Hemodynamic Effects of Pargyline in Hypertensive Patients

By Gaddo Onesti, M.D., Paul Novack, M.D., Osvaldo Ramirez, M.D., Albert N. Brest, M.D., and John H. Moyer, M.D.

The use of monoamine oxidase (MAO) inhibitors in the treatment of hypertension has been limited until recently, mainly because the available compounds had a high incidence of accompanying side reactions. However, the non-hydrazine MAO inhibitor, pargyline, appears to be an effective drug with a relatively low incidence of untoward effects. Clinical studies have substantiated that pargyline is a potent antihypertensive agent. Nonetheless, the evaluation of a new hypotensive drug is incomplete without an assessment of the hemodynamic characteristics of the compound. It is the purpose of this report to describe the cardiorenal hemodynamic response to pargyline in hypertensive patients.

Methods and Material

Pargyline was administered orally to eight hospitalized patients with essential hypertension. All antihypertensive medications were discontinued for at least 4 weeks prior to the hemodynamic studies, and each patient was maintained on a regular (5 Gm.) sodium diet.

The hemodynamic studies were performed in the fasting state except for hydration with 500 ml. of tap water given 1 hour prior to the procedure. Cardiac output was determined by the dye-dilution technic. Indocyanine green and a Gilford densitometer were used. Intra-arterial blood pressures were recorded from the brachial artery with a Statham strain-gage transducer. Pulse pressures and dye curves were recorded on a photographic oscillograph. Renal plasma flow was determined by para-aminohippurate clearance and glomerular filtration rate was measured by inulin clearance. Total peripheral resistance was calculated from the mean arterial pressure (obtained by electronic integration) and cardiac output. Renal vascular resistance was calculated from the mean arterial blood pressure and the renal blood flow according to the Gomez formula. The reported results in each case represent the average of three determinations. The renal function values were corrected to 1.73 square meters of body surface area.

After a 45-minute rest in the supine position on a standard tilt table, cardiac and renal hemodynamic studies were performed in eight subjects. The cardiac and renal studies were synchronized, with the cardiac outputs being performed during the middle of each clearance period. After three determinations each subject was passively tilted 45 degrees upright and the cardiac and renal studies were repeated in this position. Following the control determinations, pargyline was administered orally in dosages ranging from 75 to 200 mg. daily. The maximum antihypertensive response occurred from 10 to 28 days after the initiation of drug therapy; and the cardiac and renal hemodynamic studies were repeated at this time, again in the supine and tilted positions.

Results

Supine Response

The hemodynamic effects of pargyline in the supine position are recorded in table 1. Significant blood pressure reduction was observed in seven of eight patients after drug administration. The average reduction was 26 per cent ($p < 0.05$). There was no consistent change in heart rate. Cardiac output and stroke volume were not significantly altered. There was a consistent reduction in total peripheral resistance in all cases from an average of 2,451 to 1,913 dynes sec. cm.$^{-5}$ ($p < 0.01$).

During the antihypertensive response, renal blood flow decreased in five patients and increased in the remaining three. Changes in this factor were not statistically significant. The glomerular filtration rate decreased in all...
### Table 1

**Hemodynamic Response after Oral Pargyline (Supine Position)**

<table>
<thead>
<tr>
<th>Patient</th>
<th>MAP</th>
<th>CO</th>
<th>HR</th>
<th>SV</th>
<th>TPR</th>
<th>RPF</th>
<th>RBF</th>
<th>GFR</th>
<th>FF</th>
<th>RVR</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. E.C.</td>
<td>115</td>
<td>96</td>
<td>4.06</td>
<td>3.73</td>
<td>66</td>
<td>72</td>
<td>61</td>
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<td>2056</td>
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<tr>
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<td>5.70</td>
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<td>70</td>
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<td>3. M.P.</td>
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<td>86</td>
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<tr>
<td>4. R.S.</td>
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<td>68</td>
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<tr>
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<td>5.73</td>
<td>7.46</td>
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<td>77</td>
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<tr>
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<td>4.55</td>
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<td>4.98</td>
<td>5.09</td>
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<td>73</td>
<td>67</td>
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</tr>
<tr>
<td>% of control</td>
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<td>102</td>
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<td>103</td>
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<td>99</td>
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</tbody>
</table>

P value: <0.05

C, control (supine); R, response to pargyline (supine); MAP, mean arterial blood pressure (mm. Hg); CO, cardiac output (liters/min.); HR, heart rate (beats/min.); SV, stroke volume (ml./min.); TPR, total peripheral resistance (dyne.sec.cm⁻¹); RPF, renal plasma flow (ml./min.); RBF, renal blood flow (ml./min.); GFR, glomerular filtration rate (ml./min.); FF, filtration fraction; RVR, renal vascular resistance (dyne.sec.cm⁻² x 10⁵).

