SIGNAL TRANSFORMATIONS REQUIRED FOR THE GENERATION OF SACCADIC EYE MOVEMENTS

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INTRODUCTION

Saccades (high velocity, conjugate rotations of the eyes) function to bring the image of selected visual targets onto regions of the retina containing a high density of photoreceptors. Recent reviews (Robinson 1981a, Fuchs et al 1985) have documented the progress made in describing the neural signals controlling these rapid, precise movements. These papers considered the literature from the perspective of the motoneuron, emphasizing the generation of command signals by brainstem neurons having relatively direct synaptic connections with the motoneuron pools innervating the extraocular muscles. In this review, we summarize what is known about saccadic command signals found at a more central point-the superior colliculus (SC). Using this information as well as kinematic arguments, we discuss the transformations required to convert collicular signals into those needed by premotor pools of neurons. Also, we consider the functional properties of other brainstem neurons in the context of the required transformations.

SACCADIC COMMAND SIGNALS OBSERVED IN THE SUPERIOR COLLICULUS

There is now compelling evidence that the intermediate and deep layers of the SC contain neurons that are critical components of the neural circuitry initiating and controlling saccadic eye movements (see Sparks 1986, Sparks & Jay 1986, Sparks & Hartwich-Young 1988 for recent reviews). The intermediate and deep layers receive inputs from brain areas involved in the analysis of sensory (visual, auditory, and somatosensory) signals used to guide saccadic eye movements (see Huerta & Harting 1984, Sparks 1986 for references). Neurons in the intermediate and deep layers project to brainstem areas known to be important in the generation of saccadic eye movements and to nuclei containing neurons having monosynaptic connections with motoneurons (Highstein et al 1974, 1976, Harting 1977, Huerta & Harting 1984).

Electrophysiological data support the assertion that the SC is involved in saccade initiation. Many collicular neurons generate a high-frequency pulse of spike activity that precedes saccade onset by approximately 20 msec (Sparks 1978). These neurons have movement fields (i.e. they discharge before saccades with a particular range of directions and amplitudes) and, since they are topographically organized according to their movement fields (Schiller & Koerner 1971, Wurtz & Goldberg 1972a, Sparks et al 1976), form a map of saccadic motor error (defined below). Additionally, microstimulation of a discrete point in the SC reliably produces a saccade with a particular direction and amplitude that is comparable in velocity and trajectory to a visually guided saccade (Robinson 1972a, Schiller & Stryker 1972). The current required to elicit a short-latency (20-30 msec) saccade is low (5-20 μamp for stimulus trains of 40 msec duration, 500 pulses/sec).

In contrast to interpretations of early lesion studies, recent experiments attest to the importance of the SC in the generation of saccadic eye movements (see Sparks 1986 for a review). For example, reversible inactivation of collicular neurons severely impairs the direction, amplitude, and velocity of visually guided saccades (Hikosaka & Wurtz 1985, 1986, Lee et al 1988); and monkeys with combined lesions of the SC and frontal eye fields are unable to initiate saccades to visual targets (Schiller et al 1980).

Given the critical role of collicular neurons in the control of saccadic movements, how is information about the direction, amplitude, and velocity of saccades encoded by these cells? First, the discharge of collicular neurons is related to certain changes in eye position, not to movement of the eye to a particular position in the orbit. Each collicular neuron dis-

charges before saccades in its movement field regardless of the original position of the eye in the orbit (Schiller & Koerner 1971, Wurtz & Goldberg 1972a, Sparks et al 1976).

Second, it is the location of the active population of collicular neurons within the topographical map of movement fields, not their frequency of firing, that codes information about saccadic motor error (the direction and amplitude of the saccade required to direct gaze to the target). Saccade-related burst neurons are arranged topographically within the SC corresponding to a motor map first described in detail by microstimulation studies (Robinson 1972a). Although there is a spatial and a temporal gradient of activity within the population of cells discharging before a saccade (Sparks et al 1976), identical bursts of a single neuron may precede many saccades with different directions and amplitudes (Sparks & Mays 1980). Unlike medium-lead burst neurons in the pons (see below), an individual saccade-related burst neuron in the SC does not encode saccadic direction or amplitude by its firing pattern.

