Separated Renal Functions in Patients with Renal Arterial Disease, Pyelonephritis, and Essential Hypertension

By Harriet P. DusTAN, M.D., Eugene F. Poutasse, M.D., A. C. Corcoran, M.D., and Irvine H. Page, M.D.

ATHEROSCLEROSIS, fibrous proliferation, or primary dissecting aneurysm of main renal arteries or their primary branches may cause hypertension and can alter renal blood flow, filtration rate, sodium excretion, and water reabsorption.1, 2 Mechanisms of the changes in sodium and water transport are not fully understood; they may include decreases in intrarenal arterial pressure, glomerular filtration rate, distribution of peritubular blood, and, if renal atrophy is present, some change in tubular function. In dogs, partial constriction of one main renal artery provokes the excretion of urine of diminished volume and sodium concentration, and increased osmolality.3, 4 However, if the number of functioning nephrons is decreased by complete occlusion of primary branches of the main renal artery or by unilateral parenchymal disease, urine volume, filtration rate, and osmolality are, alike, decreased.5-7 These experiments indicate that differences in renal functional patterns would be found in hypertensive patients with occlusive renal artery disease depending upon whether the lesion involves the main renal artery, some of its primary branches, or is associated with pyelonephritis.

This report describes specific functions of individual kidneys of hypertensive patients with renal artery disease and compares them with those found in pyelonephritis and essential hypertension.

Methods

Separated function tests were carried out in 45 hypertensive patients. These were divided into groups on bases of clinical history, results of intravenous urography and renal angiography; this last test was performed in all. Ten were considered to have essential hypertension; 8, pyelonephritis; 10, occlusive disease of one main renal artery; 8, occlusive disease of both main renal arteries; and 9, lesions of one or more primary arterial branches which in 6 had resulted in segmental renal infarction.

Tests were performed in the afternoon during mannitol diuresis without dehydration; vasopressin infusion substituted for fluid deprivation as the stimulus for free water reabsorption. No attempt was made to regulate fluid intake during the few days prior to the test. The patients were put to bed at least 2 hours before the test and were given routine preoperative medication of a barbiturate, morphine and atropine. Urine was collected from one kidney through an occluding ureteral catheter passed approximately 4 cm. up from the ureterovesical junction and from the other continuously through the water intake valve of the cystoscope. To insulate that the ureter was completely occluded, indigo carmine was injected into the catheter; if the dye did not appear in the bladder urine, the occlusion was considered complete and urine collections were carried out over 2 15-minute periods. During the test, rates of urine flow were observed carefully; if at any time flow from the catheter decreased and that from the bladder increased, collections were interrupted, ureteral occlusion rechecked with indigo carmine and, if found to be defective, the catheter was reset. In those patients with urographic or angiographic suggestion of disparities in renal functions, the side suspected of lower urine volume was catheterized to avoid artifacts incident to collecting small urine volumes from the bladder. The procedure has been previously described in more detail.8

To measure renal functions, solutions of mannitol and vasopressin, with or without para-aminohippurate (PAH) were given intravenously. The priming solution delivered 20 mOsm of mannitol per liter of extracellular fluid (estimated as 20 per cent of the body weight) and 100 mU of vasopressin. The sustaining solution delivered 3 mOsm of mannitol and 1.5 mU of vasopressin per minute. PAH was administered in amounts sufficient to maintain plasma concentrations of 2 to 3 mg. per cent. \( C_{PAH} \) measurements were omitted in patients receiving sulfonamides.

Among the functions measured were renal plasma flow (RPF)—from the plasma clearance of PAH, glomerular filtration rate (GFR)—mannitol
clearance multiplied by 1.1, filtration fraction (FF), urine flow (V), urine osmolality (Uosm), osmolar clearance (Cosm), urine sodium concentration (UNa), and the excreted fraction of the filtered sodium load (EFNa). Analytical methods have been previously described.9, 10

Results

Values for left kidneys in essential hypertensive subjects appear in the odd-numbered columns and for the right kidneys in the even-numbered columns of table 1. Values measured in the affected or more-affected kidneys of other groups are listed in the even-numbered columns.

