thermore, even after learning had occurred, "set" was a significant factor in the monkey's response. The importance of "set" was most clearly revealed when a sequence of several hundred stimuli of one kind was followed by a stimulus of the opposite kind. For the first "opposite" stimulus, the response latency was often impaired. Thus, although the short latency of the 30- to 40-msec EMG response suggests that it is a reflex, its acquisition through learning and its sensitivity to "set" are features usually attributed to voluntary movement.

In considering the relations between sensorimotor cortex and movement, it will be useful to review Hammond's (*7*) observations on human subjects performing a movement similar to the one studied in the present experiment. In Hammond's experiments, subjects flexed the forearm and were instructed to "resist" or "let go" in response to a sudden pull. The sudden pull stretched the biceps, and a biceps EMG response occurred at a latency of 18 msec, regardless of whether the prior instruction was "resist" or "let go." This 18-msec EMG response was viewed by Hammond as a stretch reflex mediated by muscle spindle afferents, and it seems to correspond to the shortest latency EMG discharge seen in the monkey. A second phase of musclar activity began at about 50 msec in subjects who had been instructed to "resist," but was usually absent in subjects who had been instructed to "let go." This second phase of muscle activity seems to correspond to what was seen in the monkey at latencies of 30 to 40 msec. In Hammond's experiments, the short latency of the 50-msec EMG response suggested that it was a stretch reflex, but Hammond noted that ". . . this must be reconciled with the fact that prior instructions to 'let go' can interfere so rapidly and effectively with the subject's response."

In attempting to decide if neuronal activity in sensorimotor cortex can play a role in mediating the 30- to 40-msec EMG response observed in the monkey (and possibly the 50-msec response in man), it is useful to estimate the minimum delay between cortical activity and muscle discharge. Bernhard *et al.* (*8*) found that stimulation of the leg area in primate motor cortex caused discharge in ventral roots at latencies of 4.7 msec. In the present experiment, the latency from stimulation of the medullary pyramid to EMG response in the forearm muscle was 6 msec for some motor units; the antidromic latency from medullary pyramid to cortex for the large-diameter PTN's is less than 1.0 msec. Thus, the total delay from motor cortex PTN discharge to discharge of forearm muscle in the present experiment could have been as short as 7 msec. It thus appears that under the conditions of the present experiment, PTN activity at 25 msec does not necessarily occur too late to play a role in EMG activity at 35 msec. Several additional milliseconds would be required for postcentral and precentral non-PTN activity to influence motoneurons via polysynaptic pathways, but these neurons discharge well in advance of precentral PTN's and therefore have the potential capacity to influence motoneuron discharge even earlier than is the case for precentral PTN's.

How should one classify short latency muscle responses which are under volitional control? A response occurring in man within less than 100 msec of a stimulus is commonly called "reflex," but perhaps this minimum time should be shortened for the special case in which the input has a strong and direct pathway to the areas of the cerebral cortex that control the output. Phillips (*9*) has proposed that the PTN's of primate motor cortex may function in a transcortical servoloop. The observations reported here are consistent with Phillips' ideas, and point to the need for further examination of cortical "reflexes" and their possible role in motor plasticity such as that described by Hammond.

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Visual Spatial Illusions: Many Explanations

Day (*1*) has proposed an expanded version of the constancy hypothesis as an explanation of visual illusions. This illusion theory, which was first introduced by Thiery (*2*) and recently popularized by Gregory (*3*), maintains that most visual illusions result from misapplication of the constancy mechanisms. This theory proposes that some set of cues in the illusion situation triggers the constancy mechanisms which, under normal circumstances, would lead to compensation for variations in retinal size and shape which arise from changes in the relation between observer and object. These mechanisms, which usually aid veridical perception, lead to distortions in the percept when inappropriately applied in the illusion situation. One of the appeals of such an explanation lies in its proposed ability to handle all visual spatial illusions with one unitary mechanism. The weakness of such a theoretical stance is that any system which proposes a single causal process for the existence of visual illusions ignores a rather large body of data which seem to argue that illusions are multiply caused. For instance, Chiang (*4*) has suggested that diffraction and optical aberrations in the eye contribute to the magnitude of intersecting line illusions. Coren (*5*) was able to verify that such peripheral distortions can account for some 15 percent of the Poggendorff illusion. Ganz (*6*) and von Békésy (*7*) have proposed that lateral inhibition contributes to the formation of many illusions. They offer data which seem clearly to indicate that such inhibitory mechanisms would result in some of the contour displacements manifested by many of the classical illusions. [It is interesting that some of these data were collected by Day himself (*8*).] When possible sources of these inhibitory interactions

