Pharmacology Biochemistry & Behavior, Vol. 24, pp. 1623–1627, 1986. © Ankho International Inc. Printed in the U.S.A. 0091-3057/86 $3.00 + .00

Central and Peripheral Effects of Serotonin on the Immobility Response in Chickens[1]

CHARLES W. HENNIG, WILLIAM C. STEINHOFF AND JEANNE V. BOOTH

Psychology Department, Salem College, Salem, WV 26426

Received 8 August 1985

HENNIG, C. W., W. C. STEINHOFF AND J. V. BOOTH. *Central and peripheral effects of serotonin on the immobility response in chickens.* PHARMACOL BIOCHEM BEHAV 24(6) 1623–1627, 1986.—The effects of low and high doses of serotonin on tonic immobility (TI) duration and susceptibility in 10- and 45-day-old chickens were examined. High doses of serotonin reduced the number of inductions required to produce TI, regardless of the subject's age. In contrast, low and high doses of serotonin produced biphasic increases and decreases in TI duration in 10-day-old birds, but there were no apparent effects on immobility duration by either dose of this drug in older chickens. These results are discussed in terms of the formation of the blood-brain barrier in domestic fowl and the differential peripheral versus central actions by serotonin on TI susceptibility and response duration.

Tonic immobility Animal hypnosis Biphasic effects Central effects Peripheral effects Serotonin Chickens

TONIC immobility (TI), or animal hypnosis, is a catatonic-like state of extreme behavioral inhibition that is elicited in many animals by a brief period of physical restraint. After an initial struggle, animals assume a frozen posture which persists in the absence of further restraint [10]. Tonic immobility occurs in a wide variety of species ranging from invertebrates [36] to primates [20]. However, the duration of TI can be highly variable, even within species, and may last from a few seconds to several hours. Domestic fowl are often used in the study of TI because of the ease with which the response can be induced and quantified in such subjects [10,13].

Under natural conditions, tonic immobility appears to function as a terminal defensive response to predation [35,37] and has been shown to be especially sensitive to experimental manipulations designed to affect fear [11]. Similarities have also been noted between TI and forms of behavioral inhibition found in humans such as catalepsy, catatonic schizophrenia, and rape-induced paralysis [12,41].

A number of studies have examined the psychopharmacology of the TI response, especially the identification of critical neurotransmitters underlying this behavior, and they have shown the involvement of cholinergic [28, 42, 47], adrenergic [22, 24, 25], dopaminergic [8, 27, 45], and serotonergic [3, 14, 18, 21, 26, 33, 44, 46] systems with the duration of immobility episodes. However, the strongest evidence seems to favor serotonin (5-hydroxytryptamine, 5-HT) as the most important neurochemical mediator for tonic immobility. Prolonged durations of immobility during repeated testing for TI produced decreases in brain levels of 5-HT in guinea pigs [31] and rabbits [9]; whereas dietary deprivation of tryptophan, a 5-HT precursor, or injections of very large doses of p-chlorophenylalanine, a relatively specific depletor of 5-HT, significantly reduced TI durations in chickens [14,29]. In addition, numerous studies have shown that TI duration can be potentiated by drugs thought to increase the synaptic concentrations of serotonin via such processes as precursor loading with tryptophan, monoamine oxidase inhibition (pargyline, iproniazid), reuptake blockade by intraventricular administration of imipramine, and injections of 5-HT receptor agonists (d-LSD, BOL-148) and metabolites (melatonin, 5-hydroxyindole acetic acid) [14, 18, 26, 33]. Moreover, both intraventricular and intravenous administration of low doses of 5-HT increased TI durations in chickens [18,21]. Therefore, a certain level of serotonin seems critical to the occurrence of the immobility response.

