Characteristics of Hypertension in the Black Population

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SUMMARY

The renin-angiotensin system was examined in 146 black patients with essential hypertension. Classification into three categories was made according to plasma renin activity as measured by the radioimmunoassay of Angiotensin I and the accompanying sodium excretion. Differences among patients in the three renin groups (low, normal, and high) were not observed with respect to the incidence of cerebrovascular and cardiovascular events. No other discriminating variables could be identified by multivariate discriminant analysis. Low renin patients were distinguished in having a reduced sodium excretion compared to normal and high renin patients. Although total exchangeable sodium was not measured in this group of patients, other investigators have reported a higher exchangeable sodium in low renin patients than in those with normal or high values. This possibility, together with recent evidence in experimental models of low renin hypertension, that the affinity of angiotensin for its vascular receptors may be sodium dependent suggests that the incidence of vascular events may relate more specifically to angiotensin-vascular receptor interaction than to measurements of circulating renin.

Additional Indexing Words:
Plasma renin activity  Target organ dysfunction  Blood pressure

VARIABILITY of the renin-angiotensin system in essential hypertension was proposed simultaneously by Helmer and by Brown and coworkers and was subsequently confirmed by a number of other investigators. The low renin form of essential hypertension, when not associated with evidence of aldosterone excess, has been shown to exhibit other differences which suggest the possible influence of additional mineralocorticoid activity, physiologic alterations of sodium handling, disturbances of autonomic function, and unique susceptibility to adrenal inhibitory agents. Recently, Brunner et al. reported that among their series of patients with low renin activity there was no incidence of either heart attack or stroke. They concluded that patients with low circulating renin were largely spared the vasculotoxic effects of the renin-angiotensin system in hypertension. These conclusions have generated controversy on theoretical and clinical grounds.

The present study describes some of the characteristics of hypertension in the black population and provides additional information regarding vascular vulnerability in relation to various levels of plasma renin activity.

Methods

A baseline period of two years was designated in a newly established hypertension clinic at Harlem Hospital Center to provide a prospective analysis of the characteristics of hypertension in the black population, particularly with respect to the identification of renin subgroups in essential hypertension. Sources of referral included other units within the hospital, private physicians, and health workers from our community screening program. Patients with malignant hypertension were excluded from the present study.

A detailed explanation of the diagnostic and the therapeutic protocol was given and consent obtained upon entry to the clinic. Patients were assigned to one physician for continuous medical supervision and the medical history was reviewed in detail prior to the physical examination. Height (in inches) and weight (in pounds) were recorded and serial weights were taken thereafter. Blood pressure measurements were made in both arms in the supine, seated and standing positions after 15 min rest and in the left leg. Serial observations thereafter were made with the patient seated and after 2 min of quiet standing. A standard mercury manometer was used and the first and fifth (disappearance of sound) Korotkoff sounds were taken as the...
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systolic and diastolic pressures, respectively. Funduscopic findings were graded according to the Keith, Wagener, and Barker classification.27

Particular emphasis was placed upon the physical examination of target organ systems. Evidence of neurologic deficits, left ventricular enlargement, signs of pulmonary venous congestion, palpable renal abnormalities and the presence of a midepigastic bruit was sought. A chest X-ray or a cardiac series and a 12-lead electrocardiogram were obtained. The diagnosis of left ventricular enlargement was based upon criteria of the New York Heart Association.28 Evidence of myocardial infarction was obtained from the electrocardiogram (New York Heart Association Criteria)29 and the medical records. Endocrine, collagen-vascular, and other diseases were screened by serum electrolytes, creatinine, uric acid, cholesterol, fasting blood sugar, T4 and T3, plasma cortisol, latex fixation, LE preparations, antinuclear antibody, and urinary catecholamines. Assessment of renal function included urinalysis with microscopy of the sediment, urine culture, quantitative 24-hour urine protein, endogenous creatinine clearance and rapid sequence pyelography. Renal arteriography was requested when an abnormal pyelogram or midepigastic bruit suggested a renovascular abnormality. Renal biopsy was reserved for the diagnosis of renal disease of uncertain etiology.

Blood samples for the determination of plasma renin activity were obtained while the patients were receiving their customary diets and before treatment was instituted, or following three weeks off therapy. Patients who were under continuous therapy and optimally controlled and patients who presented with left ventricular failure with congestion or those with ischemic prodomes and a history of cerebrovascular accident were maintained or placed immediately on therapy with a consequent omission of the plasma renin measurement. A 24-hour urine collection for the analysis of sodium was begun on the day the blood sample was scheduled. Patients were in the fasting state and had been ambulatory for four hours before the noon hour blood sample was taken. Plasma renin activity was determined by the radioimmunoassay of Angiotensin I according to a modification of the method of Gocke et al.30 for Angiotensin II.

