serotonin ($t = 4.51$, d.f. $= 4$, $P < .02$). Serotonin was also strongly inhibitory in animals first treated with disulfiram and DEDTC (Table 1; serotonin mean compared with inhibitor drug mean: $t = 2.96$, d.f. $= 8$, $P < .02$).

These experiments show that inhibition of norepinephrine biosynthesis by disulfiram and DEDTC suppresses self-stimulation. Because central administration of norepinephrine selectively reverses the suppression, we conclude that disulfiram and DEDTC produce this effect by their inhibitory action on dopamine-β-hydroxylase and the consequent depletion of norepinephrine, and not by some other action unrelated to the metabolism of norepinephrine (*10*). Furthermore, we can rule out an important role for serotonin and dopamine in our experiments. Neither substance is depleted after disulfiram or DEDTC (*11*), and neither is capable of reversing the effects of the drugs.

According to recent models of noradrenergic function (*12*), norepinephrine in the nerve ending is contained in two pools—a small functional pool and a larger, essentially nonfunctional, reserve pool. Because the norepinephrine in the reserve pool does not transfer readily to the functional pool, noradrenergic transmission probably depends primarily on the synthesis *de novo* of norepinephrine in the functional pool. If so, inhibition of norepinephrine biosynthesis would cause noradrenergic transmission to fail after the small reserve in the functional pool was exhausted. The rapid action of centrally administered DEDTC in our experiments suggests that, in the case of self-stimulation, the reserve in the functional pool can be exhausted in a few minutes.

In animals treated with disulfiram and DEDTC, the rapid reinstatement of suppressed behavior after intraventricular administration of norepinephrine is probably due to replenishment of depleted functional pools, and not to other possible actions, such as direct combination with noradrenergic receptors. These other actions in fact appear to suppress, rather than to facilitate, self-stimulation. This conclusion is based on our observation that, in untreated rats, the 5-μg dose of norepinephrine caused mild suppression of self-stimulation. Since the functional pools are intact in untreated animals, the exogenous norepinephrine cannot act by replenishment, and therefore must suppress self-stimulation by some other means (*13*). Mild suppression similarly must be exerted on the behavior of the disulfiram-and DEDTC-treated animals, but presumably it is obscured by the strong facilitating effect of replenishment (*14*).

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13. Possibly, self-stimulation is influenced by more than one system of noradrenergic synapses in the brain; that is, in addition to the behaviorally facilitatory system, there also may be systems that inhibit behavior. The suppressive effects of hypothalamic administration of norepinephrine in the self-injection experiments of Olds *et al.* (*5*) suggest that these hypothetical, behaviorally suppressive synapses have a diencephalic location. On the other hand, the noradrenergic synapses that facilitate behavior probably are located mainly in the forebrain [L. Stein, in *Reinforcement*, J. Tapp, Ed. (Academic Press, New York, in press)].
14. Further support for the replenishment idea is provided by the observation that intraventricular norepinephrine fails to reverse the behavior-suppressant effects of reserpine [E. R. John, B. M. Wenzel, R. D. Tschirgi, *J. Pharmacol. Exp. Therap.* **123**, 193 (1958)]. Such failure may be due to the fact that reserpine impairs the capacity of the noradrenergic neuron to take up and bind norepinephrine [M. Holzbauer and M. Vogt, *J. Neurochem.* **1**, 8 (1956); J. Glowinski and J. Axelrod, *J. Pharmacol. Exp. Therap.* **149**, 43 (1965)], and thereby prevents the replenishment of the functional pools.
15. We thank A. T. Shropshire for excellent technical assistance and Ayerst Laboratories, Inc. for donating the disulfiram.