Wallnau and Gallup [46] proposed a serotonergic, midbrain-raphe model of tonic immobility in which TI duration was inversely related to the rate of firing by 5-HT neurons in the raphe area. However, recent work has suggested a revision of this model, with a shift in emphasis to the postsynaptic consequences of 5-HT manipulations [3, 43, 44]. Enhancement of 5-HT release (fenfluramine, combined administration of tryptophan and pargyline, amphetamine [38], p-chloroamphetamine [48]), and injections of the 5-HT agonist quipazine both greatly increase serotonin levels and also reduce TI duration [2–4, 43, 44]. In addition, intraventricular administration in rabbits and intravenous injections in chickens of high doses of 5-HT decreased TI duration in both species [19,33]. These results were very similar to those

[1]Portions of this paper were presented at the meeting of the West Virginia Academy of Science in Fairmont, WV, in April 1985.

found after dramatic increases in synaptic serotonin or intense stimulation of postsynaptic 5-HT receptors in rats which produced a serotonergic behavioral syndrome consisting of hyperactivity, tremor, rigidity, head weaving, and hindlimb abduction [16,30]. Therefore, the end result of 5-HT manipulations on inhibitory motor behavior such as TI seems to depend on the level of 5-HT involved. Small increases in 5-HT appear to increase TI duration, while larger doses attenuate the response. This interpretation is supported by the finding that low doses of 5-HT in chickens increased TI duration, but high doses decreased the duration of the repsonse [21].

Injections of 5-hydroxytryptophan, serotonin, or melatonin also increased susceptibility to TI in chickens and anoles by reducing the number of inductions required to elicit the response [21, 23, 26]. In contrast, p-chloroamphetamine, amphetamine, fenfluramine and quipazine all decreased susceptibility by increasing the number of inductions needed to produce TI in chickens [2, 4, 43, 44]. These findings further support the involvement of serotonin with the immobility response, but the exact nature of the relationship between 5-HT and TI is as yet unclear.

One of the unanswered questions about this relationship deals with the extent to which serotonergic effects on tonic immobility reflect peripheral versus central drug actions. Past research has shown that peripheral injections of 5-HT in young chickens produced a decrease in duration of immobility [21,33], whereas they had no effect on TI duration in rabbits [19] or anoles [23]. On the other hand, central administration of 5-HT in chickens produced an increase in TI duration [18], but a similar procedure attenuated duration of immobility in rabbits [19]. These discrepant findings may reflect differences in peripheral and central effects of 5-HT on tonic immobility, since serotonin does not readily cross the blood-brain barrier in many species of animals [1, 5, 7]. In order to test for this possibility, very young and somewhat older chickens were chosen as subjects. The blood-brain barrier in chickens only attains adult-like status after about 3 to 4 weeks of age [40]. Thus, any peripheral effect by 5-HT on TI should be apparent in all the experimental birds, whereas central effects should only be found in the younger chickens whose blood-brain barrier has not yet fully formed.

METHOD

Subjects

The subjects were 54 straight run Production Red chickens *(Gallus gallus)* obtained from Welp, Inc. (Bancroft, IA) at one day posthatch. They were group-reared in commercial brooders under normal daylight (approximately 10 hr light/14 hr dark). Water and food (Purina chick starter) were continually available. The drug treatments and testing occurred when the birds were either 10 or 45 days old.

Drugs

Serotonin creatinine sulfate (Sigma) was dissolved in distilled water and injected intraperitoneally (IP) into experimental subjects in volumes of 1 ml/kg body weight. Control animals received equivalent volumes of distilled water. The low (0.5 mg/kg) and high (15.0 mg/kg) doses of serotonin used in this experiment were chosen because they were similar to doses previously employed to produce biphasic effects on TI duration in chickens [21]. All the drug solutions were made fresh on the day of testing and coded prior to using.

Procedure

The experiment was divided into two parts. In the first part, 27 chicks were individually removed from their brooder at 10 days posthatch and were randomly assigned to one of three groups (n=9 chickens per group). These birds were weighed and given IP injections of either distilled water, 0.5 mg/kg of serotonin or 15.0 mg/kg of serotonin. In the second part, another 27 chicks were injected in the same manner at 45 days of age.

