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AN OPPONENT-PROCESS THEORY OF MOTIVATION:

I. TEMPORAL DYNAMICS OF AFFECT[1]

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A new theory of motivation is described along with its applications to addiction and aversion. The theory assumes that many hedonic, affective, or emotional states are automatically opposed by central nervous system mechanisms which reduce the intensity of hedonic feelings, both pleasant and aversive. The opponent processes for most hedonic states are strengthened by use and are weakened by disuse. These simple assumptions lead to deductions of many known facts about acquired motivation. In addition, the theory suggests several new lines of research on motivation. It argues that the establishment of some types of acquired motivation does not depend on conditioning and is nonassociative in nature. The relationships between conditioning processes and postulated opponent processes are discussed. Finally, it is argued that the data on several types of acquired motivation, arising from either pleasurable or aversive stimulation, can be fruitfully reorganized and understood within the framework provided by the opponent-process model.

First, we describe the kind of phenomenon which has caught our attention. Two fictitious examples will suffice. In the first, a woman at work discovers a lump in her breast and immediately is terrified. She sits still, intermittently weeping, or she paces the floor. After a few hours, she slowly regains her composure, stops crying, and begins to work. At this point, she is still tense and disturbed, but no longer terrified and distracted. She manifests the symptoms usually associated with intense anxiety. While in this state she calls her doctor for an appointment. A few hours later she is in his office, still tense, still frightened: She is obviously a very unhappy woman. The doctor makes his examination. He then informs her that there is no possibility of cancer, that there is nothing to worry about, and that her problem is just a clogged sebaceous gland requiring no medical attention.

A few minutes later, the woman leaves the doctor's office, smiling, greeting strangers, and walking with an unusually buoyant stride. Her euphoric mood permeates all her activities as she resumes her normal duties. She exudes joy, which is not in character for her. A few hours later, however, she is working in her normal, perfunctory way. Her emotional expression is back to normal. She once more has the personality immediately recognizable by all of her friends. Gone is the euphoria, and there is no hint of the earlier terrifying experience of that day.

In the second example, a couple have just begun sexual foreplay, and it is quite pleasurable. After a few moments of a constant level of mutual stimulation, the pleasure decreases somewhat. Normally, this decline would elicit behavior calculated to increase the intensity of mutual stimulation and to maintain the high level of pleasure. Unfortunately, at that moment a telephone rings. One partner leaves and goes into another room to answer it, and the other partner lies alone in the bed. The aban-

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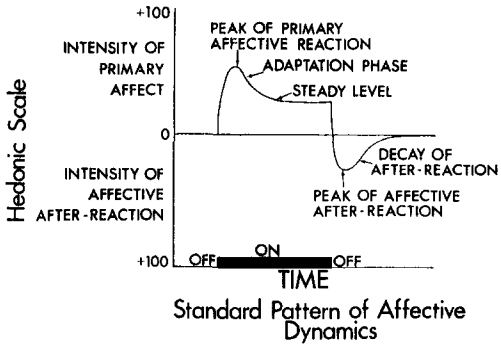

FIGURE 1. The standard pattern of affective dynamics, showing the five distinctive features: the peak of the primary affective reaction, the adaptation phase, the steady level, the peak of the affective after-reaction, and, finally, the decay of the after-reaction. (The heavy black bar represents the time during which the affect-arousing stimulus is present. The ordinate represents two hedonic scales, each departing from neutrality, one for the primary affect, the other for the affective after-reaction.)

doned partner experiences a quick decline of the pleasure, then becomes tense and irritated, and strongly craves a resumption of the sexual stimulation. Time goes by, however, and the other partner does not return. Finally, the abandoned partner gets out of bed, absentmindedly turns on the television set, and becomes absorbed in a news broadcast. Gone is the irritability and intense craving. There is no hint, in overt behavior, of the pleasurable sexual experience of a few minutes ago. A type of dispassionate normality now pervades.

We can distill from these two examples some important empirical features common to many hedonic, emotional, or affective experiences. First, following the sudden introduction of either a pleasurable or aversive stimulus, an affective or hedonic reaction begins and quickly rises to a peak. It then slowly declines to a steady level where it remains if the stimulus quality and intensity is maintained. Then, at the sudden termination of the stimulus, the affective reaction quickly disappears and gives way to a qualitatively very different type of affective reaction which reaches its own peak of intensity and then slowly disappears with time.

Figure 1 diagrams these changes in hedonic or affective state and illustrates what we call the *standard pattern of affective dynamics.* The pattern has five distinctive features: (*a*) the *peak* of the primary hedonic process or state, precipitated by stimulus onset; (*b*) a period of hedonic or affective *adaptation* during which the intensity of the hedonic state declines, even though stimulus intensity is maintained; (*c*) a *steady level* of the hedonic process which continues as long as stimulus intensity is maintained; (*d*) a *peak of affective after-reaction,* which quickly follows stimulus termination, and whose quality is hedonically very different from that of the primary hedonic state; and (*e*) finally, the afterstate decays and subsequently disappears.

This standard pattern describes both fictitious examples. In the first, the initial, primary hedonic or affective process was unpleasant and the after-reaction was pleasant. In the second, the primary process was pleasant and the after-reaction was unpleasant. The data of psychology contain literally dozens of examples of this kind. For brevity we will first describe some of the more interesting or important cases chosen from very different areas of psychological research. We can therefore demonstrate the great generality of the standard pattern of affective dynamics. Then we will describe a theoretical model for the underlying mechanism.

EXAMPLES OF EMPIRICAL PHENOMENA TO BE EXPLAINED

Table 1 presents seven examples of emotional, affective, hedonic, or motivational phenomena. Some are "behavioral," some "experiential." Some are experimental, others are observational, derived from common everyday experiences. Finally, some are precipitated by pleasant, and some by unpleasant stimuli.

Example 1 describes behavior changes seen in a dog subjected to intense aversive stimulation. The example draws on selected parts of studies reported by Katcher, Solomon, Turner, LoLordo, Overmier, and Rescorla (1969) and Church, LoLordo, Overmier, Solomon, and Turner (1966). A dog in a Pavlov harness was stimulated by sev-

TABLE 1

SELECTED EXAMPLES OF HEDONIC–AFFECTIVE PHENOMENA

Example	First few stimulations		After many stimulations	
	State A (input present)	State B (input gone)	State A′ (input present)	State B′ (input gone)
Dogs in Pavlov harness, 10-second shocks, gross behavior	terror, panic	stealth (subdued, cautious, inactive, hesitant)	unhappy (annoyed, anxious, afraid)	joy (euphoric, active, social), happy
Dogs in Pavlov harness, 10-second shocks, electrocardiograph responses	large cardiac acceleration	slow deceleration, small overshoot	small acceleration or none	quick deceleration, large overshoot
Epstein's parachutists, free fall, gross behavior, physiology	terror, autonomic nervous system arousal	stunned, stony-faced	tense, eager, expectant	exhilaration, jubilation
Opiate users, intravenous injection, moods and feelings	euphoria, rush, pleasure	craving, aversive withdrawal signs, short duration	loss of euphoria, normal feeling, relief	intense craving, abstinence agony, long duration
Dogs and M & Ms, gross behavior	pleasure, tail wagging, chewing	tenseness, motionless	—	—
Love, interpersonal stimulation, moods, feelings	ecstasy, excitement, happiness	loneliness	normal, comfortable, content	grief, separation syndrome, long duration
Imprinting, the attachment of creatures to their "mothers"	pleasure, cessation of fear, no distress	loneliness, distress cries, short duration	pleasure, no cries	loneliness, intense cries, long duration

eral 10-second shocks. The dog appeared to be terrified during the first few shocks. It screeched and thrashed about, its pupils dilated, its eyes bulged, its hair stood on end, its ears lay back, its tail curled between its legs. Expulsive defecation and urination, along with many other symptoms of intense autonomic nervous system activity, were seen. At this point, the dog was freed from the harness, it moved slowly about the room, appeared to be stealthy, hesitant, and unfriendly. Its "state" had suddenly changed from terror to stealthiness.

We now arbitrarily label the state during shock (the terror state or the peak of the primary reaction to shock) State "A." The stealthy state, right after shocks were terminated (the after-reaction), will be called State "B." In that way, we can temporarily ignore whether we have correctly labeled the states. We know that State A was not State B. Indeed, State A was very different from State B, judging by the many behavioral changes observed when the dog was suddenly released. Furthermore, State B gradually disappeared. In a few minutes, the dog appeared to be normal, like its previous, preshock, natural, self: active, alert, and socially responsive. When this happened, it was impossible to tell by looking at the dog that either State A or B had just transpired. The evidence was gone. The dog had progressed from "normalcy" to A to B, and back to "normalcy." This sequen-

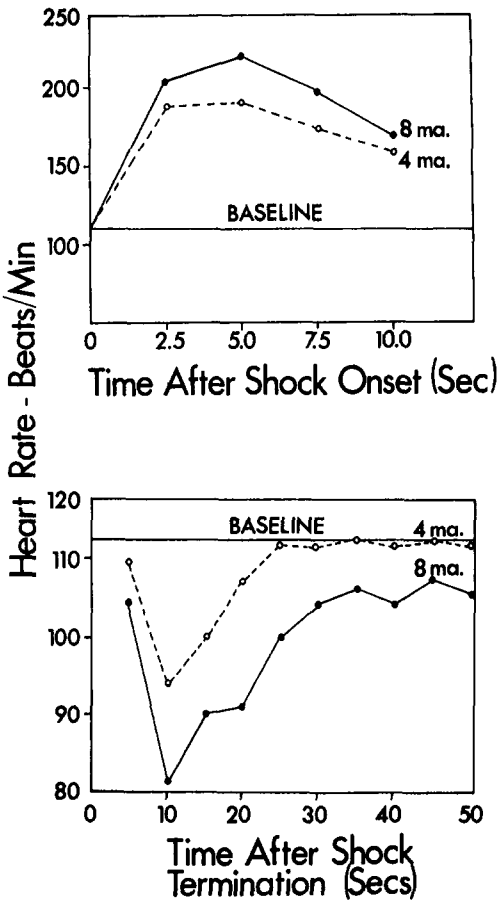

FIGURE 2. Heart rate changes as a function of shock onset, maintenance, and termination. (There is a decline following the initial peak reaction to shock onset. There is a deceleratory "overshoot" following shock termination, and then the heart rate slowly returns to baseline rate. Note that the eight-milliampere shock produces a bigger heart rate increase and a bigger deceleration than does the four-milliampere shock.) (Adapted from an article by Russell M. Church, Vincent LoLordo, J. Bruce Overmier, Richard L. Solomon, and Lucille H. Turner appearing in the August 1966 *Journal of Comparative and Physiological Psychology*. Copyrighted by the American Psychological Association, Inc., 1966.)

tial pattern will be seen in all of the examples given in Table 1. *It is, we believe, the basic pattern for the dynamics of affect.*

However, Example 1 has not yet been fully described. When the same dog was brought back for the same treatment day after day, its behavior gradually changed. During shocks, the signs of terror disappeared. Instead, the dog appeared pained, annoyed, or anxious, but not terrified. For example, it whined rather than shrieked, and showed no further urination, defecation, or struggling. Then, when released suddenly at the end of the session, the dog rushed about, jumped up on people, wagged its tail, in what we called at the time "a fit of joy." Finally, several minutes later, the dog was its normal self: friendly, but not racing about. Here again, the sequence was normal → State A → State B → normal, where State A accompanied the arousing input, and State B directly followed the sudden termination of that input, and then slowly died out.

