Pleasure Centers in the Brain

Why aren't we just as fit to survive if we're nudged ahead by pains and deficits?

Psychology has gone through a period of fantastic growth during the last 30 years, primarily because it seemed to offer college students some explanation of their malaise and some understanding of themselves which would free them from anxiety. Then why, if we are interested in the weal of these college students, do we expend our research on albino rats? Facetious though it may appear, there must be some truth in the answer that the albino rat is an excellent model of the contemporary American college student, or at least the majority of them. Neither the student, having been bred in America, nor the white rate, having been bred in a laboratory, has ever experienced a need in its life.

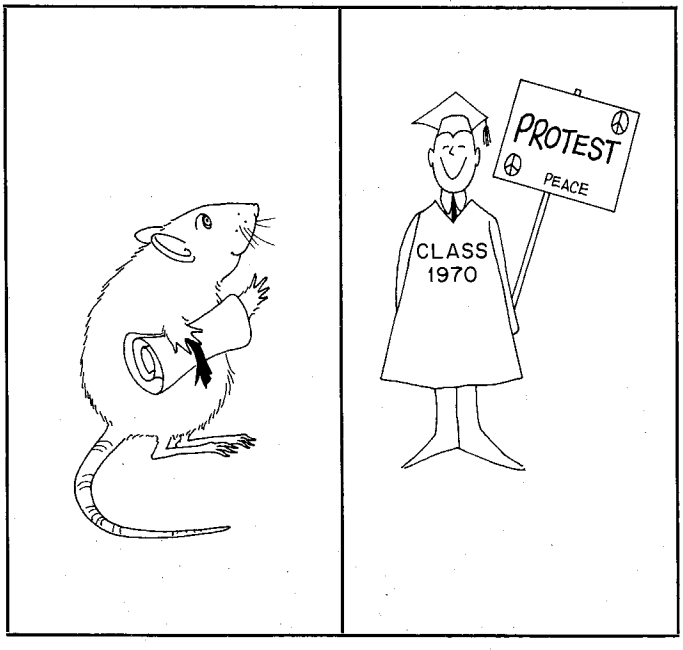
The question of what might drive behavior in the absence of needs would have bothered theoretical psychologists and Puritan preachers alike at the time I entered the field in the late 1940's. The concept of need-driven behavior is simple, compelling, and in complete accord with the simplest concept of evolution. It says that damage to the organism, or deprivation inimical to health, causes physiological processes which are experienced as discomfort, and that behavior proceeds in a random or a guided fashion until discomfort is alleviated. An uncomfortable person is in need, and need justifies a multitude of sins.

A few short steps ahead of this conception—and representing no significant advance in sophistication—is the drive-reduction theory of learning. The conception of this theory is that somewhere in the brain incoming sensory messages cross outgoing motor messages. If a sudden drive reduction occurs, a connection becomes fixed more or less permanently, so that the next time the sensory message will cause the rewarded behavior. It is possible to attack this theory on a variety of grounds, one of which is the simplicity of its attitude toward the data processing that goes on inside the brain. This theory gives rise to a law that states, "Learning occurs only when discomfort is relieved."

For an organism that seeks novelty, ideas, excitement, and good-tasting foods, the drive-reduction theory was a Procrustean bed. Whatever did not fit was shorn from our image of the man and the rat. Drugs, good foods, and sex were thought of in terms of a need—that is, a hurt generated by withdrawal. Even the coddled white rat was trapped into his forward motion by his residual pains. Behavior was a downhill course toward quiescence, and its energetics were a series of accidents from outside which countered the downhill trend.

If behavior was not aimed to repair these damages and concurrent discomforts, then why was it selected and why did it survive? This rhetorical question was given in answer to all counterarguments.

It is interesting that research on the albino rat, if it has not gone far to improve the sophistication of the



Neither the student, having been bred in America, nor the white rat, having been bred in a laboratory, has ever experienced a need in its life. What, then, might drive behavior in the absence of needs?

by James Olds

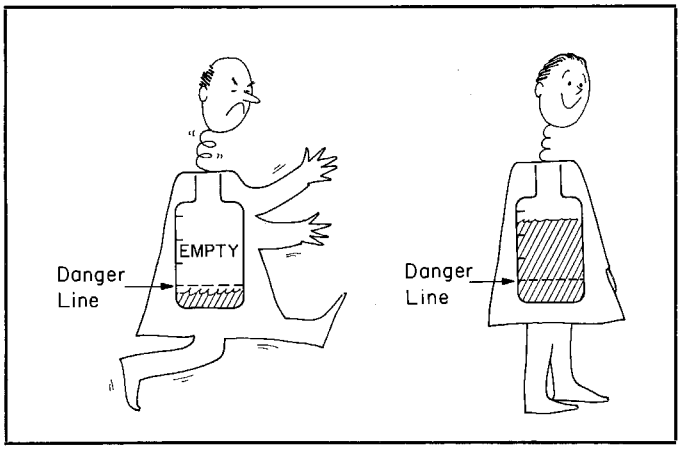
psychologist, has at least caused a minor revolution with regard to this drive-reduction theory of reward. This has been through the discovery that animals work not only to turn *off* discomforting stimuli but to turn *on* brain stimulations in an extensive set of brain regions. These regions are now considered by many to be central counterparts of the positive factors and good things of life that turn on people in everyday life.

