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PARALYSIS OF THE AWAKE HUMAN: VISUAL PERCEPTIONS

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Abstract—Subparalytic doses of curare were given to three observers. Four major perceptions were reported: (1) *displacement* or repositioning of the perceived visual world in the direction of a successfully executed eye movement; (2) *jumping* during a saccade; (3) *movement* associated with drift of the eye; (4) increased *effort* associated with each eye movement. Paralytic doses of succinylcholine were administered to a single observer. Three major perceptions were reported: (1) *displacement* in the direction of the intended eye movement without jumping; (2) a sensation that great *effort* was required to move the eye; (3) *fading* of the visual image due to effective retinal stabilization. Similar visual perceptions were observed when the eye was paralyzed with a local anesthetic; however, no *fading* or sense of *effort* was reported. No deficits in pattern vision (except for intermittent fading) were reported in any of the studies.

INTRODUCTION

When the normal eye is voluntarily moved to a new position, the optical image makes a transition on the retinal surface, yet our perception is of a stable visual and spatial world. Helmholtz (1867) first pointed out that if one pushes the globe with a finger the optical image also makes a transition on the retinal surface; however, unlike the normal perception described above, we perceive a displacement of visual space. This simple experiment led Helmholtz to conclude "our judgments as to the direction of the visual axis are simply the result of the effect of will involved in trying to alter the adjustment of the eyes". That is, our perceptions of space must in part be calculated centrally from a motor outflow to the extraocular muscles rather than from a sensory feedback. Mach (1886), Hering (1879), Jackson and Paton (1909) all later produced evidence which further suggested that our sense of visual space is independent of extraocular sensory feedback. Sperry (1950) and Holst (1954) have called this central monitoring of extraocular motor outflow a "corollary discharge" and "efference copy", respectively. We will not discuss details here, since this literature has been extensively reviewed (MacKay, 1974, 1973; Dichgans and Bizzi, 1972; Evarts, 1971; and Merton, 1964).

If a corollary discharge is in fact used centrally to calculate visual space, one would expect that extraocular paralysis would produce an illusory spatial displacement during voluntary eye movements. Several sources support this prediction. Clinically, for example, it is well known (Cogan, 1956; Jackson and Paton, 1909; Helmholtz, 1867) that pathological weakening or total paralysis of the extraocular muscles produces past pointing (spatial mislocalization). This past pointing is always in the direction of the attempted eye movement. Experimentally, Kornmueller (1931) reported that weakening or paralysis of the extraocular muscles using a local anesthetic produced past pointing and illusions of spatial displacement. He stated that when "glance intentions" were attempted, a striking apparent displacement in the direction of the intended eye movement was perceived. Brindley and Merton (1960), Siebeck (1953, 1954), and West (1932) also described a similar apparent displacement in subjects given low doses of curare.

Quite surprisingly, however, Siebeck (1953, 1954; Siebeck and Frey, 1953) found that when subjects were *totally paralyzed* with high doses of a neuromuscular blocking agent *no illusion of spatial displacement was ever reported.* The subjects reported only that they had the subjective sensation that their eyes were paralyzed. It should be emphasized that these results are in direct contradiction to the motor outflow theory.

The present series of experiments were designed to help resolve this contradiction. It is clear that to study the perceptions associated with extraocular paralysis it would be necessary to use all three forms of experimental paralysis discussed above. Accordingly, we produced: (1) partial paralysis with systemic low dose injections of curare to mimic the experiments of Brindley and Merton, West and Siebeck; (2) total paralysis using systemic injections of a neuromuscular blocking agent to replicate Siebeck's total paralysis study; and finally (3) retrobulbar blocks with local anesthetic similar to Kornmueller's procedure.

METHODS

Partial paralysis

Three of us served as observers in the low dose studies (RCE, ACR, JKS) and curare (6–24 mg) was used as the paralyzing agent. Observations were always made during the induction period of the total paralysis studies and six experiments were carried out specifically for study of low dose effects. The same procedure and precautions described below in the total paralysis section were observed. Audio tapes were made of experiments, and transcripts were later typed.

