Blood Pressure Responses to Catecholamines during Beta-Adrenergic Blockade with Propranolol in Hypertensive Subjects*

Nicholas D. Vlachakis, M.D.; Daisy DeGuia, M.D.; and Milton Mendlowitz, M.D., F.C.C.P.*

In 14 patients with essential hypertension, the responses of the heart rate and blood pressure to infusion of norepinephrine and epinephrine separately while off (control period) or on therapy with β-adrenergic receptor blockade was examined. By titrating dosage against the response of blood pressure and pulse rate, propranolol hydrochloride was administered orally at 160 mg/day in four divided doses. There was a significant decrease in systolic blood pressure and in pulse rate during propranolol therapy, whereas diastolic blood pressure decreased but not significantly so. Normal blood pressure (140/90 mm Hg or less) was attained only in five patients. The infusion of epinephrine and then norepinephrine produced a significant increase in both systolic and diastolic blood pressure during propranolol therapy, but the magnitude of the rise was significantly greater than that attained in the control period only during epinephrine infusion. We conclude that the transient hypertensive episodes which have been observed during office visits in some hypertensive patients treated with propranolol are due mainly to release of epinephrine.

Beta-adrenergic blocking agents have been suggested for the treatment of essential hypertension for many years and have been considered by some investigators as the treatment of choice in patients with malignant hypertension, renovascular hypertension, and the hyperdynamic β-adrenergic state; however, in severe hypertension, the large doses (1,000 mg of propranolol daily or more) often necessary to attain a clinically satisfactory decrease in the blood pressure, the frequency of inadequate control of blood pressure despite such large doses, and the known untoward effect on the heart and respiration have resulted in some decrease of interest in the use of these drugs alone in the treatment of essential hypertension. Even in mild and moderately severe hypertension, propranolol as the sole anti-hypertensive agent has produced a significant reduction in blood pressure only in a small number of cases.

Furthermore, transient hypertensive episodes during office visits have been observed in patients treated with propranolol alone, and therapy with propranolol did not prevent or reduce the hypertensive response to "stress." This hemodynamic response to “stress” during β-adrenergic blockade has been explained on the basis of the unmasking by propranolol of the effect of catecholamines on the β-adrenergic receptors. During mental stress, the excretion of epinephrine in the urine is increased, and in some cases the excretion of norepinephrine may also be increased. It is not known whether the hypertensive response during propranolol therapy is due to epinephrine or norepinephrine.

The present study was designed to examine the effects of administering norepinephrine and epinephrine separately on the blood pressure and heart rate in patients with essential hypertension treated with the β-adrenergic blocking agent, propranolol.

**MATERIALS AND METHODS**

Fourteen subjects with mild and moderate degrees of severity of essential hypertension were chosen from the hypertensive clinic for the study. Routine investigation consisted of history, physical examination, and laboratory procedures, including a hemogram, urinalysis, six-channel and 12-channel automated studies of blood chemistry, and assay for urinary catecholamine metabolites. A chest x-ray film and electrocardiogram were also obtained. A rapid-sequence intravenous pyelogram was obtained in all subjects. Patients with an abnormal intravenous pyelogram, renal failure, coronary arterial disease, cardiac arrhythmias, or thyroid dysfunction were excluded from the study.

The procedure was explained to each patient, a consent form was signed, and all previous medication was withdrawn.

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Following four weeks of receiving a placebo, (control period), each patient was seen in the circulatory physiology laboratory, where infusions of norepinephrine and epinephrine were administered in a protocol consisting of the following three phases.

Phase A (Basal Period)

Each subject was asked to lie quietly on a bed, and a 5-percent solution of dextrose in water was infused via a butterfly 21 infusion set. Following 45 minutes of complete relaxation, brachial blood pressure was measured with a standard pressure cuff and sphygmomanometer in the left arm, and the average of three consecutive measurements was used for the calculations. Immediately thereafter, the pulse rate was obtained; and, again, the average of three measurements, each for one minute, was used for the calculations.