Immediately after injection, each bird was placed in a cardboard box and transported to a testing room. Ten minutes after the injection, the subject was removed from the box, placed on a flat table, and quickly inverted on its right side. Gentle restraint was maintained by the experimenter with both hands for 15 sec. Then the hands were withdrawn and a standard stopwatch was activated. Any subject failing to remain immobile for at least 5 sec was given up to five successive 15-sec inductions in an attempt to elicit tonic immobility, with a 30-sec intertrial interval between each attempt. The number of inductions required to meet this criterion was recorded. If the subject did not show immobility for any of the five attempted inductions, then a duration score of zero was recorded. For those birds that did become immobile, the duration of TI was measured from the time of release until either the chicken showed a spontaneous righting response and returned to its feet, or a maximum duration of 3600 sec elapsed. Then the subject was returned to a brooder maintained for used animals. Testing was performed by experimenters who were unaware of the drug treatments that each bird received. To preclude any confounding effects of periodicity, testing was staggered over the day (1000 hr to 1600 hr) with a comparable number of birds from each group tested at different times throughout the day.

Statistics

Two-way factorial analyses of variance involving two levels of age (10 and 45 days posthatch) and three drug conditions (water, 0.5 mg/kg serotonin, and 15.0 mg/kg serotonin) were used to analyze TI susceptibility and duration data. Analyses of simple effects and orthogonal comparisons were used to test the significance of any differences between age levels and individual drug groups. Due to extreme skewness and variability in the TI duration data, a square-root transformation was performed on all duration scores prior to statistical analysis.

RESULTS

Susceptibility to TI, as measured by the number of inductions required to produce the immobility response, increased after injections of serotonin. As shown in Table 1, serotonin decreased the mean number of inductions needed to elicit the response in all the experimental groups, regardless of the subject's age. This was supported by a significant main effect of drug condition, $F(2,48)=6.68$, $p<0.01$, and no significant differences in susceptibility due to age. Subsequent orthogonal contrasts revealed that the pooled mean for the water and 0.5 mg/kg serotonin groups was significantly higher than the mean number of inductions required for the 15.0 mg/kg serotonin group, $F(1,48)=11.82$, $p<0.01$, but that the two former groups did not differ from each other.

Mean TI durations for each treatment and age group are shown in Fig. 1. As can be seen, low doses of serotonin potentiated TI duration in 10-day-old chicks, whereas large

TABLE 1

MEAN NUMBER OF INDUCTIONS REQUIRED TO PRODUCE IMMOBILITY IN 10- OR 45-DAY-OLD CHICKENS AFTER INJECTIONS OF LOW OR HIGH DOSES OF SEROTONIN (5-HT)

Drug Group*	Age of Subjects	
	10 Days	45 Days
Water	2.67	2.56
0.5 mg/kg 5-HT	1.89	2.33
15.0 mg/kg 5-HT	1.22	1.11

Maximum number of inductions=5.
*n=18 for each group, with 9 subjects per cell.

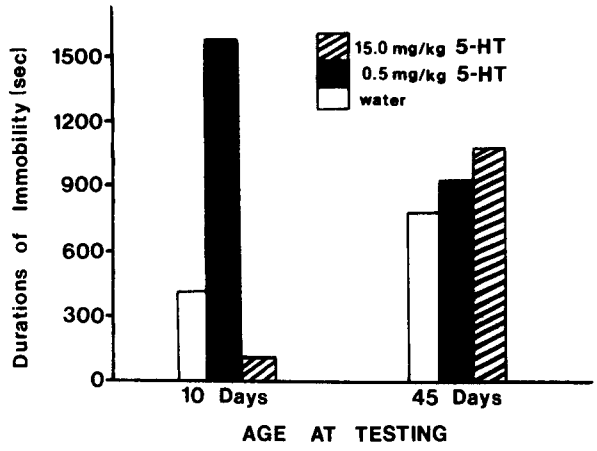

FIG. 1. Mean durations of tonic immobility (sec) in 10- or 45-day-old chickens after injections of low or high doses of serotonin (5-HT).

