Clinical Characteristics of Hypertension Associated with Unilateral Renal Disease

By George A. Perera, M.D., and Arthur W. Haelig

A comparison has been made of 20 patients with unilateral renal disease, apparently cured of their hypertension by nephrectomy, and of another group of 20 who showed persistence of elevated blood pressure readings after surgery. Evidence is presented that unilateral renal disease, when it is responsible for hypertension, produces an acute, severe and accelerated process different in many respects from the usual picture of essential hypertension or of chronic bilateral renal disorders. The diagnostic value and the possible bearing of this observation on the mechanisms of renal hypertension are discussed.

It is now established that unilateral renal pathology may be responsible for hypertension in rare instances, as demonstrated by apparent cure following the removal of the kidney which is at fault. This subject, together with descriptions of the rigid criteria necessary before conclusions can be drawn, has been reviewed extensively.1-3

The clinical features of this form of elevated blood pressure have not received much attention. For this reason, certain characteristics of the disease picture have been investigated with the hope that they may prove of additional diagnostic value and throw some light on underlying mechanisms of renal hypertension.

Clinical Material

Several hundred records were examined, including most of the published literature dealing with hypertension due to unilateral renal disease. From this material, patients were selected from the literature, from the records of the Presbyterian Hospital and from private patient records, provided that they fulfilled certain criteria. Each subject, to be included in this survey, was required to have had definite hypertension (multiple readings above 140/90), adequate examination of the ocular fundi prior to surgery, and restoration of normal blood pressure readings (at least five values less than 140/90 after discharge from the hospital and remaining normotensive for a minimum period of observation of one year) following removal of a diseased kidney. Twenty such patients were found.

Results

Some of the clinical data as well as the pathologic findings in the excised kidney are summarized in table 1. No patient in this group developed a clear-cut cerebral vascular accident and only one complained of cardiac pain, limited to a six months' period before but not after operation. Congestive failure, relieved by the surgical procedure, was clearly evident in but one instance, and was suggested in two additional cases by history alone. The findings on urinalysis were omitted intentionally from this study because of the obvious difficulty of relating abnormalities in the urine to vascular disease when their basis was primarily renal disease.

Twelve of the 20 patients, whose ages were scattered throughout seven decades, were male. None gave a story of antecedent hypertension of long duration. In 11 instances a normal blood pressure had been recorded within six years of the time of operation, and 10 of these were normotensive within four years.
On at least one occasion, diastolic values of 130 or over were noted in 17 of the series. Infection was generally responsible for the disease found in the removed kidney.

In association with the hypertension which preceded surgery, moderate to severe headaches were apparent in three-quarters of the group, four developedconvulsions, and 14 of the 20 showed definite retinopathy including hemorrhages, exudates or papilledema or a combination thereof. Of the six patients who failed to have significantretinal changes, high diastolic blood pressure values, of headaches and convulsions, of advanced retinopathy in almost all but young children, lends support to this concept. Furthermore, the conspicuously short history of illness suggests that the process is of a rapidly progressive nature from its inception.

In these patients in whom a causal relationship is established between hypertension and unilateralrenal disease, it would thus appear that the usual clinical pattern is of an accelerated nature. Conceivably, however, many com-

Table 1.—Clinical and Pathologic Data in 20 Patients with Hypertension Related to Unilateral Renal Disease

