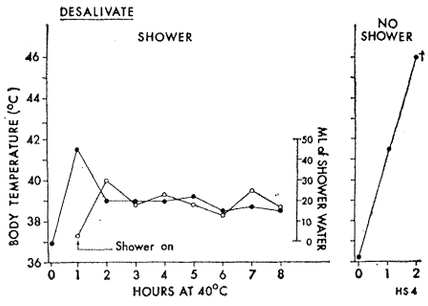

Fig. 2. Prevention by showering of lethal hyperthermia in a 40°C atmosphere by an experienced desalivate rat. Note that core temperature (solid circles) rapidly reaches 39°C and is maintained at that level when the shower is employed.

gins, it stretches upward, pointing its nose into the spray with its mouth and eyes closed.

The direct and apparently linear relation between rate of showering and increasing ambient temperature for both intact and desalivate male rats is shown (Fig. 1) for five rats tested over the range 30° to 42°C. The volume of shower water obtained during the last hour of a 1.5-hour test is shown for each animal at all temperatures tested. A solid symbol represents an intact animal; when hollow, the symbol represents the same animal after desalivation. Desalivation was by ligation and transection of all six major salivary ducts under ether anesthesia; it was confirmed by the development of prandial drinking and polydipsia in the home cage (*4*).

Note that showering begins between 30° and 32°C, the temperature at which spreading of saliva is first conspicuous in the male rat (*5*). The volume of shower water then increases for all rats with increasing temperature; the rats soak themselves at the highest temperatures, thoroughly wetting the hair of the head and neck despite the fact that some water is wasted on the bar and the floor and walls of the stall. Repeated testing of the same rat at the same temperature on different days yields comparable results (Fig. 1, especially at 38° and 40°C). Figure 1 also shows that data for animals with and without saliva overlap. Moreover, normal and desalivate core temperatures were not different when the sessions ended. These facts suggest that even when saliva is available, as in the intact rat, the animals choose the option of showers, utilize them at optimum rates, and thus swamp the contribution of endogenous water. The effectiveness of the shower for defense against heat is shown in two ways:

1) Four experienced intact rats were run for 1.5 hours at 40°C with the shower available, and then on a subsequent day without the shower. Before the tests, colonic temperatures averaged 37.8°C (range, 37.0° to 38.5°C) on the day without shower, and 37.6°C (range, 37.0° to 38.3°) on the day of the shower test. The mean core temperature at the end of the exposures to 40°C ambient was 40.4°C (range, 40.1° to 40.8°C) without the shower but only 39.1°C (range, 38.4° to 40.0°C) with the shower available. By showering, the animals maintained their core temperatures just above normal levels, and the regulated hyperthermia, seen when spreading of saliva is the only defense, was clearly reduced.

2) An experienced desalivate rat rapidly reduced its rising core temperature when the shower was made available 1 hour after exposure to 40°C (Fig. 2); thereafter it maintained itself at 39°C by taking approximately 15 ml of shower per hour. During the next test without shower the animal's core temperature rose uncontrolled in the heat and exceeded the lethal limit within 2 hours. This experiment was repeated with three other experienced desalivates; all did well when showering but became behaviorally incompetent within 2 hours when the shower was withdrawn. Clearly the shower is an effective substitute for spreading of saliva.

Drinking water was available during all these tests. Some animals did not drink while showering; others did, either from the reservoir or by consuming the shower water that condensed on the bar or the walls of the stall. Measured intakes varied widely (0.0 to 9.5 ml/1.5 hour), were inconsistent between animals, and were unrelated to increase in ambient temperature. Thirst and water reinforcement were therefore irrelevant to the behavior we discuss.

Thus rats acquire an operant with remarkable ease and effectiveness to provide themselves with exogenous water to effect loss of heat by surface evaporation. Typically they learn to take showers within 4 hours of exposure to moderate heat (36°C), and at higher temperatures they use the shower to reduce hyperthermia. Moreover, showering is an effective life-saving substitute for spreading of saliva in the desalivate rat, and it appears to be preferred by the intact rat that can either employ saliva in grooming or work for exogenous water. Our results, earlier studies (*3*) demonstrating that cold rats will work to obtain heat, and recent reports (*6*) of bar-pressing for cold air during exposure to heat emphasize the flexibility and physiological appropriateness of learned behavior for defense against extremes of ambient temperature.

