the motor cells supplied subcutaneous muscle fibers that were not accessible for penetration with microelectrodes. Since junction potentials could not be recorded from these fibers, contractions were observed under the dissecting microscope while the cell bodies were being stimulated in 20 m*M* Mg^{++} and recordings were being made from the roots.

The muscles that the identified motor cells supply are arranged simply in three layers in the body wall: (i) immediately under the skin a layer of circular muscles runs circumferentially; (ii) deep to this lie two layers of obliquely oriented fibers, spiraling around the animal in opposite directions; and (iii) deeper still lies a thick layer of powerful longitudinal muscle fibers. In addition to these sheets that make up the body wall, there is a group of subcutaneous fibers that raise the skin into ridges (see arrows in Fig. 2C, right) and another group of fibers which traverse the coelom from dorsum to ventrum and flatten the animal. Leech muscle fibers are similar to those of other annelids (*5*) and of crustacea in that they are innervated diffusely rather than at one discrete end-plate region. Thus, the fibers develop tension in a graded manner, depending on the level of depolarization achieved by excitatory junction potentials; muscle action potentials are observed on occasion, but they are not necessary for the development of tension.

All of the excitatory and inhibitory motoneurons that have been identified are shown in diagrams of the ganglion on the left of Fig. 2. Each motoneuron innervates a field confined to one of the muscle layers and always located in the same circumscribed position in the body wall. In Fig. 2 on the right, the positions of the fields of innervation of some of these cells are shown in relation to skin markings. One half of the body wall has been stretched out flat and photographed from the skin side; the long axis of the animal runs horizontally, as do the pigment bands, and the annuli can be seen running vertically (five annuli comprise one segment).

Each of the cells labeled with small letters in Fig. 2A, left, supplies longitudinal fibers running in a different part of the circumference of the segment. In Fig. 2A, right, black lines indicate the circumferential extent of three of these territories. The area that contracts is slightly longer than one segment, but the length of the individual muscle fibers is not yet known. Presumably these cells are involved in bending the animal up, down, or to one side.

In contrast, the large motor cell labeled *L* in Fig. 2A innervates the whole extent of the sheet of longitudinal muscle in the segment (indicated by the black line extending from the dorsal to the ventral midline in Fig. 2A, right). The excitatory junction potentials produced by cell *L* are large and give rise to a powerful shortening of the segment. In addition, this cell is connected to its homolog on the other side of the ganglion by an electrical synapse so that the impulses occur in both cells with a high degree of synchrony. One can speculate that this pair of *L* cells in every segment is used when the whole animal rapidly shortens, for example in response to a noxious stimulus applied to the skin. Since cell *L* and a cell supplying a restricted area of longitudinal muscle can both cause junction potentials in the same fiber, longitudinal fibers can receive innervation from at least two different motoneurons.

In Fig. 2B, left, are shown the cells which innervate circular muscle fibers; on the right, arrows point to the center of the territories of these cells. (The circumferential extent of these territories, as judged by watching contractions through the microscope, is about the length of the arrows; the longitudinal extent is about nine annuli.) The remaining motor cells, seen at left in Fig. 2C, innervate the oblique, flattener, and annulus erector muscles; inhibitory cells supplying the longitudinal and flattener musculature are also shown. The cell labeled *AE* causes the skin to be raised into ridges along the annuli as in Fig. 2C, right. The photograph shows one edge of a territory of an *AE* cell; the three annuli on the right (arrows) are within the territory and have erected in response to stimulation of the *AE* cell body.

The 28 excitatory cells described above appear to constitute a major fraction of the excitatory motoneurons in the ganglion. Together they supply all of the muscles, and an extensive search of the ganglion so far has failed to reveal additional cells that directly initiate muscular contraction when stimulated in the presence of Mg^{++}. At this stage one can infer which individual cells are active when, for example, the animal turns in a certain direction while lengthening and flattening his body. The role of the six inhibitory cells in the control of the muscular movements is not clear, but they may facilitate the elongation of the slowly relaxing longitudinal muscles.

Experiments can now be made to trace the synaptic connections between the sensory and motor cells within a ganglion and between adjacent ganglia. In this way one might begin to analyze the pathways by which the leech performs its limited repertoire of movements.

