high rates of shear, especially in narrow tubes (*2*). Since the blood circulating through the vasculature is subjected to both high and low rates of shear, both phenomena are of hemodynamic significance.

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Renin-Aldosterone System in Parkinson's Disease

Abstract. *Low blood pressure is frequent in the akinetic form of Parkinson's disease. A low renin activity in plasma as well as a low rate of aldosterone secretion is demonstrated in these patients. Renin activity in the plasma is further decreased by treatment with* L-*dihydroxyphenylalanine, thus partially accounting for the hypotensive episodes seen with this form of therapy.*

Patients with Parkinson's disease, a chronic disorder of the nervous system appearing in later life, often have a blood pressure lower than the mean for their age group. This relatively low blood pressure, present particularly in the akinetic form, is more evident in the standing position and contributes to a chronic state of fatigue and to occasional episodes of postural dizziness. The experimental treatment of these patients with the dopamine precursor L-dihydroxyphenylalanine (L-dopa) can lead to an exacerbation of hypotension to degrees that may become alarming (*1*).

Numerous studies have established a role for the renin-aldosterone system in regulation of blood pressure and in various forms of hypertension (*2*). There is considerable evidence for the participation of the sympathetic nervous system in the control of blood pressure (*3*) and of renin release (*4*). Recently, a defect in the sympathetic nervous system has been postulated in essential postural hypotension (*5*), as it had been in Parkinson's disease. We therefore studied the state of the renin-aldosterone system in the latter illness.

Renin activity in the plasma was determined by the method of Boucher *et al.* (*6*) in 35 normotensive control subjects, 8 of whom had been under a metabolic diet of 135 meq of sodium per day for 3 days before the blood sampling (*7*). Similar determinations were carried out in 31 patients with Parkinson's disease (mean age, 62.1 years) with a mean duration of illness of 9.7 years. Eleven patients were taking anticholinergic medication, but the remainder were not receiving drugs at the time of renin determination. All subjects were recumbent at time of sampling (after 12 hours of rest). Under an unrestricted salt intake, the plasma renin activity was significantly lower in patients than in controls ($P < .01$) (Table 1). This difference persisted when 17 of these patients were given a diet of 135 meq of Na per day (K, 90 meq/day) for 3 days before determination of renin activity ($P < .01$). Of the 48 measurements made, only 9 were above 5 ng liter^{-1} min^{-1}, and 28 had undetectable amounts.

Five Parkinsonian patients were chosen consecutively for more detailed studies. That the subgroup is representative of the larger group is evidenced by the fact that these three men and two women had a mean age of 63.2 years; a plasma renin activity under unrestricted salt intake of 2.2 ± 1.6 ng liter^{-1} min^{-1} and under a Na diet (135 meq) (K, 90 meq/day) of 3.6 ± 1.7 ng liter^{-1} min^{-1} was observed. Kidney functions were normal for that age range (blood urea, 27.6 ± 4.9 mg per 100 ml of blood; creatinine clearance, 82.4 ± 13.8 ml/min). Hemoglobin (15.4 ± 0.6 g), hematocrit (43.1 ± 1.6 percent), blood sodium (140.5 ± 0.7 meq/liter), blood potassium (4.4 ± 0.2 meq/liter), and Bromsulphalein test (4.9 ± 0.8 percent) were all normal, as were the individual and mean blood pressure (20 determinations per patient) in both the standing (mean, 121/78 mm-Hg) and recumbent positions (mean, 133/83 mm-Hg). Total blood volumes, red blood cell volumes, and plasma volumes were normal in all five cases with the exception of a slight (8.8 percent) decrease in plasma volume in a single patient.

Table 1. Plasma renin activity in Parkinson's disease and in normotensive healthy control subjects expressed in nanograms of angiotensin liberated per liter per minute (mean ± S.D.); *N*, number of patients.

Na diet	*N*	Angiotensin (ng liter^{-1} min^{-1})
	Control	
Unrestricted	27	9.5 ± 6.7
135 meq/day	8	9.8 ± 12.9
	Parkinson	
Unrestricted	31	2.4 ± 4.1
135 meq/day	17	2.7 ± 2.7
	Parkinson subgroup	
Unrestricted	5	2.2 ± 3.6
135 meq/day	5	3.6 ± 3.9

Amounts of aldosterone in plasma were measured in these five patients by the method of Nowaczynski *et al.* (*8*). With this method the normal mean for peripheral plasma aldosterone is 8.09 ± 1.08 ng/100 ml (diet: Na, 135 meq/day; K, 90 meq/day) with a range of 2 to 16.6 ng/100 ml. Plasma aldosterone was within the normal range in four of our five patients and elevated in one (Table 2). The secretory rate of aldosterone was determined by the double isotope dilution method (*8*). ^{3}H-Labeled aldosterone (2 μc at a specific activity of approximately 100 μc/μg) was injected into the antecubital vein. Normal range is from 50 to 210 μg of aldosterone per day secreted. The secretion was at the lower limits of normal range (Table 2). Thus in Parkinson's disease aldosterone secretion and renin activity vary in parallel, and one can rule out conditions of primary or secondary hyperaldosteronism.

In most cases of akinetic Parkinson's disease, dopamine excretion in the urine is low (*9*). This was also the case for four out of five cases in the subgroup under study (Table 2). Adrenaline values were within normal limits, but two of the patients had low noradrenaline excretion. All had low homovanillic

Table 2. Plasma renin activity, plasma aldosterone, aldosterone secretion rate, and catecholamine excretion values in five patients with Parkinson's disease under metabolic ward conditions. The patients were then treated with 3 to 5 g per day of L-dopa and the plasma renin was activity measured again.

