left-handed fathers. The former result is contrary to a learning theory of handedness, and the latter is contrary to a prenatal hypothesis. The data are consistent with a genetic hypothesis.

Because we cannot conceive of any suitable prenatal nongenetic process, we believe that the most parsimonious explanation of our data is that genetic factors are important in the origin of human handedness. However, there is also substantial evidence that handedness is related to early brain damage (*20*), and, perhaps, cultural factors (*13*). The present data certainly do not preclude the operation of such factors.

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Catecholamine Enzymes in the Degenerative Neurological Disease Idiopathic Orthostatic Hypotension

Abstract. *Discrete brain areas and sympathetic ganglia obtained at autopsy from patients with idiopathic orthostatic hypotension were assayed for tyrosine hydroxylase and dopamine β-hydroxylase. Dopamine β-hydroxylase activity was decreased 7.5-fold in sympathetic ganglia, while tyrosine hydroxylase activity was reduced more than 50-fold in the pontine nucleus locus coeruleus. These observations indicate that noradrenergic neurons of both brain and ganglion are affected in idiopathic orthostatic hypotension, but suggest that the central and peripheral biochemical deficits differ.*

Although degenerative disorders of the nervous system contribute significantly to human morbidity and mortality, little is known about the biochemical abnormalities underlying these diseases (*1*). We have been studying a rare but clinically well-defined degenerative neurological disease, idiopathic orthostatic hypotension (IOH) (*2*), the Shy-Drager syndrome (*3*). This progressive degenerative disorder characteristically strikes during middle age, causing a profound fall in blood pressure upon standing (orthostatic hypotension) that commonly results in fainting. Urinary and fecal incontinence, sexual impotence, loss of sweating, extraocular palsies, and a disorder of movement that may resemble Parkinson's disease may also occur. Including the original autopsy report in 1927 (*4*), only 19 cases have been subjected to postmortem anatomical examination (*3, 5*). These studies have revealed variable neuronal degeneration of cranial nerve nuclei, striatum, substantia nigra, pontine nuclei, inferior olives, cerebellar cortex, and spinal cord intermediolateral columns in the central nervous system, and of autonomic ganglia in the periphery.

Since many of the symptoms of IOH could result from dysfunction of catecholamine-containing neurons, and since many of the histopathological changes are found in monoaminergic systems, we elected to study two enzymes involved in catecholamine biosynthesis. Tyrosine hydroxylase (TOH), the apparent rate-limiting enzyme in catecholamine biosynthesis (*6*), catalyzes the conversion of L-tyrosine to L-dopa and is restricted to neurons synthesizing dopamine or norepinephrine. In contrast, dopamine β-hydroxylase (DBH), which converts dopamine to norepinephrine, is localized to noradrenergic nerves (*7*). This report describes the results of postmortem analysis of these enzymes and histological examination of the nervous systems of patients with IOH.

Brain tissue and sympathetic ganglia were obtained from three patients with IOH and from seven controls who died from other causes at New York Hospital–Cornell Medical Center. Because there is normally a rapid postmortem decrease in TOH activity (*8*), only patients from whom specimens could be obtained within 10 hours of death were examined. Postmortem delay (time between death and freezing of dissected tissue) was 4.9 ± 0.32 hours for the control group and 5.2 ± 2.46 hours for the IOH group ($P > .05$). At autopsy the brain and sympathetic ganglia were hemisected; one portion was used for histological examination and the other for biochemical studies. Sympathetic ganglia of the cervical and thoracic chains, the nucleus locus coeruleus of the rostral pons, substantia nigra, and head of caudate were dissected at autopsy by methods previously described (*8, 9*), with an atlas as a guide (*10*). Tissues were immediately frozen on Dry Ice and stored at −90°C. When these methods were employed enzyme activities remained stable for at least 6 months, and repeated assays of the same specimen on different days yielded results that differed by less than 10 percent. Assays of TOH and DBH activities were carried out according to slightly modified versions of methods previously described (*11, 12*). After they were weighed, samples were assayed in triplicate, and the results reported are the means of these determinations. Results were similar whether they were expressed per unit of weight or per unit of total protein. To ensure reproducibility, TOH obtained from pooled rat superior cervical ganglia and DBH obtained from pooled loci coeruleus were included in all assays as external standards.