The method of drilling holes in the skull and lowering probes by means of a guidance system to stimulate very small and well-localized brain centers was developed gradually in the first half of this century. Professor William R. Hess, still alive in Zurich, Switzerland, developed a method of fixing a plaque to the skull from which a probe penetrated deep into the brain and to which a wire from an electric stimulator might be attached. Animals were then permitted to recover from the operation; the small scalp wound healed completely around the plug. The probes into the brain were metal wires that were insulated except for the very tip, and so the point of electric stimulation was relatively small. The long, loose wire suspended from above permitted about as much movement to the animal as is permitted to a dog on a long leash. Electric stimuli could then be applied during the free behavior of the animal to see how localized electric currents might influence ongoing behaviors, and to see what responses might be evoked by stimulating locally at different brain points.

With these methods Hess discovered, in the cat, places where the basic energy-mobilizing responses of the heart, the lungs, and the preparatory musculature could be controlled. There was a large region where stimulation caused the animal to become prepared for fighting or fleeing, by an increase in heart rate, blood pressure, the rate of breathing, the amount of muscle tone, and so forth; and he found another large adjacent area where electric stimulation caused the opposite of all these actions so that the animal either became prepared for sleep or engaged in one of a number of restorative bodily processes.

The area where brain stimulation caused excitement and preparation for violent activity included parts of the posterior hypothalamus and the adjacent area of midbrain. The area where electric stimulation caused quieter bodily processes of rest and repair included the anterior hypothalamus and related sectors near the cortex.

At about the time I entered the brain-stimulation field, Neal Miller, my famous colleague who is now professor at the Rockefeller University, was the world's chief proponent of the drive-reduction theory of reward, a



The theory of drive reduction implies that learning occurs only when discomfort is relieved. But for an organism that seeks novelty, ideas, excitement, and good foods, this theory has its limitations.

theory which he still occasionally professes. He was not only a proponent of this theoretical view, but he was embarked with Jose Delgado of Yale upon an enterprise that would bring the drive-reduction theory into close relation with the work of Hess. The outcome of these studies was to show that stimulation of the posterior hypothalamus and anterior midbrain also caused a psychologically valid aversive condition so that the animal responded as if it were put into genuine discomfort by the electric brain stimulation and as though it afterward became afraid of those places where the brain stimulus had been applied. At a later date Miller and his colleagues were able to show that electric stimulation within a center —which had for other reasons come to be called the "feeding center"—of the hypothalamus caused not merely the behavioral responses of feeding but also a psychologically valid drive, because the animal would not only eat if food were available, but by stimulation would be caused to work for food when food was absent.

It was a title of a Neal Miller talk which in 1953 first caused me to believe that brain stimulation caused not only this discomfort motivation which was so palatable to the drive-reduction theorists, but also perhaps some positive or hedonic motivation which would be the antithesis of their view. Neal Miller used the title "The Motivation of Behavior—" or perhaps he said "The Reinforcement of Behavior—Caused by Direct Electric Stimulation of the Brain." On first reading the title I thought for a brief moment that he would reward the animals by turning *on* the electric brain stimulus. When I read his abstract carefully and later heard the talk and saw his movies, I realized that he was causing an aversive reaction with his electric shock to the brain, an aversive reaction which it seemed to me might be caused even more easily if he would apply his electric shocks in any part of the nervous system, even including the forepaws or the hindpaws or the surface of the skin.

Very shortly after my misreading Neal Miller's title, through a variety of fortuitous circumstances, I was sitting at a table on which there was a large enclosure about 3 feet square with sides 10 or 12 inches high. It contained an albino rat, in which a probe was implanted to stimulate in one of the regions in or near the hypothalamus. A wire suspended from the ceiling connected the animal to an electric stimulator which I controlled by means of a pushbutton hand switch.

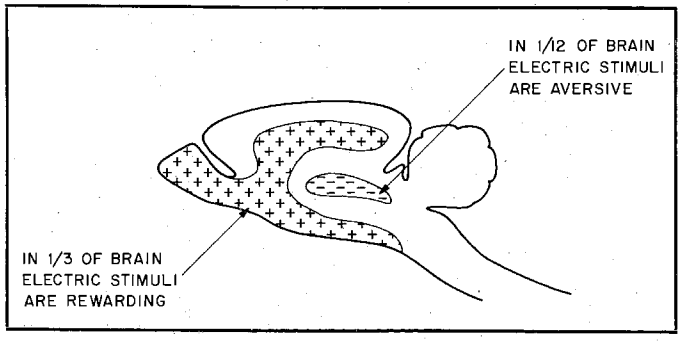
For reasons which in retrospect sound foolishly complex or ridiculously random (depending on your point of view), I had decided to stimulate the rat each time it entered one of the corners. It entered a first time, and I applied a stimulation which lasted approximately ½ second; the animal made a sortie from the corner, circled nearby, and came back. I stimulated a second time, not more than a minute or so after the first time. The animal made a second brief sortie, but came back even sooner. I stimulated a third time, and the animal stayed with an excited and happy look. (You may wonder how I know, but I have "gone among them and learned their language.") The animal kept staying and I kept stimulating, for I was already convinced that the animal had come back for more.