Measurement	Intake conditions	Results (mean ± S.E.M.)
Renin activity (ng liter^{-1} min^{-1})	Unrestricted Na	2.2 ± 1.6
Renin activity (ng liter^{-1} min^{-1})	135 meq/day Na	3.6 ± 1.7
Renin activity (ng liter^{-1} min^{-1}) (after treatment)	135 meq/day Na 3–5 g/day L-dopa	Undetectable
Plasma aldosterone (ng/100 ml)	135 meq/day Na	14.8 ± 4.5
Aldosterone secretion rate (μg/24 hr)	135 meq/day Na	42.3 ± 12.1
Urine dopamine (μg/24 hr)	135 meq/day Na	267.0 ± 70.7
Urine noradrenaline (μg/24 hr)	135 meq/day Na	22.3 ± 9.3
Urine adrenaline (μg/24 hr)	135 meq/day Na	16.0 ± 2.8
Urine homovanillic acid (mg/24 hr)	135 meq/day Na	4.6 ± 0.6

acid excretion. These results are compatible with, but of course not diagnostic of, peripheral sympathetic nervous system hypofunction. In further support of this hypothesis are the following observations (in six patients balance studies were carried out). A change from 135 to 10 meq of Na per day (K, 90 meq/day kept constant) was manifested within 3 to 4 days by the expected drop in Na excretion to less than 15 meq/day. This was followed by a marked positive balance immediately upon return to 135 meq of Na per day. The pattern of Na homeostasis is therefore within normal limits in these patients. Plasma renin activity was studied in the same six patients fed a diet of 135 meq of Na and 90 meq of K per day. The results were 2.53 ± 0.87 ng liter^{-1} min^{-1}. After a further 3 days of diet with 10 meq of Na and 90 meq of K per day, there was a rise to 13.10 ± 4.74 ng liter^{-1} min^{-1}. This rise is within the lower limits of the normal range (*2*).

Our study indicates that one of the possible underlying mechanisms for the low or low normal blood pressures in akinetic Parkinson's disease is a decreased activity of the renin-aldosterone system. This may be secondary to a deficient sympathetic nervous system or may in some way be related to the reported defect in dopamine metabolism (*9, 10*). The occurrence of basal ganglia symptoms resembling the rigidity of Parkinson's disease in cases of autonomic insufficiency or so-called "idiopathic orthostatic hypotension" where the renin-aldosterone system is also deficient (*5, 11*) has been reported. In one report (*11*), pathological findings similar to Parkinson's disease were found in the substantia nigra, locus ceruleus, and sympathetic ganglia.

After 1 and 3 months of treatment with high doses of L-dopa (3 to 5 g/day, given orally), the plasma renin activity in five Parkinsonian patients decreased and became undetectable (Table 2). In two more patients with relatively high blood pressure (above 110 mm-Hg diastolic) we succeeded in obtaining normal blood pressure readings after 3 months of L-dopa therapy alone. This decrease in plasma renin activity with L-dopa should be explored in relation to the orthostatic hypotensive episodes frequently noted during this form of therapy. The above results also warrant further studies into the relation between sodium, dopamine, and the renin-aldosterone system as well as a trial of L-dopa in the treatment of neurogenic hypertension.

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Photoperiod in Three Xanthium Populations from the Tropic of Cancer in Mexico

Abstract. *Diverse photoperiodic responses were shown by three populations of* Xanthium strumarium *L. originating between 22° and 25°N on the western coast near Culiacán, Sinaloa; in the Chihuahuan Desert near Matehuala, San Luis Potosí; and on the Gulf Coast near Ciudad Mante, Tamaulipas, respectively. A combination of differences in critical night length and in ripeness-to-flower response (maturity) appears to be the basis for reproductive adaptation of these populations to different climatic regimes that prevail at the same latitude (and photoperiodic regime).*

During an investigation of photoperiodism in populations of *Xanthium strumarium* L. (*1*) from Texas and Mexico, different requirements for night length were shown by three populations originating between 22° and 25°N. Greater similarity had been anticipated because the three populations are exposed to nearly identical sequences of day length and corresponding night length (Fig. 1) (*2*). Variation in photoperiod was suggested, however, by differences in time of collecting mature burrs at the three sites—mid-August, Culiacán, Sinaloa (24°48′N, 107°24′W); November, Matehuala, San Luis Potosí (23°39′N, 100°39′W); and late December, Ciudad Mante, Tamaulipas (22°44′N, 98°57′W). This study emphasizes the role of photoperiod in timing activities of plant populations in diverse ecosystems at the same latitude.

Ray and Alexander (*1*) have documented the latitudinal shift in photoperiodic response among *Xanthium* populations in the United States. Northern populations (Minnesota to New York) have an apparent critical night length of 7.75 to 8.5 hours for floral induction, and southern populations (central Texas to Georgia) require 9.5 to 10.5 hours. Although some variation in photoperiod was noted among U.S. populations from the same latitude, it did not encompass the range of the three populations reported below.

All three Mexican populations occur in typical roadside depressions but within three very different ecosystems (Fig. 1) (*3*). The population near Culiacán, on the western coast of the mainland, receives scant rainfall, most of it falling during July, August, and September. The Matehuala populations occur in a creosote bush–desert ecosystem (Chihuahuan Desert) where rain-