trum of the polymer was structureless in the region 400 to 1400 cm^{-1}. During the time of formation of the polymer the film is subjected to constant bombardment by atoms, ions, and radicals which chemically alter surfaces of polymers (*8*), and to photochemical irradiation from the plasma itself (*9*). Thus, films deposited at longer times, 2300 seconds, may have structural entities different from films deposited at shorter times, 600 seconds.

Flux and rejection data as functions of time for a membrane formed during a 600-second polymerization on 0.1-μm filter substrate gave the results shown in Table 1: the flux and rejection of NaCl *both* increase up to 87 hours, unlike the behavior of asymmetric cellulose acetate membranes (*10*); urea rejection, however, decreases with time. This flux trend and rejection behavior were likewise observed with films of allylamine produced at various deposition times, with nitrogen instead of argon as the additive gas. While the rejection data were very encouraging for a film produced at 600 seconds, the rejection data improved and the flux decreased at longer polymerization times (Table 1) at the same plasma conditions: 40 watts net RF power, 0.2 torr total pressure, argon additive gas, and constant deposition geometry. The data reported in Table 1 were taken after the flux and rejection were changing very slowly, generally about 20 hours after the experiment was begun. When a Millipore filter with an average pore diameter of 0.025 μm is used, longer polymerization times probably cause the larger micropores to be filled with polymer more completely (see Table 1), thereby producing fewer membrane imperfections with a concomitant improved rejection and reduction in the value of the flux. The rejection and flux properties of these membranes, and perhaps plasma formed membranes in general, can be quite effectively controlled by variations in the electrical and gas dynamic plasma conditions, a departure from the conventional variations in synthesis one would employ by traditional techniques to form or cast thin polymer films—such as polymer concentration, casting formulation, drying time, humidity, atmosphere, and curing temperature.

Table 1 shows the effect on flux and rejection properties of nitrogen instead of argon as an additive gas compared to a film formed from the pure monomer at a total plasma gas pressure of 0.20 torr, 40 watts, and 700 seconds deposition time. In the case of the pure monomer, 0.2 torr was employed, while with additive gases the monomer was at 0.1 torr partial pressure. The highest rejection was obtained from polymerization of the pure monomer. Whether this effect is generally true under all conditions of plasma deposition has yet to be confirmed. The flux for the film deposited for 600 seconds (Table 1) is actually measured to be higher than that for films deposited for 700 seconds, with argon as the additive gas (Table 1). The reason for this unexpected behavior between flux and deposition time has yet to be determined.

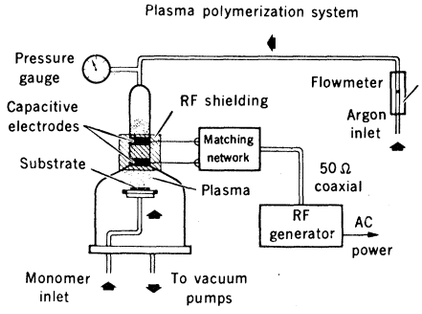

Fig. 1. Plasma polymerization system.

There are several obvious advantages and potential advantages to this synthetic approach for preparing membranes for reverse osmosis and their resultant performance properties: (i) a dry membrane is produced which does not require special handling or storage conditions; (ii) control can be maintained over the membrane thickness on selected substrates by control of plasma parameters; (iii) there is high adhesion of the polymer film on the substrate; (iv) only very thin films, 1.0 μm or less, are required to achieve high salt rejection; (v) a variety of substrates and substrate configurations are amenable to low-pressure plasma polymer deposition; (vi) membrane compaction is likely to be minimal during reverse osmosis since the polymer probably achieves a maximum density during deposition; (vii) uniform membranes with minimal imperfections—that is, pinhole free films—are capable of being formed; (viii) the plasma deposition times are short, 10 to 15 minutes in our laboratory; and (ix) it is conceivable that a large number of organic monomers can now be investigated and polymerized via plasma for preparation of reverse osmosis membranes, since most organic molecules can be readily polymerized in a low-temperature plasma (*1*).

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Motor Cortex Reflexes Associated with Learned Movement

Abstract. *In primates, sensory input can generate reflex motor cortex output in association with learned movement when the sensory input has a strong and direct connection to the motor cortex—for example, when a stimulus calling for repositioning of the hand consists of a perturbation of hand position. This finding supports the proposal that neurons of primate motor cortex may function in a transcortical servo-loop.*

Though commonly referred to as a motor area, the precentral cortex of the primate brain is a target for sensory input as well as a source of motor output (*1*). The strongest sensory input to a particular subdivision of the motor cortex arises from the body part whose movements it controls. For example, the motor cortex hand area receives its strongest input from receptors of the hand. It has been proposed that afferents to the

motor area are involved in cortical reflexes mediating reflex movements [see (*2*)]. But what of nonreflex movements? Do inputs from hand to motor cortex generate outputs in association with learned movements as well as reflexes? The present study sought to investigate this problem by observing activity of motor cortex neurons during performance of learned hand movements triggered by inputs to the hand. For comparative purposes, neuronal activity was also recorded from the postcentral sensory cortex.