In successive experiments with the same rat, first it was caused to go to any corner of the enclosure selected by an independent observer, provided only that I would apply the brain stimulus immediately after the animal took a step in the right direction. Still later it learned to run to the pre-chosen arm of a T-maze in order to get to a terminal point where electric brain stimulation was applied; the animal eliminated errors and ran faster from trial to trial. Before I was done with this, my first animal, I was convinced that his behavior was directed not to mitigate aversive conditions but rather to instigate a positive excitation. The question, however, had to be asked whether this was an accidental observation or a significant feature of brain and behavior so that it might be taken as exhibiting a fundamental law about the direction of some behavior toward, rather than away from, the excitements of the environment.

Together with Peter Milner, who was at that time my instructor in brain-stimulation methodology, I endeavored to repeat the observation in another animal. This did not at first happen with ease. Some animals with probes directed at or near the original point seemed to favor the stimulus, but others seemed to respond as if it were negative rather than positive. It soon became apparent that careful mapping of the brain would be required to zero in on the critical areas and create a situation where animals could be prepared so that the basic characteristics of the phenomenon might be studied with a variety of methods and be understood.

For this purpose we used a Skinner box in which the animal could stimulate its own brain by depressing a lever. A Skinner box (named after Harvard's famous behaviorist, B. F. Skinner) is nothing but a small enclosure with a single manipulable device such as a lever, arranged in such a fashion that the animal, by manipulating the device, causes itself to be presented with a reward. The rewardingness of the reward is then measured by the rate of the lever response. For measuring the reward properties of the electric brain stimulations in different centers, Skinner's method was ideal.

We used a very small box and a very large lever, so that



Rewarding effects of brain stimulation are neither accidental nor confined to small, obscure brain centers. Furthermore, the parts of the brain where the best positive effects are achieved are clearly separated topographically from those points of the best aversive effects.

the random rate of pedal pressing was very high during the initial period. If the rate rose rapidly, so that the animal was eventually responding at rates of about one pedal-press per second, it seemed that there were quite clearly rewarding effects of the brain stimulation; if after the first one or two self-administered stimulations the animal stayed away from the lever, these zero rates could be taken as evidence of aversive effects of the electric brain stimulation. With this arrangement it was quite easy to map the phenomenon, and this has provided a basis for an easy reproduction of the rewarding brain stimulation, not only in a large number of experiments which have been performed in my laboratories at UCLA and the University of Michigan, but also in a large number of laboratories throughout the United States and the rest of the world.

The self-stimulation experiments quickly resolved the most basic question: The rewarding effects of brain stimulation were neither accidental nor confined to small, obscure brain centers. One-quarter to one-third of the points tested yielded self-stimulation behavior to the degree that animals stimulated their brains at very high rates, ranging from one pedal-response every ten seconds in places where the effect was mild, to more than two pedal-responses every second in areas where the positive effect was very intense. Points where brain stimulation had a clearly aversive effect were far less numerous in the rat than were those with a positive effect. Only about one brain point out of every 12 tested caused a rate which was clearly depressed. Furthermore, the parts of the brain where the best positive effects were achieved were clearly separated topographically from those points of the best aversive effects.

The "rewarding" parts of the brain were all related to olfactory mechanisms and to chemical sensors. Among these were many areas where the brain itself seems to act as a detector of sex hormones and hunger factors carried in the blood. Mapping in other animals showed that the same parts of the brain were involved in rat, rabbit, cat, dog, monkey, and man. The experiments have also been conducted successfully in birds and fish, but the brains in these cases are sufficiently different from the brains of the mammals so that I would not want to say whether or not the same parts of the brain were involved in these cases.

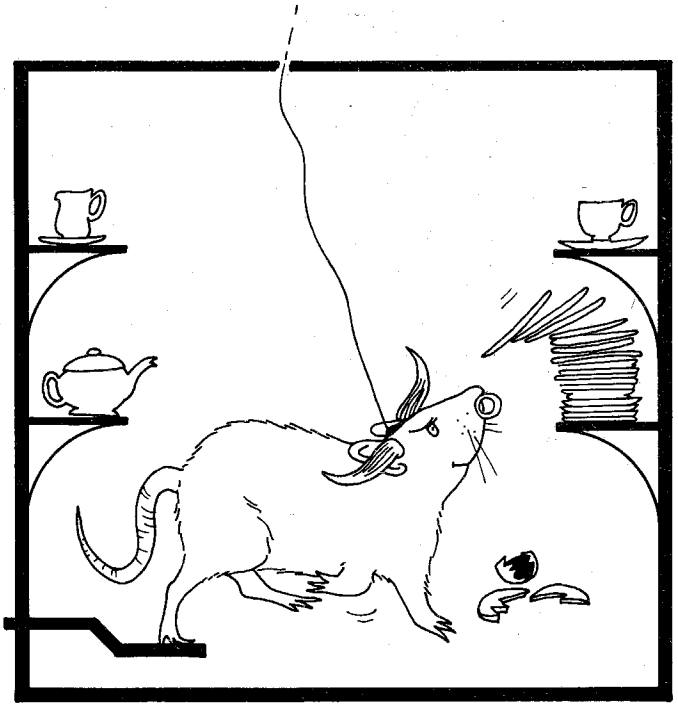
"Let's exchange pushbuttons"—a good joke, but not likely to happen.