Phase B (Infusion of Epinephrine)

After vasodilatation was produced by indirect heating with electric blankets until a positive heat balance manifested by profound perspiration was attained, an infusion of epinephrine and trimethaphancamphorsulfonate (trimethaphan camsylate) in 250 ml of a 5-percent dextrose solution was administered for 15 minutes with an infusion pump. Epinephrine was given at a rate of 0.1 µg/kg of body weight per minute and trimethaphan camphorsulfonate at a rate of 0.15 mg/kg of body weight per minute. At the end of the infusion, the blood pressure and pulse rate were measured as described in phase A.

Phase C (Infusion of Norepinephrine)

Keeping the condition in phase B constant and 20 minutes after the discontinuation of the infusion of epinephrine, an infusion of levarterenol bitartrate in 250 ml of a 5-percent dextrose solution, in addition to trimethaphan camphorsulfonate, was given for 15 minutes. Levarterenol bitartrate was administered at rates of 0.1 µg/kg to 0.15 µg/kg of body weight per minute, while trimethaphan camphorsulfonate was given in the same amount as in phase B. From our experience, levarterenol bitartrate at a dosage of 0.1 µg/kg to 0.15 µg/kg of body weight per minute is needed to increase systolic blood pressure by at least 20 mm Hg in an average hypertensive subject, which is necessary for the comparison. Patients with mild hypertension were infused with the highest dose of levarterenol bitartrate, while subjects with more severe hypertension were given 0.1 µg/kg of body weight per minute.

At the end of the infusion, the blood pressure and pulse rate were recorded, as in the previous phase. Under these conditions the blood pressure and pulse rate were found to be stable in three minutes from the beginning of infusion. In five cases, we compared the effect of administering norepinephrine alone with that of administering norepinephrine and trimethaphan camphorsulfonate and found no significant difference with respect to the levels of blood pressure and pulse rate (mean blood pressure and pulse rate were 135 mm Hg and 84 beats per minute, respectively, during norepinephrine infusion vs 132 mm Hg and 84 beats per minute during administration of norepinephrine and trimethaphan camphorsulfonate); however, the use of trimethaphan camphorsulfonate was helpful in stabilizing the circulation, and the fluctuations in results were more pronounced when norepinephrine was used alone (the standard deviation of the means for blood pressure and pulse rate ranged from ±4 to ±9 and from ±2 to ±10, respectively, during norepinephrine infusion vs ±2 to ±3 and ±0.1 to ±1 during administration of norepinephrine and trimethaphan camphorsulfonate).

Following the studies of norepinephrine and epinephrine infusion, propranolol hydrochloride (Inderal) was given orally for two to three months. Dosage was begun at 80 mg/day and was titrated against the response of the blood pressure and pulse rate, up to a maximum dose of 160 mg/day in divided doses. At the end of the propranolol therapy, studies of norepinephrine and epinephrine infusion were repeated exactly as previously described. Norepinephrine and epinephrine were infused at the same rate as in the period when the subjects received the placebo. No attempt was made to test the degree of β-adrenergic blockade by isoproterenol, and the plasma levels of propranolol were not measured; however, each patient took 40 mg of propranolol per os one hour prior to the study, and there was minimal or no significant discrepancy in the pulse rate between the recumbent and upright positions.

RESULTS

Control Period

In Table 1 are outlined the age and sex of patients, as well as the values for blood pressure and pulse rate in the control period. The infusion of epinephrine produced an increase in the mean systolic blood pressure from 164 mm Hg in the basal period to 172 mm Hg (not significant) and a decrease in the mean diastolic blood pressure from 99 mm Hg in the basal period to 86 mm Hg (P < 0.01). A hypertensive response (arbitrarily defined as an increase of 20 mm Hg or more in systolic blood pressure or a rise of 10 mm Hg or more in diastolic blood pressure) was observed in four cases. The mean pulse rate increased significantly during epinephrine infusion (P < 0.001 using the t-test for paired data). The infusion of norepinephrine was associated with a significant increase in both systolic and diastolic blood pressure (P < 0.001) and with a significant decrease in mean pulse rate (P < 0.05). A hypertensive response, as defined herein, was observed in nine subjects.