Prior to single cell recording, a monkey (*Macaca mulatta*) was trained to grasp a movable handle and position it in a correct zone; the correct zone was signaled by a lamp. After a period which varied unpredictably from 2 to 6 seconds, an external force abruptly moved the handle out of the correct zone, either toward or away from the monkey. The monkey could then gain a reward (fruit juice) by promptly returning the handle to the correct zone. There was no penalty for "overshooting" the correct zone, the only criterion for reward being the speed with which the handle reentered the correct zone. After the monkey had learned to perform the task, recordings were obtained during task performance from about 500 neurons in the hand area of the sensorimotor cortex, 200 located in the postcentral (sensory) cortex and 300 in the precentral (motor) cortex. Of the 300 precentral neurons, 75 responded antidromically to stimulation of the medullary pyramid and were therefore classified as pyramidal tract neurons (PTN's). The experimental techniques have been described (*3*).

Figure 1 shows representative neuronal response patterns and latencies occurring in association with performance of the task described above. The minimum latency from perturbation of handle to discharge of postcentral neurons was 10 msec; this latency corresponds quite well to latencies reported by Mountcastle *et al.* (*4*) and Towe and Amassian (*5*). Minimum latency for precentral non-PTN's was 14 msec. Changes of activity in precentral PTN's began still later; decreased activity began at 20 msec, and increased activity in precentral PTN's had a minimum latency of 24 msec. These differences between precentral and postcentral latencies correspond well to those found by Towe *et al.* (*6*) for precruciate and postcruciate neurons in the cat. The latency of motor cortex discharge for hand movement triggered by input to the hand is in sharp contrast to the long (100 msec) latency for motor cortex activity occurring in association with hand movement triggered by a visual stimulus (*3*) and is consistent with the particularly powerful inputs from hand receptors to motor cortex hand area.

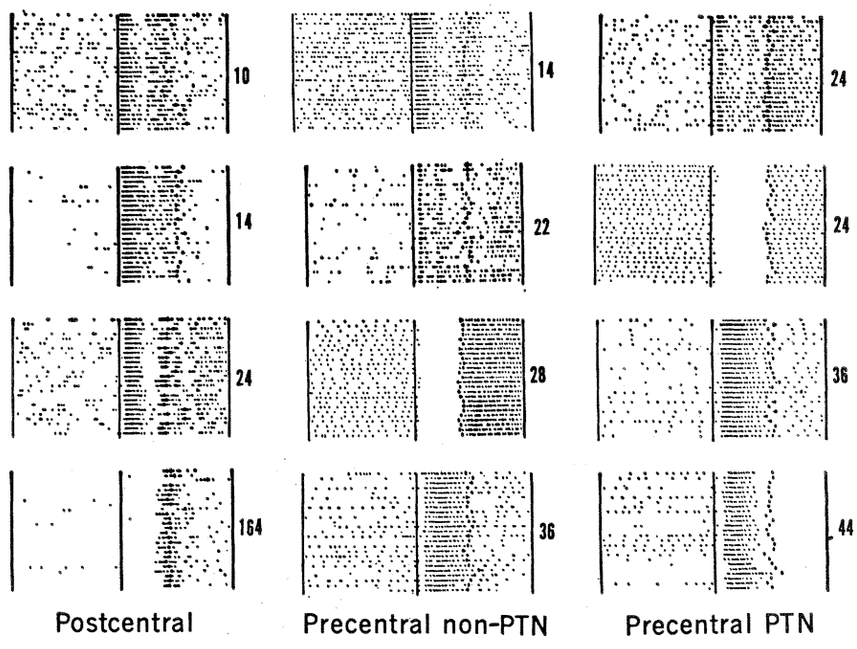

Fig. 1. Each of the 12 rasters shows neuronal activity for 500 msec before and 500 msec after the abrupt handle movement whose time of occurrence is indicated by the vertical line at the center of the raster. In each raster there are 25 rows of dots, corresponding to 25 successive trials. The individual dots in each row correspond to individual neuronal impulses. Neuronal response latency from handle perturbation to first change in neuronal discharge frequency (either increase or decrease) was computed for each neuron on the basis of 25 trials, and this latency (in milliseconds) is shown at the right of each raster. In the right half of each raster a single heavy dot indicates the time at which the handle was returned to the correct zone by the monkey's motor response. This heavy dot may be seen most clearly in the PTN raster at the lower right. Rasters in columns at left, center, and right correspond to postcentral neurons, precentral non-PTN's, and precentral PTN's, respectively. The latency values for one non-PTN (28) and one PTN (24) refer to decreases of activity; remaining latencies refer to increases of neuronal activity.

Electromyographic (EMG) records showed changes of muscle activity at a latency of as little as 12 msec in muscles whose length was changed by the abrupt movement of the handle (for example, when the handle was pulled, biceps was lengthened and had increased discharge, whereas triceps was shortened and had decreased discharge). This 12-msec latency is so short as to indicate that the response is mediated by muscle stretch receptor afferents making monosynaptic connections with motoneurons. A second phase of muscle response began at a latency of 30 to 40 msec, and a third phase began at about 80 msec. Though the first EMG response occurs too quickly to allow cerebral cortex to play a part in its occurrence, the second phase of muscle activity (occurring at a latency of 30 to 40 msec) lags the responses of all three classes of sensorimotor cortex neurons, and a role of the cerebral cortex in this phase of the response may therefore be considered.

The short latency of the 30- to 40-msec muscle response might seem to indicate that it is a reflex and should have been unaffected by learning, but this was not the case. The response of the naive monkey the *first* time the handle was jerked away from him or pushed toward him was *not* to make an opposing movement, but was to release his grip and exhibit signs of fear and surprise. Only gradually did the monkey acquire the short (30 to 40 msec) latency EMG response. Fur-

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