We wish to emphasize that the qualitative and quantitative features of States A and B during *later* shocks were *not the same* as those for States A and B during the first few shocks. This pattern of changes, occurring as a consequence of repeated exposures to the stimulus input which causes A, also is, we believe, typical of the basic pattern of affective dynamics. Because the later States A and B were *not* identical to the earlier states, we label the later ones A′ and B′, respectively.

Look at Example 2 in Table 1. A dog was in an experiment in which its heartbeat was measured by an electrocardiograph during repeated shocks to its hind feet. The observations are from Church et al. (1966). During the first few shocks, there was a large cardiac acceleration; in some dogs it was an increase of 150 beats per minute. At shock termination, the rate suddenly decreased, and within 5 seconds it descended below the baseline rate. It often fell as low as 20 to 30 beats per minute below baseline rate. Then, it slowly "recovered" to baseline rate, over a period of as long as 30 to 60 seconds. The below-baseline excursion has been called "vagal overshoot." It is a well-studied phenomenon. Figure 2 is taken from Church et al. (1966) and demonstrates these dynamic events. The figure also suggests that there may be a relationship between intensity of the A state and the magnitude and duration of the B state. Now we can define baseline, State A, State B, and the return to baseline using an electro-

cardiograph measurement instead of gross observations of emotional behavior.

States A and B changed markedly after several sessions. Shock onset now caused little, if any, increase in heart rate. Any increase was momentary. The rate often decreased even while the shock was still on. However, when shock was suddenly terminated, the "overshoot" was much larger than it was on early shock trials. Heart rate in some individual cases descended to 50 to 60 beats per minute (sometimes more than 50 beats per minute below baseline rate), and recovery to baseline took as long as two to five minutes. As Katcher et al. (1969) put it, "The deceleratory heart-rate overshoot produced by stimulus termination shows shortened latencies . . . and greater magnitude over trials [p. 172]." Thus, States A and B have changed: A′ seems to be weaker than A, B′ stronger than B, and B′ longer lasting than B. But, as in the previous examples, we can still identify the sequence: baseline → A → B → baseline.

Example 3 comes from Epstein's (1967) report of physiological, emotional-expressive, and social reactions of parachutists. When parachutists make their first jump, they are often terrified, judging by telemetered autonomic responses and photographed facial expressions. When they land safely, they look stony-faced or stunned for several minutes, then gradually resume normal composure. After the parachutists have made several jumps and are experts, their responses are different. When jumping, they are no longer terrified. They may be anxious, tense, or even eager. After they land safely, they feel exuberant, exhilarated, and good. They like the feeling, and the mood lasts sometimes for hours. Such parachutists love to jump because of this after-feeling. Again, we see that the qualitative and quantitative attributes of States A and B have changed with the repetition of eliciting conditions. However, two very different states are still observable, and with each stimulation input, we can identify the sequence: baseline → A → B → baseline.

Example 4 represents states in opiate use (Jaffe, 1965; Maurer & Vogel, 1967). Early in a history of opiate use, the user experiences the "rush" (an intensely pleasurable feeling) directly after the opiate injection, followed by a period of less intense euphoria. Then, with further passage of time, the user suffers aversive, painful, and frightening somatic withdrawal symptoms, together with a feeling of craving. Here again, we see a baseline → A → B → baseline sequence. However, with opiates, B may last a long time, sometimes for several days.

After repeated dosages of opiates over several weeks, State A begins to weaken, and at the same time State B begins to intensify and takes longer to return to baseline. State A′ is called "normal" rather than euphoric. The rush is no longer experienced. Yet, State B′ is more physiologically disturbing than B was, and lasts much longer than did State B. The craving aspect of State B′ is now extremely intense, aversive, and enduring. It is called *abstinence agony*. It can be months before B′ returns to baseline. Perhaps it never really does. It is a ghastly experience.

The repeated use of some drugs results in the behavioral phenomenon of addiction. People find themselves craving a substance in which they previously had little interest. It is the most vivid instance of acquired motivation, because of its intensity and duration. It also provides a fruitful, empirical model for analyzing many kinds of acquired motivation. Indeed, we later show, with examples taken from love and affection, social attachment, and imprinting in birds, that addiction does not differ in principle from *any* acquired motivational system. We can easily describe opiate, alcohol, barbiturate, amphetamine, or cigarette addiction (see Solomon & Corbit, 1973) within the empirical framework of the analysis we have proposed. They all have four attributes: (*a*) The B′ state lasts a long time; (*b*) the B′ state is intensely aversive; (*c*) the elicitation of State A or A′ is effective in causing immediate removal of State B or B′; and (*d*) the user learns to employ the drug which elicits States A and A′ in order to get rid of State B or B′.

A lasting cycle of addiction will *not* arise, even though A and B are repeatedly experi-

enced, if the properties of affective response to a drug are such that B fades out to baseline very quickly. This is true because another dosage of the drug is never needed in order to get rid of the aversive B state. It quickly gets rid of itself. This is the case in Example 5, a common type of pleasurable situation. A laboratory dog is sitting dejectedly in a cage. It is suddenly handed one M & M candy. It wags its tail, moves about, chews, and swallows. This reflects State A. It smacks its lips a few times, curls its tongue across its lips, then becomes motionless and tense. It orients toward the experimenter and focuses its eyes on the experimenter's hand (the one which held the single M & M). Assume this to reflect State B. Then, if another M & M is not forthcoming, the B state dies out after about 15 to 30 seconds. The dog moves away, walks around the cage, sniffs here and there, begins to "ignore" the experimenter, then sits again. It has returned to its original state. Here again, termination of one hedonic event has precipitated another state not present prior to the onset of stimulation, and the second state disappeared by itself merely with the passage of time. In this case, A is pleasant, so we infer B to be qualitatively different, and probably *craving* is the best term for it. The so-called "peanut phenomenon" in humans is comparable. Once you start eating peanuts, it is hard to stop unless the cycle is interrupted for a period longer than the time required for the peanut craving, or B state, to die out. This has been called a mini-addiction.[3] Indeed, the case of the couple interrupted during mutual sexual stimulation, described in the Introduction, is certainly similar.

In Example 6, the pleasurable input generates a condition in which the B state typically lasts a lot longer than that for an M & M or a peanut, and so the favorable conditions for addiction are present. A boy and girl "fall in love." This State A is characterized by pleasurable excitement, frequent sexual feelings, a prevailing mood of ecstasy, happiness, and good feelings. When the lovers, whose multimodal mutual stimulation will cause State A, are separated from each other, they will feel lonely, sad, and depressed (State B). Even with anticipations of reunion (symbolic, conditioned arousers of State A) loneliness may prevail. Actual reunion will simultaneously erase B and reinstate A just as described in Attribute *c* of addiction to opiates.

After several years of repeated mutual stimulation, the qualitative and quantitative changes in A and B are a matter of public lore. State A′ is characterized (if all has gone well) as contentment, normalcy, and comfort. But State B′ is now potentially of high intensity and long duration. If it should occur, it is often called grief or, as Bowlby (1952) has described it in children, the "separation syndrome." It requires a lot of time for this B′ state to decay. The partners have become addicted to one another, and when separated they experience withdrawal symptoms. As in all the previous five examples, note that the sudden termination of the stimulus that arouses A or A′ leads to the occurrence of B or B′ before the eventual return to emotional baseline or normalcy. In this case, the termination of A′, even though A′ does not manifest itself as intensely as did A, is followed by a more powerful and much more protracted B′ state. This is the same pattern seen in all the previous examples, whether the A state is pleasurable or aversive.

Example 7 is one of imprinting. If we take the duckling as our subject, it shows the same patterns of affect revealed in opiate addiction. First, right after hatching there are very few distress cries. Indeed, the duckling may appear to be quite happy with its new environment. Then, if the duckling is exposed to a white, moving object, it looks intently at it. Also, if there were any distress vocalizations, they tend to disappear. However, if the moving object is then removed from view (Hoffman, 1968; Hoffman, Stratton, Newby, & Barrett, 1970), there is a burst of distress crying which may last for several minutes and then disappear. With successive presentations and removals of the imprinting stimulus, the frequency and intensity of distress crying will increase.

[3] This term was first suggested by Eliot Stellar at a cocktail party.

The efficacy of the presentation of the moving stimulus in eliminating the distress cries appears to be optimal at the outset. Then the duckling can be shaped to push a pole in order to present itself with the imprinting stimulus. At that point, of course, the duckling is exhibiting all criteria for addictive behavior. It is "hooked" on the imprinting object, the presence of which is a positive reinforcer and the absence of which is an aversive event.

Note that the ethological description of imprinting as the sudden establishment of "following behavior," *released* by an adequate imprinting stimulus, is utterly inappropriate in the light of our analysis. The moving object releases some affective State A, presumably an unconditioned, pleasant emotional reaction to the moving stimulus. The removal of the stimulus then precipitates State B, which is an aversive event. State B intensifies with repeated stimulations. Ducklings will then work on an avoidance schedule to prevent the disappearance of the imprinting object, just as the opiate addict will develop anticipatory behavior which prevents the occurrence of at least the more intense withdrawal symptoms.

EMPIRICAL GENERALIZATIONS

First, in all seven empirical examples, as well as in the fictitious ones in our Introduction, the sudden onset of some new stimulus aroused an affect or hedonic state not present prior to onset. The state terminated when the stimulus terminated. Then, a new state appeared, *qualitatively unlike* either the prestimulation state or the state produced by the onset and maintenance of the stimulus. Finally, this new poststimulus state persisted for a while and died out. The baseline state eventually returned. In none of the examples did the subject's affective state return directly to baseline upon cessation of stimulation. Baseline was regained via some new state which became manifest at stimulus termination, and then slowly died away.

Second, in some cases the states changed in their quality and intensity with successive, repeated stimulations. Whenever this occurred, the A states became weaker and the B states stronger and longer lasting.

These two phenomena, the dynamic hedonic response pattern, and its modification with repeated experience, were seen whether the A state was pleasurable or aversive.