Propranolol Period

During propranolol therapy (Table 2), both systolic and diastolic blood pressures in the basal period decreased, but only the changes in systolic blood pressure reached a statistically significant level (P < 0.01). The decrease in mean pulse rate was also significant (P < 0.001). The infusion of epinephrine produced a significant increase in systolic and diastolic blood pressure (P < 0.001), and the magnitude of the change was significantly greater than that attained in the control period (using Student's t-test, P < 0.005 for the changes in systolic blood pressure, and P < 0.001 for diastolic blood pressure). The absolute level of diastolic
### Table 1—Blood Pressure and Pulse Rate at Rest and During Norepinephrine and Epinephrine Infusion in the Control Period

<table>
<thead>
<tr>
<th>Patient</th>
<th>Age (yr), Sex</th>
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<th>Epinephrine</th>
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<td>Systolic Diastolic</td>
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<td>170 90</td>
<td>64 56 86</td>
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<tr>
<td>Mean±SE</td>
<td>53±3</td>
<td>164±6 99±2</td>
<td>189±8* 111±2*</td>
<td>172±6 86±6**</td>
<td>83±3</td>
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*P<0.001 compared to basal period.  **P<0.01 compared to basal period.  †P<0.05 compared to basal period.

Blood pressure was also significantly greater than that attained in the control period (P < 0.005). A hypertensive response, as defined previously, was observed in all but one patient. The mean pulse rate decreased significantly during epinephrine infusion (P < 0.001), and the magnitude of the decrease was significantly greater than that in the control period (P < 0.001). The infusion of norepinephrine was associated with a significant rise in both systolic and diastolic pressure (P < 0.01) and with a significant decrease in pulse rate (P < 0.01); however, during propranolol therapy, the magnitude of the changes, as well as the absolute levels of blood pressure attained during norepinephrine infusion, were less than those in the control period, although not significantly so. A hypertensive response was observed in eight patients.

 Plasma renin activity was determined only in seven subjects (subjects 1, 2, 6 to 8, 11, and 14), with specimens obtained at midday after three hours of ambulation and on a regular diet at the end of the placebo period. A 24-hour collection of urine was...

### Table 2—Blood Pressure and Pulse Rate at Rest and During Norepinephrine and Epinephrine Infusion while Receiving Propranolol Therapy

<table>
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<tr>
<th>Patient</th>
<th>Basal Period</th>
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<th>Epinephrine</th>
<th>Pulse Rate, beats per minute</th>
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<td>Systolic Diastolic</td>
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<td>130 90</td>
<td>170 100</td>
<td>180 100</td>
<td>72 56 48</td>
</tr>
<tr>
<td>Mean±SE</td>
<td>152±5* 95±2</td>
<td>169±6** 105±4**</td>
<td>186±8†† 111±4††</td>
<td>64±2¥ 57±2**¥</td>
</tr>
</tbody>
</table>

*P<0.01 compared to control period (no propranolol).  **P<0.01 compared to basal period.  †P<0.005 compared to control period (no propranolol).  ‡P<0.001 compared to control period (no propranolol).
obtained concurrently from each patient for estimation of the excretion of sodium and potassium. Two patients (subjects 1 and 11) had normal plasma renin activity, and five had low plasma renin activity compared to our laboratory's normal renin values. Plasma renin activity was redetermined during propranolol therapy in three patients (subjects 7, 11, and 14) and was found to be significantly depressed in all of them.

**Discussion**

Beta-adrenergic blocking agents, especially propranolol, have been used in the treatment of angina pectoris,16,17 cardiac arrhythmias,18,19 hypertrophic obstructive cardiomyopathy,20 anxiety,21 hyperthyroidism,22 and hypertension.1-9 The mechanisms by which propranolol exerts its antihypertensive effect are not yet exactly delineated, but it seems that the reduction in blood pressure produced by propranolol therapy might be related to its negative inotropic and chronotropic action on the heart,1,28 its antirenin activity,6 and its effect on the central nervous system.24

