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Memory Formation for an Appetitive Visual Discrimination Task in Young Chicks

MARIE E. GIBBS AND K. T. NG

Department of Psychology, La Trobe University, Bundoora, Victoria, 3083, Australia

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GIBBS, M. E. AND K. T. NG. *Memory formation for an appetitive visual discrimination task in young chicks.* PHARMAC. BIOCHEM. BEHAV. 8(3) 271–276, 1978. – The three-phase model of memory formation in young chicks proposed by Gibbs and Ng [7] was based on a single trial passive avoidance task. Some methodological and interpretative problems associated with this task are not encountered in appetitive visual discrimination tasks. Using such a task, it is shown that 2 mM KCl induces amnesia at 10 min, ouabain at 30 min and cycloheximide at 60 min after learning. These findings are consistent with those for the single trial passive avoidance task and confirm the generality of a model of memory formation in young chicks entailing a short-term phase, a sodium pump-dependent labile phase, and a long-term, protein synthesis-dependent phase.

Visual discrimination Memory Potassium chloride Ouabain Cycloheximide

A THREE phase sequentially dependent model of memory formation has been postulated by Gibbs and Ng [6,7] on the basis of a systematic investigation of the effects of a number of pharmacological treatments on memory for a single trial passive avoidance task in day-old chickens. A short-term memory (STM) is formed within 5 min after learning and decays exponentially after 10 min following learning. The formation of STM is inhibited by 1 or 2 mM potassium chloride (KCl), isotonic KCl, 154 mM LiCl, and 4 mM glutamate, and is attributed to hyperpolarization associated with K^+ conductance changes following neural activity. It is suggested that hyperpolarization induced by sodium pump activity leads to the formation of a second phase of memory, the labile phase. Labile memory is formed by 10 min after learning and decays exponentially after 30 min following learning. Its formation may be inhibited by the sodium pump inhibitors ouabain and ethacrynic acid. Long-term, relatively permanent memory (LTM) is consolidated after 30 min following learning and shows no decay by 24 hr. Its formation is inhibited by the protein synthesis inhibitors cycloheximide (CXM) and anisomycin.

The above findings are interpretatively consistent with those from other laboratories on different species [1,12] of animals or on the same species with the same tasks [3, 4, 8, 11, 13] or different tasks [10,11]. In no case, however, has the distinction between short-term and labile memory been explored. The generality of the above model, particularly with respect to precise temporal parameters, remains to be demonstrated for other species of animals and for the same species with different tasks. We report experiments related to the latter.

The single trial passive avoidance task used by Gibbs and Ng [7] carries with it a number of methodological difficulties. Briefly, the task involves pretraining day-old chickens to peck at a chromed bead (4 mm dia.), dipped in water, attached to a metal rod bent at a right angle approximately 1 cm from the bead. On the learning trial, a similar bead dipped in the chemical aversant, methyl anthranilate is presented for 10 sec. On the retention trial, a dry bead is presented for 10 sec. Chickens are pretrained, trained, and tested in pairs.

The association to be learned is that between the aversive taste of methyl anthranilate and visual characteristics of the bead. On the learning trial, learning is assumed to have taken place if the chicken exhibits a number of behavioural signs of distaste on pecking the bead: shaking of the head and vigorous wiping of the beak on the floor of the cage. Memory for the association is inferred on the retention trial if the chicken avoids the bead. The response is therefore one of binary choice: pecking versus non-pecking.

The above operationalizations of learning and memory clearly provide no unequivocal evidence that the desired association has been formed and no index of the strength of the association within a single subject, although the possibility of generalized avoidance due to non-memory factors can be ruled out by appropriate discrimination controls. Thus, one can successfully distinguish between memory enhancement by the treatment and generalized inhibition of the peck response, and between memory inhibition and generalized enhancement of the peck response [7]. It is possible that the aversive taste may have been associated with visual cues other than those related to the lure, as well as non-visual cues. The taste of the aversant can persist for some time after learning and possibly not dissipate until after 5 min [4]. Thus the assessment of memory immediately following learning is difficult. This is significant with respect to information about the early stages of the formation fo short-term memory and about possible earlier phases of memory formation (such as the electroshock-sensitive phase of Booth [2]).