<table>
<thead>
<tr>
<th>No. and Ref.</th>
<th>Sex</th>
<th>Age (years)</th>
<th>Hypertension</th>
<th>B.P.</th>
<th>Retinitis</th>
<th>Convulsion</th>
<th>Pathology</th>
<th>Followed (Yrs)</th>
<th>Systolic Range</th>
<th>Diastolic Range</th>
<th>No. of B.P. Readings</th>
<th>Residua</th>
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<tr>
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<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>14</td>
<td>M</td>
<td>21(25)</td>
<td>150-200</td>
<td>106-134</td>
<td>0</td>
<td>0</td>
<td>+</td>
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<td>3</td>
<td>92-120</td>
<td>62-75</td>
<td>5</td>
</tr>
<tr>
<td>24</td>
<td>M</td>
<td>3</td>
<td>145-175</td>
<td>85-120</td>
<td>0</td>
<td>0</td>
<td>+</td>
<td>Partial arterial occlusion</td>
<td>3</td>
<td>92-122</td>
<td>50-76</td>
<td>21</td>
</tr>
<tr>
<td>24</td>
<td>M</td>
<td>6</td>
<td>115-240</td>
<td>90-140</td>
<td>0</td>
<td>0</td>
<td>+</td>
<td>Pyleonephritis</td>
<td>11/2</td>
<td>90-100</td>
<td>60-80</td>
<td>12</td>
</tr>
<tr>
<td>47</td>
<td>M</td>
<td>7</td>
<td>130-200</td>
<td>98-170</td>
<td>0</td>
<td>0</td>
<td>+</td>
<td>Hydrenephrosis</td>
<td>1</td>
<td>90-110</td>
<td>60-90</td>
<td>7</td>
</tr>
<tr>
<td>53</td>
<td>F</td>
<td>3</td>
<td>170-225</td>
<td>110-178</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>Pyleonephritis</td>
<td>5</td>
<td>102-110</td>
<td>70-85</td>
<td>6</td>
</tr>
<tr>
<td>60</td>
<td>F</td>
<td>19</td>
<td>175-239</td>
<td>125-150</td>
<td>0</td>
<td>0</td>
<td>+</td>
<td>Nephrocleroses</td>
<td>3</td>
<td>140/85 or less</td>
<td>70-90</td>
<td>0</td>
</tr>
<tr>
<td>710</td>
<td>F</td>
<td>20</td>
<td>210-240</td>
<td>120</td>
<td>0</td>
<td>0</td>
<td>+</td>
<td>Pyleonephritis</td>
<td>2</td>
<td>110-122</td>
<td>72-82</td>
<td>4+</td>
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<tr>
<td>8</td>
<td>F</td>
<td>23</td>
<td>130-245</td>
<td>80-145</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>Nephrocleroses</td>
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<td>110-146</td>
<td>70-86</td>
<td>6</td>
</tr>
<tr>
<td>9</td>
<td>M</td>
<td>25</td>
<td>194-220</td>
<td>118-150</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>Pyleonephritis</td>
<td>2</td>
<td>100-126</td>
<td>60-82</td>
<td>10+</td>
</tr>
<tr>
<td>111</td>
<td>M</td>
<td>32</td>
<td>150-210</td>
<td>114-130</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>Multiple infarcts</td>
<td>2</td>
<td>120-124</td>
<td>78-84</td>
<td>8</td>
</tr>
<tr>
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<td>F</td>
<td>35</td>
<td>160-190</td>
<td>90-110</td>
<td>0</td>
<td>0</td>
<td>+</td>
<td>Arterial obstruction</td>
<td>11/2</td>
<td>110-120</td>
<td>70-89</td>
<td>9</td>
</tr>
<tr>
<td>12</td>
<td>M</td>
<td>32</td>
<td>190-250</td>
<td>128-160</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>Tuberculosis</td>
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<td>104-136</td>
<td>70-86</td>
<td>15</td>
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<tr>
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<td>F</td>
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<td>169-220</td>
<td>90-130</td>
<td>0</td>
<td>0</td>
<td>+</td>
<td>Pyleonephritis</td>
<td>2</td>
<td>110-140</td>
<td>70-90</td>
<td>12</td>
</tr>
<tr>
<td>143</td>
<td>M</td>
<td>41</td>
<td>220-240</td>
<td>120-165</td>
<td>0</td>
<td>0</td>
<td>+</td>
<td>Pyleonephritis</td>
<td>2</td>
<td>115-148</td>
<td>75-85</td>
<td>12</td>
</tr>
<tr>
<td>153</td>
<td>M</td>
<td>41</td>
<td>230</td>
<td>120-130</td>
<td>0</td>
<td>0</td>
<td>+</td>
<td>Pyleonephritis</td>
<td>11/2</td>
<td>120-130</td>
<td>80-90</td>
<td>20+</td>
</tr>
<tr>
<td>16</td>
<td>M</td>
<td>50</td>
<td>160-190</td>
<td>124-146</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>Pyleonephritis</td>
<td>1</td>
<td>120-134</td>
<td>74-82</td>
<td>6</td>
</tr>
<tr>
<td>17</td>
<td>M</td>
<td>55</td>
<td>200-220</td>
<td>106-140</td>
<td>0</td>
<td>0</td>
<td>+</td>
<td>Arterial embolism</td>
<td>3</td>
<td>128-140</td>
<td>76-88</td>
<td>8</td>
</tr>
<tr>
<td>18</td>
<td>M</td>
<td>55</td>
<td>210-230</td>
<td>120-130</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>Pyleonephritis</td>
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<td>120-140</td>
<td>60-80</td>
<td>10+</td>
</tr>
<tr>
<td>19</td>
<td>F</td>
<td>58</td>
<td>165-220</td>
<td>100-140</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>Arterial embolism</td>
<td>11/2</td>
<td>120-140</td>
<td>64-88</td>
<td>8</td>
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<td>20</td>
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<td>60</td>
<td>180-240</td>
<td>120-150</td>
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<td>+</td>
<td>Pyleonephritis</td>
<td>7</td>
<td>120-140</td>
<td>70-88</td>
<td>10+</td>
</tr>
</tbody>
</table>

