Sudden Death in the Laboratory Rat: Cardiac Function, Sensory, and Experiental Factors in Swimming Deaths

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Three reported characteristics of sudden death in the wild rat, (1) bradycardia, (2) decreased survival without whiskers, and (3) increased survival with preexposure to the stressor, are demonstrated in the domestic rat. Differences in sudden death between wild and domestic rats are discussed as well as possible interpretations of the phenomenon.

INTRODUCTION

Since Richter's (1) striking report on sudden death in the wild rat there has been virtually no systematic work published on it. Single experiments appear (2–5), yet 20 years after Richter we have little more empirical information. This may be partially due to a reluctance on the part of many experimenters to use wild rodents as their experimental animals.

Richter exposed wild rats to a severe water stress and reported three main findings. First, he made electrocardiographic recordings of his dying rats and found that they died showing a slowing of the heart. Richter reported no evidence of drowning and hypothesized that ". . . the rats may have died a so-called vagus death, which is the result of overstimulation of the parasympathetic rather than the sympathetic adrenal system" (p. 196). Second, he reported that trimming his rats' whiskers would increase the prevalence of sudden death to almost 100% of the animals tested. According to Richter, ". . . the trimming of the whiskers thus proved to play a contributory, rather than an essential role" (p. 195). Third, Richter reported that " . . . by repeatedly holding the rats briefly and then freeing them, and by immersing them in water for a few minutes on several occasions . . ." (p. 196) he could prevent sudden death. He concluded from the results of this manipulation that his wild rats learned that the situation was not hopeless and thus did not give up and die. In sum, Richter attributed the occurrence of sudden death to feelings of helplessness and giving up. He traced the physiological mechanisms of this death to an overstimulation of the parasympathetic nervous system mediated by the vagus.

Recent reports indicate (2,6) that a similar phenomenon may exist in the domestic rat. Rosellini et al. (6) have demonstrated a sudden death phenomenon in the domestic rat similar to Richter's. They found that laboratory rats housed singly and handled briefly five times a week from 25 to 100 days showed a high incidence of sudden death in the Richter apparatus. Unhandled rats or those group housed showed significantly lower rates of sudden death.

The purpose of this report will be to compare in detail the phenomenon of sudden death in the singly housed-handled

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domestic rat with that reported by Richter in wild rats and on the basis of this comparison to reevaluate the phenomenon of sudden death.

EXPERIMENT 1

The primary purpose of Experiment 1 was to monitor heart rate (HR) during sudden death in the domestic rat. HR was monitored in two groups: the singly housed and handled one, which shows a high incidence of sudden death, and the group housed and unhandled one, which shows the lowest incidence of sudden death (6). In this way not only could the pattern of HR for dying animals be assessed, but it could be compared to that of survivors.

Secondly, we shortened the period of differential housing and handlng. Roselini et al. (6) reported their sudden death results after over 2½ months of housing and handling starting at 25 days. This procedure is expensive, time consuming, and perhaps unnecessary.

Method

Twenty male Holtzman rats were received at 25 days and divided into two equal groups. Ten were singly housed in cages 9½ × 8 × 7½ inches and handled five times a week. Handling consisted of gently stroking the animal for 10-15 sec while being held by the E. The remaining 10 were housed four to a cage (9½ × 7 × 17 inches). The uneven two group-housed animals were caged with two Holtzman rats of similar age used in another experiment. Group housed animals were not handled from time of arrival to time of testing. Testing for both groups began 37 days after arrival in the lab (age = 62 days) and was completed in 11 days. All animals in the colony room were maintained on ad lib food and water under a 14:10 light-dark cycle. All experimental manipulations were carried out during the lights-on period.

The stress apparatus consisted of a plexiglass cylinder 30 inches high and 8 inches in diameter. Water shunted from the plumbing system of the laboratory through a series of ½-inch copper pipes was shot into the tank from a ¼-inch-wide spigot located 1 ft above the tank. A needle valve controlled the flow of water passing through the pipes and was monitored by a water flow gauge (U.S. gauge No. 13718). A Symmons 4-500 safety mix visu-temp valve regulated the temperature of the water in the tank and presented a visual indicator of the current temperature. This arrangement allowed for the control of temperature to the nearest degree Fahrenheit and the control of water pressure to the nearest ¼ lb per square inch. The apparatus closely resembled Richter's (1).