Third, the location of the active population of collicular neurons encodes initial saccadic motor error, not dynamic saccadic motor error. When a visual target appears in the peripheral visual field, initially (before the onset of a saccade) saccadic motor error is large. During the saccade, the motor error is rapidly reduced until, if the saccade is accurate, the error is zero. However, during the saccade the site of activity within the SC does not progressively shift toward the rostral pole of the SC, as it would if the site of activity were also encoding the dynamic changes in saccadic motor error occurring during a saccade (see Fuchs et al 1985, Tweed & Vilis 1985 for additional discussion of this point).[1]

Fourth, saccadic velocity may be related to the level of activity within the active population, since reversible deactivation of collicular neurons produces dramatic reductions in saccadic velocity (Hikosaka & Wurtz 1985, Lee et al 1988). Although earlier experiments (Sparks & Mays 1980)

[1] In a recent research note, Waitzman and colleagues (Waitzman et al 1988) present findings that they interpret as supporting the hypothesis that the discharge frequency of individual neurons in the SC encodes dynamic motor error. Their major observation is that the burst frequency of many collicular neurons gradually decays during a saccade. The time course of the decay resembles the time course of the decay in motor error. This hypothesis needs to be tested in more detail. Seemingly, the hypothesis would have difficulty explaining why electrical stimulation at a constant frequency for varying time intervals (presumably disrupting the frequency code) produces saccades with a relatively constant amplitude. Moreover, it has been reported (Sparks & Mays 1980) that the duration of the burst of neurons discharging maximally before small saccades (1-3° in amplitude) outlasts, significantly, the duration of the saccade. Finally, it is possible that the observed decay in spike frequency is related to a decrease in saccadic velocity rather than to a decrease in motor error, a possibility not considered in the report.

did not reveal a relationship between the discharge of saccade-related burst neurons and saccadic velocity, this was probably due to the small range of velocities observed when movements of the same direction and amplitude were made to continuously present visual targets. Recently, under conditions in which saccadic velocity had greater variability (saccades to remembered targets or saccades to auditory targets), a positive correlation between the average firing rate of collicular cells and saccadic velocity was observed (Rohrer et al 1987). A similar relationship between the instantaneous firing frequency of collicular cells and saccadic velocity has been described also in cats (Berthoz et al 1986, Munoz & Guitton 1986), animals that show large variations in saccadic velocity, even for visually guided saccades. Since the activity of a given population of SC neurons is associated with a saccade having a particular direction and a particular amplitude, the level of activity within this population is related to the velocity of the overall movement (vectorial velocity) and not directly related to the velocity of either the horizontal or vertical component of the rotation (component velocity).

Fifth, the collicular saccadic command signal may not fully compensate for the original position of the eye in the orbit. Saccades to visual targets compensate for the initial position of the eye in the orbit since systematic errors in accuracy are not observed as a function of initial eye position (Ritchie *1976,* Pelisson & Prablanc 1988). Saccades produced by collicular stimulation do not fully compensate for the initial position. For example, stimulation of a collicular site that produces a purely horizontal saccade 10° in amplitude when the eye is in the center of the orbit will produce a saccade with a small downward component if the eye is elevated and a small upward component if, before stimulation, the eye is depressed. Similarly, centrifugal stimulation-induced saccades are smaller than centripetal ones (Segraves & Goldberg 1984, D. L. Sparks, unpublished observations). Noticeable differences in burst parameters as a function of original eye position have not been reported for neurons in the SC.

