which was similar to the AD induced by activation of the ipsilateral pyriform cortex. The duration of the seizure in different animals varied between 15 and 95 sec, depending on the stimulation parameters and the position of the electrodes. In the same animal, the duration of the AD did not greatly change when stimulation parameters remained constant. In chronic animals free to move in a behavioural cage, the paroxysmal bioelectric pattern was followed by the appearance of the well-known critical phenomena: myoclonic movements, mydriasis, salivation. Conditioning stimulation of the entopeduncular nucleus constantly inhibited the focal AD evoked in the amygdala. In chronic animals, both the bioelectric activity and the behavioural pattern were inhibited. Using parameters of pallidal conditioning stimulation which induced the maximal inhibition of the amygdaloid seizure, conditioning stimulation of the caudate nucleus, with the same parameters, was less effective than entopeduncular activation to reduce the duration of the AD. For 5 animals the mean duration of the amygdaloid AD was 37.5 ± 8.10 sec. Conditioning stimulation of the caudate and entopeduncular nuclei reduced the duration of the seizures to 26.56 ± 4.57 and 15.07 ± 1.87 sec respectively. Injection into the entopeduncular nucleus with kainic acid, a neurotoxic drug analogous to glutamate, which causes an almost complete destruction of cell bodies in the striatum[9-11], resulted in a decrease (about 50%) of the inhibition induced by caudate nucleus on the amygdaloid AD. For 3 animals the mean duration, 39.06 ± 4.5 sec, of the amygdaloid seizure was reduced by caudate pre-stimulation to 26.08 ± 3.68 sec. After entopeduncular injection of kainic acid the mean duration of the AD, controlled by caudate activation, was 31.42 ± 3.99 sec. The effect, which appeared 120 min after injection, was permanent.

Our results show that conditioning stimulation of the entopeduncular nucleus inhibits, to a greater extent than the stimulation of the caudate nucleus, focal paroxysmal activity evoked in the ventro-basal complex of the amygdala both in acute and chronic animals. Permanent lesion in the entopeduncular neurons induces a decrease of the inhibitory effect of the caudate nucleus on the amygdaloid AD. It may be assumed that the globus pallidus represents one possible pathway through which the neostriatum controls the focal paroxysmal activity of the amygdala.

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Coxsackievirus B_4 infection of spinal sympathetic ganglion[1]

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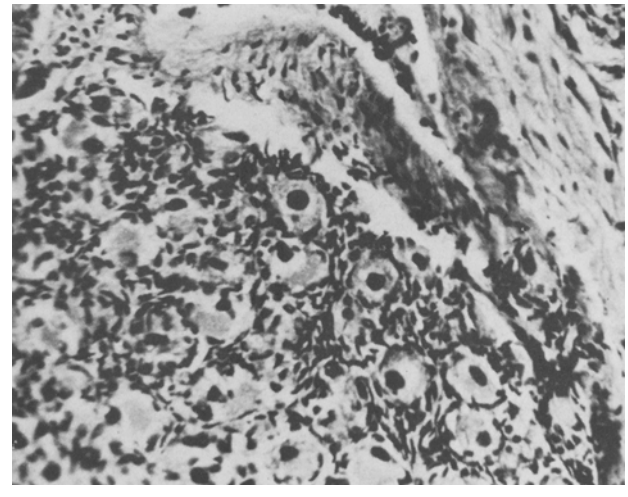
Summary. Coxsackievirus B_4 infection of a spinal sympathetic ganglion of a squirrel monkey is described. Chromatolysis and neuronophagia were extensive. It is suggested that such viral sympathetic ganglial infections may be responsible for dysfunction of organ systems.

Pathologic changes have been found with Coxsackievirus B infections in all types of organs and tissues of experimental animals during studies in this laboratory for over 15 years. In a previous report[2], we described infection of many sympathetic ganglia produced by Coxsackievirus B_4 in mice. Infection of a spinal sympathetic ganglion was recently observed in a squirrel monkey inoculated i.v. with Coxsackievirus B_4. This finding is important because the observation reveals that a viral infection can produce ganglionitis in a primate.

Material and methods. 9 young squirrel monkeys, *Saimiri sciureus*, aged 6 days to 175 days and held individually in isolator cages, were inoculated i.v. or i.p. with 0.5 ml of Coxsackievirus B_4 grown in Vero cells and having a titer of $10^{-6.5}$ $TCID_{50}$ per ml. The animals were killed by pentobarbital injection from 7 to 120 days after inoculation. Each animal was carefully examined grossly and all organs were removed immediately after death. In 1 monkey a small mass (approximately $1 \times 1 \times 2$ mm) was noted near the posterior thoracic wall where the sympathetic ganglia are normally found. The mass had the gross appearance of a lymph node. Because of its rather unusual gross appearance and its location, it was collected along with other tissues. A



Spinal sympathetic ganglion of a monkey killed 88 days after Coxsackievirus B_4 inoculation, showing inflammatory cell extension into surrounding tissue and vascular congestion and perivascular edema. H&E, × 112.

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.