taste paired with LiCl can be anticipated and avoided—that the anticipation of a taste, rather than the taste itself, can be aversive. The usual taste-aversion experiment demonstrates only escape from an aversive taste. As a historical sidelight, this is a particularly clear demonstration in the rat of what Tolman called an "insight" or "foresight" mechanism (a sign-gestalt-expectation). More than 40 years ago, Miller showed that such a mechanism could be deduced from Pavlovian conditioning principles in the form of Hull's fractional anticipatory goal response (*13*).

Experiment 2 adds the finding that PRF training can reduce (immunize the rat against) the suppressive effects of the anticipation of the conditioned aversive taste and that such training attenuates the suppression of drinking of such a taste solution. If rats are reinforced intermittently and inconsistently with a particular flavored solution, they will avoid that flavor less and drink more of it when it is subsequently paired with gastrointestinal illness. Such a finding has potential practical as well as theoretical implications. One practical application might be to therapeutic situations in which taste aversions and anorexia frequently result from drug or radiation treatments or chemotherapy (*14*). The theoretical implications are for broadening the range of generalization and transfer of persistence in responding across motivational-reward systems (*15*).

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References and Notes

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2. For example, J. Garcia and R. A. Koelling, *Psychon. Sci.* **4**, 123 (1966); J. Garcia, F. R. Ervin, R. A. Koelling, *ibid.* **5**, 121 (1966).
3. Exceptions are P. J. Best, M. Best, and R. N. Ahlers [*ibid.* **25**, 281 (1971)] and E. W. Holman [*Learn. Motiv.* **6**, 358 (1975)]. Both of these experiments were investigations of transfer from taste-aversion conditioning to an operant lever press with the aversive taste as the reward. In the first experiment, baseline lever-press training was with water rather than with the later-conditioned (saccharin) taste. The procedures of the second were similar to those in our first experiment, but, contrary to our findings, extinction was not hastened with the aversive taste (saccharin) as reward.
4. C. J. Levy, M. E. Carroll, J. C. Smith, K. G. Hofer, *Science* **186**, 1044 (1974).
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6. For clarity of presentation, we reported only the total speed (centimeters per second) over the entire runway, although all measures showed the effects and start speed was perhaps the most sensitive measure.
7. Analysis of variance on extinction data revealed a significant groups effect [$F(3, 36) = 5.41$, $P < .005$] and a groups by trials interaction [$F(12, 144) = 2.45$, $P < .01$]. Subsequent Newman-Keuls tests of the overall means for the four groups indicated that the saline control group ran faster than the vinegar-LiCl ($P < .01$), the saccharin-LiCl ($P < .05$), and the water-LiCl ($P < .05$) groups, and that the saccharin-LiCl and water-LiCl groups ran faster than the vinegar-LiCl groups (P's $< .05$).
8. The group effect [$F(3, 36) = 12.23$, $P < .001$] indicated a significant difference in vinegar intake among the four groups after runway extinction. Newman-Keuls tests showed that the saline control animals drank more vinegar solution than the vinegar-LiCl ($P < .01$), the saccharin-LiCl ($P < .05$), and the water-LiCl ($P < .05$) groups, and that the latter two groups, in turn drank more than the vinegar-LiCl group (P's $< .05$).
9. Examples are J. Garcia and R. A. Koelling, *Radiat. Res. Suppl.* **7**, 439 (1967); W. A. McLarin, J. A. Farley, B. B. Scarborough, *Radiat. Res.* **18**, 473 (1963); S. H. Revusky and E. W. Bedarf, *Science* **155**, 219 (1967).
10. This additional precaution was probably unnecessary in view of recent research showing that (i) after a single preliminary exposure to sucrose, neither additional exposures nor length of delay between successive exposures has any additional attenuating effect on taste-aversion conditioning [J. W. Kalat and P. Rozin, *J. Comp. Physiol. Psychol.* **83**, 198 (1973)], and (ii) under conditions of severe water deprivation, preliminary exposure to saccharin does not greatly attenuate the taste-aversion conditioning [M. Domjan, *Learn. Motiv.* **3**, 389 (1972)].
11. Analysis of variance yielded a significant effect of reward [$F(1, 48) = 45.93$, $P < .001$] and a significant interaction of reward and trials, [$F(9, 432) = 18.99$, $P < .001$], indicating a clear PRF extinction effect. The interaction of poison (LiCl versus NaCl) and trials was significant [$F(9, 432) = 4.37$, $P < .001$], indicating that the poisoned groups (CRF-LiCl and PRF-LiCl) extinguished faster than the saline controls (CRF-NaCl and PRF-NaCl). Perhaps more important is the significant interaction of reward and poison [$F(1, 48) = 5.95$, $P < .025$]. Subsequent Newman-Keuls tests showed that the PRF-LiCl group extinguished at about the same rate as PRF-NaCl.
12. After extinction, the poisoned groups (CRF-LiCl and PRF-LiCl) consumed less saccharin solution than the saline controls [$F(1, 48) = 346.09$, $P < .001$]. The PRF animals (PRF-LiCl and PRF-NaCl) drank more solution than their CRF counterparts [$F(1, 48) = 12.38$, $P < .001$]. Newman-Keuls tests among the means of the four groups showed that the PRF-LiCl group drank more saccharin solution than the CRF-LiCl group ($P < .01$); the PRF-NaCl and CRF-NaCl groups did not differ from each other.
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Asymmetry in Facial Expression

