

Pet Ownership, but Not ACE Inhibitor Therapy, Blunts Home Blood Pressure Responses to Mental Stress

Karen Allen, Barbara E. Shykoff, Joseph L. Izzo, Jr

Abstract—In the present study, we evaluated the effect of a nonevaluative social support intervention (pet ownership) on blood pressure response to mental stress before and during ACE inhibitor therapy. Forty-eight hypertensive individuals participated in an experiment at home and in the physician's office. Participants were randomized to an experimental group with assignment of pet ownership in addition to lisinopril (20 mg/d) or to a control group with only lisinopril (20 mg/d). On each study day, blood pressure, heart rate, and plasma renin activity were recorded at baseline and after each mental stressor (serial subtraction and speech). Before drug therapy, mean responses to mental stress did not differ significantly between experimental and control groups in heart rate (94 [SD 6.8] versus 93 [6.8] bpm), systolic blood pressure (182 [8.0] versus 181 [8.3] mm Hg), diastolic blood pressure (120 [6.6] versus 119 [7.9] mm Hg), or plasma renin activity (9.4 [0.59] versus 9.3 [0.57] ng · mL⁻¹ · h⁻¹). Lisinopril therapy lowered resting blood pressure by ≈35/20 mm Hg in both groups, but responses to mental stress were significantly lower among pet owners relative to those who only received lisinopril ($P < 0.0001$; heart rate 81 [6.3] versus 91 [6.5] bpm, systolic blood pressure 131 [6.8] versus 141 [7.8] mm Hg, diastolic blood pressure 92 [6.3] versus 100 [6.8] mm Hg, and plasma renin activity 13.9 [0.92] versus 16.1 [0.58] ng · mL⁻¹ · h⁻¹). We conclude that ACE inhibitor therapy alone lowers resting blood pressure, whereas increased social support through pet ownership lowers blood pressure response to mental stress. (*Hypertension*. 2001;38:815-820.)

Key Words: blood pressure ■ social support ■ stress ■ lifestyle ■ pets

Antihypertensive agents are known to reliably lower resting blood pressure, but most of these drugs have little effect on blood pressure responses to physical or mental stressors.^{1,2} Because individuals who experience pronounced, frequent, or enduring autonomically mediated cardiovascular responses to stress may be at risk for the development of cardiovascular disease,^{3,4} variables that moderate or mediate reactivity to stress are important to consider. Several laboratory- and community-based studies have focused on the potential role of social support in buffering reactivity to mental stress⁵⁻⁹ and have found that when social support participants are perceived as supportive and nonevaluative, a beneficial effect is found.

Although considerable attention has been devoted to the definition and measurement of social support as it relates to health,^{10,11} most of this literature assumes that benefits are provided only by humans. In recent years, however, several studies have documented that pet animals also can have an important supportive role and a positive influence on the health of their owners. Pet ownership is a significant predictor of 1-year survival after myocardial infarction.^{12,13} Relative to the support of friends and spouses, the presence of a pet elicits significantly lower blood pressure and heart rate reactivity during mental stress.⁵ In addition, elderly individ-

uals with pets are buffered from the impact of stressful life events and make fewer visits to physicians¹⁴; among persons with AIDS, pet owners have a lower incidence of depression than do those without pets.¹⁵ Finally, service dogs have a positive influence on the well-being, self-esteem, and community integration of persons with disabilities.¹⁶

In the present study, we extended previous laboratory stress-reactivity and social support research to include home data in randomly assigned pet owners administered the ACE inhibitor lisinopril. We hypothesized that the acquisition of a pet would reduce heart rate, blood pressure, and renin responses to psychological stress among a group of hypertensive individuals in a high-stress profession (stockbrokers). The design we used made it possible to demonstrate the independence of blood pressure reactivity from basal blood pressure.

