Learned Control of Blood Pressure in Patients with High Blood Pressure

By DONALD A. KRISTT, M.D. AND BERNARD T. ENGEL, PH.D.

SUMMARY
Five patients with documented histories of essential hypertension of at least ten years' duration participated in a triphasic study of training to control systolic blood pressure (SBP). Phase 1 was a seven week period during which patients took their BP (systolic and diastolic) at home and mailed these data to us daily. Phase 2 was a three week period during which patients were taught to control SBP using a noninvasive technique: patients were trained to raise, to lower and to alternately lower and raise SBP. Phase 3 was a three month period during which patients again took their BP at home and mailed these data to us daily. Results: (1) all patients learned SBP control: average increase — 15%; average decrease — 11%; (2) during SBP control heart rates, breathing rates, triceps brachii muscle tension and EEG activity did not change; (3) follow-up tests at one and three months showed evidence of retained SBP control; (4) baseline SBP fell from 153 mm Hg during laboratory training to 135 mm Hg at the three month follow-up; (5) phase 3 home BP's fell 18/8 mm Hg from phase 1 levels; (6) at home patients also were able to reduce SBP from 141 mm Hg (average) to 125 mm Hg (average) by means of the lowering technique learned in the laboratory.

Additional Indexing Words:
Biofeedback  Operant conditioning  Essential hypertension

SEVERAL LINES OF EVIDENCE suggest that the central nervous system plays an important role in either the genesis or the maintenance of high blood pressure. Exploration of the human brain has identified numerous areas that mediate blood pressure (BP) changes when electrically stimulated, suggesting that there is an anatomic basis for such a role.1 Psychological factors also may be reflected in changes in BP2,3 which some believe to be pathogenetically significant.4,5 In addition, the current therapeutic approach to hypertension emphasizes the use of pharmacologic agents that interfere with autonomic control of the cardiovascular system implying some role for the nervous system in maintaining abnormally elevated BP.

Recent investigations also have shown that BP can be brought under voluntary control in the laboratory using operant conditioning techniques.6-11 This means that a human subject can be taught both to raise and to lower BP to achieve an appropriate level of BP. With regard to individuals with high blood pressure some degree of success has been reported in conditioning lowering of BP.12-14 Relaxation and meditation techniques also have been applied and show promise for BP lowering.15-19 Over-all BP control — i.e., lowering and raising — by hypertensive patients has not been explored. The purpose of this report is to show that hypertensive patients can learn to modify their BP using operant conditioning techniques in the laboratory, and that such learning will persist throughout a three month follow-up period in the laboratory and in a non-laboratory setting, i.e., the home.

Materials and Methods

Patients
Five volunteer patients were recruited from the Hypertension Clinic of the Baltimore City Hospitals. All five patients had been seen in the clinic for at least ten years prior to study, during which time the diagnosis of high blood pressure of unknown origin was confirmed repeatedly. The principal selection criteria were: 1) willingness to adhere to the experimental protocol; and 2) willingness to consent to hospitalization for the three week training period. The five subjects reported here comprise the total study group for this project. Informed consent was obtained from each patient. All patients were seen by one investigator (D.K.) in the clinic for at least 6 months prior to referral to the project. The purpose of this delay was to enable the patient to become familiar with his physician so that the novelty of this aspect of the experiment could be minimized. Biographical and pertinent medical information is summarized in table 1.
Table 1

