stationary stimulus. But the effect described above has also been obtained under conditions of optical stabilization (*4*) and in any event one would expect the whole visual field to tilt when the eye movement occurred, rather than its effect being limited to producing an apparent rotation of the test spiral. Thus it seems likely that this result reflects a long-term modification of the responsiveness of the nervous system.

This suggestion is supported by the fact that the presence of the effect is limited to the part of visual system which was stimulated by real movement (Fig. 2). Fourteen observers fixated a point 6.1° lateral to the moving spiral during a 15-minute exposure. After a 30-minute delay (*5*) each reported on the presence or absence of apparent motion when the spiral was presented in one of nine positions (in random order) relative to the position of the originally moving stimulus. Each position was tested twice for each observer, with the stationary stimulus exposed for 2 seconds on each trial. The results show that unless the stationary test stimulus falls within about 1.5° of the location where the objectively moving stimulus had been shown there is no aftereffect (*6*). In free observation the specificity of the perceptual change is quite compelling. Looking directly at the stimulus one sees nothing unusual, but when the eyes return to the fixation point the spiral suddenly begins to move; the motion can be started and stopped merely by shifting one's line of regard by a few degrees.

Thus an observer exposed to the rotating spiral for 15 minutes under these experimental conditions leaves the laboratory with a localized change in his vision. His perception is apparently unchanged, but if he looks at a pattern identical to the one he watched rotate, and if the pattern falls on the same part of his retina—and its topographic central projection—his perception is altered and he sees an illusory motion.

These findings show that at least in a simple case vision can be modified by previous visual stimulation. It may be that the phenomenon is best considered as a form of habituation, specific both to stimulus and to place (*7*).

The physiological basis of such a visual storage mechanism is obscure, although Morrell (*8*) and Chow *et al.* (*9*) have shown evidence of plasticity in firing patterns of single units of cat visual cortex and dorsal lateral geniculate body. The topographic specificity of the modification of perception seems to suggest that at least some of the events responsible for the long-term effects of localized stimulation occur in the same population of cells which was stimulated by real motion. An electrophysiological study of motion-sensitive cortical neurons during prolonged stimulation and testing might yield information on this problem.

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5. Figure 1 shows that there is little change in the strength of the aftereffect during the additional time between 30 minutes and 24 hours. This experiment has also been made using 24-hour delay and central fixation, with the same results (*2*).
6. An independent similar experiment on the immediate aftereffect of short exposure to motion has yielded results similar to those reported here [R. Sekuler and A. Pantle, *Vision Res.* **7**, 427 (1967)].
7. Inasmuch as the phenomenon represents form-contingent motion adaptation it may be related to the specific color adaptation effects shown by N. Hepler [*Science* **162**, 376 (1968)] and C. McCollough [*ibid.* **149**, 1115 (1965)].
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Operant Control of Neural Events in Humans

Abstract. *Human subjects were trained by traditional methods of instrumental conditioning to change the amplitude of a late component of the auditory evoked potential with and without oscilloscopic feedback of their performance.*

Fox and Rudell (*1*) trained hungry cats to change the amplitude of a late component of their visual evoked response by reinforcing them with milk whenever the response reached a specified amplitude. We have now trained human subjects in a similar task; the experimental design was somewhat modified to satisfy conditions created by the use of human subjects.

The aforementioned workers and others have aptly described the general aims of the operant control approach and the disadvantages of earlier attempts (*2*) to decode brain waves via the demonstration of neural correlates of behavior. This report, as part of the operant control program, aims to specify brain wave components as potential information carriers as demonstrated by their ability to yield to operant control.

Human subjects were used in the hope of getting at the mechanisms responsible for the operant control of evoked potential components. We expected that, by asking successful subjects how they were able to "control their brain waves," we might obtain suggestive information. Furthermore, we wished to confirm our belief that the operant conditioning of neural events is a general enough phenomenon to be reliably observed in humans as well as in cats. There are advantages in the use of humans; application of scalp electrodes can obviate long hours of surgical placement required in animals less inclined to restraint, and human subjects can be instructed quickly before and interviewed easily after a session.