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References and Notes

1. M. Otsuka, E. A. Kravitz, D. D. Potter, *J. Neurophysiol.* **30**, 725 (1967); E. Kandel and H. Wachtel, in *Physiological and Biochemical Aspects of Nervous Integration*, F. D. Carlson, Ed. (Prentice-Hall, Englewood Cliffs, N.J., 1968), p. 17; D. Kennedy, in *ibid.*, p. 285.
2. J. G. Nicholls and D. A. Baylor, *J. Neurophysiol.* **31**, 740 (1968).
3. J. del Castillo and B. Katz, *J. Physiol.* **124**, 553 (1954); *ibid.*, p. 560; I. A. Boyd and A. R. Martin, *ibid.* **132**, 74 (1956); B. Katz and R. Miledi, *ibid.* **168**, 389 (1963); J. G. Blackman, B. L. Ginsborg, C. Ray, *ibid.* **167**, 402 (1963).
4. A. O. W. Stretton and E. A. Kravitz, *Science* **162**, 132 (1968).
5. T. Hidaka, Y. Ito, H. Kuriyama, N. Tashiro, *J. Exp. Biol.* **50**, 417 (1969).
6. I thank Dr. John G. Nicholls for advice and encouragement in all phases of this work, Dr. D. A. Baylor for helpful discussion, and Dr. D. Purves for assistance with the dye injection techniques. Supported by NIH predoctoral fellowship 6M-34, 676-01 and PHS grant NB-06145.
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Visual Motion Perception: Experimental Modification

Abstract. *If a human observer fixates a moving spiral pattern for 15 minutes, a negative aftereffect of motion is perceived when he inspects a stationary spiral 20 hours later. The illusory motion is seen only when the stationary test stimulus falls upon the portion of the retina which had been stimulated by real motion. Thus previous stimulation can cause a relatively long-term modification of vision.*

In the classic aftereffect of visual motion the observer views a repetitively moving pattern for a short time, the motion of the pattern is then stopped, and an illusion of the pattern moving in the opposite direction is experienced (*1*). Under the usual conditions the aftereffect dissipates within a few seconds, and perhaps for this reason the effect was considered by 19th-century physiologists as a "motion afterimage,"

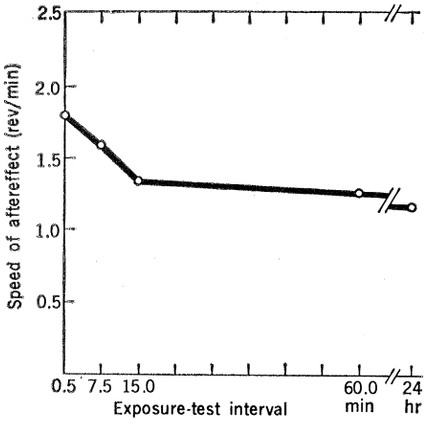

Fig. 1. Speed of aftereffect as a function of exposure-test delay.

similar to the familiar negative color afterimages. However, I have found that it is possible to generate a motion aftereffect, which is seen hours or days after exposure to real movement, when the observer inspects the stimulus which he originally viewed in real motion. This demonstration suggests the existence of a long-term storage capability in vision.

Observers were 45 male university students who had no previous knowledge of the motion aftereffect. They were seated 2.4 m from a black and white spiral pattern (3.5 turn) on a disk 20.3 cm (5.1° visual angle) in diameter. A black knob (0.6 cm in diameter) provided a fixation point at the center of the spiral. The spiral was aligned vertically, with its center 55 cm from the floor, and was surrounded by a white screen 100 cm^2. It was illuminated at 13.9 lu/m^2 by a tungsten photoflood lamp located above and behind the observer. Motion was generated by rotating the disk clockwise at 80 rev/min.

The observer was told only that the experiment was concerned with visual perception and that he would simply be asked to report what he saw. He heard the following instructions: "In a minute I am going to turn on a motor which will make the spiral turn. You see that there is a black knob in the center of the spiral. I would like you to look at the knob, and keep looking there until I tell you to stop. It is extremely important that you keep looking right at the center and not look away at all." Each observer then fixated the center of the spiral for 15 minutes. At the end of this period, alternate observers were assigned to one of two experimental groups.

In group 1 ($N = 15$), the spiral was stopped and the observer was permitted to observe the aftereffect at will for 10 minutes before leaving the room. For group 2 ($N = 15$), the lights were turned out and the observer sat in the dark for 10 minutes, after which the lights were turned on again and he left the room. He was then asked if he noticed anything unusual about his vision, which none did. A third group of 15 observers underwent a control procedure identical to that of group 2, except that the spiral was not rotated—the observer simply fixated the center of a stationary spiral for 15 minutes, then sat in the dark for 10 minutes.

