Infusion of Epinephrine Augments Pressor Responses to Mental Stress

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Circulating epinephrine may facilitate neural release of norepinephrine both during and after periods of sympathoadrenal activation by stimulation of prejunctional \(\beta\)-adrenergic receptors. The present study was undertaken to examine possible effects and aftereffects of epinephrine on the hemodynamic reactivity to mental stress. To this end, two strictly standardized mental stress tests were performed in 14 normotensive men during and 1 hour after double-blind infusion of epinephrine (50 ng kg\(^{-1}\) min\(^{-1}\)) or placebo given in random order. During epinephrine infusion, the systolic pressor response to psychosocial stress was augmented (+17 versus +10 mm Hg during epinephrine and placebo, respectively; \(p=0.02\)). This was associated with an attenuated post-stress recovery, with the result that the stress exposure induced a prolonged elevation of systolic blood pressure. Heart rate was elevated and diastolic blood pressure lowered during epinephrine infusion without any change in the reactivity to stress. One hour after the end of the epinephrine infusion resting heart rate was still maintained on a higher level independently of level of arousal, but heart rate and blood pressure responses to stress were unaffected. The findings are consistent with the hypothesis that high circulating epinephrine levels amplify pressor responses to mental stress but do not support the suggestion that short-term infusion of epinephrine causes prolonged augmentation of blood pressure responses to psychosocial stress. *(Hypertension 1991;18:467–474)*

Several studies have demonstrated that exogenous and endogenous epinephrine may facilitate neural release of norepinephrine.\(^1\)–\(^9\) This mechanism is, at least partly, related to the stimulatory effect of epinephrine on prejunctional \(\beta\)-adrenergic receptors, which in humans appear to belong to the \(\beta_2\)-adrenergic receptor subtype.\(^2\)–\(^4\),\(^10\) Furthermore, there is evidence that circulating epinephrine may be taken up into the postganglionic sympathetic nerves and be released as a co-transmitter with norepinephrine for 24 hours after its uptake.\(^2\)–\(^4\),\(^11\)–\(^13\) Thereby, epinephrine may augment the simultaneous release of endogenous norepinephrine and increase \(\alpha\)-adrenergic receptor-mediated vasoconstriction.\(^11\)–\(^13\)

It has been suggested that endogenously released epinephrine could increase the neural discharge of norepinephrine both during and after periods of sympathoadrenal activation and thereby contribute to development of stress-linked hypertension.\(^14\)–\(^16\) In humans, amplification of blood pressure responses to vasoconstrictor stimuli such as the cold pressor test and isometric handgrip exercise have been observed during infusion of epinephrine.\(^17\) Furthermore, increased vasoconstriction responses to lower body negative pressure (LBNP) have been demonstrated 30 minutes after infusion of epinephrine.\(^11\)–\(^13\) In fact, a significant pressor effect has been observed for up to 18 hours after a 6-hour infusion of epinephrine.\(^18\)

Although it has been assumed that this mechanism may be of importance also for pressor reactions to psychosocial stress, no studies have as yet been published to specifically show if blood pressure responses to psychological stressors are augmented by high epinephrine levels. The present study was therefore undertaken to examine the possible effects and aftereffects of epinephrine on the hemodynamic reactivity to mental stress. To this end, two strictly standardized mental stress tests were performed in healthy male subjects during and 1 hour after infusion of epinephrine into the high physiological range.

**Methods**

**Subjects**

Fourteen young, normotensive male subjects participated in the study. All subjects were apparently healthy nonsmokers who were not being treated with any medication and who were without cardiovascular...
disease. The subjects were recruited among medical students and hospital employees. Their mean age, weight, and height were 26.8 years (range 22–35), 77.4 kg (67–90), and 186.4 cm (178–195), respectively. Informed consent was obtained from each subject before inclusion in the study after the rationale, nature, and potential risks of the research had been carefully explained. The protocol was approved by the Ethics Committee of the University of Göteborg, and the study was performed according to the declaration of Helsinki.

The participants were instructed to avoid heavy exercise or emotional excitement and to be in a fasting condition for 4 hours before the experiment. All subjects were asked to refrain from taking methylxanthine-containing products on the day before the experiment to avoid interference with the catecholamine analyses.

