creasing pressure in order to regain hydrostatic balance. The experiments of Jacobs (*10*) indicate that this species is capable of relatively rapid compensation to sudden changes of pressure of half an atmosphere. The gas is produced by a well-developed gland and giant cells located at the base of the pneumatophore. Protrusion of extensible processes which would increase the surface area, and therefore their resistance to sinking, might aid in this process. It is generally agreed that vertical movements of scattering layers are related to changes in deep-sea illumination (*1*). It is therefore of interest to note that Mackie recently reported that the sister species, *Nanomia cara,* is phototactic, and responds to light stimuli by swimming (*15*). The pigment effector cells which he describes may very well play a role in controlling vertical movements. Apparently, then, though full documentation is lacking, this type of siphonophore is ideally suited to perform long diurnal migrations with little expenditure of energy.

Siphonophores make up a large portion of the plankton in the warmer oceans of the world. The species of our bathyscaphe observations, *Nanomia bijuga* is cosmopolitan in distribution (*11*) and is considered the most common physonectid in the waters adjacent to the California coast (*16*). Certain anomalies of siphonophore distribution are also informative. For example, few species are reported from arctic and antarctic waters; the latter region develops deep scattering layers only spasmodically.

Thus it appears that physonectid siphonophores fulfill all the prerequisites of a major scattering organism. In view of this evidence we suggest that the primary cause of diffuse zones of scattering recordable on echo sounders in mid-depths off the California coast—and very probably throughout the warm water oceans of the world—are such organisms.

That this has not been considered before is attributable primarily to the fragile nature of these colonies so that it is impossible to sample them adequately with conventional nets (*17*). On contact with mesh or bridle, the tentacles stick by their stinging cells, the individuals making up the colony break away, and most are lost through the large meshes of high-speed nets which have been most commonly used in scattering-layer research. Further, because of their pellucid nature they are extremely hard to photograph and could easily have been missed in studies of the deep scattering layer with cameras and underwater television (*18*). On the other hand, several biologists who have descended in bathyscaphes have reported seeing siphonophores at depths of the deep scattering layer, although they did not relate them to zones of scattering (*19*).

The *Trieste* siphonophore observations indicate that we are dealing with a zone of mid-water predators—a living net—stretched across the world's oceans. Obviously populations of such magnitude must play a key role in the overall economy of the oceans (*20*).

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Electroconvulsive Threshold Elevation: From Daily Stimulation of Adrenalectomized Animals

Abstract. *The elevation of electroconvulsive threshold, which develops in cats during repeated daily measurements thereof might result from an increased production of deoxycorticosterone. Bilateral adrenalectomy followed by maintenance on deoxycorticosterone and cortisone in fixed dosages did not prevent subsequent elevation of the threshold in either cats or miniature dogs. The elevation rate in the adrenalectomized dogs exceeded that in the intact control dogs. This elevation, which resembles tolerance, in the intact cat or miniature dogs, is not dependent on an increased production of adrenocortical hormones; it may more likely be the result of cerebral rather than extracerebral adaptation.*

A significant increase in electroconvulsive thresholds (ECT) develops in cats during periods when regular daily determinations are made. After the elevation has occurred, threshold returns almost to original levels if occasional rather than daily measurements are made (*1*).

This "tolerance" to repeated electrically induced convulsions might result from an increased production of deoxycorticosterone (*1*), which has been reported to cause elevation of the threshold in rats (*2*). Electroconvulsions might cause an increased production of this hormone, since adrenal hyperplasia has been described in rats so treated (*3*). This hypothesis could be tested by removing the adrenals and maintaining the animals on fixed doses of corticosteroids before and during the periods of daily electrostimulation. The purpose of this report is to show that the elevation in the electroconvulsive

threshold occurs also in adrenalectomized cats and dogs.

Two cats and two miniature dogs (mixed Chihuahua and toy terrier) were adrenalectomized by way of a posterolateral approach in two stages. After the operation, the animals received 3.5 to 5.0 mg of deoxycorticosterone acetate (DOCA) and 7.5 to 10 mg of cortisone acetate daily. Two to three weeks later an electrode was fixed in the skull over each cerebral hemisphere and determinations of the electroconvulsive threshold were initiated (*1*). In the morning and afternoon of each day the voltage from a Grass S-4 stimulator was delivered to the epidural surface of each hemisphere in 3-volt increments at successive 5-minute intervals, until a generalized tonic-clonic convulsion ensued. Biphasic pulses 2 msec in duration were delivered for 5 seconds at the rate of 200 per second. The milliamperage was derived from pulse heights measured on an oscilloscope during stimulation (*1*). After the threshold was determined, a second convulsion was induced at the usual time interval and voltage increment because this seemed to enhance the rate of elevation of the threshold in normal cats. Thus, all the animals had both a threshold and a suprathreshold convulsion twice daily. After threshold studies were completed, the hormone injections were discontinued and distilled drinking water was substituted for tap water. Prolonged survival of adrenalectomized animals under these conditions would indicate either incomplete removal of the adrenals or the presence of functioning accessory adrenal cortical tissue (*4*). Two cats and two miniature dogs, that were neither adrenalectomized nor injected with steroids, were used as controls.