Radioimmunoassay
Preparation of antigen. Synthetic asp1-ileu5-Angiotensin I was coupled to rabbit serum albumin using ethyl carbodiimide as described by Goodfriend and Levine.31 Immunization with 0.5 to 1.0 ng Angiotensin I injected in divided doses into toe pads, intramuscularly and intraperitoneally, was performed in 12 rabbits of various breeds at 10-day intervals for six weeks. Four series of immunizations were made and antibody titers, performed two weeks after each series, reached levels of 1:100,000 after the second and third series and did not increase appreciably thereafter.

Immunoassay. Blood samples of patients were drawn into tubes containing EDTA 20 mg in solution, chilled immediately on ice and centrifuged at 0°C. Tris buffer 0.1 M at pH 7.5, lysosome 300 mg%, neomycin sulfate 2g/L, and chlorhexidine 0.002% was used as the diluent throughout the assay.

The incubation mixture consisted of one ml plasma containing EDTA 20μl dimercaprol (10% solution in benzoyl benzoate) and 10μl 8-hydroxyquinoline (0.34M), with adjustment of pH to 5.7 with IN HCl. The mixture was incubated for 3 hours in a water bath at 37°C.

The reaction mixture contained either standard angiotensin solution (0.01 to 0.4 ng/ml) or patient’s plasma (undiluted) 10 or 20μl, 125I-Angiotensin I 4000cpm/0.05 ml, 50μl antibody diluted to 1:75,000, and Tris buffer for a total reaction mixture of 1.2 ml. Control tubes containing no antibody were employed to detect nonspecific binding of 125I-Angiotensin I to plasma in each assay. No blank subtraction was made; duplicates were used throughout. After mixing, the assay mixture was incubated for 18 hours at 4°C and a dextran-charcoal separation was carried out. Figure 1 shows a typical standard curve.

Plasma of 53 normal subjects who were approximately equal in the ratio of male to female, black to white, and age (16-48 years) was obtained under circumstances identical to that of the patients. Generated Angiotensin I derived from these subjects served as control data for the patients.

Therapeutic Protocol
Four progressive stages of therapy were established for all patients in the hypertension clinic. Reference was not made to the measured plasma renin activity in instituting the therapy. Stage I consisted of spironolactone 50-100 mg daily for four weeks. Patients whose serum creatinine was 2 mg/100 ml or more were treated with an alternate agent, chlorthalidone 100 mg/day, to obviate potassium retention. If the blood pressure was not or near optimal control after this period, stage II therapy was begun: spironolactone was continued and alpha-methyldopa was added in 250 mg increments up to a maximum of 2 g per day for four weeks. In stage III, guanethidine was substituted for alpha-methyldopa if blood pressure control had not been achieved; increments of 25 mg were prescribed until the patient was receiving up to 600 mg. If symptoms of postural hypotension were present or control was not still optimal after four weeks, hydralazine 100-200 mg per day was added for a stage IV regimen which consisted then of triple therapy.

Statistical Applications
A one-way analysis of variance was performed using the following variables for comparison among the renin subgroups: 1) age, 2) systolic blood pressure, 3) diastolic blood pressure, 4) cholesterol, 5) fasting blood sugar, 6) serum sodium, 7) serum potassium, 8) uric acid, 9) creatinine, 10)

![Figure 1](http://circ.ahajournals.org/)

A typical standard curve shows the ratio of Angiotensin I bound to free Angiotensin I as increments of standard angiotensin, 10 to 400μg, are added to the system.
Results

Of the 295 patients admitted consecutively to the hypertension clinic, 156 patients were characterized according to the plasma renin activity and concomitant 24 hour urinary sodium excretion before treatment or after an interval of three or more weeks since previous antihypertensive therapy. The remaining 139 patients did not fulfill these criteria for the following reasons: existing treatment resulted in good control of blood pressure (13); existing treatment had poor response and patients were symptomatic (left ventricular failure with congestion or previous urinary sodium, 11) urinary potassium, 12) creatinine clearance, and 13) hematocrit. A two-way analysis of variance using the same variables was performed to compare data within and between two age categories (third and fourth decade; fifth and sixth decade). Each variable was tested for interaction. In the two cases where interaction was observed (diastolic blood pressure and creatinine), the main effects were tested by analysis of variance on cell means using harmonic means. A chi-square test (with sine transformation) was used to compare the incidence of vascular events, postural changes, and renal abnormalities among the renin subgroups. A stepwise discriminant analysis (corrected BMD 07M program) was performed on the following linearly related variables: age, systolic and diastolic blood pressures, plasma renin activity, and creatinine clearance.

