-patient interaction at Emergency Units.

References

1. IV Diretrizes Brasileiras de Hipertensão Arterial. Rev Bras Hipertens. 2002; 9: 359-408.
2. III Consenso Brasileiro de Hipertensão Arterial. Rev Bras Clin Ter. 1998; 24: 231-272.
3. Batlouni M. Os diuréticos efetivamente determinam alterações metabólicas que limitam seu uso no tratamento da hipertensão arterial? Hiperativo. 1998; 5: 62-6.
4. Zampaglione N, Pascale C, Marchisio M, Cavallo-Perin P. Hypertensive urgencies and emergencies. Prevalence and clinical presentation. Hypertension. 1996; 27: 144-7.
5. Mansur AP, Ramires JAF, Avakian SD, De Paula RS, Pileggi F. Efeito comparativo do diazepam, nifedipina, propranolol e da associação nifedipina e propranolol, por via sub-lingual, em pacientes com crise hipertensiva. Arq Bras Cardiol. 1991; 57: 313-7.
6. Bales A. Hypertensive crisis. How to tell if it´s an emergency or an urgency. Postgrad Med. 1999; 105: 119-30.
7. Epstein M. Diagnosis and management of hypertensive emergencies. Clin Cornerstone 1999; 2: 41-54.
8. Varon J, Marik PE. The diagnosis and management of hypertensive crises [review]. Chest 2000; 118: 214-27.
9. Blumenfeld JD, Laragh JH. Management of hypertensive crises: the scientific basis for treatment decisions. Am J Hypertens. 2001; 14(Pt 1): 1154-67.
10. Varon J, Fromm RE. Hypertensive crises. The need for urgent management. Postgrad Med. 1996; 99: 189-91, 196-6, 199-200.
11. Franco RJS. Crise hipertensiva: definição, epidemiologia e abordagem diagnóstica. Rev Bras Hipertens. 2002; 9: 340-5.
12. Almeida FA. Emergências hipertensivas: bases fisiopatológicas para o tratamento. Rev Bras Hipertens. 2002; 9: 346-52.
13. Praxedes JN, Santello JL, Amodeo C, Giorgi DMA, Machado CA, Jabur P. Encontro multicêntrico sobre crises hipertensivas – relatório e recomendações. J Bras Nefrol. 2001; 23 (Suppl III): 1-20.
14. Nobre F, Chauchar F, Viana JM, Pereira GJV, Lima NKC. Avaliação do atendimento do hipertenso em serviço de urgência e em ambulatório de hipertensão. Arq Bras Cardiol. 2002; 78: 156-8.
15. Grossman E, Ironi AN, Messerli FH. Comparative tolerability profile of hypertensive crisis treatments. Drug Saf. 1998; 19: 99-122.
16. The sixth report of the Joint National Committee on Detection, Evaluation and Diagnosis of High Bood Pressure (JNC VI). Arch Intern Med. 1997; 157: 2443-6.
17. Sigurdsson JA, Bengtsson C. Symptoms and signs in relation to blood pressure and antihypertensive treatment. A cross-sectional and longitudinal population study of middle-aged Swedish women. Acta Med Scand. 1983; 213: 183-90.
18. Moritz RD, Queiroz LP, Pereira MR, Scotinni MA. Estudo comparativo do uso da nifedipina e do captopril em urgências hipertensivas. Arq Bras Cardiol. 1989; 52: 323-6.
19. Gus M, Andrighetto AG, Balle VR, Pilla MB. Abordagem terapêutica de pacientes com queixa de pressão arterial elevada em um setor de emergência cardiológica. Arq Bras Cardiol. 1999; 72: 321-3.
20. Lima SG, Nascimento LS, Santos Filho CN, Patú RC, Luna MJC, Santos PCO. Atendimento de hipertensão arterial sistêmica no setor de emergência cardiológica. In: XII Congresso Pernambucano de Cardiologia; 2002, Ago; Recife, PE. Recife: Isa Pontual Design e Comunicação; 2002: 40.
21. Lima SG, Nascimento LS, Santos Filho CN, Carvalho LHF, Farias CM. Uso de sintomáticos no controle da pressão arterial elevada na unidade de emergência. Arq Bras Cardiol. 2002; 79 (suppl III): 30.
22. Weiss NS. Relation of high blood pressure to headache, epistaxis, and selected other symptoms. The United States Health Examination Survey of Adults. N Engl J Med. 1972; 287: 631-3.
23. Kruszewski P, Bieniaszewski L, Neubauer J, Krupa-Wojeiechowska B. Headache in patients with mild to moderate hypertension is generally not associated with simultaneous blood pressure elevation. J Hypertens. 2000; 18: 437-44.
24. Tavares A, Kohlmann Jr O. Tratamento da crise hipertensiva. Hiperativo. 1998; 5: 120-5.
25. Fernandes LC, Martins PD, Specialli JG, Gorayeb R, Coelho EB, Nobre F. Cefaléia e hipertensão: causa ou conseqüência? Rev Bras Hipertens. 2002; 9: 83-6.
26. Primeira Diretriz sobre Dor Torácica. Arq Bras Cardiol. 2002; 79 (suppl II): 1-22.
27. López M. Pressão arterial. In: López M, Medeiros JL. Semiologia Médica – As Bases do Diagnóstico Clínico. 3ª ed. Rio de Janeiro: Atheneu; 1990: 228-58.
28. Rodrigues CIS. Tratamento das emergências hipertensivas. Rev Bras Hipertens. 2002; 9: 353-8.
29. Almeida FA, Ribeiro AB, Marson O et al. Tratamento da crise hipertensiva com captopril. Arq Bras Cardiol. 1981; 37: 425-9.
30. Gifford Jr RW. Management of hypertensive crises. JAMA. 1991; 266: 829-35.
31. Fuchs FD. Nifedipina no tratamento da hipertensão arterial: o fim da controvérsia? Rev Bras Hipertens. 2001; 8: 230-3.
32. Fuchs FD, Lubianca Neto JF, Neves JM. Urgência e emergência hipertensivas. Arq Bras Cardiol. 1991; 56: 243-6.
33. Hirschl MM, Seidler D, Mullner M et al. Efficacy of different antihypertensive drugs in the emergency department. J Hum Hypertens. 1996; 10 (suppl III): 143.
34. Ware JH, Antman EM. Equivalence trials. N Engl J Med. 1997; 337: 1159-61.
35. Sample size. In: Friedman LM, Furberg CD, DeMets DL. Fundamentals of Clinical Trials. 3th edition. New York: Springer-Verlag New York, Inc; 1998: 94-129.
36. Bennet NM, Shea S. Hypertensive emergency: case criteria, sociodemographic profile and previous care of 100 cases. Am J Public Health. 1998; 78: 636-40.
37. Freitas JB, Tavares A, Kohlmann Jr O, Zanella MA, Ribeiro AB. Estudo transversal sobre controle da pressão arterial no Serviço de Nefrologia da Escola Paulista de Medicina – UNIFESP. Arq Bras Cardiol. 2002; 79: 117-22.
38. Zyczynshi TM, Coyne KS. Hipertensão e questões sobre adesão ao tratamento e resposta dos pacientes. Current Hypertension Reports Brasil. 2001; 1: 11-6.
39. Dondici Filho J, Gomes JC, Castro EG, Luz NST, Abzaid A. Redução aguda da pressão arterial: estudo comparativo entre nifedipina e clonidina. Arq Bras cardiol. 1991; 56: 127-30.
40. Vaughan CJ, Delanty N. Hypertensive emergencies. The Lancet. 2000; 356: 411-7.
41. Ferreira SRG, Zanella MT. Epidemiologia da hipertensão arterial associada à obesidade. Rev Bras Hipertens. 2000; 7: 128-35.
42. Rodriguez MC, Mateos FH, Fernandez CP, Martell NC, Luque MO. Hypertensive crises: prevalence and clinical aspects. Rev Clin Esp. 2002; 202: 255-8.
43. Pickering T. Headache and hypertension – something old, something new. J Clin Hypertens. 2000; 2: 345-7.
44. Ramsay LE, Williams B, Johnston GD et al. Guidelines for management of hypertension: report of the third working party of the British Hypertension Society. J Hum Hypertens. 1999; 13: 569-92.
45. Olmos RD, Lotufo PA. Epidemiologia da hipertensão arterial no Brasil e no mundo. Rev Bras Hipertens. 2002; 9: 21-3.
46. Stevens VJ, Obarzanek E, Cook NR et al. Trials for the hypertension prevention research group. Long-term weight loss and changes in blood pressure: results of the trials of hypertension prevention, phase II. Ann Intern Med. 2001; 134: 1-11.
47. Payne KA, Esmonde-White S. Estudos de observação sobre uso e adesão à medicação anti-hipertensiva: a escolha dos medicamentos é um fator na adesão ao tratamento? Current Hypertension Reports Brasil. 2001; 1: 17-27.
48. Angeli P, Chiesa M, Caregaro L et al. Comparison of sublingual captopril and nifedipine in immediate treatment of hypertensive emergencies. Arch Intern Med. 1991; 151: 678-82.
49. Materson BJ, Reda DJ, Cushman WC et al. Single-drug therapy for hypertension in men. A comparison of six antihiypertensive agents with placebo. N Engl Med. 1993; 328: 914-21.
50. The Badajoz cooperative study. The Badajoz Cooperative Group. Captopril in a single daily dose in arterial hypertension in the elderly. An Med Interna. 1993; 10: 119-22.
51. Ferrara LA, Marino Di L, Russo O, Marotta T, Mancini M. Doxazosin and captopril