THE EXPLANATORY MODEL

In our opinion, the simplest theoretical model that organizes these typical motivational phenomena (and countless other phenomena of innate and acquired motivation) is an opponent-process system. It was borrowed from previously developed accounts of sensory dynamics (Hurvich & Jameson, 1957). The primary *a* process for a given hedonic state is aroused by its adequate stimulus. We then imagine a single opponent loop generating the secondary *b* process and having an hedonic sign *opposite* to that of the state aroused by the input. The loop generating the *b* process is activated whenever any input evokes a sufficient hedonic consequence. The *b* process is sluggish, so it has a relatively long latency, recruits slowly, and dies out slowly. Finally, the *b* process is strengthened by use and weakened by disuse.

Because many formal properties of the patterns of both sensory and affective phenomena seem so similar, we must show at the outset that they are *not* identical and that the standard pattern of affective dynamics is *not* a direct consequence of the pattern of sensory dynamics. Otherwise, one could explain all hedonic or affective dynamics in terms of sensory events. For example, in the case of the woman who discovered the lump in her breast, one might argue that her steady-level anxiety state was less intense than her initial peak of terror at the discovery of the lump because of sensory adaptation; that is, the perceived presence of and magnitude of the lump decreased. We can refute such an interpretation, leaning heavily on the fact that the *time course* of typical sensory dynamics is of a completely different order of magnitude from that of the standard pattern of affective dynamics reflected in Figure 1 and Table 1. Sensory changes usually occur in

a matter of milliseconds, seconds, and minutes, whereas the emotional changes usually occur in minutes, hours, days, weeks, and months. There is, therefore, a theoretical

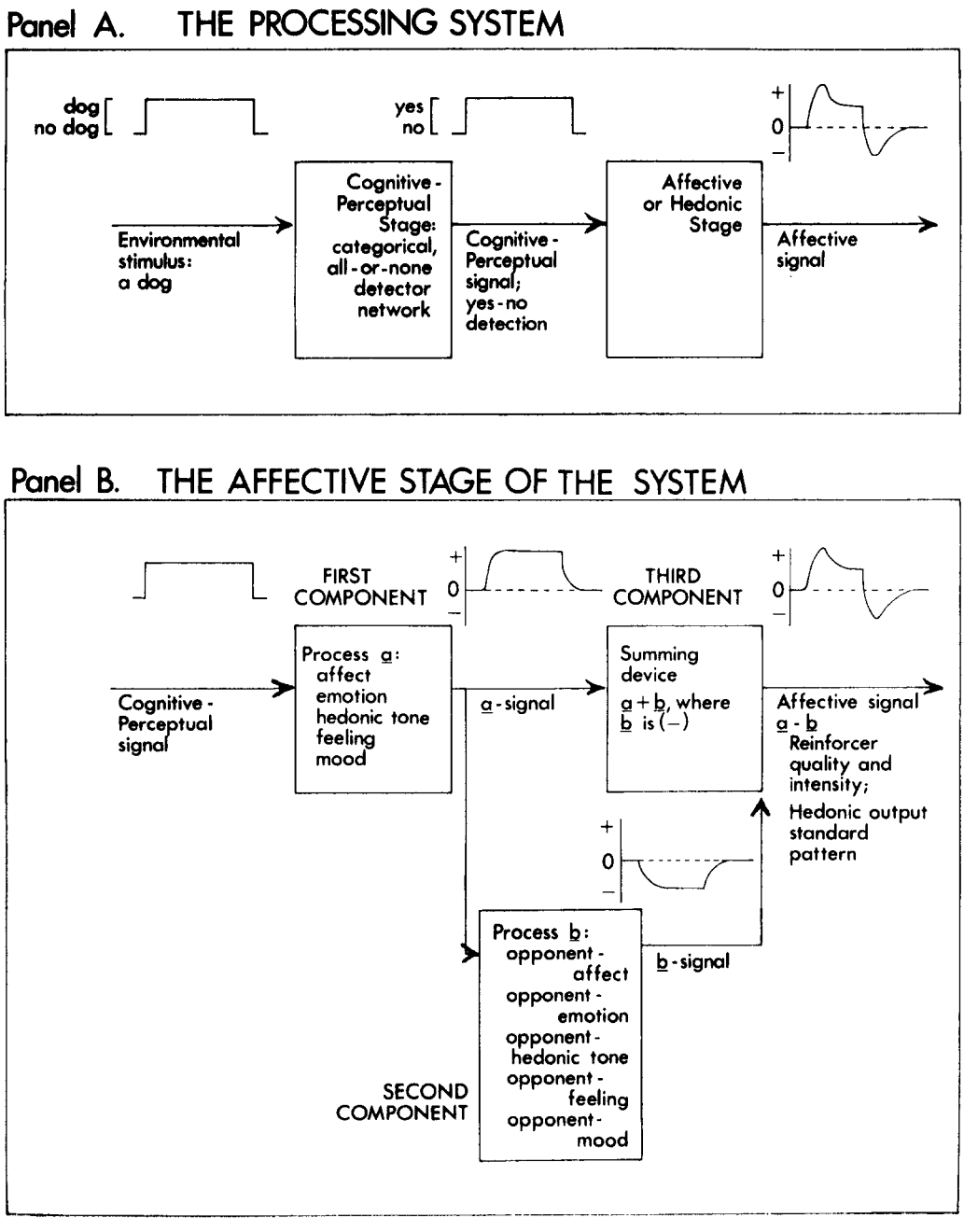

FIGURE 3. Panel A: The detection of an environmental stimulus by a cognitive-perceptual mechanism, and the arousal of an affective stage yielding an affective signal which shows the standard pattern of Figure 1. Panel B: An analysis of the three components of the affective mechanism. (In the first component the *a* process is aroused. The second component, the *b* process, is aroused via the arousal of *a*. Then the third component, a summing device, combines the *a* and *b* signals to generate the standard pattern of affective dynamics.)

necessity for a distinct opponent-process mechanism for affect and motivation independent of mechanisms for sensory dynamics.

Figure 3 illustrates the type of system that we have in mind. Panel A shows two stages of information processing, a cognitive-perceptual stage that converts the stimulus to an informational signal, and an affective or hedonic stage that converts the informational signal to an affective signal. The affective system in Figure 3 receives a square wave input, and follows it with a dynamic affective response of the standard pattern shown in Figure 1.

Perhaps an example will clarify the point of Figure 3. The sight of a dog is a fear-arousing stimulus for a cat. The dog represents a complex, multidimensional display, and the perception of the dog is categorical, all-or-none. The stimulus sequence is as follows: The dog enters the cat's environment, remains for a while, and then leaves. The cognitive-perceptual sequence is: The dog is detected, continues to be detected as long as it remains, and then ceases to be detected when it leaves. There is no adaptation (i.e., the dog does not become less doglike), nor is there any appreciable sensory after-reaction at stimulus removal (i.e., no negative [antidog?] afterimage). The affective sequence, in contrast, will show the primary reaction and after-reaction components: intense fear at first, subsiding to a steady level, and then, when the dog is gone, the appearance of another state, very different from the first. The after-reaction (relief?) then gradually dies out.

Figure 3, Panel A, shows that the affective system follows its square wave input with an output having the complex dynamic features of Figure 1. How can we account for this behavior? The affective system in Panel A is shown as a single stage. Now, we will open the "black box" for this stage and look inside. Our proposal for the mechanism responsible for affective dynamics is shown in Figure 3, Panel B. Here, the affective stage is analyzed into its three component parts. The cognitive-perceptual stage has acted as a categorical detector. The informational signal enters the affective system as input to the first component, the *a* process, which has a short time constant. The signal from the *a* process activates the second component or *b* process, which responds with a slow rise and a slow decay. The third component is a summing device that adds the *a* and *b* signals, and it generates as its output the affective signal, which shows the sequence of the peak primary reaction A, adaptation, steady level, after-reaction B, and decay of B. Thus, we see that the input from the perceptual-cognitive stage has an affective, hedonic side effect, Process *a*. When it does, the opponent loop is activated, calling into play the opponent process (which has a hedonic quality in some way opposite to, and very different from, that of Process *a*). The opponent process, which we call Process *b*, reduces the hedonic intensity of the state which the input initially aroused. When the perceptual-cognitive input ceases, the opponent process reveals itself as "pure" State B, because the *b* process takes a while to decay.

The opponent process is a *slave process*: It is activated indirectly via the activation of the *a* process. Presumably, the slave process has an evocation threshold, a latency, a recruitment or augmentation time, and a decay function, all characteristic of a given opponent-process system. We will also see later that the opponent process can, under proper conditions, be activated by events in memory, as a consequence of Pavlovian conditioning procedures.

The block diagram of the affect-control system in Figure 3 yields the temporal dynamics of affect shown in Figure 4, Panel A. There we first see a baseline state. Then the affect-arousing stimulus is presented and it stays on for 10 seconds. Next, it is suddenly terminated. This simple event sequence activates the underlying opponent processes. First, there is a quick rise of Process *a* to a peak intensity. Shortly afterward, there is a slow recruitment of Process *b*. When the stimulus is terminated, Process *a* quickly goes to zero, but Process *b*, having a sluggish decay property, perseverates and dies out slowly. The resultant manifest dynamics of affect are a conse-

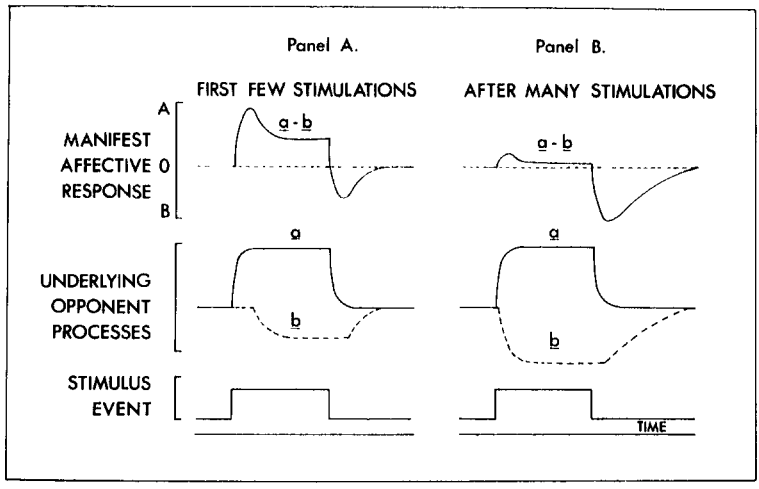

FIGURE 4. Panel A: The operation of the summing device for the first few stimulations. (The summation of the underlying opponent processes, a and b, yields the manifest affective response.) Panel B: The operation of the summing device after many repeated stimulations.

quence of subtracting the b process from the a process. The subtraction of the two quantities yields Manifest State A when $a > b$ and yields Manifest State B when $b > a$. The intensity of the manifest state is given by the quantity $|a - b|$.

