conclude that we do not need one, we remember judgments made by very competent analysts in the late 1940's and early 1950's to the effect that, if there existed ten computers that could execute instructions at a rate of 1000 per second, then these ten, together, could solve all the problems anyone could possibly imagine. The crux of the matter seems to be that, when an apparently useful device becomes available, the fact of its availability sets a great many more people thinking about the applications than addressed that question during its development. P. E. Haggerty of the Texas Instruments Company remarked, in an address several years ago (*10*), that electronics will become "pervasive." Automated production of large-scale integrated electronics systems is a force making this pervasiveness possible.

Let me list some of the areas in which this "pervasiveness" is now either a fact or an expectation. There is the ubiquitous computer terminal at airline reservation desks. Undergoing rapid development are the terminal in contact with a high-powered computer system for the scientific user; the medical monitoring terminal coupled to the hospital's central computer; management information systems that enable executives to call for digested business data on the basis of which they can make rapid decisions; and, of course, various computer-assisted education programs. We can look forward to the development of computer terminals that will assist doctors in making diagnoses and recording data; to the development of on-line recorders of business transactions, for more effective retail operations; and, eventually, to virtual elimination of the need for money in the form of cash. The reader can extend this list almost indefinitely; the question I contemplate with interest is this: What impact will really inexpensive electronics have on the ingenious toy designers and the generation of young programmers that will follow?

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11. I am indebted to many people for very stimulating discussions on the subject of large-scale integration and the revolution in electronics; the individuals who influenced my thinking the most were H. Freitag, D. E. Rosenheim, and M. G. Smith of our laboratory at the IBM Thomas J. Watson Research Center.

Learning of Visceral and Glandular Responses

Recent experiments on animals show the fallacy of an ancient view of the autonomic nervous system.

Neal E. Miller

There is a strong traditional belief in the inferiority of the autonomic nervous system and the visceral responses that it controls. The recent experiments disproving this belief have deep implications for theories of learning, for individual differences in autonomic responses, for the cause and the cure of abnormal psychosomatic symptoms, and possibly also for the understanding of normal homeostasis. Their success encourages investigators to try other unconventional types of training. Before describing these experiments, let me briefly sketch some elements in the history of the deeply entrenched, false belief in the gross inferiority of one major part of the nervous system.

Historical Roots and Modern Ramifications

Since ancient times, reason and the voluntary responses of the skeletal muscles have been considered to be superior, while emotions and the presumably involuntary glandular and visceral responses have been considered to be inferior. This invidious dichotomy appears in the philosophy of Plato (*1*), with his superior rational soul in the head above and inferior souls in the body below. Much later, the great French neuroanatomist Bichat (*2*) distinguished between the cerebrospinal nervous system of the great brain and spinal cord, controlling skeletal responses, and the dual chain of ganglia (which he called "little brains") running down on either side of the spinal cord in the body below and controlling emotional and visceral responses. He indicated his low opinion of the ganglionic system by calling it "vegetative"; he also believed it to be largely independent of the cerebrospinal system, an opinion which is still reflected in our modern name for it, the autonomic nervous system. Considerably later, Cannon (*3*) studied the sympathetic part of the autonomic nervous system and concluded that the different nerves in it all fire simultaneously and are incapable of the finely differentiated individual responses possible for the cerebrospinal system, a conclusion which is enshrined in modern textbooks.

Many, though not all, psychiatrists have made an invidious distinction between the hysterical and other symptoms

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that are mediated by the cerebrospinal nervous system and the psychosomatic symptoms that are mediated by the autonomic nervous system. Whereas the former are supposed to be subject to a higher type of control that is symbolic, the latter are presumed to be only the direct physiological consequences of the type and intensity of the patient's emotions (see, for example, *4*).

Similarly, students of learning have made a distinction between a lower form, called classical conditioning and thought to be involuntary, and a superior form variously called trial-and-error learning, operant conditioning, type II conditioning, or instrumental learning and believed to be responsible for voluntary behavior. In classical conditioning, the reinforcement must be by an unconditioned stimulus that already elicits the specific response to be learned; therefore, the possibilities are quite limited. In instrumental learning, the reinforcement, called a reward, has the property of strengthening any immediately preceding response. Therefore, the possibilities for reinforcement are much greater; a given reward may reinforce any one of a number of different responses, and a given response may be reinforced by any one of a number of different rewards.

Finally, the foregoing invidious distinctions have coalesced into the strong traditional belief that the superior type of instrumental learning involved in the superior voluntary behavior is possible only for skeletal responses mediated by the superior cerebrospinal nervous system, while, conversely, the inferior classical conditioning is the only kind possible for the inferior, presumably involuntary, visceral and emotional responses mediated by the inferior autonomic nervous system. Thus, in a recent summary generally considered authoritative, Kimble (*5*) states the almost universal belief that "for autonomically mediated behavior, the evidence points unequivocally to the conclusion that such responses can be modified by classical, but not instrumental, training methods." Upon examining the evidence, however, one finds that it consists only of failure to secure instrumental learning in two incompletely reported exploratory experiments and a vague allusion to the Russian literature (*6*). It is only against a cultural background of great prejudice that such weak evidence could lead to such a strong conviction.

The belief that instrumental learning is possible only for the cerebrospinal system and, conversely, that the autonomic nervous system can be modified only by classical conditioning has been used as one of the strongest arguments for the notion that instrumental learning and classical conditioning are two basically different phenomena rather than different manifestations of the same phenomenon under different conditions. But for many years I have been impressed with the similarity between the laws of classical conditioning and those of instrumental learning, and with the fact that, in each of these two situations, some of the specific details of learning vary with the specific conditions of learning. Failing to see any clear-cut dichotomy, I have assumed that there is only one kind of learning (*7*). This assumption has logically demanded that instrumental training procedures be able to produce the learning of any visceral responses that could be acquired through classical conditioning procedures. Yet it was only a little over a dozen years ago that I began some experimental work on this problem and a somewhat shorter time ago that I first, in published articles (*8*), made specific sharp challenges to the traditional view that the instrumental learning of visceral responses is impossible.

Some Difficulties

One of the difficulties of investigating the instrumental learning of visceral responses stems from the fact that the responses that are the easiest to measure —namely, heart rate, vasomotor responses, and the galvanic skin response —are known to be affected by skeletal responses, such as exercise, breathing, and even tensing of certain muscles, such as those in the diaphragm. Thus, it is hard to rule out the possibility that, instead of directly learning a visceral response, the subject has learned a skeletal response the performance of which causes the visceral change being recorded.

One of the controls I planned to use was the paralysis of all skeletal responses through administration of curare, a drug which selectively blocks the motor end plates of skeletal muscles without eliminating consciousness in human subjects or the neural control of visceral responses, such as the beating of the heart. The muscles involved in breathing are paralyzed, so the subject's breathing must be maintained through artificial respiration. Since it seemed unlikely that curarization and other rigorous control techniques would be easy to use with human subjects, I decided to concentrate first on experiments with animals.

Originally I thought that learning would be more difficult when the animal was paralyzed, under the influence of curare, and therefore I decided to postpone such experiments until ones on nonparalyzed animals had yielded some definitely promising results. This turned out to be a mistake because, as I found out much later, paralyzing the animal with curare not only greatly simplifies the problem of recording visceral responses without artifacts introduced by movement but also apparently makes it easier for the animal to learn, perhaps because paralysis of the skeletal muscles removes sources of variability and distraction. Also, in certain experiments I made the mistake of using rewards that induced strong unconditioned responses that interfered with instrumental learning.

One of the greatest difficulties, however, was the strength of the belief that instrumental learning of glandular and visceral responses is impossible. It was extremely difficult to get students to work on this problem, and when paid assistants were assigned to it, their attempts were so half-hearted that it soon became more economical to let them work on some other problem which they could attack with greater faith and enthusiasm. These difficulties and a few preliminary encouraging but inconclusive early results have been described elsewhere (*9*).