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We were all fully aware of risks and were familiar with the procedure. In experiments using less than 15 mg of curare no respiratory embarrassment occurred. Those which used more than 15 mg did require some respiratory assistance. Frequently, the observers were able to walk about the room describing some of the reported effects and we all agreed that the low doses of curare were not at all unpleasant.

Total paralysis

One of us (JKS) served as the observer for all of the total paralysis experiments. At the time of the experiments, JKS was a healthy, 155-lb, 24-yr-old male. A complete physical, ocular and vestibular examination revealed no abnormalities.

During the experiments, JKS was supine on an operating table with the back inclined to 30° (Fig. 1). A 7 × 10 ft screen was viewed through a first surface mirror located above the *O*'s head. Simple patterns were projected on the screen during the experiments. The *O*'s left eye was always occluded. Audio tapes were made and transcripts were later typed.

Eye movements were monitored by projecting a beam of light onto the screen via a mirror mounted on a tight-fitting scleral contact lens. A video tape record of the projected dot of light was made during critical portions of the experiment. This system was sensitive to movements of less than 15′ of an arc. Often movements due to artificial respiration and heart beat were visible making it rather difficult to precisely measure the magnitude of an eye movement. However, we could always determine whether and when a voluntary movement occurred.

A total of five total paralysis experiments were conducted. Only the final three completely suppressed voluntary eye movements. The first two studies used curare given in 6-mg increments until JKS wished to discontinue the experiment or the study was complete. Usually JKS was able to signal that he wished to continue by moving his finger or hand. After two attempts, we found this method to be unsatisfactory for the following reasons.

Curare has the disadvantage of being slow acting and the depth of paralysis is difficult to control for short periods of time. This made it difficult for both JKS and experimenters to judge how much more curare was necessary to produce total paralysis. As a result, total paralysis was never achieved using curare. A depolarizing blocking agent such as succinylcholine has the advantage that it is extremely fast-acting and the depth of paralysis is easily controlled. Its one disadvantage is that it produces muscle fasciculations during induction which can be rather painful. These, however, may be eliminated by a small predose of curare. Therefore, the final three experiments used succinylcholine as the blocking agent, preceded by a 6-mg dose of curare. All of these latter studies did achieve total paralysis.

Before the succinylcholine was administered an arterial tourniquet was placed on the right arm, thus preventing local blood flow and paralysis. This procedure made it possible for JKS to communicate by flexing his hand even during total paralysis. A well-practiced protocol was followed which allowed JKS to skip various experiments, to add procedures, or to stop the experiments.

All experiments were carried out in a fully-equipped hospital experimental room, and normal medical precautions associated with any anesthetic procedure were observed. Prior to the studies, the observer was briefed on the subjective sensations of the paralysis and artificial ventilation. The possibility that endotracheal intubation might be required was discussed. JKS did not eat or drink after midnight of the day preceding the experiment. Blood pressure, pulse, respiration, EKG, and expired CO_2 were usually all monitored. An anesthesiologist was present at all times during the experiments and carried out all anesthetic procedures. When necessary, positive pressure ventilation was administered with oxygen from a standard anesthesia machine using a circle absorber breathing circuit and face mask. The airway was supported manually, and ample suction was available.

All medication was injected through or added to a saline intravenous drip. Two to three mg of atropine were given 15 min prior to all total paralysis experiments. Prostigmine was used as the reversing agent in experiments employing more than 6 mg curare.

JKS felt the succinylcholine experiments were very unpleasant, but bearable. No particular discomfort was associated with artificial respiration as long as somewhat deeper, faster respiratory movements than normal were used and PCO_2 was maintained at 5%. JKS never reported any abnormal state, discomfort, or confusion which might have interfered with his visual perceptions. The most critical factors to the success of an experiment were that it be carried out as quickly as possible, and JKS's knowledge that he could communicate with the tourniqueted arm.

Retrobulbar block

These experiments were designed to mimic the neuromuscular block studies in every respect. The same author (JKS) was used and the same stimuli were observed. This study consisted of three separate procedures. Two were done on successive eyes on a single day. The last retrobulbar block immediately followed recovery from the last total paralysis experiment.