The investigations of human patients with implanted electrodes have been carried out in the course of three different kinds of therapeutic procedures: those related to the severe mental psychotic ailments; those related to the cure by means of very small brain lesions of severe intractable pain, and by means of similar lesions of Parkinson's palsy; and finally there have been those involved in providing temporary relief for cancer victims who had previously been maintained on morphine. Reports of experience from human patients have often been confused, but they have been repeatedly positive; patients have stimulated themselves and have been maintained in far better and happier condition with less deterioration than was ever achieved with drug therapy.

Lest the younger of you fear for yourselves, and the older of you fear for your children, I do not foresee even in these times anyone so avant-garde that he will readily tolerate having his head drilled while having his ears pierced. Probes in the brain over long periods of time create scar tissues, and scar tissues become epileptic foci; the method will never be used except in cases where therapy is acutely required.

One of my friends who was rewriting Aldous Huxley's *Brave New World* and combining it with his own version of Orwell's *1984* brought his novel to an unlikely end by having his two main characters (still a girl and boy, although for some reason the difference was both less conspicuous and less important) both implanted with wires which come from under their long hair and into their pocket stimulators. He shyly suggests, "Let's exchange pushbuttons." I am of the view that it's a good joke, but it's not a danger.

Back to the rats. The very rapid and intense pedal-response rates were not as immediately convincing to my colleagues as they were to me. People asked whether the brain stimulation might be simply arousing and exciting so that the large animal in the small box would be something like a bull in a china shop and that with such a big lever every behavior would be a pedal-press behavior. Other people suggested that even if there were some disposition on the part of the rat to come back for more stimulation, this might be something induced by the previous stimulus, so the animal, having an aversive aftereffect—something like an itching caused by the first stimulus—would come back and alleviate it by pressing



Is a rat in a small Skinner box equipped with a large pedal like a bull in a china shop, where any kind of stimulation results in pedal-press behavior?

a second time, and a third, and so forth, much in the way one scratches a mosquito bite.

To answer these questions—which suggested that perhaps the positive observations were only a sham and not the true substance of a positive reward—we ran a series of behavioral tests. In a maze, animals were trained to run from Start to Goal, where they received only brain shock for reward. Hungry rats ran faster for the brain shock than they did for food. They eliminated errors from trial to trial, thereby indicating that this was no bull-in-a-china-shop phenomenon. They ran purposefully without errors when first tested in the morning, 24 hours after the last previous brain shock, thereby disproving the argument that some aversive consequence of a preceding brain stimulus caused the animal to seek more.

At this point people began to concede that perhaps there was a set of mechanisms in the brain concerned with positive drives which competed with or were an adjunct to the control of behavior by negative needs and aversive mechanisms. But the question needed to be asked whether or not this was some junior partner to be in charge of entertainment and cultural enlightenment after the needs were all cared for, or whether this might be a basic force in behavior, a full competitor with pain and the basic needs.

The first experiment to answer this question showed that animals would cross a grid that administered painful shocks to their feet in order to get to a pedal where they

could stimulate their brains. Animals took four times as much electric footshock when they were pursuing the brain reward as a normal hungry rat is willing to tolerate when it is in pursuit of food.

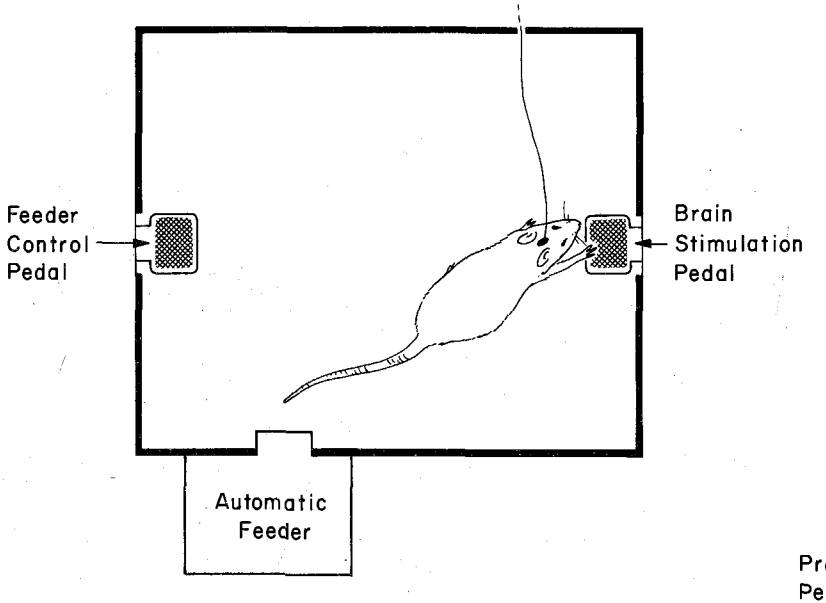
In another experiment rats, to all intents and purposes, gave up food to the detriment of health and underwent the danger of starvation in order to stimulate their brains. In this experiment, animals in a food pedal box were permitted 45 minutes daily (just time enough to get a meal that would maintain them in a healthy condition). When they were offered in this box the alternative of electric brain stimulation, they quickly renounced food almost altogether, and would have died of starvation but for the benign intervention of the experimenter. Other experiments have showed that rats would press one pedal as much as 100 times only for the sake of getting access to a second pedal with which they could stimulate their brains.