Experimental Design

The general design of the study is outlined in Figure 1. Briefly, all subjects participated on 2 study days at least 1 week apart. According to a randomized, double-blind schedule, they received infusions of either epinephrine or placebo for 35 minutes during the first part of the experiment. On each day two mental stress tests were performed with an interval of 1 hour between; the first test was performed during the infusion and the second 1 hour after the end of the infusion. During each stress test, the subjects performed 10 minutes of forced mental arithmetic according to a strictly standardized procedure (see Reference 19 for reproducibility data on this method). Two investigators conducted the tests (M.P. and S.J.), but each subject always met the same investigator during each stress test.

The experiments were performed either in the morning (9:00 AM to noon) or in the afternoon (1:00–4:00 PM). Each subject was investigated at the same time of day on both occasions, and since each subject served as his own control, this design controlled for possible effects of circadian rhythms in stress reactivity.

Procedure

On reporting to the laboratory, the subject was placed in a comfortable chair in a semirecumbent position in a quiet room. One venous cannula (Venflon, Viggo, Helsingborg, Sweden) was inserted percutaneously into an antecubital vein of the dominant arm for infusion of epinephrine. Another venous cannula was inserted into a vein of the dorsum of the hand of the same arm for blood sampling. A 50 cm polyethylene catheter was connected to each indwelling cannula, and the catheters were led through the wall to an adjacent control room. This technique allowed us to start and stop the infusions and collect blood samples without the subjects knowing exactly when this was done. The intravenous lines were kept patent by slow saline infusions.

A blood pressure cuff for noninvasive assessment of blood pressure was applied on the nondominant arm. Electrodes for computerized vector-cardiography were fixed to the chest, arms, and legs according to a modified Frank system (i.e., leg electrodes moved to the anterior superior iliac spines).

Epinephrine Infusion

The epinephrine infusate was prepared by diluting 0.5 mg epinephrine tartrate (Adrenalin, Apoteksbolaget, Stockholm, Sweden) in 50 ml isotonic saline. It was administered at a rate of 50 ng×kg⁻¹×min⁻¹. The total individual infusion volume ranged between 335 and 450 µl/min. Placebo (isotonic saline) was administered in the same volume for each subject.

Mental Stress

Fifteen minutes after the start of the infusion, the investigator entered the room, which was now fully lit, and gave the subject an oral instruction lasting approximately 40 seconds. The subject then performed forced mental arithmetic for 10 minutes with serial subtractions of seven from 700 while trying to keep pace with a metronome at a rate of approximately 90/min. The investigator encouraged the subject to increase the speed by short repeat comments. After a positive and reassuring comment, the examiner reduced the light and left the room. The stress...
task was then followed by a 10-minute post-stress baseline period during which the infusion was continued. The infusion was then stopped, and the recording was continued for another 10-minute rest period.

After conclusion of the first part of the experiment, the subjects remained in the same position for 1 hour but were allowed to read or listen to the radio. Thereafter, a second stress test was performed in exactly the same way as the first except for the infusions.

**Hemodynamic Monitoring**

Systolic and diastolic blood pressure were measured at 1-minute intervals using an automatic noninvasive oscillometric blood pressure monitor (Dinamap model 845, Criticon Instruments, Tampa, Fla.), which has been validated against intra-arterial blood pressure recordings in our laboratory.20 Heart rate was monitored continuously throughout the experiment by computerized vectorcardiography (MIDA 1000, MIDA System, Ortivus Medical AB, Täby, Sweden).

**Blood Sampling and Biochemical Analyses**

Blood samples for analysis of plasma catecholamines were obtained on the following time points: at 10, 25, 35, 45, 55, 105, 115, and 125 minutes (see Figure 1). All blood samples were obtained from the distal indwelling venous cannula. For technical reasons, a few blood samples could not be obtained at the precise time points specified in the protocol; when there were a lower number of observations, this is indicated in Table 2.

The first 5 ml of blood were always discarded. Eleven milliliters of blood for analysis of catecholamines was drawn into prechilled tubes containing 220 μl glutathione-EDTA (60 mg/ml and 90 mg/ml) and immediately was placed on melting ice. The samples were centrifuged for 5 minutes at +4°C and 2,000g within 2 minutes of collection. Plasma aliquots were stored at −70°C until assay. Plasma catecholamines (epinephrine and norepinephrine) were analyzed by high-performance liquid chromatography with electrochemical detection (Electrochemical Detector 640, Waters, Millipore Ltd, Griesenhein, FRG). All coded samples from each study subject were always analyzed in the same assay run.