Normally, in a retrobulbar block, local anesthetic is injected into the extraocular muscle cone, producing a nerve block of the fifth, sixth and third nerves and a muscle block of the superior, inferior, medial and lateral recti. The superior oblique is not affected by the procedure since it and its innervation lie outside the muscle cone. The block renders the globe immobile (except for movements in the superior oblique plane), completely anesthetic, and relaxes the ciliary muscles.

In our study 2% procaine was used to anesthetize locally the lower margin of the skin around the orbit. A blunt 23 gauge, retrobulbar needle was inserted through the skin, behind the eye into the muscle cone and 2–5 cm^3 of 2% procaine was infused. A detailed description of the method is given in Atkinson (1965). The block was administered by an ophthalmologist. JKS reported only minor discomfort.

Edema of the orbital area made it difficult to record eye movements using the scleral contact lens system described above. Therefore, the experimenters monitored movements visually throughout this phase of the work.

RESULTS

Partial paralysis

After doses as low as 6 mg of curare, spontaneous microsaccades were almost immediately abolished, eye movements were slowed, and diplopia was reported. As dose levels increased, vertical, lateral, and medial eye movements were suppressed in that order. Total extraocular paralysis was not achieved in the low dose experiments even with 24 mg of curare (which was enough to require respiratory assistance).

The perceptions associated with a 6-mg injection were very striking and often confusing. Initially all of the observers had difficulty describing what they saw and frequently contradicted themselves. However, after a period of about 15 min of observation they came to the same general conclusions.

When an attempt was made to make a fast saccade upward for example, the visual world would disappear or "jerk" and reappear above its original spatial