And in another set of experiments it has been found that one experience of this positively reinforcing brain stimulation can last for a very long time, having consequences for two or three days. Even a period of two seconds of brain stimulation has modified the animals' behavior for as long as seven days in the experiments of Carol Kornblith in our own laboratory. In her study, animals were stimulated briefly in the least preferred part of a large enclosure. Often this caused the least preferred place to become the most preferred place, or at least it modified greatly the amount of time which the animal spent in that part of the experimental chamber, and the change lasted for a very long time.

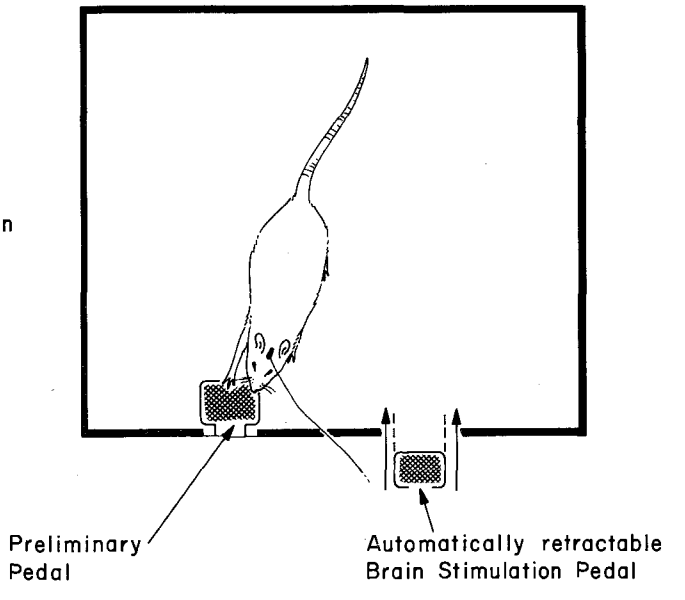
If we grant the very strong influence exerted by these positive brain stimulations on behavior, the problem arises of how these forces interact with negative, aversive influences, and what is their relation to basic drives such as hunger, thirst, and sex.

The problem of interaction between positive and negative mechanisms was first brought to the forefront by experiments of Professor Warren Roberts, now at the University of Minnesota, indicating that there were parts of the brain where electric stimulation simultaneously and paradoxically caused both rewarding and punishing effects. Either the animal would first work to turn the stimulation on, and once it was on, work rapidly to terminate it; or the animal would, if forced to remain in a place where stimulation was available, pedal-press very rapidly as if seeking to obtain it, but rapidly escape from the box if any escape could be found. In the latter case it appeared that an ambivalent and ambiguous stimulation was being applied, a simultaneous stimulus of opposing neurons which could not normally be activated at the same time.

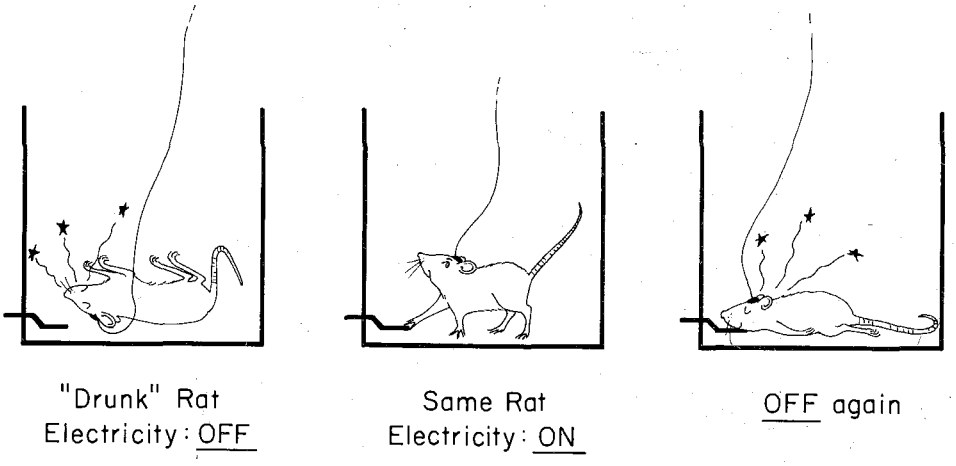
In mapping the brain areas that yielded pure positive and negative reinforcement, and those that yielded this mixed phenomenon, we found that input pathways to some of the brain nuclei would yield pure reinforcement of one sign—*either positive or negative*—and output pathways from these same areas would yield just the opposite effect; stimulation of the nuclear masses themselves would yield mixed positive-negative behavior. This suggested



Hungry rats, offered the alternatives of food and positively reinforcing brain stimulation, quickly renounce food almost altogether.



Rats will press one pedal as many as 100 times to get access to a second pedal with which to stimulate their brains—demonstrating that they will work for the reward of brain stimulation.