Essential Hypertension

Absolute rates of RPF and GFR were depressed and FF was elevated. Differences between the two sides were usually within the accuracy of the method used, i.e., within 10 per cent. Differences in V ranged from 0.03 to 0.4 ml. per minute. The maximum difference of 0.4 ml. per minute indicated that in only 1 patient was V on one side as much as 10 per cent less than that from the other. Similar consistency in the values between the two sides obtained for RPF, GFR, FF, Uosm, and Cosm. Differences in UNa were less than 10 per cent on the two sides except in patient no. 9 in whom a 15 per cent difference was found. At low values of EFNa disparities ranged from 14 to 37 per cent (patients nos. 2, 3, 9); at higher levels, disparities did not exceed 10 per cent.

Pyelonephritis

Disparities were observed in V, RPF, and GFR. In the 6 patients in whom RPF was measured, FF was higher on the more affected side; Uosm was lower on the more affected side in 4 of the 8 and Cosm was lower in all. UNa was nearly the same on the two sides; differences in EFNa were insignificant except in patients nos. 17 and 18, in whom it was 25 per cent and 26 per cent higher on the more affected side.

Unilateral Renal Artery Disease

Nine of these 10 patients showed similar functional patterns. On the unaffected side, by reference to average normal values, RPF was slightly depressed in 7 of the 8 patients in whom it was measured; GFR was normal or increased; FF was increased in all. On the affected side, V was depressed and this depression was out of proportion to decreases of RPF and GFR; FF was strikingly less. In all of these 9 patients, UNa was much lower on the side of the lesion; the least depression, when compared with the opposite side, was 14 per cent and the maximum depression was 88 per cent. Without exception EFNa was strikingly reduced and Uosm increased.

The remaining patient of this group, no. 28, a 9-year-old girl with fibrous intimal proliferation of the right main renal artery, did not show the distinctive renal functional characteristics detailed above. Urine volume, RPF, and GFR were lower on the affected side, but FF, UNa, Uosm, and EFNa were the same on the two sides.

Bilateral Main Renal Artery Disease

Differences in V, RPF, and GFR were found between the two sides. In 3, FF was significantly lower on the side of the lower GFR. Values for Uosm were disparate in 7; in 5 it was higher on the side of the lower GFR (as observed in patients with unilateral artery disease) but in 2 patients with severe functional loss (GFR, respectively 1.3 and 4 ml. per minute) the lesser Uosm was on the side of the lower GFR; Cosm was lower on the more affected side. Values for UNa were dissimilar in all patients; in 6, the lower UNa was found on the side of the lower GFR, while in 2 it was higher on that side. Differences in EFNa paralleled the differences in UNa.

Lesions of Primary Arterial Branches

Branch Lesions. Six patients had unilateral lesions and 2, bilateral (nos. 44 and 45). Renal functions resembled those found in pyelonephritis, except in patient no. 44, in whom the similarities of functions on the two sides can be explained by the presence of bilateral renal infarcts that happened to result in the equal depressions of V, RPF, and GFR. In the remaining patients, significant depressions on the affected or more affected side were observed. For the rest of the functions values...
### Table 1