Success with Salivation

The first clear-cut results were secured by Alfredo Carmona and me in an experiment on the salivation of dogs. Initial attempts to use food as a reward for hungry dogs were unsuccessful, partly because of strong and persistent unconditioned salivation elicited by the food. Therefore, we decided to use water as a reward for thirsty dogs. Preliminary observations showed that the water had no appreciable effects one way or the other on the bursts of spontaneous salivation. As an additional precaution, however, we used the experimental design of rewarding dogs in one group whenever they showed a burst of spontaneous salivation, so that they would be trained to increase salivation, and rewarding dogs in another group whenever there was a long interval between spontaneous bursts, so that they would be trained to decrease salivation. If the reward had any unconditioned effect, this effect might be classically conditioned to the experimental situation and therefore produce a change in salivation that was not a true instance of instrumental learning. But in classical conditioning the reinforcement must elicit the response that is to be acquired. Therefore, conditioning of a response elicited by the reward could produce either an increase or a decrease in salivation, depending upon the direction of the unconditioned response elicited by the reward, but it could not produce a change in one direction for one group and in the opposite direction for the other group. The same type of logic applies for any unlearned cumulative aftereffects of the reward; they could not be in opposite directions for the two groups. With instrumental learning, however, the reward can reinforce any response that immediately precedes it; therefore, the same reward can be used to produce either increases or decreases.

The results are presented in Fig. 1, which summarizes the effects of 40 days of training with one 45-minute training session per day. It may be seen that in this experiment the learning proceeded slowly. However, statistical analysis showed that each of the trends in the predicted rewarded direction was highly reliable (*10*).

Since the changes in salivation for the two groups were in opposite directions, they cannot be attributed to classical conditioning. It was noted, however, that the group rewarded for increases seemed to be more aroused and active than the one rewarded for decreases. Conceivably, all we were doing was to change the level of activation of the dogs, and this change was, in turn, affecting the salivation. Although we did not observe any specific skeletal responses, such as chewing movements or panting, which might be expected to elicit salivation, it was difficult to be absolutely certain that such movements did not occur. Therefore, we decided to rule out such movements by paralyzing the dogs with curare, but we immediately found that curare had two effects which were diastrous for this experiment: it elicited such copious and continuous salivation that there were no changes in salivation to reward, and the salivation was so viscous that it almost immediately gummed up the recording apparatus.

Heart Rate

In the meantime, Jay Trowill, working with me on this problem, was displaying great ingenuity, courage, and persistence in trying to produce instrumental learning of heart rate in rats that had been paralyzed by curare to prevent them from "cheating" by muscular exertion to speed up the heart or by relaxation to slow it down. As a result of preliminary testing, he selected a dose of curare (3.6 milligrams of *d*-tubocurarine chloride per kilogram, injected intraperitoneally) which produced deep paralysis for at least 3 hours, and a rate of artificial respiration (inspiration-expiration ratio 1:1; 70 breaths per minute; peak pressure reading, 20 cm-H_2O) which maintained the heart at a constant and normal rate throughout this time.

In subsequent experiments, DiCara and I have obtained similar effects by starting with a smaller dose (1.2 milligrams per kilogram) and constantly infusing additional amounts of the drug, through intraperitoneal injection, at the rate of 1.2 milligrams per kilogram per hour, for the duration of the experiment. We have recorded, electromyographically, the response of the muscles, to determine that this dose does indeed produce a complete block of the action potentials, lasting for at least an hour after the end of infusion. We have found that if parameters of respiration and the face mask are adjusted carefully, the procedure not only maintains the heart rate of a 500-gram control animal constant but also maintains the vital signs of temperature, peripheral vasomotor responses, and the $p\mathrm{CO}_2$ of the blood constant.

Since there are not very many ways to reward an animal completely paralyzed by curare, Trowill and I decided to use direct electrical stimulation of rewarding areas of the brain. There were other technical difficulties to overcome, such as devising the automatic system for rewarding small changes in heart rate as recorded by the electrocardiogram. Nevertheless, Trowill at last succeeded in training his rats (*11*). Those rewarded for an increase in heart rate showed a statistically reliable increase, and those rewarded for a decrease in heart rate showed a statistically reliable decrease. The changes, however, were disappointingly small, averaging only 5 percent in each direction.

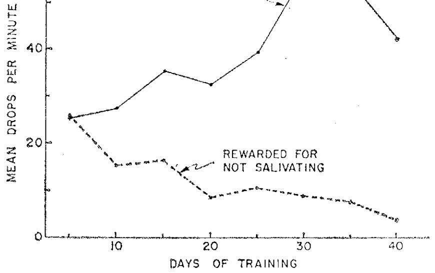


Fig. 1. Learning curves for groups of thirsty dogs rewarded with water for either increases or decreases in spontaneous salivation. [From Miller and Carmona (*10*)]

The next question was whether larger changes could be achieved by improving the technique of training. DiCara and I used the technique of shaping—in other words, of immediately rewarding first very small, and hence frequently occurring, changes in the correct direction and, as soon as these had been learned, requiring progressively larger changes as the criterion for reward. In this way, we were able to produce in 90 minutes of training changes averaging 20 percent in either direction (*12*).

Key Properties of Learning: Discrimination and Retention

Does the learning of visceral responses have the same properties as the learning of skeletal responses? One of the important characteristics of the instrumental learning of skeletal responses is that a discrimination can be learned, so that the responses are more likely to be made in the stimulus situations in which they are rewarded than in those in which they are not. After the training of the first few rats had convinced us that we could produce large changes in heart rate, DiCara and I gave all the rest of the rats in the experiment described above 45 minutes of additional training with the most difficult criterion. We did this in order to see whether they could learn to give a greater response during a "time-in" stimulus (the presence of a flashing light and a tone) which indicated that a response in the proper direction would be rewarded than during a "time-out" stimulus (absence of light and tone) which indicated that a correct response would not be rewarded.

Figure 2 shows the record of one of the rats given such training. Before the beginning of the special discrimination training it had slowed its heart from an initial rate of 350 beats per minute to a rate of 230 beats per minute. From the top record of Fig. 2 one can see that, at the beginning of the special discrimination training, there was no appreciable reduction in heart rate that was specifically associated with the time-in stimulus. Thus it took the rat considerable time after the onset of this stimulus to meet the criterion and get the reward. At the end of the discrimination training the heart rate during time-out remained approximately the same, but when the time-in light and tone came on, the heart slowed down and the criterion was promptly met. Although the other rats showed less change than this, by the end of the relatively short period of discrimination training their heart rate did change reliably ($P < .001$) in the predicted direction when the time-in stimulus came on. Thus, it is clear that instrumental visceral learning has at least one of the important properties of instrumental skeletal learning—namely, the ability to be brought under the control of a discriminative stimulus.

Another of the important properties of the instrumental learning of skeletal responses is that it is remembered. DiCara and I performed a special experiment to test the retention of learned changes in heart rate (*13*). Rats that had been given a single training session were returned to their home cages for 3 months without further training. When curarized again and returned to the experimental situation for nonreinforced test trials, rats in both the "increase" and the "decrease" groups showed good retention by exhibiting reliable changes in the direction rewarded in the earlier training.