Prior to immersion in the tank each animal was weighed and then anesthetized with ether during which time three safety pin electrodes were inserted subcutaneously. The reference electrode was attached to the back of the animal slightly below the neck. The two others were attached to the underside of the animal, one in the area of the right foreleg and one on the left side at the bottom of the rib cage. HR monitoring was accomplished by connecting these electrodes via leads through a mercury commutator and specially designed weighted pulley to a Grass model 7 polygraph. The cummutator and pulley system allowed the animal a relatively high degree of freedom of movement while swimming and diving and prevented entanglement in the leads. Movement artifact in the polygraphic recording was greatly reduced through a preamplifier system (7). HR was evaluated by the second author counting the QRS spikes. A second "counter" naive to the purposes of the study independently evaluated 35 15-sec samples of the HR recordings. The counts were found to be highly reliable ($r = 0.99$).

During immersion in the tank, water temperature was maintained at 95° F and water pressure at 5.5 psi. The water pressure utilized in this experiment was 1 psi higher than that utilized in Experiments 2 and 3, since the weighted pulley made it slightly easier for the rat to swim on the surface of the water. Increasing the pressure offset this advantage. Each session consisted of a maximum of 25 min in the tank. Previously reported data and extensive pilot work has indicated that survival in the tank for 21 min perfectly predicts survival for long periods of time (1,6).

Results

Seventy percent of the animals in the single-housed and handled group died soon after immersion in the tank while none of those group housed and unhand-

led died. Mean time to death for the dying animals was 4 min, 26 sec. There was no significant correlation between weight before testing and death ($r_{pbi} = 0.38$, $t = 1.74$, 18 *df*, two-tailed, $P > 0.05$) or were there significant weight differences between the groups ($t = 1.15$, 18 *df*, $P > 0.05$). These data replicate that previously reported (6) and indicate that shorter periods of differential handling and housing will result in similar results.

Extensive observation of these animals indicates the following pattern of behavior. Upon immersion most animals have some difficulty in remaining on the surface of the water. This difficulty results in their sinking feet first as a result of being pushed under by the water jet and the resulting turbulence. Shortly after immersion many animals make a head first dive to the bottom of the tank. Summed over the previously reported study (6) and Experiments 2 and 3 reported here, 39% of the animals dived. The percentage in this study was quite low (5%) as a result of the constraining influence of the HR leads and pulley. In general, most rats do not dive more than once, although the range is 0-5. Most dives are 5-10 sec in duration, although dives of up to 25 sec have been reported. Virtually all diving occurs during the first 10 min of swimming. A correlational measure for diving and death is not reported for this study given the minimal number of dives.

Soon after immersion two distinct patterns of swimming emerge. Some animals experience increasing difficulties in staying above the surface. They often swim in an uncoordinated and spasmodic fashion, sinking hind feet first under the surface of the water until they are spending most of their time underwater. This perfectly predicts death. Ultimately these animals fail to regain the surface and after convulsive-like movements cease moving. For practical purposes, this cessation of movement can be considered the time of death.

This pattern is in sharp contrast to those animals who also experience minor difficulties upon immersion but soon adapt and spend most of their time on the surface swimming smoothly. These animals may sink for short periods of time or may make long dives, yet they easily regain the surface. As previously reported (6) this pattern maintained for 20 min is a perfect predictor of survival for long periods in the tank.

HR recordings were of mixed quality due to the strenuous movement of the animals and turbulence of the water around the electrodes; however, computation of HR based on the occurrence of the QRS complex was possible. The patterns of HR over time for surviving and dying animals were strikingly different. HR for surviving animals showed a relatively stable pattern over the 20-min immersion period. Immersion in the tank did not result in a consistent immediate HR response; some rats showed an increase while others showed a decrease. After 20 min of swimming most showed an HR very close to that immediately prior to immersion. Figure 1(A) is representative of this group.

This was in sharp contrast to the dying animals who always showed an immediate drop in HR upon immersion and a gradual slowing over time marked by greatly fluctuating rates. Electrical activity of the heart continued for several minutes after the cessation of movement. Figure 1(B) is representative of this group.