When we subtract the underlying processes, a and b from each other, we obtain the manifest affective response pattern shown in Figure 5. Immediately after stimulus onset, a is large and b is zero, thus yielding a peak of State A. As b is slowly recruited, the quantity $|a - b|$ decreases, yielding a decline in the magnitude of State A. This is the adaptation phase shown in Figure 1, the standard pattern of affective

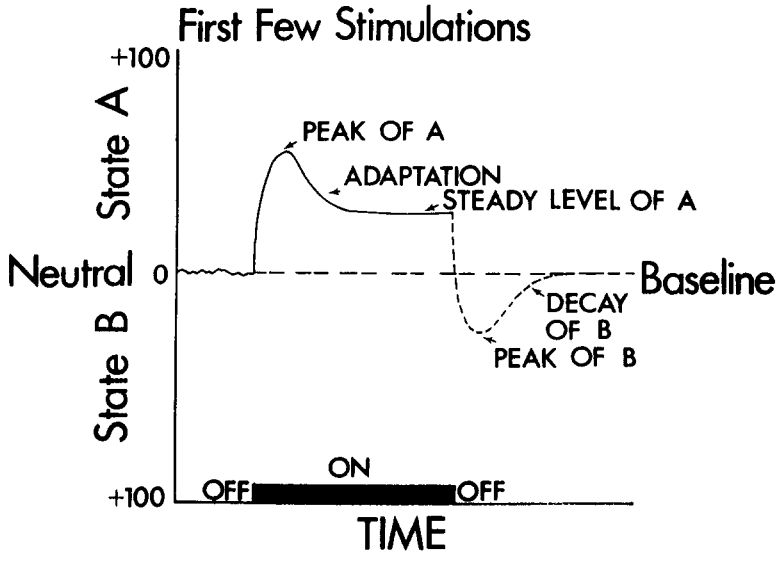

FIGURE 5. The manifest temporal dynamics generated by the opponent-process system during the first few stimulations. (The five features of the affective response are labeled.)

dynamics. When the *b* process reaches an asymptote, there will be a steady level of State A. Then, when the stimulus is terminated, the intensity of *a* goes quickly to zero, but the *b* process dies away slowly. At this moment, $b > a$, yielding the peak and subsequent decay of State B. At this point, State B is pure *b*.

The theory represented in Figures 3 and 4 gives a rough, qualitative account of all of the data of Table 1, for the first few stimulations only. First, it explains peaks of intensity of affect or hedonic quality at stimulus onset: The opponent process has not yet had enough time to get into action. So we see phenomena like terror, the rush, ecstasy, etc. Or, rate the painfulness of a 30-second shock. The peak painfulness is at onset. For example, see the peak of heart rate in Figure 2.

The other major event explained by the theory is the emergence of the *after-reaction,* postulated to be a function of the opponent process, which becomes manifest after the termination of stimulus input. This emergence is due to pure *b* perseverating in time after the *a* has quickly disappeared. The B state slowly decays, and baseline is eventually attained.

In each of the seven empirical examples of Table 1, the manifest B state was, in some unspecified way, related to A, but not the same as A. The model designates the relationship as *oppositeness.* But in what way can we say, for example, that loneliness is opposite to the pleasure produced by the presence of a loved one? Surely, they are hedonically opposed. This concept is built into the theoretical model. If A is pleasant, then B must be unpleasant. If A is a positive reinforcer, then B is a negative reinforcer. Other affective attributes of A and B remain an empirical question. The model tells us how to identify the attributes of oppositeness. All one has to do, for any given A state, is observe the attributes of affect which are revealed at the peak of B. Therefore, the model puts a constraint on what we call oppositeness. For example, the opposite of love must be the attributes of grief. To say that, "Hate is the opposite of love" is simply wrong, when at the death of a loved one, one experiences grief, not hate. The kinds of oppositeness which are generated by each A state are still, in many cases, waiting to be studied and named. Here is a vast, neglected area of investigation.

We postulated that the *b* process is a slave process. That means that at first it cannot be aroused directly by ordinary sensory inputs, but instead can arise only *indirectly* via the arousal of an *a* process (see Figure 3) and the subsequent activation of the opponent loop. This fits common sense. Try to imagine being grief-stricken without having loved someone. Try to imagine craving and abstinence agony without drug use. Try to imagine the exhilaration of the parachutist without any jump. On the other hand, we will point out later that it may be possible to arouse a *b* process directly by electrical or chemical stimulation of the brain, or to eliminate it by surgery, or to condition it by Pavlovian procedures. Initially, however, it is a slave process, inaccessible to direct environmental inputs, but indirectly arousable via hedonic and affective processes elicited by environmental inputs.

STRENGTHENING OF OPPONENT PROCESSES BY REPEATED STIMULATION

We have not yet explained the changes in hedonic dynamics brought about by repeated affect-arousing stimulations over a relatively long period of time. There are important differences between the A and B states on the left side of Table 1 as compared to the right side. Fortunately, one postulate brings order into the data: *The opponent process is strengthened through use and weakened through disuse,* but the primary affective process is *not* seriously affected by use. A *b* process will acquire more power if frequently elicited. It will show a shorter latency of response to *a*, a quicker rise, a higher asymptote, and a longer decay time. In contrast, an *a* process is a relatively stable, unconditioned reaction. This seems reasonable for a system which is designed to minimize deviations from affective neutrality. Why should an opponent process not act like a defensive or immunization pro-

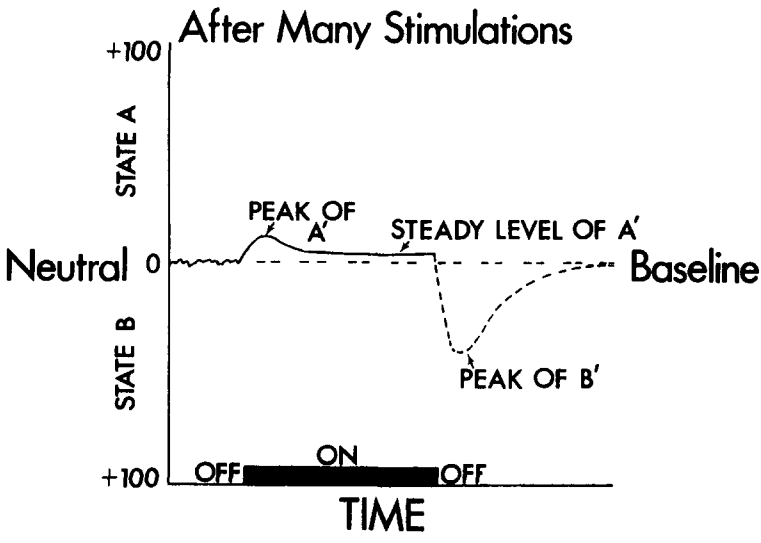

FIGURE 6. The manifest temporal dynamics generated by the opponent-process system after many repeated stimulations. (The major features of the modified pattern are labeled.)

cess, which produces antibodies more efficiently and in larger numbers in the face of repeated challenge? In the same vein, *disuse* should weaken a *b* process, and it should slowly return to its original magnitude whenever its *a* process has not occurred for a long time.

Figure 4, Panel B, shows how the opponent processes will interact after the *b* process has been strengthened by repeated use. A comparison of Panels A and B explains why the left-hand portion of Table 1, describing "early stimulation," differs from the right-hand portion of Table 1, describing "later stimulations."

The effect of repeated experiences on the hedonic response is confined to strengthening the *b* process. During later stimulations the *b* process increases at a faster rate at stimulus onset and reaches a greater asymptotic intensity. In addition, the *b* process requires a much longer time to decay after the later stimulus terminations. As a consequence of these changes in the *b* process, the resultant pattern of the affective reaction changes so that the peak of A is considerably attenuated, and the peak of B becomes much greater and longer lasting. Figure 4, Panel B, shows these simple algebraic summations of the *a* and *b* processes after many repeated stimulations.

Figure 6 shows the pattern of emotional dynamics after many repeated stimulations and should be contrasted with the pattern shown in Figure 5. Three phenomena are corollaries of the *use postulate*. First, the peak of A′ will be less intense because the latency of the *b* process is decreased and its intensity is increased. Second, the steady level of A′, $|a - b|$, during maintained stimulation will be close to baseline and perhaps even below it in some cases. Third, the peak of B′ should be intense and long lasting, compared to what it was during early stimulation (Figure 5).

The events in Figure 6 do *not* follow from a simple *affective contrast* model. If a principle of simple hedonic or affective contrast were operating, then the peak of B in Figure 5 should be greater in intensity than it is in Figure 6, because the intensity of A directly prior to stimulus termination is greater in Figure 5 than it is in Figure 6. However, if we run down the descriptive adjectives for States A, B, A′, and B′ in Table 1, even as loosely defined as they are, the opponent-process model works well, and a simple contrast model fails.

More convincingly, the objective data from the electrocardiograph experiments with dogs fit the opponent-process model. In Table 1 we saw the following:

A, large acceleration;
B, small overshoot, short duration;
A′, small or absent acceleration;
B′, large overshoot, longer lasting.

These findings fit the postulate that repetitions of affect-arousing stimulation, in this case shocks, will strengthen the opponent process which dampens the primary cardiac acceleration process. Furthermore, there are many signs of qualitative changes; e.g., signs of pleasure and euphoria appear in the period following termination of the shock session, although they were absent in earlier sessions. Our model cannot yet deal precisely with these qualitative changes which are produced by many repetitions of the same stimulus. A′ is less intense than A, and B′ is more intense and longer lasting than B. The model deduces these quantitative changes (Figure 4). In many cases, however, A′ is qualitatively different from A, even though they have the same hedonic sign. Similarly B′ and B are sometimes qualitatively different, even though they have the same hedonic sign. When the model is fully developed, it must contain a rationale for these qualitative changes.

The observations on the right-hand side of Table 1 also support the assumption that many repetitions of pleasures will strengthen their aversive opponent processes. Aversive states, manifesting themselves after the sudden termination of pleasurable inputs, become more intense with repeated experiences. Mild loneliness later becomes grief. Mild craving later becomes abstinence agony and intense craving. In addition, after many repetitions the steady level of pleasure produced by the continued presence of the pleasurable stimulus input has decreased. The confirmed opiate user experiences a "loss of euphoria," and the rush is gone. The pleasure-affective systems seem eventually to yield to opponent processes which keep departures from hedonic equilibrium relatively small. The aversive opponent process, when it is manifest, is more intense and longer lasting than it once was. So frequently repeated pleasure has its costs, psychologically, in an increased potentiality for displeasure.

Similarly, for A′ states aroused by aversive input, there is a "cost," but this cost is an increased potentiality for pleasure. Table 1 lists in the column under B′ the opponent affects for the aversive A′ states. Words like "joy," "exhilaration," and "good feeling" appear. These are the emotional costs of aversiveness. The model thus requires that any prolonged or repeated departures from hedonic or affective neutrality, regardless of hedonic sign, have a cost. Any significant departure from hedonic or affective neutrality should have correlates in increased autonomic and central nervous system activity aimed at reducing that departure. The cost of this activity will not only be psychological, but also will be physiological (metabolic, hormonal, and neural).