doses of this drug attenuated duration of the response. In contrast, neither dose of 5-HT produced much of a change in the duration of TI in older birds. These findings are supported by a significant main effect of drug condition, $F(2,48)=3.67$, $p<0.05$, and a significant age × drug interaction, $F(2,48)=4.98$, $p<0.025$. Analysis of simple effects for each age level revealed a significant difference between drug conditions in 10-day-old birds, $F(2,48)=8.03$, $p<0.001$, but found no such difference in 45-day-old chickens. Subsequent orthogonal comparisons on the data from the younger birds revealed that the pooled mean duration for the water and 0.5 mg/kg serotonin groups was significantly greater than the mean of the 15.0 mg/kg serotonin group, $F(1,48)=7.60$, $p<0.01$, while the mean duration of the low dosage serotonin group was significantly higher than that of the water control group, $F(1,48)=8.46$, $p<0.01$. Durations of tonic immobility were somewhat longer in 45-day-old chickens than in the younger birds, but this difference was not significant.

DISCUSSION

The present study demonstrated that serotonin affected both susceptibility to TI and duration of the immobility response in chickens, but the 5-HT seemed to act independently on each of these two measures of immobilization. Serotonin reduced the number of inductions required to elicit tonic immobility. This finding replicated previous results [21]. A new development was that serotonin produced the same effect on TI susceptibility in both 10- and 45-day-old chicks, despite the fact that the blood-brain barrier has been shown to restrict the access of 5-HT to the central nervous system in chickens older than 3 to 4 weeks of age [40]. This latter finding suggests that any serotonergic effect on immobility responses in the 45-day-old birds would probably be peripheral. Therefore, the increase in TI susceptibility in the present study, which seems to be similar in chickens of either age, is most likely due to peripheral actions of serotonin. In addition, such an explanation would also be consistent with past research which showed that peripheral injections of 5-HT increased TI susceptibility in anoles [23], even though this drug should not penetrate their blood-brain barrier [39].

Serotonin also produced changes in the duration of tonic immobility in chickens. The present study found that low doses of 5-HT potentiated TI duration, while high doses attenuated the response. However, serotonin only affected TI duration in the 10-day-old chicks. This drug had no apparent influence on duration of immobility in the older birds, probably because not enough 5-HT could penetrate the blood-brain barrier to have a central effect on their behavior. This possibility suggests that serotonin's biphasic effect on TI duration is most likely due to the central action of serotonin.

The present findings replicated previous research with TI duration in young chickens that were given peripheral injections of various dosages of serotonin [21], but went one step further. They also helped to explain some past inconsistencies in the effects of serotonin on TI duration when the test species, routes of drug administration, and drug dosages differed across studies. For example, intraventricular administration of low doses of 5-HT potentiated TI duration in chickens [18], whereas central administration of high doses of 5-HT in rabbits decreased immobility durations [19]. These results can be explained by findings in the present study which suggest that biphasic effects on TI duration are produced by central actions of low and high doses of 5-HT in subjects. Peripheral injections of high doses of 5-HT in young chicks also attenuated TI duration [33], but similar injections of 5-HT in adult rabbits and anoles had no apparent effect on duration of immobility [19,23]. The former result can be explained by the fact that the blood-brain barrier in chickens is not formed completely until 3 or 4 weeks of age, so that large doses of 5-HT can still reach the central nervous system in young birds [40] and attenuate TI duration. In contrast, the lack of effect by large doses of 5-HT on immobility duration in anoles and rabbits can be explained by the fact that peripheral serotonin should not be able to penetrate the blood-brain barrier and reach the central nervous system in these adult animals [7,39]. The findings of the present study suggest that TI duration should only be influenced by central actions of serotonin.

Although the involvement of central serotonergic action alone was not evaluated in the current experiment, the findings presented in this study support the inference that serotonin's biphasic effect on TI duration acts through some central control mechanism since peripheral serotonin alone had no effect on immobility duration. However, none of these results explain the cause of the differential duration effects with low and high doses of serotonin. Wallnau and Gallup [46] suggested that TI duration depended on the ex-