Escape and Avoidance Learning

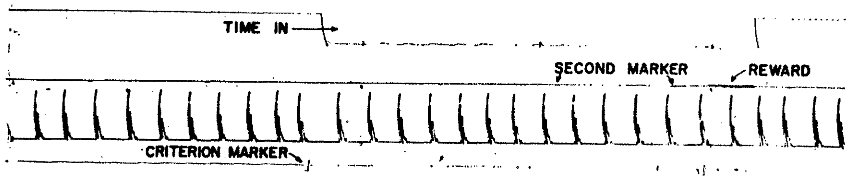
Is visceral learning by any chance peculiarly limited to reinforcement by the unusual reward of direct electrical stimulation of the brain, or can it be reinforced by other rewards in the same way that skeletal learning can be? In order to answer this question, DiCara and I (*14*) performed an experiment using the other of the two forms of thoroughly studied reward that can be conveniently used with rats which are paralyzed by curare—namely, the chance to avoid, or escape from, mild electric shock. A shock signal was turned on; after it had been on for 10 seconds it was accompanied by brief pulses of mild electric shock delivered to the rat's tail. During the first 10 seconds the rat could turn off the shock signal and avoid the shock by making the correct response of changing its heart rate in the required direction by the required amount. If it did not make the correct response in time, the shocks continued to be delivered until the rat escaped them by making the correct response, which immediately turned off both the shock and the shock signal.

For one group of curarized rats, the correct response was an increase in heart rate; for the other group it was a decrease. After the rats had learned to make small responses in the proper direction, they were required to make larger ones. During this training the shock signals were randomly interspersed with an equal number of "safe" signals that were not followed by shock; the heart rate was also recorded during so-called blank trials—trials without any signals or shocks. For half of the rats the shock signal was a tone and the "safe" signal was a flashing light; for the other half the roles of these cues were reversed.

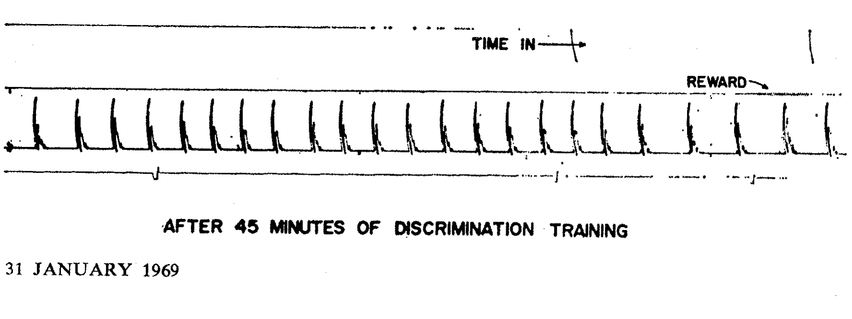
The results are shown in Fig. 3. Each of the 12 rats in this experiment changed its heart rate in the rewarded direction. As training progressed, the shock signal began to elicit a progressively greater change in the rewarded direction than the change recorded during the blank trials; this was a statistically reliable trend. Conversely, as training progressed, the "safe" signal came to elicit a statistically reliable change in the opposite direction, toward the initial base line. These results show learning when escape and avoidance are the rewards; this means that visceral responses in curarized rats can be reinforced by rewards other than direct electrical stimulation of the brain. These rats also discriminate between the shock and the "safe" signals. You will remember that, with noncurarized thirsty dogs, we were able to use yet another kind of reward, water, to produce learned changes in salivation.

Transfer to Noncurarized State: More Evidence against Mediation

In the experiments discussed above, paralysis of the skeletal muscles by curare ruled out the possibility that the subjects were learning the overt performance of skeletal responses which were indirectly eliciting the changes in the heart rate. It is barely conceivable, however, that the rats were learning to send out from the motor cortex central impulses which would have activated the muscles had they not been paralyzed. And it is barely conceivable that



BEGINNING OF DISCRIMINATION TRAINING



AFTER 45 MINUTES OF DISCRIMINATION TRAINING

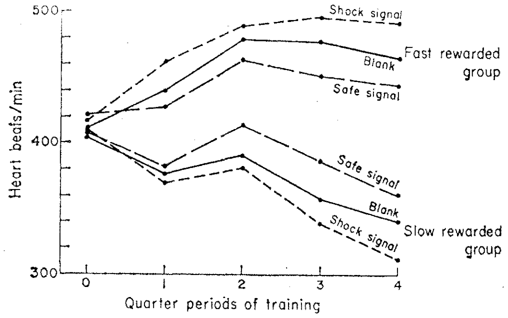


Fig. 2 (left). Electrocardiograms at the beginning and at the end of discrimination training of curarized rat rewarded for slow heart rate. Slowing of heart rate is rewarded only during a "time-in" stimulus (tone and light). [From Miller and DiCara (*12*)] Fig. 3 (above). Changes in heart rate during avoidance training. [From DiCara and Miller (*14*)]

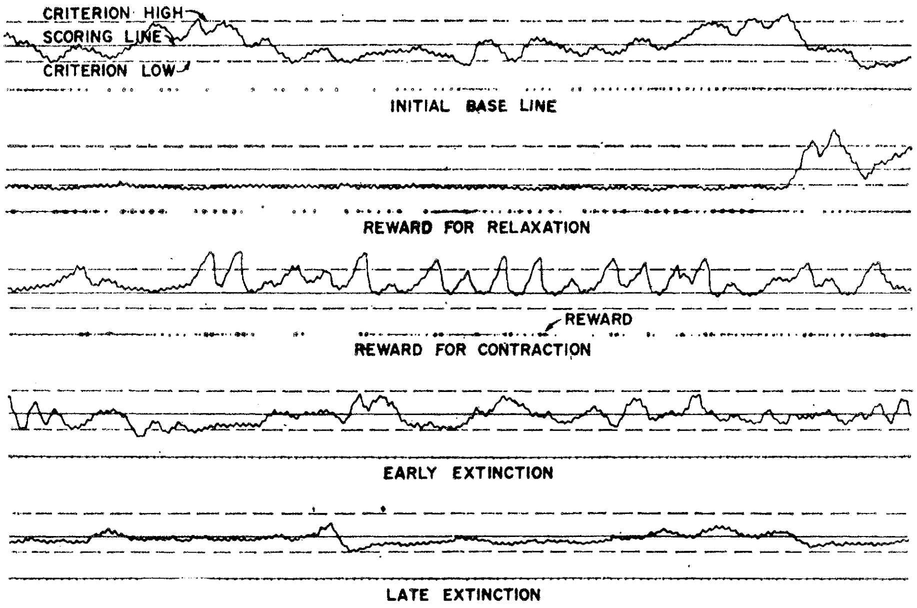


Fig. 4. Typical samples of a record of instrumental learning of an intestinal response by a curarized rat. (From top to bottom) Record of spontaneous contraction before training; record after training with reward for relaxation; record after training with reward for contractions; records during nonrewarded extinction trials. [From Miller and Banuazizi (*18*)]

these central impulses affected heart rate by means either of inborn connections or of classically conditioned ones that had been acquired when previous exercise had been accompanied by an increase in heart rate and relaxation had been accompanied by a decrease. But, if the changes in heart rate were produced in this indirect way, we would expect that, during a subsequent test without curare, any rat that showed learned changes in heart rate would show the movements in the muscles that were no longer paralyzed. Furthermore, the problem of whether or not visceral responses learned under curarization carry over to the noncurarized state is of interest in its own right.

In order to answer this question, DiCara and I (*15*) trained two groups of curarized rats to increase or decrease, respectively, their heart rate in order to avoid, or escape from, brief pulses of mild electric shock. When these rats were tested 2 weeks later in the noncurarized state, the habit was remembered. Statistically reliable increases in heart rate averaging 5 percent and decreases averaging 16 percent occurred. Immediately subsequent retraining without curare produced additional significant changes of heart rate in the rewarded direction, bringing the total overall increase to 11 percent and the decrease to 22 percent. While, at the beginning of the test in the noncurarized state, the two groups showed some differences in respiration and activity, these differences decreased until, by the end of the retraining, they were small and far from statistically reliable ($t =$ 0.3 and 1.3, respectively). At the same time, the difference between the two groups with respect to heart rate was increasing, until it became large and thus extremely reliable ($t = 8.6$, d.f. = 12, $P < .001$).