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The methodological problems outlined above can be overcome to some degree by appropriate control experiments [7]. More important is the theoretical issue associated with the quantification of the strength of memory. The need for quantification is implied in the concept of trace and is embodied in the notions of development and decay of traces. Because the binary choice response entailed in the single trial passive avoidance paradigm used in these experiments does not permit indices of strength of learning and memory for a single subject, reasonably complex assumptions are required in treating the proportion of chickens avoiding the lure during retention tests as a measure of strength of memory. These are discussed in detail in Gibbs and Ng [7]. Since these assumptions are not directly amenable to empirical tests, their validity, as does the validity of the model as a whole, rests in part on demonstrating that the postulates of the model have generality across tasks where these assumptions are not made.

Rogers, Drennen and Mark [10] showed that CXM inhibited memory for a visual appetitive discrimination task 24 hr after learning, when administered 5 min after learning to 5–6 or 11-day-old chickens. Twenty-five μl of 0.20 mg CXM was administered intracranially to the centre of each forebrain. The results were confirmed by Rogers *et al.* [11] using 9-day-old chickens. In the latter study, no memory deficit was induced by CXM when tested 30 min after learning. These results are consistent with Gibbs and Ng's [7] argument that LTM is not fully developed until after 30 min following learning.

Of some concern, however, is the report by Rogers *et al.* [11] that chickens treated with 0.2 μg ouabain, administered intracranially in 25 μl volumes to the centre of each forebrain 10 min before learning the same visual discrimination task, showed no evidence of discrimination after 60 pecks. Furthermore, chickens retested 30 min after the initial block of 60 pecks again showed no evidence of discrimination in the first 20 pecks but developed some discriminatory behaviour by 40 pecks. However, chickens retested 1 hr or 24 hr after the initial block of 60 pecks showed immediate and almost perfect discrimination! These results were confirmed with ethacrynic acid (4 μg). With this drug, however, memory for the discrimination appeared as early as 20 min after learning. The authors interpreted the results as due to ouabain (and ethacrynic acid) temporarily rendering labile memory inaccessible, although labile memory has been formed. This was said to be supported by the observation that coupling CXM with ouabain produced loss of memory at 24 hr but left memory intact at 1 hr, again with no evidence of learning during the learning trial. The authors suggest that, at least with appetitive discrimination, formation of labile and long-term memory may be by parallel processing. This contrasts markedly with our postulation of sequentially dependent phases in the formation of memory for a single trial passive avoidance task. The experiments reported in this paper constitute in part an attempt to validate these findings.

METHOD

Animals

Five–six-day-old White-Leghorn Black-Australorp cockerels were obtained from a local poultry farm on the morning of hatching, and housed in pairs in wooden boxes 20 × 25 cm and opened at the top. Food and water were available ad lib and the room kept at constant temperature (27–30°C) and humidity. Ten different chickens were used for each treatment-retention condition.

Procedure

The task was essentially the same as the appetitive visual discrimination task described by Rogers *et al.* [10]. Each day prior to the experimental day, chickens were given experience in feeding in isolation in test cages similar to the experimental cage. For these sessions, millet grain was scattered on a perspex floor. On each experimental day chickens were deprived of food for 3 hr prior to being trained to discriminate between pebbles and grains of chick mash. The grains were scattered on a perspex floor to which glued pebbles of approximately the same size as the grains. Choices between grain and pebble were scored manually in blocks of 20 pecks, and only the initial peck at a given grain or pebble was counted. A chicken was assumed to have learned the discrimination if it made 4 or less errors in 20 pecks. Chickens were allowed a minimum of 60 pecks. Typically, all chickens achieved the learning criterion within 80 pecks or less, and in less than 5 min. After training, chickens were returned to their home cage and not fed unless retention tests were carried out longer than 3 hr after training.

Retention tests were carried out at various times between 10 min and 180 min or 24 hr after training. The number of errors in the first 20 pecks was taken as a measure of retention, but the test was extended for a further 40 pecks to ensure that chickens treated with amnesic drugs were capable of relearning the discrimination.