29 August 1968; revised 19 November 1968 ■

Steady Potential Correlates of Positive Reinforcement: Reward Contingent Positive Variation

Abstract. *A positive reinforcement with food produced high-voltage bursts of alpha activity over the posterior marginal gyrus in a cat deprived of food and water. This synchronization was always associated with a large (180 to 300 microvolt), positive steady potential shift comparable to that occurring during the onset of sleep. Since this shift was contingent upon the relative appropriateness and desirability of food reward, it was termed reward contingent positive variation.*

In cats deprived of food, click or flash stimuli reinforced with food produce either negative or positive epicortical steady potential shifts with reference to the skull. Their magnitude diminishes as a function of the volume of food eaten. Hence, it was suggested that they reflect the degree of drive and motivation (*1*). A negative steady potential shift maximum at the vertex occurs in normal human subjects whenever a conditional stimulus is followed by an unconditional one that is expected to involve an action or decision. Such steady potential shifts return to the base line at the instant that the response is performed. Since the appearance of this steady potential is contingent on the significance of an association of unconditional stimulus, it was termed "contingent negative variation." Later it was termed "expectancy wave" since its magnitude is a function of the probability of occurrence of the unconditional stimulus (*2*). In cats deprived of food which were trained to press a lever for milk rewards, the electrocorticogram (ECoG) over the parietooccipital region shows dramatic fluctuations from desynchronized to highly synchronized patterns

(180 to 200 μv; 6 to 9 cycle/sec) after the presentation and during the consumption of food. This burst of alpha activity was termed postreinforcement synchronization (PRS) by Clemente *et al.* (*3*); PRS activity depends on the appropriateness and desirability of food reward (*3–6*). A striking similarity between the PRS patterns and those of sleep onset (*5–7*) and between concomitant changes in patterns of averaged auditory (*6*), somatosensory (*8*), and photic responses (*9*) recorded from the cortex during bursts of PRS and sleep led to the suggestion that PRS results from a transient inhibition of the reticular activating system. We have studied the time course of PRS and steady potential shifts during instrumentally conditioned appetitive behavior in the cat. Both phenomena were compared to those that occur during the onset of sleep in a satiated subject.