In short, while greater changes in heart rate were being learned, the response was becoming more specific, involving smaller changes in respiration and muscular activity. This increase in specificity with additional training is another point of similarity with the instrumental learning of skeletal responses. Early in skeletal learning, the rewarded correct response is likely to be accompanied by many unnecessary movements. With additional training during which extraneous movements are not rewarded, they tend to drop out.

It is difficult to reconcile the foregoing results with the hypothesis that the differences in heart rate were mediated primarily by a difference in either respiration or amount of general activity. This is especially true in view of the research, summarized by Ehrlich and Malmo (*16*), which shows that muscular activity, to affect heart rate in the rat, must be rather vigorous.

While it is difficult to rule out completely the possibility that changes in heart rate are mediated by central impulses to skeletal muscles, the possibility of such mediation is much less attractive for other responses, such as intestinal contractions and the formation of urine by the kidney. Furthermore, if the learning of these different responses can be shown to be specific in enough visceral responses, one runs out of different skeletal movements each eliciting a specific different visceral response (*17*). Therefore, experiments were performed on the learning of a variety of different visceral responses and on the specificity of that learning. Each of these experiments was, of course, interesting in its own right, quite apart from any bearing on the problem of mediation.

Specificity: Intestinal versus Cardiac

The purpose of our next experiment was to determine the specificity of visceral learning. If such learning has the same properties as the instrumental learning of skeletal responses, it should be possible to learn a specific visceral response independently of other ones. Furthermore, as we have just seen, we might expect to find that, the better the rewarded response is learned, the more specific is the learning. Banuazizi and I worked on this problem (*18*). First we had to discover another visceral response that could be conveniently recorded and rewarded. We decided on intestinal contractions, and recorded them in the curarized rat with a little balloon filled with water thrust approximately 4 centimeters beyond the anal sphincter. Changes of pressure in the balloon were transduced into electric voltages which produced a record on a polygraph and also activated an automatic mechanism for delivering the reward, which was electrical stimulation of the brain.

The results for the first rat trained, which was a typical one, are shown in Fig. 4. From the top record it may be seen that, during habituation, there were some spontaneous contractions. When the rat was rewarded by brain stimulation for keeping contractions below a certain amplitude for a certain time, the number of contractions was reduced and the base line was lowered. After the record showed a highly reliable change indicating that relaxation had been learned (Fig. 4, second record from the top), the conditions of training were reversed and the reward was delivered whenever the amplitude of contractions rose above a certain level. From the next record (Fig. 4, middle) it may be seen that this type of training increased the number of contractions and raised the base line. Finally (Fig. 4, two bottom records) the reward was discontinued and, as would be expected, the response continued for a while but gradually became extinguished, so that the activity eventually returned to approximately its original base-line level.

After studying a number of other rats in this way and convincing ourselves that the instrumental learning of intestinal responses was a possibility, we designed an experiment to test specificity. For all the rats of the experiment, both intestinal contractions and heart rate were recorded, but half the rats were rewarded for one of these responses and half were rewarded for the other response. Each of these two groups of rats was divided into two subgroups, rewarded, respectively, for increased and decreased response. The rats were completely paralyzed by curare, maintained on artificial respiration, and rewarded by electrical stimulation of the brain.

The results are shown in Figs. 5 and 6. In Fig. 5 it may be seen that the group rewarded for increases in intestinal contractions learned an increase, the group rewarded for decreases learned a decrease, but neither of these groups showed an appreciable change in heart rate. Conversely (Fig. 6), the group rewarded for increases in heart rate showed an increase, the group rewarded for decreases showed a decrease, but neither of these groups showed a change in intestinal contractions.

The fact that each type of response changed when it was rewarded rules out the interpretation that the failure to secure a change when that change was not rewarded could have been due to either a strong and stable homeostatic regulation of that response or an inability of our techniques to measure changes reliably under the particular conditions of our experiment.

Each of the 12 rats in the experiment showed statistically reliable changes in the rewarded direction; for 11 the changes were reliable beyond the $P <$ .001 level, while for the 12th the changes were reliable only beyond the .05 level. A statistically reliable negative correlation showed that the better the rewarded visceral response was learned, the less change occurred in the other, nonrewarded response. This greater specificity with better learning is what we had expected. The results showed that visceral learning can be specific to an organ system, and they clearly ruled out the possibility of mediation by any single general factor, such as level of activation or central commands for either general activity or relaxation.

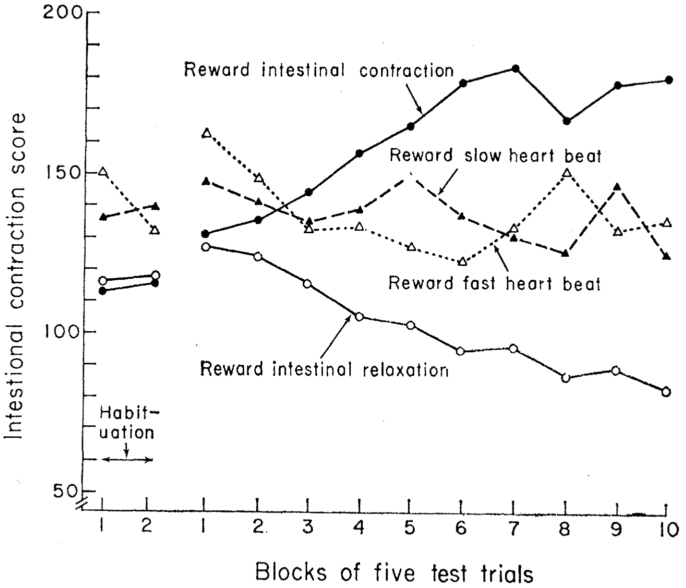
In an additional experiment, Banuazizi (*19*) showed that either increases or decreases in intestinal contractions can be rewarded by avoidance of, or escape from, mild electric shocks, and that the intestinal responses can be discriminatively elicited by a specific stimulus associated with reinforcement.

Kidney Function

Encouraged by these successes, DiCara and I decided to see whether or not the rate of urine formation by the kidney could be changed in the curarized rat rewarded by electrical stimulation of the brain (*20*). A catheter, permanently inserted, was used to prevent accumulation of urine by the bladder, and the rate of urine formation was measured by an electronic device for counting minute drops. In order to secure a rate of urine formation fast enough so that small changes could be promptly detected and rewarded, the rats were kept constantly loaded with water through infusion by way of a catheter permanently inserted in the jugular vein.

All of the seven rats rewarded when the intervals between times of urine-drop formation lengthened showed decreases in the rate of urine formation, and all of the seven rats rewarded when these intervals shortened showed increases in the rate of urine formation. For both groups the changes were highly reliable ($P < .001$).