The conclusion of Sackeim *et al.* (*1*) that there is "greater right-hemispheric involvement in the production of emotional expression" is unwarranted. They found that observers judge double-left composite faces as showing more intense emotion than double-right composite faces. However, they failed to consider the possibility that peripheral neural and anatomical differences rather than differences in the activity of the right and left cerebral hemispheres could explain such results. Facial surgeons note (*2*) that the two sides of the face differ in the size of the muscles, in fatty deposits (*3*), and in the neural supply from the facial nerve nucleus to the facial muscles. Without controls for such differences, the findings of Sackeim *et al.* cannot be interpreted as being due solely to differences in the impulses sent from the two cerebral hemispheres to the facial nuclei.

There is also reason to question whether Sackeim *et al.* were justified in talking about lateralization in emotional expressions, since they studied a different type of facial movement. Neurologists distinguish between voluntary facial movements (by which they usually mean the ability to perform requested actions) and spontaneous emotional expressions. The evidence is clear that these two types of facial activity depend upon different neural pathways (*4*). The potential independence of these two types of facial actions is dramatically shown in clinical cases in which lesions in the pyramidal system (for example, the precentral gyrus) impair requested facial movements but leave emotional facial movements intact, whereas lesions in nonpyramidal systems produce the reverse pattern. This evidence emphasizes the need for caution in generalizing from studies of requested facial movements to emotional expression and vice versa. Thus, it is crucial to know whether the facial movements studied by Sackeim *et al.* were requested or more spontaneous emotional expressions.

Sackeim *et al.* did not accurately describe the photographs they used, which W. V. Friesen and I supplied to them. They wrote that the pictures showed "posed" emotions, or "subjects deliberately attempting to convey particular emotions." Posing may involve either deliberate performance or some attempt to reexperience an emotion to produce the expression. If our photographs had been posed it would be unclear which kind of facial movements Sackeim *et al.* had studied. With few exceptions, however, the photographs they used were not even poses, but the most deliberate

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performance of requested facial movements. In describing how the photographs were taken, Friesen and I wrote that the photographic models "were not told to feel an emotion, but rather given instructions such as lower your brow so that it looks like this, . . . or tighten your lower eyelid" (*5*, p. 170). Because our photographs were of requested facial movements, not of emotional poses, there must be even more caution in generalizing to spontaneous facial expression of emotion.