To summarize, each visually guided or spontaneously occurring saccade is preceded by the discharge of a relatively large population of neurons in the intermediate and deep layers of the SC. The location of this active population encodes initial motor error, the direction and amplitude of the change in eye position required for target acquisition. The rate of firing of neurons in the active population may be a determinant of the vectorial velocity of the ensuing saccade. Apparently, the saccadic command signal originating in the SC does not fully compensate for the differences in innervation required to produce comparable rotations from different starting positions (see below).

THE OCULOMOTOR PLANT AND ITS INNERVATION

The neural signals observed in the SC are quite different from the innervation signals required for saccadic rotations of the eye. These differences become apparent when the properties of the oculomotor plant (the globe, extraocular muscles, orbital suspensory tissues or any other passive orbital tissues influencing rotation of the eye) and kinematic constraints upon the rotations of the eye are considered.

The Oculomotor Plant

Mathematical models of the oculomotor plant and the innervation required to produce saccadic eye movements have been developed by several investigators (Cook & Stark 1967, Clark & Stark 1974, Collins 1975, Bahill et al 1980, Robinson 1981b). These models are based upon available measurements of muscle and globe parameters but also must employ a number of approximations and simplifying assumptions. Because of a lack of experimental data on the behavior of the globe and muscles in situ, the models are seriously underdetermined (Robinson 1981b). Thus, if parameters are chosen carefully, all the models generate normally appearing saccades, although some require unrealistic neural inputs. Nonetheless, there is considerable agreement on two points.

First, all of the models recognize the highly overdamped characteristics of the plant and require a pulse and step of innervation to produce a saccade. The *pulse* of innervation produces a phasic increase in muscle tension that overcomes the viscous drag of the orbital tissues and moves the eye at a high velocity. The step of innervation causes the sustained change in muscle tension required to hold the eye in the new position and to overcome the elastic restoring innervations of the orbital tisue. In addition, Robinson's model of the plant (1964) has a third time constant so that the innervation required is a pulse of high-frequency activity followed by an exponential slide in instantaneous frequency to a final step level of innervation.

Second, the total innervation needed to hold the eyes in a fixed position is strongly dependent upon eye position (Collins 1975, Robinson 1975a); the innervation required to produce a saccade of a given amplitude depends upon both initial position and saccade direction (Robinson 1975a, Optican & Robinson 1980). This indicates that information about changes in eye position present in the SC is not sufficient to produce accurate saccades; information about absolute eye position must be used as well.

Kinematic Considerations

Lawful relationships observed in kinematic studies must be accounted for by the mechanical properties of the plant or by the innervation signals supplied to the plant. If the small translations associated with ocular rotations are ignored, the eye can be modeled as a perfect ball-and-socket joint requiring three independent variables to describe eye position unambiguously. According to Donders' law, however, saccadic eye movements are accomplished using only two degrees of freedom of rotational movements. This law states that the relationship of the vertical meridian of the eye with respect to a vertical reference (e.g. a plumb line) is invariant for a given eye position, regardless of how the eye got to that position. Thus, the torsion, or twist of the eye about the line of sight, when the eye is directed 10° above and 10° to the left of the primary position, is the same whether that position is achieved by a 10° upward and then a 10° leftward movement, or whether the movements are executed in opposite order. Listing's law specifies the amount of this cyclorotation, relative to the vertical, at any given position. Donders' law *is* not a universal property of rigid bodies free to rotate in three dimensions. Such rotations are not commutative; the order of the rotations about the horizontal and vertical axes is important in determining the amount of torsion at the final position. Examples of this effect are shown by Nakayama (1975) and Tweed & Vilis (1987). Moreover, the eye is not mechanically constrained to observe Donders' law. That voluntary saccades are accomplished using only two degrees of freedom is a consequence of the pattern of innervation. Donders' law does not hold for movements occurring, for example, during sleep (Nakayama 1975), during pursuit eye movements (Westheimer & McKee 1973, Ferman et al 1987b), or during vergence movements (Nakayama 1983).