### Table 2

**Hemodynamic Response after Oral Pargyline (Erect Position)**

<table>
<thead>
<tr>
<th>Patient</th>
<th>MAP</th>
<th>CO</th>
<th>HR</th>
<th>SV</th>
<th>TPR</th>
<th>RPF</th>
<th>RBF</th>
<th>GFR</th>
<th>FF</th>
<th>RVR</th>
</tr>
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<tbody>
<tr>
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<td>2084</td>
<td>1653</td>
</tr>
<tr>
<td>2. R.W.</td>
<td>150</td>
<td>101</td>
<td>4.59</td>
<td>4.66</td>
<td>69</td>
<td>90</td>
<td>66</td>
<td>52</td>
<td>2611</td>
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</tr>
<tr>
<td>3. M.P.</td>
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<td>90</td>
<td>4.62</td>
<td>4.00</td>
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<td>90</td>
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<td>4.21</td>
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<td>54</td>
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</tr>
<tr>
<td>5. H.M.</td>
<td>138</td>
<td>118</td>
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<td>3.83</td>
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<td>82</td>
<td>40</td>
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<tr>
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<td>3.40</td>
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<td>82</td>
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<td>41</td>
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<td>7. M.B.</td>
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<td>126</td>
<td>4.80</td>
<td>6.09</td>
<td>98</td>
<td>92</td>
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<td>8. C.C.</td>
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<td>50</td>
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<tr>
<td>% of control</td>
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<td>77</td>
<td>74</td>
<td>66</td>
<td>88</td>
<td>82</td>
</tr>
</tbody>
</table>

P value: <0.01

C, control (erect); R, response to pargyline (erect); MAP, mean arterial blood pressure (mm. Hg); CO, cardiac output (liters/min.); HR, heart rate (beats/min.); SV, stroke volume (ml./min.); TPR, total peripheral resistance (dyne.sec.cm⁻¹); RPF, renal plasma flow (ml./min.); RBF, renal blood flow (ml./min.); GFR, glomerular filtration rate (ml./min.); FF, filtration fraction; RVR, renal vascular resistance (dyne.sec.cm⁻² x 10⁵).
cases \((p < 0.05)\), with the filtration fraction tending to decrease \((p < 0.05)\). Renal vascular resistance was reduced in five cases and increased in the remaining three.

**Erect Response**

The hemodynamic effects of pargyline in the erect position are recorded in table 2. A significant blood pressure reduction was observed in each case after drug administration, but the changes in heart rate were again inconsistent. The average blood pressure reduction was 31 per cent \((p < 0.01)\). Cardiac output and stroke volume were not substantially altered except in one patient (case 1), in whom a profound hypotensive effect was accompanied by a marked fall in cardiac output. In contrast, total peripheral resistance was consistently reduced in all cases from an average of 2,872 to 2,000 dynes sec. cm.\(^{-5}\) \((p < 0.01)\).

Renal blood flow decreased in five cases and increased in the remaining three. Changes in this parameter were not statistically significant. The glomerular filtration rate decreased in all cases \((p < 0.01)\), with the filtration fraction tending to decrease \((p < 0.01)\). Renal vascular resistance increased in two cases and decreased in the remaining six.

**Effect of Tilting**

In the control studies (table 3), passive head-up tilting produced no consistent effect on mean arterial blood pressure. The tilting maneuver did result in a decrease in cardiac output and stroke volume along with an increase in heart rate and total peripheral resistance. Decrease in renal blood flow averaged 14 per cent \((p < 0.05)\) in the upright position, and glomerular filtration rate also tended to decrease. Renal vascular resistance was consistently increased by tilting \((p < 0.01)\).