Our experiments were under the control of a PDP-8 computer (Digital Equipment Corporation). One hundred stimuli (tonal pips) were presented every 4 seconds, and the evoked response was averaged. We selected for each subject a negative-going peak at about 200 msec (*3*) as the criterion component. The computer's next operation was the presentation of a second hundred stimuli, after which it calculated and stored the mean difference between the voltage of the average responses 200 msec before the stimulus (base line) was given and the voltage during the 20 msec selected earlier as the criterion. During training, the computer would reinforce a subject (with money) for increasing the calculated mean difference by 1 standard deviation. Differences rather than pure criterion amplitudes were evaluated to ensure that artifacts of long duration would not be rewarded. In the third phase, stimuli were presented as before, and a running record of reinforcements

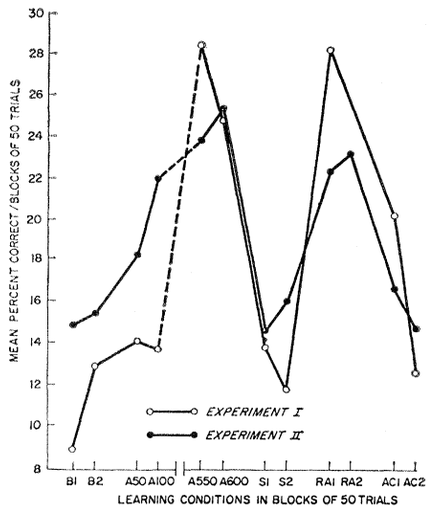

Fig. 1. Percentage of responses reaching criterion as a function of experimental conditions.

was kept. Before the subjects were given instructions, a last set of 100 samples was given so that we could determine the subject's "chance level" of performance. (This last, nonreinforced set is referred to later as the B1 and B2 blocks or *base*.) The subjects were then told to find some "state of mind which would change their brain waves" and that a signal light would inform them of a success, each worth 10 cents. (All subjects were told to be as still as possible.) The subjects in experiment 1 could watch their own performance on an oscilloscope. Subjects in experiment 2 sat in dim light.

Acquisition trials followed. (A100 hereafter refers to acquisition trials 50 through 100). The term *final acquisition* means the last hundred acquisition trials. After A600, the trial block including trials 550 and 600, subjects were told to suppress making correct responses; the signal light would continue flashing, but it now meant the loss of 10 cents. (These trials, 100 in all, are called later S1 and S2 or *suppress*.) Then 100 more acquisition trials were run (later called *reacquisition* or blocks RA1 and RA2). Finally, trials to control for artifact were run (hereafter, AC1 and AC2 or *artifact*). In AC1 and AC2, the tone stimulus was removed altogether (in experiment 1) or attenuated (in experiment 2) to a level whose evoked component in the criterion segment was, according to our pilot data, essentially absent—down by 6 μv. We reasoned that a subject generating a myogenic artifact does not need a stimulus-evoked wave to change but only knowledge of where to generate the artifact. The display provided that knowledge in experiment 1, and the attenuated stimulus did the same in experiment 2. In view of recent data (*4*), our special concern for controlling artifacts seems justified.

In all cases, silver–silver chloride electrodes were fastened with collodion to vertex and mastoid (ground). The raw signal was amplified (80,000 = gain) by a Grass preamplifier set to pass signals between 1.5 and 100 hz. The amplifier signal went to A–D channels of the PDP-8 and also, in experiment 2, to the A–D channels of the Mnemotron CAT 400B. The tone, triggered by the PDP-8, was a 20-msec train of 0.1-msec square pulses. The burst was amplified with an audio preamplifier whose output was delivered to Koss earphones. The PDP-8 computer displayed only single sweeps after B1; the CAT was used to collect average evoked responses during the five training conditions.