**Summary of Medical History**

<table>
<thead>
<tr>
<th>Pt.</th>
<th>Age</th>
<th>Sex</th>
<th>Race</th>
<th>HBP (yrs)</th>
<th>Pretraining medication (mg)</th>
<th>Cardiac DX</th>
<th>CXR</th>
<th>Other</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>68</td>
<td>M</td>
<td>B</td>
<td>&gt;20</td>
<td>H 25 q.i.d. HY 50 q.d. G 25 t.i.d.</td>
<td>None</td>
<td>Left ventricular prominence;aortic atherosclerosis</td>
<td>Diabetes mellitus</td>
</tr>
<tr>
<td>2</td>
<td>46</td>
<td>F</td>
<td>W</td>
<td>&gt;10</td>
<td>HY 50 h.i.d. G 37.5 q.d.</td>
<td>Sinus bradycardia</td>
<td>LV prominence</td>
<td>Malignant hypertension</td>
</tr>
<tr>
<td>3</td>
<td>70</td>
<td>F</td>
<td>B</td>
<td>&gt;10</td>
<td>HY 25 q.d. DIG 0.1 q.d.</td>
<td>1° AVB; LVH; CHF</td>
<td>WNL</td>
<td>R CVA-1968; diabetes mellitus</td>
</tr>
<tr>
<td>4</td>
<td>49</td>
<td>F</td>
<td>B</td>
<td>&gt;10</td>
<td>HY 50 h.i.d. A 500 t.i.d.</td>
<td>Angina pectoris; CHF</td>
<td>Cardiomegaly</td>
<td>Obesity; intermittent claudiation</td>
</tr>
<tr>
<td>5</td>
<td>61</td>
<td>F</td>
<td>W</td>
<td>&gt;20</td>
<td>HY 50 h.i.d.</td>
<td>1° AVB; sinus bradycardia</td>
<td>Aortic atherosclerosis; cardiomegaly</td>
<td>Obesity</td>
</tr>
</tbody>
</table>

Abbreviations: HBP = high blood pressure; DX = diagnosis; CXR = chest X-ray; AVB = atrioventricular block; LVH = left ventricular hypertrophy; CHF = congestive heart failure; WNL = normal; CVA = cerebrovascular accident; H = Hydralazine; HY = hydrochlorothiazide; G = guanethadine; A = alpha-methyldopa; DIG = digitoxin.

Experimental Design

This study was arranged in three phases (fig. 1A): Phase 1 was a pretraining assessment period, phase 2 was the laboratory training period, and phase 3 was a posttraining assessment period. Phases 1 and 3 included self-determination of BP by the patients.

**Pretraining**

At least five weeks prior to hospitalization, patients were taught to record their own BP at home. This they did four times each day until admission. Furthermore, these home BPs were mailed to us daily by the patient. This procedure enabled us to monitor the faithfulness of the patient’s adherence to our instructions, and the daily mailing reduced the likelihood that the patient would record spurious values based on data from previous days. Following this pretraining period patients were admitted to the Gerontology Research Center research ward of the Baltimore City Hospitals for 3 weeks. During hospitalization patients were maintained on reduced dosages of their BP drugs to com-

**A. STUDY**

**PHASE:** PRE (7 wks)  
**CONDITION:** DAILY BLOOD PRESSURE  
**TRAINING (3 wks).Decode error occurred: content is not available.**  
**POST (12 wks):** 1 mo TEST 3 mo TEST  
**DURATION:**  

**B. SESSION**

**TRIAL:** BASE REST TRAINING REST 1 to 10 11 to 20 REST 21 to 30  
**NUMBER:**

**C. TRIAL**

**PERIOD:** INFLATION AND STABILIZATION FEEDBACK DEFLECTION REST  
**DURATION:** 17 sec 24 HEART BEATS 1 sec 15 sec  

Figure 1

Schematic diagram of experimental design: A) over-all structure of the study; B) detail of a single session; C) detail of a single trial.

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pensate for the improved care and reduced activity during hospitalization.

Laboratory Training

The technique we used to monitor systolic blood pressure (SBP) is a noninvasive procedure first described by Tursky et al.29 In this method a standard BP cuff is placed around the upper arm and inflated to a fixed pressure where it is maintained for about 30 sec. A microphone is placed over the brachial artery at the distal edge of the cuff and Korotkoff’s sounds (KS) are detected. The cuff pressure equals average SBP when 50% of the heart beats are accompanied by KS. When 25% to 75% of the heart beats are accompanied by KS, cuff pressure is within \( \pm 2 \) mm Hg of average SBP. We used this technique for two purposes: first, to measure SBP as described above; and second, to train patients to control SBP on a beat-to-beat basis as described below.