We are assuming that prolonged exercise of an opponent-process system, whether it be pleasurable or aversive, might cause physiological stress in the same sense that Selye (1950) uses the term stress. That is, many physiological resources might be required in order to keep the opponent process strong. If we follow Selye's argument, we would expect that such a constant demand might lead to the exhaustion of a particular overworked opponent-process system or to the debilitation of other defensive systems.

Thus we come to a major implication of our opponent-process model: There probably are stresses caused by pleasurable stimulation just as there are stresses caused by aversive stimulation. Therefore, there should be adaptational costs as a consequence of *both* kinds of stresses. Furthermore, if we look for them, we should find *diseases of adaptation for both,* due to the correlated, physiological side effects of long-duration often-elicited intense *b* processes.

In the past, psychologists have identified psychological stress with aversion, pain, and unpleasantness. Theories of mental disease, psychosomatic disease, and behavior disorders usually emphasize that aversiveness means stress, stress means aversiveness, and

both cause emotional disorders, psychosomatic illnesses, and behavioral malfunctions. Our opponent-process theory of motivation puts all this to question. From our point of view, stresses caused by aversive stimulation are only one half of the story. There should be emotional disorders, psychosomatic diseases, and behavior disorders caused by long-lasting, repeated, and intense *b* processes in general, whether these opponent processes are pleasurable or aversive.

Take the case of the parachutists. Their operant behavior, when B′ is strong, will be reinforced by the pleasurable experience of B′. But the cost of neutralizing the innate aversiveness of a fall through space might be high, both physiologically and emotionally, just as Epstein (1967) has postulated. Even so, one might imagine, if there were no other pleasurable sensory inputs elsewhere available to the individual, that he might put himself through repeated aversive stimulation in order to experience the pleasurable B′ state which would be both intense and lasting. From our point of view, this type of apparent *masochistic* behavior is not a mental disorder but is, rather, a reflection of the *normal* functioning of a healthy, automatic, affect-control system. Thus, also, from this point of view, there is nothing abnormal or strange about addiction. It is only a socially vivid example of the normal hedonic and motivational functionings of an efficiently operating affect-control system.

Motivational systems involving pleasurable A states and aversive A states are similar. In both cases the onset, maintenance, and termination of the stimulus results in a certain amount of pleasure and a certain amount of displeasure. They differ mainly in whether pleasure or displeasure comes first. In the case of the pleasurable A states, we can assume that the subsequent aversive B state functions as a drive that energizes the performance of operants, and that the pleasurable A states may positively reinforce these operants. Electrical self-stimulation of rewarding brain sites, chemical self-stimulation with opiates, and love relationships should work this way. In contrast, when the A state is aversive, and when the A state stimulation is absent, nothing functions as a drive to energize an operant upon which the A state is contingent. So we have the problem of how to get the behavior started. The B state for an aversive A state is not an energizer. Instead, it is a positive reinforcer. There is thus an important *asymmetry* between motivational systems for pleasurable and aversive A states. Some outside energizing influence is needed to get behavior started when the operant is followed by an aversive A state. Examples of such behaviors include "thrill-seeking" behaviors, such as parachute jumping, mountain climbing, automobile racing, etc., all of which involve an aversive component, followed by a pleasurable feeling of exhilaration. Why should one initiate an activity when its immediate effect is aversive, i.e., when a punishment contingency exists? Some competing outside influence, such as social pressure from peers, is required. However, after many repeated stimulations, such outside influences may not be needed. Because the aversive A state is then weak and the subsequent, positively reinforcing B state is strong, the A state will function as a positive reinforcer. We know that this can happen when a weak shock signals the onset of food for cats (Masserman, 1943). Note that an outside influence is not needed when the A state is pleasurable. These operants are energized by the aversive B state.

RELATION OF THE THEORY TO OTHER CONCEPTS

To the reader well versed in the history of theories of learning, two aspects of our model should now be apparent. First, the phenomena of acquired motivation produced merely by the repetition of affect-arousing stimuli are nonassociative in nature. For example, the person repeatedly dosed with morphine does not have to know anything and is not required to be subjected to Pavlovian stimulus contingencies, nor to contingencies between operants and outcomes, in order to develop an increasing tendency to suffer when the morphine is withdrawn. The model is therefore very different from previous theories of acquired motivation,

all of which have emphasized associational processes.

Second, the model, in relating its hypothetical mechanism to the phenomena of operant conditioning, explicitly assumes that operants are energized only by aversive states and that they can be reinforced either by the onset of pleasurable states or by the termination of aversive states. This is the case whether the pleasure or aversiveness comes from A states or B states. For example, the reinforcing effects of shock termination will not only be due to the elimination of the aversive A state but also, and perhaps more importantly, it will be due to the subsequent pleasurable B state. Woodworth and Schlosberg's (1954) concept of "safety," Mowrer's (1960) concept of "relief," and Denny's (1971) concept of "relaxation" are all emphasizing this concept of reinforcement in aversive situations.

The assumption that operants are energized only by aversive states fits most comfortably with the theoretical position taken by Hull (1943, 1952) and elaborated by Mowrer (1947), Miller (1948), Spence (1956), and Brown (1961). The assumption that operants can be reinforced by the onset of pleasurable states fits easily with the position taken by Young (1955) and by Pfaffman (1960). Of course, the assumption that operants are reinforced by the termination of aversive states is most like the drive-reduction position maintained especially by Hull (1943) and by Mowrer (1947). These assumptions are really not at issue for us. We take all three to be axiomatic, a point of departure, and we go on from there to spell out the dynamics of pleasurable and aversive states.

CONDITIONABILITY OF A STATES AND B STATES

The opponent-process theory would encompass an even larger array of data on acquired motivation if it could be safely assumed that A states, or B states, or both could be brought under the control of previously neutral stimuli as a consequence of experience. A Pavlovian conditioning procedure then would result in the establishment of conditioned stimuli which could evoke either state as a conditioned response.

Assume that we have a valid measurement of the intensity of affect produced by the onset and maintenance of a 10-second shock in dogs and that we can measure the opponent process when it reveals itself directly after shock termination. Now we repeatedly pair a conditioned stimulus (tone) with shock onsets. If a Pavlovian conditioning process is effective, then the conditioned stimulus would then become an A-state elicitor. We call such a conditioned stimulus a CS_A. (In the Pavlovian framework such a conditioned stimulus would be called a CS+.) If we present a test trial with CS_A, with no shock presented, we should see CS_A arouse a conditioned State A (or a weak relative). When CS_A is suddenly terminated, we should see State B appear, peak, and then decay to baseline in time. The hedonic recovery from such a test with CS_A alone should therefore appear to be *biphasic*.

Contrast this with what should occur if we establish a conditioned stimulus by pairing a signal with the peak of State B, which will occur directly following shock termination. That paradigm is Pavlovian *backward conditioning*: The conditioned stimulus always follows termination of the unconditioned stimulus closely in time. Such a conditioned stimulus should become a Pavlovian CS−, or in our terminology, a CS_B. It should be able to elicit a conditioned state very similar to State B. A test with CS_B (without a shock preceding it) should result in the conditioned State B appearing at the onset of CS_B. Then, after CS_B termination, State B should decay monotonically, and baseline should be reestablished. In other words, a monophasic recovery following CS_B termination should occur, whereas a biphasic recovery ought to occur following CS_A termination. Why a monophasic recovery for B? Because, if the *b* process is a slave process with respect to *a*, and yet the resultant states are also conditionable, then we ought to be able to arouse the *b* process directly by onset of a CS_B, but then, after CS_B has terminated, all State B can do is die away. But a CS_A, if successful in

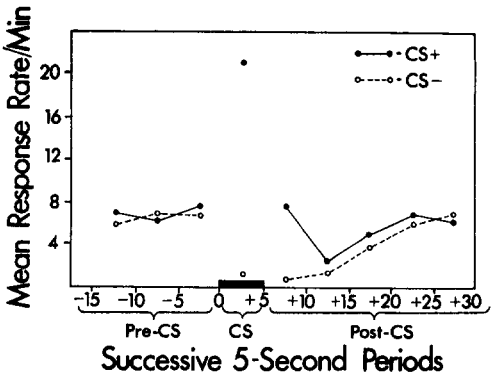

FIGURE 7. Data from Rescorla and LoLordo (1965). (The solid lines and dots show the biphasic response following termination of the fear-arousing conditioned stimulus [CS]. In this graph the CS+ is a CS_A. The dotted lines and open circles show the monotonic recovery following the termination of the fear-suppressing conditioned stimulus. In this graph the CS− is a CS_B.)

eliciting the conditioned *a* process, will also indirectly arouse its opponent process. Therefore, State B will be seen immediately after CS_A is suddenly terminated, yielding a biphasic recovery. But a peak of State A will never be seen after a CS_B is terminated: Only a return of State B to baseline will occur.

In commonsense terms, what the opponent-process theory is saying for the case of conditioned fear is peculiar. The sudden termination of a *danger* signal (CS_A) should cause the conditioned subject to act as though it had received a safety signal. But the sudden termination of a *safety* signal (CS_B) should *not* cause the conditioned subject to act as though it had received a danger signal. An analogous asymmetry should of course exist for appetitively conditioned stimuli, though we are not aware of the existence of experimental data on this point.

Some Pavlovian conditioning data fit these expectations very well for aversive stimulation. Rescorla and LoLordo (1965) trained dogs to avoid shocks on a Sidman nonsignalized avoidance schedule (shock–shock interval = 10 seconds, response–shock interval = 30 seconds). The avoidance response rate was used as a measure of fear intensity. When the dogs' avoidance response rate had stabilized, they were tested during avoidance performance with a CS_A which had previously been paired with shock onsets during separate Pavlovian fear-conditioning sessions. The onset of the CS_A during the shock-free tests resulted in a large increase in the avoidance response rate. This was interpreted to mean that the avoidance response was energized by the increment of conditioned fear caused by the presentation of the CS_A. The most interesting phenomenon to us is the fact that recovery from a CS_A test was biphasic. The avoidance response rate, when CS_A was terminated, dropped quickly below baseline, then recovered slowly to baseline in 30 to 45 seconds (see Figure 7, solid line with solid circles).