The fact that they found no left-right differences in judgments of the happy photographs is important since, unknown to Sackeim *et al.*, these were the only photographs of spontaneous emotional expression rather than deliberately performed facial actions. In making the happy photographs, we caught the models off guard during a spontaneously occurring happy moment in the photographic session. It might be argued that the reason no left-right differences were found in these happy pictures was not because they showed spontaneous rather than requested actions, but because positive emotions alone are not asymmetrical in appearance (*6*). However, Ekman, Hager, and Friesen (*7*) found that asymmetrical deliberate smiles are usually more intense on the left than on the right side, and they replicated findings by Lynn and Lynn (*8*) that asymmetrical spontaneous smiles are relatively infrequent and are not usually more intense on a particular side of the face. These findings suggest that facial asymmetry (with left stronger than right) is apparent only with deliberate and not spontaneous expressions, but studying muscle movements involved in the negative emotions is necessary to generalize beyond the smile.

The issue of left-right differences is not resolved for either emotional expression or requested facial movements. Both types of action need to be studied, ideally in the same subjects and in situations that clearly differentiate the type of facial movement elicited. The methods must control or bypass the type of peripheral differences in facial anatomy that cloud results based solely on observers' judgments of emotion. For now, more caution is needed in interpreting the findings from studies based on observers' judgments of emotion to either requested facial movement or emotional expression, and in generalizing from studies of the former to the latter.

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References and Notes

1. H. A. Sackeim, R. C. Gur, M. C. Saucy, *Science* **202**, 434 (1978).
2. As a facial surgeon with wide experience with face-lifts, parotid gland operations, and surgery to remedy facial paralysis, R. Crumley (personal communication, 1 February 1980) has stated that he has little doubt that asymmetry of facial structure is the rule rather than the exception.
3. Data on consistent differences in the relative size of the left and right sides of the face are reported in P. H. Burke, *Hum. Biol.* **43**, 536 (1971).
4. A. Meihlke, *Surgery of the Facial Nerve* (Saunders, Philadelphia, 1973); R. E. Myers, *Ann. N.Y. Acad. Sci.* **280**, 745 (1976); K. Tschiasshy, *Ann. Otol. Rhinol. Laryngol.* **62**, 3, 677 (1953).
5. P. Ekman and W. V. Friesen, *Unmasking the Face* (Prentice-Hall, Englewood Cliffs, N.J., 1975); *Pictures of Facial Affect* (Consulting Psychologists Press, Palo Alto, Calif., 1976).
6. There are still ambiguities about asymmetry in smiling. R. Campbell [*Cortex* **14**, 327 (1978)] found asymmetries in requested smiles, but has said, "I did find expressor asymmetries. . . . These have turned out to be due to the left side of face . . . being rated 'more sad' in a 'relaxed,' unposed still photo than the right side of face. In fact, of the eighteen right-handers I used I did *not* find that a posed smile was stronger on the left of the face. So the discussion in the paper is a bit misleading" (personal communication, 3 December 1977). G. E. Schwartz (personal communication) has found asymmetries in electromyographic activity in the facial area relevant to the smile, but it is not certain that he was studying spontaneous emotional expression.
7. P. Ekman, J. C. Hager, W. V. Friesen, in preparation.
8. J. G. Lynn and D. R. Lynn, *J. Abnorm. Soc. Psychol.* **33**, 291 (1938); *ibid.* **38**, 250 (1943).
9. I thank W. V. Friesen, J. Hager, H. Oster, and M. O'Sullivan for their comments.

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The conclusion of Sackeim *et al.* (*1*) that the greater perceived intensity of emotional expression on the left side of the face "points to greater right-hemispheric involvement in the production of emotional expression" is probably premature.

Having access to the same set of stimuli as Sackeim *et al.*, we tested the equality of the sides of the face directly. Of the 110 slides of facial affect (*2*), 34 were selected on the basis of their "measurability." Measurability was determined by the extent to which precise measurements could be taken from vertical midline of the face to the edge where the head and ear merge. The remaining 76 slides were rejected because such things as interfering sideburns or hairlines made it difficult to obtain accurate measurements.