In the laboratory the patient was recumbent in a hospital bed located in a soundproof room. The experimenter and his equipment were located in an adjacent room. A pair of electrodes were placed on the patient’s chest to detect the ECG. A small crystal microphone was placed over the brachial pulse in the antecubital fossa. A standard BP cuff was applied over the microphone. A KS was detected if it occurred between 250 and 500 msec following an R wave of the ECG.

In front of the patient was a vertical array of red, yellow and green cue lights. Illumination of the red light signaled the patient to lower his SBP. The green light indicated SBP raising was expected. The yellow light remained on as long as the patient continued to perform correctly, which meant producing KS during a raising attempt or decreasing KS — i.e., inhibiting their production — during BP lowering. The yellow light, which provided beat-by-beat feedback to the patient of his ability to modify his SBP, served as a reinforcer since keeping it lit was evidence to the patient that he was successfully controlling his SBP. Another source of reinforcement and feedback was a digital meter adjacent to the light panel which gave the patient a cumulative numerical score of his performance; each successful response advanced the meter by two points.

The first week of training was used to teach the patients to raise SBP. During the second week patients were trained to lower SBP. In the third week patients were trained to lower and to raise SBP alternately within a single session. Thus, during this alternating condition the patients were demonstrating their abilities to control their SBP. There were approximately 14 sessions per week. Patients always were aware of the contingencies which controlled the reinforcement light. They were instructed to modify their BP in accordance with the prevalent experimental conditions. They were never told how to raise or to lower SBP. They were told only that other experiments had shown that it was possible to do so, and that subjects did best when they evolved their own techniques. The subject knew that he could gauge his success on a given trial by noting his cumulative numerical score on the panel meter. In addition, at the end of each session the subject was shown his data and he was told how well or how poorly he had done.

A usual training session (fig. 1B) consisted of a series of pretraining baseline trials and 3 blocks of ten training trials each. A two minute rest period occurred between trial blocks. Training would not begin until the pretraining baseline SBP was stable — i.e., did not vary more than 2 mm Hg from trial to trial for five consecutive trials. The visual feedback was available only during training trials.

A trial (fig. 1C) consisted of a 17 sec cuff inflation period, a 24 heart beat recording period in which panel lights and the digital meter were functional, and a rapid deflation followed by 15 sec of rest in which cue lights were off. During the rest period the numerical score indicated by the digital meter for that trial remained illuminated. It would reset at the beginning of the next trial. The number of successes per trial and the duration of each trial (i.e., the time for 24 R-R intervals) were recorded by means of digital printout recorders for subsequent analyses.

During each session of each condition (raise, lower, alternate) the criterion was made progressively more difficult depending upon the subject’s performance on the preceding trial. For example, if the subject successfully raised his BP on a given trial, the cuff pressure was increased by 2–4 mm Hg on the next trial. If the patient failed to produce between 25% and 75% KS on this trial, the criterion was either not changed or returned to the previous level.

Posttraining

While still in the hospital the patients were taught to use a BP cuff to perform a BP lowering maneuver. The technique required them to make the KS (heard at SBP) disappear while cuff pressure was maintained at the level of SBP. They then determined the new (lower) SBP by slowly releasing the cuff until KS again appeared. Following discharge the patients practiced this technique from 4–30 times each day and made daily records of their performance. In addition, they recorded daily diastolic (DBP) and SBP as they did prior to training for the duration of the three month follow-up period. They returned to the laboratory for a single session test of their ability to alternately raise and lower SBP at the end of the first and third months of follow-up.

Response Measures

During the laboratory phase of the study, in addition to BP and heart rate, we also recorded several other responses. Breathing rate was recorded during two of the alternate sessions. Respiratory excursions were detected by a strain gauge mounted on a belt placed around the chest. Electroencephalograms (EEG) from bilateral occipital placements were recorded during one alternate session; electromyograms (EMG) over the triceps brachii were recorded during one alternate session. The sessions during which breathing rate, EEG and EMG were measured came late in training so that the patient could be expected to evince reliable control of SBP.

