

Tonic Immobility in the Chicken: Catalepsy Potentiation by Uncontrollable Shock and Alleviation by Imipramine

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As compared to birds given the opportunity to control shock, inescapable aversive stimulation was shown to facilitate a cataleptic like state in chickens. In a second experiment imipramine was found to alleviate tonic immobility. It was suggested that the etiology of the cataleptic state in humans may relate to the maladaptive firing of a primitive predator defense.

INTRODUCTION

There is no widely accepted cause of catalepsy, a pathological state often associated with catatonia and characterized by a "hypnotic gaze" and waxy flexibility of the limbs. Nor are there many animal models of the human cataleptic state. The present report describes a possible model for catalepsy and the facilitative effects of a behavioral paradigm upon that model. The proposed model is tonic immobility, or the so-called state of animal hypnosis, which is a nonassociative response observed in fish, frogs, lizards, birds, rats, and rabbits (1). The hypnotic state is, like catalepsy in humans, characterized by a tonic immobility and waxy flexibility (see Fig. 1). The reaction is elicited by manual restraint and a variety of procedures known to produce directional effects upon fear (e.g., electric shock (2), conditioned fear and "safety" (3), adrenalin injections (4), tranquilizer ingestion (5), and simulated predation (6)) have been shown to affect the response. It is believed that the

behavior is unlearned because it occurs the first time the animal is manually restrained in the absence of any previous training. Although some believe that the reaction may represent the prototype of human hypnosis, others (7) hold that the response may represent the terminal reaction in a series of sequentially dependent predator defenses.

The behavioral paradigm which we found to enhance tonic immobility involves repeated exposure to inescapable aversive events. The procedure produces what has been called learned or conditioned helplessness (8). It has been postulated that helplessness is produced by un-signalled inescapable shock and is defined by interference with subsequent avoidance or escape learning. For example, Seligman and Maier (9) trained dogs to escape shock by pressing a head panel, while a yoked set of subjects received an equal shock density but were unable to escape. Twenty-four hours later those subjects which previously received escape training learned a shuttlebox avoidance task in 4-5 trials. However, the yoked subjects, whose prior training did not include experience with shock control, failed to respond on any of the 10 trials. Although no previous

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Fig. 1. Chicken in a state of animal hypnosis, catalepsy, or tonic immobility. Legs are rigid but display tremor and waxy flexibility. The chicken may actually be immobilized in any position, including standing, but we have found the lateral position preferable.

report has appeared for an avian species, this effect has been observed in a number of animals (8) including man (10).

EXPERIMENT 1

The present experiment was designed to assess the relative effects of escapable and inescapable electric shock upon an apparent state of catalepsy, tonic immobility.

Method

Subjects. Thirty-six experimentally naive Production Red chickens (*Gallus gallus*) participated in

the experiment. They were acquired from a local hatchery at one day of age and were group-reared in commercial brooders. Food (Purina Chick Chow) and water were always available during rearing under conditions of a 14-hr photoperiod.

Conditioning. At 10 days of age, the chicks were randomly assigned to either shock-escape, shock-inescape, or no-shock conditions. Each shock-escape subject was permanently yoked to a subject which could not escape by placing each member of a pair of birds in one of two identical shuttleboxes connected to the same shock source. A film programmer scheduled the onset of 15 trials, presented at varying intervals (the mean time of which was 90 sec) given each day for five days. Electromechanical equipment controlled shock presentation and recorded response latencies. The third group was treated the same as the other two groups, but they were never shocked.

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Shock intensity was 3.5 mA, generated by an AC shock source (Lafayette Model No. A-615AR).

In the two shock conditions shock onset was simultaneous, but after two seconds, the experimenter withdrew a partition separating the two chambers of one shuttlebox and 90 sec was allowed for the subject to escape to the opposite side thereby triggering a floor switch which terminated shock to both animals. If the bird failed to execute the shuttle response in 90 sec, he was gently prodded to the opposite side. In both groups, shock onset was unsignalled and uncontrollable. Controllability only became a factor for the escapable groups 2 sec after shock onset.

Testing. Twenty-four hours following the 75th trial, the animals were individually returned to the experimental room and tested for duration of tonic immobility. The reaction was induced by placing each subject in the shuttle apparatus and applying lateral manual restraint for 15 sec. If the subject failed to become immobile on the first attempt he was given up to five inductions. The duration of tonic immobility was measured from the time restraint ended until the subject rose to his feet.

RESULTS

The escape acquisition means are provided in Table 1. A repeated measures analysis of variance revealed a significant trials effect ($F = 2.437$, $df = 74,814$, $p < 0.001$), indicating that the shock-escape animals had learned the task and therefore qualified as having learned to control, to the extent possible, their aversive environment.