Several stimulations applied to a pleasure center in an animal prostrated by alcohol will restore muscle tone and awareness, and the animal will then continue to self-stimulate his brain for as long as current is available. When the current is cut off, the animal sinks back into his stupor.

inhibitory relations within the nuclear masses between positive and negative neuronal mechanisms. In one test we found an inhibitory chain where stimulation which was rewarding in a first area inhibited behavior related to a second area whose stimulation was aversive; stimulation in the second area which was aversive inhibited behavior related to a third area where stimulation was rewarding. One was plus, two was minus, and three was plus again. And one inhibited two, and two inhibited three. Stimulation of the third area rewarded the animal directly, and also augmented rewarding behavior when it was induced by stimulation of other parts of the brain. Much to our surprise, it also augmented punishment behavior induced by stimulating aversive points or when that behavior was induced by more normal means.

By these and further experiments we were led to the conclusion that mechanisms of positive and negative emotion interact with one another inhibitorially in the brain, in such a fashion that a predominance of one could inhibit the other, and vice versa. Furthermore, we were led to the conclusion that they might be acting through an area like "3." If 3 activity were augmented by rewarding stimuli and depressed by aversive stimuli, then 3 might derive an algebraic sum of rewards and punishments so that the animal would have a unitary state, somewhere between very good and very bad; and this would modify future behavior probabilities. This model generated interesting experiments which gave it some support, and it is still a viable theory held by me and some of my colleagues. But as with many good theories in the behavioral sciences, it is still in a state of limited probability.

The overlap of areas yielding positive or rewarding effects with areas where electric stimulation caused aversive reactions led us to wonder whether different drugs and different neuronal messenger chemicals might be involved in activating the two different kinds of neurons. As a first step toward testing for such differences, experiments were performed in which many different drugs were tested for the influence on self-stimulation behavior. The most interesting outcome of these tests was that use of a family of popular and intoxicating drugs repeatedly increased self-stimulation over escape behavior. Either these drugs didn't affect self-stimulation while abolishing escape behavior, or some of these drugs actually augmented self-stimulation behavior. We found that an animal which has been prostrated by a large dose of alcohol will lie flaccidly without muscle tone and yield no response when we apply aversive stimulation. Surprisingly, several stimulations applied through a self-stimulation electrode will restore muscle tone, and the animal will arise and self-stimulate for as long as the current is available. If the current is then turned off by the experimenter, the animal will quickly sink back into stupor and flaccidity.

Pentabarbitol, the favorite of sleeping pill enthusiasts, has effects remarkably like those of alcohol. Amphetamine, which activates many behaviors, also activates self-stimulation, and there are tests which strongly suggest that amphetamine has a particular relation to self-stimulation behavior. The relatively popular mild tranquilizers—Miltown and Librium—both also favor self-stimulation behavior over escape behavior; Librium and a family of drugs like it cause remarkable accelerations in self-stimulation behavior, even though this drug has a generally quieting effect on the animal.

It was surprising at first, but I suppose it should not have been, that the main drugs which are currently used to control agitation in the major psychoses—namely, chlorpromazine and reserpine—both have a highly selective effect against self-stimulation behavior. These drugs permit escape behaviors to continue in doses which totally abolish the rewarding effects of brain stimulation, or at least the resulting behaviors.

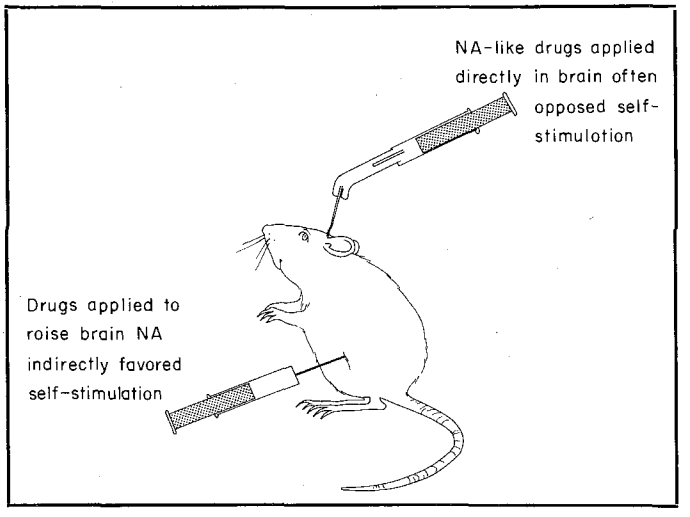
Studies at a more fundamental level have been directly

concerned with those chemicals which carry messages from nerve to nerve. The primary messenger, so far as current knowledge and speculation is concerned, is acetylcholine, abbreviated ACH. The secondary messenger, again so far as current evidence and speculation is concerned, is noradrenalin, abbreviated NA. Drugs applied to the rat for augmenting ACH in the brain generally decreased self-stimulation; this led to the speculation that ACH might be more important as a messenger in the negative or aversive systems. Drugs applied to augment NA in the brain regularly increased self-stimulation, so this secondary transmitter might be more important in the positive or rewarding apparatus.

Clear evidence that the problem would not be all that simple came from studies which showed a difference between direct and indirect augmentation of NA in the critical centers. Drugs which were applied peripherally to raise brain NA regularly increased self-stimulation. However, NA and NA-like drugs, when they were applied directly in the critical brain centers, often decreased or counteracted behavioral excitations which were caused by stimulating these centers. Furthermore, many recent studies have suggested the possibility that NA might be mainly an inhibitory chemical involved in counteracting rather than instigating neuronal activity.