The implications of Donders' and Listing's laws for models of the neural mechanisms generating saccades have been discussed by Westheimer (1954, 1957, 1981) and Nakayama (1975, 1983). They state that the neural circuitry must specify an absolute, as opposed to a relative, eye position. If the saccade generator simply issued instructions for relative rotations (i.e. move 3° down and 6° to the right), torsional positions violating Donders' law would often be observed, since the amount of torsion observed would depend upon the presaccadic position of the eye in the orbit (Nakayama 1975, Westheimer 1981).

Orbital Geometry and Muscle Interactions

The implications of kinematics for neural control depend on orbital geometry and muscle interactions as well. Robinson (1975a) developed a

computer model of the plant that analyzed the interactions among the six extraocular muscles. This muscle interaction model provides values for the length,, force, and the innervation signal required to generate that force, for each muscle when the eye is in any orbital position. He emphasized that the rotation produced by contraction of any single extraocular muscle depends upon the state of contraction of all the remaining muscles. Moreover, muscles interfere with each other, and there is considerable cross-coupling between muscles (Robinson 1975a). Thus, the innervation patterns required to obey Listing's and Donders' laws cannot be deduced by considering the planes of action of each of the muscles individually. An important outcome of Robinson's model is that iso-innervation curves predicted for each of the six extraocular muscles when constrained by Listing's law are considerably straighter and more nearly parallel to the horizontal and vertical meridians than would have been expected by observing the pulling actions of individual muscles at various eye positions. From the point of view of generating saccadic commands, Robinson (1975a) noted that the innervational participation of a muscle is different from its mechanical participation.

A recent study by Miller (1989) also indicates that orbital geometry may profoundly affect the signal transformations required in brainstem circuits. He used magnetic resonance imaging to generate pictures of the recti muscles and optic nerve of human observers while the eye was in a number of different orbital positions. He reported that there is almost no side-slip of the recti muscles and that the muscle planes, but not necessarily the axes of rotation, are essentially fixed in the orbit. Two models consistent with his observations were considered. In both, side-slip of the muscles is prevented by a "harness" composed of fascia bands connecting the muscles with each other and with the bony orbit. In one model, the muscle sheaths act as a pulley fixed to the orbit such that the axis of rotation moves with the eye. Ignoring the problem of recruitment of motoneurons, this model would permit saccadic commands to be computed in retinotopic or global coordinates, independent of the position of the eye in the orbit. For example, contraction of the lateral rectus would cause rotation along an axis perpendicular to the plane of the horizontal meridian of the eye over a wide range of elevations. In contrast, the second model assumes no significant linkage between the muscle "harness" and the orbit; neural commands would need to be organized in orbital rather than retinotopic coordinates. Models with intermediate characteristics can also be envisioned.

In summary, the most realistic models of the oculomotor plant suggest that either a pulse/step or a pulse/slide/step innervation signal should be observed. Furthermore, the amplitude of the total innervation of both the pulse and the step should vary for identical saccades with different starting

positions. This implies that, ultimately, saccade commands must be commands to move the eye to a particular orbital position, not commands to produce relative rotations. Finally, cross-coupling between muscles and orbital geometry are important determinants of the coordinates in which innervation signals must be organized.

MOTONEURON ACTIVITY

The Pulse/Slide/Step

The pattern of activity observed at the single motoneuron level is a pulse/slide/step waveform, the pattern of innervation suggested by Robinson's early model (1964) of the plant. A high frequency burst of motoneuron activity precedes, by about 8 msec, the onset of saccades in the on-direction (Fuchs & Luschei 1970, 1971, Schiller 1970, Robinson 1970). The pulse of activity is not rectangular, as some models assume, but decays with both a short and a long time constant to the steady-state level of activity associated with the new position (Fuchs & Luschei 1970, Robinson 1970, Goldstein 1983).