Although cardiac output is decreased to the same extent by short-term intravenous25 and long-term oral administration of propranolol,9,26 arterial blood pressure is reduced only with long-term administration, due probably to adaptation of the peripheral circulation. Initial studies of hypertensive subjects suggested that the reduction of blood pressure with propranolol therapy was correlated with the pretreatment level of cardiac output.28 It has been also suggested that patients with tachycardia and labile hypertension have a hyperdynamic β-adrenergic state and a better hypotensive response to propranolol;9 however, recent hemodynamic studies failed to show a relation between the antihypertensive effect of propranolol and the pretreatment cardiac output state.9 When propranolol has been used as the sole antihypertensive agent, a significant reduction in blood pressure has been attained only in a small number of patients with mild and moderate severity of essential hypertension.8,9 In our series a significant decrease in systolic blood pressure was observed, while diastolic blood pressure did not change significantly; and a clinically satisfactory reduction in blood pressure (140/90 mm Hg) was attained in only five out of 14 patients. Although the plasma renin activity was determined only in seven patients, the unsatisfactory decrease in blood pressure may be due to the fact that the majority of the patients had low plasma renin activity and some had normal plasma renin activity. A pressor response to propranolol has been observed by Drayer et al27 in 11 percent of 188 patients treated with propranolol for 21 days. All of these patients belonged to the subgroups with low or normal plasma renin activity, and their plasma renin activity did not decrease significantly during propranolol therapy.

Transient hypertensive episodes during propranolol therapy were observed in our series and have been reported by others.8 During mental stress, pain, and apprehension-provoking situations, a twofold to threefold increase in urinary excretion of epinephrine14 was found, although excretion of norepinephrine may also be increased in certain cases.15 Administration of epinephrine in large doses produces vasoconstriction with hypertension and bradycardia,28 whereas in smaller amounts, administration of epinephrine stimulates mainly β-adrenergic receptors.29,30 The rise in blood pressure produced by administration of epinephrine during β-adrenergic blockade seen in the present study has also been reported by others31 and has been explained on the basis of the unmasking by propranolol of the effects of epinephrine on the α-adrenergic receptors.11-13 Hemodynamic studies during infusion of catecholamines5 demonstrated that propranolol converted the dilator effect of epinephrine into vasoconstriction, while the constrictor response to norepinephrine remained unchanged. In studying the effect of propranolol on the pressor responses to noxious stimuli in 11 hypertensive patients, Nicotero et al8 showed that administration of propranolol failed to affect either the basal blood pressure or the pressor response to cold and ischemic pain or to prevent the increase of urinary catecholamine excretion in response to such stimuli.

Blum et al32 have very recently reported the occurrence of progressive or precipitous elevation of blood pressure in eight out of 44 psychiatric patients treated with high doses of propranolol. The hypertension was accompanied by pallor, coldness, and clamminess of the skin and by marked tension and outbursts of psychomotor unrest. In all cases the hypertension responded immediately to the administration of the α-adrenergic blocking agents, phentolamine or phenoxybenzamine.

In the present study, administration of norepinephrine, with an effect on α-adrenergic and β-adrenergic receptors,28 produced a significant increase (P < 0.005) in both systolic and diastolic blood pressures during propranolol therapy; however, these increases did not reach the levels attained in the control period. On the other hand, administration of epinephrine produced a significantly greater rise in blood pressure, as compared to that in the control period (P < 0.005 for systolic and P < 0.001 for diastolic blood pressure).

Similar studies in our laboratory with a diuretic
agent or a peripheral vasodilator drug clearly demonstrated a significant reduction in the blood pressure during catecholamine infusion. The addition of such a drug to \( \beta \)-adrenergic blockade has shown a greater hypotensive effect and will probably lessen the increase in blood pressure during stress. In studying the infusion of norepinephrine and epinephrine into 17 dogs receiving combined administration of phenoxybenzamine and pronethalol, Garrett et al. found a significantly smaller increase in blood pressure during the combined therapy than in the control period. Similar results have also been reported by Gagnon and by others.

In the present study the doses may not be comparable to the physiologic release of catecholamines in man. Furthermore, epinephrine and norepinephrine were administered separately, while in stressful situations of real life, release of both epinephrine and norepinephrine may be increased. Goldenberg et al. observed that the vasoconstrictive effect of norepinephrine was abolished and blood pressure fell when epinephrine was simultaneously administered in equal doses in normotensive subjects not receiving any medication. It seems likely that stress producing epinephrine from the adrenal medulla becomes more vasoconstrictive with propranolol therapy than without and can cause the blood pressure to rise at least transiently; however, paradoxical elevation of blood pressure may occur in any patient treated with \( \beta \)-adrenergic blocking agents when excessive amounts of catecholamines are released, especially in patients with pheochromocytoma or schizophrenic disorders.