are removed or attenuated, the resultant illusion magnitudes are reduced, although some effects still remain (*9*). Some cognitive components that are independent of constancy mechanisms have also been demonstrated. For instance, adaptation level theory, based on comparative judgmental processes, has not only qualitatively predicted some illusions, but is also capable of describing quantitative variations in the magnitude of some illusions as a function of parametric stimulus variations (*10*). The well-established diminution of illusion magnitude with inspection also seems difficult to explain in terms of the constancy hypothesis alone. Day suggests that such practice effects are due to "shifts in the significance of a set of stimuli." This should lead, however, to a decrease in illusion magnitude under some circumstances and an increase under others, depending upon the particular shifts in significance involved. For all of the illusion configurations thus far tested, however, repeated judgments have invariably resulted in a diminution of illusion magnitude (*11*). In addition, these practice effects seem to be responsive to traditional learning variables such as the spacing of exposure trials (*12*) or the availability of information about the nature and extent of the distortion obtained through erroneous eye movements (*13*). This argument should not be taken as denying the involvement of such constancy mechanisms in the formation of some of the visual illusions. Day has clearly summarized a large body of data that provide support for the contention that constancy factors influence some of the obtained visual distortions (*2, 3, 14*). However, it is clear that, despite this body of supportive data, there is an even larger collection that supports the nonparsimonious and esthetically ugly position that the visual geometric illusions are multiply caused and maintained by a number of different peripheral and central factors. Any single causal mechanism is apt to explain only part of the data.

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The statement that "one of the appeals of such an explanation lies in its proposed ability to handle all visual spatial illusions with one unitary mechanism" is quite unjustified since no such proposition was made or implied. In fact a cautionary statement to the contrary was made in the following terms: "I do not claim that this explanation, which I call the general constancy theory, satisfactorily encompasses all known illusions, but merely that it is more comprehensive than alternative explanations" (*1*).

My central aim was to set out a principle couched in terms of the sensory stimuli that initiate and preserve perceptual constancy, not in terms of the neural processes which they invoke. It is reasonable to expect that neural interactions involving lateral inhibitory processes are correlated with spatial illusions, even though attempts to isolate them have been unsuccessful (*2*). It is also reasonable to expect that the same neural processes invoked under slightly different conditions (when the sensory image varies) are correlated with perceptual constancy. Further, I have suggested that it might be more profitable first to isolate the neural mechanisms associated with the constancies since these are likely also to be associated with illusion (*3*). The general principle and neural processes do not represent mutually exclusive approaches, merely different ones.

The proposal originally put forward by Chiang (*4*) that diffraction and optical aberrations of the eye contribute to intersecting line illusions must be contested. Both Restle (*5*) and Cumming (*6*) have offered forceful evidence in refutation of the proposal. Restle has shown that the Poggendorff illusion is large and positive under conditions in which optical aberrations would not be expected to contribute.

The statement that "for all the illusion configurations thus far tested, however, repeated judgments have invariably resulted in a diminution of illusion magnitude" is demonstrably untrue. In my extensive experiments on the so-called "practice effect," I showed that increases in the magnitude of the Müller-Lyer illusion frequently occurred and that often there was no change in illusion after repeated judgments (*7*). Parker and Newbigging have more recently isolated the basis of illusion decrement (*8*). When practice effects do occur it is likely, as Coren and Girgus suggest, that they are responsive to traditional learning variables, as I concluded (*7*). It is entirely reasonable to suppose that we learn to use those stimuli which reduce the magnitude of an illusion. But it is quite wrong to state that diminution always occurs with repeated judgments.

The general criticism made by Coren and Girgus is that spatial illusions are multiply determined. This contention which was in fact emphasized in my article does not, of course, rule out the operation of a general principle. To argue as I have that different classes of illusion derive from different classes of perceptual constancy, each dependent for its preservation on various stimuli, is of course to argue that illusions do have multiple causes. However, multiple determinants do not rule out the operation of a general principle.

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