Statistical Analyses

Laboratory Training

Statistical analyses were limited to the last four sessions of each training condition since we were interested in stable behavior. There were five baseline trials and 30 training trials during each of these sessions. The baseline value for SBP was that value of SBP which was stable (\( \pm 2 \) mm Hg) over five consecutive trials. Baseline heart rate (HR) was the average for these five trials. Paired consecutive scores during each training session were averaged. Thus, the reduced data sets comprised a matrix of 60 scores (4 sessions \( \times 15 \) paired consecutive trial scores/session), and a vector of four baseline values for each subject and for each experimental condition. Two measures of performance were derived. First, because the criteria were made progressively more
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difficult throughout each session, we expected a monotonic change in SBP across the session. To test the significance of this effect we tested the statistical significance of the linear component of trend. This component is arithmetically identical to the regression coefficient. However, since there is no need to make an assumption of linearity (as one would if he were fitting a straight line to a set of data), we did not do so.

The second measure of performance we used was a count of the number of trials in which the subject’s SBP (or HR) exceeded his baseline SBP (or HR) in the appropriate direction. The probability of obtaining a particular frequency was then tested by using a one-tailed (SBP) or two-tailed (HR) test based on the binomial distribution.

Pre and Posttraining

Pre and posttraining BP values were compared within each subject by means of the t-test. During the posttraining period the patient not only monitored his BP but also tried to lower it as described previously. Analysis of these data was performed by computing an average difference score for each day, and then performing a t-test on the scores.

Laboratory Training

Blood Pressure

All subjects were trained to raise SBP during the first week in the laboratory, and they were trained to lower SBP during the second week. Figure 2 shows the average SBPs produced by the subjects during the last four training sessions of each of these weeks. Table 2 reports the statistical analyses of the lowering and raising data. Trend tests showed that SBP increased across trials reliably for all patients except patient 2. In this patient there was a pattern of substantial fall in SBP (average = 13 mm Hg) early in the session followed by a gradual trial-by-trial raising of SBP (fig. 2). This patient, who had a history of malignant hypertension, noted at one point that "maybe in the back of my mind I really don’t want to raise my pressure." During lowering training patient 1 showed a pattern of substantial elevation of SBP early in the session followed by a gradual and significant trial-by-trial lowering of SBP. All other patients modified their SBPs across trials significantly and in the appropriate directions. With the exception of patient 1, analyses of the changes of SBP from baseline showed that those effects paralleled the trend findings (table 2). Thus it may be concluded that all patients showed evidence of being able to lower SBP and four of the five patients were consistently able to raise SBP after these two weeks of training.

During the final week the ability of the patients to control SBP was tested in a series of sessions in which they were required to lower, raise and then lower SBP again. These sessions also consisted of 30 feedback

Table 2

<table>
<thead>
<tr>
<th>Patient</th>
<th>Baseline SBP (mm Hg)</th>
<th>Coefficient of linear trend (mm Hg/paired trial)</th>
<th>Percent responses above baseline</th>
<th>Baseline SBP (mm Hg)</th>
<th>Coefficient of linear trend (mm Hg/paired trial)</th>
<th>Percent responses below baseline</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>168</td>
<td>154–182</td>
<td>0.6*</td>
<td>0.9*</td>
<td>149</td>
<td>12--142</td>
</tr>
<tr>
<td>2</td>
<td>132</td>
<td>142–171</td>
<td>0.4</td>
<td>0.4*</td>
<td>149</td>
<td>12--142</td>
</tr>
<tr>
<td>3</td>
<td>150</td>
<td>144–155</td>
<td>0.9*</td>
<td>0.9*</td>
<td>149</td>
<td>12--142</td>
</tr>
<tr>
<td>4</td>
<td>164</td>
<td>149–175</td>
<td>1.1*</td>
<td>1.1*</td>
<td>149</td>
<td>12--142</td>
</tr>
<tr>
<td>5</td>
<td>124</td>
<td>109–134</td>
<td>1.3*</td>
<td>1.3*</td>
<td>149</td>
<td>12--142</td>
</tr>
<tr>
<td>Mean</td>
<td>151.6</td>
<td>0.86</td>
<td>74.2</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

*P < .01.