tent to which 5-HT levels inhibited midbrain-raphe serotonergic neurons, but this hypothesis could not explain why some 5-HT drugs attenuated TI duration [2–4, 43, 44], while others potentiated durations of immobility [14, 18, 21, 26, 33]. This led to a revision of the previous hypothesis. It was suggested that TI duration was controlled by the postsynaptic consequences of serotonin manipulations such as receptor stimulation [3,44]. However, this alternative does not explain how low doses of 5-HT increase TI duration, whereas high doses attenuate the response. One possible explanation for these biphasic effects is that there are two types of serotonin receptors, which react in opposite ways to this neurotransmitter. Evidence for two different types of serotonin receptors was reported by Peroutka and Snyder [34]. They labeled these receptors as 5-HT_1 and 5-HT_2. The 5-HT_1 receptors seem inhibitory in nature and are highly sensitive to even small amounts of serotonin, whereas the 5-HT_2 receptors act in an excitatory manner and are stimulated only by very large amounts of serotonin. Low doses of 5-HT may act on the former type of receptor to inhibit motor behavior and prolong TI duration, while large doses of 5-HT may act on the latter form of receptor to excite motor responses and attenuate duration of immobility. Such a dichotomy may explain the differential effects of serotonergic drugs on TI duration [3, 21, 44] and may even permit us to better understand some of the more complex interactions between different neurochemical systems that influence TI duration. Adrenergic influences on response duration [22, 24, 25] may also work through 5-HT_2 receptors, since these receptors have been shown to excite adrenergic neurons in the reticular formation [17]. In addition, dopaminergic antagonists such as spiroperidol and haloperidol are known to affect both TI duration [27,45] and 5-HT_2 receptors [34].

The present study demonstrated that serotonin affected both TI susceptibility and duration. However, susceptibility appeared to depend on the peripheral action of serotonin, whereas the duration of immobility seemed to be influenced by the type of postsynaptic 5-HT receptor that was stimulated in the central nervous system. In addition, there is some evidence that behaviors other than TI are also differentially affected by various dosages of serotonin. Low doses of 5-HT are known to facilitate sleep and motor inhibition, whereas high doses of 5-HT can cause arousal and other forms of excitation such as the serotonin behavioral syndrome [6, 15, 30, 32]. These similarities should permit some interesting generalizations about possible biphasic effects by serotonin on other behaviors that are also influenced by the same neurochemical system.