Drugs and Injections

All drugs were made up in sterile NaCl (154 mM, 0.9%). A 10 μl volume of 2 mM KCl (BDH), 0.027 mM ouabain (Sigma; 0.4 μg/chicken), 3.5 mM CXM (Upjohn; 20 μg/chicken), or 154 mM NaCl was administered intracranially by freehand injection into the centre of each side of the forebrain, to a depth of 3–3.5 mm, using a Hamilton repeating dispenser syringe. Ouabain, CXM, and NaCl were administered 10 min before learning, and KCl and NaCl 5 min before learning.

RESULTS

For each chicken, the number of errors in each block of 20 pecks until attainment of the criterion of 4 or less errors per block was recorded, as well as the time taken on each block and the total time taken to reach criterion. During retention testing, the number of errors per 20 pecks for 3 consecutive blocks of 20 pecks each were recorded.

If the three phase model is generalizable to memory for the appetitive visual discrimination task, predictions from the model are reasonably unequivocal, and involve comparisons between saline- and drug-treated groups at each learning-retention interval. All comparisons were therefore tested using the technique of planned contrasts on means, with a type-1 error rate of 0.05 [9].

Effects of KCl

Table 1 gives the mean number of errors and the associated standard deviation for each block of 20 pecks for each group of chickens during training, retention, and

TABLE 1

MEANS AND STANDARD DEVIATIONS OF NUMBER OF ERRORS

	Training Blocks of Trials					Retention Blocks of Trials			
Saline	0 - 20	21 - 40	41 - 60	61 - 80	Time (Sec)	0 - 20	21 - 40	41 - 60	N
10 min	14.9 (3.8)	8.1 (3.8)	3.0 (2.2)	1.8 (2.5)	219.0 (102.9)	2.8 (1.8)	1.1 (1.3)	1.1 (2.5)	10
30 min	15.2 (2.9)	7.8 (4.6)	3.9 (2.2)	1.0 (2.3)	206.0 (106.3)	2.4 (1.4)	1.8 (1.6)	0.8 (0.9)	10
60 min	14.6 (4.2)	7.7 (5.9)	3.7 (3.5)	0.4 (1.0)	122.0 (60.5)	1.9 (1.3)	0.6 (0.7)	1.0 (0.9)	10
120 min	17.0 (2.4)	11.7 (6.4)	3.7 (3.4)	1.0 (2.5)	105.0 (69.2)	2.4 (1.9)	1.5 (1.3)	0.5 (0.7)	10
180 min	14.7 (3.2)	7.0 (1.9)	3.6 (1.8)	0.6 (1.3)	112.0 (77.6)	3.2 (2.0)	1.6 (1.6)	1.6 (1.3)	10
24 hr	15.8 (1.5)	8.4 (3.3)	2.7 (1.1)	0	91.0 (35.7)	2.6 (1.2)	1.2 (1.1)	0.9 (0.9)	10
Means	15.37	8.45	3.43	0.8	142.3				
KCL									
10 min	14.7 (4.7)	11.1 (5.8)	4.7 (2.9)	1.5 (1.4)	210.0 (71.8)	7.7 (3.3)	3.3 (2.5)	2.1 (2.2)	10
30 min	13.7 (4.4)	7.1 (3.6)	3.0 (1.7)	0.6 (1.3)	173.0 (84.5)	8.3 (2.4)	3.0 (2.2)	2.9 (1.3)	10
60 min	16.3 (2.3)	9.5 (3.2)	4.0 (3.2)	0.8 (1.7)	178.0 (70.7)	11.8 (2.0)	5.5 (1.7)	1.9 (1.7)	10
120 min	14.8 (3.7)	8.5 (4.8)	5.0 (5.0)	2.7 (3.3)	151.0 (49.3)	8.1 (3.4)	2.7 (1.5)	1.5 (1.5)	10
180 min	12.3 (3.3)	6.7 (2.6)	3.1 (2.4)	0.4 (1.3)	212.0 (108)	10.1 (2.4)	4.1 (2.5)	2.8 (1.5)	10
24 hr	17.0 (2.6)	8.6 (4.0)	2.9 (3.8)	0	131.0 (59.2)	9.6 (2.6)	5.0 (2.6)	2.8 (1.8)	10
Means	14.83	8.58	3.78	1.0	175.83				

Means and standard deviation (in parentheses) of number of errors per block of 20 pecks on training and retention trials for groups of 10 chickens each treated with either 2 mM KCl or 154 mM NaCl 5 min before training and tested for retention at various times after training. Mean and SD for time taken to reach the learning criterion of 4 errors or less per 20 pecks is also given.