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was variable although all patients were able to modify SBP in one direction at least. Thus, the data from the last week of training indicate that all patients were able to control SBP, some more effectively than others.

Heart Rate, Breathing Rate, EMG and EEG

During each trial we obtained a measure of HR as well as SBP. Despite the changes in SBP which the subjects produced, heart rate did not change systematically with SBP as measured by the coefficient of linear trend (table 4). The only consistent effect seen in the HR data occurred during the raising trials of the first week when HR usually was above baseline.

Table 5 summarizes the data for single sessions of alternate raising and lowering of SBP in which occipital EEG (percent alpha-wave activity per trial) and breathing rate also were recorded in patients 1, 2, 3, 4. There were no noteworthy differences in breathing rate or in the extent of alpha-wave activity between the lowering and raising trials. There was a decline in average alpha activity from baseline to training, however, which suggests that the patients were attending to the feedback light. The absence of sleep spindles further supports the inference that patients were attending to the cues. Electromyograms of the triceps brachii failed to show observable differences between SBP lowering or raising.

Follow-up

Laboratory

Patients returned to the laboratory after one month and after three months of training. They were tested in a single alternating session on each occasion. Figures 4 and 5 present the SBP data during these sessions in which the patients alternately lowered and raised SBP. Extensive statistical analyses of these data are not warranted since there are too few observations. However, visual inspection of the data suggests the following conclusions. 1) The ability of the patients to

Table 3

Individual Responses during Training to Alternate Systolic Blood Pressure

<table>
<thead>
<tr>
<th>Patient</th>
<th>Baseline SBP (mm Hg)</th>
<th>Lower Coefficient of linear trend (mm Hg/paired trial)</th>
<th>Percent responses below baseline</th>
<th>Raise Coefficient of linear trend (mm Hg/paired trial)</th>
<th>Percent responses above baseline</th>
<th>Lower Coefficient of linear trend (mm Hg/paired trial)</th>
<th>Percent responses below baseline</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>165</td>
<td>-2.2*</td>
<td>40</td>
<td>2.6*</td>
<td>80*</td>
<td>-2.7*</td>
<td>45</td>
</tr>
<tr>
<td>2</td>
<td>151</td>
<td>-3.9*</td>
<td>80*</td>
<td>1.0**</td>
<td>5</td>
<td>-2.7*</td>
<td>100*</td>
</tr>
<tr>
<td>3</td>
<td>162</td>
<td>-2.8*</td>
<td>55</td>
<td>2.2*</td>
<td>40</td>
<td>-2.2*</td>
<td>70**</td>
</tr>
<tr>
<td>4</td>
<td>135</td>
<td>-2.2*</td>
<td>90*</td>
<td>2.2**</td>
<td>70**</td>
<td>-0.4</td>
<td>95**</td>
</tr>
<tr>
<td>5</td>
<td>142</td>
<td>-0.8*</td>
<td>65**</td>
<td>1.0**</td>
<td>65**</td>
<td>0.8</td>
<td>20</td>
</tr>
<tr>
<td>Mean</td>
<td>151.0</td>
<td>-2.38</td>
<td>66.0</td>
<td>1.98</td>
<td>52.0</td>
<td>-1.44</td>
<td>66.0</td>
</tr>
</tbody>
</table>

*P < .01.  
**P < .05.
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Table 4