The 34 stimuli chosen consisted of five male and two female models and the same six expressions used by Sackeim *et al.* Each slide was projected onto a white background, to a chin-to-eyebrow height of approximately 13 cm. Measurements were taken from the edge where the ear and head meet (easily distinguishable in a two-dimensional photograph) to the vertical midline of the head. A comparison of means for the left and right sides of the face was thinner ($\bar{X}$ = 6.44 cm, standard deviations = .70) than the right side ($\bar{X}$ = 6.75 cm, standard deviations = .58) (Wilcoxon matched-pairs signed-ranks tests, $T = 30$, $P < .001$). In 25 of the 34 slides the left side was thinner than the right, in two they were equal, and in only seven did the left side exceed the right in width.

The significant difference in the width of the left and right sides of the face leads us to suggest that the greater apparent intensity of expression on the left side of the face is not a function of differential right-hemispheric control of expression but rather results from the fact that the left side of the face provides a smaller area on which to distribute the same features.

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2 April 1979

In the example set of original and derived left- and right-side composite photographs of a posed facial expression furnished by Sackeim *et al.* (*1*), the original full-face photograph is not uniformly illuminated. The left side of the model's face shows a greater proportion of shadow than does the right side. Thus the derived left-side composite photograph appears darker and more "dramatic" than does the composite right-side photograph. Disparities in amount of shadow present between right and left composite photographs could influence subjects' assessments of expression intensity irrespective of actual facial differences.

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Reference

1. H. A. Sackeim, R. C. Gur, M. C. Saucy, *Science* **202**, 434 (1978).

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We reported (*1*, *2*) that voluntary expressions of emotion are rated as more intense on the left than on the right side of the human face. We interpreted our finding as supporting the hypothesis of hemispheric asymmetry in the control of facial emotional expression.

Nelson and Horowitz, Spinrad, and Ekman submit that our finding may be

due to artifact. Nelson and Horowitz suggest that the greater intensity of left-side expression "results from the fact that the left side of the face provides a smaller area on which to distribute the same features." They found that the left side was narrower in 25 of 34 photographs (seven posers) taken from the same set of 110 photographs (14 posers) from which we had selected 70 photographs (14 posers). If it is true that, in general, the left side of the human face is narrower than the right side, not only might this account for our finding, but it would be an important discovery in itself. However, we question this.

To make the case that asymmetry in facial width accounts for our finding, one must show that in our sample of photographs the two sides of the face differed significantly in size and that differences in size are correlated with judgments of emotional intensity. We originally selected photographs from the larger set so that males and females and the emotions expressed would be equally represented. On the basis of preliminary testing, we excluded photographs of poor quality, marked asymmetry in illumination, and asymmetry in width resulting from head tilt. To test Nelson and Horowitz's claim, we adapted their procedure by projecting our slides of left- and right-side composites to a chin-to-eyebrow height of 13 cm. We measured for each composite both the distance between the external canthi and the width of the face at its widest horizontal plane at or below the level of the eyes. The first measure uses convenient and standard points of reference (*3*), while the second permits the expression of asymmetry in facial morphology in the assessment of facial size (*4*). The distance between the external canthi did not differ in left-side [mean ($\bar{X}$) = 9.72 cm, standard deviation (S.D.) = 0.98] and right-side ($\bar{X}$ = 9.78, S.D. = 0.95) composites [$t(69) < 1$]. Likewise, the width at the widest part of the face did not differ for left-side ($\bar{X}$ = 13.28 cm, S.D. = 1.33) and right-side ($\bar{X}$ = 13.19 cm, S.D. = 1.39) composites [$t(69) < 1$]. Furthermore, the differences between left- and right-side composites in both measures of facial width were not correlated with the differences in mean-rated emotional intensity of the composites [$r(68) = .01$ and $r(68) = .05$ for the two measures of width, respectively].