Discussion

Behavioral and HR data clearly differentiate dying from surviving animals. This is

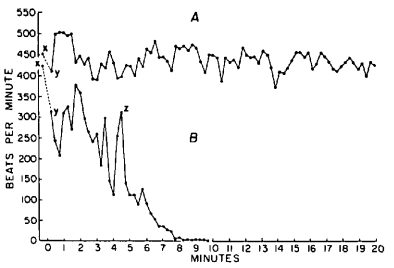

Fig. 1: Heart rate plotted at 15-sec intervals for one surviving (A) and one dying (B) animal. X = 15 sec prior to immersion; Y = first 15 sec of swimming; Z = cessation of movement.

true whether the survivor is from the single handled or the group unhandled conditions. No HR data are available for group handled animals who die since none died in this experiment. Previous observations (8) indicate that animals from the unhandled groups or other rearing conditions who die do so showing highly similar behavioral patterns to those reported here.

The general slowing of HR shown by the dying animals is similar to the "vagal death" reported by Richter. The extent of the similarity is difficult to determine because of the sketchiness of Richter's reports (1,9). We are currently trying to develop a better methodology for this kind of recording, which will further reduce movement artifact and will allow for more detailed analyses of the ECG data.

Finally, it seems unnecessary to house and to handle animals differentially for 2½ months to show the effects of environment on sudden death. Similar results were obtained after 5-6 weeks and it seems possible that this period could be further reduced. The precise determination of how long environmental conditions must be maintained and which developmental periods are crucial might lead to important clues relating to the mechanism of these effects.

EXPERIMENT 2

In this experiment an attempt was made to assess the effect of removal of the whiskers on sudden death in domestic rats.

Method

Twenty male Holtzman rats were received at 25 days and singly housed until 85 days under a 14:10 light-dark cycle. This experiment was run concurrently with Experiment 1 and thus used a 2-month housing and handling period. All experimental manipulations were carried out during the lights-on period. Each animal was handled for 10-15 sec a day five times a week during this time and maintained on ad lib food and water. Between 80-85 days animals were randomly assigned to the whisker or the no whisker groups and were placed in the Richter tank. One animal died prior to testing and one was not run as a result of illness, leaving two groups of nine. Animals in the no whisker group had their whiskers clipped with scissors immediately prior to immersion. Animals in the whisker group were placed in the tank after being held for a short period of time approximately equal to the time necessary to clip a rat's whiskers. Each animal was weighed prior to testing and a record was kept of the number and length of each dive made in the tank. Most of the rats were not allowed to die in this experiment. Extensive pilot work and behavioral data reported in Experiment 1 had indicated that it was possible to perfectly predict on the basis of swimming behavior in the tank which rats would die. Rats showing an increasing inability to stay on the surface of the water over a period of 1 to 3 min often accompanied by spasmodic attempts to regain the surface were classed as "diers" and removed from the tank. Previous observations indicate that these rats would have died within the next few minutes. This behavior is in striking contrast to "survivors" who spend the majority of their swimming time above the surface using a smooth coordinated swimming stroke. These animals were allowed to stay in the water 20-25 min and then were classed as "survivors" and removed from the tank.

Three rats were allowed to die and were subsequently autopsied; three that survived the first 20 min were immediately sacrificed and also autopsied. During immersion, water temperature was maintained at 95°F and water pressure at 4.5 psi. The swimming tank and plumbing system were identical to that used in Experiment 1.

Results

Five of 9 of the animals in the whiskers-intact group "died" as compared with 8 of 9 in the dewhiskered group. This difference is not significant ($X^2 = 1.11$, Yates corrected, 1 df $P > 0.05$). As in previous experiments there was no correlation between the weight of the animal prior to running and death ($r_{pbi} = 0.10$; $t = 0.80$, 1 df, $P > 0.05$) or was there a significant relationship between dives and death ($t = 0.16$, 1 df, $P > 0.05$).

Discussion

The cutting of whiskers was found to increase the incidence of death but nonsignificantly. This lack of whiskers, however, was not essential for the occurrence of the phenomenon. Here again the data resemble that reported by Richter for wild rats. Richter's explanation for this increase seems appropriate for both the domestic and wild rat. The whiskers or vibrissae are an important sensory organ for the rat. The elimination of this sensory input impairs the rats' coping ability in the water tank and in a variety of other situations (10,11). In watching our dewhiskered rats, it seems that they have greater difficulty in locating the surface of the water or perhaps the side of the tank.

Of note is the slightly lower incidence of death in the whiskers-intact group (56%) than that reported for this same group in other experiments [70%—Experiment 1; 80%—Experiment 3; 80% and 70% —Rosellini et al. (6)]. This difference is small and may be due to sampling error inherent in replicating the same condition repeatedly in groups with a small N. We believe, however, that there is a point to be made about variations in our data and will return to it in the final discussion.