Nonlinear Relationship Between Muscle Innervation and Eye Position

It is clear from an analysis of the plant (Collins 1975, Robinson 1975a) that the total innervation to a given muscle is a nonlinear increasing function of the rotation in the on-direction. In order to move the eyes in a series of equal amplitude saccades to more eccentric positions, successively greater innervation increments are required. The firing patterns of individual motoneurons show a simpler relationship to eye position than that suggested by analyses of total innervation. For steady fixation, the activity of any motoneuron is linearly proportional to eye position along its on-direction. The static firing rate of a motoneuron can be described by two parameters: a threshold position and the slope of the line of best fit for the firing rate vs. eye position plot (Robinson 1970, Goldstein & Robinson 1986). The major mechanism for generating a nonlinear relationship between muscle innervation and eye position appears to be recruitment of more and more motoneurons. For static positions, the recruitment order of extraocular motoneurons is relatively fixed (Robinson 1970, Keller 1981, Fuchs et al 1988). Motoneurons with larger rate-position slopes tend to be recruited later as the eye moves in the on-direction and, according to Robinson (1970), all motoneurons for a given muscle are recruited by the point at which the eye has moved approximately 20° beyond the primary position in the on-direction. If this observation is true, increased muscle innervation beyond 20° must be generated by increased activity

rather than recruitment of additional motoneurons. This implies that the total innervation will be a linear function of eye position once all motoneurons are recruited. Van Gisbergen (1988) plotted total neural innervation as a function of eye position for a hypothetical motoneuron pool. He assumed that the recruitment thresholds of the motoneuron pool were distributed homogeneously over the observed range of recruitment thresholds and that the threshold and the slope of the rate/position curve were correlated (Robinson 1970, Keller 1981). The resulting curve resembled, somewhat, the isometric muscle-tension relation described by Collins (1975) in that it was nonlinear up to the point at which all motoneurons were recruited.

The nonlinear relationship between innervation and eye position during the dynamic phase of saccades is poorly understood. For small saccades, the burst frequency of motoneurons is related to saccadic velocity and amplitude. But for saccades larger than 10°, burst frequency saturates and becomes unrelated to saccadic amplitude. Because of this saturation, the major determinant of saccadic amplitude for large saccades is burst duration. With respect to recruitment, Robinson (1970) and Goldstein (1983) reported that, during saccades, burst rate depends upon the starting position of the saccade relative to the unit's threshold position; the velocity signals reaching individual neurons in the abducens nucleus may not add linearly to position signals (Goldstein 1983). Whether or not the pulse innervation of the entire motoneuron pool is dependent upon saccadic starting position is unknown (van Gisbergen 1988). Nonetheless, it should be noted that the effective saturation of the pulse in generating force occurs at a lower firing rate than that exhibited by motoneurons. Motoneurons may burst at instantaneous rates *of 600* spikes/sec (Robinson 1970), yet artificial stimulation suggests that little additional tension is generated above 200-300 Hz (Robinson 1981b, Nelson et al 1986).

Organization of Commands in Horizontal and Vertical Coordinates

Hepp & Henn (1985) measured the frequency of firing of mononeurons innervating the six extraocular muscles during periods of fixation within 30° of the primary position. They constructed plots of the horizontal and vertical eye positions associated with particular rates of firing of single motoneurons. As expected, the iso-frequency plots of individual motoneurons innervating the horizontal recti formed a family of curves that were approximately parallel with the vertical meridian. Iso-frequency plots of motoneurons innervating the inferior rectus and most motoneurons with upward on-directions were approximately parallel with the horizontal meridian. (For technical reasons, it was not possible to distinguish inferior oblique from superior rectus motoneurons). The iso-frequency gradients of moto-

neurons innervating the superior oblique muscle depended upon horizontal eye position only when the innervated eye was abducted. The discharge frequency of motoneurons innervating the oblique muscles and the vertical recti was more highly correlated with the horizontal or vertical position of the eye in the orbit than would be expected based upon knowledge of the axis of rotation produced by these muscle pairs acting independently.

