operation in all groups. The amplitude of the summation of S waves in the right precordial lead and R waves in the left precordial lead increased significantly in all denervated SHR ($p < 0.05$). In contrast, a decreased amplitude was seen in the same summation in the control groups. The ratio of heart weight to b.wt in SHR was larger than that in WKR. However, there were no significant differences between the denervated and the non-denervated groups in SHR and WKR.

Histologically, the cell diameters in the anterior, lateral and posterior portions of the left ventricle, and also in septum and right ventricle were measured at the cross position of the nuclei. The diameters of all portions of left ventricle and septum in the denervated SHR were significantly larger than those in the non-denervated SHR (table). Electron microscopic findings showed a decrease in glycogen storage, swelling of mitochondria, disruption of cristae and edematous changes in the myocardial cells. These results suggest that cardiac hypertrophy in SHR is enhanced by denervation of the heart despite the lack of elevation of blood pressure. The mechanism by which cardiac hypertrophy is increased after denervation is not clear. However, a report says that the left stellate ganglectomy may lead to a reduction in free lysosomal enzyme activity, implying a possible reduction in protein degradation in the cell[4]. In conclusion, our data supply evidence that cardiac hypertrophy in SHR may be accelerated by left cervical sympathetic denervation.

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Effect of adrenalectomy on sound-induced seizure susceptibility and intensity in genetically susceptible rats

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Summary. Neither seizure susceptibility nor intensity was altered by sham-operation or by adrenalectomy in adult rats that are genetically susceptible to sound-induced seizures. Thus, sound-induced seizures in genetically susceptible rats are analogous to those in genetically susceptible mice to the extent that removal of the adrenal glands does not alter established seizure characteristics in either species.

There are several mouse and rat models which exhibit characteristic seizures in response to loud sounds. These audiogenic seizures have been found to occur in mice following acoustic priming or ethanol withdrawal, and in several strains of mice that have a genetically determined predisposition to this type of seizure[1-3]. There is also a strain of rats which has a genetically determined sound-induced seizure susceptibility. This rat model has generally been called the audiogenic seizure (AGS) susceptible rat. More recently, however, we have come to realize that this designation is too restrictive and these animals might more appropriately be called epilepsy-prone rats.

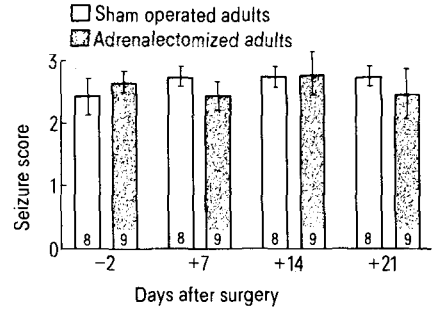
Adrenalectomy, inhibition of glucocorticoid synthesis or administration of a glucocorticoid receptor antagonist prevents the development of audiogenic seizures induced by acoustic priming or by ethanol withdrawal in mice[1-3]. Neither adrenalectomy[4] nor inhibition of glucocorticoid synthesis[1] alters susceptibility to audiogenic seizure in genetically susceptible mice. There are a number of similarities and a number of differences between rats and mice that are genetically susceptible to sound-induced seizures[5]. Therefore, experiments were carried out to determine the effect of adrenalectomy on seizure susceptibility and intensity in the epilepsy-prone rats with a genetically determined predisposition to sound-induced seizures.

Methods. Adult male and female rats from the Shreveport Veterans Administration Medical Center Colony of Sprague-Dawley derived epilepsy-prone rats were employed in this experiment. Since not all animals in this colony are susceptible to sound-induced seizures, each animal was tested 4 separate times to assure susceptibility to sound-induced seizures. The acoustic stimulus was administered to individual rats in a cylindrical metal chamber approximately 40 cm in diameter and 50 cm in height. A sound level of approximately 115 dB relative to 2×10^4 dyne/cm^2 was generated by 2 Edwards bells ringing simultaneously. The sound stimulus was initiated within 15 sec after each animal was placed into the chamber. Seizure intensity was assessed through use of the audiogenic response score (ARS) rating scale (0–9, where each higher number represents a greater seizure intensity) which has been described previously[6].

Seizure incidence

	Days after surgery			
	−2*	+7	+14	+21
Sham-operated	8/8	8/8	8/8	8/8
Adrenalectomized	9/9	9/9	9/9	9/9

* Minus 2 indicates 2 days before surgery.



Seizure intensity (mean ± SEM) in sham-operated and adrenalectomized rats. -2 indicates 2 days before surgery. The numbers inside the bar graphs indicate the number of animals. There were no statistically significant differences.