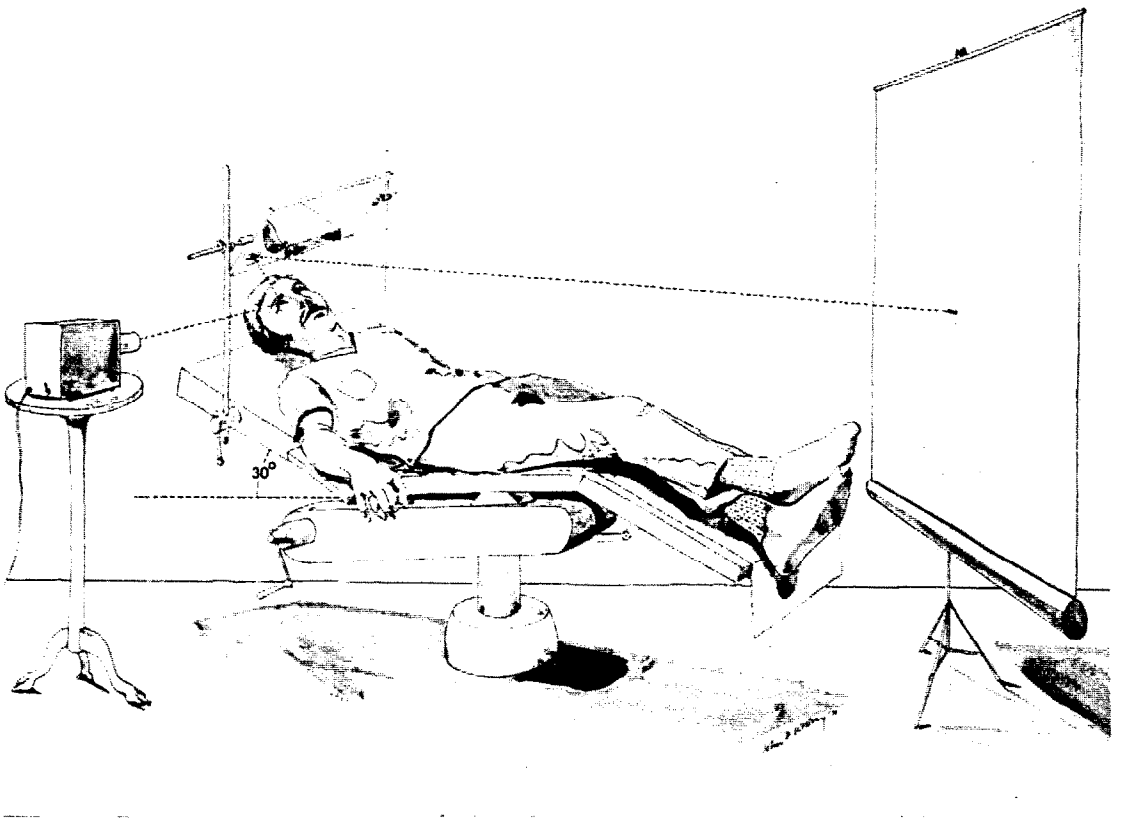

Fig. 1 Artist conception of total paralysis, experimental set-up. Observer was supine on operating table. He viewed screen through a first surface mirror. The projector illuminated a small mirror mounted on a stalk attached to a tight fitting scleral, contact lens. This produced a dot on the screen which followed the observers eye movements. Not shown are the projectors used to project stimulus patterns, words, etc. and the video tape system for recording the eye movements.

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locus. This was described as a sensation of displacement rather than actual movement. "The world did not move... it was not as if you had taken the stimulus and moved it across the screen... when I moved my eyes up, the whole screen was displaced up... (the stimulus) disappeared and then popped up again in another place."

The *displacement* was preceded either by a very rapid jump or a blanking out of the visual input during the saccades. ACR and RCE felt that it was a jerk or jump and JKS felt that it was sometimes a jerk and sometimes a blanking out. This perception of blanking out or rapid jerk will be called *jumping* in later discussions.

A third perception frequently reported was a *slow movement* of the stimulus. This was accompanied by a slow drift of the eye, and almost always occurred when the fixation point was away from the midline. Often the *jumping* and *displacement* perceptions discussed above and the *slow movement* perception were interleaved. The observer would make a saccade away from the midline, and his eye would slowly drift back towards the midline, creating a very confusing perceptual picture. He would first see *jumping*, then *displacement*, followed by a slow drifting *motion*. Only after repeated attempts were the observers able to separate the three perceptions.

Two additional perceptions were found when dose levels of 15 mg of curare or more were used. As the level of paralysis increased, observer JKS[2] mentioned that he had great difficulty in making voluntary saccades. The sensation was not one of heaviness or weighing, but to "move my eyes took quite a bit of effort...". He later mentioned that the sensation was much like trying to look past the most peripheral limit of your eyes. During these periods requiring great effort JKS reported a visual *fading* similar to that seen in a fixed retinal image. Presumably paralysis had produced just such stabilization.

When all three of the observers attempted to ballistically touch an object, they always missed by several inches in the direction they had turned their eyes. For example, if the observer fixated on an object to the right of his center (head fixed straight ahead), he would past point by several inches to the right. Also, when all three of the observers closed their eyes and attempted to touch their nose, they very systematically overshot by at least a half an inch. In a later experiment, we injected curare distal to an arterial tourniquet and found the same nose mislocalization effect immediately after the injection. These data suggest that the past pointing discussed above had both a somatosensory and a visual component. A similar effect has been noted in other curare experiments (Peter Cohen, personal communication), and we are presently carrying out more detailed experiments.

Total paralysis

All total paralysis experiments used succinylcholine for the paralysing agent. The eyes were completely immobile during the observations periods at least to the resolution of the scleral mirror system.

In the first experiment JKS reported no *movement* or *displacement* during attempted saccades. "I tried to move my eyes as hard as I possibly could and nothing happened, the world was just there... I simply could not move my eyes". The earlier curare experiments described above made it clear that effort was a critical factor. Therefore, the study was repeated, the JKS was reminded at frequent intervals to exert great effort. JKS moved a finger on the tourniquet-protected arm to indicate an attempted eye movement. Careful study of the video tape showed no actual eye movements during these periods. Again JKS reported that he was very much aware that his eyes were paralysed. "I know I did not move my eyes, I was trying very hard." However, unlike the first total paralysis experiment, "when I looked to the right I felt that if I had to touch anything... I would have had to reach over to the right." JKS felt that his perceptions were much the same as seen during the low dose experiments, but this *displacement* was not punctuated by *jumping*. That is, no jerk, jump, or blanking out of the visual input was perceived during the attempted saccade during total paralysis. The jumping had been very striking in the low dose experiments and had made the displacement illusion quite apparent. JKS emphasized that the displacement perception was not necessarily visual in nature, and found it very difficult to describe.

Later, a third total paralysis experiment was carried out, and again JKS reported the same perceptions described in the second total paralysis experiment. When he attempted to move his eyes to the right, they felt paralysed, yet the visual world was spatially relocated to the right. As before, he emphasized the perception was not visual.

In agreement with the low dose experiments, image fading became a real problem when the head was not moved. However, due to inadvertant movements associated with artificial respiration, the image never faded for long periods of time.

Tests were carried out for visual field deficits and decrements in visual acuity. None were detected at any time during the total paralysis studies. JKS could read yes/no questions and answer them correctly with the tourniqueted arm; he could recognize familiar patterns and faces, etc. Other than the fading described above, JKS never reported any subjective loss of pattern vision or confusion of thought.

Retrobulbar block

The first study consisted of two successive blocks, first the left eye and then following its recovery an hour later, the right eye. The second experiment used only the left eye and was carried out on the same day as the last paralysis experiment. All retrobulbar block studies were designed to mimic the neuromuscular block experiments.

Shortly after the injection, and prior to total extraocular paralysis, JKS reported that with each attempted saccade the visual world transiently jumped (*jumping*) followed by a spatial relocation in the direction of the eye movement (*displacement*). *Movement* opposite to the direction of slow eye drifts was also occasionally reported. These perceptions were similar to those seen in the low dose curare experiments and

[2] RCE and ACR never received more than 15 mg of curare over a short period of time.

Table 1. Summary of experiments and results

Experiment	Blocking Agent	Total Dose(mg)	Perceptions: Movement	Displacement	Jumping	Past Pointing	Effort	Fading
Low Dose	Curare	6–15	X	X	X	X		
Low Dose	Curare	15–24	X	X	X	X	X	X
Total Paralysis	Succinylcholine	60–160		X			X	X
Partial Retrobulbar Block	Procaine	40–100	X	X	X	X		
Total Retrobulbar Block	Procaine	40–100		X		X		

were present only during the prodomal stages of the procedure.

When total extraocular paralysis was achieved, JKS reported the same perception of *displacement* without noticeable *jumping*, as seen in the succinylcholine experiments. Again he emphasized the non-visual quality of this spatial relocation, but added that it was most dramatic when he viewed an active movement of his arm or leg. When patterns were viewed so that body parts could not be used as references, rather large attempted eye excursions were necessary before the displacement became perceptible. However, if JKS looked at his moving hand even the slightest attempted eye movement produced a perceived displacement. Past pointing during the total block was very strong. During one study JKS attempted to touch an object in the periphery and overshot by 20 in.

Unlike the total neuromuscular paralysis experiments, *fading* was never a serious problem during the total retrobulbar block. Also, during the retrobulbar block, JKS was unable to state whether his eye had successfully moved or not, even though some movement was present. After his eye was totally immobile he frequently substituted head movements for eye movements. He was totally unaware of this substitution and again was unable to perceive the actual state of his extraocular paralysis. During these studies he never felt that great *effort* was required to attempt an eye movement. This stands in strong contrast to the total neuromuscular paralysis experiments, where JKS was acutely aware of his extraocular paralysis and great *effort* was required to attempt an eye movement.

These data and the data from the total and partial paralysis are summarized in Table 1.

DISCUSSION

Unfortunately, human experiments designed to directly answer the questions we have addressed ourselves to are inherently limited. Most studies dealing with the perception of space provide the experimenter with at least one independent variable which he can control and a second variable which can be accurately measured (e.g. eye position). In the most interesting portion of our studies we had only JKS's response to yes or no questions as data. We found that the correspondence between physical objective straight ahead and subjective straight ahead was dependent upon JKS's attempts to move his eyes. Therefore, objective indication of eye position was meaningless and asking JKS to indicate if any object was left or right or straight ahead would have proven fruitless. Finally, any detailed psychophysical task would have required hours of total paralysis—a very unrealistic requirement.

With these limitations in mind, we designed the study to be as direct and simple as reasonably possible, with repeated well-rehearsed, short experiments, using for the most part a single trained observer, making subjective observations.

Fading

When the eyes were sufficiently paralyzed (15 mg curare or more), JKS reported *fading* of the visual input. *Fading* was not a serious problem in the retrobulbar block studies, but this was probably due to frequent head and body movements, which were not possible in the curare studies using more than 15 mg of curare. This fragmentation or *fading* has been extensively studied using various stabilization methods (see Jones, Webster and Keesey, 1972) all of which make the reasonable assumption that the sole purpose of small eye movements is to physically shift the visual image. In previous experiments dealing with stabilized images the microsaccades were actually intact, but the visual image was compensated to produce the stabilized retinal image. In our experiments, we simply abolished the eye movements and produced what appeared to be the same result: *fading*. We can conclude, therefore, that small eye movements and small stimulus movements are equivalent for pattern vision.

Jumping

This perception was described variously by the observers as a jerk, jump, rapid movement, or blanking out. All later agreed that *jumping* was an adequate

descriptive term. *Jumping* was always reported immediately following a successfully executed saccade in the low dose curare studies and during the incomplete stages of the retrobulbar blocks. It was never seen during the total paralysis experiments or during the complete retrobulbar block.

It is well known that during a saccade eye movement, visual input is perceptually suppressed (see Volkman, Shick and Riggs, 1968). A number of workers have demonstrated that in the normal human, this suppression begins *prior* to the perceptual onset of the actual saccade (Duffy and Lombrose, 1968; Volkman *et al.*, 1968). This finding lead to the conclusion that saccadic suppression is induced by a direct central component (i.e. a corollary discharge). More recent work, however (Brooks and Holden, 1973; Matin, Clymer and Matin, 1972; Mitrani, Mateeff and Yakimoff, 1971; MacKay, 1970), has demonstrated that saccade suppression can be explained by backward masking (metacontrast), triggered by a rapid image movement over the entire retinal mosaic, rather than by a corollary discharge. MacKay (1973) has reviewed this literature.

In our experiments, we routinely observed that as curare doses increased, the saccadic eye movement velocity slowed down. If one accepts that the rapid movement of the retinal image serves as the trigger for saccadic suppression, it seems reasonable that by slowing down the eye movement we have reduced or eliminated the normal suppressive mechanism, leading to our *jumping* perception. The very fact that such a transformation can take place suggests that image movement rather than a corollary discharge serves as the trigger for saccadic suppression, but the strongest support comes from the total paralysis and total retrobulbar block experiments. During both, neither *jumping*, smearing, nor suppression were ever seen in spite of the fact the observer made repeated unsuccessful eye movements. We, therefore, conclude that if there are any direct central suppressive components, they must be very weak, and that a rapid image movement over the entire visual mosaic must serve as the trigger for "perceivable" saccadic suppression.

Effort

In the total paralysis studies JKS was aware that his eyes were paralyzed and frequently mentioned that any attempted eye movement required great *effort*. In the retrobulbar block studies JKS had no sense of where his eye was located physically in its socket, and *effort* as perceived in the total paralysis studies was never a problem. We, therefore, equate the perception of *effort* with an intact eye position sense.

One obvious reason for the difference between the two studies might be that the retrobulbar blocks were always unilateral, and all the normal perceptions of eye rotation were intact in the other eye while the neuromuscular block studies always affected the two eyes equally. Subjectively, however, JKS did not feel this fully explained the differences.