The absence in our sample of consistent asymmetry in facial width accords with Burke's (*3*) findings that there are left-right differences within individuals, but little consistency across individuals. Nelson and Horowitz's results are likely due to small sample variation or the method they used to determine facial midline, or both (*5*).

On the basis of the illustrative figure accompanying our report (*1*), Spinrad suggests that our left-side composites were "darker and more 'dramatic,' " artifactually creating an impression of greater intensity. We had determined during preliminary testing that subjects could not distinguish by illumination left- and right-side composites, under the lighting conditions in which judgments of emotional intensity were obtained. To examine Spinrad's claim, we collected ratings of illumination differences between left- and right-side composites under viewing conditions that would maximize any differences (*6*). Left-side composites were judged brighter than right-side composites [$t(69) = 2.46$, $P < .02$]. However, ratings of illumination differences varied considerably, and these differences and the differences in mean ratings of emotional intensity were not correlated [$r(68) = -.11$]. Therefore, illumination differences were opposite in direction to Spinrad's expectation, and they did not influence the asymmetry in perceived emotional intensity.

Ekman first suggests that our finding was an artifact of differences in size of muscles, in fatty deposits, and in peripheral neural supply in the face. We know of no objective data indicating consistent facial asymmetry in these morphological characteristics (*7*). Burke (*3*), cited by Ekman, took four stereophotogrammetric measurements of size on each side of the face of 48 children. He found, "The mean values . . . show remarkable right-left similarity. The mean differences are all less than 1 mm, but when *individual* records are examined, there is quite marked right-left difference [emphasis added]" (*3*, p. 545; *8*). Considering the meager evidence for consistent peripheral neural and anatomical differences and the large body of studies, cited in our report (*1*), indicating hemispheric specialization in the regulation of emotion (*9*), we believe our hypothesis—that facial asymmetry in intensity of emotional expression is related to hemispheric specialization—was justified.

Ekman also questions our justification for generalizing from our finding with voluntary facial movements to involuntary or spontaneous emotional expressions. We did not make this generalization. Indeed, referring to the evidence for dissociation in the control of voluntary and involuntary emotional expressions, we stated (*1*, p. 435), "Whether or not the same asymmetry in intensity of expression would be obtained with more spontaneously occurring emotional expressions is a matter for future investigation." There has been little investigation of facial asymmetry with spontaneous emotional expressions. Ekman cites Lynn and Lynn's two studies (*10, 11*) as having examined spontaneous smiles, but incorrectly states that they found that "asymmetrical spontaneous smiles are relatively infrequent and are not usually more intense on a particular side of the face." Lynn and Lynn did not assess the "intensity" of smiles but rather examined the extent of lateral facial movement during smiles; they reported, "It was noticed that 91.5 per cent of all the smiles, during the initial periods of upward motion, show one mouth corner traveling farther and hence faster than the other. At some later time during the upward course, the initially slower mouth corner usually catches up with and often passes the mouth corner which led at first" (*12*).

Finally, Ekman questions whether by using the stimuli that he supplied (*13*) we were entitled to generalize even to posed emotions, since the expressions were produced by requests for deliberate and particular facial movements (*14*, p. 170). It is clear, however, that Ekman's models were producing facial expressions that were reliably perceived as emotions by raters (*1, 2*) and that these expressions were voluntarily produced. The models were not instructed to create more intense expressions on one side of the face and, thus, their photographs were suitable for the study of asymmetry in voluntary expression. The question of whether one can generalize from our finding with Ekman's stimuli to other types of voluntary or posed expression is an empirical issue. Since our report was published, we (*15*) have found that asymmetry in the emotional expression of the resting face of the right-handed is related to lateral eye dominance and to family history for left-handedness. Two other studies (*16*) have found that facial asymmetry in the resting face is related to handedness. In addition to the study conducted by Ekman *et al.* (*17*), five other investigations (*18–21*) have confirmed and extended our original finding by demonstrating consistent asymmetry in the voluntary expression of emotion. Two of these (*19*) showed relations between facial expressive asymmetry and handedness. These studies used a variety of different methods to obtain photographs of voluntary emotional expressions, further supporting our hy-

pothesis that facial asymmetry in emotional expression reflects hemispheric asymmetry of function.