in mildly hypercholesterolemic hypertensive patients. The doxazosin-captopril in hypercholesterolemic hypertensives study. Hypertension. 1993; 21: 97-104.
52. Mancia G, Buoninconti R, Errico M, Freda M, Giordano MP, Grana Q. Efficacy and tolerability of nicardipine retard and captopril in hypertension in the aged. Results of a multicenter study. Minerva Med. 1992; 83: 731-8.
53. Franco RJ, Curi PR, Khlman JO et al. O captopril na hipertensão arterial leve a moderada resistente ao tratamento com diurético. Um estudo multicêntrico. Arq Bras Cardiol. 1992; 58: 237-42.
54. Tuck ML, Katz LA, Kirkendall WM, Koeppe PR, Ruoff GE, Sapir DG. Low-dose captopril in mild to moderate geriatric hypertension. J Am Geriatr Soc. 1986; 34: 693-6.
55. Frishman WH, Greenberg S. Angiotensin converting enzyme inhibitors as initial monotherapy in severe hypertension. Quinapril and captopril. Am J Hypertens. 1991; 4(Pt 1): 827-31.
56. Blumenfeld JD, Laragh JH. Management of hypertensive crises: the scientific basis for treatment decisions [review]. Am J Hypertns. 2001; 14: 1154-67.