Autopsies of the rats actually dying in the tank revealed no evidence of drowning. When compared with survivors of the water stress who were subsequently sacrificed and autopsied, 2 of 3 showed increased congestion of the lungs and all showed hearts engorged with blood. This may be indicative of right heart failure; however, this conclusion is speculative given the small N. No other differences were noted.[1]

EXPERIMENT 3

In trying to support his "helplessness hypothesis" of sudden death Richter showed " . . . by repeatedly holding the rats briefly and then freeing them, and by immersing them in water for a few minutes on several occasions . . . " (p. 196) he could prevent most of the sudden deaths in his wild rats. It is difficult to know from the brevity of Richter's description exactly what the parameters or nature of this manipulation were; however, there do seem to have been two types: (a) holding and freeing the rats repeatedly and (b) immersing and removing them several times in the tank for short periods prior to final testing.

Manipulation (a) resulted from Richter's observations that wild rats sometimes died

[1]We are grateful to Prof. H. Sheldon of the pathology department of McGill University Medical School who performed the autopsies and evaluated the results.

in his hand prior to immersion in the tank. We have never observed this death in our domestic rats. Richter's second strategy was relevant to our results. Our purpose in evaluating this intervention was not to discover the exact parameters through which we could prevent sudden death, but rather it was to see if manipulations similar to Richter's would have similar effects.

Method

Fifty rats were received at 25 days of age from the Holtzman Corporation and singly housed until 90 days. This experiment was run concurrently with Experiment 1 and thus used a 2-month handling and housing period. Each animal was handled for 10-15 sec a day during this time and maintained on ad lib food and water under a 14:10 light-cycle. All experimental procedures were carried out during the lights-on period. Animals were randomly assigned to one of five groups. Four animals died prior to the start of testing at 90 days. As a result, 4 of the 5 groups contained only nine animals per group instead of the originally intended 10. All animals had intact whiskers. Separate testing procedures were followed for each of these groups as follows.

Group 1—baseline. These animals were placed in the tank without any prior exposure to the water stress.

Group 2 —3 × 30 sec. These animals were placed in the tank three times for 30 sec prior to testing. Each 30-sec immersion was followed by a 30-sec rest period in a plastic cage. The three immersions and rest periods were immediately followed by testing for sudden death.

Group 3 —6 ×5 sec. These animals were placed in the tank six times for 5 sec prior to testing. Each 5-sec immersion was followed by a 5-sec rest period in a plastic cage. The six immersions and rest periods were immediately followed by testing for sudden death.

Group 4—6 ×1 min. These animals were placed in the tank six times for 1 min prior to testing. Each 1-min immersion was followed by a 1- min rest period in a plastic cage. The six immersions and rest periods were immediately followed by testing for sudden death.

Group 5—spaced immersion. These animals were exposed to the water stress situation over a period of 3 days. On day 1, they were immersed for three 1-min periods separated by 1-min rest periods in a plastic cage. Approximately 24 hr later this procedure was repeated. On the third day, approximately 24 hr after the second exposure to the water stress, these animals were placed directly in the tank and tested for sudden death.

As explained in Experiment 2, none of the animals were allowed to die in this experiment. The duration of immersions prior to testing was not included in the 20-min survival criterion. Water temperature was maintained at 95°F throughout the experiment while water pressure was regulated at 4.5 psi. All animals were weighed before each session.

Results

Figure 2 presents the percentage death for each group in this experiment. Both the baseline and 6 × 5 sec showed high proportions of sudden death (8 /10 and 7 /9, respectively). The 6 × 1-min and spaced immersion groups show a significantly

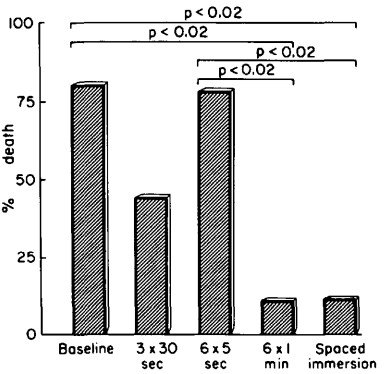

Fig. 2. Percentage death in each group run in Experiment 3.

lower proportion of death (1/9, $X^2 = 6.47$, Yates corrected, $P < 0.02$, 1 *df*). The 3 × 30-sec group showed an intermediate number of deaths (4/9) but was not significantly different from any of the others. There was no correlation between weight and death ($r_{ppi} = 0.02$; $t = 0.14$, 44 *df*, $P > 0.05$) or was there a significant correlation between dives and death ($t = 1.60$, 44 *df*, $P > 0.05$).

