
From the American College of Emergency Physicians Clinical Policies Subcommittee (Writing Committee) on Asymptomatic Hypertension in the ED:
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Approved by the ACEP Board of Directors, September 23, 2005

INTRODUCTION
Hypertension is ubiquitous. It affects approximately 50 million individuals in the United States and 1 billion individuals worldwide. Hypertension accounts for 35 million office visits in the United States, making it the most common primary diagnosis, yet 30% of those with this condition are unaware of their condition.2,3

The health risks caused by prolonged untreated hypertension are serious. As noted in the recent report by the Joint National Committee on Prevention, Detection, Evaluation and Treatment of High Blood Pressure (JNC 7 Report) for individuals aged 40 to 70 years, each increment of 20 mm Hg
in systolic blood pressure or 10 mm Hg in diastolic blood pressure doubles the risk of cardiovascular disease events independent of other factors.\textsuperscript{1-4} JNC 7 defined an important new category in the spectrum of blood pressure assessment, termed “prehypertension,” which includes individuals with a systolic blood pressure of 120 to 139 mm Hg or a diastolic blood pressure of 80 to 89 mm Hg. These patients are at twice the risk of developing hypertension as those with values below this range, highlighting the need for careful screening in the primary care setting.\textsuperscript{5}

The definitions of hypertension used in this report are those developed by JNC 7 (see Table),\textsuperscript{1} and include normal, prehypertension, stage 1 hypertension, and stage 2 hypertension. Acute hypertensive emergencies are not addressed by this policy.

DEFINITIONS

The definitions of hypertension used in this report are those developed by JNC 7 (see Table),\textsuperscript{1} and include normal, prehypertension, stage 1 hypertension, and stage 2 hypertension. Acute hypertensive emergencies are not addressed by this policy.

METHODOLOGY

This clinical policy was created after careful review and critical analysis of the peer-reviewed literature. A MEDLINE search of English-language articles published between January 1992 and January 2005 was performed using combinations of the key words “hypertension” and “emergency department.” Terms were then exploded as appropriate. Abstracts and articles were reviewed by subcommittee members, and pertinent articles were selected. These articles were evaluated, and those addressing the questions considered in this document were chosen for grading. Subcommittee members also supplied references from bibliographies of initially selected articles or from their own files. Expert peer reviewers supplied articles with direct bearing on this policy.
The reasons for developing clinical policies in emergency medicine and the approaches used in their development have been enumerated. This policy is a product of the American College of Emergency Physicians (ACEP) clinical policy development process, including expert review, and is based on the existing literature; where literature was not available, consensus of emergency physicians was used. Expert review comments were received from individual emergency physicians as well as individual members of the American College of Physicians, American Society of Hypertension, American Society of Nephrology, and Emergency Nurses Association. Their responses were used to further refine and enhance this policy. Clinical policies are scheduled for revision every 3 years; however, interim reviews are conducted when technology or the practice environment changes significantly.

All publications were graded by at least 2 of the subcommittee members into 1 of 3 categories of strength of evidence. Some articles were downgraded on the basis of a standardized formula that considers the size of study sources of bias (Appendix A).

During the review process, all articles were given a baseline “strength of evidence” by the subcommittee members according to the following criteria.

**Strength of evidence Class I**—Interventional studies including clinical trials, observational studies including prospective cohort studies, aggregate studies including meta-analyses and randomized clinical trials only.

**Strength of evidence Class II**—Observational studies including retrospective cohort studies, case-controlled studies, aggregate studies including other meta-analyses.

**Strength of evidence Class III**—Descriptive cross-sectional studies, observational reports including case series and case reports, consensus studies including published panel consensus by acknowledged groups of experts.

Strength of evidence Class I and II articles were then rated on elements subcommittee members believed were most important in creating a quality work. Class I and II articles with significant flaws or design bias were downgraded on the basis of a set formula (Appendix B). Strength of evidence Class III articles were downgraded if they demonstrated significant flaws or bias. Articles downgraded below strength of evidence Class III were given an “X” rating and were not used in formulating recommendations in this policy. An Evidentiary Table was constructed and is included in this policy.

Recommendations regarding patient management were then made according to the following criteria:

**Level A recommendations.** Generally accepted principles for patient management that reflect a high degree of clinical certainty (ie, based on strength of evidence Class I or overwhelming evidence from strength of evidence Class II studies that directly address all of the issues).