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Dorsal Root Potentials after C-Fiber Stimulation

Abstract. *A pure volley from C-fibers, set up by electrical stimulation of a cutaneous nerve and subsequent selective blocking of the A-fibers, generates in the cat spinal cord a dorsal root potential (C-DRP). Its polarity is the same as that of the dorsal root potential elicited by stimulation of the A-fibers (A-DRP), thus probably providing presynaptic inhibition of primary afferents. The size of the C-DRP increases in proportion to the size of the C-volley. A preceding A-DRP reduces the C-DRP.*

The myelinated cutaneous afferents exert onto each other strong presynaptic depolarizations which are probably paralleled by inhibitory actions (*1*). The spinal organization of the primary afferent depolarization (PAD) of mechanoreceptor afferents points to the functional significance of this inhibitory interaction (*2*). Afferent C-fibers take part in the nervous transformation of many kinds of peripheral stimuli. I have studied, therefore, whether these fibers also generate PAD's by testing whether a C-volley reaching the spinal cord

evokes a dorsal root potential (DRP), the electronic spread of the PAD. C-volleys were generated by electrical stimulation of a cutaneous nerve. Slowly rising electrical currents applied more proximally to the nerve were used to block reversibly the impulses in myelinated fibers.

Eight cats either had the spinal cord cut at cervical 3 to 5 (C_3 to C_5) during a short ether narcosis and were subsequently immobilized by Flaxedil, or they were anesthetized by pentobarbital sodium (Nembutal, initial dose 40 mg/kg) and their spinal cords were cut at lumbar 1 (L_1). The lumbar spinal cord was exposed in a pool of paraffin oil, and the left ventral roots from L_5 to S_1 (sacral 1) were cut. The DRP's were recorded from L_7 dorsal rootlets (100 to 360 μ in diameter) cut 10 mm from their cord entry and put on a pair of platinum wire electrodes. The time constant of the a-c–coupled preamplifier was 2 seconds. Usually five or more successive DRP's were added in a computer (CAT 400B) at a repetition rate of one per 10 seconds or slower. The medial branch of the sural nerve of the left hind leg was exposed in a pool of paraffin oil. Three nerve sections (15 to 20 mm long) were dissected free at a distance of 15 mm from each other for the installation of electrode pairs for stimulation, blocking, and recording (Fig. 1a). Records taken from the nerve near its entry into the sciatic bundle monitored the composition of the afferent volley. Stimuli about 10 volts and 0.5 msec long (approximately 100 times the A-fiber's threshold) produced maximum C-volleys. A-fibers were selectively blocked at electrodes *B* by a current starting 1 second before the stimulus, increasing slowly to about 50 μa, where the current remained until the DRP had been recorded; it was switched off thereafter. This current preferentially blocked the A-fibers by depolarization (*3*). The C-fibers remained unblocked, probably because the strength of the current entering these thin fibers was too low to alter appreciably their membrane potential. This is consistent with the fact that much higher current strength is required for stimulation of C-fibers than of A-fibers. The slow rise of the blocking current was necessary in order to prevent stimulation of A-fibers. To minimize current spread to the recording sites, the generators of stimuli and blocking current were isolated from ground. More than a thousand stimulus-blocking cycles could be applied at a rate of one per 10 seconds without appreciable deterioration of the nerve responses.

Table 1. Comparison of the peripheral conduction velocity of the C-volley and an estimate calculated from the poststimulus latency of the DRP.

Experiment	Conduction velocity (m/sec)	
	Peripheral	Central
1	1.0	1.01
2	0.97	0.97
3	1.18	1.03
4	0.99	0.97
5	.93	.93
6	.97	1.01
7	1.09	0.92
8	0.94	.95

In Fig. 1 (b–n) the effect of blocking the volley in the myelinated fibers is demonstrated. Increasing the blocking current has little effect on the C-volley (Fig. 1, f–i), whereas the A-volley decreases (Fig. 1, b–d) and finally disappears completely (Fig. 1e). Parallel to this decrease the early deflection in the DRP records (Fig. 1, k–n) vanishes. Instead a late component shows up, which is only just detectable in Fig. 1k. My evidence relates this change in potential to the afferent C-volley, thus the notation "C-DRP" is proposed. Obviously, in Fig. 1, k–m, the C-DRP is more or less suppressed by the preceding A-DRP.

In cats anesthetized with Nembutal (30 mg per kilogram of body weight, given intravenously), the A-DRP was always prolonged (broken line in Fig. 1k), thus masking the small residual C-DRP. However, when the A-DRP was reduced, a C-DRP appeared in these experiments too (Fig. 2, a–d). The DRP in Fig. 2a was recorded after a volley in both the sural A- and C-fibers. With increasing block of the A-volley the C-DRP appeared (Fig. 2, b–d). The complete series is shown in Fig. 2e (solid circles), in which the C-DRP peak value is plotted in relation to that of the preceding A-DRP. The open circles in Fig. 2e are from another experiment.