Patients with IOH and controls were comparable not only with respect to postmortem delay time, but also with respect to age: controls were 62.3 ± 6.75 years, and the IOH patients were 58.7 ± 9.02 years ($P > .05$). All patients and controls were American-born Caucasians. There were five males and two females in the control group and two males and one female in the IOH group. Primary dis-

eases in the control group included acute myocardial infarction (two), multiple myeloma (one), metastatic adenocarcinoma of the gall bladder (one), disseminated lupus erythematosus (one), pneumonia, bronchiectasis, arteriosclerotic cardiovascular disease, and Paget's disease (one), and acute tubular necrosis with uremia, acute pancreatitis, bronchopneumonia, and peripheral neuropathy (one). Both patients and controls had been treated with a variety of agents, and no single drug was received by everyone in either group. The patients with IOH were treated with a number of drugs that may alter blood pressure, including 9-α-fluorocortisone, hydroxyamphetamine, tranylcypromine, and L-dopa, but four of the seven controls had also received such agents, including dopamine, L-dopa, norepinephrine, and isoproterenol.

Microscopic examination of the postmortem specimens revealed degenerative changes in the locus coeruleus, sympathetic ganglia, and intermediolateral columns of the spinal cord in all IOH cases. The locus coeruleus exhibited neuronal loss, mild fibrillary gliosis, extracellular melanin, and melanin within macrophages. Lewy bodies were noted in pigmented neurons of the locus coeruleus in cases A and C (Fig. 1). Compared to the controls, sympathetic ganglia in all three IOH cases contained fewer neurons associated with increased numbers of capsule cells and amounts of interstitial connective tissue (Fig. 1). In addition, there was an apparent decrease in myelin and axon numbers in the IOH ganglia. Intracytoplasmic and interstitial hyaline bodies resembling those previously reported to occur in patients with IOH (5) were noted in case C (Fig. 1). Gliosis and an apparent decrease in neurons in the intermediolateral cell columns of the spinal cord were mild in case C and moderate in cases A and B. Only case B demonstrated neuronal loss and gliosis in the substantia nigra, putamen, and outer segment of the globus pallidus.

Dopamine β-hydroxylase activity was examined in sympathetic ganglia and in central noradrenergic neurons with the nucleus locus coeruleus. Enzyme activity was profoundly depressed in sympathetic ganglia of all patients with IOH. Although mean activity was 1.15 ± 0.202 nmole per milligram (wet weight) per hour in the control group, it was at or below blank values (0.15 nmole mg^{-1} $hour^{-1}$) in patients with IOH (Table 1). Thus, mean DBH activity was at least 7.5 times lower in IOH ganglia than in ganglia from controls. Preliminary studies suggest that these differences are not attributable to the presence of inhibitors or the absence of activators of DBH activity in IOH ganglia. Although there were marked differences in ganglionic DBH activities, there was no significant difference in locus coeruleus enzyme activity between the two groups. Mean control activity was 2.64 ± 0.581 nmole mg^{-1} $hour^{-1}$, while IOH activity was 2.39 ± 0.529 nmole mg^{-1} $hour^{-1}$ ($P > .05$; Table 1).