**Level B recommendations.** Recommendations for patient management that may identify a particular strategy or range of management strategies that reflect moderate clinical certainty (ie, based on strength of evidence Class II studies that directly address the issue, decision analysis that directly addresses the issue, or strong consensus of strength of evidence Class III studies).

**Level C recommendations.** Other strategies for patient management based on preliminary, inconclusive, or conflicting evidence, or in the absence of any published literature, based on panel consensus.

There are certain circumstances in which the recommendations stemming from a body of evidence should not be rated as highly as the individual studies on which they are based. Factors such as heterogeneity of results, uncertainty about effect magnitude and consequences, strength of prior beliefs, and publication bias, among others, may lead to such a downgrading of recommendations.

**Scope of Application.** The guideline is intended for physicians working in hospital-based EDs.

**Inclusion Criteria.** This clinical policy is intended for ED patients older than 18 years.

**Exclusion Criteria.** Excluded from this policy are patients presenting to the ED with acute hypertensive emergencies. Also excluded are individuals with acute presentation of conditions known to be caused by hypertension such as strokes, myocardial infarction, and new-onset renal dysfunction.

**CRITICAL QUESTIONS**

1. Are ED blood pressure readings accurate and reliable for screening asymptomatic patients for hypertension?

The association of hypertension with poor long-term health outcomes is well established. However, the issue of how to approach the incidental finding of asymptomatic hypertension in the ED remains a quandary. On 1 hand, the patient and the emergency physician are brought together to address that patient’s emergency complaint, not long-term health maintenance issues, which are generally believed by both the patient and provider to be beyond the scope of an ED visit. On the other hand, failing to recognize and address hypertension in the ED may represent a missed opportunity to avert catastrophic health events. Confounding this issue is the concern that blood pressure elevation in the ED may be an aberrancy attributed to the ED environment and the patient’s own pain and/or anxiety.

When confronted with an elevated blood pressure reading in a patient who is otherwise asymptomatic, the emergency physician must determine whether the reading is accurate and reliable. Patients are often apprehensive or in pain, and these variables may affect the blood pressure readings. If elevated blood pressure in the ED represents inadequately treated hypertension, the emergency physician may have an opportunity to discuss these findings with the patient and ensure close follow-up with a primary physician.

Clement et al, in a prospective multicenter observational study with 1,963 patients comparing ambulatory blood pressure measurements and the frequency of adverse cardiovascular
events, demonstrated an association between elevated baseline ambulatory blood pressure and adverse outcome. The study assessed the association of hypertension with endpoints of stroke, myocardial infarction, sudden death, new angina pectoris, congestive heart failure, and peripheral vascular disease. Patients with elevated ambulatory blood pressure were more likely to have adverse events within a mean follow-up period of 5 years. Unfortunately, the conclusions of this well-designed trial have limited applicability to the ED setting because the study was performed in an office or ambulatory setting where variables of pain and anxiety are less prevalent. Furthermore, all patients received 3 blood pressure readings during separate office visits, which is not feasible in the ED setting.

Three studies were identified by our literature search that address the question of whether elevated blood pressure readings obtained in the ED are reproducibly elevated in follow-up.8-10 Backer et al8 followed 407 ED patients with elevated blood pressure and no previous diagnosis of hypertension. The degree of blood pressure elevation was classified according to JNC VI criteria (stage I, II, or III). The investigators found that the proportion of patients with at least 1 elevated blood pressure measurement in follow-up increased with increasing stage of initial blood pressure. Chernow et al9 prospectively identified 239 patients with systolic blood pressures greater than 159 mm Hg or diastolic blood pressures greater than 94 mm Hg. Of those patients referred for follow-up, 35% were found to be hypertensive, 33% had borderline hypertension, and 32% had normal blood pressure readings on follow-up. Slater et al10 also demonstrated a correlation between elevated blood pressure in the emergency setting and elevated blood pressure in follow-up. How many blood pressure readings should be obtained before the measurements are considered adequate for screening purposes? According to JNC 7, at least 2 measurements of blood pressure should be obtained in the office setting after the patient has been sitting quietly in a chair for at least 5 minutes.1