Discussion

These data show that under certain circumstances sudden death can be prevented in groups at high risk by short exposures to the water stress interspersed with rest periods. These exposures can immediately precede final immersion and testing for sudden death (e.g., 6 × 1 min) or can be spaced over a period of days (spaced immersion). While it is not possible to deduce from this experiment the exact parameters of preexposure necessary, it seems likely that for manipulations occurring in one session, 5-6 immersions in the tank separated by rest periods of close to 1 min will significantly reduce the incidence of sudden death (cf. group 6 × 1 min). Increasing the number of trials or the duration of rest periods might reduce the incidence of death to 0; reducing the number of preexposures or the duration of rest periods increases the percentage of death (cf. groups 3 × 30 sec and 6 × 5 sec).

In discussing similar data, Richter maintained that wild rats preexposed to the water stress learned that their situation was not hopeless and therefore did not give up and die. The data presented here are consistent which such an interpretation with the proviso that a certain minimum of trials and intertrial intervals are necessary for such learning. We believe that this proviso is consistent with a learning interpretation since it is tantamount to saying that learning is better with more trials and more spacing of trials, two generally accepted facilitators of learning (12).

There is, however, at least one other hypothesis consistent with the data. This hypothesis does not posit the learning of hope, but rather the learning of an adequate motor response, i.e., swimming in turbulent water. Rats gradually preexposed to the water stress are able to adapt their inherent swimming ability to a special situation. A variant of this hypothesis might argue that preexposure results in the adaptation or sensitization of some unspecified physiological parameter necessary for survival in this situation. Without specification of this parameter, this interpretation is problematic as is the final interpretation of the results of this experiment.

FINAL DISCUSSION AND CONCLUSIONS

The sudden death phenomenon in domestic rats reported here is very similar to that reported by Richter in wild rats. First, of course, is that a large proportion of Holtzman rats singly housed and handled after weaning will die suddenly in a water stress situation. Those surviving the first 21 min will continue to swim for long periods of time. Second, the rats who die suddenly show a definite slowing of the heart. This is similar to Richter's report of a vagal death, although the extent of the similarity is unclear.[2] Third, there is no

[2]Personal communications with Prof. Richter have failed to elucidate this due to the misplacing of his original data recorded over 20 years ago. We are grateful to Prof. Richter for trying to locate them and for his kind advice.

evidence of drowning in these rats. Fourth, preexposure to the water stress situation can prevent this death, and finally, the shaving of whiskers increases the incidence of death but is not essential to the occurrence of the phenomenon.

Some of our data differs from Richter's hypotheses and conclusions. First, sudden death does not seem to be necessarily linked to wild species. We can guess that Richter observed only a small percentage of sudden death in his domestic rats because his housing and handling conditions were not appropriate. Second, Richter implies that his rats died during dives. He writes:

> The first rat swam around excitedly on the surface for a very short time, then dove to the bottom, where it began to swim around nosing its way along the glass wall. Without coming to the surface a single time, it died two minutes after entering the tank (p. 154).

It is unclear from his report whether this is characteristic or not. However, we have never observed a rat dying in such a dive. All of our rats have succeeded in surfacing after diving, and if they do die subsequently, it is with the feet-first sinking pattern described previously.

Third, Richter suggests that " . . . the sudden death phenomenon may be a one-time occurrence both in rats and man—in any particular kind of death" (p. 197). Pilot data recently collected in our laboratory (13) indicate that domestic rats removed before death and then placed in the water situation several days later may "die again". Repeated exposures begin to lessen the probability of death, but this approximates the spaced immersion condition reported in experiment 3. One thing is clear; in the domestic rats tested in our laboratory the sudden death phenomenon need not be a one time occurrence resulting in either death or immunity.

Fourth, unlike Richter, we have never observed a domestic rat dying while being held. Anecdotal reports indicate that this sometimes occurs; however, we know of no reliable way to produce this phenomenon. It seems likely that human contact is much more of a stressor for the wild than for the domestic rat and that this may result in a much higher incidence of sudden death from contact in the wild rat.