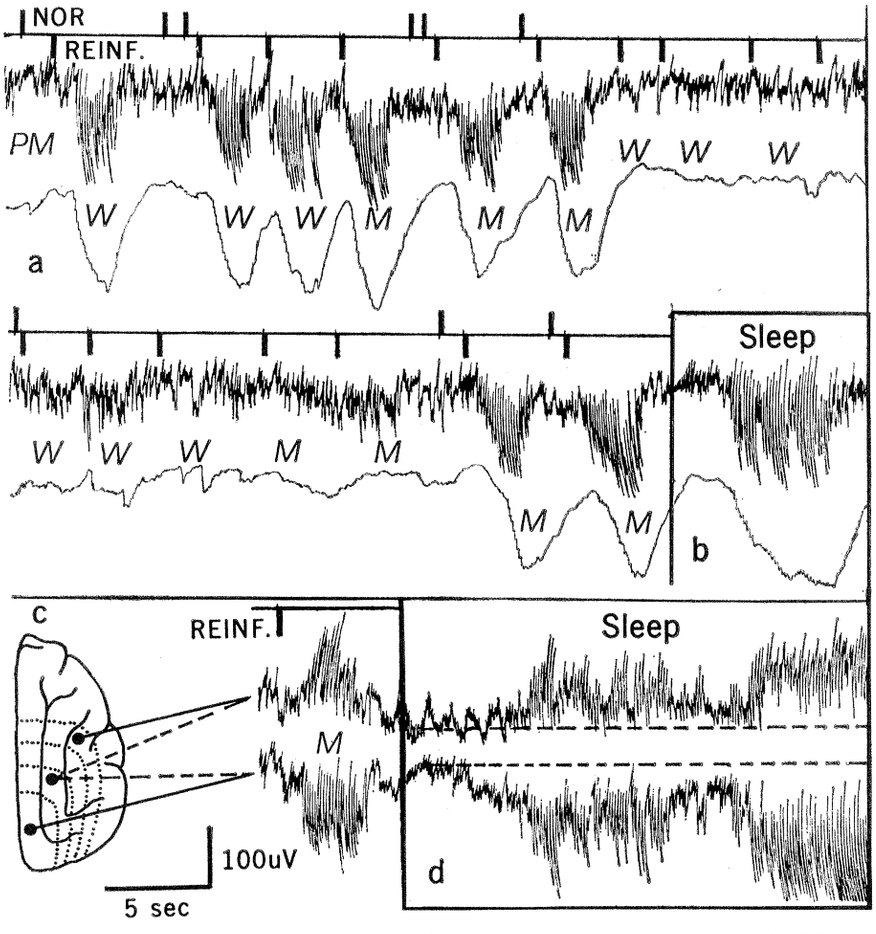

Fig. 1. (a) The effect of reduction in quality of reward on the occurrence of PRS and RCPV. Upper curve represents bar press potentials; *NOR*, nonreinforced; *REINF*, reinforced lever press; the remaining two curves are d-c recordings from the posterior marginal gyrus (*PM*) with reference to the occipital crest. The upper record is filtered to half amplitude response at 15 cycle/sec, and the lower one at 3 cycle/sec. In all recordings the positivity with regard to the reference electrode is downward. Before the beginning of this continuous record the subject had already received ten water rewards (*W*) which resulted in a full development of PRS and RCPV. These responses were not affected by substitution of milk (*M*) for water. Subsequent substitution of water for milk completely abolished PRS and RCPV, although the subject consumed the rewards. Note also a sustained negative steady potential shift during reintroduction of water. Subsequent reintroduction of milk rewards restored the PRS and RCPV. (b) A burst of alpha activity and associated positive steady potential shift during the onset of sleep in the same satiated subject. The base line was moved upward since there was an approximate 100 μv positive steady potential shift after the subject stopped to press the lever. (c) A simultaneous recording of RCPV from the posterior marginal and anterior ectosylvian gyri with reference to the medial suprasylvian gyrus. The dotted lines in the diagram of the cat's cortex represent an approximate distribution of the isopotential lines around the positive posterior marginal focus. They explain the mirror effect in RCPV recording. (d) In the same leads two bursts of alpha activity and associated steady potential shifts during the onset of sleep. Note the cumulative effect on the development of a strong positive focus of about 300 μv over the posterior marginal gyrus (measured with reference to the anterior ectosylvian gyrus).

Ninety experiments were carried out in six adult cats, of either sex, trained to press a lever for 0.5 ml of milk presented on a schedule such that pressing the lever produced the reward aperiodically, averaging once every 12 seconds. Cats were anesthetized with pentobarbital; in each cat, four to nine epidural, nonpolarizable electrodes were implanted over frontal and parietooccipital cortex. Two reference electrodes were used; one in the bone marrow over the frontal sinus, and another in the occipital crest. Electrodes (1 mm in diameter) were formed by compressing AgCl with pure Ag under vacuum (*10*). The characteristics of a pair of electrodes, 1 cm apart in physiological saline were as follows: resistance, 1000 ohms, measured with 10 cycle/sec impulses; phase angle, $-10°$; potential difference, less than 1 mv; cumulative drift, less than 1 μv/minute. The electrodes were connected to a miniature socket and fixed to the skull with dental cement. Low noise cable (*11*) was used to feed the signal into a Grass polygraph equipped with model 5P1 low level d-c amplifiers. In ten experiments, responses to substitution of water for milk rewards and vice versa were measured by feeding the d-c signal from a driver amplifier into a 5U-2 d-c integrator preamplifier coupled with a Grass model U1-1 unit integrator. Its output was in turn fed into a Grass d-c driver amplifier and written out on a separate channel. The integration system was adjusted to produce a 20-mm pen deflection in response to 100 μv positive steady potential shift lasting 1 second. This value was arbitrarily accepted as one unit of reward contingent positive variation (RCPV) in response to 0.5 ml of milk.