Individual Heart Rate Responses during Systolic Blood Pressure Training

<table>
<thead>
<tr>
<th>Patient</th>
<th>Average (beats/min)</th>
<th>Range</th>
<th>Coefficient of linear trend (beats/min/paired trial)</th>
<th>Percent responses above baseline</th>
<th>Average (beats/min)</th>
<th>Range</th>
<th>Coefficient of linear trend (beats/min/paired trial)</th>
<th>Percent responses below baseline</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>57.2</td>
<td>55.5-58.3</td>
<td>-0.1**</td>
<td>83*</td>
<td>58.4</td>
<td>52.3-60.9</td>
<td>-0.1**</td>
<td>65**</td>
</tr>
<tr>
<td>2</td>
<td>50.0</td>
<td>48.5-51.6</td>
<td>-0.2**</td>
<td>73*</td>
<td>45.5</td>
<td>39.6-52.1</td>
<td>0.1</td>
<td>58</td>
</tr>
<tr>
<td>3</td>
<td>83.4</td>
<td>77.9-88.0</td>
<td>0.0</td>
<td>54*</td>
<td>81.6</td>
<td>73.8-84.7</td>
<td>0.1</td>
<td>62</td>
</tr>
<tr>
<td>4</td>
<td>68.3</td>
<td>67.3-69.2</td>
<td>0.0</td>
<td>75*</td>
<td>72.7</td>
<td>70.0-78.9</td>
<td>-0.4*</td>
<td>60</td>
</tr>
<tr>
<td>5</td>
<td>56.2</td>
<td>47.0-63.0</td>
<td>-0.1</td>
<td>54*</td>
<td>49.2</td>
<td>43.4-56.0</td>
<td>0.0</td>
<td>24*</td>
</tr>
<tr>
<td>Mean</td>
<td>63.0</td>
<td></td>
<td>-0.08</td>
<td>73.2</td>
<td>61.5</td>
<td></td>
<td>-0.06</td>
<td>53.8</td>
</tr>
</tbody>
</table>

**P < .01.
*P < .05.
(two tailed tests).

control SBP is maintained throughout the observation period. 2) With the exception of patient 3, baseline BP falls from the alternate sessions during the last week of training to the 3 month follow-up sessions. Even in patient 3 baseline SBP during the third month follow-up is slightly less than it was during the laboratory training period. These falls in baseline SBP range from 3 to 18% with an average of 11%. 3) Heart rate (which is not presented in a figure) is similar to that during alternate training. There is little change from baseline and there is no trend across trials as appears in SBP. Thus, these data suggest strongly that the patients were able to retain their abilities to control SBP after one month and after two month intervals during which no formal training procedures occurred.

Home Blood Pressures

Figure 6 and table 6 compare home BP records before and after training for patients 1 to 4. SBP decreased significantly in all four patients. Diastolic pressures also decreased significantly in two of these patients. In patient 5 pretraining home BP determinations are not available. However, her home BP records during the follow-up period showed a decrease in SBP of 14 mm Hg from the first to the last week and a decrease in DBP of 8 mm Hg in the same period.

In addition to merely recording blood pressures at home, the patients also practiced controlling their SBP 4 to 30 times each day during the follow-up period. These results also were mailed to the

Table 5

Systolic Pressure, Heart Rate, Alpha-wave Activity and Breathing Rate during SBP Alternation Sessions*

<table>
<thead>
<tr>
<th>Patient</th>
<th>SBP</th>
<th>HR</th>
<th>EEG</th>
<th>Breathing rate</th>
<th>SBP trend</th>
<th>HR trend</th>
<th>EEG</th>
<th>Breathing rate</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>155</td>
<td>60.0</td>
<td>38</td>
<td>12</td>
<td>-4.3</td>
<td>-0.1</td>
<td>43</td>
<td>11</td>
</tr>
<tr>
<td>2</td>
<td>151</td>
<td>64.5</td>
<td>51</td>
<td>12</td>
<td>-1.6</td>
<td>-0.3</td>
<td>27</td>
<td>13</td>
</tr>
<tr>
<td>3</td>
<td>145</td>
<td>44.6</td>
<td>48</td>
<td>10</td>
<td>-4.0</td>
<td>0.0</td>
<td>34</td>
<td>10</td>
</tr>
<tr>
<td>4</td>
<td>122</td>
<td>65.1</td>
<td>97</td>
<td>14</td>
<td>-0.8</td>
<td>-0.5</td>
<td>86</td>
<td>13</td>
</tr>
<tr>
<td>Mean</td>
<td>143.2</td>
<td>58.6</td>
<td>58.5</td>
<td>12.0</td>
<td>-2.68</td>
<td>-0.22</td>
<td>47.5</td>
<td>11.8</td>
</tr>
</tbody>
</table>

*These sessions comprised only one lower and one raise condition.

