portion of each tissue was fixed in 10% formalin and dehydrated in ethanol. Paraffin sections were stained with hematoxylin and eosin for histologic study.

Results and discussion. Although these investigations were not designed to study viral infection of the sympathetic ganglia, an interesting pathologic change was found in the tissue mass identified as a spinal sympathetic ganglion removed from 1 of the 9 monkeys. This monkey was killed 88 days after i.v. inoculation with Coxsackievirus B_4. The ganglion cells were relatively well preserved, but chromatolysis and neuronophagia were extensive. There was diffuse infiltration of histocytes and lymphocytes around the ganglion cells (figure). The inflammatory cells extended into surrounding tissue, and dilatation of vessels with vascular congestion and perivascular edema were present (figure). In the affected areas, demyelination was also noted.

Viral ganglionitis as described in this report must produce functional disturbances of sympathetic ganglia and, in turn, autonomic nervous system disturbances which could result in important organ dysfunction. It is also possible that such viral autonomic nervous system disease may be responsible for obscure disease manifestations or may even be the cause of so-called idiopathic diseases in man. Some clinical disturbances of the gastrointestional system, respiratory system or cardiovascular system of unknown etiology, for example, may be due to dysfunction of the autonomic nervous system produced by viral infection of sympathetic ganglia, especially since viral infections are so common in man. Furthermore, Coxsackievirus B[3], Coxsackievirus A[4] and several types of ECHO viruses[5] have been recovered from patients with clinical diagnosis of nonparalytic poliomyelitis or 'aseptic' meningitis[6]. Certain serotypes of Coxsackie and ECHO viruses have been etiologically considered in some illnesses resembling paralytic poliomyelitis[7].

There is a need to study in great detail viral infections of the autonomic nervous system, at least those due to neurotropic viruses, and the resultant changes in function of various organ systems. The finding of viral sympathetic nervous system ganglionitis in the monkey reported here and in a number of mice reported in the past[2] reveals the great potential of such observations.

It is well to note that histologic changes were also found in other organs of these infected monkeys, including the heart, lung, liver, kidney, brain, meninges and pancreas.

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Adrenergic neuroplasticity is maintained in the nutritional rehabilitated adult rat

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Summary. This work examined the capacity of intact catecholaminergic axons to grow, in response to lesions, in the brain of adult rats following nutritional rehabilitation. The partially deafferented epithalamic habenula was used as a model to study neuronal plasticity. Noradrenergic neurons appear to maintain their plasticity in rats recovered from their postnatal undernutrition.

It has been well documented that in certain regions of the mature central nervous system, axonal sprouting and formation of new synapses occur in sites vacated by the degeneration of nearby axons (Kerr[4] and Eccles[5]). These plastic changes may play an important role in the recovery of function lost by neuronal injury. Several lines of evidence, however, suggest that neuroplasticity-induced recovery may be greatly influenced by the neurochemical and physiological state of the organism[6]. Consequently, more information is needed on this fundamental, and still unresolved, issue in order to identify factors which limit or facilitate the process of axonal sprouting.

Postnatal undernutrition of humans and laboratory animals has provided evidence, although equivocal, of diminished learning capacities and behavioral maladaptation, which persisted long after nutritional rehabilitation[7,8]. It is not clear whether the reported disorders may have been associated with retarded synaptic plasticity, since studies on the effects of undernutrition on synaptogenesis and neuronal complexity have yielded inconsistent results[9–13]. The present experiments were designed to study the effect of early postnatal undernutrition on axonal sprouting capacity in response to damage in the mature brain, following nutritional rehabilitation. Attention was focused on catecholaminergic neurons, since they have been implicated in modulating various behavioral patterns[14]. The habenula, an epithalamic limbic structure, was used in this work to study neuroplasticity; partial deafferentation of the habenula by stria medullaris lesions has been found to induce adrener-

Catecholamines in deafferented adult habenula after nutritional rehabilitation

Neonatal nourishment	Sham-treatment (ng/mg protein)	Stria medullaris (ng/mg protein)	Lesions* (% change)
	Norepinephrine		
Well-fed control	9.8±0.6	13.7±0.6	↑39**
Deprived	10.5±0.7	13.8±1.1	↑31**
	Dopamine		
Well-fed control	2.6±0.5	3.0±0.6	
Deprived	3.1±0.7	2.8±0.4	

Results given as mean±SEM for groups of 10 rats each. * Survival time was 4 weeks. ** The change compared to sham-treated animals is significant $p<0.01$, using the one-way analysis of variance.

gic sprouting in this region[15]. In addition, the habenula is of particular interest since it may play a role in the control of feeding[16].