In order to determine how the change in rate of urine formation was achieved, certain additional measures were taken. As the set of bars at left in Fig. 7 shows, the rate of filtration, measured by means of ^{14}C-labeled inulin, increased when increases in the rate of urine formation were rewarded and decreased when decreases in the rate were rewarded. Plots of the correlations



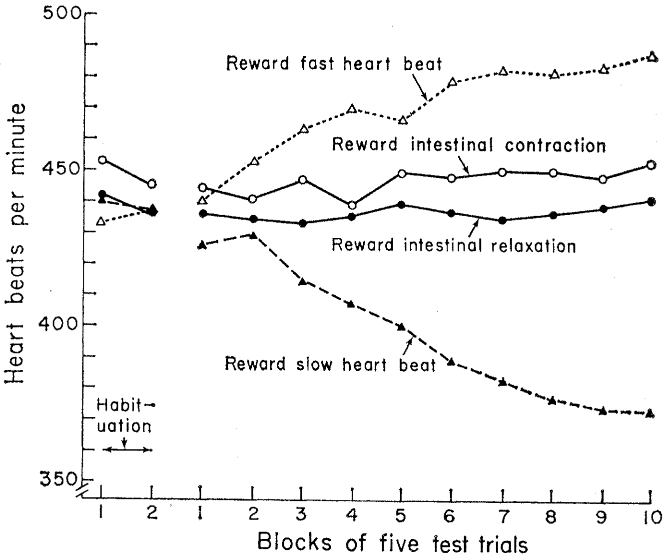


Fig. 5 (left). Graph showing that the intestinal contraction score is changed by rewarding either increases or decreases in intestinal contractions but is unaffected by rewarding changes in heart rate. [From Miller and Banuazizi (*18*)] Fig. 6 (right). Graph showing that the heart rate is changed by rewarding either increases or decreases in heart rate but is unaffected by rewarding changes in intestinal contractions. Comparison with Fig. 5 demonstrates the specificity of visceral learning. [From Miller and Banuazizi (*18*)]

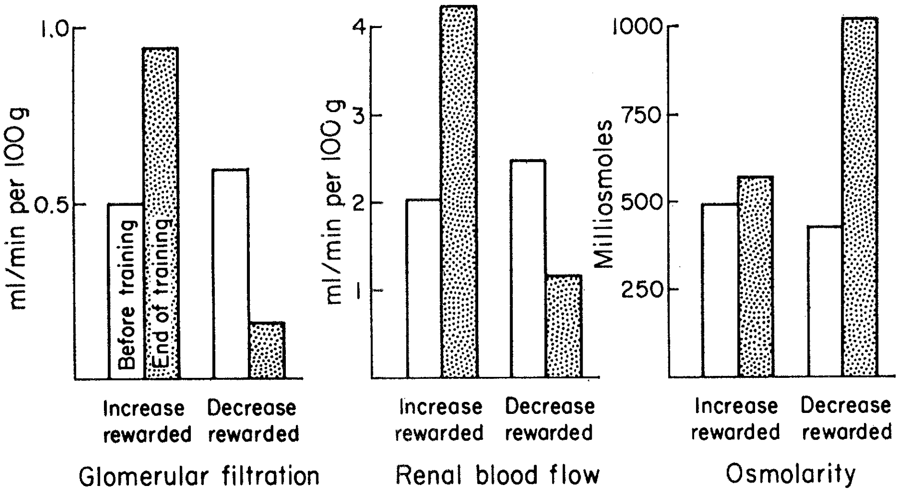


Fig. 7. Effects of rewarding increased rate of urine formation in one group and decreased rate in another on measures of glomerular filtration, renal blood flow, and osmolarity. [From data in Miller and DiCara (*20*)]

showed that the changes in the rates of filtration and urine formation were not related to changes in either blood pressure or heart rate.

The middle set of bars in Fig. 7 shows that the rats rewarded for increases in the rate of urine formation had an increased rate of renal blood flow, as measured by ^{3}H-*p*-aminohippuric acid, and that those rewarded for decreases had a decreased rate of renal blood flow. Since these changes in blood flow were not accompanied by changes in general blood pressure or in heart rate, they must have been achieved by vasomotor changes of the renal arteries. That these vasomotor changes were at least somewhat specific is shown by the fact that vasomotor responses of the tail, as measured by a photoelectric plethysmograph, did not differ for the two groups of rats.

The set of bars at right in Fig. 7 shows that when decreases in rate of urine formation were rewarded, a more concentrated urine, having higher osmolarity, was formed. Since the slower passage of urine through the tubules would afford more opportunity for reabsorption of water, this higher concentration does not necessarily mean an increase in the secretion of antidiuretic hormone. When an increased rate of urine formation was rewarded, the urine did not become more diluted—that is, it showed no decrease in osmolarity; therefore, the increase in rate of urine formation observed in this experiment cannot be accounted for in terms of an inhibition of the secretion of antidiuretic hormone.

From the foregoing results it appears that the learned changes in urine formation in this experiment were produced primarily by changes in the rate of filtration, which, in turn, were produced primarily by changes in the rate of blood flow through the kidneys.

Gastric Changes

In the next experiment, Carmona, Demierre, and I used a photoelectric plethysmograph to measure changes, presumably in the amount of blood, in the stomach wall (*21*). In an operation performed under anesthesia, a small glass tube, painted black except for a small spot, was inserted into the rat's stomach. The same tube was used to hold the stomach wall against a small glass window inserted through the body wall. The tube was left in that position. After the animal had recovered, a bundle of optical fibers could be slipped snugly into the glass tube so that the light beamed through it would shine out through the unpainted spot in the tube inside the stomach, pass through the stomach wall, and be recorded by a photocell on the other side of the glass window. Preliminary tests indicated that, as would be expected, when the amount of blood in the stomach wall increased, less light would pass through. Other tests showed that stomach contractions elicited by injections of insulin did not affect the amount of light transmitted.

In the main experiment we rewarded curarized rats by enabling them to avoid or escape from mild electric shocks. Some were rewarded when the amount of light that passed through the stomach wall increased, while others were rewarded when the amount decreased. Fourteen of the 15 rats showed changes in the rewarded direction. Thus, we demonstrated that the stomach wall, under the control of the autonomic nervous system, can be modified by instrumental learning. There is strong reason to believe that the learned changes were achieved by vasomotor responses affecting the amount of blood in the stomach wall or mucosa, or in both.

In another experiment, Carmona (*22*) showed that stomach contractions can be either increased or decreased by instrumental learning.

It is obvious that learned changes in the blood supply of internal organs can affect their functioning—as, for example, the rate at which urine was formed by the kidneys was affected by changes in the amount of blood that flowed through them. Thus, such changes can produce psychosomatic symptoms. And if the learned changes in blood supply can be specific to a given organ, the symptom will occur in that organ rather than in another one.

Peripheral Vasomotor Responses

Having investigated the instrumental learning of internal vasomotor responses, we next studied the learning of peripheral ones. In the first experiment, the amount of blood in the tail of a curarized rat was measured by a photoelectric plethysmograph, and changes were rewarded by electrical stimulation of the brain (*23*). All of the four rats rewarded for vasoconstriction showed that response, and, at the same time, their average core temperature, measured rectally, decreased from 98.9° to 97.9°F. All of the four rats rewarded for vasodilatation showed that response and, at the same time, their average core temperature increased from 99.9° to 101°F. The vasomotor change for each individual rat was reliable beyond the $P < .01$ level, and the difference in change in temperature between the groups was reliable beyond the .01 level. The direction of the change in temperature was opposite to that which would be expected from the heat conservation caused by peripheral vasoconstriction or the heat loss caused by peripheral vasodilatation. The changes are in the direction which would be expected if the training had altered the rate of heat production, causing a change in temperature which, in turn, elicited the vasomotor response.

The next experiment was designed to try to determine the limits of the specificity of vasomotor learning. The pinnae of the rat's ears were chosen because the blood vessels in them are believed to be innervated primarily, and perhaps exclusively, by the sympathetic branch of the autonomic nervous system, the branch that Cannon believed always fired nonspecifically as a unit (*3*). But Cannon's experiments involved exposing cats to extremely strong emotion-evoking stimuli, such as barking dogs, and such stimuli will also evoke generalized activity throughout the skeletal musculature. Perhaps his results reflected the way in which sympathetic activity was elicited, rather than demonstrating any inherent inferiority of the sympathetic nervous system.

In order to test this interpretation, DiCara and I (*24*) put photocells on both ears of the curarized rat and connected them to a bridge circuit so that only differences in the vasomotor responses of the two ears were rewarded by brain stimulation. We were somewhat surprised and greatly delighted to find that this experiment actually worked. The results are summarized in Fig. 8. Each of the six rats rewarded for relative vasodilatation of the left ear showed that response, while each of the six rats rewarded for relative vasodilatation of the right ear showed that response. Recordings from the right and left forepaws showed little if any change in vasomotor response.