The hemodynamic effects of passive head-up tilting during the hypotensive response to pargyline are recorded in table 4. Tilting again resulted in a fall in cardiac output and stroke volume with an increase in heart rate, the changes being similar in magnitude to the control effects. In contrast with the control studies, however, significant increase in total peripheral resistance with tilting did not occur. The resultant average drop in mean arterial pressure was 18 per cent \((p < 0.05)\). Renal blood flow and glomerular filtration rates were sharply reduced by tilting, the magnitude of the reduction (average 23 per cent) being much greater than that observed during the control studies. Renal vascular resistance did not show any significant changes with tilting during treatment with pargyline.

**Discussion**

The hemodynamic studies obtained with oral pargyline demonstrate that the drug lowers blood pressure by decreasing peripheral vascular resistance, whereas cardiac output is not significantly affected either in the supine or the standing position (see tables 1 and 2). Although a greater orthostatic antihypertensive response is obtained, significant blood pressure reduction occurs in the supine position as well.

The cardiorenal hemodynamic response to tilting observed during the control study is similar to that previously described in normal subjects and in patients with essential hypertension (table 3). It would appear that the physiologic postural reflexes, which under normal circumstances maintain the mean arterial pressure following tilting, are impaired by pargyline therapy. This is amply demonstrated by the failure of peripheral resistance to increase after tilting in the patients receiving pargyline (table 4). As a result, significant orthostatic reduction in mean arterial pressure occurs.

The hemodynamic patterns observed after pargyline therapy are similar to those reported with another MAO inhibitor, RO-4-1038. Since the various pharmacologic members of individual antihypertensive drug groups tend to be similar hemodynamically, it is likely that other MAO inhibitors will present this common pharmacodynamic pattern, i.e., predominant orthostatic blood pressure reduction due primarily to decreased peripheral resistance and unrelated to changes in cardiac out-
Table 3

**Effect of Passive Head-up Tilting (Control)**

<table>
<thead>
<tr>
<th>Patient</th>
<th>S MAP T</th>
<th>S CO T</th>
<th>S HR T</th>
<th>S SV T</th>
<th>S TPR T</th>
<th>S RPF T</th>
<th>S RBF T</th>
<th>GFR T</th>
<th>FF T</th>
<th>RVR T</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. E.G.</td>
<td>115</td>
<td>115</td>
<td>4.06</td>
<td>4.41</td>
<td>66</td>
<td>76</td>
<td>61</td>
<td>58</td>
<td>2265</td>
<td>2084</td>
</tr>
<tr>
<td>2. R.W.</td>
<td>143</td>
<td>150</td>
<td>5.38</td>
<td>4.59</td>
<td>59</td>
<td>59</td>
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<td>2098</td>
</tr>
<tr>
<td>4. R.S.</td>
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<td>183</td>
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<td>4.62</td>
<td>80</td>
<td>83</td>
<td>62</td>
<td>54</td>
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<td>5. H.M.</td>
<td>136</td>
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<td>8. C.G.</td>
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</table>

S, supine (control); T, tilted (control); MAP, mean arterial blood pressure (mm Hg); CO, cardiac output (liters/min.); HR, heart rate (beats/min.); SV, stroke volume (ml/min.); TPR, total peripheral resistance (dyne sec. cm⁻²); RPF, renal plasma flow (ml/min.); RBF, renal blood flow (ml/min.); GFR, glomerular filtration rate (ml/min.); FF, filtration fraction; RVR, renal vascular resistance (dyne sec. cm⁻² x 10⁸).

Table 4

**Effect of Passive Head-up Tilting During the Hypotensive Response to Pargyline**

<table>
<thead>
<tr>
<th>Patient</th>
<th>S MAP T</th>
<th>S CO T</th>
<th>S HR T</th>
<th>S SV T</th>
<th>S TPR T</th>
<th>S RPF T</th>
<th>S RBF T</th>
<th>GFR T</th>
<th>FF T</th>
<th>RVR T</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. E.G.</td>
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<td>3.73</td>
<td>2.61</td>
<td>72</td>
<td>96</td>
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</tr>
<tr>
<td>2. R.W.</td>
<td>142</td>
<td>101</td>
<td>5.70</td>
<td>4.66</td>
<td>70</td>
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<td>1951</td>
<td>1789</td>
</tr>
<tr>
<td>4. R.S.</td>
<td>124</td>
<td>120</td>
<td>4.57</td>
<td>4.21</td>
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<td>58</td>
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<td>2283</td>
</tr>
<tr>
<td>5. H.M.</td>
<td>114</td>
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<td>4.86</td>
<td>3.83</td>
<td>68</td>
<td>82</td>
<td>62</td>
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<tr>
<td>6. L.G.S.</td>
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<tr>
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</table>