These recommendations are made for purposes of diagnosing hypertension in the outpatient setting. Such controlled conditions are difficult to reproduce in an ED. However, evidence from several studies suggests that 2 separate blood pressure measurements in the ED setting are adequate for screening patients with elevated blood pressure readings.11-13 Mamon et al11 developed a protocol to improve hypertension detection and referral in the ED. Of the 203 patients enrolled, 71 patients had an elevated initial blood pressure measurement. Although the protocol involved 3 separate blood pressure measurements, post hoc analysis revealed that 68 of 71 hypertensive patients would have been detected if only 2 blood pressure measurements were obtained. Edmonds et al12 prospectively followed 140 ED patients who had vital signs measured by 2 independent observers. These investigators found a mean difference between observers of 1.3 mm Hg and an expected range of agreement of 24.2 mm Hg in systolic blood pressure, with similar findings for diastolic blood pressure. This study highlights that interobserver variability may limit the reproducibility of vital sign measurements. Pitts and Adams13 demonstrated a spontaneous decline in diastolic blood pressure on repeat measurements and postulated that an initial “alerting reaction” may contribute to a high initial blood pressure reading. Overall, the evidence suggests that in the ED, 2 blood pressure measurements are adequate for screening purposes.

The technique for measuring blood pressure is important. For years, direct intraarterial measurement of blood pressure has been the criterion standard. Because this measurement is impractical in the emergency setting, blood pressure determination by the auscultatory method using a mercury sphygmomanometer has been the traditional method of measurement against which other methods are compared.1,14 Borow and Newburger15 demonstrated that automated oscillometric determination of blood pressure reliably approximates central aortic pressure. However, blood pressure readings from commercial oscillometric devices may vary significantly from readings obtained by the auscultatory method. Park et al16 obtained blood pressure measurements from 7,208 school-aged children from 5 to 17 years of age. On average, systolic oscillometric readings were 10 mm Hg higher than systolic readings obtained with the auscultatory method. Diastolic readings obtained with the oscillometric method were on average 5 mm Hg higher than readings obtained with the auscultatory method. Because of variability in blood pressure readings obtained by commercial oscillometric devices and the need for quality and precision in these devices, those who oversee equipment purchases should know whether the oscillometric device used in their ED meets the Association for the Advancement of Medical Instrumentation standards.17

1. Patient Management Recommendations: Are ED blood pressure readings accurate and reliable for screening asymptomatic patients for hypertension?
   **Level A recommendations.** None specified.
   **Level B recommendations.** If blood pressure measurements are persistently elevated with a systolic blood pressure greater than 140 mm Hg or diastolic blood pressure greater than 90 mm Hg, the patient should be referred for follow-up of possible hypertension and blood pressure management.
   **Level C recommendations.** Patients with a single elevated blood pressure reading may require further screening for hypertension in the outpatient setting.

2. Do asymptomatic patients with elevated blood pressures benefit from rapid lowering of their blood pressure?
   There is little data that directly addresses the need for rapid lowering of elevated blood pressure in the ED. Much of the published research is on long-term trials and does not address the impact of acute management in asymptomatic patients. The VA Cooperative Trial of 1967, a randomized placebo-controlled trial of 143 patients with diastolic blood pressure of 115 to 130 mm Hg, demonstrated no adverse outcomes with treatment versus placebo during the initial 3 months of study.18 Suggestive of the need for definitive follow-up, 4 patients did develop...
significant complications within 4 months of enrollment, including sudden death, ruptured aortic aneurysm and death, severely elevated blood urea nitrogen level, and congestive heart failure. Another prospective randomized controlled trial compared the blood pressure response in 74 asymptomatic patients who had diastolic blood pressure 116 to 139 mm Hg and were receiving oral clonidine loading followed by maintenance dosing versus initiating therapy with maintenance dosing.19 Patients given repeated doses of clonidine were required to have a diastolic blood pressure decrease by 20 mm Hg or fall to a diastolic blood pressure of 105 mm Hg before discharge. All other patients received only the initial oral dosing followed by daily maintenance therapy. There was no clinically important difference in blood pressure response or clinical outcome between study groups during 7 days.19 There are a number of case studies and case reports of patients with poor outcomes, including hypotension, myocardial ischemia and infarction, strokes, and death, precipitated by rapidly lowering elevated blood pressures in asymptomatic patients.20-24 Many of these events were associated with the use of nifedipine.