All subjects deprived of food and water for 23 hours when placed in the test cage displayed high-frequency (18 to 24 cycle/sec), low-voltage (20 to 30 μv) ECoG activity in frontal (anterior and posterior sigmoid, anterior marginal gyri) and parietooccipital leads (medial and posterior suprasylvian, anterior, medial, and posterior ectosylvian; medial and posterior marginal gyri). During the unreinforced and prior to reinforced lever pressing, the ECoG remained desynchronized, and the steady potential base line showed little or no significant fluctuations. However, after five to ten reinforcements, characteristic bursts of large amplitude (180 to 200 μv) alpha activity of 6 to

9 cycle/sec occurred, that is, a typical PRS gradually developed over the parietooccipital region during consumption of reward. During PRS the frontal cortex remained desynchronized. Simultaneously with PRS bursts, large positive steady potential shifts, that is, RCPV of 150 to 300 μv, occurred over the same cortical region, recorded with reference to either frontal sinus or occipital crest. A simultaneous monitoring of PRS and RCPV over posterior marginal gyrus with two separate d-c channels, one set for half-amplitude response at 15 cycle/sec, and the other at 3 cycle/sec, showed that RCPV resulted from a temporal summation of single and relatively small 10 to 30 μv positive potentials carried by each alpha wave (Fig. 1a). Thus, each RCPV response represented a faithful envelop of a PRS burst. After consumption of reward both PRS and RCPV disappeared, and a large negative steady potential shift developed. It reached the negative peak just prior or during the lever press. Both PRS and RCPV occurred only in an illuminated environment, although all our subjects were habituated to perform in completely darkened cages, and the rates of lever pressing and food consumption in a darkened or an illuminated cage were not significantly different. The ECoG remained desynchronized, and the steady potential base line showed a sustained negative shift, when novel environmental stimuli were introduced that did not disrupt the subject's performance (for example, knocking the cage, or blowing fish odor or cigarette smoke into the cage through the ventilation system). A reduction in quality of reward markedly affected both PRS and RCPV (Figs. 1a and 2). When water was introduced as reward at the beginning of an experimental session, large PRS and RCPV responses usually developed after five to ten reinforcements. With Student's *t*-test, no differences were found between the average RCPV responses induced by milk and water. A substitution of milk for water during the experiment had no significant effect. However, when water was substituted for milk during the same experiment, there was a complete abolishment or a significant reduction of both PRS and RCPV responses when compared to experiments in which water or milk was used initially ($P < .001$) although the rewards were consumed. Reintroduction of milk rewards promptly restored both responses.

Both PRS and RCPV are restricted to the parietooccipital region. The maximum voltage (150 to 280 μv) of RCPV developed bilaterally over the posterior marginal gyri with reference to frontal sinus, frontal cortex, or occipital crest. The frontal sinus, however, was a poor reference since it picked up the electroretinogram. The isopotential lines, whose approximate distribution (in about 50 μv steps) was based on exploration of 16 various electrode locations, ran bilaterally in a concentric manner with regard to the posterior marginal foci (Fig. 1c). The RCPV gradually decreased in positivity if the electrodes were located at increasing distances from the focus in the anterior, lateral, and posterior direction. A simultaneous recording from the posterior marginal and anterior ectosylvian gyri with reference to an electrode located over the medial suprasylvian gyrus produced a mirror reversal effect in RCPV phenomena, since the reference became less positive than the posterior marginal gyrus, but it still became more positive than the anterior ectosylvian gyrus, during a process of a presumable "irradiation" of RCPV responses from their posterior marginal focus (Fig. 1c). In satiated subjects during the onset of ECoG and behavioral sleep, similar large amplitude bursts of alpha activity associated with large (180 to 300 μv) positive shifts occurred over the same cortical region (Fig. 1, b and d). These shifts produced a cumulative effect on the development of relatively stable 200 to 300 μv positive foci over the posterior marginal gyri. Simultaneously, the adjacent parietooccipital and frontal cortex showed a gradually increasing positive steady potential shift of about 100 to 200 μv, and displayed a synchronized ECoG activity. Finally, delta waves developed predominantly over the parietooccipital region. Immediately after the establishment of delta patterns the average positivity over the posterior marginal foci (measured in 1 second intervals for 1 minute) was significantly smaller than that generated by several consecutive bursts of alpha activity during the onset of sleep ($P < .01$). Mild environmental stimuli (for example, gentle knocking on the cage) introduced during the pattern of a delta-wave sleep triggered a burst of alpha activity over the posterior marginal foci similar to that occurring during the onset of sleep. This resulted in a transient but significant enhancement of the positivity over the posterior marginal foci as if a homeostatic mechanism set to maintain sleep was activated. However, strong novel stimuli abolished the posterior marginal positive sleep foci, woke up the subject, and produced a diffuse negative steady potential shift of about 200 to 500 μv associated with ECoG desynchronization. Similar negative steady potential shifts and ECoG patterns were observed upon transition from slow-wave sleep to paradoxical, rapid eye movement sleep. The last two observations are in agreement with other reports (*12*).