Methods

Participants and Setting

Participants were hypertensive patients with a high-stress occupation. A pretest-posttest control group design was used¹⁷ in 48 volunteers who had uncomplicated stage II+ hypertension (resting blood pressure ≥160/100 mm Hg). Of the 24 men (18 white and 6 black) and 24 women (18 white and 6 black), all were interested in

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Six-Month Results for Lisinopril and Pet Ownership: F Values for Main Effects and Interactions With SBP, DBP, HR, and PRA

Effect	SPB	DBP	HR	PRA
Pet condition				
MAT	6.832*	3.33	7.623*	19 431.66*
Speech	7.165*	6.695*	12.228*	28.48*
Social support				
MAT	6.125*	4.728*	1497	0.373
Speech	5.232*	5.345*	0.913	0.403
Stressors				
MAT	1826.17†	2096.74†	1588.90†	1795.66†
Speech	1314.56†	957.60†	1597.13†	2055.72†
Pet condition×MAT	307.903†	250.01†	229.33†	370.72†
Pet condition×speech	212.71†	113.48†	237.04†	364.34†
Social support×MAT	63.00†	63.54†	71.92†	0.017
Social support×speech	33.83†	21.17†	35.76†	0.053
Pet condition×MAT×social support	53.28†	105.52†	89.35†	0.133
Pet condition×speech×social support	27.96†	11.86†	35.15†	0.154

* $P < 0.05$.† $P < 0.001$.

stress reduction and agreed to acquire a pet if chosen to do so. Participants were randomized to a control group without pets ($n=24$) or an experimental group ($n=24$) who subsequently acquired pets.

Design

All participants completed baseline mental stress sessions in their homes after 1 month of observation. All participants then were treated with lisinopril (20 mg/d). Those assigned to the pet owner group acquired their animals at the time drug therapy began. All participants were evaluated again at 6 months with a second home mental stress session. Dependent measures were systolic blood pressure (SBP), diastolic blood pressure (DBP), heart rate (HR), and plasma renin activity (PRA). Participant ratings of stress and coping were assessed both before and after each stressor.

Stressors

Stressors included mental arithmetic task (MAT) and speech, both of which have been used in numerous laboratories¹⁸ to produce substantial increases in cardiovascular response.

Physiological Recording Instrument

HR, SBP, and DBP were recorded automatically once each minute throughout the experiment with a portable Propaq monitor (model 106 EL; Protocol Systems Inc).

Procedures

After participants provided written informed consent, they were seated in a quiet room, and the Propaq blood pressure cuff was attached. Resting HR, SBP, DBP, and PRA then were assessed and recorded. In addition, participants completed a questionnaire about social support.¹⁹ Later in the same day, participants performed 2 psychologically stressful tasks in their homes according to a standard laboratory paradigm. HR, SBP, and DBP were recorded once each minute throughout the experiment. Renin was assessed 3 times (ie, after the initial rest and after each of the stressful tasks).

After this home experiment, all participants in both the experimental and control groups began lisinopril therapy (20 mg/d) with the goal of blood pressure within a normal range ($<130/90$ mm Hg). Black participants also received 12.5 mg/d hydrochlorothiazide. At the time they began drug therapy, individuals in the experimental group were instructed to acquire a pet cat or dog.

Six months later, the data collection procedure just described was repeated (in both the physician's office and in the homes of participants). Pet owners performed the second phase of the home experiment in the presence of their pets, which roamed freely throughout the room in which data collection took place.

Data Analysis

The main analysis was a repeated measures ANOVA before and during drug therapy, with tasks (MAT and speech) as within-subjects factors and pet ownership status and score on self-report social support scale (categorized as high or low) as between-subjects factors. All analyses were performed separately for SBP, DBP, HR, and PRA. Additional repeated measures ANOVAs addressed home versus office blood pressure values and a comparison of MAT performance before and with drug therapy and pet ownership.

An expanded Methods section can be found in an online data supplement available at <http://www.hypertensionaha.org>.