A 1990 study described a 6% decrease in mean arterial blood pressures of 54 ED patients with asymptomatic hypertension with initial diastolic blood pressures greater than 90 mm Hg before pharmacologic therapy.25 Interestingly, when these patients were subdivided into diastolic pressures between 90 mm Hg and 114 mm Hg (n=22) and those greater than 115 mm Hg diastolic (n=32), only the latter group demonstrated a significant decrease with either the systolic and diastolic blood pressures or the mean arterial blood pressures. More recently, a study attempted to describe the causality for blood pressure improvement in the ED patient population not receiving pharmacologic intervention. This retrospective medical record review of 195 patients demonstrated that a majority of patients with diastolic blood pressure greater than 90 mm Hg declined spontaneously on a second measurement during the same visit. A mean decline of 11.6 mm Hg in diastolic blood pressure was observed, with regression to the mean explaining 7.1 mm Hg of this change.13 Because regression to the mean exhibits greater impact on results at extremes of measurement, the authors conclude that emergency physicians must be aware of this phenomenon to avoid potential pitfalls with unnecessary therapy. They propose that physicians average repeated observations before initiating any interventions.

The data on the utility of screening patients with asymptomatic hypertension for end-organ damage in the ED is limited. Asymptomatic patients with elevated blood pressures may be screened to identify evidence of end-organ damage, including microvascular injury manifested by retinal changes, left ventricular hypertrophy, and renal injury. These patients may have improved long-term outcomes from antihypertensive treatment aimed at gradually lowering their blood pressure.1,3,18,20 A focused history and physical examination can detect signs and symptoms of end-organ damage that may not be readily apparent. Neurologic, cardiac, and renal reviews of systems can reveal signs not present in the patient’s chief complaint, such as subtle vision changes, mild confusion, dyspnea on exertion, and oliguria. The physical examination may include neurologic, fundoscopic, and cardiovascular examinations. In addition to the history and physical examination, screening for target organ damage in an asymptomatic patient may include urinalysis, serum creatinine level, ECG, and chest radiography. A search for evidence to validate each of these screening tests revealed little data, but relevant studies are reviewed here. At least 1 study has suggested that a negative urine dip stick test result for both protein and hematuria in 143 ED patients with hypertension ruled out an acute elevation in creatinine level (sensitivity 100%; 95% confidence interval [CI] 83% to 100%). Although the sensitivity of this test was 100%, the wide CI creates some question in using this test alone as a screening tool to identify acute renal damage.20 In a 1978 medical record review by Bartha and Nugent,27 116 patients with hypertension had routine chest radiographs and ECGs. Only 2 of the 116 patients had therapeutic or diagnostic interventions based on the chest radiograph or ECG, and none influenced hypertensive management. Bartha and Nugent concluded that routine chest radiographs and ECGs could not be defended in the workup of hypertension.

The VA Cooperative Trial18 demonstrated that 27 (39%) of 70 patients treated with placebo and 2 (3%) of 73 patients treated with antihypertensive drugs experienced adverse events within 20 months (absolute risk reduction 36%, 95% CI; number needed to treat = 3). However, there were no adverse events in either group within the first 3 months of treatment versus placebo. Without the presence of acute end-organ damage, no literature demonstrated that patients who received pharmacologic intervention in the ED had better outcomes than those referred for repeat blood pressure measurements, subsequent screening for end-organ damage, and treatment.18-22,28

JNC VI made the statement, “Elevated blood pressure alone, in the absence of symptoms or new or progressive target organ damage, rarely requires emergency therapy.”3 Of note, JNC 7 recommendations state that marked elevations of blood pressure without target organ damage “usually do not require hospitalization, but should receive immediate combination oral antihypertensive therapy.”21 This statement appears to be consensus in origin because no supportive references were cited. Furthermore, the term “immediate” is used in the context of an outpatient setting and not to acute ED management. We could find no evidence demonstrating improved patient outcomes or decreased mortality or morbidity with acute management of elevated blood pressure in the ED.

There is no evidence that clearly delineates appropriate ED management of patients with asymptomatic hypertension. Despite this lack of evidence, providers sometimes feel compelled to initiate treatment. However, before initiating this therapy, the treating physician should be aware that as many as
one third of patients with diastolic blood pressures greater than 95 mm Hg on initial ED visit have been found to normalize before arranged follow-up. When follow-up is available, the emergency physician may provide the greatest benefit to the patient by identifying the patient at risk with an elevated blood pressure and advising them to arrange prompt and definitive follow-up with their primary physician.

2. Patient Management Recommendations: Do asymptomatic patients with elevated blood pressures benefit from rapid lowering of their blood pressure?

**Level A recommendations.** None specified.

**Level B recommendations.**

1. Initiating treatment for asymptomatic hypertension in the ED is not necessary when patients have follow-up.

2. Rapidly lowering blood pressure in asymptomatic patients in the ED is unnecessary and may be harmful in some patients.