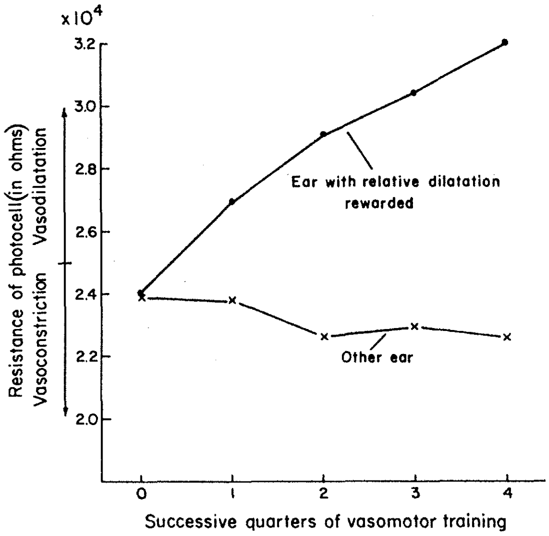
It is clear that these results cannot be by-products of changes in either heart rate or blood pressure, as these would be expected to affect both ears equally. They show either that vasomotor responses mediated by the sympathetic nervous system are capable of much greater specificity than has previously been believed, or that the innervation of the blood vessels in the pinnae of the ears is not restricted almost exclusively to sympathetic-nervous-system components, as has been believed, and involves functionally significant parasympathetic components. In any event, the changes in the blood flow certainly were surprisingly specific. Such changes in blood flow could account for specific psychosomatic symptoms.

Blood Pressure Independent of Heart Rate

Although changes in blood pressure were not induced as by-products of rewarded changes in the rate of urine formation, another experiment on curarized rats showed that, when changes in systolic blood pressure are specifically reinforced, they can be learned (*25*). Blood pressure was recorded by means of a catheter permanently inserted into the aorta, and the reward was avoidance of, or escape from, mild electric shock. All seven rats rewarded for increases in blood pressure showed further increases, while all seven rewarded for decreases showed decreases, each of the changes, which were in opposite directions, being reliable beyond the $P < .01$ level. The increase was from 139 mm-Hg, which happens to be roughly comparable to the normal systolic blood pressure of an adult man, to 170 mm-Hg, which is on the borderline of abnormally high blood pressure in man.

Each experimental animal was "yoked" with a curarized partner, maintained on artificial respiration and having shock electrodes on its tail wired in series with electrodes on the tail of the experimental animal, so that it received exactly the same electric shocks and could do nothing to escape or avoid them. The yoked controls for both the increase-rewarded and the decrease-rewarded groups showed some elevation in blood pressure as an unconditioned effect of the shocks. By the end of training, in contrast to the large difference in the blood pressures of the two groups specifically rewarded for changes in opposite directions, there was no difference in blood pressure between the yoked control partners for these two groups. Furthermore, the increase in blood pressure in these control groups was reliably less ($P < .01$) than that in the group specifically rewarded for increases. Thus, it is clear that the reward for an increase in blood pressure produced an additional increase over and above the effects of the shocks per se, while the reward for a decrease was able to overcome the unconditioned increase elicited by the shocks.

For none of the four groups was there a significant change in heart rate or in temperature during training; there were no significant differences in these measures among the groups. Thus, the learned change was relatively specific to blood pressure.



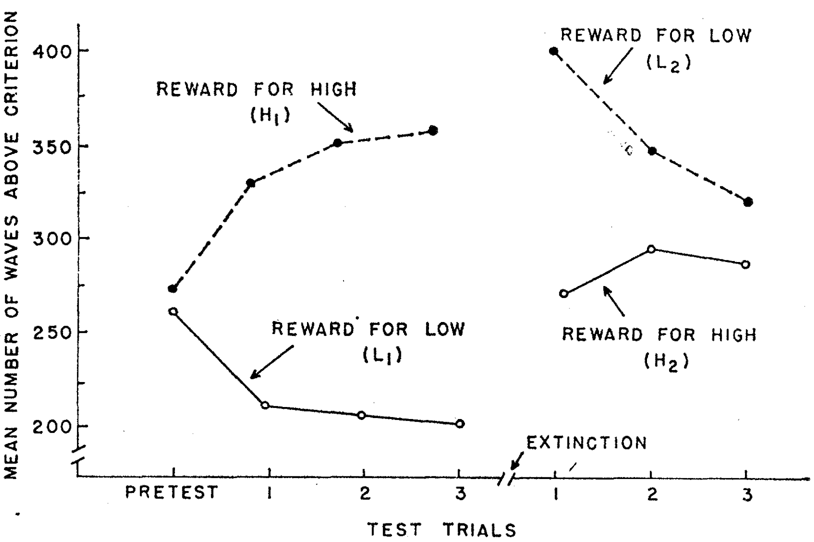


Fig. 8 (left). Learning a difference in the vasomotor responses of the two ears in the curarized rat. [From data in DiCara and Miller (*24*)] Fig. 9 (right). Instrumental learning by curarized rats rewarded for high-voltage or for low-voltage electroencephalograms recorded from the cerebral cortex. After a period of nonrewarded extinction, which produced some drowsiness, as indicated by an increase in voltage, the rats in the two groups were then rewarded for voltage changes opposite in direction to the changes for which they were rewarded earlier. [From Carmona (*29*)]

Transfer from Heart Rate to Skeletal Avoidance

Although visceral learning can be quite specific, especially if only a specific response is rewarded, as was the case in the experiment on the two ears, under some circumstances it can involve a more generalized effect.

In handling the rats that had just recovered from curarization, DiCara noticed that those that had been trained, through the avoidance or escape reward, to increase their heart rate were more likely to squirm, squeal, defecate, and show other responses indicating emotionality than were those that had been trained to reduce their heart rate. Could instrumental learning of heart-rate changes have some generalized effects, perhaps on the level of emotionality, which might affect the behavior in a different avoidance-learning situation? In order to look for such an effect, DiCara and Weiss (*26*) used a modified shuttle avoidance apparatus. In this apparatus, when a danger signal is given, the rat must run from compartment A to compartment B. If he runs fast enough, he avoids the shock; if not, he must run to escape it. The next time the danger signal is given, the rat must run in the opposite direction, from B to A.

Other work had shown that learning in this apparatus is an inverted U-shaped function of the strength of the shocks, with shocks that are too strong eliciting emotional behavior instead of running. DiCara and Weiss trained their rats in this apparatus with a level of shock that is approximately optimum for naive rats of this strain. They found that the rats that had been rewarded for decreasing their heart rate learned well, but that those that had been rewarded for increasing their heart rate learned less well, as if their emotionality had been increased. The difference was statistically reliable ($P < .001$). This experiment clearly demonstrates that training a visceral response can affect the subsequent learning of a skeletal one, but additional work will be required to prove the hypothesis that training to increase heart rate increases emotionality.

Visceral Learning without Curare

Thus far, in all of the experiments except the one on teaching thirsty dogs to salivate, the initial training was given when the animal was under the influence of curare. All of the experiments, except the one on salivation, have produced surprisingly rapid learning—definitive results within 1 or 2 hours. Will learning in the normal, noncurarized state be easier, as we originally thought it should be, or will it be harder, as the experiment on the noncurarized dogs suggests? DiCara and I have started to get additional evidence on this problem. We have obtained clear-cut evidence that rewarding (with the avoidance or escape reward) one group of freely moving rats for reducing heart rate and rewarding another group for increasing heart rate produces a difference between the two groups (*27*). That this difference was not due to the indirect effects of the overt performance of skeletal responses is shown by the fact that it persisted in subsequent tests during which the rats were paralyzed by curare. And, on subsequent retraining without curare, such differences in activity and respiration as were present earlier in training continued to decrease, while the differences in heart rate continued to increase. It seems extremely unlikely that, at the end of training, the highly reliable differences in heart rate ($t = 7.2$; $P < .0001$) can be explained by the highly unreliable differences in activity and respiration ($t =$ .07 and 0.2, respectively).