Abbreviations: SBP (mm Hg); SBP trend (mm Hg/paired consecutive trial); HR (beats/min); HR trend (beats/min/paired consecutive trial); EEG (percentage time alpha-wave activity); breathing rate (breath/min).

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The mean lowering of SBP that each patient achieved is shown in Table 7. The average amount of lowering for all patients was 16 mm Hg. In order to estimate the validity of the home SBP control finding, three of the patients (1, 2 and 3) were tested while in the clinic by a staff physician other than one of us. These results were: patient 1, -5 mm Hg; patient 2, -10 mm Hg; patient 3, -10 mm Hg.

Discussion

The results of this study confirm and substantially extend the findings of Benson et al.\textsuperscript{12} that patients with high blood pressure can learn to lower their SBP while in the laboratory. We have shown that these patients not only can be taught to lower pressure but also can be taught to raise pressure and alternately to lower and to raise pressure. Furthermore, the results of this study show that the skills learned in the laboratory persist for at least three months.

Our findings also offer some suggestions about the mechanisms of learned SBP control. Heart rates did not undergo changes parallel to those of SBP in the laboratory initially or in either of the follow-up sessions. During training of BP lowering or raising, BP changed gradually and steadily in the 45 min training period.
periods occasionally reaching levels of 40–50 mm Hg above or below baseline, still without alterations in HR.* Moreover, at home the decline in DBP paralleled that for SBP. These findings suggest that our patients may have been controlling SBP by regulating peripheral vascular resistance directly. We plan to try to identify these mechanisms in subsequent studies.

The laboratory findings that brain alpha-wave activity, breathing rate and triceps brachii muscle tension do not change during SBP control periods in highly trained subjects is interesting. Since each of these indices has been used as a criterion of relaxation, and since there were no differences between the SBP raise and SBP lower periods, the findings suggest that differential relaxation was not the mechanism by which our patients controlled SBP. These findings are especially relevant since other investigators15, 17–19 have been training patients with high blood pressure to relax in order to clinically control their BP. Although we wish to emphasize that learning techniques such as we have used, and relaxation techniques such as those just cited are not mutually exclusive, they do seem to be different. At least one of the relaxation methods that has been employed, transcendental meditation, produces decreases in heart rate and increases in EEG alpha-wave activity.21 As we have just noted, none of these responses corresponded with SBP in our patients. Another point of difference between these two techniques is the return of BP within two weeks to pretraining levels following cessation of four weeks of relaxation therapy.19 These observations also would support the view that the effect of hospitalization in our study (with decreased patient activity and the increased attention given to the patients) is an unlikely mechanism to account for the prolonged and progressive declines in BP we have reported.

One factor which we believe to be important in the outcome of our study is the home blood pressure recording technique which each of our patients learned. This technique had several effects. From our point of view it enabled us to assess each patient’s BP. The requirement that the patient return his results to us daily permitted us to maintain a regular check on the patient’s cooperation, and it reduced the risk that he would enter data on his work sheet without actually taking his BP. From the patient’s point of view it afforded him a regular check on his BP so that he could objectively chart his own progress throughout the study. In addition the self-control maneuver which he utilized during the posttraining period gave the patient evidence confirming his ability to regulate his own BP. A similar finding was reported by Benson et al.19 with patients who practiced meditation throughout one month follow-up period. In their study patients with borderline high blood pressure who learned to meditate, and who continued to practice meditation, showed a drop of 7/4 mm Hg. It also is interesting to compare these data and the data of the present study with the findings of Brady et al.18 in which no sustained depressor effect was observed after their patients discontinued relaxation practice.