3. When ED treatment for asymptomatic hypertension is initiated, blood pressure management should attempt to gradually lower blood pressure and should not be expected to be normalized during the initial ED visit.

**Level C recommendations.** None specified.

**REFERENCES**


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<tr>
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<tr>
<td>Chobanian et al (JNC 7 report)</td>
<td>2003</td>
<td>Review, consensus</td>
<td>Review of current literature on prevention, detection, evaluation, and treatment of high blood pressure; recommendations based on available literature and expert consensus</td>
<td>Expert consensus and recommendations</td>
<td>New blood pressure classification (see Table): goals of therapy: (&lt;140/90), (&lt;130/80) if patient has diabetes or renal disease; hypertensive urgencies or emergencies with target organ damage require IV therapy and hospitalization; marked elevation of blood pressure without target organ damage usually does not require hospitalization but per the report should receive immediate combination oral antihypertensive therapy</td>
<td>Consensus/review: no references given to support the combination of oral therapy for markedly elevated blood pressure without target organ damage</td>
<td>III</td>
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<tr>
<td>Clement et al</td>
<td>2003</td>
<td>Prospective multicenter observational</td>
<td>Sphygmomanometric blood pressures were obtained in patients with known hypertension in both the office and ambulatory setting</td>
<td>Cardiovascular events, including stroke, myocardial infarction, sudden death, new angina pectoris, congestive heart failure, or peripheral vascular disease</td>
<td>Both office and ambulatory measurements of systolic and diastolic blood pressure significantly predicted the primary endpoint of fatal or nonfatal cardiovascular events</td>
<td>Blood pressures were not obtained in the emergency setting, thus variables such as acute pain are unlikely to affect measurements; 3 blood pressure readings were obtained on separate visits, which is not feasible in an emergency setting</td>
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<tr>
<td>Backer et al</td>
<td>2003</td>
<td>Prospective cohort</td>
<td>407 patients with elevated blood pressure measurement in the ED and no previous diagnosis of hypertension were classified according to JNC criteria (stage I, II, or III) and prospectively followed</td>
<td>Identification of patients who had at least 1 abnormal blood pressure measurement recorded in follow-up</td>
<td>The proportion of patients with at least 1 abnormal measurement in follow-up increased with increasing stage of initial blood pressure; pain as a chief complaint was unrelated to likelihood of having an elevated blood pressure in follow-up</td>
<td>Only 65% of patients had repeat blood pressure measurements at follow-up: the number of repeat blood pressure measurements was variable; the authors concluded that patients with a single elevated blood pressure measurement in the ED are at risk for diagnosis of primary hypertension and should be referred for follow-up evaluations</td>
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<td>Chernow et al⁹</td>
<td>1987</td>
<td>Prospective observational</td>
<td>All patients admitted to the ED of a university hospital over a 1-y period were prospectively screened for hypertension; a total of 239 patients were identified as having an elevated blood pressure</td>
<td>An elevated blood pressure was defined as a measurement of &gt;159/94 mm Hg</td>
<td>Of the 239 patients enrolled, follow-up was obtained in 107 (45%) of cases; overall, 35% of patients referred for follow-up were found to be hypertensive, 33% had borderline hypertension, and 32% had normal blood pressure measurements on follow-up</td>
<td>Follow-up information was available in only 45% of patients; the authors concluded that the ED may be a useful environment to screen for hypertension; patients found to have an elevated blood pressure should be referred for follow-up evaluation and management</td>
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<td>Slater et al¹⁰</td>
<td>1987</td>
<td>Retrospective observational</td>
<td>2,000 medical records were reviewed; 60 patients with diastolic blood pressures &gt;95 mm Hg were called back to the ED to have blood pressure checks in a quiet environment; 15 patients had elevated diastolic blood pressure measurements that were communicated to the primary physician</td>
<td>Pharmacologic treatment of hypertension initiated by the primary physician after independent assessment in follow-up</td>
<td>14 of 15 patients with elevated diastolic blood pressure measurements on a return visit to the ED had blood pressure treatment initiated by the primary physician</td>
<td>Retrospective observational design; small sample size of 60 patients; a single, elevated blood pressure reading may be a useful indicator of hypertension and warrants follow-up with the primary care physician</td>
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<td>Mamon et al¹¹</td>
<td>1987</td>
<td>Prospective observational</td>
<td>203 patients seen in the nonurgent area of an ED were enrolled; inclusion criteria were an initial blood pressure measurement ñ140/90 if &lt;50 y of age and ñ160/95 if ≥50 y of age; if the initial blood pressure taken by a triage nurse was elevated, then 2 repeat blood pressure measurements were obtained by an EMT and the patient was asked whether he or she: (1) had a history of hypertension, (2) had been treated in the past year for hypertension, or (3) had a usual source of medical care</td>
<td>Elevated blood pressure was defined as blood pressure ≥140/90 if &lt;50 y of age and ≥160/95 if ≥50 y of age; this differed from JNC criteria at the time the study was performed</td>
<td>Of the 203 patients enrolled, 71 had an elevated initial blood pressure measurement; in post hoc analysis, 68 of the 71 patients would have been detected if only 2 blood pressure measurements were performed; results of the EMT interview were compared with patient records and found to be 90%-100% sensitive and 79%-96% specific</td>
<td>No outcome measures were identified (eg, percentage of hypertensive patients who were referred for follow-up before and after implementation of the study protocol); The study findings suggest that the ED may be a useful site for high blood pressure screening</td>
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<td>Study</td>
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<td>Edmonds et al¹²</td>
<td>2002</td>
<td>Prospective observational</td>
<td>140 ED patients had vital sign measurements obtained by 2 independent observers; the vital sign measurements were analyzed to determine the degree of variability</td>
<td>The mean value of each vital sign and degree of vital sign variability as determined by Bland-Altman statistics, mean difference between observers and expected range of agreement</td>
<td>The mean systolic blood pressure of 127.1 mm Hg was associated with a mean difference between observers of 1.3 mm Hg (1.0%) and an expected range of agreement of ±24.2 mm Hg (±19%); the mean diastolic blood pressure of 77.4 mm Hg had a mean difference between observers of 0.3 mm Hg (0.4%) and an expected range of agreement of ±19.9 mm Hg (±25.7%)</td>