![Figure 2](http://circ.ahajournals.org/)

**Figure 2**

Plasma renin activity is plotted against the concomitant 24 hour urinary sodium excretion for normal subjects (closed circles) and patients with essential hypertension (open circles). The bands (dotted lines) which enclose the normal range depict an inverse relationship between plasma renin activity and sodium excretion.

<table>
<thead>
<tr>
<th>Clinical Characteristics</th>
<th>No.</th>
<th>Sex</th>
<th>F</th>
<th>Known duration* of hypertension</th>
<th>Diastolic Blood pressure</th>
<th>SBP</th>
<th>DBP</th>
</tr>
</thead>
<tbody>
<tr>
<td>Low</td>
<td>46</td>
<td>31</td>
<td>13</td>
<td>110 ± 12</td>
<td>84 ± 7.0</td>
<td>25</td>
<td>25</td>
</tr>
<tr>
<td>Normal</td>
<td>37</td>
<td>47</td>
<td>11</td>
<td>111 ± 12</td>
<td>84 ± 8.4</td>
<td>35</td>
<td>20</td>
</tr>
<tr>
<td>High</td>
<td>36</td>
<td>40</td>
<td>11</td>
<td>108 ± 20</td>
<td>81 ± 4.9</td>
<td>20</td>
<td>9</td>
</tr>
</tbody>
</table>

*p<0.003

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*SNS = not significant; I = grade I; II = grade II.
cerebrovascular accident with ischemic prodromes) (25); no previous treatment and patients were symptomatic (29); blood sample was obtained less than three weeks off therapy (13); gap in protocol or failure to complete the 24 hour urine collection prior to therapy (55); or patients were normotensive on observation (4). Ten of the 156 patients who had a renin determination did not have essential hypertension: three patients had chronic renal disease and presumptive secondary hypertension, three patients had contraceptive-related hypertension, and four additional patients were normotensive on observation.

By diagnostic exclusion, 146 patients had essential hypertension. Classification into three renin groups (low, normal, and high) was based upon the results of the radioimmunoassay of Angiotensin I in relation to the 24 hour urinary sodium excretion as measured in a representative sample of 53 normal subjects (fig. 2). Fifty-three patients (36%) fell within the normal renin range on the constructed nomogram. Fifty-six patients (39%) had low plasma renin activity, and 37 patients (25%) had high renin levels compared to the normal subjects. Some patients with nominally low plasma renin levels (less than 0.1 ng/ml/hr) but whose urinary sodium excretions were 200 mEq/day or more may represent additional low renin patients; however, no dietary manipulations were made to ascertain this possibility. Although plasma aldosterone determinations were not made, serum potassium levels were not significantly different among the three renin groups and the lowest individual value measured was 3.5 mEq/L.

**Clinical Characteristics**

Table 1 depicts the clinical characteristics of the patients in the three renin categories. A stepwise multivariate discriminant analysis did not select age as a discriminating variable. An analysis of variance indicated no significant difference in age among the three renin groups. Thirty-eight percent of the patients had never been treated (low: 17; normal: 24; high: 15 patients). Seventeen patients had been under continuous treatment at the time of admission to clinic; however, 14 patients had been receiving therapy for less than one year and each of them had been referred because of unsatisfactory blood pressure control. The remaining patients had been receiving intermittent therapy. While it was not possible to document the efficacy of previous treatment among the three renin categories, when a comparison of the ratio of the duration of disease to the years of intermittent treatment was made, there was no significant difference among low, normal, and high renin patients.

**Chemical Parameters**

Chemical parameters (table 2) were comparable in all three renin categories except for a reduced urinary sodium excretion and creatinine clearance in low renin as compared to normal or high renin patients. There were no differences in serum sodium and hematocrit. Diabetes mellitus was diagnosed in two patients in each of the low and normal renin groups and in three of the high renin patients.