A somewhat troubling finding is the variance in our data in these experiments and in those previously reported (6). For instance, the water pressure used in Experiment 1 of the Rosellini et al. (6) study was found to result in sudden death for all animals over all groups in Experiment 2. When this pressure was reduced the differing percentages of sudden death as a function of rearing conditions was reported. We have seen similar variance in pilot work and in the experiments reported here (cf. Experiment 2). We believe that part of this may be due to batch differences in the Holtzman rats that we use and that part may be due to sampling error inherent in replicating the same condition repeatedly in groups with a small *N*. The use of a more highly inbred strain might reduce this variance.

The most basic question, perhaps, is why or how do these rats die? This question rephrased for the case of domestic rats reads, why does differential housing and handling postweaning result in widely differing rates of sudden death? In the case of wild rats, Richter suggested that it occurred ". . . as a result of hopelessness . . ." (p. 197). As stated his hypothesis is difficult to test for either wild or domestic strains and especially difficult to differentiate from the motor learning one previously discussed (cf. Experiment 3). Recently, however, there has developed an impressive empirical literature concern-

ing "learned helplessness" in animals and man that may provide for a more direct test (4). It is possible to expose a rat to a variety of situations that will result in his giving up in the face of aversive stimulation or conversely his being immunized from giving up. Taking animals so trained and exposing them to the water stress will allow for a much more direct assessment of whether helplessness is involved in sudden death. Recent work (6,14) has shown that this may be a promising approach, yet to explain the reported results for domestic rats, there will ultimately have to be some link established between differential handling and housing and helplessness.

A second approach has been suggested by Wolf (15–18). He has argued that some instances of sudden death in humans and the phenomenon in rats might be mediated by the diving or oxygen conserving reflex. This reflex results in a marked bradycardia and has been demonstrated in a wide variety of species including man. It can generally be induced by placing one's face under water. Our polygraphic recordings have confirmed existence of this reflex in our animals, and there can be little doubt that it must have some role in the sudden death of our domestic rats since they spend progressively more time under water prior to dying. Animals who survive the water stress spend less time under water and thus have this reflex elicited less. This leaves unanswered, however, the question of why some animals sink a lot, eliciting the bradycardia of the diving reflex, and others do not. It also begs the question of why rearing conditions are highly correlated with the ability to stay on the surface or not. Finally, it is unclear whether the cardiac slowing described above is causal in sudden death or a by-product of death by asphyxiation. Both the cardiac and behavioral data previously presented are similar to that of organisms that have been experimentally asphyxiated (19,20).[3] Further experimentation is necessary, however, to determine whether the death is the result of respiratory or cardiac factors and why these are highly correlated with differential rearing.

A third possible explanation stems from the stimulation in infancy literature. This literature may be interpreted as indicating that handling rodents in infancy reduces emotional reactivity and allows for more adaptive physiological stress responses (21,22). One would predict from this a lower incidence of death in handled groups. This is, of course, inconsistent with our results. Moreover, early stimulation results are reliable only for handling preweaning; handling or gentling postweaning has not yielded consistent effects (23).

A fourth explanation argues that living in a group exposes an animal to repeated problem-solving situations and will thus result in better problem solving ability and a greater chance of survival. Adding the corollary that handling impairs this survival independent of housing condition can account for the reported data (6). Unfortunately we know of no data supporting the idea that group housing helps problem-solving or do we know why handling should hurt. In addition, this kind of interpretation as well as many others is made difficult by pilot studies completed in our lab (24), which indicate that differential handling and housing is a generally significant manipulation with respect to sudden death in several strains of rats and mice; the direction of these results, however, is not always the same as reported

[3] I am indebted to one of the journal reviewers for this suggestion.

here in the Holtzman rat. These studies are now being replicated.

Finally, there is some evidence to suggest that various handling and housing procedures postweaning result in differential resting levels of certain hormones, including corticosterone (25,26). That adrenal activation is involved in our water stress situation seems highly likely; moreover, the 20-min "critical period" upon immersion seems consistent with hormonal mediation. This view, however, is highly speculative given the lack of experimental evidence.

It is still premature to attempt to generalize the results reported here to sudden death in humans. One is struck, however, by anecdotal reports indicating sudden death without evidence of drowning in people falling overboard at sea and those shipwrecked in addition to those in many other situations (4,27–31). The development of an adequate animal model of sudden death and its prevention may yield interesting hypotheses concerning the nature of sudden cardiac death, voodoo death, and related phenomena.