<td>Small sample size; the authors concluded that significant interobserver variability may limit the reproducibility of vital sign measurements</td>
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<td>Pitts and Adams¹³</td>
<td>1998</td>
<td>Retrospective observational</td>
<td>2 ED patient groups in which at least 2 vital sign measurements were obtained were selected for study; in group 1, 76 of 220 consecutive medical records that included at least 2 vital sign measurements were selected; group 2 included 125 consecutive patients with hypertension (diastolic blood pressure &gt;90 mm Hg)</td>
<td>Mean, SD, and calculated versus observed regression to the mean of repeat vital sign measurements</td>
<td>The mean diastolic blood pressure in the patient sample was 78.3 mm Hg, with a SD of 17.9 mm Hg; from this difference, a correlation coefficient of 0.73 was calculated, predicting a spontaneous decline of 7.3 mm Hg between repeat blood pressure measurements; a decline of 11.6 mm Hg was observed in the hypertensive group, substantiating the concept of regression to the mean</td>
<td>Retrospective design; the study supports the concept that most patients presenting to the ED with asymptomatic blood pressure will demonstrate a spontaneous decrease in blood pressure; a significant portion of this effect may be explained by regression to the mean</td>
<td>II</td>
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<tr>
<td>Park et al¹⁶</td>
<td>2001</td>
<td>Prospective observational</td>
<td>7,208 school-aged children aged 5-17 y had blood pressure measurements obtained by the auscultatory method and oscillometric method (Dinamap model 8100; Critikon, Tampa, FL)</td>
<td>The auscultatory method of blood pressure determination with a mercury sphygmomanometer</td>
<td>Oscillometric blood pressure readings were 10 mm Hg higher (95% CI –4 to 24 mm Hg) than auscultatory systolic pressure readings; diastolic oscillometric readings were 5 mm Hg higher (95% CI –14 to 23 mm Hg) than auscultatory diastolic pressure readings</td>
<td>Although a single proprietary oscillometric device was used, the sample size of 7,208 enhances the strength of the study</td>
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<tr>
<th>Study</th>
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<tr>
<td>Frein et al (VA Cooperative Study Group)</td>
<td>1967</td>
<td>Randomized placebo-controlled trial</td>
<td>143 patients with diastolic blood pressure 115-129 mm Hg were randomized to treatment with antihypertensive drugs versus placebo; adverse events were followed during the next 20 months</td>
<td>Adverse events during 20 months</td>
<td>27 (39%) of patients treated with placebo and 2 (3%) of 73 patients treated with antihypertensive drugs experienced adverse events within 20 months; however, there was no difference in adverse events between the 2 groups during the first 3 months (95% CI)</td>
<td>This study finds that without initiation of therapy for hypertension, the risk of adverse events significantly increases after 3 months; importantly, the authors found no difference in outcomes during the first 3 months after study enrollment either with or without therapy</td>
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<td>Zeller et al</td>
<td>1989</td>
<td>Randomized controlled trial</td>
<td>Randomized controlled trial of 74 asymptomatic patients with diastolic blood pressure of 116-139 mm Hg with no antihypertension treatment during the past 3 days; all patients received 0.2 mg clonidine and then were allocated to 3 arms: 0.1 mg q 1 h x 4 or placebo q 1 h x 4 until decrease of 20 mm Hg or diastolic blood pressure &lt;105, or discharged home; all were discharged on clonidine and chlorthalidone depending on response; follow-up done in 1, 2, 3, and 7 days</td>
<td>Difference in blood pressure on follow-up between the 3 groups</td>
<td>No clinically significant difference in blood pressure response in all 3 groups</td>
<td>64 patients made 1-day follow-up, 44 patients made 1-week follow-up; number lost to follow-up reportedly equal across groups</td>
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<td>Shayne and Pitts</td>
<td>2003</td>
<td>Literature review</td>
<td></td>
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<td>Limited by design; the authors conclude that there is a lack of evidence to support treatment of asymptomatic hypertension in the ED</td>
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<td>Gallagher</td>
<td>2003</td>
<td>Brief commentary and literature review</td>
<td></td>
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<td></td>
<td>Limited by design; conclusion that treatment of asymptomatic hypertension does not benefit the patient and may increase the risk of harm</td>
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<th>Outcome Measure/Criterion Standard</th>
<th>Results</th>
<th>Limitations/Comments</th>
<th>Class</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ram^22</td>
<td>1995</td>
<td>Literature review</td>
<td></td>
<td></td>
<td></td>
<td>Limited by design; severe asymptomatic hypertension (diastolic blood pressure 130-140 mm Hg) does not require acute management with parenteral agents in the ED; rapid lowering of severe asymptomatic hypertension may be harmful</td>
<td>III</td>
</tr>
<tr>
<td>Wachter^23</td>
<td>1987</td>
<td>Case series</td>
<td>Retrospective review of 51 patients treated for hypertension identified 3 cases of morbid events as a result of nifedipine administration</td>
<td>Onset of new clinical finding after nifedipine administration</td>
<td>After oral nifedipine: 1 patient developed hypotension, acute mental status change, and ECG changes; 1 patient developed dizziness, nausea, and ECG changes; 1 patient developed epigastric pain, dizziness, nausea, diaphoresis, and ECG changes</td>
<td>Limited by design; the authors express concern over multidose delivery of nifedipine in patients with elevated blood pressure</td>
<td>III</td>
</tr>
<tr>
<td>O’Mailia et al^24</td>
<td>1987</td>
<td>Case report</td>
<td>3 cases are described that demonstrate clinical sequelae, including cardiac ischemia and infarct after administration of nifedipine to patients with hypertensive urgency</td>
<td>Onset of new clinical finding after nifedipine administration</td>
<td>After oral nifedipine: 1 patient developed hypotension, chest pain, and ECG changes consistent with ischemia; 2 patients developed chest pain, hypotension, and ECG changes and cardiac enzymes consistent with acute myocardial infarction</td>
<td>Limited by design; the authors suggest that nifedipine be used with caution on patients at risk for cardiac ischemia</td>
<td>III</td>
</tr>
<tr>
<td>Lebby et al^25</td>
<td>1990</td>
<td>Retrospective cohort</td>
<td>54 records met inclusion criteria of 94 candidates with diagnosis of hypertension or hypertensive urgency; repeat blood pressure measurements were taken an average of 51.5 minutes apart; patients could not have received drug intervention before the repeat measurement</td>
<td>Repeat blood pressure measurement</td>
<td>Systolic and diastolic blood pressure decreased an average of 6% without pharmaceutical intervention (11 mm Hg systolic and 8 mm Hg diastolic)</td>
<td>40 of 94 original patients excluded because of no documented repeat blood pressure measurement; the authors conclude that a short observation period is warranted before pharmaceutical treatment</td>
<td>III</td>
</tr>
</tbody>
</table>
### Evidentiary Table (continued)