All observers returned for a second session between 20 and 26 hours later. They had been told on the 1st day that there was "another part of this experiment." The stimulus was covered before the observer entered the room. He was seated as in the first session and given the following instructions: "In a minute I will take the card away and you will see the spiral that you looked at yesterday. I want you to look back at the center of the spiral, where you looked before, and tell me what you see."

All observers in both experimental groups reported apparent movement of the spiral on the 2nd day. The typical observer in group 1 would exclaim "It's still moving," and observers in group 2, who had not been allowed to see the aftereffect on the 1st day, reported that the spiral definitely seemed to be moving. In all cases the motion described was in the opposite direction to the previous real motion of the spiral. None of the control observers, who had inspected a stationary spiral in the first session, reported the motion aftereffect on the second day. The basic finding described above has been replicated with an additional 121 observers in other experiments (*2*).

The strength (speed) of the motion aftereffect can be measured by means of a compensation method in which the observer controls, with a rheostat, an objective motion of the spiral in the direction opposite of the aftereffect. The null point, where aftereffect and real motion cancel, is taken as the speed of the aftereffect (*3*). Seventy-two additional observers were given a 15-minute exposure to real movement under conditions similar to those for group 2. The speed of the resulting apparent movement was measured after a period between exposure and testing of 0.6, 7.5, 15, or 60 minutes, or 24 hours (Fig. 1). Each observer served in a single delay condition.

Subjectively the apparent movement is striking and unambiguous. One sees a clear aftereffect like the one following short exposure to real motion in the classic demonstration. Although the instructions merely request description of the stimulus, it is conceivable that some observers may have expected to see motion on the 2nd day. This seems unlikely because the effect was not reported by control observers, and was reported by observers in experimental group 2, who never experienced the aftereffect before the second test session and should have had no way to suggest to themselves the direction of the apparent motion. More importantly, they were clearly taken by surprise to find the spiral moving.

Another explanation would involve eye movements. A tendency for the eye to counteract the original rotation might become an actual (torsional) eye movement during later inspection of the

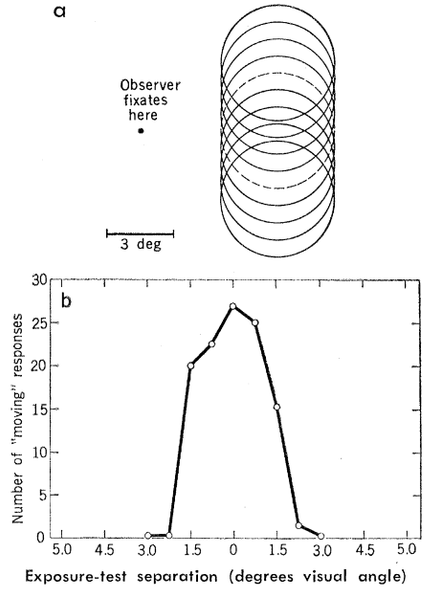

Fig. 2. (a) Test for topographic specificity of the aftereffect. During both real movement and test for aftereffect the observer fixated at the indicated point. During stimulation with real movement the spiral was located in the position indicated by the dashed circle. The stationary test spiral was later presented at one of the positions indicated by the solid circles, or at the position where the moving spiral had been. Since the fixation point was constant this procedure varies the overlap between the retinal area stimulated by real motion and that stimulated by the stationary pattern. (b) Number of reports of movement as a function of the center-to-center separation between the location of the moving spiral and the location of the stationary test spiral.

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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References and Notes

1. For reviews see A. Wohlgemuth, *Brit. J. Psychol.* suppl. 1 (1911); H. C. Holland, *The Spiral Aftereffect* (Pergamon, New York, 1965).
2. R. H. Masland, thesis, McGill University (1968).
3. This procedure could not be used in the first experiment because the instructions to the observer might be construed as suggesting the result. For details of the tracking method see (*2*) and M. M. Taylor [*Percept. Mot. Skills* **16**, 119 (1963)].
4. N. Hepler and R. H. Masland, in preparation.
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)] ; ➡ C. McCollough [*ibid.* **149**, 1115 (1965)].
8. F. Morrell, in *The Neurosciences*, G. C. Quarton, T. Melnechuk, F. O. Schmitt, Eds. (Rockefeller Univ. Press, New York, 1967), p. 452.
9. K. L. Chow, D. F. Lindsley, M. Gollender, *J. Neurophysiol.* **31**, 729 (1968).
10. Supported by National Research Council of Canada grant to Dr. D. Donderi, whose guidance is acknowledged. During this work, R.H.M. held a National Research Council of Canada studentship.
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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