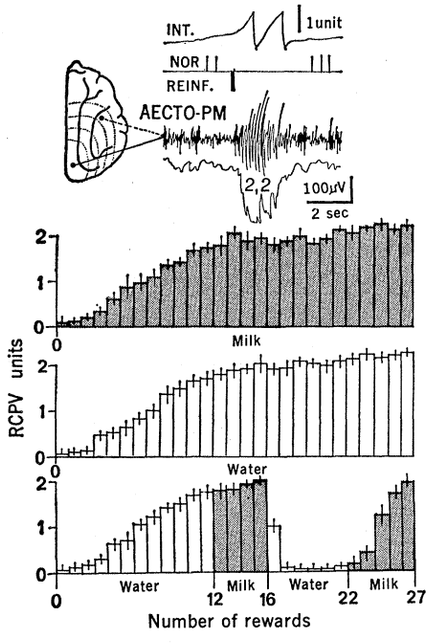

Fig. 2. The manipulation of RCPV [recorded from the anterior marginal gyrus (*PM*) with reference to the anterior ectosylvian gyrus (*AECTO*)] with water and milk rewards. Ordinate, average changes in RCPV expressed in units with standard errors; abscissa, number of reinforced lever presses. Each value is an average of four experiments performed in one subject. (Top) Development of RCPV during the first 27 milk reinforcements; (middle) the same when water was used as reinforcer; (bottom) the experiment started with water producing a well-developed RCPV. After 12 rewards the substitution of milk for water did not produce any significant changes. Subsequent substitution of water for milk rewards abolished RCPV. Reintroduction of milk rewards restored RCPV. The upper ECoG insert: *INT*, integrated RCPV; *NOR*, nonreinforced lever press; *REINF*, reinforced lever press. The upper of the two ECoG recordings is a-c and the lower one is d-c, filtered to half amplitude response at 3 cycle/sec. Positivity is downward with reference to *AECTO*.

The reticular activating system was implicated in generating the negative epicortical steady potential (*12*). Experiments in which lesions were made suggest that PRS depends on the integrity

of the internal inhibitory (synchronizing) system located in the basal forebrain (*5*). A cholinergic link of muscarinic type was postulated between this system and the reticular activating system (*13*), and between the latter and cortical neurons (*14*). Centrally acting anticholinergic drugs, scopolamine and atropine (but not peripherally acting methyl-scopolamine) abolished and physostigmine (a cholinesterase inhibitor) restored both PRS and RCPV phenomena (*15*). All these observations and our results are compatible with the view that RCPV represents a steady potential correlate of a Pavlovian active internal inhibitory process.

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16. Supported by PHS grant NB 06385 and PHS training grant GM 81-09 to J.L.Y. and J.T.H. We thank Miss S. L. Allen for her assistance.