The epoch of the PDP-8 was 500 msec, beginning 200 msec before stimulus. The long base line was to insure a sample large enough to be averaged to a straight line. The CAT, also with a 500-msec epoch, began sampling 50 msec before the stimulus so as to continue sampling after the PDP-8 epoch ended, in order to allow further monitoring for artifact. This meant sacrificing the first 150 msec sampled by the PDP-8. The delayed CAT sweep was achieved with Tektronix waveform and pulse generators.

The percentages of correct responses as a function of learning condition are elevated in the appropriate conditions (Fig. 1). The raw B1 and B2 scores added to the S1 and S2 scores were compared (in a within-subject one-tailed t-test) to the sum of the A550, A600, R1, and R2 scores of experiment 2. (For experiment 1, the sum of B1, B2, S1, and S2 scores was compared with the sum of A500, A550, A600, and R1 scores, since no R2 trials were run.) Conditions were pooled to control for any changes occurring over time.

The differences in scores were significant in both experiments. In experiment 1, $t = 2.55$; d.f. $= 4$, and $.025 < P < .05$; in experiment 2, $t = 4.69$, d.f. $= 6$, and $.0005 < P < .005$. In view of the decrease in scores (Fig. 1) on the artifact control blocks, the significant differences obtained testify to the successful control of responding attained by subjects. From the *artifact* traces of Fig. 2, it would seem unlikely that a large artifact occurred systematically which could account for the elevated amplitudes obtained by successful responses. During the criterion segments, these traces do not show any negative-going peaks.

Differences in mean amplitudes of the criterion segments between *base* and *suppress* (pooled) and *acquisition* and *reacquisition* (pooled) approached significance ($t = 1.71$, d.f. $= 4$, and $.05 < P < .10$). Amplitude data were collected only in experiment 2. Absolute amplitudes rather than peak-to-peak differences were tested, inasmuch as the average amplitude of arbitrarily selected 20-msec samples of voltage before stimulus occurrence across learning conditions and across subjects is a flat, straight line, the major difference between neighboring conditions being 0.37 μv.) Records of the averaged evoked potentials of experiment 2 for subjects 2 through 5 and 7 are shown in Fig. 2. There is no uniform pattern of response. Subject 2 seems to generate a new component in the critical segment. Subjects 4 and 5 seem to change the latency of an existing component, whereas subject 7 seems to increase the amplitude of all components in *final acquisition* compared to *base*.

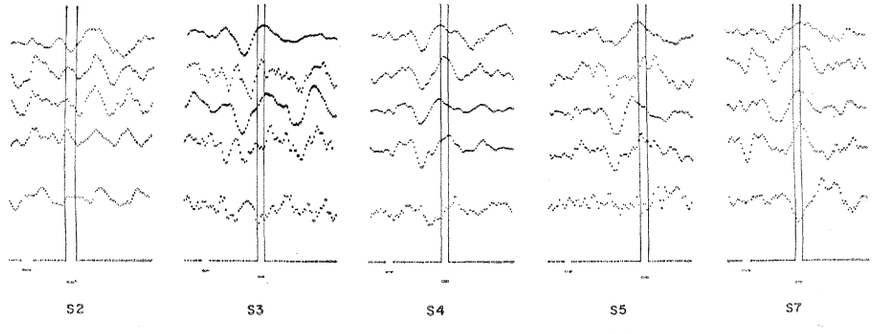

Fig. 2. Average evoked potentials in five experimental conditions. Top trace, B1 and B2; next, A550 and A600; middle, S1 and S2; next, R1 and R2; bottom, AC1 and AC2. First pulse shows stimulus location. Height is arbitrary. Second pulse shows criterion segment; its height represents 12.5 μv.

Subject 3's records show no evidence of reacquisition although performance measured in number of correct responses does show an increase in R1 and R2 over B1, B2, S1, and S2. Subjects 3 and 5 demonstrate increased variability in acquisition and reaquisition.