**Separate Renal Functions in Patients with Essential Hypertension, Pyelonephritis, and Occlusive Renal Artery Disease**

<table>
<thead>
<tr>
<th>Patient no.</th>
<th>V (ml./min./1.73 M²)</th>
<th>RPF</th>
<th>FF</th>
<th>U_{Osm} (mOsm/Kg. H₂O)</th>
<th>C_{Osm} (ml./min./1.73 M²)</th>
<th>U_{Na} (mEq./L.)</th>
<th>EF_{Na} (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Essential hypertension</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1</td>
<td>3.90</td>
<td>3.87</td>
<td>183</td>
<td>182</td>
<td>44</td>
<td>39</td>
<td>0.24</td>
</tr>
<tr>
<td>2</td>
<td>3.81</td>
<td>3.41</td>
<td>166</td>
<td>151</td>
<td>45</td>
<td>43</td>
<td>0.27</td>
</tr>
<tr>
<td>3</td>
<td>2.81</td>
<td>2.72</td>
<td>103</td>
<td>106</td>
<td>30</td>
<td>33</td>
<td>0.29</td>
</tr>
<tr>
<td>4</td>
<td>6.07</td>
<td>5.81</td>
<td>178</td>
<td>162</td>
<td>47</td>
<td>43</td>
<td>0.27</td>
</tr>
<tr>
<td>5</td>
<td>3.18</td>
<td>3.33</td>
<td>68</td>
<td>73</td>
<td>22</td>
<td>23</td>
<td>0.32</td>
</tr>
<tr>
<td>6</td>
<td>3.73</td>
<td>3.88</td>
<td>194</td>
<td>190</td>
<td>49</td>
<td>46</td>
<td>0.25</td>
</tr>
<tr>
<td>7</td>
<td>5.08</td>
<td>5.05</td>
<td>250</td>
<td>279</td>
<td>56</td>
<td>53</td>
<td>0.23</td>
</tr>
<tr>
<td>8</td>
<td>3.58</td>
<td>3.30</td>
<td>96</td>
<td>91</td>
<td>31</td>
<td>28</td>
<td>0.32</td>
</tr>
<tr>
<td>9</td>
<td>3.57</td>
<td>3.85</td>
<td>206</td>
<td>199</td>
<td>67</td>
<td>62</td>
<td>0.33</td>
</tr>
<tr>
<td>10</td>
<td>5.19</td>
<td>4.90</td>
<td>152</td>
<td>150</td>
<td>41</td>
<td>39</td>
<td>0.28</td>
</tr>
<tr>
<td>Pyelonephritis</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>11</td>
<td>5.16</td>
<td>1.94</td>
<td>218</td>
<td>76</td>
<td>48</td>
<td>18</td>
<td>0.22</td>
</tr>
<tr>
<td>12</td>
<td>4.47</td>
<td>2.68</td>
<td>214</td>
<td>24</td>
<td>41</td>
<td>24</td>
<td>396</td>
</tr>
<tr>
<td>13</td>
<td>11.90</td>
<td>3.48</td>
<td>316</td>
<td>83</td>
<td>86</td>
<td>24</td>
<td>27</td>
</tr>
<tr>
<td>14</td>
<td>12.40</td>
<td>1.76</td>
<td>152</td>
<td>22</td>
<td>59</td>
<td>8</td>
<td>0.39</td>
</tr>
<tr>
<td>15</td>
<td>8.98</td>
<td>2.97</td>
<td>211</td>
<td>66</td>
<td>73</td>
<td>24</td>
<td>0.35</td>
</tr>
<tr>
<td>16</td>
<td>6.04</td>
<td>.74</td>
<td>330</td>
<td>30</td>
<td>51</td>
<td>6</td>
<td>0.15</td>
</tr>
<tr>
<td>17</td>
<td>6.07</td>
<td>3.33</td>
<td>70</td>
<td>32</td>
<td>462</td>
<td>405</td>
<td>9.41</td>
</tr>
<tr>
<td>18</td>
<td>4.54</td>
<td>1.75</td>
<td>291</td>
<td>96</td>
<td>56</td>
<td>20</td>
<td>0.19</td>
</tr>
<tr>
<td>Unilateral main renal artery disease</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>19</td>
<td>3.27</td>
<td>2.18</td>
<td>193</td>
<td>177</td>
<td>59</td>
<td>50</td>
<td>0.31</td>
</tr>
<tr>
<td>20</td>
<td>3.85</td>
<td>2.01</td>
<td>266</td>
<td>166</td>
<td>69</td>
<td>42</td>
<td>0.26</td>
</tr>
<tr>
<td>21</td>
<td>7.60</td>
<td>0.99</td>
<td>266</td>
<td>56</td>
<td>71</td>
<td>12</td>
<td>0.27</td>
</tr>
<tr>
<td>22</td>
<td>5.04</td>
<td>2.11</td>
<td>236</td>
<td>184</td>
<td>71</td>
<td>41</td>
<td>0.30</td>
</tr>
<tr>
<td>23</td>
<td>7.91</td>
<td>2.66</td>
<td>169</td>
<td>120</td>
<td>89</td>
<td>48</td>
<td>0.53</td>
</tr>
<tr>
<td>24</td>
<td>10.60</td>
<td>1.90</td>
<td>236</td>
<td>123</td>
<td>75</td>
<td>33</td>
<td>0.32</td>
</tr>
<tr>
<td>25</td>
<td>7.62</td>
<td>1.52</td>
<td>449</td>
<td>267</td>
<td>92</td>
<td>43</td>
<td>0.20</td>
</tr>
<tr>
<td>26</td>
<td>9.34</td>
<td>0.60</td>
<td>81</td>
<td>15</td>
<td>254</td>
<td>489</td>
<td>7.98</td>
</tr>
<tr>
<td>27</td>
<td>6.25</td>
<td>3.03</td>
<td>238</td>
<td>238</td>
<td>62</td>
<td>53</td>
<td>0.26</td>
</tr>
<tr>
<td>28</td>
<td>3.87</td>
<td>3.07</td>
<td>277</td>
<td>240</td>
<td>73</td>
<td>62</td>
<td>0.26</td>
</tr>
<tr>
<td>Bilateral main renal artery disease</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>29</td>
<td>7.47</td>
<td>1.26</td>
<td>302</td>
<td>133</td>
<td>87</td>
<td>31</td>
<td>0.29</td>
</tr>
<tr>
<td>30</td>
<td>2.13</td>
<td>0.21</td>
<td>127</td>
<td>8</td>
<td>21</td>
<td>1.4</td>
<td>0.15</td>
</tr>
</tbody>
</table>
of FF were disparate in 3, $U_{Na}$ was practically equal; $U_{Na}$ and $EF_{Na}$ were not different except in 1 patient (no. 39) in whom $U_{Na}$ was 10 per cent and $EF_{Na}$ 22 per cent higher on the affected side.