9 September 1968; revised 8 November 1968

Old Faithful: A Physical Model

Since 1938 the Rangers at Yellowstone National Park have used the duration of eruption of Old Faithful to predict the time interval between eruptions. The relation, displayed by a graph on the wall of the Old Faithful Ranger Station, was established by the U.S. National Park Service, reported by Rinehart (*1*), and rediscovered by Geis (*2*). Using the above relations and Rinehart's (*1*) seismic data, Geis (*3*) postulated a physical model for Old Faithful that is similar in many respects to Bunsen's discredited geyser model (*4*). Recent work of White (*5*) showed that (i) large voids and geyser tubes are effects rather than causes of geyser action; (ii) most of the water erupted from geysers comes from fractures and porous and permeable rock deep underground, rather than from large chambers, which are not the source indicated by research drilling; (iii) hot water predominantly of meteoric origin circulates to depths of a few thousand meters underground where it is heated to temperatures far above the surface boiling point, and this heated water, rising in a huge convection system, in turn heats the rock in the upper several hundred feet of the system, and carries with it all the energy required for geyser action; and (iv) after a geyser eruption has occurred, the local underground rock is left relatively chilled due to extraction of heat from the system as water flashed to steam.

Geis (*3*) proposes that the underground configuration of Old Faithful is in the shape of a "U" with one end open to the surface and the other opening into a single closed underground chamber which generally is completely emptied during an eruption but sometimes is incompletely emptied. Geis further states: "An eruption would take place when the U portion of the cavity was sufficiently full to splash a quantity of water over into the hot, *dry* [my italics], back half of the cavity. The water would immediately flash boil to steam, forcing the water out of the U section of the cavity" (*3*).

If this model is correct the temperature in the cavity will immediately come to, and remain fixed at, the boiling point of water as soon as liquid is splashed into the cavity. If this boiling temperature is kept constant during flash boiling, and if the cavity is spherical, then the rate of heat flow into the cavity is given by the formula derived by Ingersoll, Zobell, and Ingersoll (*6*):

$$q = 4\pi k R T_s \left[1 + \frac{R}{(\pi \alpha t)^{1/2}}\right] \quad (1)$$

where q is the rate of heat flow, k is the thermal conductivity, R is the radius of the cavity, T_s is the initial difference in temperature between the cavity (kept at constant temperature) and the surrounding rock, α is the thermal diffusivity, and t is the time. Integration of Eq. 1 in respect to time yields an equation giving the total heat Q flowing from the surrounding rock into the cavity for any time interval, t_1 to t_2. If we assume generously large values for T_s, equal to 10°C, and R, equal to 224 cm, and reasonable values for k and α (*7*), the amount of heat that could be supplied to the cavity to flash boil water during the 1st second after splashing would be about 4.46×10^6 cal and less than about half that amount during each succeeding second.

At the initiation of an eruption, Geis (*3*) demonstrates, the water level in the "dry cavity" side of the U is at the same level as that in the open end, so that the total gas pressure in the closed cavity must be equal to atmospheric pressure. Thus, water splashed into the "dry" cavity would start boiling at about 92°C, the average boiling point at the elevation of Old Faithful, the initial enthalpy of evaporation would be 544 cal/g, and the specific volume of the steam that formed would be about 2200 cm³/g. Therefore, the maximum volume of steam produced would be 0.22×10^7 cm³ in the first second. Even after 30 seconds of continuous "flash boiling," a maximum of only 1.2×10^7 cm³ of steam could be produced regardless of whether a large or small amount of water had splashed into the cavity. This is only about 25 percent of the volume of water that must be displaced. In actuality much less steam would form, because the pressure in the "sealed" cavity must increase as soon as liquid water started flashing to steam. This, in turn, would raise the boiling point of water and would decrease the term T_s in Eq. 1. The pressure in the cavity would be balanced by the weight per unit area of a column of water rising in the open end of the U. An increase of just 3 m in the head of water in the geyser tube