Materials and methods. Timed pregnant Long Evans rats were obtained from Charles River Co. (Boston, MA). A total of 5 litters consisting of 10 pups each, were used in the experiments. 5 pups from each litter were undernourished by depriving access to the mother, with the remaining 5 serving as well-fed littermate controls. Experimental and control groups always contained an equal ratio of male to female pups. The regime of postnatal undernutrition has been characterized and typically leads to a deficit of about 50% b.wt by 21 days (weaning)[17]. Actual weights obtained here were: control 57 ± 1 g, undernourished 29 ± 1 g. From 21-60 days of age all rats were permitted free access to standard laboratory rat chow, which led to growth 'catch-up' to within 5% of the well-fed controls. Actual weights obtained for test and control rats were 360 ± 8 g and 380 ± 4 g, respectively. At 60 days of age, bilateral stria medullaris (SM) lesions were placed in 1 group of test rats and in 1 group of control rats. The remaining were sham-treated animals in which the SM was maintained intact, as previously described[15]. 4 groups were thus obtained: 1. well-fed control, sham treated; 2. well-fed control, lesioned; 3. undernourished-rehabilitated, sham treated; and 4. undernourished-rehabilitated, lesioned. All animals were allowed feeding ad libitum for 4 additional weeks, after which they were sacrificed by decapitation. The brains were frozen immediately in dry ice, and the habenular nuclei were removed by microdissection[15]. Following homogenization in 0.1 N perchloric acid, aliquots were taken for protein[18] and catecholamine[19] assays.

Results. The levels of NE and DA were essentially the same in both sham-treated groups, namely the well-fed controls and the neonatally deprived rats, following nutritional rehabilitation. In response to SM lesions, however, NE increased significantly ($p < 0.01$) in both well-fed controls and deprived rats (table). The NE and DA in the habenula of males and females were identical, therefore results for both sexes were pooled for each experimental group.

Discussion. The increase in NE is most likely to be associated with noradrenergic axons sprouting, rather than with the accumulation of this amine in neuronal terminals. This assumption is based on morphological evidence provided by a recent study, in which proliferating catecholaminergic terminals in the deafferented habenula were visualized by histofluorescence[15]. These data indicate that noradrenergic plastic capacity in the habenula is retained during nutritional rehabilitation. Interestingly, plastic retention has also been reported in the mature cerebellar Purkinje cell dendrites following nutritional recovery[13]. It is not known, however, if other brain regions maintain neuronal plasticity under similar conditions of postnatal undernutrition and recovery. Furthermore, whether or not the newly formed adrenergic synapses are functional, is still an open question. Thus, while early undernutrition appears to have no apparent lasting effect on noradrenergic axon sprouting in the habenula, the possibility is not ruled out that loss of neuronal plasticity is associated with behavioral retardation.

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Variations in cardiac noradrenaline content during sodium loading in hypertension prone and resistant rats

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Summary. The cardiac catecholamine content of Sabra rats and their 2 genetically derived substrains, hypertension prone and resistant rats, was studied by high pressure liquid chromatography and electrochemical detection. Both in the control period and after sodium and DOCA administration the cardiac noradrenaline level is higher in hypertension resistant rats than in Sabra rats, and also higher than in hypertension prone rats. This finding suggests that a reduction of the cardiac sympathetic nervous tone is involved in the genetic resistance to sodium.

Numerous studies suggest an enhanced activity of the peripheral sympathetic system in various models of experimental hypertension. A decrease in the content of endogenous noradrenaline (NA) in the heart has been described in deoxycorticosterone (DOCA) and salt hypertension[1,2]. Both impaired storage[3,4] and increased turnover of noradrenaline[5,6] have been described in the hearts of DOCA-salt hypertensive rats. It has been shown recently that the faster disappearance of endogenous noradrenaline or exogenous labelled noradrenaline observed in vivo in these hypertensive rats should be attributed to an increased release due to enhancement of nerve impulse flow rather than to an impaired storage ability[7]. The relevance of the above abnormalities to hypertension remains uncertain, since spontaneous hypertensive rats appear to have unchanged levels of endogenous noradrenaline, associated with a decreased rate of NA synthesis[8]. Moreover, several studies have suggested that the sodium overload per se