<table>
<thead>
<tr>
<th>Study</th>
<th>Year</th>
<th>Design</th>
<th>Intervention(s)/Test(s)/Modality</th>
<th>Outcome Measure/Criterion Standard</th>
<th>Results</th>
<th>Limitations/Comments</th>
<th>Class</th>
</tr>
</thead>
<tbody>
<tr>
<td>Karras et al(^{26})</td>
<td>2002</td>
<td>Prospective observational</td>
<td>143 patients with diastolic blood pressure (\geq 115) mm Hg were evaluated with serum creatinine and urine dipstick tests</td>
<td>Whether urine dipstick is an adequate screening test for acute serum creatinine level elevation</td>
<td>Of 143 patients, 24 met criteria for acutely elevated serum creatinine ((&gt;1.2) or acute elevation (&gt;25%) above baseline); the presence of either hematuria or proteinuria on dipstick identified these patients with 100% sensitivity and 29.7% specificity; specificity rose to 42.4% when an abnormal dipstick result was defined as hematuria or (\geq 1%) proteinuria; (95% CI 83% to 100%)</td>
<td>The wide CI creates some question in using this test alone as a screening tool to identify acute renal damage</td>
<td>II</td>
</tr>
<tr>
<td>Bartha and Nugent(^{27})</td>
<td>1978</td>
<td>Retrospective medical record review</td>
<td>Reviewed records of 116 patients entering a hypertension clinic who had a routine chest radiograph and/or ECG as part of their hypertension evaluation</td>
<td>Evaluate the utility of routine chest radiographs and ECGs in the evaluation of patients with hypertension</td>
<td>A routine chest radiograph or ECG led to diagnostic or therapeutic interventions in only 2 instances, were not useful as baseline examinations, were never used for prognostic purposes, and not once influenced hypertension management</td>
<td>Retrospective design; small sample size</td>
<td>III</td>
</tr>
<tr>
<td>Thach and Schultz(^{28})</td>
<td>1995</td>
<td>Literature review</td>
<td>Review of available evidence on lowering blood pressure in asymptomatic hypertension</td>
<td>Whether treatment of asymptomatic hypertension affected patient outcomes</td>
<td>Found no available evidence to support lowering blood pressure in asymptomatic patients in hours to 1-2 days prevents complications; acute treatment of asymptomatic hypertension has not shown improved blood pressure on short-term follow-up</td>
<td>Few studies and little evidence found to answer this specific question</td>
<td>III</td>
</tr>
</tbody>
</table>