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References and Notes

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2. H. A. Sackeim and R. C. Gur, *Neuropsychologia* **16**, 473 (1978).
3. P. H. Burke, *Hum. Biol.* **43**, 536 (1971).
4. The differences in facial width between left-side and right-side composites on the two measures were correlated substantially [$r(68) = .72$, $P < .001$].
5. The degree to which the tip of the nose deviates to the left or right is associated with handedness [P. R. Sutton, *Nature (London)* **198**, 909 (1967)]. Choice of the tip of the nose as a landmark could artifactually inflate asymmetry in facial size. In our laboratories, to establish facial midline we determine the best fitting line to a set of four points in the face, none of which are known to have consistent lateral deviation.
6. Composites were presented for 2 minutes in pairs on a solid white background with no external illumination. Slides were projected from a distance of 2 m with 300-W lamps to three raters who sat 1 m from the wall. For each pair, raters made difference judgments on a −5 to +5 scale. The correlations between pairs of raters were $r_{1,2} = .73$, $r_{1,3} = .76$, and $r_{2,3} = .80$.
7. R. Crumley, the facial surgeon cited by Ekman, knows of no data indicating *consistent* lateral asymmetry in these morphological characteristics (personal communication, 21 November 1979).
8. The only mention in Burke's report of a possible consistent difference was "The measurements on asymmetry suggest that in the maxillary area of the face, there is a tendency for the larger side to be on the left" (*3*, p. 546).
9. This literature is reviewed by H. A. Sackeim, M. S. Greenberg, A. L. Weiman, R. C. Gur, J. P. Hungerbuhler, and N. Geschwind (in preparation); findings are presented relating side of brain damage to uncontrollable outbursts of emotion.
10. J. G. Lynn and D. R. Lynn, *J. Abnorm. Soc. Psychol.* **33**, 291 (1938);
11. ———, *ibid.* **38**, 250 (1943).
12. In both studies Lynn and Lynn (*10, 11*) found associations between personality functioning and concordance between direction of smile asymmetry and handedness. In 1943 (*11*), they reported an association between direction of smile asymmetry and lateral eye dominance. These findings also support the hypothesis that hemispheric specialization is associated with asymmetry of facial expression.
13. P. Ekman and W. V. Friesen, *Pictures of Facial Affect* (Consulting Psychologists Press, Palo Alto, 1976).
14. ———, *Unmasking the Face* (Prentice-Hall, Englewood Cliffs, N.J., 1975).
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16. R. Campbell, *Cortex*, in press; M. Rappeport and M. Friendly, paper presented to the Canadian Psychological Association, Ottawa, 6 to 9 June 1978.
17. P. Ekman, J. C. Hager, W. V. Friesen, in preparation.
18. R. Campbell, *Cortex* **14**, 327 327 (1978). [Campbell has informed us (personal communication, 1 February 1980) that she has not questioned her data concerning asymmetries of posed smiles. "It seems to me . . . that the asymmetry of a posed smile is a real effect, but it is small compared with the asymmetry of a relaxed face."] W. Heller, unpublished manuscript; J. Rosen, D. K. Zeidler, in preparation; Borod and Caron and Rubin and Rubin (*19*). In related areas of research, Moscovitch and Olds (*20*) found more spontaneous unilateral facial gestures on the left than on the right side of the face during speech. Schwartz *et al.* (*21*) found asymmetry in facial electromyographic activity as a function of emotional conditions.
19. J. C. Borod and H. S. Caron, paper presented to the International Neuropsychological Society, New York, 31 January to 3 February 1979; R. Rubin and N. Rubin, *Neuropsychologia*, in press.
20. M. Moscovitch and J. Olds, paper presented to the International Neuropsychological Society, Amsterdam, Netherlands, 4 to 7 June 1979.
21. G. E. Schwartz, G. L. Ahern, S. Brown, *Psychophysiology* **16**, 561 (1979).
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Neurobiology of Amnesia