There is rather good proportionality between the size of the C-volley and the peak value of the C-DRP (Fig. 2f), and no sign of occlusion was seen when the volley was maximum in the sural nerve. Thus the DRP is expected to increase further with additional C-input from other nerves. The conduction velocities of the C-fibers calculated from the poststimulus latencies of the C-volleys and the corresponding conduction distances always were around 1 m/sec (Table 1). The latency of the C-DRP is the sum of the nerve conduction time to the cord plus an unknown central delay. The latter was neglected, and a lower limit of the conduction velocity between stimulus electrodes and spinal cord was

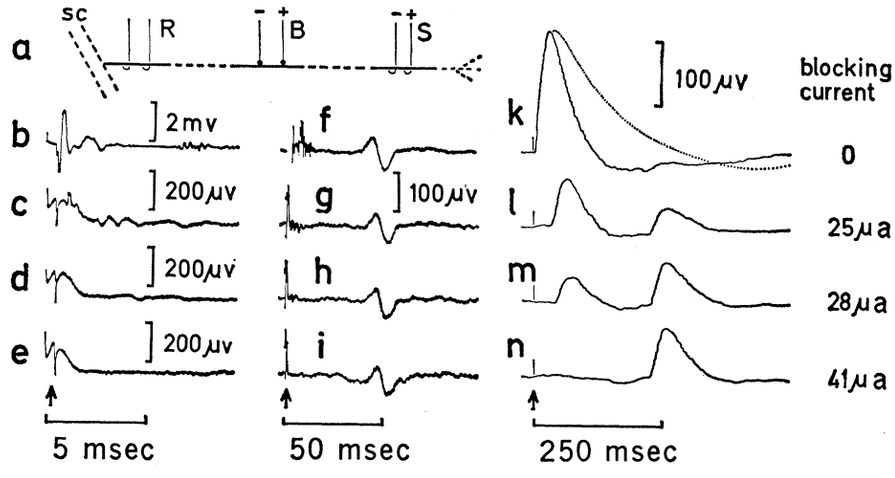

Fig. 1. The differential blocking of the A-volley and its effect on the dorsal root potentials. (a) Arrangement on the sural nerve of the electrodes for stimulation (*S*), blocking of the A-fibers (*B*), and recording (*R*); *sc*, sciatic. (b,f) Nerve records taken at *R* and displayed at different gains and sweep speeds appropriate to depict the A- and C-volleys, respectively. The stimulus given at *S* was 10 volts, 0.5 msec long. (k) Dorsal root potential (DRP, average of five successive records) evoked by the volleys shown to the left. (c–e, g–i, and l–n) Corresponding recordings, but with a blocking current applied at *B*, rising to various levels (as indicated in the last column) before the stimulus was applied. Note different gains in b and c–e, respectively. Stimuli are indicated by arrows below the records. Unanesthetized cat with spinal cord cut at C_3. The dotted line in k indicates the DRP after intravenous injection of Nembutal (30 mg per kilogram of body weight).