2 days after the 4th seizure test, approximately half of the animals were adrenalectomized through a midline dorsal incision under pentobarbital anesthesia. Controls were sham-operated. Thereafter, both groups had free access to food and were given a choice between tap water and 0.9% saline to drink. At weekly intervals following the surgery, each animal was tested to determine seizure susceptibility and intensity. Differences between means were tested using a 2-tailed t-test.

Results and discussion. The table shows that neither sham-operation nor adrenalectomy altered the fraction of animals susceptible to sound-induced seizures. The figure shows that neither sham-operation nor adrenalectomy altered seizure intensity in the seizure susceptible rats. Thus, sound-induced seizures in the genetically susceptible rats are analogous to those in genetically susceptible mice to the extent that removal of the adrenal glands does not affect the established seizure patterns in either species. In some other respects these two models differ considerably. For example, seizure intensity in the rat is inversely related to the level of noradrenergic and 5-hydroxytryptaminergic activity in the central nervous system. Dopaminergic tracts, in the rat, are not involved[6]. In the mouse, strong evidence suggests a role for dopamine in seizures whereas the evidence for a role of noradrenaline is more equivocal[5]. The biological basis for sound-induced seizure susceptibility in the rat has not been fully elucidated. However, recent data suggests that at least 2 biological substrates are involved. We believe one substrate to be an abnormality in cochlear function and another to be a deficit in noradrenergic and 5-hydroxytryptaminergic activity in the central nervous system[7].

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Lasting effects of acute dehydration and post-weaning undernourishment on cortical spreading depression in adult rats[1]

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Summary. Acute dehydration (D) early in life made adult rats less susceptible to cortical spreading depression (SD) than control (C) rats. Post weaning undernourished (U) rats tended to be more susceptible than controls. The association of D and U (DU group) made rats more susceptible to SD than U-rats. It is suggested that this association gives rise to a more complex pathological state than that which would result from the summation of the effects of its components.

The nervous system of undernourished animals appears to have a greater susceptibility to seizures when compared with that of normal animals. The threshold current of electroconvulsive shocks needed to provoke seizures in undernourished rats is significantly lower than that for well-nourished rats, and these seizures are of longer duration[3]. The propagation rate of cortical spreading depression (SD) is increased in rats undernourished from birth until weaning[4] or adulthood[5]. Adult rats undernourished during the nursing period develop hippocampal epileptiform kindling significantly faster than normal animals[6]. In children, undernutrition is often associated with secondary pathologies, which can exacerbate the precarious health condition of the infants. The episodes of systemic dehydration produced by diarrheas are among these pathologies[7], and so undernourishment and dehydration are often associated in childhood. The effects of this association (dehydration + undernutrition) upon brain excitability have not been studied systematically. Rocha[8] reported that acute episodes of dehydration impaired the performance of well-nourished rats in an avoidance-conditioning situation but that the already impaired performance of undernourished rats was not further diminished. In the present work we have studied the effects of dehydration and chronic post-weaning undernutrition on the susceptibility of the cerebral cortex to SD - a phenomenon which is closely associated with epilepsy[9].

Materials and methods. 49 Sprague-Dawley rats of both sexes were divided into 4 groups: C (control, 14 rats), D (dehydrated, 12 rats), U (undernourished, 13 rats) and DU (dehydrated and undernourished, 10 rats). The groups C and D received a normal diet, containing 20% casein as protein source. The groups U and DU were fed a protein-free diet during the 2 weeks after weaning. Subsequent to this period they were fed a diet containing 5% casein. All diets contained about 390 kcal/100 g. 3 sessions of dehydration were performed in the D and DU groups, as described by Rocha[8]. These occurred on the 18th, 21st and 24th days of life. Briefly, 0.2 ml of the laxative, 4-4'-(2-picoliliden)-bis-phenylsulphate, was administered by means of an oesophageal cannula introduced through the mouth. This caused a diarrheic state, with resultant loss of water and electrolytes. After receiving the laxative the rats were maintained without water and food for 10 h. They were weighed before and after each session and those which did not lose at least a mean value of 5% of their body weight in the 3 sessions were not used. The recordings were made when the rats were 60–110 days old. Under anesthesia (urethane, 750 mg/kg + chloralose, 60 mg/kg, i.p.), the animal's head was fixed in a stereotaxic apparatus and 3 trephine holes drilled. SD was elicited by placing a cotton pledget (1-2 mm diameter) soaked in 2% KCl on the cortical surface. Electrocorticograms (ECoG) were performed at 1 point and the slow potential changes (SPC)