Results

Physiological Responses: Experimental Findings

ANOVAs for SBP, DBP, and HR revealed that before individuals began drug therapy or acquired pets, there were main effects ($P < 0.01$) for social support and for tasks (MAT and speech), as well as for social support×task interactions, but no effects by assigned pet ownership status. Before drug therapy and pets, ANOVA results for PRA also revealed main effects ($P < 0.01$) for MAT and speech but not for social support.

The Table includes SBP, DBP, HR, and PRA ANOVA summary data after 6 months with lisinopril and pets; except for DBP during MAT, main effects are shown for pet ownership condition as well as social support and tasks for SBP and DBP. Results for HR and PRA are similar but include no main effects for social support. In addition, the Table shows significant 2-way interactions (between pet condition and tasks and between social support and tasks) for all dependent variables, as well as 3-way interactions among

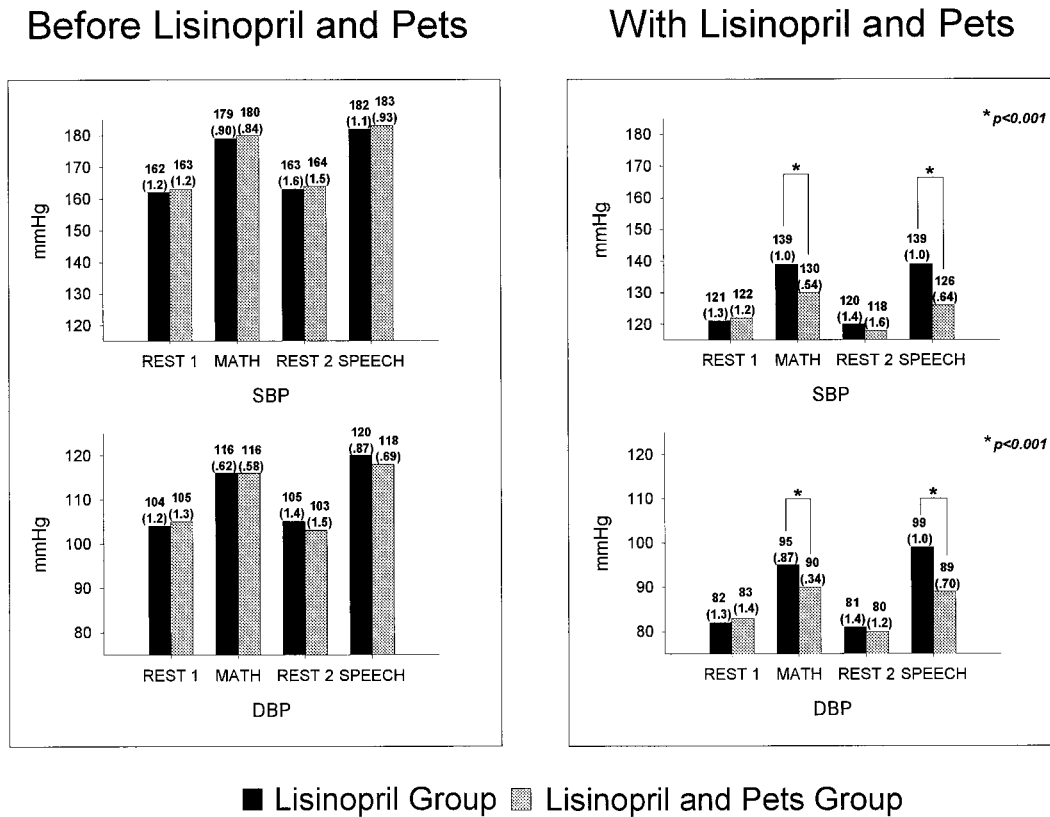


Figure 1. SBP and DBP reactivity (mean [SEM]) in response to MAT and speech both before and during lisinopril therapy and the presence of a pet.

pet condition, social support, and tasks for all dependent variables except PRA.