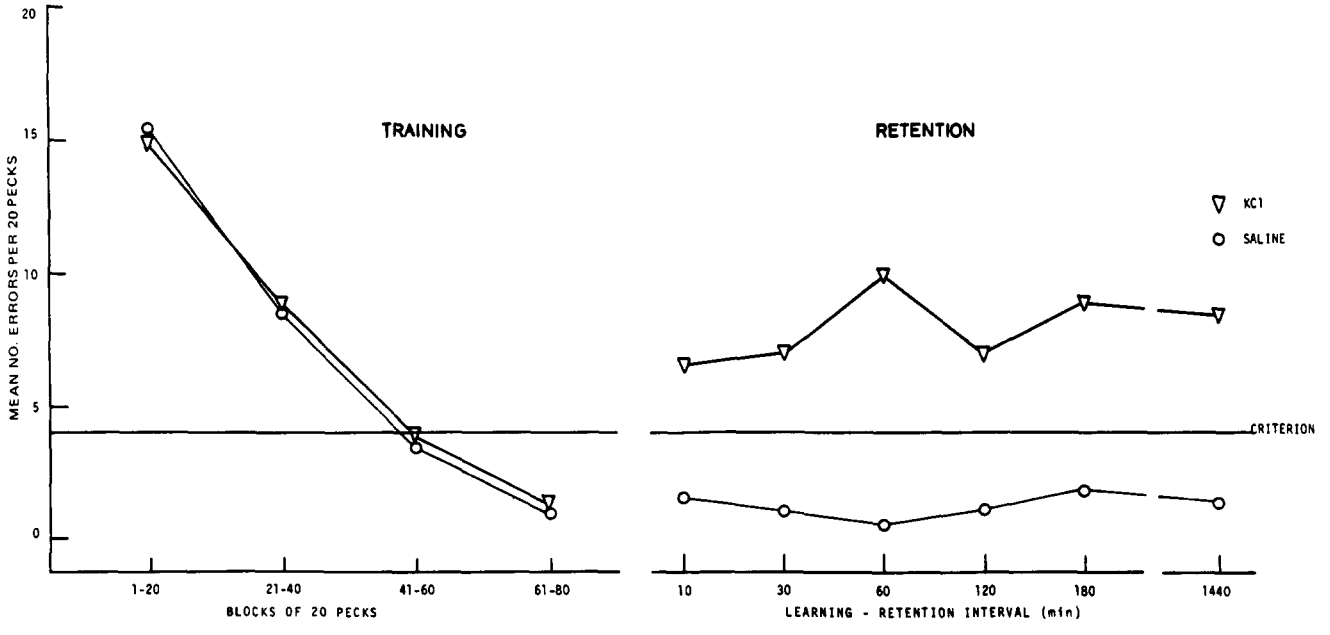

FIG. 1. Effect of KCl on retention for appetitive visual discrimination task, after training to a criterion of learning of 4 errors or less in 20 pecks. One hundred fifty-four mM NaCl or 2 mM KCl were administered intracranially 5 min before commencement of training and retention intervals defined from the end of 20 pecks when the criterion is met. Ten different animals were used for each learning-retention interval.