REFERENCES

1. Axelrod, J. and J. K. Inscoe. The uptake and binding of circulating serotonin and the effect of drugs. *J Pharmacol Exp Ther* **141:** 161–165, 1963.
2. Boren, J. L. and G. G. Gallup, Jr. Amphetamine attenuation of tonic immobility in chickens. *Physiol Psychol* **4:** 429–432, 1976.
3. Boren, J. L., G. G. Gallup, Jr., S. D. Suarez, L. B. Wallnau and G. J. Gagliardi. Pargyline and tryptophan enhancement of tonic immobility: Paradoxical attenuation with combined administration. *Pharmacol Biochem Behav* **11:** 17–22, 1979.
4. Boren, J. L., S. D. Suarez and G. G. Gallup, Jr. p-Chloroamphetamine: Effects on tonic immobility, activity, and temperature in chickens. *J Comp Physiol Psychol* **95:** 991–1002, 1981.
5. Cserr, H. F. and M. Bundgaard. Blood-brain interfaces in vertebrates: A comparative approach. *Am J Physiol* **246:** R277–R288, 1984.
6. Dewhurst, W. G. and E. Morley. Action of sympathomimetic and allied amines on the central nervous system of the chicken. *Br J Pharmacol Chemother* **25:** 705–727, 1965.
7. Erspamer, V. Recent research in the field of 5-hydroxytryptamine and related indolealkylamines. *Prog Drug Res* **3:** 290, 1961.
8. Ettinger, R. H. and R. W. Thompson. The role of dopaminergic systems in the mediation of tonic immobility (animal hypnosis) in chickens. *Bull Psychon Soc* **12:** 301–302, 1978.
9. Farabollini, F., G. Carli and C. Lupo. Changes in brain serotonin and 5-hydroxy-indolacetic acid levels in the rabbit following tonic immobility. *Physiol Behav* **32:** 205–209, 1984.
10. Gallup, G. G., Jr. Animal hypnosis: Factual status of a fictional concept. *Psychol Bull* **81:** 836–853, 1974.
11. Gallup, G. G., Jr. Tonic immobility: The role of fear and predation. *Psychol Rec* **27:** 41–61, 1977.
12. Gallup, G. G., Jr. and J. D. Maser. Tonic immobility: Evolutionary underpinnings of human catalepsy. In: *Psychopathology: Experimental Models*, edited by J. D. Maser and M. E. P. Seligman. San Francisco: Freeman, 1977, pp. 334–357.
13. Gallup, G. G., Jr., R. F. Nash and A. M. Wagner. The tonic immobility reaction in chickens: Response characteristics and methodology. *Behav Res Methods Instrum* **3:** 237–239, 1971.
14. Gallup, G. G., Jr., L. B. Wallnau, J. L. Boren, G. J. Gagliardi, J. D. Maser and P. H. Edson. Tryptophan and tonic immobility in chickens: Effects of dietary and systemic manipulations. *J Comp Physiol Psychol* **91:** 642–648, 1977.
15. Gerson, S. C. and R. J. Baldessarini. Minireview: Motor effects of serotonin in the central nervous system. *Life Sci* **27:** 1435–1451, 1980.
16. Grahame-Smith, D. G. Studies *in vivo* on the relationship between brain tryptophan, brain 5-HT synthesis and hyperactivity in rats treated with a monoamine oxidase inhibitor and L-tryptophan. *J Neurochem* **18:** 1053–1066, 1971.
17. Haigler, H. J. and G. K. Aghajanian. Peripheral serotonin antagonists: Failure to antagonize serotonin in brain areas receiving a prominent serotonergic input. *J Neural Transm* **35:** 257–273, 1974.
18. Harston, C. T., D. H. Sibley, G. G. Gallup, Jr. and L. B. Wallnau. Effects of intraventricular injections of imipramine and 5-hydroxytryptamine on tonic immobility in chickens. *Bull Psychon Soc* **8:** 403–405, 1976.
19. Hatton, D. C., R. Tankle, T. Lanthorn and M. E. Meyer. Serotonin and tonic immobility in the rabbit. *Behav Biol* **24:** 97–100, 1978.
20. Hennig, C. W. Tonic immobility in the squirrel monkey *(Saimiri sciureus). Primates* **19:** 333–342, 1978.
21. Hennig, C. W. Biphasic effects of serotonin on tonic immobility in domestic fowl. *Pharmacol Biochem Behav* **12:** 519–523, 1980.
22. Hennig, C. W., E. B. Carl, S. Aldrich, J. K. Fazio and C. A. Hughes. Differential effects of alpha-adrenergic antagonists on tonic immobility in domestic fowl. *Pharmacol Biochem Behav* **15:** 739–742, 1981.
23. Hennig, C. W., W. P. Dunlap and C. T. Hartson. Tonic immobility and skin color in anoles: Effects of serotonin precursors and metabolites. *Physiol Behav* **22:** 1079–1088, 1979.
24. Hennig, C. W., W. P. Dunlap, C. T. Harston and A. A. MacPhee. Tonic immobility and the alpha-adrenergic system in chickens. *Physiol Behav* **24:** 21–25, 1980.
25. Hennig, C. W., J. K. Fazio, C. A. Hughes, W. R. Castaldi and B. D. Spencer. Duration of tonic immobility in chickens as a function of alpha-adrenergic receptor stimulation and blockade. *Pharmacol Biochem Behav* **20:** 731–738, 1984.