The results of the Hepp & Henn (1985) study were, in general, consistent with Robinson's (1975a) analysis of muscle interactions. According to this model, the static innervation signals required by each extraocular muscle are approximately parallel to either the horizontal or the vertical meridian. This suggests that the characteristics of the oculomotor plant itself may simplify the neural computations needed to satisfy Listing's law. Based upon the findings of Hepp & Henn (1985) and Robinson's (1975a) analysis, it is possible that Listing's law would he obeyed approximately, without explicitly computing a torsional command, if signals were simply organized in horizontal and vertical coordinates. The plausibility of this idea depends upon how well Listing's law is actually obeyed.. Ferman and colleagues (1987a,b) tested the validity of Listing's law directly. They concluded that the control of torsion by the saccadic system is usually not precise, and that Listing's and Donders' laws are only obeyed approximately. Long-term fluctuations of up to 5° of torsion were observed with the eye in the primary position, and systematic deviations from values predicted by Listing's law were seen in secondary and tertiary positions.

In summary, the signals needed by the oculomotor plant and supplied by motoneurons are quite different from those found in the SC. Although collicular cells generate a presaccadic pulse of activity, they do not provide a step signal related to steady eye position. Collicular neurons specify relative rotations of the eye, not commands to move the eye to an absolute orbital position. There is no evidence that the magnitude of the saccade-related burst associated with saccades of a particular direction and amplitude depends upon initial eye position. Finally, the map of collicular activity is not organized along either the planes of action of the extraocular muscles or in horizontal and vertical coordinates.

FORMATION OF INNERVATION SIGNALS BY BRAINSTEM CIRCUITS AND THE REQUIRED TRANSFORMATIONS OF COLLICULAR SIGNALS

Formation of the Pulse/Slide/Step Signal

BRAINSTEM SIGNALS Four major functional classes of neurons are observed in the paramedian pontine reticular formation (PPRF), a brain

region critical for the generation of horizontal saccades. Medium-lead burst neurons (MLBs) generate a high-frequency burst before ipsilateral saccades. Omnipause neurons fire at a relatively constant rate during fixation and pause during saccades in all directions. Tonic neurons discharge at a frequency proportional to horizontal eye position, and long-lead burst neurons (LLBNs) generate a low-level increase in activity and a vigorous burst before saccades (see Raphan & Cohen 1978, Fuchs et al 1985 for reviews).

Robinson used three of these neural elements in a local feedback model (1975b) designed to simulate horizontal saccades. The model has two inputs: a signal of the desired horizontal position of the eyes (DHP) and a trigger signal. An important feature of the model is that the input specifying saccade amplitude does so by providing a signal of final eye position in the orbit, not the required displacement of the eye. Saccades are initiated by a trigger signal that briefly inhibits the pause cells, permitting the MLBs to discharge at a rate proportional to horizontal motor error (the difference between DHP and an internal estimate of current horizontal eye position, CHP). In the model, the pulse of activity generated by MLBs is transmitted directly to motoneurons and to a neural integrator. The neural integrator converts the pulse into a step of activity (observed in the activity of tonic cells) that is sent to motoneurons and used as the estimate of CHP. Once activated, this circuit drives the eye at a high velocity until the representation of CHP matches the DHP signal. At that point, the eye stops on target and the pause cells are allowed to resume firing, thereby inactivating the saccadic generator until a new trigger signal arrives.

Robinson's original model produces a pulse (output of MLBs) and step (output of tonic neurons) of innervation as observed in motoneuron activity. A modification of the model proposed by Optican & Miles (1985) also generates the slide component of motoneuron activity. Given such a circuit, the task of more central components of the saccadic system is to provide the input signals required by the pontine network: a trigger signal, and a signal of the DHP. Provided with these signals, the PPRF circuit automatically generates the pulse/slide/step signal that is transmitted to the motoneurons. The computations required to extract these signals from those observed in the SC are discussed below.