**Target Organ Dysfunction**

Evidence of target organ dysfunction was present in each of the renin groups of patients (table 3). Arteriosclerotic heart disease was diagnosed in ten patients; evidence of a myocardial infarction was present in three low, four normal, and two high renin patients, and one additional low renin patient had angina pectoris. Left ventricular enlargement was present in 22% of patients (low: 14 patients; normal: 13; high: 5) according to the electrocardiogram and the chest X-ray. Eleven patients had left ventricular enlargement diagnosed solely on the chest X-ray (low: 5 patients; normal: 2; high: 4). Additional findings on the electrocardiogram which could not be ascribed with certainty to a specific form of heart disease were: 1) left bundle branch block: one high renin and two low renin patients; 2) atrioventricular conduction

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**Table 2**

<table>
<thead>
<tr>
<th>Plasma renin activity</th>
<th>Serum creatinine (mg/100 ml)</th>
<th>Serum potassium (mEq/L)</th>
<th>Serum uric acid (mg/100 ml)</th>
<th>Serum cholesterol (mg/100 ml)</th>
<th>Fasting blood sugar (mg/100 ml)</th>
<th>Creatinine clearance (ml/min/m²)</th>
<th>Urinary sodium excretion (mEq/24 hrs)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Low</td>
<td>1.08 ± 0.22</td>
<td>4.2 ± 0.44</td>
<td>5.74 ± 1.6</td>
<td>224 ± 54</td>
<td>105 ± 20</td>
<td>90 ± 22</td>
<td>100 ± 36</td>
</tr>
<tr>
<td>Normal</td>
<td>1.04 ± 0.19</td>
<td>4.3 ± 0.48</td>
<td>5.37 ± 1.3</td>
<td>216 ± 55</td>
<td>101 ± 15</td>
<td>99 ± 24</td>
<td>141 ± 90</td>
</tr>
<tr>
<td>High</td>
<td>1.07 ± 0.22</td>
<td>4.2 ± 0.36</td>
<td>5.80 ± 1.5</td>
<td>220 ± 54</td>
<td>103 ± 17</td>
<td>101 ± 23</td>
<td>153 ± 69</td>
</tr>
</tbody>
</table>

*Mean ± SD.*

†NS = not significant.

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Table 3

<table>
<thead>
<tr>
<th>Plasma renin activity</th>
<th>Cerebrovascular accident</th>
<th>Arteriosclerotic heart disease</th>
<th>Left ventricular enlargement</th>
</tr>
</thead>
<tbody>
<tr>
<td>Low</td>
<td>9 (16%)</td>
<td>4 (9%)</td>
<td>14 (25%)</td>
</tr>
<tr>
<td>Normal</td>
<td>5 (7%)</td>
<td>4 (8%)</td>
<td>13 (23%)</td>
</tr>
<tr>
<td>High</td>
<td>2 (5%)</td>
<td>2 (5%)</td>
<td>5 (14%)</td>
</tr>
<tr>
<td>Total</td>
<td>16 (11%)</td>
<td>10 (7%)</td>
<td>32 (22%)</td>
</tr>
</tbody>
</table>

*1 patient with angina pectoris (low renin); 9 patients with myocardial infarction.

defect in the absence of digitalis: one high renin patient; and 3) nonspecific T-wave abnormalities in 24 low renin, 13 normal renin, and seven high renin patients. Sixty-four patients had normal electrocardiograms (low: 15 patients; normal: 27; high: 22).

Sixteen patients (11%) had evidence of a previous cerebrovascular accident. There were more low renin patients with stroke (9) than normal (5) or high (2) renin patients but this variation was not significant. Three additional hospitalized patients with recent strokes were studied to see whether this trend continued. Two of these hypertensive patients had low plasma renin activity and one had a high level.

Assessment of renal function other than creatinine clearance and sodium excretion revealed no further distinguishing features among the renin categories. Overt renal abnormalities were equally distributed among the three renin groups. Examination of the urine sediment revealed 10–50 WBC/HPF and less than 3 RBC/HPF in eight patients (low: one patient; normal: three; high: four) and positive urine cultures in eight patients (low: three; normal: two; high: three). Proteinuria (> 150 mg/24 hr) was present in 20 patients. Nine of 49 low renin patients had proteinuria (170–398 mg/day) without evidence of urinary tract infections. Seven of 51 normal renin patients and four of 30 high renin patients had proteinuria (185–925 mg/day and 177–408 mg/day, respectively) without known infections. One additional high renin patient had 1265 mg protein/day when she presented with an abnormal urine sediment and a positive culture. The differences among the renin groups were not significant.