**Discussion**

The method of collection employed in these studies involves collecting urine from one kidney through an occluding ureteral catheter and from the other via bladder. This technic has been criticized on several counts. The first is that ureteral occlusion may not be complete throughout the test, even though it is shown to be complete at the beginning. Thus, urine leakage would go undetected, since it would be mixed with urine from the opposite kidney. Further, the possibility has been suggested that ureteral catheterization might, of itself, modify renal function.\textsuperscript{11} If either of these situations had regularly obtained, significant disparities should have been found in patients with essential hypertension, for urine flow from the catheterized side would have always been less. This was not the case, and minor decreases of function that were observed were not necessarily on the side of ureteral catheterization. It should be emphasized that, although the catheter is seated firmly in the ureter, there is no obstruction to free urine flow. Reflex depression of renal function arising from ureteral manipulation, while possible, has not been a problem.\textsuperscript{12} Yendt et al.\textsuperscript{13} have suggested that urine from the uncatheterized side may lose sodium through the bladder wall and this loss would vitiate the results of the test. This does not seem to be the case as shown by the similarities of $U_{Na}$ on the two sides in patients with essential hypertension, pyelonephritis, and branch lesions. That this has not been a problem is, perhaps, because the bladder is constantly drained and urine is not allowed to remain in it.

Further evidence for reliability appears in table 2. In this patient, tests were performed 3 days apart. At the first test the right ureter was catheterized; because urine flow from this side was unexpectedly higher, the test was
repeated and the left ureter was catheterized. Again the higher rate of urine flow was from the right kidney. The depression of GFR observed during the second test was due to 2 days of chlorothiazide treatment.14

In previous studies of separate renal functions11, 13, 15-17 specimens were collected by bilateral ureteral catheterization; to check for leakage the bladder was also catheterized. One difficulty with bilateral catheterization is the doubled hazard of leakage during ureteral peristalsis. Another is that at low rates of urine flow significant leakage could occur but the volume (say, 3 ml. in 30 minutes) could be so small as to escape drainage via the bladder catheter. By means of this procedure, the bladder is drained at the end of the collection period and by analysis an attempt is made to determine the kidney from which this urine originated. However, retention of urine in the bladder could allow for transfer of electrolytes through the bladder mucosa13, 20 and so change the composition of the urine that it would not be recognizable as belonging to either kidney.

Obviously, neither method is ideal because of the possibility of escape of urine around the catheters into the bladder. Our experience suggests that unilateral ureteral catheterization gives results as valid as does bilateral catheterization.

Essential Hypertension

The finding of similarities of functions of the two kidneys in patients with essential hypertension confirms and extends earlier observations of Chasis and Redisch.15 It is not in accord with the recent studies of Baldwin et al.,17 which showed disparities of separate renal functions in 40 of 50 patients considered to have essential hypertension but not examined by renal angiography. Accordingly, they have concluded that significant disparities in functions of the two kidneys develop in the course of essential hypertension. This conclusion was based on comparison of these results with those of 21 normotensive subjects.18 In the normotensive group, arbitrary limits of normality were established, and interpretation of data from the hypertensive group was based on these limits. When our data were analyzed by the same criteria, we found no significant disparities as concerned RPF, GFR, and Uosm. V was significantly different in patient no. 2, FF in nos. 1 and 7; UNa in no. 9, and EFNa in nos. 2, 3, 9. The reason that the results of Baldwin et al. are different from those of Chasis and Redisch,15 those of Connor et al.,11 and those reported here is not apparent. It seems unlikely that four fifth of a group of hypertensive patients would suffer from clinically inapparent renal lesions. The fact that our studies were performed during mannitol diuresis is not an explanation; this could, however, obscure differences in sodium and water excretions but not in hemodynamic functions.