Gold and Sternberg have recently suggested that a common neurobiological mechanism may underlie many examples of retrograde amnesia in experimental animals (*1*). This conclusion was based on their report that phenoxybenzamine (PBZ), an α-adrenergic antagonist, attenuated the amnesia produced by several different treatments: electrical stimulation of frontal cortex or amygdala, injection of pentylenetetrazol, diethyldithiocarbamate, or an inhibitor of brain protein synthesis cycloheximide (CXM). They suggested that formation of memory may depend in part on brain catecholamine concentrations and that amnesia in many if not all cases is related to effects on catecholamines.

This conclusion seems difficult to reconcile with previous research on amnesia and the biology of memory. In particular, a large body of evidence has suggested that brain protein synthesis during or shortly after training is required for the formation of long-term memory (*2*). If protein synthesis is required for the development of long-term memory, it is difficult to understand why an adrenergic blocking agent should reverse the amnesic effects of protein synthesis inhibition. Accordingly, we have reevaluated the report that PBZ can attenuate the amnesia produced by CXM.

In the original study (*1*), memory was assessed with the passive avoidance task—that is, retention was measured by the time taken to enter an area where footshock had previously been delivered. In this task, drugs can change response latencies for many reasons (*3*). Thus, if combined treatment of PBZ and CXM made animals ill, then response latencies at retest might be high for animals given both drugs. This result would give the appearance of improved retention but could more appropriately be explained as sickness. We have replicated the original findings of Gold and Sternberg, who tested mice 1 day after training (Table 1). We also tested mice 7 days after training, after the general health of the mice had had time to recover. In this case, however, the impairment in retention produced by CXM was not "attenuated" by PBZ (Table 1). Finally, we trained mice for 20 trials in the Deutsch carousel, an automated discrimination training apparatus in which the response measure (a choice between two objects) need not be confounded by changes in locomotor activity or by illness (*4*). When 20 retention trials were given the next day, we found that PBZ did not attenuate the amnesic effect of CXM (Table 1).

Accordingly, the effects of PBZ on CXM-produced amnesia seem best interpreted as illness or some other temporary effect caused by the injection of both PBZ and CXM. A variety of known side effects of inhibitors of brain protein synthesis have been evaluated and have been dissociated from their amnesic effects (*2*). In particular, the possibility that CXM impairs memory by disrupting catecholamine metabolism has been re-

Table 1. Retention performance of mice as affected by saline, phenoxybenzamine (PBZ) (2 mg per kilogram of body weight), cycloheximide (CXM) (160 mg/kg), or a combination of PBZ and CXM. For passive avoidance training, the first drug was given 30 minutes before training and the second immediately after training. Discrimination training was the same except that the second drug was given 10 minutes before training. The number of mice per group is indicated in the headings. Mann-Whitney *U* tests were used to evaluate passive avoidance differences; *t*-tests were used for discrimination data ($\alpha = .05$).

Group	Passive avoidance latency (median sec)		Correct discriminations ($\bar{X} \pm$ S.E.M.) ($N = 18$)
	Retention on day 1 ($N = 45$)	Retention on day 7 ($N = 48$)	
1. Saline-saline	283*	215*	11.2 ± 0.8*
2. Saline-CXM	104†	56.5†	9.1 ± 0.6†
3. PBZ-saline	364*	156*	11.8 ± 0.7*
4. PBZ-CXM	245*	78.5†	8.9 ± 0.6†

*Significantly different from group 2. †Significantly different from group 1.

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