26. Hennig, C. W., C. T. Harston, W. P. Dunlap, E. M. Hill and M. D. McNichols. The effects of serotonin metabolites on tonic immobility in chickens *(Gallus gallus)*: Dose and time response curves. *Physiol Psychol* **8:** 65–71, 1980.
27. Hennig, C. W. and B. D. Spencer. The effect of spiroperidol on tonic immobility in domestic fowl. *Bull Psychon Soc* **21:** 317–320, 1983.
28. Hughes, R. A. Anticholinergic drugs, blood-brain-barrier and tonic immobility in chickens. *Physiol Behav* **29:** 67–71, 1982.
29. Hughes, R. A. P-Chlorophenylalanine attenuates tonic immobility duration in chickens. *Physiol Behav* **36:** 427–430, 1986.
30. Jacobs, B. L. Minireview: An animal model for studying central serotonergic synapses. *Life Sci* **19:** 777–786, 1976.
31. Liberson, W. T., J. Bernsohn, A. Wilson and V. Daly. Brain serotonin content and behavioral stress. *J Neuropsychiatry* **5:** 363–365, 1964.
32. Lloyd, O. L. Inhibition of median raphe neurone metabolism by cerebrospinal fluid (CSF) containing 5-hydroxytryptamine and melatonin. *Biochem Pharmacol* **23:** 1913–1915, 1974.
33. Maser, J. D., G. G. Gallup, Jr. and L. E. Hicks. Tonic immobility in chickens: Possible involvement of monoamines. *J Comp Physiol Psychol* **89:** 319–328, 1975.
34. Peroutka, S. J. and S. H. Snyder. Multiple serotonin receptors: Differential binding of [^{3}H]5-hydroxytryptamine, [^{3}H]lysergic acid diethylamide and [^{3}H]spiroperidol. *Mol Pharmacol* **16:** 687–699, 1979.
35. Ratner, S. C. Comparative aspects of hypnosis. In: *Handbook of Clinical and Experimental Hypnosis*, edited by J. E. Gordon. New York: Macmillan, 1967, pp. 550–587.
36. Ratner, S. C. Immobility of invertebrates: What can we learn? *Psychol Rec* **27:** 1–13, 1977.
37. Sargeant, A. B. and L. E. Eberhardt. Death feigning by ducks in response to predation by red foxes *(Vulpes fulva)*. *Am Midl Nat* **94:** 108–119, 1975.
38. Schrold, J. and R. F. Squires. Behavioural effects of d-amphetamine in young chicks treated with p-Cl-phenylalanine. *Psychopharmacologia* **20:** 85–90, 1971.
39. Shivers, R. R. The blood-brain barrier of a reptile, *Anolis carolinensis*. A freeze-fracture study. *Brain Res* **169:** 221–230, 1979.
40. Spooner, C. E. and W. D. Winters. Evidence for a direct action of monoamines on the chick central nervous system. *Experientia* **21:** 256–258, 1965.
41. Suarez, S. D. and G. G. Gallup, Jr. Tonic immobility as a response to rape in humans: A theoretical note. *Psychol Rec* **29:** 315–320, 1979.
42. Thompson, R. W., J. Piroch, D. Fallen and D. Hatton. A central cholinergic inhibitory system as a basis for tonic immobility (animal hypnosis) in chickens. *J Comp Physiol Psychol* **87:** 507–512, 1974.
43. Wallnau, L. B. Tonic immobility in domestic fowl: Anticataleptic effects of quipazine. *Pharmacol Biochem Behav* **12:** 347–352, 1980.
44. Wallnau, L. B., G. D. Bordash and P. Corso, Jr. The effects of tryptophan and manipulations of serotonergic receptors on tonic immobility in chickens. *Pharmacol Biochem Behav* **14:** 463–468, 1981.
45. Wallnau, L. B., C. L. Carnrike, Jr. and G. I. Dewey. Tonic immobility: Effects of dopamine receptor blockade and stimulation. *Pharmacol Biochem Behav* **10:** 177–181, 1979.
46. Wallnau, L. B. and G. G. Gallup, Jr. A serotonergic, midbrain-raphe model of tonic immobility. *Biobehav Rev* **1:** 35–43, 1977.
47. Woodruff, M. L., D. C. Hatton, M. B. Frankl and M. E. Meyer. Effects of scopolamine and physostigmine on tonic immobility in ducks and guinea pigs. *Physiol Psychol* **4:** 198–200, 1976.
48. Wong, D. T., J. S. Horng and R. W. Fuller. Kinetics of serotonin accumulation into synaptosomes of rat brain—Effects of amphetamine and chloroamphetamines. *Biochem Pharmacol* **22:** 311–322, 1973.