Pyelonephritis and Lesions of a Branch of the Main Renal Artery

The functional patterns found in patients with pyelonephritis and branch lesions were
similar, which suggests a common mechanism. As would be expected RPF and GFR were lower on the affected or more-affected side, except in patient no. 44, who had bilateral renal infarcts. In each instance changes in V were directly proportional to changes of GFR, and this is reflected in the tendency of U_{osm} to be similar on the two sides. Slightly lower values for U_{osm} were found on the more-affected side in 4 pyelonephritic patients. U_{Na} on the two sides was practically equal and only in 2 patients (nos. 17 and 39) was EF_{Na} considered to be significantly different.

The disparities in renal functions observed in these diagnostic groups suggest a quantitative decrease in numbers of nephrons but without a qualitative change in the function of those remaining. As concerns the findings in pyelonephritis, they are in accord with those of Michie et al.\textsuperscript{16} whose studies led them to conclude, “in chronic pyelonephritis complete loss of nephron function predominates over specific impairment of glomerular and tubular function.”

The experimental counterparts of these renal lesions are unilateral pyelonephritis and segmental ischemia in dogs as studied by Bricker et al.,\textsuperscript{7} Blake\textsuperscript{6} and Klapproth et al.\textsuperscript{6} In each situation, function tests indicated a decrease in numbers of functioning nephrons with a supranormal GFR in the remaining nephrons, as evidenced by increased GFR/T_{m}PAH and GFR/T_{m}glucose of the experimental kidney. Constant findings, also, were increased C_{osm}/GFR and a lower U_{osm}. In our clinical studies, U_{osm} was not constantly decreased in the poorer functioning kidney, nor was C_{osm}/GFR greater. Since we did not measure Tm/PAH or Tm/glucose, we have no data concerning the magnitude of GFR in the residual nephrons. Perhaps the fact that mannitol was the major urinary solute obscured differences that might have been observed under another condition, such as water diuresis.

Main Renal Artery Lesions

In 9 of the 10 patients with unilateral occlusive disease of a main renal artery, disparities in renal functions were similar to those produced in dogs by narrowing one renal artery.\textsuperscript{3,4} In both species, the affected kidney excretes urine of smaller volume, higher osmolality and lower sodium concentration than its mate. The lower U_{Na} results from an enhanced sodium reabsorption as evidenced by the small amount of the filtered sodium that is excreted (EF_{Na}). This enhancement of sodium reabsorption on the affected side would, as Berliner et al. postulate,\textsuperscript{19} increase the tonicity of the medullary interstitial fluid and promote water reabsorption. Accordingly urine flow would not be directly dependent on GFR, as seen in patients with pyelonephritis or branch arterial lesions, but would be depressed out of proportion to depressions of GFR. The fact that U_{osm} was not greatly increased on the affected side is probably due to the mannitol diuresis with its augmented natriuresis and low urine urea concentration.\textsuperscript{20} This would impair sodium reabsorption, diminish the effectiveness of urinary urea in the concentrating mechanism\textsuperscript{21} and thus, mask that kidney’s tendency for enhanced water reabsorption.

Patient no. 28 did not have the renal functional changes that would have been expected to result from renal artery stenosis. A 15 per cent reduction in GFR on the affected side was not accompanied by a reduction in U_{Na}. One might assume that the decrease in GFR and filtered sodium load was not great enough, in the presence of mannitol diuresis, to result in greater sodium reabsorption. However, in one other patient (no. 19) a 15 per cent reduction in GFR on the affected side was associated with a 35 per cent reduction in U_{Na}. That sodium reabsorption is affected by some factor other than the filtered sodium load is suggested by these findings and by the observation in patients with bilateral main renal artery lesions that the lower U_{Na} is not always on the side of the lower GFR.