Figure 1 provides SBP and DBP reactivity for each task, and Figure 2 provides HR and PRA (both before and during drug therapy). At the beginning of the study, all participants had stage II hypertension ($>160/100$ mm Hg), and after 6 months, average blood pressures in both the lisinopril-only and the lisinopril-and-pet groups were within the normal range. At month 1, MAT and speech elicited significant increases in physiological responses of both groups. After 6 months, however, relative to those without pets, individuals with pets had significantly lower reactivity scores; their reactivity was diminished by half.

Comparisons of Office With Home Blood Pressure

Before participants received the medication and acquired pets, there was a significant difference between office and home mean SBP ($F=2089.36$, $P<0.001$) and DBP ($F=4499.23$, $P<0.001$) values. When individuals were administered medication and acquired pets, blood pressure was again higher in the office than in the home (SBP [$F=344.94$, $P<0.001$] and DBP [$F=1657.37$, $P<0.001$]). There were no other main effects associated with these differences.

Performance Data

MAT performance was computed for each participant as a ratio between the number of correct answers and the number of attempted answers. We were interested in a comparison of ratios before participants had received lisinopril and pets with

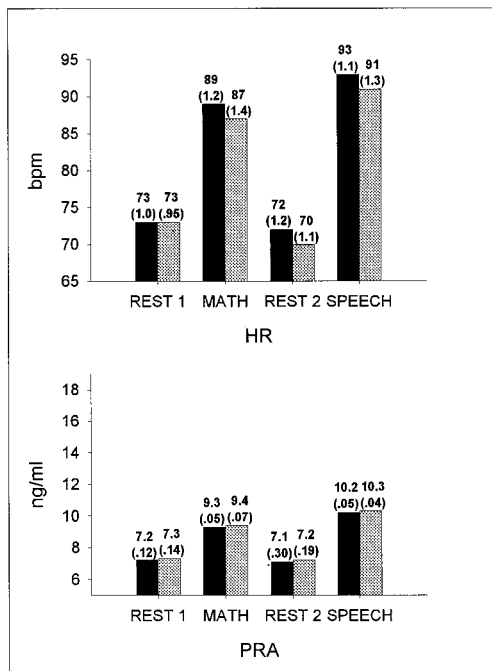
ratios after participants received lisinopril and acquired pets. Results revealed a main effect for ratio ($F=166.90$, $P<0.001$) and an interaction effect for lisinopril and pet ownership ($F=140$, $P<0.001$). That is, at the second data collection point (after the acquisition of pets), pet owners had significantly greater improvements in their task performances than did individuals without pets.

Discussion

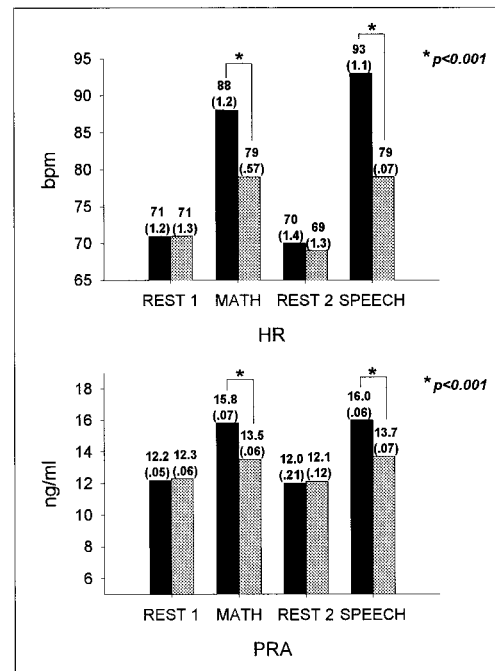
In the present study, we examined the effect of pet ownership on cardiovascular responses to psychological stress among a group of hypertensive individuals in high-stress professions. On the basis of these results, we conclude that reactivity and basal blood pressure are influenced by independent mechanisms; that is, ACE inhibitor therapy lowers only resting blood pressure, whereas the addition of a social support intervention lowers responses to stress. These results extend earlier findings that ACE inhibition fails to diminish BP or HR responses to stressful tasks¹ to demonstrate a beneficial influence of social factors that may buffer stress responses for persons with hypertension who are treated with ACE inhibitors.