The present study affords considerable evidence that the hypertension associated with unilateral renal disease is not only severe in intensity but clinically of the accelerated or so-called “malignant” type.* The frequency of comparable subjects who exhibit a low-grade or “benign” course fail to reach the point of surgical intervention and hence have been excluded automatically from consideration. To answer this objection, the records were reviewed of 20 consecutive adult patients encountered in this clinic or seen in consultation who were believed to have had hypertension on the basis of unilateral renal disease but whose blood pressure failed to be restored to normal values following nephrectomy. The findings on funduscopic examination were.

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* A suggestion first made to us by Dr. Paul Wermer of this institution.
recorded in 10 instances; not one showed a retinopathy. Thirteen gave a history of hypertension of more than four years' duration. In contrast to the series which responded by apparent "cure," diastolic values of 130 or over were recorded in but five of these 20 patients; only seven complained of headaches; and the renal pathologic diagnosis of pyelonephritis was made in only five instances as opposed to a high incidence of nephrosclerosis and congenital hypoplasia. Sensitization of the vascular system or changes in the opposite kidney could explain the failure of this group to respond but could not account for the more benign clinical course of these patients.

The observation that hypertension associated with unilateral renal disease is of an acute, severe and rapidly progressive type, deserves added confirmation. The evidence for it is sufficiently strong already to suggest that the presence or absence of such a pattern might be of diagnostic value in determining the likely candidates to respond to nephrectomy. As hypertensive vascular disease rarely if ever begins after the age of about 50, the sudden appearance of a striking elevation of diastolic blood pressure in older age groups, known previously to have been definitely normotensive, should lead to the suspicion of another cause. The last seven patients in this clinic who developed hypertension abruptly after the age of 50 were shown subsequently to have sustained a unilateral renal insult (infection, vascular thrombosis or embolism) in every instance. All have responded to date to nephrectomy.

It is suggested also that these data may have some bearing on the problem of mechanisms. The accelerated phase is encountered in approximately 5 per cent of patients with essential hypertension, retinal hemorrhages, exudates and papilledema develop in all combined phases of hypertensive vascular disease in 25 per cent at the most, and then only in terms of the total life history, certainly not as a rule in the first few years of the disease. Certain renal disorders, such as chronic glomerulonephritis or polycystic disease, exhibit generally a comparatively benign hypertensive course until kidney dysfunction becomes advanced. These comments suggest the strong possibility that the hypertension of unilateral renal disease is in some way different from hypertensive vascular disease, as well as from certain chronic kidney disorders, and is perhaps more closely related to the direct elaboration of a pressor substance against which no compensatory mechanisms have been established. Conceivably acute situations, such as acute glomerulonephritis and the divers factors which lead to acute urinary suppression (for example, carbon tetrachloride poisoning), fall into a similar category, but differ from unilateral renal disease in that patients either die promptly or recover completely.

The possibility must be entertained that some forms of experimental renal hypertension resemble the unilateral renal disease in man, whereas others give rise to a more chronic process on some fundamentally different basis. This should be taken into consideration before analogies are drawn between experimental data and human disease.

**Conclusions**

1. An analysis of 20 hypertensive patients with unilateral renal disease, apparently cured by nephrectomy, affords strong evidence that this condition gives rise to an acute, severe and accelerated type of hypertensive process.

2. This observation may be of diagnostic help and may serve to distinguish the patients likely to respond to nephrectomy.

3. It is suggested that the mechanisms involved in hypertension associated with unilateral kidney disease differ from those related to certain chronic bilateral renal disorders and to hypertensive vascular disease.

**References**


HYPERTENSION AND UNILATERAL RENAL DISEASE


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