Although the rats in this experiment showed some learning when they were trained initially in the noncurarized state, this learning was much poorer than that which we have seen in our other experiments on curarized rats. This is exactly the opposite of my original expectation, but seems plausible in the light of hindsight. My hunch is that paralysis by curare improved learning by eliminating sources of distraction and variability. The stimulus situation was kept more constant, and confusing visceral fluctuations induced indirectly by skeletal movements were eliminated.

Learned Changes in Brain Waves

Encouraged by success in the experiments on the instrumental learning of visceral responses, my colleagues and I have attempted to produce other unconventional types of learning. Electrodes placed on the skull or, better yet, touching the surface of the brain record summative effects of electrical activity over a considerable area of the brain. Such electrical effects are called brain waves, and the record of them is called an electroencephalogram. When the animal is aroused, the electroencephalogram consists of fast, low-voltage activity; when the animal is drowsy or sleeping normally, the electroencephalogram consists of considerably slower, higher-voltage activity. Carmona attempted to see whether this type of brain activity, and the state of arousal accompanying it, can be modified by direct reward of changes in the brain activity (*28*, *29*).

The subjects of the first experiment were freely moving cats. In order to have a reward that was under complete control and that did not require the cat to move, Carmona used direct electrical stimulation of the medial forebrain bundle, which is a rewarding area of the brain. Such stimulation produced a slight lowering in the average voltage of the electroencephalogram and an increase in behavioral arousal. In order to provide a control for these and any other unlearned effects, he rewarded one group for changes in the direction of high-voltage activity and another group for changes in the direction of low-voltage activity.

Both groups learned. The cats rewarded for high-voltage activity showed more high-voltage slow waves and tended to sit like sphinxes, staring out into space. The cats rewarded for low-voltage activity showed much more low-voltage fast activity, and appeared to be aroused, pacing restlessly about, sniffing, and looking here and there. It was clear that this type of training had modified both the character of the electrical brain waves and the general level of the behavioral activity. It was not clear, however, whether the level of arousal of the brain was directly modified and hence modified the behavior; whether the animals learned specific items of behavior which, in turn, modified the arousal of the brain as reflected in the electroencephalogram; or whether both types of learning were occurring simultaneously.

In order to rule out the direct sensory consequences of changes in muscular tension, movement, and posture, Carmona performed the next experiment on rats that had been paralyzed by means of curare. The results, given in Fig. 9, show that both rewarded groups showed changes in the rewarded direction; that a subsequent nonrewarded rest increased the number of high-voltage responses in both groups; and that, when the conditions of reward were reversed, the direction of change in voltage was reversed.

At present we are trying to use similar techniques to modify the functions of a specific part of the vagal nucleus, by recording and specifically rewarding changes in the electrical activity there. Preliminary results suggest that this is possible. The next step is to investigate the visceral consequences of such modification. This kind of work may open up possibilities for modifying the activity of specific parts of the brain and the functions that they control. In some cases, directly rewarding brain activity may be a more convenient or more powerful technique than rewarding skeletal or visceral behavior. It also may be a new way to throw light on the functions of specific parts of the brain (*30*).

Human Visceral Learning

Another question is that of whether people are capable of instrumental learning of visceral responses. I believe that in this respect they are as smart as rats. But, as a recent critical review by Katkin and Murray (*31*) points out, this has not yet been completely proved. These authors have comprehensively summarized the recent studies reporting successful use of instrumental training to modify human heart rate, vasomotor responses, and the galvanic skin response. Because of the difficulties in subjecting human subjects to the same rigorous controls, including deep paralysis by means of curare, that can be used with animal subjects, one of the most serious questions about the results of the human studies is whether the changes recorded represent the true instrumental learning of visceral responses or the unconscious learning of those skeletal responses that can produce visceral reactions. However, the able investigators who have courageously challenged the strong traditional belief in the inferiority of the autonomic nervous system with experiments at the more difficult but especially significant human level are developing ingenious controls, including demonstrations of the specificity of the visceral change, so that their cumulative results are becoming increasingly impressive.

Possible Role in Homeostasis

The functional utility of instrumental learning by the cerebrospinal nervous system under the conditions that existed during mammalian evolution is obvious. The skeletal responses mediated by the cerebrospinal nervous system operate on the external environment, so that there is survival value in the ability to learn responses that bring rewards such as food, water, or escape from pain. The fact that the responses mediated by the autonomic nervous system do not have such direct action on the external environment was one of the reasons for believing that they are not subject to instrumental learning. Is the learning ability of the autonomic nervous system something that has no normal function other than that of providing my students with subject matter for publications? Is it a mere accidental by-product of the survival value of cerebrospinal learning, or does the instrumental learning of autonomically mediated responses have some adaptive function, such as helping to maintain that constancy of the internal environment called homeostasis?

In order for instrumental learning to function homeostatically, a deviation away from the optimum level will have to function as a drive to motivate learning, and a change toward the optimum level will have to function as a reward to reinforce the learning of the particular visceral response that produced the corrective change.

When a mammal has less than the optimum amount of water in his body, this deficiency serves as a drive of thirst to motivate learning; the overt consummatory response of drinking functions as a reward to reinforce the learning of the particular skeletal responses that were successful in securing the water that restored the optimum level. But is the consummatory response essential? Can restoration of an optimum level by a glandular response function as a reward?

In order to test for the possible rewarding effects of a glandular response, DiCara, Wolf, and I (*32*) injected albino rats with antidiuretic hormone (ADH) if they chose one arm of a T-maze and with the isotonic saline vehicle if they chose the other, distinctively different, arm. The ADH permitted water to be reabsorbed in the kidney, so that a smaller volume of more concentrated urine was formed. Thus, for normal rats loaded in advance with H_2O, the ADH interfered with the excess-water excretion required for the restoration of homeostasis, while the control injection of isotonic saline allowed the excess water to be excreted. And, indeed, such rats learned to select the side of the maze that assured them an injection of saline so that their glandular response could restore homeostasis.

Conversely, for rats with diabetes insipidus, loaded in advance with hypertonic NaCl, the homeostatic effects of the same two injections were reversed; the ADH, causing the urine to be more concentrated, helped the rats to get rid of the excess NaCl, while the isotonic saline vehicle did not. And, indeed, a group of rats of this kind learned the opposite choice of selecting the ADH side of the maze. As a further control on the effects of the ADH per se, normal rats which had not been given H_2O or NaCl exhibited no learning. This experiment showed that an excess of either H_2O or NaCl functions as a drive and that the return to the normal concentration produced by the appropriate response of a gland, the kidney, functions as a reward.

When we consider the results of this experiment together with those of our experiments showing that glandular and visceral responses can be instrumentally learned, we will expect the animal to learn those glandular and visceral responses mediated by the central nervous system that promptly restore homeostasis after any considerable deviation. Whether or not this theoretically possible learning has any practical significance will depend on whether or not the innate homeostatic mechanisms control the levels closely enough to prevent any deviations large enough to function as a drive from occurring. Even if the innate control should be accurate enough to preclude learning in most cases, there remains the intriguing possibility that, when pathology interferes with innate control, visceral learning is available as a supplementary mechanism.

Implications and Speculations

We have seen how the instrumental learning of visceral responses suggests a new possible homeostatic mechanism worthy of further investigation. Such learning also shows that the autonomic nervous system is not as inferior as has been so widely and firmly believed. It removes one of the strongest arguments for the hypothesis that there are two fundamentally different mechanisms of learning, involving different parts of the nervous system.