Selkurt\textsuperscript{22} has suggested that some hemodynamic function is partly responsible for the rate of sodium excretion. He studied renal hemodynamic functions and sodium excretion of dogs in which one kidney was perfused at
high or low arterial pressure levels, with or without a pulsatile flow. He found that increased intrarenal arterial pressure greatly enhanced natriuresis and that low intrarenal arterial pressure depressed natriuresis. These changes in perfusion pressure did not result in significant changes in GFR. Further, changes in pulse pressure were without effect. These results indicate that the narrowed pulse pressure found distal to a renal artery lesion does not play a role in the functional changes observed in such a kidney and that the filtered sodium load is not the only determinant of sodium excretion in these patients. Rather, decreases in intrarenal arterial pressure may account, in part, for the enhanced sodium reabsorption characteristic of occlusive arterial lesions. As concerns this possibility, mention should be made that at operation in patient no. 28, aortic pressure was 210/120 and renal artery pressure distal to the lesion was 164/120; this is the smallest gradient between aortic and renal artery pressure levels that we have observed in patients with occlusive renal artery disease.

**Clinical Significance of Separated Renal Function Tests**

Connor, Thomas, Haddock, and Howard have greatly extended the observations of Connor, Thomas, Berthrong, and Howard and again conclude that in patients with hypertension, the finding that one kidney excretes urine of lower volume and sodium content indicates that the kidney is responsible for the hypertension. Our data show that this is commonly, but not necessarily, the case. The renal functional patterns observed in patients with renal hypertension are not dependent upon the vasopressor factor responsible for the raised arterial pressure, but are determined by the location of the lesion which, in some way, causes hypertension, probably through renin release. Accordingly, results of function tests in some patients with bilateral main renal artery lesions may be the same as those of patients with unilateral lesions and also the functional effects of partial or total occlusion of major branches of the main renal artery resemble those of pyelo-

nephritis. Neither branch nor bilateral lesions are uncommon, and in such patients function tests are neither diagnostic nor prognostic. They do provide useful information on relative functional status, but anatomic diagnosis depends on renal angiography.

**Summary**

Function tests of the individual kidneys have been performed during mannitol diuresis and vasopressin infusion in hypertensive patients with essential hypertension, pyelonephritis, and occlusive lesions of one or both main renal arteries or their primary branches.

In patients with essential hypertension, glomerular filtration rate and renal plasma flow on the two sides, though depressed, were practically equal, as were urine flow, water, solute, and sodium excretions.

Pyelonephritis and branch arterial lesions alike depressed urine flow, glomerular filtration rate, and renal plasma flow in the affected or more-affected kidneys; water, total solute, and sodium excretions were in proportion to glomerular filtration rate. These findings indicate a decrease in numbers of functioning nephrons without a qualitative change in function of those remaining.

Oclusive lesion of one main renal artery decreased glomerular filtration rate and renal plasma flow on the affected side; urine flow was relatively more depressed than filtration rate, urinary osmolality was higher, and urinary sodium concentration was sharply decreased, as was the excreted fraction of the filtered sodium load.

Bilateral occlusive main arterial lesions sometimes had effects similar to those of unilateral lesions in the sense of greater functional deficits on the more-affected sides. However their functional patterns were not consistent.

The enhanced renal sodium reabsorption observed in patients with unilateral and bilateral main renal artery diseases could not be explained solely by decreases in filtered sodium load; this suggests that decreases in intrarenal arterial pressure also affect sodium excretion.

Changes in renal functions caused by ar-
terial disease depend on the site of the lesion and not on the pressor mechanism it may evoke.

References

“I rode over to Teddington,” wrote Wesley in his diary, “Dr. Hales sent after dinner to desire our company and showed us several experiments,” and then this comment: “How well do philosophy and religion agree in a man of sound understanding.”—Diary of John Wesley, July, 1753.
Separated Renal Functions in Patients with Renal Arterial Disease, Pyelonephritis, and Essential Hypertension
HARRIET P. DÜSTAN, EUGENE F. POUTASSE, A. C. CORCORAN and IRVINE H. PAGE

Circulation. 1961;23:34-41
doi: 10.1161/01.CIR.23.1.34
Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 1961 American Heart Association, Inc. All rights reserved.
Print ISSN: 0009-7322. Online ISSN: 1524-4539

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://circ.ahajournals.org/content/23/1/34

Permissions: Requests for permissions to reproduce figures, tables, or portions of articles originally published in Circulation can be obtained via RightsLink, a service of the Copyright Clearance Center, not the Editorial Office. Once the online version of the published article for which permission is being requested is located, click Request Permissions in the middle column of the Web page under Services. Further information about this process is available in the Permissions and Rights Question and Answer document.

Reprints: Information about reprints can be found online at:
http://www.lww.com/reprints

Subscriptions: Information about subscribing to Circulation is online at:
http://circ.ahajournals.org//subscriptions/