**Data Reduction**

Hemodynamic data from the stress experiment were reduced by dividing baseline and stress periods into 2.5-minute epochs, and by computing mean values for heart rate and systolic and diastolic blood pressure for each period. Data from the plasma catecholamine analyses were not reduced.

Reactivity was defined for each stress experiment as the difference between the mean level during stress and the mean from the corresponding prestress baseline period. Recovery was defined as the difference between the stress and post-stress levels observed during the 10 minutes after cessation of stress.

**Statistical Analysis**

Standard statistical methods were used. To assess the overall effects of both treatments, two two-way analyses of variance (ANOVA) for repeated measures (with subject as random factor) were performed for each variable; one for the first part of the experiment (0–55 minutes) and another one for the second part (95–125 minutes). Each ANOVA model included treatment (placebo or epinephrine), period (22 or 12 2.5-minute periods), and the treatment × period interaction term. To evaluate the effects of the stress tests as such, one-way ANOVAs for repeated measures were performed for each of the two treatments; one for the first stress test (15–45 minutes) and one for the second test (95–125 minutes). Each model included 12 2.5-minute periods.

To compensate for possible violations of the assumptions of sphericity, all degrees of freedom were corrected according to the conservative Greenhouse-Geisser procedure.21

In addition, the magnitude of stress-induced changes (reactivity) during the first and second stress tests of each experiment day were analyzed by paired Student's t test. In all instances two-tailed tests were used, and the test was considered significant at p<0.05.

**Results**

**During the Infusion**

As shown in Table 1, there were no significant differences in any of the hemodynamic variables or plasma catecholamine levels at rest between the epinephrine and placebo experiments. After 15 minutes of epinephrine infusion, the plasma epinephrine concentration had increased from 0.3 to 2.4 nmol/l [t(11)=5.12, p=0.003]. Parallel to the increase in circulating epinephrine, the plasma norepinephrine concentration increased from 2.5 to 3.2 nmol/l [t(13)=2.53, p=0.025]. These changes were accompanied by an increase in heart rate by approximately 5 beats/min [t(13)=6.62, p=0.0001] and a decrease in diastolic blood pressure by 5 mm Hg [t(13)=9.32, p=0.0001] (Table 1 and Figure 2). There was no significant change of systolic blood pressure during this first part of the infusion period. During the corresponding period of placebo infusion, no significant variations were observed in any of the hemodynamic or plasma catecholamine variables.

The first mental stress test caused significant reactions in all three hemodynamic variables during both types of infusions as indicated by significant period effects by one-way analyses of variance (ANOVA) [F:s(11,143)>9.57, p<0.0002 throughout] (Table 2 and Figure 2). Further, two-way ANOVA revealed a significant infusion × period interaction for systolic blood pressure [F(21,273)=5.40, p=0.0003] (Table 2). Accordingly, the stress-induced increase of systolic blood pressure was significantly greater during the epinephrine infusion [+17 mm Hg, t(13)=7.27, p<0.0001] than during the placebo infusion [+10
mm Hg: t(13)=4.01, p=0.001 [epinephrine versus placebo t(13)=2.68, p=0.02].

ANOVA also indicated significant infusion × period interactions for heart rate [F(21,273)=3.30, p=0.01] and diastolic blood pressure [F(21,273)=2.93, p=0.01]. However, the slight difference in stress reactivity between the two conditions did not attain statistical significance for either heart rate or diastolic blood pressure [epinephrine versus placebo, t(13)<1.4, p>0.10 for both measures].

Heart rate increased by 17 beats/min [t(13)=5.73, p<0.0001] and 14 beats/min [t(13)=8.66, p<0.0001] after epinephrine and placebo, respectively. Diastolic blood pressure increased by 13 mm Hg [t(13)=7.45, p<0.0001] after epinephrine and 11 mm Hg [t(13)=6.00, p<0.0001] after placebo. Plasma norepinephrine increased significantly in response to the stress test during the placebo infusion [t(13)=2.55, p=0.02].

During the 20-minute post-stress recovery period, systolic blood pressure and heart rate remained significantly elevated after epinephrine compared with the placebo condition (Table 1 and Figure 2). Also, heart rate was maintained on a higher level throughout the second stress experiment [F(1,13)=5.52, p=0.04]. However, systolic and diastolic blood pressure had returned to levels similar to those observed after the placebo infusion.