The effects of the different shock condi-

TABLE 1. Mean Shuttle-Response Times in Seconds Averaged Over Blocks of 15 Trials in a Two-Way Escape Learning Task

Trials	Mean Latency	Standard Deviation
1-15	23.23	25.33
16-30	23.37	30.56
31-45	11.11	17.73
46-60	9.02	18.35
61-75	5.73	10.01

tions upon tonic immobility are seen in Table 2. Subjects exposed to conditions designed to produce learned helplessness remained immobile over 4.5 times longer than chickens allowed to control shock duration. Unshocked subjects showed a mean duration of immobility of only 17.33 sec. Using an analysis of variance, the difference in immobility duration between groups was statistically reliable ($F = 7.32$, $df = 2, 33$, $p < 0.005$). Duncan's post hoc test for multiple comparisons revealed that subjects receiving inescapable shock were immobile significantly longer than those with a prior history of escapable shock ($p < 0.01$).

TABLE 2 Mean Durations of Tonic Immobility in Seconds for each Treatment Condition

Treatment	Mean Duration	Standard Deviation
Inescapable Shock	794.25	849.70
Escapable Shock	173.58	326.31
No Shock	17.33	35.71

EXPERIMENT 2

In order to further examine the claim that the tonic immobility response is an appropriate model for catalepsy, an attempt was made to manipulate immobility with a drug reported to be efficacious in the treatment of the pathological state in humans. Zarcone (11) has recently described imipramine as the drug of preference for a constellation of cataleptic like symptoms. Imipramine has been shown to be effective in the treatment of cataplexy, but not narcolepsy (12). Fekete and Kurti (13) inhibited the cataleptic state induced by chlorpromazine and tetrabenazine by injections of 8 and 12 mg/kg of imipra-

mine, respectively. Unpublished observations in our laboratory indicate that low doses of chlorpromazine and tetrabenazine also enhance the duration of tonic immobility. If administration of this tricyclic antidepressant were to reduce the duration of tonic immobility, the applicability of the model would be supported.

Method

Subjects. Forty experimentally naive Production Red chickens participated. They were acquired and maintained as described in Experiment 1. On the day of the experiment they were approximately 3 weeks of age and weighed an average of 340 g, with a standard deviation of 41.4 g.

Procedure. It was decided to administer a constant concentration of imipramine to all subjects in each group and weigh them after the behavioral test. This decision was made because handling is known to affect tonic immobility (14). The birds were randomly assigned to 4 groups of 10 birds each. All injections were administered into the right thigh muscle.

Ten birds received an average dose of 8.8 mg/kg and ten others 17.4 mg/kg. A control group received 0.5 cc of distilled water. Following injection these subjects were taken to individual testing rooms, where, 10 min later, they were tested for duration of tonic immobility as described in Experiment 1. A fourth group was given an average dose of 17.4 mg/kg, but it was not tested until 60 min postinjection.

Results

Figure 2 indicates that both levels of imipramine, relative to the control subjects, produced a reduction in the duration of tonic immobility in the birds tested 10 min after injection. Analysis of variance was performed on these three groups and was found to be significant ($F = 4.23$, $df = 2/37$, $p < 0.05$). One hour later, however, immobility had returned to the level of the control animals. Analysis of variance revealed that the 17.4 mg/kg group tested at 10 min and the 1-hr group were significantly different ($F = 9.60$, $df = 2.37$, $p < 0.01$).

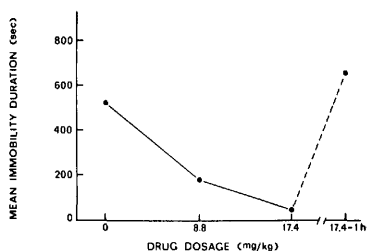


Fig. 2. Effects on duration of tonic immobility of 8.8 and 17.4 mg/kg doses of imipramine 10 min following intramuscular injection. The effect of a 17.4 mg/kg dose is also shown 1 hr after injection.

It is interesting that almost immediately following the imipramine injection, the chickens became extremely ataxic and assumed awkward, almost flacid postures. However, rather than prolong the immobility response, the ataxia was associated with a reduction in tonic immobility duration.

DISCUSSION

Inescapable shock significantly enhanced tonic immobility, while imipramine acted to inhibit the behavior. These data have several implications. There have been a number of suggestions that catalepsy and tonic immobility may be identical, but these have been based on noting mere superficial similarities between the two states. The imipramine data coupled with the suggestion that similar response contingencies may operate in the facilitation of these behaviors bolsters the analogy (1,15). Seligman (16) has discussed conditioned helplessness relative to depression and phobias but not catalepsy. Recent reports of drug-induced catalepsy have ap-

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peared for antibrain antibodies (17), for 6-hydroxydopamine (18), and for monkeys suffering from long-term social isolation (19).