In three patients (1, 2, 4) the reduced medication schedule that was employed in the hospital has been subsequently maintained. As a result the home posttraining BP records may be understating the magnitude of sustained lowering effects. The most striking example of BP reduction in the face of significant reduction in antihypertensive medications is patient 4. This patient entered training with mean home BP of 168/117 and was being treated with alpha-methyldopa, 500 mg t.i.d., and hydrochlorothiazide 50 mg b.i.d. Clinically her BP had been progressively rising and would have required a change in her medication schedule. Following training her

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*For example, during the last raising session patient 3 had a maximum change of +55 mm Hg: Baseline SBP = 152, heart rate = 88 beats/min; in trial 4, SBP = 156, heart rate = 90; in trial 16, SBP = 160, heart rate = 90; in trial 24, SBP = 205, heart rate = 87. During the last lowering session patient 2 had a maximum change of −45 mm Hg: Baseline SBP = 161, heart rate = 40; in trial 4, SBP = 138, heart rate = 42; in trial 16, SBP = 127, heart rate = 41; in trial 30, SBP = 116, heart rate = 46.

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**Table 6**

<table>
<thead>
<tr>
<th>Patient</th>
<th>Pretraining SBP (mm Hg)</th>
<th>Pretraining DBP (mm Hg)</th>
<th>Posttraining SBP (mm Hg)</th>
<th>Posttraining DBP (mm Hg)</th>
<th>Decline in SBP (mm Hg)</th>
<th>Decline in DBP (mm Hg)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>151</td>
<td>78</td>
<td>142</td>
<td>76</td>
<td>9*</td>
<td>2</td>
</tr>
<tr>
<td>2</td>
<td>171</td>
<td>93</td>
<td>162</td>
<td>92</td>
<td>9*</td>
<td>1</td>
</tr>
<tr>
<td>3</td>
<td>160</td>
<td>90</td>
<td>141</td>
<td>83</td>
<td>19*</td>
<td>7*</td>
</tr>
<tr>
<td>4</td>
<td>168</td>
<td>117</td>
<td>132</td>
<td>97</td>
<td>30*</td>
<td>20*</td>
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<tr>
<td>Mean</td>
<td>162.5</td>
<td>94.5</td>
<td>144.2</td>
<td>87.0</td>
<td>18.2</td>
<td>7.5</td>
</tr>
</tbody>
</table>

*P < .01.

---

**Table 7**

<table>
<thead>
<tr>
<th>Patient</th>
<th>SBP before lowering (mm Hg)</th>
<th>SBP after lowering (mm Hg)</th>
<th>Amount of lowering (mm Hg)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>138</td>
<td>128</td>
<td>10*</td>
</tr>
<tr>
<td>2</td>
<td>170</td>
<td>158</td>
<td>12*</td>
</tr>
<tr>
<td>3</td>
<td>141</td>
<td>119</td>
<td>22*</td>
</tr>
<tr>
<td>4</td>
<td>129</td>
<td>115</td>
<td>14*</td>
</tr>
<tr>
<td>5</td>
<td>126</td>
<td>103</td>
<td>21*</td>
</tr>
<tr>
<td>Mean</td>
<td>141</td>
<td>125</td>
<td>15.8</td>
</tr>
</tbody>
</table>

*P < .01.
mean home BP was 131/96 and she was taking only 250 mg t.i.d. of the alpha-methyldopa, and the hydrochlorothiazide dosage was unchanged.

Acknowledgment

We wish to acknowledge the assistance of L. R. Baker, M.D., R. Quilter and R. Mathias.

References

Learned control of blood pressure in patients with high blood pressure.
D A Kristt and B T Engel

*Circulation*. 1975;51:370-378
doi: 10.1161/01.CIR.51.2.370
*Circulation* is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
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Print ISSN: 0009-7322. Online ISSN: 1524-4539

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