\(^{26}\) EMT, Emergency medical technician; \(^{27}\) IV, intravenous; \(^{28}\) q, every.
### Appendix A. Literature classification schema.*

<table>
<thead>
<tr>
<th>Design/Class</th>
<th>Therapy†</th>
<th>Diagnosis‡</th>
<th>Prognosis§</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Randomized, controlled trial or meta-analyses of randomized trials</td>
<td>Prospective cohort using a criterion standard</td>
<td>Population prospective cohort</td>
</tr>
<tr>
<td>2</td>
<td>Nonrandomized trial</td>
<td>Retrospective observational</td>
<td>Retrospective cohort Case control</td>
</tr>
<tr>
<td>3</td>
<td>Case series Case report Other (eg, consensus, review)</td>
<td>Case series Case report Other (eg, consensus, review)</td>
<td>Case series Case report Other (eg, consensus, review)</td>
</tr>
</tbody>
</table>

*Some designs (eg, surveys) will not fit this schema and should be assessed individually.

†Objective is to measure therapeutic efficacy comparing ≥2 interventions.

‡Objective is to determine the sensitivity and specificity of diagnostic tests.

§Objective is to predict outcome including mortality and morbidity.

### Appendix B. Approach to downgrading strength of evidence.

<table>
<thead>
<tr>
<th>Downgrading</th>
<th>1</th>
<th>2</th>
<th>3</th>
</tr>
</thead>
<tbody>
<tr>
<td>None</td>
<td>I</td>
<td>II</td>
<td>III</td>
</tr>
<tr>
<td>1 level</td>
<td>II</td>
<td>III</td>
<td>X</td>
</tr>
<tr>
<td>2 levels</td>
<td>III</td>
<td>X</td>
<td>X</td>
</tr>
<tr>
<td>Fatally flawed</td>
<td>X</td>
<td>X</td>
<td>X</td>
</tr>
</tbody>
</table>