Many of the drive-reduction theorists would be quick to jump to the suggestion that reward therefore might be mainly an inhibitory neuronal process, a process whereby one system of neurons utilizing norepinephrine would inhibit another set of neurons whose influence would be mainly energizing and perhaps even aversive. While this possibility is not totally unreasonable, I feel that our current knowledge of NA effects in the brain is advancing so rapidly that we must suspend judgment in this area. Progress is being spurred not only by our researches which connect NA to reward, but also by recent advances in many laboratories suggesting that NA and its close relative, serotonin, are very importantly involved in the control of sexual behavior and aggression; it appears possible that both sex and aggression are augmented by drugs which selectively depress levels of serotonin without simultaneously depressing levels of NA.

The paradoxical overlap of brain areas yielding



Noradrenalin (NA) is believed to be the secondary messenger in carrying information from nerve to nerve. When drugs which raise the level of NA in the brain are given to rats, self-stimulation is increased. However, when NA-like drugs are applied directly to the brain, the opposite effect occurs. Obviously, the chemical basis for positive and aversive responses is not simple.

radically different kinds of motivation, which first appeared in approach-escape tests, was further exhibited when the feeding centers in rats studied by Neal Miller and Jose Delgado were eventually studied with a view to understanding whether their stimulation would yield rewarding or possibly aversive effects. Before the rewarding tests were made, two important centers related to feeding were already known. These were roughly outlined topographic entities in the brain where the probability of affecting feeding behavior by destruction of brain tissues or by electric stimulation was at a highly likely level.

In one of these areas, known as the "satiety center," destruction of tissues caused animals to overeat and become obese. Careful studies of this phenomenon convinced scientists that this center was normally involved in the termination of eating behavior after the animal had

become satisfied. Whereas lesions caused eating to go on and on, stimulation in or near this center when it was not lesioned caused eating behavior to stop.

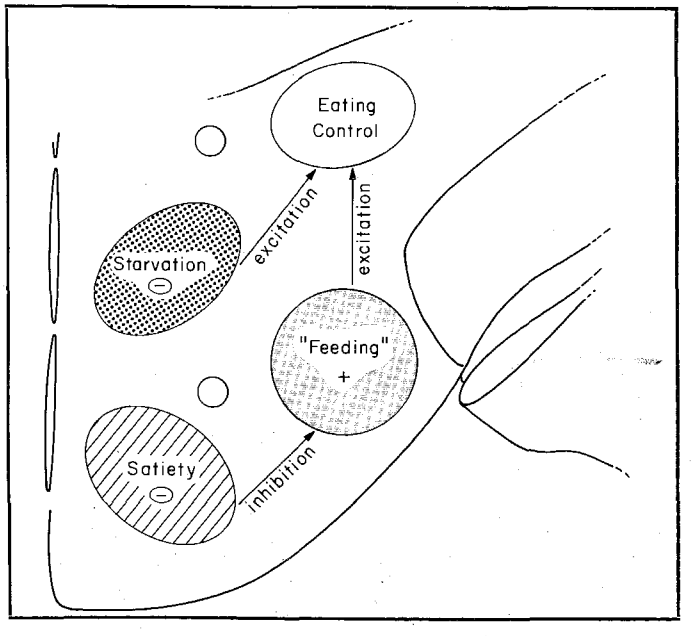
In a nearby area lies the second center related to feeding, where lesions cause the animal to stop eating altogether; unless the experimenter takes special care, these lesions cause the animal to die of starvation. Stimulation in or near these feeding center points causes the animal to eat voraciously during the period of stimulation.

Prior to the entry of our work into this field, I believe a relatively simple interaction was assumed. One view was that when neurons in the *lateral feeding center* became excited, a state of high drive (an aversive condition for the organism) goaded the animal into eating behavior. The ingestion of food, on the other hand, would be detected by receptors in the mouth and in the stomach, and would also modify the chemical state of the blood, and this information would be processed and projected to the *medial satiety center*, where it might be supposed to cause a positive state of the animal, and to inhibit the aversive lateral drive mechanism.