The present findings suggest that procedures designed to bring about conditioned helplessness increase the probability that catalepsy will eventuate. Indeed, manual restraint qualifies as an operation producing helplessness in its own right since while restrained any attempts to escape are ineffectual. The condition of helplessness could conceivably develop when an organism learns that all of its behaviors are ineffectual by way of coping with his environment, and learning may be a major factor in the etiology of catalepsy. Behavioral therapy for conditioned helplessness exists (8,16) and may decrease the likelihood of catalepsy in some pathologies.

Alternatively it might be argued that subjects receiving escape training were taught to run in the shuttlebox, and therefore on the test trial show briefer immobility times because of competing conditioned tendencies to escape. Contrary to this contention, however, it is important to note that during training shock was the discriminative stimulus for running (escape) and no shock was given on the test trial. At no time did subjects in the escapable group show evidence of running in the shuttlebox prior to the onset of shock. Similarly, the yoked inescapable birds also engaged in considerable motoric activity during shock, which according to a competing response interpretation should produce considerable if not equivalent conditioned interference on the test trial, yet they remained immobile much longer than their escapable counterparts.

A second implication relates to the literature which describes inescapable shock as more aversive, or more aptly, more "distressing" (8, p. 360) than escapable

shock (20). Tonic immobility has been shown to be potentiated by stress-producing situations which generate fear reactions (2,3). The fact that birds which were never shocked remained immobile for an average of only 17.33 sec testifies to the considerable influence of aversive stimulation, even when controllable. The important comparison is, however, between the animals experiencing equal amounts of escapable or inescapable shock. If inescapable shock is more distressing than escapable shock, then based on previous findings this ought to be reflected in an increased duration of tonic immobility. This effect is clearly seen in the mean duration of the inescapable groups and the trend toward reduced distress in the shock-escape group.

Learned helplessness represents one possible interpretation of these data. An alternative but related interpretation is motivational. Desiderato and Neuman (21) have shown that a stimulus paired with inescapable shock is more fear producing than the same stimulus associated with escapable shock. Since conditioned fear (stress reaction) is known to potentiate tonic immobility (2), the situational cues of the shuttlebox could function as conditioned stimuli for fear (22), and animals given inescapable shock should remain immobile longer than those receiving escapable shock. Thus, the enhanced tonic immobility reactions might be a reflection of heightened fear caused by learning that shock is inescapable. On the other hand, preliminary data in our laboratory suggest that when chickens are trained in the same situation as described here but tested for tonic immobility in a markedly different environmental context, a history of inescapable shock still results in longer durations than in animals receiving escapable shock (\bar{X} s = 912.7 sec and 389.5 sec res-

pectively). The effect of uncontrollable shock on tonic immobility may therefore be independent of situational cues, but this conclusion awaits further verification.

A third implication of these data relate to the effects of inescapable aversive events. Rather than producing negative interference by way of retarding the elaboration of a new behavior, the effect of these procedures was to facilitate the cataleptic state. Inescapable shock effects have not previously been demonstrated in avian species nor has positive transfer been found to an innate behavior.

A final implication concerns the importance of an ethological orientation to behavioral pathology. Considerable evidence exists (6,7) in support of the notion that under natural conditions tonic immobility serves in the capacity of a predator defense. We feel that there may be reason to believe that so-called catalepsy in humans may represent the behavioral manifestation of a primitive predator defense which may misfire under conditions of exaggerated stress, e.g., when coping behaviors fail to control a hostile, aversive environment.

SUMMARY

Thirty-six chickens were given 75 escapable or inescapable shock trials in a shut-

tlebox. Twenty-four hours after the last trial (Day 6) the animals were placed in the shuttlebox and manually restrained. Those subjects receiving inescapable shock remained in a cataleptic-like state (tonic immobility) more than 4.5 times longer than the subjects which received escapable shock. A second study demonstrated that imipramine, a compound known to alleviate catalepsy in humans, dramatically reduces the duration of tonic immobility in chickens.

These data suggest that catalepsy develops when a stressed organism learns that his coping behaviors are ineffectual. Given the large number of species exhibiting tonic immobility, it is further suggested that in man the reaction becomes manifest when extreme stress precipitates "catalepsy." In our view, the development of catalepsy results from exposure to uncontrollable aversive events, whereas the origin of catalepsy relates to conditions which were in effect during the evolution of our species.

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