Rapid sequence intravenous pyelograms revealed the following findings in seven of 47 low renin patients: delayed dye excretion (4); minimal bilateral calyceal blunting (1); nephrocalcinosis (1); and congenital double collecting systems (1). Four of 42 normal renin patients had abnormal pyelograms: delayed dye excretion (2) and discrepant renal size (2). The latter two patients underwent selective renal arteriography with arterial and venous sampling for renin activity. Normal renal arterial vessels were observed in both patients and renal vein renin ratios between the two kidneys were normal. Four of 31 high renin patients had pyelograms which showed delayed dye excretion (1), unilateral renal calculus (1), minimal bilateral calyceal blunting (1) and minimal unilateral hydronephrosis (1). Twenty-seven patients failed to have an intravenous pyelogram.

Statistical Analysis

In order to evaluate the influence of age on the manifestations of target organ dysfunction, three patients in the seventh decade were eliminated and chi-square analysis was performed again. No significant difference was observed. For the remaining variables, a two-way analysis of variance was performed in which patients in the third and fourth decade of age were compared with those in the fifth and sixth decade of age and the age differences among the three renin groups were tested for significance. By this method it became clear that the reduced creatinine clearance observed in the low renin patients was confined to the older patients (P < 0.01). Age was not a factor in the reduction in urinary sodium excretion observed in low renin patients when compared to normal and high renin patients. A difference in systolic but not in diastolic blood pressures emerged in all the renin groups between the younger and older patients (P < 0.001).

Therapeutic Findings

Prior to therapy, the diastolic blood pressure rose 10 mm Hg or more above the level measured in the seated position upon assuming upright posture in 53% of normal and 46% of high renin patients. In only 27% of low renin patients was blood pressure raised upon standing (P < 0.02). However, these patients were not more prone to postural hypotension after sustained control of their blood pressures with either diuretic or sympatholytic agents.

Fifty-eight patients responded to spironolactone as the sole therapeutic agent with good blood pressure control. There was no significant difference in the degree of response among the renin groups. The mean decline in diastolic blood pressure, seated, was: low renin patients, 22 ± 12 mm Hg; normal renin patients 16 ± 9 mm Hg; and high renin patients, 22 ± 12 mm Hg. However, the percentage of low renin patients whose response to diuretic therapy or salt restriction alone following initial diuretic therapy (57%) exceeded that of normal (40%) or high (38%) renin patients. Nine patients received 300 mg spironolactone daily. Patients who responded to this level differed from those receiving 50–100 mg/day not only in the rapidity of response (7–10 days as compared to
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4–6 weeks) but also in their inability to maintain control when spironolactone was reduced after several months from 300 mg to 150 mg daily. Control was regained with resumption of high dose therapy.

Thirty-eight patients responded well to spironolactone in combination with alpha-methyldopa. The mean decline in blood pressure was: low renin patients (14), 26 ± 12 mm Hg; normal renin patients (14), 25 ± 9 mm Hg; and high renin patients (10), 18 ± 17 mm Hg (NS). The remaining patients were maintained on combination drug regimens requiring chlorthalidone (12), hydralazine (2), or guanethidine (10). Fourteen patients failed to follow the therapeutic plan. Twenty patients were started at the second level of therapy because of either the height of the initial blood pressure or a previous history of resistance to control. There was no significant difference in the distribution of these patients among the renin groups (low: five patients; normal: ten; high: five).

Plasma renin activity during therapy was not determined as part of the protocol. However, these data were available in 16 patients. On diuretic therapy (12 patients) plasma renin activity rose as anticipated; however, eight patients remained within their initial renin group. Three patients shifted from the low to the normal renin group with large increases in sodium excretion and two of these subsequently were managed with sodium restriction alone. One additional low renin patient who received 300 mg spironolactone daily had a marked rise from 0.7 to 8.4 ng/ml/hr plasma renin activity 4 months after the initiation of therapy but two months later returned to a level of 0.3 ng/ml/hr without any change in treatment and with the blood pressure optimally controlled at both points. On a combination therapy of spironolactone and either alpha-methyldopa or guanethidine three patients had marked reductions in plasma renin activity; however, one patient had a twofold rise which shifted him to a high renin category. Two months later he required increased therapy for control.

Discussion

Low plasma renin activity has been reported to be present in approximately one fourth of patients with essential hypertension when data from several investigators are averaged.18 However, Helmer observed that 53% of black patients studied had low renin levels compared to 31% of white patients. Brunner et al.13 reported a 27% over-all incidence of low plasma renin activity; however, 42% of patients in this category were black patients. In the present study 39% of patients in a black population had low plasma renin activity in relation to the concurrent sodium excretion. The preponderance of low renin patients in a population known to have a greater prevalence of hypertension as well as a greater attendant morbidity raises the question whether the low renin segment of the black hypertensive population might have a relatively lower morbidity than have patients with higher renin levels as has been proposed by Brunner and colleagues.18 The results of the present study do not confirm this postulate. Manifestations of target organ dysfunction were not significantly different among patients in the three renin categories.