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SUMMARY

Sudden death in the domestic rat was found to parallel that in the wild rat with respect to HR response, the function of the whiskers, and the result of preexposure to the stressor. Sudden death does not seem necessarily related to the wildness of the animal, although possible differences between this type of death in wild and domestic rats were discussed. Psychological and physiological explanations of sudden death were briefly evaluated.

REFERENCES

1. Richter CP: On the phenomenon of sudden death in animals and man. Psychosom Med 19:191–198, 1957
2. Lynch JJ, Katcher AH: Human handling and sudden death in laboratory rats. J Nerv Ment Dis 159:362–365, 1974
3. Ewing LS: Fighting and death from stress in the cockroach. Science 155:1035–1036, 1967
4. Seligman MEP: Helplessness: On Depression, Development, and Death. San Francisco, Freeman, 1975
5. Von Holst D: Renal failure as the cause of death in Tupaia belangeri exposed to persistent social stress. J Comp Physiol 78:236–273
6. Rosellini RA, Binik YM, Seligman MEP: Sudden death in the laboratory rat. Psychosom Med 38 (No. 1):55–58, 1976
7. Mundl WJ: A cardio preamplifier for the free roving rat. Psychophysiology 2:75–78, 1965
8. Binik YM, Rosellini R: Unpublished data
9. Richter CP: The phenomenon of unexplained sudden death in animals and man, in WH Gantt (ed), Physiological Bases of Psychiatry. Springfield, Ill., Charles C Thomas, 1958
10. Vincent SM: The functions of the vibrissae in the behavior of the white rat. Behav Monogr 1(5):7–76
11. Gregoire S, Smith DE: Mouse-killing in the rat: effects of sensory deficits on attack behavior and stereotyped biting. Anim Behav 23(1):186–192, 1973

12. Kimble GA (ed): Hilgard and Marquis' Conditioning and Learning. New York, Appleton-Century-Crofts, 1961
13. Binik YM, Shustack B: Unpublished data
14. Corley KC, Mauck HP, Shiel FO'M: Cardiac responses associated with"yoked-chair" shock avoidance in squirrel monkeys. Psychophysiology 12 (No. 4):439–444, 1975
15. Wolf S: Bradycardia of dive reflex—possible mechanism of sudden death. Trans Am Clin Climatol Assoc 76:192, 1964
16. Wolf S: Sudden death and O_2 conserving reflex. Am Heart J 71:840, 1966
17. Wolf S: The end of the rope: the role of the brain in cardiac death. Can Med Assoc J 97:1022–1025,1967
18. Wolf S: The turned off heart. Med Times 69:132–146, 1968
19. Elsner R, Franklin DL, Van Citters RL, Kenney DW: Cardiovascular defense against asphyxia. Science 153:941–949, 1966
20. Hofer MA, Engel M, Weiner H: Development of cardiac rate regulation and activity after neonatal immunosympathectomy. Commun Behav Biol 6:59–62, 1971
21. Levine S: Infantile stimulation: a perspective, in A Ambrose (ed), Stimulation in Early Infancy. London, Academic Press, 1969
22. Daly Martin: Early stimulation of rodents: a critical review of present interpretations. Br J Psychol 64:435–460, 1973
23. Morton JRC: Effects of early experience "handling and gentling" in laboratory animals, in MW Fox (ed), Abnormal Behavior in Animals. Philadelphia, WB Saunders, 1968
24. Binik YM, Rosellini R, Dyer L: Unpublished data
25. Brown GM, Martin JB: Corticosterone, prolactin, and growth hormone responses to handling and new environment in the rat. Psychosom Med 36(3):241–247, 1974
26. Seggie JA, Brown GM: Stress response patterns of plasma corticosterone, prolactin, and growth hormone in the rat, following handling or exposure to novel environment. Can J Physiol Pharmacol 53(4):629–637, 1975
27. Wheaton JL: Fact and fantasy in sensory deprivation studies. Aeromedical Reviews (5-59). Air University School of Aviation Medicine, USAF. Brook AFB, Texas, pp. 13–24
28. Bettelheim B: The Informed Heart. New York, Avon Books, 1971
29. Canon WB: "Voodoo" death. Am Anthropol 44:169–181, 1942
30. Engel GL; Sudden and rapid death during psychological stress, folklore or folkwisdom? Ann Intern Med 74:771–782, 1971
31. Green WA, Goldstein S, Moss AJ: Psychosocial aspects of sudden death. Arch Intern Med 129:725–731, 1972