Both intact and adrenalectomized cats developed elevations of the threshold during the first 2 weeks of daily stimulation (Table 1). In the two adrenalectomized cats threshold values increased 58.9 and 72.8 percent during this interval. The intact cats developed elevations in threshold of 69.7 and 147.8 percent under these conditions.

In the adrenalectomized cat (No. 235A) that survived sufficiently long, the elevated threshold declined toward the original while the animal was still receiving maintenance steroid injections. This cat died 23 days after the steroids were discontinued, indicating that functioning adrenal cortical tissue was not present. The other adrenalectomized cat (203A) died while measurements were being made each day. No remnants of the adrenal glands were found at autopsy in either of these animals.

Table 1. Threshold elevations in adrenalectomized (A) steroid maintained and intact cats and dogs (C).

Animal No.	Surgical treatment	Daily dose (mg)		Thresholds: mean and S.D. (ma)		Increase (%)	*P**	Return threshold (ma)	Time since last daily stimulation (days)
		Cortisone	DOCA	First four stimuli	Last four stimuli of period specified				
				Period: 13 to 14 days					
Cat 200♂†	C−			8.2 ± 0.3	20.5 ± 0.9	147.8	<.001	Died	
Cat 203♂†	A+	7.5	3.5	7.4 ± 0.3	11.8 ± 1.2	58.9	<.01	Died	
				Period: 14 to 15 days					
Cat 238♂	C−			6.5 ± 0.7	11.1 ± 0.2	69.7	<.005	7.1	23
Cat 235♂	A+	10	5	3.5 ± 0.2	6.05 ± 0.32	72.8	<.005	3.9	24
				Period 9 to 10 days					
Dog 20♀	C−			8.3 ± 0.4	9.8 ± 0.7	18.07	<.05	7.7	18
Dog 11♀	A+	10	5	4.5 ± 0.1	10.05 ± 0.85	123.3	<.005	6.0	28
				Period 6 to 7 days					
Dog 22♀	C−			3.2 ± 0.4	4.8 ± 0.1	52.4	<.10	3.5	24
Dog 14♂	A+	10	5	5.2‡	13.5 ± 0.5	159.6	<.05	5.0	27

* Based on *t*-tests; four observations in all cases except dogs 14-A and 22-C where two were used. † These animals had received a series of electroconvulsions before this study. ‡ Both observations the same value.

Both adrenalectomized miniature dogs maintained on steroids developed a more marked elevation than the intact dogs (Table 1). The former group acquired elevations of 123.3 and 159.6 percent. Their respective control dogs developed increases of 18.0 and 52.4 percent during comparable intervals. After the elevations, the thresholds returned toward normal in both the adrenalectomized and control dogs after daily stimulation was discontinued and only occasional stimulation was given (Table 1).

Both adrenalectomized dogs (14A and 11A) succumbed 13 and 14 days, respectively, after steroid injections were discontinued. No remnants of the adrenal glands were found in either dog at autopsy.

An analysis of variance of mixed design (*5*) was made on the data in Table 1 concerning the differences that developed between the initial and final determinations in all the animals. This analysis confirms that the elevation which developed in both the control and adrenalectomized animals was significant (*6*).

Although the difference in rates of elevation in the intact and adrenalectomized dogs should be confirmed in more animals, it is not likely that the difference in this study was caused by excessive amounts of DOCA. This possibility seems remote because these dogs had been receiving the steroid for several weeks before their initial threshold measurement, and the latter was in the general range for intact dogs. Moreover, the dogs maintained on steroids did not develop edema, a sign of excessive administration of these substances. Finally their threshold returned to initial ranges when occasional rather than daily convulsions were induced during continued administration of the steroids.

This study indicates that, in normal dogs and cats, the elevation of the electroconvulsive threshold is not dependent on an increased production of adrenal steroids. Thus, the increase in threshold associated with daily electroconvulsions is more likely due primarily to cerebral, rather than to extracerebral, adaptive changes. The possibility that the brain responds by increased formation of a neurohumoral inhibitor has been discussed previously (*1*), but evidence for such a mechanism has not been obtained.

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