Medium-lead burst neurons identified in the rostral midbrain [the interstitial nucleus of Cajal (INC) and the rostral interstitial nucleus of the medial longitudinal fasciculus (riMLF)] have functional properties similar to those observed in the PPRF except that during saccades their discharge seems to be related to the vertical, instead of the horizontal, component of eye movements (Buttner et al 1977a,b, King & Fuchs 1977, 1979, King

et al 1981).[2] Robinson's model, as modified by Optican & Miles, could be extended to this population of vertical bursters to generate a pulse/slide/step signal for motoneurons providing innervation signals for vertical rotations of the eye. Since pause cells in the PPRF stop firing before both horizontal and vertical saccades, and microstimulation of the pause region inhibits the occurrence of both horizontal and vertical saccades (Keller 1977, Evinger et al 1977), the pause cells control both the horizontal and vertical pulse generator circuits.

The vertical pulse generator circuit is more complex than the horizontal circuit. Cells in the riMLF and INC project to motoneurons innervating the vertical recti and oblique muscles (Buttner-Ennever & Buttner 1978) and two subsets of vertical MLBs organized in planes similar to those of the anterior and posterior semicircular canals have been functionally identified (Hepp et al 1986). During visually elicited saccades with the head stationary, **MLBs** with upward or downward on-directions are found bilaterally in the riMLF (Hepp et al 1986). However, these neurons can be subdivided into two subpopulations by observing their activity during sinusoidal head rotation in the dark. For example, during head rotation in the plane of the right-anterior/left-posterior semicircular canals, MLBs in the right riMLF with downward on-directions and MLBs in the left riMLF with upward on-directions are most vigorously activated. The firing rates observed during rotations in this plane are significantly higher than firing rates associated with saccadic eye movements in the light. In a companion study, Vilis and colleagues (Vilis et al 1986) reported that unilateral microinjections of muscimol, a GABA agonist, into the riMLF disrupted saccades along specific planes. Injections in the right riMLF impaired visually elicited saccades to targets in the upper left and lower right quadrants of the visual field. Conversely, injections into the left riMLF impaired saccades to targets in the upper right and lower left quadrants.

As suggested by Robinson (1972b), saccades are controlled, at least in part, by neural circuits subserving the phylogenetically older vestibulo-ocular-reflex (VOR). Accordingly, rapid vertical rotations of the eyes can be executed in two modes (Villis et al 1986). During vestibular nystagmus, quick phases in the plane of head rotation (movements that may not obey Listing's law) are produced by preferentially activating subsets of riMLF

[2] Robinson & Zee (1981) hypothesized that the on-directions of vertical burst neurons were organized in the canal planes. They noted that if so, the on-directions of vertical burst cells could not be determined by observing their activity only during visually guided saccades, since the saccadic system operates under the constraints of Listing's law and does not generate rotations in the planes of the anterior and posterior canals.

burst neurons. Visually elicited saccades are produced by bilateral activation of burst neurons in the riMLF. Presumably, variations in the symmetry of bilateral activation are associated with differences in the torsional position of the eye. Appropriate innervation ratios for these burst neurons are required if the saccadic system is to approximate Listing's law.

REQUIRED TRANSFORMATIONS OF COLLICULAR SIGNALS Robinson's model of the saccadic system (1975b) as revised by Optican & Miles (1985) will automatically produce the pulse/slide/step of innervation if the horizontal and vertical burst generator circuits are provided with a trigger signal, a signal of the desired horizontal position of the eye in the orbit, and a signal of the desired vertical position of the eye in the orbit.