In response to the second stress test, performed 60 minutes after the end of the infusions, ANOVA indicated significant reactions in heart rate and blood pressure [F(6,114)=7.14, p<0.0003 throughout]. There were, however, no significant interactions between period and condition for any of the hemodynamic variables, that is, the epinephrine infusion had no effect on the reactivity to the second stress experiment (Table 2). The stress-induced increase in heart rate was 16 beats/min [t(13)=8.26, p<0.0001] after epinephrine and 14 beats/min [t(13)=8.46, p<0.0001] after placebo (t test epinephrine versus placebo, NS). The corresponding responses of systolic and diastolic blood pressure were 10/10 mm Hg [t(13)=3.95, p=0.002 and t(13)=6.62, p=0.0001] after epinephrine, and 8/9 mm Hg [t(13)=4.25, p=0.0009 and t(13)=4.89, p=0.0003] after placebo (epinephrine versus placebo, NS throughout).

Stress-induced increases of plasma norepinephrine and epinephrine only attained statistical significance after the placebo infusion [F(2,24)=4.49, p=0.04 and F(2,24)=4.71, p=0.03, respectively]. There were no significant differences between the catecholamine responses to the second stress test after the two

One Hour After the Infusion

One hour after the epinephrine infusion, plasma epinephrine concentrations were still slightly but significantly elevated as indicated by a significant condition effect [F(1,11)=12.79, p<0.004] (Table 2 and Figure 2). Also, heart rate was maintained on a higher level throughout the second stress experiment [F(1,13)=5.52, p=0.04]. However, systolic and diastolic blood pressure had returned to levels similar to those observed after the placebo infusion.

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Figure 2. Line graphs show hemodynamic responses to the first and second stress experiments during and 1 hour after infusion of epinephrine (solid lines) and placebo (broken lines). Mean and SEM.

infusions. We did not observe any differences in any of the variables in post-stress recovery between the two types of infusion.

Discussion

In the present study, infusion of epinephrine elevated venous plasma epinephrine levels to approximately 3 nmol/l, which is in the high physiological range. As observed earlier by other investigators, the epinephrine infusion also significantly elevated plasma norepinephrine levels. Systolic blood pressure responses to mental stress were significantly augmented during the epinephrine infusion. This change was associated with an attenuated post-stress recovery, with the result that the stress exposure induced a prolonged elevation of systolic blood pressure. In contrast, the heart rate recording showed a parallel upward shift during and after the infusion, without evidence of increased stress reactivity. As expected, diastolic blood pressure decreased during the epinephrine infusion, but the reactivity was unaltered when expressed in absolute values. One hour after the epinephrine infusion, heart rate or systolic and diastolic blood pressure responses to mental stress were not significantly different from those observed after placebo. However, there were slight but significant upward shifts of heart rate both before, during, and after the second stress test. Taken together, these findings indicate that systolic pressor responses are amplified by high levels of circulating epinephrine.

The augmented pressor responses during the infusion are in line with some previous investigations. The observations are also compatible with the hypothesis that epinephrine facilitates norepinephrine release by stimulation of presynaptic β2-adrenergic
receptors. The fact that the stress-induced increase in plasma norepinephrine was not significantly augmented does not necessarily speak against this interpretation since the venous sampling used in the present study does not fully reflect the sympathetic norepinephrine outflow.

However, the unaltered amplitude of the stress-induced blood pressure changes 1 hour after the epinephrine infusion appear to be at variance with studies of effects on vasoconstrictor responses to physical stressors. In a series of extensive studies, Floras and coworkers have demonstrated augmented pressor responses to LBNP in the forearm 30 minutes after both local and systemic infusions of epinephrine. Similar results were obtained by Fellows et al. In the latter study, heart rate responses to LBNP were augmented 30 minutes after infusion of epinephrine, whereas the forearm vasoconstrictor response was only amplified 15 but not 30 minutes after the end of the infusion.