TABLE 2

MEANS AND STANDARD DEVIATIONS OF NUMBER OF ERRORS

	Training Blocks of Trials					Retention Blocks of Trials			
Saline	0 - 20	21 - 40	41 - 60	61 - 80	Time (Sec)	0 - 20	21 - 40	41 - 60	N
10 min	15.1 (3.4)	7.7 (4.1)	3.5 (3.1)	0.4 (1.3)	121.0 (63.5)	2.5 (1.3)	1.0 (0.8)	1.2 (1.1)	10
30 min	16.7 (2.4)	10.2 (6.6)	3.9 (4.6)	0.8 (1.8)	86.0 (34.1)	2.0 (1.9)	1.2 (1.5)	1.8 (1.5)	10
60 min	18.4 (1.2)	10.8 (6.1)	6.3 (5.3)	2.8 (4.3)	107.0 (49.5)	2.2 (2.4)	1.5 (1.4)	2.9 (2.8)	10
120 min	11.2 (2.7)	4.0 (2.3)	2.8 (1.5)	0.6 (1.6)	187.5 (92.2)	2.4 (1.4)	1.5 (0.9)	1.0 (0.9)	10
180 min	12.4 (3.7)	6.0 (2.5)	3.9 (2.3)	1.9 (2.5)	198.5 (80.8)	2.2 (1.4)	1.5 (1.8)	0.9 (1.0)	10
24 hr	16.2 (1.6)	7.8 (2.8)	2.3 (1.2)	0	92.0 (53.9)	3.3 (1.5)	1.8 (1.2)	1.6 (1.4)	10
Means	15.0	7.75	3.78	1.25	132.0				
Ouabain									
10 min	15.4 (2.8)	7.3 (2.9)	2.7 (1.6)	0	116.0 (27.6)	3.1 (1.9)	2.2 (1.4)	2.1 (1.0)	10
30 min	16.3 (3.4)	6.6 (4.3)	2.6 (2.1)	0.5 (1.3)	201.0 (130.8)	8.9 (2.5)	2.6 (1.9)	1.1 (0.9)	10
60 min	16.3 (1.8)	9.4 (3.0)	3.3 (2.2)	0.1 (0.3)	136.0 (82.1)	11.5 (3.0)	5.8 (2.0)	2.4 (1.6)	10
120 min	16.8 (1.9)	9.3 (2.7)	2.3 (1.1)	0	113.0 (64.3)	10.9 (2.1)	4.6 (1.6)	2.4 (1.6)	10
180 min	16.4 (2.5)	8.3 (2.8)	3.3 (3.1)	0.4 (1.3)	145.0 (58.4)	9.8 (2.2)	4.0 (1.7)	1.3 (0.9)	10
24 hr	16.4 (3.0)	10.4 (4.9)	5.1 (4.3)	0.7 (1.3)	164.0 (66.7)	11.4 (4.6)	4.7 (2.6)	2.1 (1.4)	10
Means	16.27	8.55	3.22	0.28	145.83				
CXM									
10 min	16.4 (2.7)	7.5 (1.8)	2.5 (1.0)	0	103.0 (52.1)	3.0 (1.4)	2.1 (1.3)	1.2 (1.3)	10
30 min	13.7 (3.2)	6.3 (1.7)	2.8 (1.4)	0	110.0 (59.4)	2.9 (1.6)	2.2 (2.5)	1.9 (1.7)	10
60 min	14.0 (3.4)	7.3 (3.4)	2.3 (1.3)	0	146.0 (67.7)	5.5 (1.8)	2.5 (1.6)	1.2 (1.3)	10
120 min	14.4 (3.0)	7.8 (5.1)	3.4 (3.3)	0.4 (1.0)	124.0 (42.5)	10.4 (6.1)	4.1 (3.3)	1.9 (1.3)	10
180 min	16.5 (3.2)	10.4 (5.4)	6.7 (5.9)	1.2 (1.9)	162.0 (68.1)	10.4 (1.8)	2.9 (0.9)	1.1 (0.7)	10
24 hr	14.5 (2.4)	8.3 (2.1)	3.2 (2.1)	0.7 (1.6)	107.5 (56.6)	11.3 (2.3)	4.3 (1.5)	2.8 (1.4)	10
Means	14.91	7.93	3.5	0.37	130.25				

Means and standard deviations (in parentheses) of number of errors per block of 20 pecks on training and retention trials for groups of 10 chickens each treated with 0.027 mM ouabain, 3.5 mM CXM, or 154 mM NaCl, 10 min before training and tested for retention at various times after training. Mean and SD for time taken to reach the learning criterion of 4 errors or less per 20 pecks is also given.

relearning trials, as well as the mean and SD of time taken to reach criterion during training.

Inspection of the table reveals no substantial differences between and within drug groups in the training data. On the average, however, the KCl-treated groups differed significantly in mean time taken to reach criterion from the saline-treated groups (mean times are 175.83 and 142.50 sec respectively; $p<0.05$). The rate of learning in terms of errors per block of 20 pecks is virtually identical for the two drug conditions (Fig. 1). While KCl may have slowed down the average rate of pecking, it may be assumed that KCl-treated chickens have attained the same level of learning as saline-treated chickens prior to retention testing. Furthermore, the two sets of chickens would appear to have been exposed to approximately the same number of pecks. Any difference in retention tests may therefore be attributed to memory processes alone.