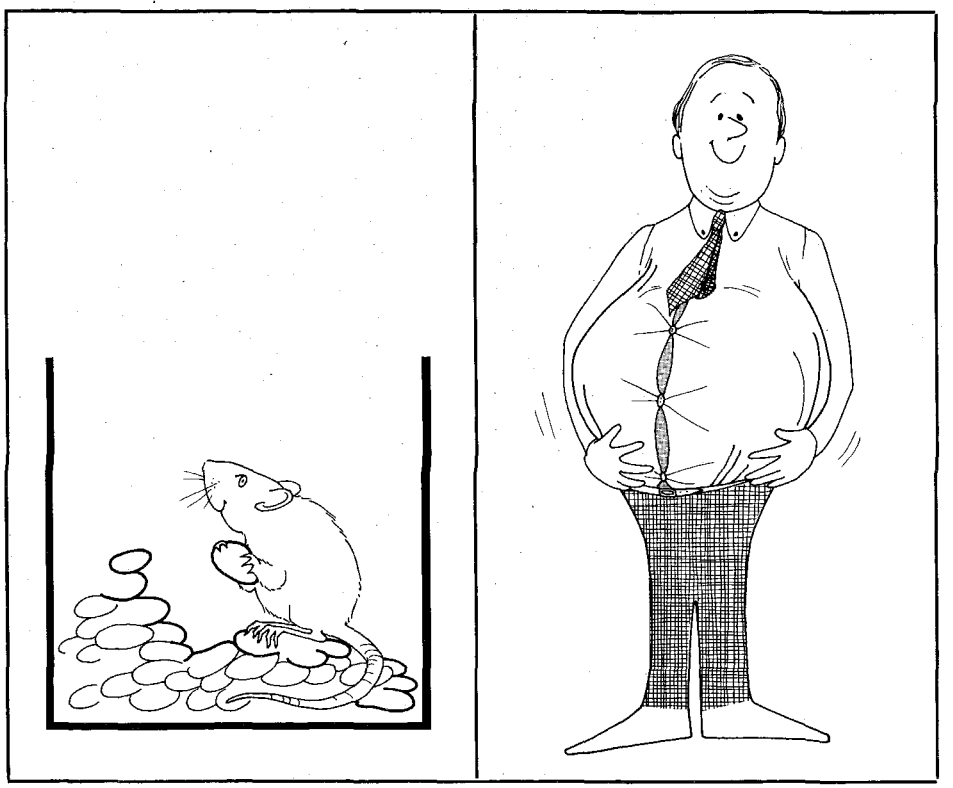
One of the most surprising findings to date, and one which has dramatically changed the concept of the control of eating behavior, and therefore the control of obesity, was the discovery that stimulation of the feeding center was among those yielding the strongest self-stimulation behavior and on all of our measures the strongest kind of positive rewarding effects. As you might or might not guess, the electric stimulation that caused satiety—and therefore was expected to cause bliss—did not induce any positive reactions at all, but rather turned out to be one of the areas where electric stimulation produced prompt aversive or withdrawal reactions.

So we no longer see hunger as a simple aversive mechanism, or as an aversive mechanism at all. Instead we would say that eating is a positive feedback mechanism. That is, eating behavior, once triggered, tends to continue. Eating begets eating, and once it gets going it has a marked tendency to intensify itself. This is experienced from the point of view of the Epicurean subject as a very satisfactory state of affairs. However, it can be a very sad state of affairs for the person who wants to lose weight and has weak limiting centers. Because food engenders a self-reactivating drive, we now know that the main cure for this (main) kind of obesity is simply to get and keep the patient away from food stimuli.

We conceive of two limiting centers brought to bear on the feeding process. One is the satiety center, which meters the input in one way or another, and gradually or abruptly converts the process from a rewarding one into a neutral and finally into an aversive one, so that eating which was rewarding at the beginning is negatively reinforcing at the end. We are now convinced that there is another center which comes very little into play in the white albino rat, or in the majority of the American college students, and this we might think of as a starvation center. We have only recently found intimations of this; it is not too far displaced from the other two long-known topographic entities related to feeding. Within this area electric stimulation causes eating behavior, but in this



It appears that there are two limiting centers bearing on the feeding process. The satiety center meters the input and converts the process from rewarding to neutral to aversive. A starvation center produces an aversive mechanism when the animal's food supply reaches a danger level.



Creatures with hoards—the rat's food pellets or a man's fat—have been able to survive the lean years. The animal that waits to eat until he is starving is always living on the edge of demise.

case the stimulation is either neutral or aversive in its effects on behavior.

We now conceive of hunger as being instigated either by accidental encounters between the subject and the succulent stimuli emanating from the food, or, barring that, eventually triggered by an aversive mechanism brought into play when the animal reaches a danger level so far as food supplies are concerned. In either case, once the eating mechanism has been triggered, it moves forward under its own power and would go on indefinitely if other extraneous control devices were not brought to bear. The satiety mechanism of the medial hypothalamus represents precisely this kind of a control device.

This research formed the model for a set of researches on the other drives; now a drinking center and a sexual center have been added to the array of vague entities in the lateral hypothalamus. In these regions other consummatory behaviors are triggered by electric stimulation, and a common denominator among all of the drive centers so far discovered has been that the electric stimulations there are also yielding positive rewarding effects on behavior.

So we assume that positive emotional mechanisms are indeed involved in the control of behavior—but why do they exist? Why were animals not just as fit to survive if they were nudged ahead by their pains and their deficits?

I believe the clue lies in our analysis of feeding mechanisms. Why does the animal keep on eating after the starvation trigger is gone? Why does the animal start eating even if he is not starving, but is only stimulated by the sight or smell or taste of food? The answer could well be that creatures with hoards, whether these were laid up at home as the rat hoards pellets in his home cage, or laid up within the animal's body as man often keeps his pounds of fat on his midriff, were able to survive the lean years. Therefore, in relation to certain objects not so plentiful that they would be available when needed, mechanisms for hoarding promoted the survival of the species. The abstract animal (who never lived so far as I can make out in phylogenetic history) who waited until demise was imminent and then began looking to satisfy his need was on the edge of demise at all times. You might say he was "just" living. His lucky cousin, who is no abstraction, stocked up his larder during the fat years, in preparation for the lean ones, and you might say he enjoyed it. Instead of "just" living, the positive reinforcement creature was *really* living.