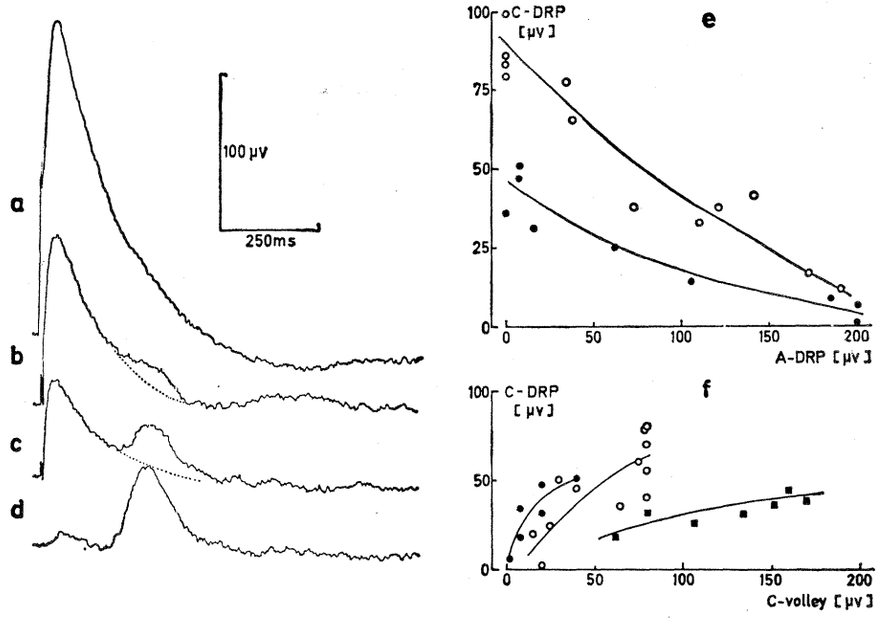

Fig. 2. Factors influencing the size of the C-DRP. (a) An average DRP (16 superpositions) following a sural nerve stimulus (35 volts, 0.2 msec) producing maximum A- and C-volleys. This experiment was on a cat anesthetized with Nembutal (40 mg/kg). In b–d the stimuli were the same as in a, but the A-volley was blocked to an increasing extent. In e the peak values of the C-DRP's are plotted against those of the preceding A-DRP's. The points are from the same experiments as a–d; the circles are from the measurements shown in Fig 1. In f the relation between the size of the C-volley (abscissa) and of the C-DRP (ordinate) is shown. The points are either from single measurements (○) or averaged values from 10 to 16 successive trials (●, ■).

estimated, which was always around the peripheral value (Table 1).

Generally, the C-DRP was greatly reduced by a preceding A-DRP (Fig. 1, k–n; Fig. 2, a–e). This was also the case when the A-volley was generated by a separate stimulus given to other cutaneous nerves, the effect of depression persisting for more than 300 msec. In similar experiments the influences of a C-DRP onto an A-DRP and onto a second C-DRP were tested. The depression of the A-DRP by the preceding C-DRP was less pronounced, but the time course was similar. When pairs of C-fiber stimuli were delivered to the sural nerve, the second C-DRP was strongly suppressed with intervals between stimuli of up to more than 500 msec.

Thus, an afferent C-volley generates a DRP having the same polarity as that caused by an A-volley; that is, the proximal electrode is more negative than the distal one is throughout the potential change. These findings are in contrast to those of Mendell and Wall (*4*), who claimed that afferent C-volleys produce a "positive DRP" (that is, a presynaptic hyperpolarization). As judged from their illustrations, most of the positive DRP's occurred too soon after the peripheral stimulus to have been evoked by C-fiber activity. Their conclusions form one of the basic postulates in a recent pain theory (*5*). According to this "gate control" theory, presynaptic hyperpolarization of afferent fibers by a C-input will facilitate the central effects of all sensory impulses. My experiments do not support this view.

Further investigations in which intraspinal potential field measurements, spinal reflex interactions, and presynaptic excitability testing of single afferents fibers are used should identify the origin of the C-DRP, and determine whether this DRP is also linked to presynaptic inhibition.

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Exencephalia: Its Occurrence in Untreated Mice

Abstract. *Exencephalia has been reported in irradiated CF1 mice but there are no reports of its occurrence in untreated mice of this strain. In the course of establishing disease-free breeding colonies from CF1 female mice delivered of their offspring by cesarean section, exencephalia was seen frequently. During a 2-week period, 90 litters were delivered; 11 contained exencephalic fetuses, at the rate of one per litter (11 of 90 litters, 12.2 percent; 11 of 1056 fetuses, 1.04 percent). The prevalence of this anomaly in untreated mice of this strain could contribute to overestimates of the effectiveness of low doses of radiation.*

In view of reports of exencephalia in irradiated CF1 mice (*1*) it is important to report the natural occurrence of this anomaly in this strain, because lack of evidence of its prevalence in untreated mice could contribute to overestimates of the effectiveness of low doses of radiation.

Doses of x-irradiation of 200 r produce exencephalia in a high incidence (26 percent) in CF1 mice (*2*), and low incidences (0.7 to 2.3 percent) have been noted after small exposures to radiation (15 r) (*3*). Naturally occurring exencephalia has been reported in several strains of mice (T_1, DB′, Cd/Cd, and AKR-T × Brachy) (*4*), but there are no reports of the occurrence of this anomaly in untreated CF1 mice.

In the course of establishing disease-free breeding colonies at the Argonne National Laboratory, many CF1 female mice were delivered of their offspring by cesarean section. Naturally occurring exencephalia was seen frequently enough to make it necessary to measure its incidence more precisely.

The breeders used to initiate our laboratory's colonies were obtained from Carworth, Inc. Those used for this study were first-, second- and third-generation descendants of the original stock. All were housed in plastic shoebox-type cages (*5*) with white pine shavings as bedding, at a room temperature of 22° to 24°C, a 30 to 40 percent relative humidity, and a light cycle of 12 hours light and 12 hours dark. The diet was Wayne Lab-Blox (*6*) and water. The cesarean sections were performed between the 14th and 20th day of gestation.