There was no consistent response to the question, "What did you do to get rewarded?" The responses were of three types: (i) imagined sights such as "I imagined seeing a pin stick me in the head each time I heard the tone"; (ii) imagined sounds such as, "I heard a [second] tone"; and (iii) special attention to various aspects of the stimulus, for example, "I tried not to hear just one tone but an *on* part and an *off* part." Of the 12 subjects in experiments 1 and 2, four responses were of type (i), four were of type (ii), and two were of type (iii), with two subjects reporting that they had to change their strategy from time to time, sometimes imagining a sound, sometimes a sight, with a variety of specific imagery.

The operant control demonstrated here is far from large. These subjects at best did not exceed 30 percent successful responses when chance success was about 16 percent.

It should be noted that the averaged amplitude increases in *acquisition* and *reacquisition* are not apparent although the increases in success scores (Fig. 1) are. Such results agree with the small absolute size of the effect of reinforcement and may be explained if one assumes that, during acquisition trials, a subject may show an increase in criterion responses while missing criterion on unsuccesful trials by a wider margin than during unsuccessful trials of *base* and *suppress*.

Our data do not offer any simple explanation of the operant control phenomenon. Clearly, subjects are not able to quickly perfect the response. Some subjects volunteered the information that immediate feedback on the oscilloscope in experiment 1 was more a hindrance than a help. They said it distracted them from the intense concentration that they needed to do well in the task. If some simple behavior regularly resulted in the rewarded neural event, subjects watching the oscilloscope should have been more quickly able to discover the right technique. There seems, however, to be little difference in the scores of the two experiments. The variety of verbal reports and the various types of changes seen in the average evoked potentials in the criterion segment and elsewhere argue against the idea that subjects can learn a simple motor response whose somasthetic feedback or efferent command generates the rewarded amplitude change. In view of the controls for systematic movements, such an interpretation becomes even less tenable. The use of earphones as the vehicle of stimulus presentation makes it unlikely that learned changes in receptor orientation are the simple explanation of the phenomena. This general kind of interpretation might, however, be successfully revised to account for the phenomenon by basing it upon the notion that subjects can learn to attend (or not to attend) to the stimulus, behavior whose neural correlate could be an enhanced component (*5*). Yet Fox and Rudell reported two successfully conditioned voltage changes of opposite direction. It seems unlikely that control of attention could be mastered with such specificity.

The lack of uniformity in verbal and neural responses makes it difficult to propose a specific mechanism for the operant control reported, even if we have eliminated notions involving a regularly occurring neural correlate (efferent or afferent) of a movement. It is likely that subjects are learning to generate some internal state which may mediate an altered evoked potential by either increasing the overall excitability of many neuron populations, or by increasing the size of a particular population so that when the population is activated, its greater effective size yields an enhanced voltage (*6*). The verbal reports of the subjects suggest that behavior they call "imagining" can bring the relevant state about.

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Pulmonary Gas Transplant Time

The experimental evidence presented by Wagner *et al.* (*1*) does not imply that there is an interaction between the bulk flow and diffusion. On the contrary, the C-shaped curve in Fig. 1 suggests that the mechanism of transport is by convection alone [a more detailed discussion of convective dispersion is given in (*2*)]. The fact that the shortest transit time is about half that obtained from a calculation based upon the average velocity would also result if the velocity profile in the ducts are nearly parabolic; when the Reynolds number of the flow is less than about 2100, the velocity profile is nearly parabolic and the maximum velocity within the duct is nearly twice the average velocity. Furthermore, because Taylor diffusion (*3*) involves only radial diffusion, the minimum transit time does not change; that is, the minimum transit time remains equal to the distance between the larynx and the alveolus, divided by the velocity of the fastest streamline.

This is not to say that Taylor diffusion is not occurring. Ross (*4*) has indicated the gross structure of the dog's bronchial tree. However, to determine whether or not Taylor diffusion is important, the characteristic time associated with convective dispersion must be compared with that for the decay of radial variations in concentrations. Taylor diffusion would be expected to occur in the small diameter bronchi.

However, before the question of transport mechanism can be resolved, the characteristic times must be compared for each branch in the bronchial tree followed by the appropriate dispersion analysis; then the results should be carefully compared with the shape of the residence time distribution curve associated with a step change in stream concentration.

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