One possibility for the lack of significant aftereffects of epinephrine in the present study may be that the time lag between infusion and the second stress test was too long. However, since augmented pressor responses have been observed up to 18 hours after infusion of epinephrine, alternative explanations must be considered. In the present study, we used a slightly higher dose of epinephrine than the one used by Floras and coworkers (50 ng/kg/min versus approximately 20 ng/kg/min). Since inappropriately high doses of epinephrine can inhibit norepinephrine release by stimulating inhibitory a2-adrenergic receptors, it may be argued that the epinephrine outflow is a sign of the vasoconstrictor responses to LBNP. The hemodynamic response to LBNP is characterized by reflex neurogenic vasoconstriction. In contrast, mental stress induces a hemodynamic pattern with increased cardiac output and a slight decrease of total peripheral resistance. In fact, in the forearm (which is the vascular bed studied by Floras and Fellows) mental stress leads to a rather marked vasodilation with a decrease of vascular resistance by approximately 30% (S. Jern, unpublished observations), whereas the LBNP procedure increases resistance in the order of 55-70%. Similarly, the stimuli used by Vincent and coworkers (isometric exercise and cold pressor test) are also known to induce vasoconstrictor responses. Thus, it cannot be ruled out that the aftereffects of epinephrine are related to the hemodynamic response pattern elicited by different stimuli. In fact, the proportion of circulating catecholamines taken up by the human heart and the forearm is very different, and it is conceivable that stimuli mainly associated with a peripheral vasoconstriction may be differently affected than stimuli associated with a predominantly b-adrenergic cardiac response.

Also, the aftereffects of epinephrine may be different in hypertensive patients than in the normotensive subjects investigated in the present study. In fact, in a recent study by Grassi et al responses to LBNP and the cold pressor test were unchanged after epinephrine in normotensive subjects, but the responses were augmented in subjects with borderline hypertension. It is worthy of note that some of the previous positive studies have been performed in borderline hypertensive subjects.
It has recently been demonstrated that the augmentation of pressor responses after epinephrine is abolished by pretreatment with the neuronal uptake-1 blocker desimipramine, thus supporting the notion that the amplified response is due to uptake and subsequent co-release of epinephrine together with norepinephrine.\(^{13}\) However, a central effect of epinephrine cannot be ruled out, since there are at least two studies in which a rather marked increase in effenter sympathetic nerve activity has been demonstrated after infusion of epinephrine.\(^{9,29}\) Furthermore, epinephrine is known to be a stimulator of renin release,\(^{30}\) and angiotensin II augments the vasoconstrictor response to sympathetic nerve stimulation and LBNP, partly because of facilitation of norepinephrine release.\(^{31,32}\) Thus, several mechanisms may be activated by epinephrine, and psychological stress may interact differently with these changes than do vasoconstrictor stimuli.

Another possible explanation for the present observations is related to the effects of epinephrine on postsynaptic \(\beta_2\)-adrenergic receptors.\(^{33,34}\) Under normal conditions stress-induced \(\beta_2\)-mediated muscular vasodilation by epinephrine may partly cancel the vasoconstrictor effects of neurally released norepinephrine, with the result that there is little change of systemic vascular resistance during stress. During the active infusion, the high levels of circulating epinephrine probably exerted a vasodilatory action by stimulating postsynaptic \(\beta_2\)-adrenergic receptors, as evidenced by a decrease in diastolic blood pressure. However, since the stress-induced endogenous release of epinephrine on top of the high circulating epinephrine levels probably produced very little additional increase in \(\beta_2\)-mediated vasodilation, \(\alpha\)-mediated vasoconstriction due to neurally released norepinephrine would be unopposed. This mechanism may have contributed to the augmented pressor response of stress during the infusion of epinephrine.

Excessive sympathetic nervous activity is one possible pathophysiological mechanism in essential hypertension.\(^{35,36}\) It has been suggested that endogenously released epinephrine may exert a prohypertensive effect hours after its plasma concentrations have returned to basal levels and thereby contribute to development of stress-linked hypertension.\(^{11,14,16}\) In an interesting study, Blankestijn and coworkers\(^{18}\) have provided support for a clinical relevance for this hypothesis. A significant pressor effect was observed during 18 hours after a 6-hour infusion of epinephrine, and this pressor effect was more pronounced during periods of sympathetic activation. However, these authors have not specifically provided data to show that the effect was confined to periods of psychological arousal rather than other causes of sympathoadrenal activation. The findings of the present study confirm an amplification of pressor responses to psychological stress during periods of high epinephrine levels. On the other hand, our results do not support the hypothesis that short periods of elevated physiological levels of epinephrine cause prolonged amplifications of stress-induced blood pressure responses after the plasma epinephrine concentration has returned to basal levels. However, it may be that longer periods of elevated epinephrine levels are needed to significantly alter pressor responses to stress after normalization of plasma epinephrine levels.

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