REFERENCES

31 Harris WS, Schoenfeld CD, Brooks RH, et al: Effect of \( \beta \)-
adrenergic blockade on the hemodynamic responses to epinephrine in man. Am J Cardiol 17:484, 1966

**Felis Domestica**

The cat has been man’s companion since time immemorial. It is thought that cats appeared in Europe about 700 - 800 BC. According to a census of 1961 there were about 22 million cats in urban households in the United States, in addition to several millions owned by farmers. In the latter instance the cat proved to be an invaluable guardian of staple grains, wheat, corn, rice etc against rodents. In ancient China and Japan cats were kept for the protection of cocoons of silkworms against rats 1,000 years BC. The average life-span of cats is 14-15 years. Their perception of sound is far beyond human hearing. By means of 27 muscles of the external ear the latter can be turned in several directions. Visual adaptation of their eyes is aided by contraction of the pupils to a vertical slit in bright light and their maximal dilatation in dim light. The strange shinning of the cat’s eyes when bright light strikes them is due to the tapetum, a reflecting layer in the choroid. In the religious shrines of ancient Egypt the cat was exalted as a sacred creature; one of their goddesses, Bast, was depicted with the head of a cat. Throughout the ages, cats, paradigms of self-possession, perseverance, tidiness and relaxation, were pampered as pets by humans, including celebrities of religion, science and the seven arts. Cats lack the vociferous xenophobia of dogs. They are faithful servants of man and able to express their affection by rubbing their bodies against one’s arms or legs, or by purring which is produced by a peculiar vibration of their vocal cords with the passing air flow. It is well to cite Manolson, F (C is for Cat, New York, Basic Books, 1965). Catgut is made from the intestine of sheep. These strands of sheep gut were originally used as strings on an Italian musical instrument called a kit. Kitgut doesn’t really sound right, so they started calling it catgut. Lord Lister was the first who used it. Another great English pioneer, Blackley, CH (Exper Res, London, 1880) first recorded the occurrence of hypersensitivity to epidermal scales or dander of animal hair. Symptoms due to allergy to cat dander may develop through direct contact or through staying at a location where cats were or had been, also through exposure to material containing cat hair. Pertinent symptoms include pruritus, infrequent urticaria, conjunctival congestion, itching of the eyes, paroxysmal sneezing, rhinorrhea, tightness in the chest, cough and wheezing. Emmons, CW et al (Am J Hyg 61:40, 1955) found that 18 percent of cats studied harbored *Histoplasma capsulatum*. The latter may be detected in the saliva, urine and feces of infected animals. In 1932, Foshay, L, quoted by Daniels, WB et al (Ann Int Med 37:697, 1952) first identified ulceroglandular disease resulting from the scratch of cats. Thousands of cases of cat-scratch fever have been observed since the report of Debre, R et al (Bull Mem Soc Med Hop Paris, 66:76, 1950). The causal agent is a virus. In addition to localized manifestations and regional lymphadenitis, the salient features of this disease are malaise, headache, chills and fever. Its course is usually benign. Encephalomyelitis is a rare complication. Positive skin test to intracutaneous injection of antigen from a suppurating regional lymphnode is diagnostic. The cat is highly susceptible to tularemia. Rare instances of tularemia transmitted through cat scratch have been reported. Three cases of febrile infection following cat bite were described by Francis, DP et al (JAMA 233:42, 1975). Such infection may result in severe localized cellulitis and associated with septicemia. The causal agent was *Pasteurella multocida*, a gram-negative coccobacillus often present in the oral cavity of healthy cats. Penicillin or a penicillin analogue is considered effective therapy. In reference to cancer, Levy, JA (JAMA 229:1654, 1974) asserts that leukemia virus can be found in the mucus of the oral cavity and of the trachea, and in the urine of unaffected cats and that this cat virus grows well in human cell cultures; even so, no clear-cut association of cat virus with human cancer has been proved. The first presumable case of human rabies in the continental United States in 15 years, originating from catbite was reported in the Medical News section of the JAMA (233:407, 1975). It is hoped that this article is not bringing about ailurophobia.

Andrew L. Banyai, M.D.

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