Rescorla and LoLordo (1965) used a discriminative conditioning feature, with a conditioned stimulus paired with a long shock-free intertrial interval. When the conditioned stimulus was tested during the dogs' avoidance performance, it suppressed the avoidance response rate. Then, when the conditioned stimulus was terminated, the rate slowly returned to baseline in 30 to 45 seconds (see Figure 7, dotted lines with hollow circles). The recovery from the conditioned stimulus was exactly what we should expect if the conditioned stimulus had been conditioned to the B state. Intuitively, one might think that the termination of the conditioned stimulus, which is, after all, a safety signal, ought to cause a sudden intensification of fear above the original fear baseline. This does not happen, and adds strength to the theoretical analysis we have presented. However, there is a serious problem in applying the model to these data on Pavlovian differential conditioning. The conditioned stimulus was always presented following an intertrial interval of 1½ minutes or longer. Therefore, by no stretch of the imagination could we consider the conditioned stimulus to have been regularly paired with the peak of B, because the peak of B would have occurred very shortly after the previous shock termination. How, then, can we manage this conceptual problem? If we are to stay within the confines of our model, we must

make two assumptions. First, after many conditioning trials during which hundreds of shocks have been sustained, the *b* process would be very strong and last a long time (perhaps for many minutes, or even for hours). Second, we must assume contemporaneous conditioning, an event–state conditioning process, where the conditioned stimulus is the event and the state is B. This is an assumption for which we have no empirical support.

The findings of Moscovitch and LoLordo (1968) add more unambiguous support for the conditioning of a B state. Using the same experimental situation as did Rescorla and LoLordo (1965), they employed a Pavlovian backward-conditioning paradigm to establish a CS_B. According to our analysis, this should pair the CS_B onset with the peak of B and create a maximally powerful CS_B. Recovery from a short-duration test with such a CS_B should be monotonic, because there is no mechanism whereby the *b* process could engender the *a* process. This is indeed what happens, as shown in Figure 8. Tests with CS_B caused a reduction in avoidance response rate to a level about 1/5 of that during the baseline performance. Recovery to baseline after CS_B termination took more than 30 seconds (see Moscovitch & LoLordo, 1968, p. 675, Figure 1), and it was monotonic. Moscovitch and LoLordo referred to the backward conditioned stimulus as a "safety signal"; they reported their dogs to be relaxed; "there were no indications of freezing or fright [p. 675]." This relaxation occurred in spite of the fact that the dogs were still in the avoidance-training situation, which was frightening enough to motivate a steady rate of avoidance responses (about six per minute). So their backward conditioned stimulus met all the criteria for a conditioned stimulus for a B state.

To convince oneself that B-state conditioning has indeed occurred, it might not be sufficient merely to show that the backward conditioned stimulus will suppress an ongoing Sidman avoidance response. The decrease could reflect a reduction of fear, a decreased expectation of shock, or both rather than some pleasant hedonic state with

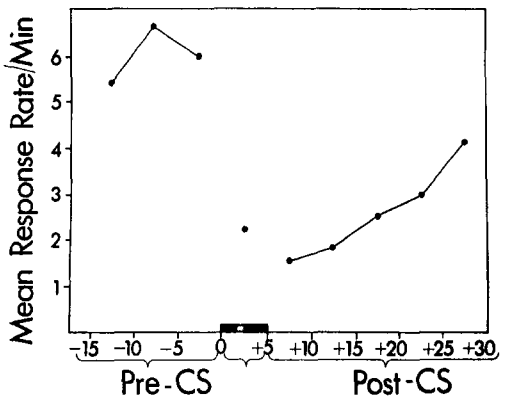

FIGURE 8. Data from Moscovitch and LoLordo (1968). (Here we see the response to a Pavlovian backward conditioned stimulus [CS]. The recovery from the conditioned stimulus presentation is monotonic and is a CS_B.)

positive reinforcement properties. Two lines of evidence would help. First, the gross behavior of the subjects, such as tail wagging in the presence of the backward conditioned stimulus, would suggest a qualitatively different state from that which the avoidance situation normally evokes. Second, the demonstration that a backward conditioned stimulus would subsequently reinforce the development of a new operant in a nonfearful situation might strengthen the conviction that the conditioned stimulus was really a CS_B of a pleasurable sort.

We have seen that Pavlovian laws seem to apply well to the conditioning of both A states and B states precipitated by aversive stimulation. We should then expect the same laws to hold for A states and B states precipitated by pleasurable sensory events. First, the conditioned stimulus events for pleasurable A states should be positive reinforcers, should be able to reinforce operants, and finally should be able to counter and sometimes even temporarily eliminate the B state. Therefore, in opiate addiction, presentation of a drug container, a syringe, a needle prick, or a room full of satisfied addicts should all tend to function as CS_As which would oppose the B state by arousing a conditioned A state. Such conditioned opposition would of course be weaker than that produced by the opiate itself, but it should be detectable. Many

opiate addicts actually give themselves "sham" injections. Even the familiar social surroundings where the drugs are obtained and used should have CS_A properties. They should temporarily reduce craving and abstinence panics. Clinical evidence suggests that this is indeed the case (Maurer & Vogel, 1967; Wikler, 1971).

The action of CS_B events should be even more interesting in addiction, because they should augment the B state and produce more intense craving. Such CS_Bs would be those paired with the peak of B, the most intense craving state. Thus, we would expect a variety of social and personal events to become conditioned arousers of craving. In general, they should be events and places associated with *lack* of the addictive drug. A jail cell, confinement, lack of money, all of these should be able to arouse a conditioned B state to augment an existing unconditioned B state. Perhaps the occurrence of the conditioned B state might precede the actual emergence of the unconditioned B state. This conditioning phenomenon should lead to increased frequency of dosage. For someone trying to "kick a habit," the careful avoidance of these CS_B events, which elicit B states, should be very helpful.

The conditioning process results in the addict being "hemmed in" to a great extent. If we assume that the events in Figures 7 and 8 occur also for the addictions, then increased craving would be a consequence of conditioned stimulus events, no matter whether a CS_A or a CS_B were presented. In the case of the CS_A, the craving should occur following the termination of the CS_A (see the biphasic recovery in Figure 7). In the case of CS_B, the craving should occur during and after the CS_B presentation (see the monotonic recovery in Figure 8). Thus, the Pavlovian conditioning of A states and B states, during the acquisition of an addictive cycle, overdetermines the craving for the drug. The addict, even if he tries to reinforce himself symbolically, i.e., with a variety of CS_A onsets, will experience an increased aversive B state afterward.

Conditioned *b* processes can become very powerful relative to *a* processes, just as unconditioned *b* processes can become very powerful. Take the work of Kimmel (1971), who used the galvanic skin response as an anxiety index. In analyzing the phenomena of galvanic skin response conditioning with aversive unconditioned stimuli, he found that the magnitude of anticipatory galvanic skin responses in response to CS_As often increased on the very early trials of conditioning. However, on later trials they decreased, even though the same unconditioned stimulus reinforcer was being regularly presented. Kimmel called this phenomenon the inhibition of emotional behavior, a manifestation of the organisms' adaptive adjustment. His idea is that the fear of the unconditioned stimuli has become manageable by the action of a fear-inhibition mechanism. Kimmel's concepts seem to be close to our own, if we change the inhibition concept to an hedonic opponent-process concept. We do not know experimentally whether the phenomena demonstrated by Kimmel also manifest themselves in appetitive conditioning. We suspect that they do.

The observations of Kimmel (1971), taken in conjunction with an opponent-process theory, suggest a variant of the experiment by Rescorla and LoLordo (1965) which would be a very strong test of the opponent-process theory. One would train dogs to perform a regular Sidman avoidance response. Then, during the conditioning phase of the experiment, a long series of trials with the unconditioned stimulus alone could be presented. Theoretically, this should strengthen the *b* process for fear. Then the discriminative conditioning phase of the experiment could be carried out just as Rescorla and LoLordo did. When the CS_A is presented on a test trial while the dogs are performing this avoidance response, the CS_A now, in contrast to what Rescorla and LoLordo found, should be relatively impotent in energizing the avoidance response. In other words, the CS_A onset would be a weak fear elicitor (Kimmel, 1971). In contrast, the termination of CS_A as well as the presentation of the CS_B, when presented during avoidance responding, should powerfully suppress

avoidance behavior. This suppressive effect should persist for a relatively long time after termination of the test with CS_B. These deductions stem from the assumption that the long series of pretreatment trials with the unconditioned stimulus alone has strengthened the *b* process, so that when the conditioned stimuli are finally introduced in the later discriminative conditioning, CS_A will be paired with a shock which elicits very little fear (*b* is large, so *a* minus *b* is small), but CS_B will be paired with the intense peak of the B state which is the opponent of fear (joy, euphoria).

A further deduction from opponent-process theory suggests a way to reverse what has heretofore been a strong empirical generalization. In most discriminative-conditioning experiments the elicitation power of the CS+ grows quickly as a function of trials of conditioning, but the inhibitory properties of the CS− require many more trials to manifest themselves. In the variant of the Rescorla and LoLordo (1965) experiment which we suggested as a strong test of our opponent-process model, the reverse should be true if discriminative conditioning is started after a long series of trials on which the unconditioned stimulus alone has been presented. The effectiveness of the unconditioned stimulus should be greatly attenuated when conditioning is started, and a conditioned stimulus paired with an ineffective unconditioned stimulus should acquire excitatory properties very slowly. In contrast, a conditioned stimulus coming directly after unconditioned stimulus termination would be associated with an intense B state. Such a conditioned stimulus should quickly acquire CS_B properties.

The postulates of an opponent-process theory of affect, combined with Pavlovian laws for the conditioning of A states and B states, give us a new way of looking at the phenomena of acquired motivation.

ANALYSIS OF SELECTED MOTIVATIONAL PHENOMENA IN LIGHT OF THE OPPONENT-PROCESS THEORY

There are many areas of psychological research which can be profitably reanalyzed within the confines of the opponent-process model. For example, we have found the model useful in increasing our understanding of addiction, childhood attachments, love and affection, imprinting in precocial birds, rewarding and punishing electrical stimulation of the brain, so-called thrill-seeking behavior, the dynamics of fear and avoidance behavior, the pleasures and displeasures associated with thermoregulatory behavior, some aspects of masochistic behavior, depression and euphoria, and taste cravings and obesity, to mention a few. We have selected only two of these phenomena for intensive and detailed analysis in order to demonstrate the applicability and use of the opponent-process model to deduce new phenomena and experiments. In the first, the A state is pleasurable, and in the second it is aversive.

Addiction and Stimulation of A and B States with Drugs

We have discussed the main features of an opponent-process theory of affect. These are as follows: (*a*) Process *a*; (*b*) Opponent Process *b*, a slave process; (*c*) the strengthening of the *b* process as a function of repetitions of the *a* process; and (*d*) the conditioning of A and B states by Pavlovian contingencies.

We believe any addiction can be better understood by reference to these features. First, the addictive substance must be capable of giving pleasure, at least sometime during its early use. Actually, there may be some aversive effects from the first few doses, but these are overshadowed by the pleasure effects coming either from the drug itself or from other available reinforcers. If not, the dosage will *not* be repeated. If dosage is repeated, the opponent process will begin to strengthen. Withdrawal symptoms and craving will intensify and become longer lasting. They will be aversive enough and persistent enough so that the user will try ways of getting rid of them.