No memory loss at any learning-retention interval is observed for saline-treated chickens, the mean number of errors in the first block of 20 pecks during retention tests being always less than 4 (Fig. 1). KCl-treated chickens, however, showed some degree of memory loss at all learning-retention intervals. All differences from saline-treated chickens are significant. Amnesia is in every case partial, the mean number of errors being always less than the naive level observed during the first 20 training pecks. The important finding, however, is that KCl induces amnesia as early as 10 min after learning, and the amnesia is maintained for up to 24 hr.

Effects of Ouabain and CXM

Table 2 summarizes the data for training, retention and relearning trials for each group of chickens treated with saline, ouabain or CXM 10 min before training. Once again, no differences are apparent for rate of learning or final level of learning between and within drug groups. The learning functions for the 3 drug conditions are virtually identical (Fig. 2). There is no significant difference in mean time taken to reach criterion during the training trials across the 3 drug conditions (mean times were NaCl: 132.0 sec; ouabain: 145.83 sec; CXM: 130.25 sec).

As shown in Fig. 2, saline-treated chickens showed no retention deficits at any learning-retention interval. The performance of ouabain-treated chickens differed significantly from saline-treated chickens at all learning-retention intervals from 30 min onward. Performance at 10 min after learning was identical to that observed for saline-treated animals. On the other hand, CXM yielded significantly worse performance compared with saline only at 60 min or longer after learning, retention levels at 10 and 30 min being comparable with those obtained with saline. Again, amnesia is partial where it occurs, especially with CXM at the 60 min learning-retention interval.

DISCUSSION

The task used in the series of experiments reported here differs in a number of significant ways from the single trial passive avoidance task used by Gibbs and Ng [7] to