S, supine (with pargyline); T, tilted (with pargyline); MAP, mean arterial pressure (mm Hg); CO, cardiac output (liters/min.); HR, heart rate (beats/min.); SV, stroke volume (ml/min.); TPR, total peripheral resistance (dyne sec. cm⁻²); RPF, renal plasma flow (ml/min.); RBF, renal blood flow (ml/min.); GFR, glomerular filtration rate (ml/min.); FF, filtration fraction; RVR, renal vascular resistance (dyne sec. cm⁻² x 10⁸).
put. This pattern is similar to that observed with alpha methyl dopa. In contrast, guanethidine and the ganglioplegic drugs exert their antihypertensive effects via a predominant reduction in cardiac output, whereas peripheral resistance is not significantly altered. 

Methyl dopa produces a consistent decrease in renal vascular resistance without significant change in renal blood flow or glomerular filtration rate. In contrast, the ganglion-blocking agents and guanethidine cause a significant reduction in renal blood flow and glomerular filtration rate. As compared with these other antihypertensive agents, pargyline appears to have a more detrimental effect upon renal hemodynamics than methyl dopa but a somewhat more favorable effect than guanethidine or ganglioplegic drugs.

During recent years, numerous experimental studies have been conducted in an attempt to elucidate the antihypertensive mechanism of the monoamine oxidase inhibitors. Studies on the effect of MAO inhibitors on the isolated superior cervical ganglion and on ganglionic transmission in the intact animal plus clinical observations that certain MAO inhibitors produce parasympatholytic side effects have suggested to some investigators that the hypotensive properties of the MAO inhibitors may result from ganglionic blockade. From a hemodynamic standpoint, however, the dissimilarity between the cardiorenal effects of the ganglion-blocking drugs and those of pargyline is not likely due to ganglionic blockade.

It has also been postulated that the MAO inhibitors may produce sympathetic blockade at a site distal to the autonomic ganglia. The difference in hemodynamic effects observed between pargyline and guanethidine or bretylium suggests that the antihypertensive action of the MAO inhibitors is probably not related to interference with catecholamine storage or release at the postganglionic nerve terminals. On the other hand, the available studies do not rule out the possibility that the antihypertensive response is related to some alteration in catecholamine metabolism. At this time, it would appear that the mechanism of action of the MAO inhibitors remains uncertain despite numerous pharmacologic, clinical, and hemodynamic studies.

Summary

Pargyline exerts its antihypertensive effect via a predominant reduction in peripheral vascular resistance, whereas cardiac output is not altered significantly. The effect on renal blood flow is inconsistent, but a significant reduction in glomerular filtration rate does occur. Therefore the drug should be used with appropriate caution in patients with significant impairment of renal function. The hemodynamic response observed with pargyline is similar to that obtained with other MAO inhibitors.

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into our individual lives and within which a lawyer may and does give advice to his 
client and acts in his behalf. This has been called “lawyer’s law.” In a similar manner 
a physician advises and works for his patient. This could be called “doctor’s medicine”— 
if not confused with some mixture in a bottle. One may lose confidence in a lawyer, 
but not in the law; in a doctor or in what is in the bottle, but not in medicine as an 
institution. Were we to renounce our confidence in government under law, our society 
would revert to anarchy or totalitarian dictatorship; loss of confidence in medicine as 
an institution would expose us to the plagues and pestilence of the Middle Ages and 
very likely carry us back to some primitive tribal way of life.—Introduction, EDWARD D. 
CHURCHILL, M.D. Listen to Leaders in Medicine. Edited by ALBERT LOVE and JAMES 
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GADDO ONESTÍ, PAUL NOVACK, OSVALDO RAMIREZ, ALBERT N. BREST
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