Cause of psychosomatic symptoms. Similarly, evidence of the instrumental learning of visceral responses removes the main basis for assuming that the psychosomatic symptoms that involve the autonomic nervous system are fundamentally different from those functional symptoms, such as hysterical ones, that involve the cerebrospinal nervous system. Such evidence allows us to extend to psychosomatic symptoms the type of learning-theory analysis that Dollard and I (*7*, *33*) have applied to other symptoms.

For example, suppose a child is terror-stricken at the thought of going to school in the morning because he is completely unprepared for an important examination. The strong fear elicits a variety of fluctuating autonomic symptoms, such as a queasy stomach at one time and pallor and faintness at another; at this point his mother, who is particularly concerned about cardiovascular symptoms, says, "You are sick and must stay home." The child feels a great relief from fear, and this reward should reinforce the cardiovascular responses producing pallor and faintness. If such experiences are repeated frequently enough, the child, theoretically, should learn to respond with that kind of symptom. Similarly, another child whose mother ignored the vasomotor responses but was particularly concerned by signs of gastric distress would learn the latter type of symptom. I want to exphasize, however, that we need careful clinical research to determine how frequently, if at all, the social conditions sufficient for such theoretically possible learning of visceral symptoms actually occur. Since a given instrumental response can be reinforced by a considerable variety of rewards, and by one reward on one occasion and a different reward on another, the fact that glandular and visceral responses can be instrumentally learned opens up many new theoretical possibilities for the reinforcement of psychosomatic symptoms.

Furthermore, we do not yet know how severe a psychosomatic effect can be produced by learning. While none of the 40 rats rewarded for speeding up their heart rates have died in the course of training under curarization, 7 of the 40 rats rewarded for slowing down their heart rates have died. This statistically reliable difference (chi square $= 5.6$, $P < .02$) is highly suggestive, but it could mean that training to speed up the heart helped the rats resist the stress of curare rather than that the reward for slowing down the heart was strong enough to overcome innate regulatory mechanisms and induce sudden death. In either event the visceral learning had a vital effect. At present, DiCara and I are trying to see whether or not the learning of visceral responses can be carried far enough in the noncurarized animal to produce physical damage. We are also investigating the possibility that there may be a critical period in early infancy during which visceral learning has particularly intense and long-lasting effects.

Individual and cultural differences. It is possible that, in addition to producing psychosomatic symptoms in extreme cases, visceral learning can account for certain more benign individual and cultural differences. Lacey and Lacey (*34*) have shown that a given individual may have a tendency, which is stable over a number of years, to respond to a variety of different stresses with the same profile of autonomic responses, while other individuals may have statistically reliable tendencies to respond with different profiles. It now seems possible that differential conditions of learning may account for at least some of these individual differences in patterns of autonomic response.

Conversely, such learning may account also for certain instances in which the same individual responds to the same stress in different ways. For example, a small boy who receives a severe bump in rough-and-tumble play may learn to inhibit the secretion of tears in this situation since his peer group will punish crying by calling it "sissy." But the same small boy may burst into tears when he gets home to his mother, who will not punish weeping and may even reward tears with sympathy.

Similarly, it seems conceivable that different conditions of reward by a culture different from our own may be responsible for the fact that Homer's adult heroes so often "let the big tears fall." Indeed, a former colleague of mine, Herbert Barry III, has analyzed cross-cultural data and found that the amount of crying reported for children seems to be related to the way in which the society reacts to their tears (*35*).

I have emphasized the possible role of learning in producing the observed individual differences in visceral responses to stress, which in extreme cases may result in one type of psychosomatic symptom in one person and a different type in another. Such learning does not, of course, exclude innate individual differences in the susceptibility of different organs. In fact, given social conditions under which any form of illness will be rewarded, the symptoms of the most susceptible organ will be the most likely ones to be learned. Furthermore, some types of stress may be so strong that the innate reactions to them produce damage without any learning. My colleagues and I are currently investigating the psychological variables involved in such types of stress (*36*).

Therapeutic training. The experimental work on animals has developed a powerful technique for using instrumental learning to modify glandular and visceral responses. The improved training technique consists of moment-to-moment recording of the visceral function and immediate reward, at first, of very small changes in the desired direction and then of progressively larger ones. The success of this technique suggests that it should be able to produce therapeutic changes. If the patient who is highly motivated to get rid of a symptom understands that a signal, such as a tone, indicates a change in the desired direction, that tone could serve as a powerful reward. Instruction to try to turn the tone on as often as possible and praise for success should increase the reward. As patients find that they can secure some control of the symptom, their motivation should be strengthened. Such a procedure should be well worth trying on any symptom, functional or organic, that is under neural control, that can be continuously monitored by modern instrumentation, and for which a given direction of change is clearly indicated medically—for example, cardiac arrhythmias, spastic colitis, asthma, and those cases of high blood pressure that are not essential compensation for kidney damage (*37*). The obvious cases to begin with are those in which drugs are ineffective or contraindicated. In the light of the fact that our animals learned so much better when under the influence of curare and transferred their training so well to the normal, nondrugged state, it should be worth while to try to use hypnotic suggestion to achieve similar results by enhancing the reward effect of the signal indicating a change in the desired direction, by producing relaxation and regular breathing, and by removing interference from skeletal responses and distraction by irrelevant cues.

Engel and Melmon (*38*) have reported encouraging results in the use of instrumental training to treat cardiac arrhythmias of organic origin. Randt, Korein, Carmona, and I have had some success in using the method described above to train epileptic patients in the laboratory to suppress, in one way or another, the abnormal paroxysmal spikes in their electroencephalogram. My colleagues and I are hoping to try learning therapy for other symptoms—for example, the rewarding of high-voltage electroencephalograms as a treatment for insomnia. While it is far too early to promise any cures, it certainly will be worth while to investigate thoroughly the therapeutic possibilities of improved instrumental training techniques.

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Gnathostomulida from America

This is the first record of the new phylum from North America.

R. J. Riedl

Members of the new animal phylum (*1*), Gnathostomulida, recently discovered in Europe, have now been found in unexpected abundance and diversity along the east coast of the United States.

Two million animal species have been described, but the rate at which new descriptions accumulate indicates that these two million are only about 50 percent of the extant species on earth. The increase in new species of birds (8600 known species) has sunk to less than 0.3 percent a year, but in many other classes (for example, Turbellaria with 2500 known species) the rate of increase indicates that undescribed species probably total more than 80 percent. Although only about half of the existing kinds of animals have been described, 80 percent of the families, 95 percent of the orders (*2*), and nearly all of the animal classes are presumably already known. Therefore a new phylum should be rare indeed.

About 24 phyla now divide the metazoa at the upper level of classification. A new phylum might give us a chance to increase our knowledge of principles of structural organization by 1/24. As a matter of fact, the second third of this century brought us many more new animal taxa of the higher categories than the systematics of the first third of the century allowed us to predict. This was based partly on more extensive collecting and more adequate methods and equipment, but also on the notable renaissance of interest in systematics (*3*).

The last phylum discovered prior to Gnathostomulida was Pogonophora. From the history of its discovery, given in Ivanov's voluminous monograph (*4*), we learn that at first there was a period of taxonomic uncertainty lasting one generation. This was followed by a progressive increase in numbers of discovered species, then of new genera, and finally of further new species (Fig. 1). The third stage was the initiation of studies of the embryology, ecology, ultrastructure, and physiology of the Pogonophora, encouraged naturally by the new organization and combination of principal structures characteristic of the new phylum.

The history of the gnathostomulids follows the same sequence of events as that of the Pogonophora. According to reports, the first representative gnathostomulid was seen by Remane in 1928 in the Bay of Kiel; but the first publication was by his collaborator, Ax, and appeared nearly a generation later in 1956 (*5*), with the addition of a second species and genus. The late '50's and the early '60's (Fig. 1) saw the first wave

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