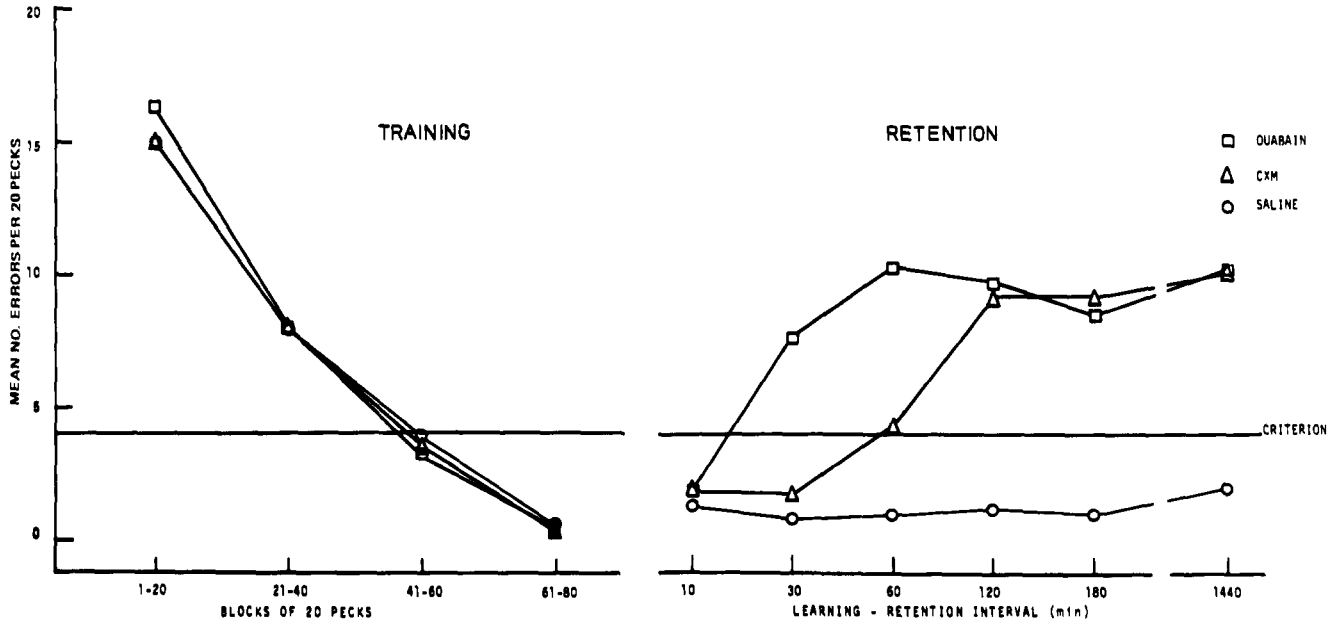

FIG. 2. Effects of ouabain and CXM on retention of appetitive visual discrimination task, after training to a criterion of learning of 4 errors or less in 20 pecks. One hundred fifty-four mM NaCl, 0.027 mM ouabain or 3.5 mM CXM were administered intracranially 10 min before commencement of training and retention intervals defined from the end of 20 pecks when the criterion is met. Ten different animals were used for each learning-retention interval.

generate evidence in support of their three-phase model of memory formation. The appetitive visual discrimination task is essentially a multi-trial task [10] and carries with it the advantage of providing a basis for objectively controlling the level of learning for each individual animal. This permits in turn the possibility of a more precise measure of memory and of memory loss than is possible with the single trial passive avoidance task, without having to make a number of assumptions difficult to substantiate empirically [7]. The visual discrimination task also avoids possible sources of error associated with the presentation of the lure in the task used by Gibbs and Ng. The main disadvantage of the task is that the multi-trial nature of learning allows for the overlapping of memory processes associated with each elemental trial. The final operational representation of memory would be the end product of the integration of a number of individual memory traces, more or less temporally spaced. Propositions concerning temporal parameters associated with the development and decay of memory and temporal characteristics of the effects of amnesia-inducing treatments may have to rely on the assumption of a functionally unitary trace arising from the multiple learning trials. This may pose a problem if the time taken to reach criterion learning and/or the number of trials required vary substantially from individual to individual or, more significantly, from treatment condition to treatment condition. This is especially serious when dealing with stages of memory formation in close temporal proximity to the learning experience.

Notwithstanding the above qualifications, the results from the present experiments are consistent with the three phase model of memory formation postulated by Gibbs and Ng [6,7]. In particular, 2 mM KCl, ouabain, and CXM appear to inhibit different phases in the memory formation sequence; 2 mM KCl inducing partial amnesia as early as 10 min after learning, ouabain 30 min after learning, and CXM 60 min after learning. These temporal parameters correspond closely to those obtained with the same drugs using single trial passive avoidance learning and associated discrimination paradigms. The retention losses are maintained in each case for at least 24 hr. The differences in time of onset of the deficits cannot be attributed to delayed action of ouabain or CXM. The same retention function has been obtained with ouabain administered as early as 15 min before learning [6,7] and with CXM administered as early as 30 min before learning in a passive avoidance task [7]. Furthermore, CXM has been shown to yield approximately 90% inhibition of ^{14}C-leucine incorporation *in vivo* into chicken forebrain protein by 10 min after administration [5].

The results with ouabain contradict those obtained by Rogers *et al.* [11]. Ouabain-treated chickens in the present experiments showed no evidence of learning difficulties but suffered retention losses 30 min and later following learning. The reverse was true for the chickens in the Rogers *et al.* experiments. The difference in the volume of drug administered (10 μl per hemisphere here as against 25 μl per hemisphere in the other) may be a relevant factor, but the mechanisms underlying the difference in effects are not clear. Rogers *et al.* did not define a criterion for learning and the absence of unequivocal evidence of learning in the ouabain-treated chickens by the end of the training period makes the interpretation of their results in terms of memory processes difficult. The possibility that their 30 min results with ouabain may be a continuation of the learning sequence initiated during the training period and disrupted by ouabain cannot be dismissed. Conclusions regarding parallel processing may be premature.

We offer the tentative conclusion that the model of memory formation we proposed for the single trial passive avoidance task in day-old chickens applies to memory formation for the appetitive visual discrimination task, in

terms of stages in the formation sequence, temporal parameters associated with these stages, and the possible mechanisms underlying the stages. The last possibility requires further substantiation with drugs that challenge the postulated physiological and biochemical effects of the amnesia-inducing drugs used here [7], but the present results are encouraging with respect to the generality of the model.

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