It is here that we have to qualify the theory with a *specificity assumption.* We assume that, because the *b* process is the opponent of *a*, the quickest and most effec-

tive way of getting rid of the B state is to use the substance which directly produces the A state. We do not know, however, why this should be so, but it is the way addictive drugs work. Thus, behavior resulting in the obtaining and use of the A-arousing substance will be strongly reinforced because it produces A and it simultaneously terminates B. This will lead to further strengthening of the *b* process. Therefore, amounts of the substance will have to be increased in order for the sum, $(a - b)$, to be greater than zero, or above baseline. This is the drug tolerance phenomenon (Jaffe, 1965; Wikler, 1953). Increase of dosage then will reinforce all the behavior upon which it is contingent, and the *b* process will be further strengthened by more frequent use. This is the addictive cycle.

During early doses, when the quantity $|a - b|$ is large, previously neutral stimuli should become conditioned to A if they directly precede the onset of State A. They will become CS_A stimuli, Pavlovian elicitors of the A state or components of it. These stimuli, as CS_As for a pleasurable Drug State A, will be *positive* secondary reinforcers, capable of reinforcing new operants.

As the *b* process becomes stronger, the Pavlovian conditioning of previously neutral stimuli which directly follow *a*-process termination, and which also directly precede the peak of B, should readily occur. We will then see the emergence of strong CS_Bs, aversive in quality. They will be the conditioned stimuli for the craving and withdrawal symptoms. Thus they will be secondary negative reinforcers and will energize escape and avoidance behaviors. As the CS_Bs become stronger they will become more anticipatory, and so redosage and restimulation will start to occur at shorter intervals. The addict then will be using the drug partly to remove conditioned B-state arousals precipitated by the presence of CS_Bs. Therefore, increased dosage frequency, until some asymptote is reached, should occur whenever: (*a*) The *b* process becomes stronger, and (*b*) CS_Bs are established by Pavlovian conditioning contingencies. Increased frequency of dosage should occur even if drug amounts per dosage do not substantially change. Finally, if the addict, under the influence of other motivation, stops using the drug, the *b* process will weaken gradually with the passage of time.

The dynamics of behavior change which we have outlined are typical of most addictions (see Solomon & Corbit, 1973). Naturalistic observations appear to fit the deductions from the opponent-process model. Could the opponent-process model for acquired motivation help us to understand better the problems of the addict who wants to quit? Could we devise a regime to help him quit? In a well-addicted drug user, the major behavioral engineering task would seem to be to weaken B′. This is the state which energizes the strong escape and avoidance behaviors which terminate craving each time it occurs. The well-addicted drug user is exhibiting *avoidance* behavior much of the time rather than escape behavior. He would rather not experience the B state; he indulges so frequently that he rarely lets it occur, and if it occurs, he quickly gets rid of it with another dosage. However, in order to weaken B′, the addict must lessen the constant, repeated challenges to the opponent process. One way is to stop arousing A, i.e., cold turkey withdrawal, total cessation all at once. Another way is to decrease the intensity and frequency of A inputs. In other words, fade out A by gradually cutting down on drug use.

There is an analogy in color vision. Take a saturated red, and very gradually diminish the saturation until it is gray. A green afterimage does not occur, but the time of fade-out has to be equal to, or longer than, the duration of the green afterimage when red is suddenly terminated rather than faded out. If the time of fade-out is shorter than that, there will be a green afterimage because some finite quantity of the opponent process will still be operative due to its own intrinsic decay time. Therefore, reasoning by analogy, in order to weaken the *b* process in addiction by a fade-out technique, we must first know how long the craving *would have lasted* had the addict gone through cold turkey withdrawal until the craving had disappeared. This could be a very long time,

and it must be estimated. If the fade-out technique involves a gradual decrease of drug use frequency and amount, which lasts for a shorter period than the cold turkey period, the technique is bound to fail. That is because, after the last small dose is taken and terminated, the B′ state would still come in strongly enough to energize behavior leading to another redosage.

The opponent-process model warns of still another problem for the addict who wants to quit. Even if the drug user were successful in reducing the amount of B, there is still the positive reinforcing effect of A to contend with. Together with social reinforcers, it was probably important initially in reinforcing the user's behavior. Now the *a* process again has a chance to reinforce drug use, because the quantity $|a - b|$ gets larger as *b* gets smaller. Thus a strong positive reinforcer (A) for drug use will again become available for any experienced user late in the cold turkey withdrawal or the fade-out regimen. This trade-off, deduced by the model, makes the "kicking" of an addiction very difficult. Late in addiction, the aversive craving state energizes the behavior more forcefully, but after partial withdrawal, the positively reinforcing state reinforces the self-administration operants.

How does one handle the problem of countering a positive reinforcer? First, by the action of punishment. To weaken the drug-use behavior at a time when $|a - b|$ is large, punishment is one indicated alternative. Punishment probably should *not* be used early in withdrawal, or early in fade-out, because the drug-use behavior is composed of avoidance and escape operants driven by the aversive B′ (the withdrawal agony and craving). Punishment at that time might intensify the operants, as it frequently does for any operants controlled by aversive states (the vicious circle phenomenon). Perhaps late in withdrawal, strong rewards for abstinence might be used together with punishment for drug taking. But such rewards would have to be hedonically stronger than is the drug-produced reward A at the time when *a* is strong and *b* is weak.

Another technique suggested by the opponent-process theory of motivation is the use of antagonistic drugs. It should be possible to break the addictive cycle with a drug which antagonizes or eliminates the aversive *b* process. The addict medicated with such a *b* process antagonist would still experience the pleasurable A state, but the withdrawal agony and craving would be gone. He might, under the impetus of other motivational influences, become a casual user rather than a driven addict. An even more effective therapy could be developed if we had two drugs, an *a*-process antagonist as well as a *b*-process antagonist. The former could be relatively short acting, but the latter would have to be long acting in order to counteract the long-lasting aversive *b* process.

The opponent-process model deduces that there will be great difficulty in eliminating the addictive cycle. Even though all the conditions for weakening the *b* process are optimal, trouble can ensue. In addiction, State B is aversive. If aversive B states are not completely specific to their A states, then other aversive states might be reacted to as though they were the B state. A person who has reduced his craving by means of protracted abstinence might respond to some types of other aversive emotional states as though they were intense craving states, thus remotivating the drug-seeking behavior. We need to know a great deal more about the narrowness of specificity of A and B states before addiction is satisfactorily understood. The cross-tolerance phenomenon tells us that specificity in many cases is not narrow. For example, the alcohol addict develops a strong cross tolerance to barbiturates even though he does not use barbiturates. Therefore, he requires a larger barbiturate dose to produce a specified behavioral effect than he would if he had not already acquired alcohol tolerance. The opponent-process model suggests that the *b* process elicited by the primary affective consequences of alcohol is somehow similar to that elicited by barbiturates. The opponent-process model is concerned with affective and hedonic processes. Therefore, two drugs, no matter how chemically unre-

lated they may be, if they arouse very similar affective states, will be candidates for cross tolerance. Furthermore, this reasoning deduces that drug-aided withdrawal treatments must fail if the drug used to antagonize the *b* process is one which itself generates an affect similar to that produced by the original addictive agent. Finally, an addict should be very quickly and easily addicted to such a treatment drug. Thus, there are grave problems associated with the use of *b*-process antagonists.

There is another difficulty. The action of CS_A and CS_B stimuli, whose properties were acquired over thousands of conditioning events usually involved in long-standing addiction, must be considered in the process of withdrawal and "cure." The CS_B stimuli will be conditioned arousers of B, the craving. The CS_A stimuli will be conditioned arousers of the positive reinforcing State A. A major problem during withdrawal ought to be CS_B events, the conditioned elicitors of craving. They would be stimuli associated with the absence of the drug: no pushers, empty pockets, new surroundings, nonusers (they cannot offer you drugs), etc. Their presence should produce some degree of conditioned craving.

It appears possible, at first glance, to use CS_A stimuli to enable an addict to endure intense craving episodes. For example, a nicotine addict might take out a pack of cigarettes, take out a match, put a cigarette in his mouth, then suck on it without lighting it, going through all the usual acts, including inhaling, blowing out, etc. This would arouse a conditioned *a* process, and therefore should temporarily result in *less* craving than that felt before these acts. However, when the acts are terminated, their aftereffect should be the appearance of a conditioned B state which would be superimposed on the existing unconditioned one. Then, of course, the craving should temporarily intensify. Once again, we see that the behavior of the addict is tightly hemmed in by processes, both conditioned and unconditioned, which overdetermine the persistence of the behavior leading to a redose (Solomon & Corbit, 1973).

In conclusion, nature has devised a powerful way of maintaining behavior which initially produces pleasure, even though eventually the pleasurable consequences of the behavior may become minimal while the aversive consequences of absence of that behavior become more pronounced. We should not expect a quick cure for addiction, then, until we learn how to suppress or antagonize *both* opponent processes.

The powerful control of behavior resulting from repeated pleasures is not limited to drug pleasures. Any intense pleasure, the termination of which is followed by an enduring aversive state, should lead to persistent, recurrent behavior that is extremely hard to eliminate. Addiction is, therefore, a possible consequence of any repeated pleasure.

Drug addiction is not the only behavioral phenomenon simply organized by an opponent-process model. We cannot examine all of these cases in detail, but we are certain that the reader in each case can develop the explanations imposed by the opponent-process model. One example is the reinforcing electrical stimulation of the brain. Assume only that the onset of such stimulation produces a pleasurable A state and that the sudden termination of the same stimulation reveals an aversive B state which is of relatively short duration (about 30 seconds). Another example is imprinting in precocial birds. Assume only that the sight of a moving white object arouses an unconditioned A state which is a strong positive reinforcer and that the removal of that object reveals a very aversive, long-lasting B state which intensifies rapidly with repeated elicitation. All of the known data on both distress calling in ducklings and following behavior (as well as other operants) are simply explained (Hoffman & Solomon, 1974). Indeed, most of the major phenomena of human attachment behavior are subsumed in this analysis.

Aversive Learning and Performance

We have previously presented evidence that both A states and B states are conditionable. Stimuli directly preceding the peak of A will become CS_As for the elicita-

tion of a conditioned A state. Stimuli directly preceding the peak of B will become CS_{Bs} for the elicitation of a conditioned B state. We have been referring to hedonic or affective processes, and so our conditioned A states, in the case of shocks, will be conditioned aversive states (fears). Our conditioned B states, in the case of termination of shocks, will be conditioned pleasure states (happiness, relief, euphoria, exhilaration). The onset of the A state will negatively reinforce operants while the onset of the B state will positively reinforce operants.