Doyle et al.25 and Stroobandt et al.26 reported no significant difference in the occurrence of heart attacks and strokes in patients in various renin categories. Christlieb and colleagues27 observed fewer of these events in the low renin group. However, in each of these studies plasma renin activity was measured by bioassay and the renin subgroupings were defined without relation to the concurrent sodium excretion. Although Mroczek et al.28 reported findings similar to the present study in a large black population, a correlation between plasma renin activity and sodium excretion in normal subjects was not demonstrated, a finding that imposes an inherent limitation on the classification.

Influences other than plasma renin activity were assessed to determine what bearing they might have on the disorders observed in the cerebrovascular, cardiovascular and renovascular systems. A ratio of duration of disease to the duration of previous treatment showed no comparative differences in the means of the subgroups; however, the intermittency of treatment was so pervasive that an accurate assessment of the efficacy of therapy could not be made. Prospective analysis of treatment under a defined protocol indicated similar magnitudes of blood pressure control among the three renin groups. A longer period of observation and a more extensive analysis of changes within the renin system during therapy will be required to assess the influence of renin as a risk factor when blood pressures are optimally controlled in all groups.

Although the difference in age among patients in the three renin subgroups was not statistically significant and multivariate discriminant analysis did not select age as a discriminating variable, the possible relationship was further tested by eliminating the three patients in the seventh decade and repeating statistical comparison of vascular events. There was still no significant difference in the number of events among the three groups. An age factor was found to be operative in the relative reduction of creatinine clearance in low renin patients: the reduced clearances were confined to patients in the fifth decade and beyond. Age was not shown to be a factor,
however, in the lower mean sodium excretion measured in low renin compared to normal or high renin patients. These differences could not be assigned to overt renal abnormalities since, though minor abnormalities were present in a limited number of patients, they were equally distributed among the renin categories.

In a recent review of the experimental evidence Giese pointed out the variety of factors in addition to renin which appear to be operative in the production of arteriolar lesions; whether these factors are also central in the development of arteriolar lesions remains to be clarified. The relative weight of renin as a contributor may not be susceptible to evaluation until methods are available to measure angiotensin-vascular receptor interaction in human hypertension. Studies in experimental models indicate that measurements of plasma renin activity do not wholly reflect angiotensin receptor affinity since low renin models have been shown to require from two to four times more angiotensin antibody to ablate the pressor response to angiotensin than other experimental forms of hypertension. This apparent dichotomy has been shown to be related to the influence of sodium upon the vascular receptor affinity for angiotensin. Whether this mechanism was operative in our low renin patients is unknown. A higher exchangeable sodium has been reported in patients with low renin compared to normal or high renin forms of hypertension.

Subtle differences in sodium handling may be present in low renin patients which may contribute to a high sodium pool. Reduced sodium excretion was noted in our low renin patients and those reported by Woods et al. prior to treatment. Conversely, diuretic therapy in low renin patients has been shown to initiate an exaggerated natriuresis as compared to other hypertensive patients. Furthermore, Helmer and Judson have reported subtle differences in sodium excretion during treatment between black and white low renin patients.

The low renin patients may not be homogeneous in the mechanisms involved in their hypertension. In the few patients treated with 300 mg spironolactone therapy the response was more immediate than in those patients controlled with 50–100 mg spironolactone daily. Although this may represent a difference in the dose of the natriuretic agent only, the fact that none of the patients who were controlled for several months on the higher dose could be sustained on a 50% dose reduction may implicate additional mineralocorticoid input as has been suggested by others. Our data are insufficient to establish this point.

The results of the present study confirm the usefulness of the renin framework for distinguishing variations among patients with essential hypertension. However, plasma renin activity, when considered as an isolated delineator of potential vascular consequences, appears to be overshadowed by the dynamics inherent in the system. Further investigation will be required to determine whether recent evidence in animal models that angiotensin affinity for its vascular receptors is sodium dependent can be applied to the human form of low renin hypertension. This prospect provides hope for a better understanding of the genetic-environmental interaction which may promote the onset and temporal developments of hypertension and its consequences throughout the vascular network.

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