Activity of TOH was assayed in peripheral noradrenergic neurons in sympathetic ganglia, in central noradrenergic neurons of the locus coeruleus, and in central dopaminergic neurons of the substantia nigra and the head of caudate. Activity varied over a wide range in control ganglia, from 2.2 to 97.5 pmole per milligram (wet weight) per hour, possibly as a result of marked postmortem lability (8) as well as the heterogeneity of disease processes and medications in this group (Table 1). Two different sympathetic ganglia from each IOH patient were assayed. Tyrosine hydroxylase activity varied markedly, even between ganglia from a single patient (Table 1), but no consistent differences between the control and IOH groups emerged. In con-

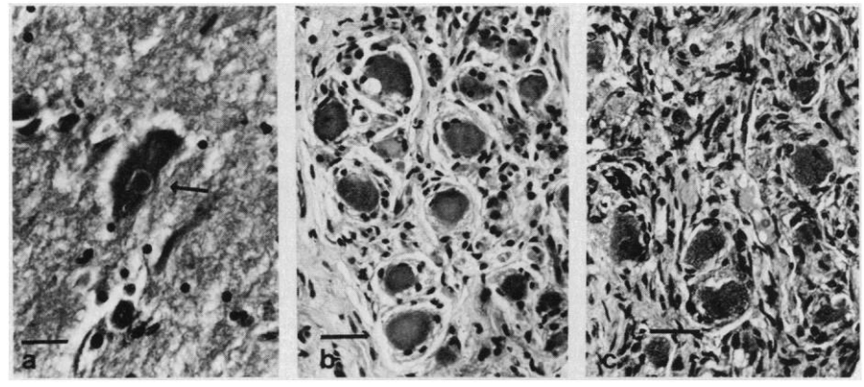

Fig. 1. (a) Locus coeruleus from a patient with idiopathic orthostatic hypotension (case C): an intracytoplasmic Lewy body surrounded by a clear halo is seen within a melanin-containing, degenerating neuron (arrow). Bar = 160 μm. (b) Sympathetic ganglion from a control patient: autonomic neurons are surrounded by a small number of capsule cells. Bar = 250 μm. (c) Sympathetic ganglion from a patient with IOH: compared to the control ganglion in (b), there is a smaller number of neurons accompanied by a moderate increase in number of capsule cells and amount of interstitial connective tissue. Bar = 250 μm.

Table 1. Dopamine β-hydroxylase and tyrosine hydroxylase activities in postmortem specimens from seven control patients and three patients with idiopathic orthostatic hypotension. Patients are listed individually with numbers for controls and letters for the IOH group. Figures for enzyme activities are the means of triplicate determinations and are expressed as nanomoles per milligram (wet weight) per hour for DBH and picomoles per milligram (wet weight) per hour for TOH. Blanks are $\leqq 0.15$ nmole mg^{-1} $hour^{-1}$ for DBH and $\leqq 0.2$ pmole mg^{-1} $hour^{-1}$ for TOH. Note that two ganglia from each patient with IOH were assayed for TOH. Abbreviation: NA, not available. See text for details.

Patient	DBH activity		TOH activity			
	Sympathetic ganglion	Locus coeruleus	Sympathetic ganglion	Locus coeruleus	Substantia nigra	Caudate (head)
Control						
1	0.85	NA	48.2	NA	NA	NA
2	1.27	0.89	5.6	Blank	Blank	Blank
3	1.27	4.13	18.4	0.4	0.4	0.8
4	1.05	3.32	97.5	32.9	2.7	19.0
5	1.98	2.43	62.8	14.5	22.9	23.7
6	NA	3.99	NA	10.9	3.4	10.6
7	0.50	1.06	2.2	0.3	0.3	0.4
IOH						
A	Blank	2.56	Blank 15.8	Blank	12.4	36.7
B	Blank	3.20	6.9 2.7	Blank	Blank	Blank
C	Blank	1.40	79.6 29.5	Blank	45.0	19.4

trast, TOH activity in the locus coeruleus was markedly depressed in the IOH patients. Enzyme activity was at or below blank values (0.2 pmole mg^{-1} $hour^{-1}$) in each locus from the IOH patients, whereas mean activity was 11.8 ± 5.98 pmole mg^{-1} $hour^{-1}$ in the control group (Table 1). Thus, TOH activity was at least 50 times lower in loci coeruleus of IOH patients. In control patient 2 activity in all brain areas was also at blank levels, but this subject died with disseminated lupus erythematosus and had multiple areas of brain necrosis (Table 1).