Saccade-related burst neurons (SRBNs) observed in the SC could serve as the trigger input required by Robinson's model (Robinson 1975b) of the saccadic system (Sparks & Mays 1980). The axons of **SRBNs** comprise a major efferent pathway from the SC to subsequent premotor neurons, particularly the **PPRF. SRBNs** meet the requirements for a trigger input; they generate a high-frequency burst of activity tightly linked to saccade onset. The burst precedes saccade onset by an appropriate time. Keller's (1980) observations that 10 out of 10 SRBNs recorded in the SC are antidromically activated by stimulation of the region of the PPRF containing pause units, supports the suggestion that the SC provides a trigger input to the PPRF. Only 1 of 11 other collicular neurons with saccade-related discharges, but lacking the high-frequency burst, was antidromically activated by PPRF stimulation.

Robinson's model (1975b) does not specify how a signal of DHP is generated, but the formation of such a signal requires additional processing. Neurons in the SC generate commands for changes in eye position or desired displacement, rather than commands to move the eye to a particular position in the orbit. The motor error signals recorded in these areas must be decomposed into appropriate horizontal and vertical components [desired horizontal displacement (DHD), and desired vertical displacement (DVD)], and an estimate of CHP must be added to the DHD signal to form a signal of DHP. Modified versions of Robinson's model assume that the horizontal and vertical pulse/step generators are driven by signals of desired displacement instead of desired position. The extraction of desired displacement signals is discussed below.

Recruitment and Compensation for Presaccadic Eye Position

Little experimental attention has been given to the question of how signals reaching the motoneuron pools control recruitment. Tonic units in the

PPRF (thought to project to motoneurons) are recruited in a relatively continuous manner out to approximately 25° in the on-direction; some of these cells have nonlinear rate position curves, displaying increased slopes at more lateral positions (Luschei & Fuchs 1972, Keller 1974). King and colleagues (1981) did not find a significant correlation between the slope of the rate/position curve and the recruitment threshold for vertical burst-tonic neurons near the INC. In a recent study, Fuchs and colleagues (1988) studied the recruitment order of identified motoneurons and internuclear neurons in the monkey abducens nucleus. Motoneurons in the abducens nucleus innervate the ipsilateral lateral rectus muscle and display a linear increase in firing rate associated with progressively larger temporal rotations of the eye. The axons of internuclear neurons travel in the medial longitudinal fasciculus to the subdivision of the oculomotor nucleus containing motoneurons that innervate the medial rectus of the contralateral eye. Abducens internuclear neurons are involved in the coordination of yoked, conjugate movements of the two eyes and show increases in firing rate when the ipsilateral eye moves temporally and the contralateral eye moves nasally. Fuchs and collaborators (1988) showed that, in contrast to motoneurons, there is only a weak relationship between the rate-position slope and recruitment threshold for internuclear neurons. This suggests that the recruitment order of motoneurons may be established at the level of the motoneuron pool. A single recruitment mechanism shared by all conjugate subsystems might explain the lack of strong eye position effects found in the saccadic subsystem.

In addition to a shared recruitment mechanism for generating the nonlinear innervation signal, separate neural circuits may form supplementary signals to compensate for the presaccadic eye position. Ritchie (1976) reported that rhesus monkeys with lesions of the posterior vermis made hypermetric centripetal saccades and hypometric centrifugal saccades. Optican & Robinson (1980) also studied the effects of cerebellar lesions upon saccades in different parts of the oculomotor range. Their lesions, larger than those made by Ritchie, included the posterior vermis, paravermis, and fastigial nuclei. Monkeys with these lesions made hypermetric saccades, but centripetal saccades were significantly less hypermetric than centrifugal ones. Optican & Robinson (1980) concluded that the cerebellum acts as an interface between visual commands and motor performance by providing (directly or indirectly) different innervation signals to the motoneurons for the same retinal error signal, depending upon the orbital position and direction of the impending saccade. The route by which such signals are conveyed to motoneurons is unknown.

In the same study, Optican & Robinson (1980) found that animals with an intact cerebellum can adjust the gain of the saccadic pulse and step to

compensate for surgically induced alterations in the mechanical properties of the muscle. