optimism that these spatial frequency filters behave almost linearly with contrast over the range studied. Therefore, simulations of a hierarchy of these filters may provide a good first approximation to many of the visual cues required for the perception of complex objects. Furthermore, linear modeling may be as useful for predictive modeling of suprathreshold phenomena as it has been at threshold.

ARTHUR P. GINSBURG
MARK W. CANNON
Aviation Vision Laboratory,
Air Force Aerospace Medical Research
Laboratory, Wright-Patterson
Air Force Base, Ohio 45433
MICHAEL A. NELSON
Systems Research Laboratory,
Dayton, Ohio 45440

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10. These results are not in agreement with the data of Hamerly, Quick, and Reichert (*4*) for the perceived contrast of two superimposed sine-wave gratings of frequencies f and $3f$. They reported a linear addition of perceived contrast when the two stimuli were presented simultaneously at contrasts above 0.05. Our square-wave results indicate that its higher harmonics do not add linearly to the fundamental. Further experiments are needed to determine the cause of this difference. D. Tolhurst [*J. Physiol. (London)* **226**, 231 (1972)] found adaptation effects with suprathreshold square-wave gratings that he attributed to inhibition between the fundamental and third harmonic frequencies. Our results suggest little or no such interaction under our experimental conditions.
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18 September 1979; revised 1 February 1980

Saccadic Eye Movements and Body Sway

Abstract. *Different conditions of moving retinal images show differential influences on postural stability. A surrounding pattern moved during steady fixation increases body sway, but similar image motions generated by voluntary saccades do not. Mechanisms for postural control do not respond to visual feedback during saccades. Analogous principles in saccadic suppression and space constancy are discussed.*

Maintaining a steady upright posture depends on a fine balance between opposing muscular actions. This balance is controlled reflexively in accord with sensory feedback from proprioceptive joint and muscle afferents, vestibular organs, and the visual system. When an imbalance of forces results in sway of the body, the head and eyes move with respect to the surroundings so that optical images move across the retinas (*1*). Feedback for the multiloop control of posture from this movement of the retinal image is useful for reducing slow body sways to which the other systems are not sensitive (*2*). One's steadiness while standing on one foot illustrates the stabilizing effect of visual feedback. Steadiness improves when surrounding objects are visible and is diminished by closing the eyes.

Although vision normally stabilizes posture, in restricted circumstances it provides "false" feedback. Motion of the retinal image analogous to that induced by sway can be imposed experimentally, as by coherent movement of the whole visual surroundings, and the false signal can evoke inappropriate countersway. We noted that similar motions of optical images across the retinas accompanied lateral sways or small horizontal saccadic eye movements. Can the eye movements cause a false feedback signal?

The processing of visual information during a saccadic eye movement is a classic question of perception. In the course of a saccade, the rapid motion of optical images across the retina should appear blurred as a result of temporal summation, yet the blur is seldom seen. Nor does one get the impression that the world moves when the gaze is redirected voluntarily (*Raumkonstanz* or space constancy). In terms of visual information transfer, the perception of blur and of motion is thought to be reduced significantly by loss of visual sensitivity (saccadic suppression) during the course of an eye movement. Studies of visual detection or acuity find reduced sensitivity accompanying saccades, and other visual functions such as target localization may be altered around the times of eye movements (*3*). To our knowledge, however, prior studies have not investigated postural stability—a context of spatial orientation that may interact with voluntary eye movements.

We reasoned that investigating body sway could contribute to our understanding of visual information processing during a saccade since the mechanisms that control sway are behaviorally and neurologically different from those governing the perceptual functions previously studied (*4*). We explored the possibility that visual aspects of body sway control are different in the presence of (i) the voluntarily produced motion of the retinal image accompanying a saccadic eye movement and (ii) an otherwise similar motion produced in the stationary eye by moving the visual surroundings.

Each subject viewed a large (180° horizontal by 120° vertical) grating pattern (0.08 cycle/deg) of gray and white vertical stripes. Near the center were two fixation lights separated horizontally by 4° of arc. The subject was instructed to fixate whichever light was illuminated. Voluntary saccades were directed by illuminating the lights alternately. Externally produced motions of the retinal image were generated while the subject fixated one light. For these, the grating pattern was moved horizontally through 4° of arc in 33 msec, closely approximating the average speed and duration of 4° saccades. Subjects stood on a rigid plate mounted on strain gages (stabilimeter). As the body's center of gravity shifted, forces exerted on each strain gage changed correspondingly. Subjects were instructed to stand on one foot to enhance lateral body sway (*5*).

Figure 1 presents the results, normalized with respect to individual differences. For normalizing, we took Fourier transforms of the lateral sways during 2-second sampling intervals and averaged the power spectra measured successively during each 16- or 32-second recording period ($N = 8$ or 16). These recording periods were either baselines, during which both the pattern and the eyes remained stationary, or tests, during which the conditions of interest were included. Our dependent measure was the ratio of test to baseline power spectra, namely, the gain in sway induced by a test condition. If the test conditions were without effect, the ratio between test and baseline measures would have an expected value of unity (0 dB) for all frequency components. Positive values on the ordinate of Fig. 1 indicate larger sways during the test periods. The abscissa represents frequencies, condensed into bins of 0.5-Hz width. Each curve is a frequency spectrum connecting the median values of sway gain (across subjects) for each condition of interest.

In Fig. 1A, we compare sway baseline data with the data obtained during a test period including saccadic eye movements. The amount of time while standing on one foot increased baseline sway marginally at low frequencies and more markedly from 3.5 to 4.5 Hz. In some tests, the saccades were directed three times per second, and in other tests, aperiodically every few seconds (data were collapsed for plotting). Comparing the tests' results with the control data indicates that retinal image motions accompanying saccades have little influence on body sway. Results are consistent, too, with the everyday experience of walking while reading; these saccades do not disrupt balance.

Figure 1B shows what happened when the pattern surrounding the subject was moved to simulate a saccade (circles and triangles). All sway component frequencies from 1 to 4 Hz show significantly increased gain (beyond the 95 percent confidence limits of control data). The 3-Hz component of sway shows the most reliable increase. To determine whether the frequency dependence resulted because the pattern changed from stationary to moving three times per second (circles), we presented single occurrences of the saccadelike pattern motion at aperiodic intervals of several seconds (triangles). Gain was increased significantly at all frequencies and markedly so at about 3Hz (*6*). Stability was altered so remarkably under these conditions that subjects typically began to fall, and in order to avoid falling they lowered the raised foot.

Our data indicate that body sway depends on whether movement of the retinal image is voluntarily or externally produced. We might ask whether the lack of a visual influence on posture during saccades can be explained by visual masking. Masking is a powerful interfering effect that may be induced by retinal image motions, neural correlates for which can be found as far distal as the retina. Consequently, it has been emphasized as a mechanism involved in saccadic suppression (*7*). In this case, however, the image motions generated in the different conditions are comparable, and effects similar to masking (such as the periphery effects or shift effects on retinal ganglion cell activities) (*8*) should therefore be comparable regardless of how they were generated. Hence these results cannot be explained by visual masking.

How could voluntarily produced retinal image motions be distinguished from externally produced (but otherwise similar) ones in terms of their feedback roles? Since, during rotation of the eye, the angular moments of inertia might introduce retinal shear, it has been proposed that shear may briefly disrupt the neural activities of the retina (*9*). The inadequacy of a shear model is easily demonstrated. If one stands on one foot, occludes one eye, and then quickly rotates the open eye by pushing it with a finger (*10*), one tends to lose balance and fall to the side. Although retinal shears are set up by the atypical manner of eye rotation, visual stimulation nevertheless influences body sway. The use of voluntary finger movements to effect an eye movement hints that neither motor acts per se nor eye motion per se are effective in eliminating evoked sway.

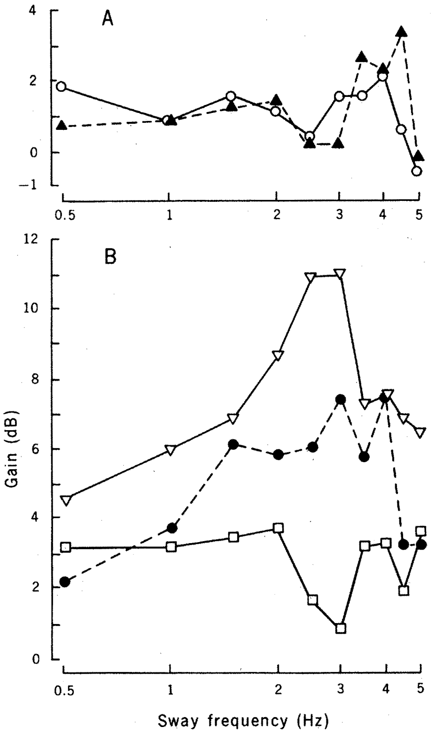

Fig. 1. Dependence of body sway responses on various testing conditions. Thirteen adult human subjects participated in the experiments, but not all were tested under every condition. Each frequency spectrum plots median gains (across subjects) derived from the average (within subject) lateral sway power spectra. Confidence intervals are not shown on the graphs to avoid clutter. The average confidence intervals are ±1.5 dB for (A) and ±2 dB for (B) (68 percent confidence intervals of medians, calculated nonparametrically) (*13*). (A) ▲, Relative gain in sway for preliminary versus final baselines, during each of which the pattern and the eyes were both stationary. The lapse of time between preliminary and final periods is a control for fatigue effects. ○, Gain in sway (decibels relative to the preliminary baseline) when the surrounding pattern remained stationary but subjects executed 4° voluntary saccades as directed by alternating fixation lights. (B) ●, Gain in sway when the subjects fixated one light but the pattern was changed from stationary to moving three times per second. Each movement of the pattern required 33 msec to travel 4° of arc, simulating the visual motion of voluntary saccades. ▽, Gain in sway when the subjects fixated steadily but the pattern changed from stationary to a saccadelike motion at irregular intervals. □, Gain in sway when subjects made voluntary saccades three times per second; during each 300-msec fixational pause the pattern was changed from stationary to a saccadelike motion.

Another possibility is that execution of a motor act that normally affects seeing can briefly shift attention away from visual feedback cues. We assumed that voluntary blinks are as effective as voluntary saccades (*11*) for shifting attention in this way, and on these grounds tested whether blinks and saccades could result in equivalent body sway. Our preliminary findings show, however, that sway increases with a different frequency dependence during blinking and during eye movements.

A further possibility is suggested by certain theoretical treatments of saccadic suppression and the perceived stability of the world during saccades. These propose that motor commands to move the eyes modify sensory activity evoked by retinal image motion (*12*). In effect, the nervous system is believed to anticipate that retinal image motion will accompany voluntary eye movements and to cancel its impact as false feedback.

If a cancellation effect originates in the central nervous system, it is likely to either precede or outlast the actual eye movement. Thus, an indirect test for central cancellation is whether sway evoked by externally presented motion can be altered when saccades occur in close temporal proximity. In supplementary experiments, voluntary saccades and externally presented pattern movements were interleaved with slight temporal offsets. Subjects made saccades between the fixation lights (three per second), and during each 300-msec fixational pause the grating pattern moved. Frequency components of sway below 2.5 Hz were increased marginally, whereas those at higher frequencies were not (squares in Fig. 1B). Gains in the mid-frequency range were never as great as those evoked by pattern motions presented during steady fixation. At 3 Hz (the former peak), gain in sway was less than 1 dB. When the eye was in a fixational pause with close temporal proximity to a saccade, the pattern motions were plainly seen but evoked different (smaller) sway responses. These findings are consistent with a special role of the motor commands per se.

We infer that the central nervous system responds differently to visual image motion accompanying a saccade than to that which is externally produced. Neither visual masking effects nor the effects of retinal shear can explain our results.

Currently accepted views of the changes in visual sensitivity during saccades attribute saccadic-suppression effects predominantly to sensory factors. These include visual masking, optical blur, and perhaps proprioceptive feedback from the extraocular muscles. The role of efferent commands in reducing visual sensitivity is believed to be minor. For the visual control of sway, however, the relative contributions of afferent and efferent factors appear to be reversed. The need to omit inappropriate responses to self-produced visual motion during eye movements appears to be satisfied in distinctive ways by the mechanisms underlying visual sensitivity and those underlying postural stability.

KEITH D. WHITE
Department of Psychology, University of Florida, Gainesville 32611
R. B. POST
H. W. LEIBOWITZ
Department of Psychology,
Pennsylvania State University,
University Park 16802

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13 November 1979

Opioid and Nonopioid Mechanisms of Stress Analgesia

Abstract. *Inescapable foot shock in rats caused profound analgesia that was antagonized by naloxone or dexamethasone when shock was delivered intermittently for 30 minutes, but not when it was delivered continuously for 3 minutes. Thus, depending only on its temporal characteristics, foot-shock stress appears to activate opioid or nonopioid analgesia mechanisms. Certain forms of stress may act as natural inputs to an endogenous opiate analgesia system.*

Early work on the analgesic effect of electrical brain stimulation led to the suggestion that portions of the central nervous system normally function to inhibit pain (*1*). This hypothesis gained considerable support from the discovery of opioid peptides with analgesic properties (*2*) and a distribution encompassing regions of the nervous system implicated in stimulation-produced and opiate analgesia (*3*). Suggesting that opioid peptides act as chemical mediators in this endogenous system of pain inhibition are studies showing that an opiate antagonist can block stimulation-produced analgesia (*4*), that cross-tolerance develops between stimulation-produced and opiate analgesia (*5*), and that opioid peptide concentration in human cerebrospinal fluid is altered by chronic pain and by analgesic central or peripheral stimulation (*6*).

To establish that opioids serve a biologically significant role in pain inhibition, the natural factors activating their release need to be identified. So far, only fragmentary or contradictory evidence related to this point is available (*7*). Perhaps the most promising lead comes from the recent demonstration that various stressors can cause analgesia (*8, 9*). Whether opioids mediate stress analgesia, however, has remained in doubt. Some reports indicate that stress analgesia is attenuated by the opiate antagonist naloxone (*8, 10, 11*) and that cross-tolerance develops between opiate and stress analgesia (*10, 12*). Moreover, stressors that induce analgesia alter brain and plasma indices of opioid activity (*13*). Other studies, however, indicate that stress analgesia neither manifests cross-tolerance with morphine nor is antagonized by naloxone (*14, 15*).

Consideration of the stress paradigms used in these studies reveals both qualitative and quantitative differences that may account for the disparity in the findings. The principal aim of the present experiment was to investigate the possibility that both opioid and nonopioid systems influence stress analgesia and that quantitative characteristics of a given stressor can determine which system is predominantly engaged. We find that, depending on its temporal parameters, inescapable foot shock can cause either an opioid or a nonopioid type of analgesia, as defined by susceptibility to naloxone blockade (*16*).

Because the analgesic effects of stress (*17*) and acupuncture (*18*) are reduced by hypophysectomy, it has been suggested that pituitary hormones mediate these forms of analgesia. Some pituitary cells contain both adrenocorticotropic hormone (ACTH) and β-endorphin (*19*) and release them concomitantly in response to stress (*20*). The synthetic glucocorticoid dexamethasone blocks the stress-induced rise in plasma ACTH (*21*) and β-endorphin (*22*) and decreases acupuncture analgesia in mice (*23*). Therefore, a second aim of our experiment was to in-

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