Nigrostriatal TOH activity was depressed only in patient B with IOH, and this patient had Parkinsonian symptoms and nigrostriatal neuronal loss (Table 1). In the other patients with IOH, none of whom had significant extrapyramidal dysfunction or histopathological changes, nigrostriatal TOH activity was within the normal range.

In our observations there is a remarkably good correlation between the histological and biochemical abnormalities. The results suggest that noradrenergic neurons of the central as well as the peripheral nervous system are severely affected in this degenerative neurological disease. Our results also suggest that different noradrenergic neurons are affected differently in IOH. In the IOH patients, DBH activity was undetectable in sympathetic ganglion neurons, whereas TOH activity was normal. Conversely, in the locus coeruleus TOH activity was undetectable, whereas DBH activity was normal. Dopamine β-hydroxylase and tyrosine hydroxylase are thus altered in a nonparallel fashion in these two populations of abnormal neurons, which indicates that the biochemical alterations are not simply secondary to neuronal dropout.

Although noradrenergic neurons were abnormal in brain as well as periphery in all the IOH patients, the nigrostriatal dopaminergic system was apparently affected only in the single patient with extrapyramidal signs and symptoms. It appears that not all catecholaminergic neurons manifest biochemical deficits in patients with IOH, and that noradrenergic and dopaminergic cells may have differing susceptibilities.

Although the data do not yet permit definition of the underlying mechanisms in IOH, the observations may be relevant to the general problem of the regulation of neurotransmitter biosynthesis. Considerable evidence from animal models suggests that in normal noradrenergic neurons TOH catalyzes the rate-limiting step in norepinephrine biosynthesis (*6*). Our observations, on the other hand, suggest that in sympathetic ganglia from patients with IOH, DBH activity may be undetectable while TOH activity is normal. This raises the possibility that in diseased human neurons DBH may become rate limiting in norepinephrine biosynthesis.

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Tournaments and Slavery in a Desert Ant

Abstract. *Many species of ants engage in physical fighting when territorial borders are challenged. In contrast, colonies of the honeypot ant species* Myrmecocystus mimicus *conduct ritualized tournaments, in which hundreds of ants perform highly stereotyped display fights. Opposing colonies summon their worker forces to the tournament area by means of an alarm-recruitment system. When one colony is considerably stronger than the other, the tournament quickly ends, and the weaker colony is raided and its ants "enslaved." This is the first example of intraspecific slavery recorded in ants.*

Ritualized aggressive behavior, sometimes entailing tournaments and sometimes pure displays, has been described in many invertebrate and vertebrate animal species (*1*). That it has never been observed in ants is surprising, because many ant species are territorial and frequently conduct physical combats with conspecific neighboring colonies. One would expect to find a more ritualized form of aggression in those species that are furnished with strong mandibles or stings but are also vulnerable because of a thin cuticle. I now report such a case of ritualization in the ant *Myrmecocystus mimicus* during territorial combats with conspecific neighbors and describe how territorial aggression in an ant species can lead to intraspecific "slavery," of a kind hitherto unknown in the social insects.

Myrmecocystus mimicus is one of the honeypot ant species, which are abundant in the mesquite-acacia community in the southwestern United States. Like other members of its genus, *M. mimicus* has a thin cuticle, apparently a necessary adaptation to its special honeypot biology. The members of the honeypot caste function as living storage containers; and when their crops are heavily filled, their gasters can be expanded to almost the size of a cherry (*2*).

In some parts of our study area in Arizona (near Portal) foraging grounds of neighboring colonies of *M. mimicus* frequently overlap. As a result, there are often massive territorial confrontations. However, in contrast to most ant species studied, the territorial conflicts do not consist of deadly physical fights, but, rather, of elaborate tournaments in which few ants are injured. Hundreds of ants participate in these affairs, which take place along the challenged territorial border. They can last for several days, being