Interestingly, we found that an individual's assessment of his or her general social environment was predictive of that person's BP and HR responses to stress. Even though we did not investigate the effect of the presence of friends on reactivity, participants who perceived that they belonged to a group and had friends to confide in had a lower reactivity to stress than did their counterparts who reported few social

Before Lisinopril and Pets



With Lisinopril and Pets



■ Lisinopril Group ▨ Lisinopril and Pets Group

Figure 2. HR and PRA reactivity (mean [SEM]) in response to MAT and speech both before and during lisinopril therapy and the presence of a pet.

contacts. These results suggest that persons with low social support systems are likely to benefit in particular from the enhanced environment that pets can provide.

Improved task performance was also associated with pet ownership. At the beginning of the study, participants in both the control group (lisinopril only) and the experimental group (lisinopril and pet ownership) had 74% correct performance. At the second data collection point, however, participants with pets had 92% correct performance, whereas their counterparts without pets remained at 75%. This finding is notable because improved task performance suggests that participants did not abandon the task because they perceived their pets to be pleasant distractions. In addition, although ACE inhibitor therapy has been associated with either improved or impaired cognitive function,²⁰ in our study lisinopril alone did not have any influence on cognition, and cognitive improvement occurred only when lisinopril was paired with the presence of pets.

One explanation for our findings is that the presence of pets provided the kind of nonevaluative social support that is critical to buffering physiological responses to stress. Social support theorists^{10,19} have suggested that positive feeling states may enhance an individual's capacity to adapt to stress. We believe that pets may evoke such feelings in their owners. This conclusion was confirmed by an exit conference in which the nominal group technique²¹ was used to structure responses to the question, "How has your pet changed your life?" Interestingly, all of our participants attended this

voluntary meeting, and all dog owners and most cat owners brought their pets with them to the session. The consensus response with the highest rating was: "Having this pet makes me better able to see what is really important and to put things into perspective." When asked about increased responsibility and similar issues, participants responded that the positive aspects far outweighed any added expense or responsibility and that they would never give up their pets. Because only persons who would agree to acquire pets were eligible to participate in our study, however, we cannot comment on whether individuals who are not inclined to like animals would develop similar relationships with pets.

The issue of demand characteristics (expectations unintentionally conveyed from the investigator to the participants) is an important factor in behavioral research. Consequently, at the beginning of our project, we did not reveal our hypotheses about pets but rather said the focus of the study was on the general relationship between social factors and health. In the debriefing session at the conclusion of the study, we asked participants to identify whether they thought pets influenced their resting blood pressure, their responses to stress, or both. Except for 2 participants who answered "both," all said that they believed their pets helped diminish their resting blood pressure. This was reinforced by the fact that participants performed home monitoring over the course of the study and that lisinopril therapy did in fact cause a dramatic reduction in blood pressure. The pet owners, however, attributed this reduction to a combination of drug therapy and pet owner-

ship. Because of this fortuitous misunderstanding on the part of our participants, we do not believe that demand characteristics contributed to our findings about stress responses. Because the participants (both with and without pets) were highly motivated to reduce their blood pressure and believed that the drug would work, however, we cannot totally rule out that demand characteristics as well as a placebo effect contributed to their reductions in resting blood pressure. That is, wanting to please experimenters who came to their homes, combined with a strong belief in the treatment, could have influenced responses to lisinopril, although research suggests that actual placebos have little effect on ambulatory blood pressure monitoring,²² so we believe this was unlikely.

We acknowledge that the study population sample was highly selected for homogeneity. We were interested in looking at stress responses in a group of individuals who experienced similar job stress and who lived alone. Because there were many stockbrokers available who were motivated to be in a behavioral study and were willing to acquire pets, we decided to focus on them. Although chronic stress may be associated with an elevated renin level, we did not select participants on this basis or for any physiological characteristic other than stage II hypertension. The baseline PRA that we report may appear higher than is often observed, but it is consistent with a previous related study.¹ In addition, our work supports earlier studies that document a positive relationship between psychological stress and renin reactivity.^{23,24} In the present study, because the main interest was in a change from baseline, the reported difference in change scores between groups is the important area of focus. However, because it is known that individuals with high renin levels often have a very positive response to ACE inhibition therapy,²⁵ the possibility exists that our findings would generalize only to other “high-renin” individuals. Among our participants, the degree of response to lisinopril was consistent with renin response to stress before drug therapy. We can only speculate whether elevated renin is another manifestation of generalized neural hormonal activation that likely includes increased sympathetic nervous system and adrenocortical activity.

The present study has several limitations. Although the study design could have been strengthened by the addition of an “intervention” placebo group, we did not include one because of the nature of our social intervention. We do not believe that a placebo drug would have been equivalent to the acquisition of a pet. Consequently, we cannot comment definitively on the possibility that the pet acted as a “placebo effect” or on the relationship between the power of suggestion and the power of pets. Another limitation of the study is that because participants had stage II hypertension at the beginning of the study, it was not ethically possible to randomly assign only pet ownership for 6 months before lisinopril therapy. This arm of the design would be especially important in future investigations among populations with borderline hypertension, because it would help determine whether manipulation of the social environment can reduce the need for drug therapy. Yet another drawback to our study is that although our findings reveal much about blood pressure response to acute psychological stress in the home,

we cannot generalize the findings to other stressful settings, such as work environments. Because differences between office and home resting blood pressures were not influenced by pet ownership or lisinopril therapy, it is logical to consider whether stress reactivity outside the home might also not be influenced by pets. We believe, however, that because reactivity and resting blood pressure are independent of each other, reactivity outside the home has the potential to be influenced by social factors.

In the present study, we were able to change the social environment of our participants by adding a pet to their lives. Enhancement of social support with human friends is much more complex and difficult to achieve and, to our knowledge, has not been successfully carried out in an experimental design. Because pets, unlike human friends, are perceived as always being nonjudgmental and accepting of their owners, they are ideal candidates for social support intervention. Physiologically, pets had greater influence on sympathetic responses than did ACE inhibition alone. Consequently, our findings suggest that higher center influences can modify stress responses and that coping skills may be related to increased cortical inhibition of the brain stem. Although we do not advocate the substitution of pets for human companionship, we conclude that for persons who like animals and have few social contacts, pets can enhance isolated lives and provide health benefits.

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References

1. Dimsdale JE, Mills P, Ziegler M, Leitz K, Nelesen R. Converting enzyme inhibition and blood pressure reactivity to psychological stressors. *Hypertension*. 1992;20:210–213.
2. Mills P, Dimsdale JE. Cardiovascular reactivity to psychological stressors: a review of the effects of beta-blockade. *Psychosomatics*. 1991; 32:209–220.
3. Clarkson TB, Manuck SB, Kaplan JR. Potential role of cardiovascular reactivity in atherogenesis. In: Matthews KA, Weiss SM, Detre T, Dembroski TM, Falkner B, Manuck SB, Williams RB, eds. *Handbook of Stress, Reactivity, and Cardiovascular Disease*. New York, NY: Wiley; 1986:35–47.
4. Gullette ECD, Blumenthal JJ, Babyak M, Jiang W, Waugh RA, Frid DJ, O'Connor CM, Morris JJ, Krantz DS. Effects of mental stress on myocardial ischemia during daily life. *JAMA*. 1997;277:1521–1526.
5. Allen K, Blascovich J, Tomaka J, Kelsey RM. Presence of human friends and pet dogs as moderators of autonomic responses to stress in women. *J Pers Soc Psychol*. 1991;61:582–589.
6. Christenfeld N, Gerin W, Linden W, Sanders M, Mathur J, Deich JD, Pickering TG. Social support effects on cardiovascular reactivity: is a stranger as effective as a friend? *Psychosom Med*. 1997;59:388–398.
7. Fontana AM, Diegnan T, Villeneuve A, Lepore S. Nonevaluative social support reduces cardiovascular reactivity in young women during acutely stressful performance situations. *J Behav Med*. 1999;22:75–91.
8. Kamarck TW, Manuck SB, Jennings JR. Social support reduces cardiovascular reactivity to psychological challenge: a laboratory model. *Psychosom Med*. 1990;54:42–58.
9. Lepore SJ, Allen KA, Evans GW. Social support lowers cardiovascular reactivity to an acute stressor. *Psychosom Med*. 1993;55:518–524.
10. Cohen S, Syme SL. *Social Support and Health*. San Diego, Calif: Academic Press; 1985.
11. Shumaker SA, Czajkowski SM. *Social Support and Cardiovascular Disease*. New York, NY: Plenum; 1994.

12. Friedmann E, Katcher AH, Lynch JJ, Thomas SA. Animal companions and one-year survival of patients after discharge from a coronary care unit. *Public Health Rep.* 1980;95:307–312.
13. Friedmann E, Thomas SA. Pet ownership, social support, and one-year survival after acute myocardial infarction in the Cardiac Arrhythmia Suppression Trial (CAST). *Am J Cardiol.* 1995;76:1213–1217.
14. Siegel JM. Stressful life events and use of physician services among the elderly: the moderating role of pet ownership. *J Pers Soc Psychol.* 1990;58:1081–1086.
15. Siegel JM, Angulo FJ, Detels R, Wesch J, Mullen A. AIDS diagnosis and depression in the multicenter AIDS cohort study: the ameliorating impact of pet ownership. *AIDS Care.* 1999;11:157–169.
16. Allen K, Blascovich J. The value of service dogs for people with severe ambulatory disabilities: a randomized controlled trial. *JAMA.* 1996;275:1001–1006.
17. Campbell DT, Stanley JC. *Experimental and Quasi-Experimental Designs for Research.* Boston, Mass: Houghton Mifflin Company; 1963.
18. Kelsey RM. Electrodermal lability and myocardial reactivity to stress. *Psychophysiology.* 1991;28:619–632.
19. Cohen S, Hoberman HM. Positive events and social supports as buffers of life change to stress. *J Appl Soc Psychol.* 1983;13:99–125.
20. O'Brien AA, Bulpitt CJ. The effects of ACE inhibitors on cognitive function. *Drugs Aging.* 1995;6:173–180.
21. Delbecq AL, Van de Ven AH, Gustafson DH. *Group Techniques for Program Planning.* Middleton, Wis: Green Briar Press; 1986.
22. Waeber B, Brunner HR. Clinical value of ambulatory blood pressure monitoring in the assessment of antihypertensive therapy. *Blood Press Monit.* 1999;4:263–266.
23. Dimsdale JE, Ziegler JB, Mills P. Renin correlates with blood pressure reactivity to stressors. *Neuropsychopharmacology.* 1990;3:237–242.
24. Spence JD, Manuck SB, Munoz C, Cheung H, Huff M, Dennis B, Borkowski K. Hemodynamic and endocrine effects of mental stress in untreated borderline hypertension. *Am J Hypertens.* 1990;3:859–862.
25. Case DB, Wallace JM, Keim HJ, Weber MA, Sealey JE, Laragh JH. Possible role of renin in hypertension as suggested by renin-sodium profiling and inhibition of converting enzyme. *N Engl J Med.* 1977;296:641–646.

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