The assumptions about the conditionability of opponent processes suggest a reinterpretation of escape and avoidance learning and extinction. On the first few avoidance training trials, the aversive A state will be very intense, and its onset will energize a variety of escape behaviors. If signalized shocks are used, the signal will quickly become a conditioned A-state elicitor. If a Sidman, unsignalized, training procedure is used, the responses or acts of "staying still" or "doing the incorrect thing" will become conditioned A-state elicitors, and so long bursts of energetic activity will ensue. Early in training, shock termination will result in a weak *b* process, without much positively reinforcing effect, and it will fade quickly.

As shocks are repeated, two affective processes will evolve. First, the shocks will decrease in their aversiveness as the opponent *b* process strengthens. Second, the positively reinforcing effect of the B state will increase, so that successful operants will have an opportunity to be reinforced by two events, the termination of the aversive A state and the onset of the pleasurable B state. These operants will be circumscribed, as contrasted with the wild, disorganized, quickly altering bursts of behavior occurring during the first few shocks. Furthermore, if the *b* process strengthens very quickly, some subjects will not learn. They will be the failures so frequently referred to in footnotes (see Turner & Solomon, 1962). They will become "used to "shocks before B-state reinforcement can accurately select the successful avoidance response for them. Their behavior will first be chaotic. Then they will appear to "give up" and become unresponsive to shocks. They will act as if shocks no longer "bother" them very much. Probably they do not.

Provided that the responses narrow down sufficiently to those successful in preventing shocks, a new stage of learning will ensue. Inactivity and other danger signals will control a conditioned aversive A state. The correct avoidance response will control the appearance of the peak of B, the pure B state. The *b* process will be further strengthened by the repeated elicitation of conditioned A states even when the shocks are no longer experienced. The A state conditioning will then start to extinguish due to the nonoccurrence of the shocks. Meanwhile, the correct avoidance response will continue to be reinforced, differentiated, and perfected as an automatic act, as long as it will produce the pleasant B state. At this point the subject's behavior will have other attributes, too. Because the conditioned aversiveness of all stimuli in the training situation is now extinguishing, the subject may willingly enter the experimental room and apparatus. He will perform his avoidance behavior without signs of fear. Yet he will appear overjoyed when the training session is over. This reflects both the weakening of conditioned A and the continued strength of unconditioned and conditioned B. The subject has a new source of positive reinforcement in his environment, the CS_B (see Weisman & Litner, 1969a). As Weisman and Litner (1969b) have shown, the CS_{Bs} have the power to reinforce new operants. They are, then, conditioned stimuli for a B state which is pleasurable. These stimuli must be serving a function like that of the correct behavior of the ski-jumper which produces an exhilaration mood when the danger is over. Another example is that of the parachutists (Epstein, 1967).

The model predicts that avoidance behavior will eventually extinguish, but may take a long time. Extinction may, as Hullians claim, be motivated only by the aversiveness of effort, but the contrary aversive forces are also great. Should the subject fail to

perform a correct avoidance response during an extinction series, he will reinstate a powerful CS_A which has not been experienced since the early training trials. Arousal of the A state will then strengthen the *b* process further. Because no shocks occur, the conditioned A state will weaken. The aversiveness of failing to perform the avoidance operant will consequently decrease.

Avoidance behavior will cease only when all response-produced and external danger signals no longer produce an aversive A state strong enough to energize the avoidance behavior. The more effortful the behavior, the sooner this should happen. The sooner this happens, the more likely is the subject to appear to be frightened by his own inactivity even though he fails to respond. Toward the end of an extinction series, then, behavior should be variable. A long "vacation" away from the training situation would weaken the *b* process, so $(a - b)$ would increase and frequent "spontaneous recoveries" should be seen, with avoidances occurring early in a new session. At the same time, the joyous behavior, which once characterized the end of a session, should disappear. Sources of positive affect thus dry up late in the extinction of avoidance responding, but sources of aversive affect are still lurking and are relatively weak. There should be no such phenomenon as extinction of avoidance behavior with equanimity.

Most of these phenomena deduced from an opponent-process model have occurred at one time or another in avoidance-training experiments, but they have tended to be overlooked or ignored by most writers, including one of us (Solomon & Brush, 1956; Solomon & Wynne, 1953; Turner & Solomon, 1962).

The analysis of escape and avoidance learning and performance in terms of opponent-process theory has generality beyond the mere habituation to electrical shocks in the laboratory. The intense aversiveness of the first free fall through space, the first exposure to the intense heat of a sauna bath, the first ski jump, or the first killing of another man in war might initially suggest that escape and avoidance behavior would occur. Instead, the subject often perseveres in re-exposing himself to the aversive event. He looks like the rat which fails to learn to avoid shocks in the laboratory and appears to be "unmoved" by those shocks. Why would these failures of escape and avoidance occur? There must be some outside motivational influence which keeps the subject in the aversive situation long enough to allow the strengthening of the *b* process. Once this has occurred, the quantity $(a - b)$ is reduced and the B state after stimulus termination is more pleasurable and long lasting. There is then a reasonable trade-off between the immediate occurrence of the weakened, aversive A state and the somewhat delayed occurrence of the strengthened, pleasurable B state. According to our theoretical model, many seemingly masochistic behaviors should have the etiology we have described.

A States Which Have Little or No Opponent Process

A few hedonic disequilibriums may cause very little corrective, oppositional reaction in the nervous system. A nonopposed system would manifest no peak of the A state, no adaptation, and no appearance of a B state after stimulus termination. One possible example is the hedonic state engendered by marijuana. As a chemical stimulus it precipitates a mildly pleasurable A state. However, there is no reported peak or adaptation, nor are there aversive withdrawal symptoms or craving. Furthermore, tolerance often does not develop with repeated dosages. The concommitance of *all* these attributes would be exactly what the opponent-process model would predict for a pleasure without an opponent. Nausea is an example of an aversive A state which may have no opponent B state. Perhaps some aesthetic pleasures have no opponent process.

We have already talked about the variations in the intensity and duration of B states as a function of the particular kind of A state elicited. For example, we mentioned the intense and long-lasting aversive B states in the cases of grief and opiate

withdrawal. In contrast, we mentioned the relatively short duration of B states associated with the taste of M & Ms and peanuts, and rewarding electrical stimulation of the brain. In addition to these inherent variations in intensity and duration of B states elicited by different types of stimuli, repeated elicitation causes large changes in the intensity and duration of B states in some cases and not in others. Therefore, differences in the initial strength of *b* processes must reflect a parameter of our model, and the effects of repeated elicitation of A states must operate on this parameter. In the case of A states having no opponent process, the value of the parameter is zero.

We have been constantly amazed by the huge variations in the strengths and durations of different *b* processes after repeated elicitations. Some *b* processes, even when well exercised, last but a few minutes. Some examples are taste cravings, the aversive *b* process for electrical stimulation of the brain, cardiac deceleration after shock termination, the "fit of joy" when the dog is released from a shock box, etc. Some last a few hours. Examples are the exhilaration following dangerous or endurance-challenging exercise, or distress calling in precocial birds when the imprinting object is gone. Yet others last for months. Examples are loneliness and grief, craving following withdrawal from either opiates, alcohol, or barbiturates.

At first glance it may appear that we have completely undercut our theory. We have speculated that some A states may not arouse B states, and we have provided no principles by which one could designate in advance whether or not a particular A state would be a part of an opponent-process pair. At present, we look on this as an empirical problem, and we use the following argument. Opponent processes defend a hedonic equilibrium. They are part of the biological defense system mediated by the brain. In the realm of foreign body reactions to bacteria, viruses, and poisons we often can detect defense systems. Indeed, the actions of antigens and antibody formation bear many resemblances to the opponent-process systems we have described. However, not all poisons and not all foreign bodies engender defense reactions. We can be defenseless. These substances are, therefore, called deadly poisons. We think the same situation holds in the defense of hedonic equilibrium, and so we should not be surprised to discover that a given A state goes relatively unopposed. Perhaps one day we will have a theoretical rationale for the parameter and operator involved here.

DISCUSSION

We have argued that there are certain systems in the brain, the business of which is to suppress or reduce all excursions from hedonic neutrality, whether those excursions be appetitive or aversive, pleasant or unpleasant. The systems operate to decrease the intensity of subjective "hedonic quality," "affect," "emotion," "arousal," or the objective reinforcing properties of stimuli. The systems function independently of operants or instrumental acts. They are fully automatic. Thus, whereas operants tend to maximize positive reinforcement and to minimize negative reinforcement, the affect-controlling systems of the central nervous system minimize both. They are brought into play whenever significant departures from affective equilibrium occur as a consequence of stimulation onset and maintenance. When such a system is effective, it will reduce the intensity of the affective experience even while the input is still there. This reduction will be manifest for both positive and negative reinforcers, and for pleasant and unpleasant stimuli. The theoretical model refers to both subjective and objective psychological phenomena.

Reduction in affective or hedonic intensity is postulated to be brought about by the activation of an opponent loop, precipitated into action whenever affective, hedonic, or emotional states are aroused. The opponent loop opposes the stimulus-aroused affective state. Furthermore, the opponent process is postulated to be sluggish in its latency, recruitment, and decay (a heavily damped circuit, an inertia-laden system). The opponent loop itself is postulated to generate an hedonic process which is, in

some abstract sense, the opposite to that precipitated by the stimulus input which has initially aroused affect. The opponent process will manifest its quality and intensity when the stimulus is suddenly terminated. The persistence of the opponent process will be seen for some time because of its sluggish decay property.

Furthermore, we have postulated that the opponent process is strengthened through use and weakened through disuse. These changes are nonassociative in nature. This makes the affect-control systems similar to some immunological mechanisms in their properties.

Even though the model is not yet as precise as some mathematical models, from it, nevertheless, one can unambiguously deduce many of the known phenomena of acquired motivation. As examples of this, we have illustrated in detail how the model organizes information on aversive behavior control and the drug addictions.

The theory postulates no conditioning or learning mechanisms responsible for the occurrence of the acquired motivations we have discussed. The acquisition is automatic, merely by virtue of the repeated occurrence of affect-producing stimuli. However, the interrelationships between the affect-control mechanisms and Pavlovian conditioning processes, and the interrelations of both with operant behavior, are discussed.

The novel feature of the opponent-process theory is that it sees the behavioral phenomenon of addiction as an empirical model for all acquired motivation. Addiction is not viewed as an abnormality. Instead, it is the inevitable consequence of a normally functioning system which opposes affective or hedonic states. We assume, for example, that love is an addiction phenomenon characterized by habituation to the presence of the loved one and intensified aversion in the absence of the loved one. In the same vein, we assume that imprinting in precocial birds is an addiction phenomenon. In the case of aversive stimulation, we assume that masochistic phenomena are the consequence of a normally functioning system which opposes affect. These phenomena are characterized by habituation to the presence of the feared or unpleasant event and intensified pleasure after the termination of that event.

Finally, we have pointed out some new lines of empirical research suggested by the opponent-process theory of motivation.

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