A second explanation for the differences comes from Brindley and Merton (1960) and Skavenski (1972). Brindley and Merton have demonstrated that a subject is subjectively unable to assess the position of his eye after the conjunctival sac is anesthetized. Thus, we receive "subjective" eye position information from the lids pressing against the eye. Recently, however, Skavenski has used an objective psychophysical task to demonstrate that subjects are able to detect at least a change in eye position after the conjunctival sac is anesthetized. Thus, we must receive additional eye position information from either extraocular stretch receptors or retrobulbar mechanoreceptors.

These conclusions are both entirely consistent with our data. During the retrobulbar block experiment both the conjunctival sac and the retrobulbar sensory receptors were anesthetized, and JKS had no subjective eye position sense. During the total paralysis experiments all ocular and extraocular sensory receptors were intact, and JKS was acutely aware that his eye was paralyzed.

Our results also emphasize that *felt position of the eye is not the same as the perceived position of objects in space*. It is possible to sense that the eye is fixed straight ahead in its socket, yet the visual world is perceived as being displaced to the right. This agrees with Siebeck and the reports of patients with pathologically paralyzed extraocular muscles (Cogan, 1956) and we feel is a rather important distinction not usually made in the behavioral literature.

Displacement and movement

The perception of *displacement* with attempted saccades was described in all of the studies. It should be emphasized that this is not the same perception as *movement*. *Displacement* was seen immediately after a saccade attempt in the total paralysis and retrobulbar block studies and after a successful saccade in the low dose experiments. *Movement* was reported when the eyes were seen drifting after some attempt was made to fixate on a peripheral point or when the head was rotated. A sufficient condition for perception of *movement* seems to be the successive stimulation of a limited area of retinal receptors. The perception of *displacement* requires that some attempt be made to contract the extraocular muscles without a corresponding physical shift of the visual fields. Therefore, *displacement* must be the perception associated with a corollary discharge to the extraocular muscles.

Siebeck, in his total paralysis experiments, did not make the distinction between movement and displacement and was most likely expecting to see a movement perception. As mentioned in the results section pure displacement is not normally experienced and thus very difficult to describe. It is not necessarily visual in nature, but simply the feeling that if you wanted to touch a given object you would have to reach to the right, left, up or down, etc. It was apparent in the retrobulbar and low dose studies that the threshold for detection of this perception was relatively high. All of the observers were quite surprised when they attempted to touch a point and missed it by several inches. Nevertheless, JKS, who was used in our total paralysis studies, had considerable practice in testing his ability to detect displacement in both the low dose and retrobulbar experiments, where hand and body movements were possible. The last total paralysis and retrobulbar block studies were carried out in the same day for a semi-direct comparison

and JKS was convinced that the *displacement* seen in each was identical.

CONCLUSION

Our data and others referenced above lead us to the conclusion that at least three independent systems are operating to produce the three major classes of perceptions reported here.

First, an eye position system must be responsible for our perceptions associated with eye position (*effort*). This system receives its input from sensory receptors located in the conjunctival sac and from retrobulbar sensory receptors possibly located in the extraocular muscles.

Secondly, a pattern visual system must be responsible for our perceptions associated with pattern vision (*fading*, *jumping*, and *movement*). This system receives its input solely from the retinal mosaic and differentially responds to moving stimuli in one of three ways: (1) when the entire retinal mosaic is activated by a small rapid image movement (e.g. via a microsaccade), we "see" a pattern; (2) when the entire retinal mosaic is activated by a large rapid image movement (e.g. via a saccade) pattern vision is inhibited or suppressed; and finally (3) when a limited area of the retinal mosaic is activated by an image movement we see *movement*. Using these three criteria, the pattern system is capable of distinguishing between self-induced retinal image *movements* and *movements* of objects in the external world. Thus, the pattern system with information solely from the retinal mosaic can produce a perceptually stable *visual* world.

Finally, a spatial system must be responsible for our perceptions associated with spatial localization (*displacement*, *past pointing*). This system receives its input from the retinal mosaic and the motor signals on their way to the extraocular muscles (i.e. the corollary discharge). Using these inputs the spatial system is capable of distinguishing between self-induced spatial *displacements* and *displacements* of objects in the external world. Thus, we conclude that the spatial system, with the corollary discharge and information from the retinal mosaic can produce a perceptually stable *spatial* world.

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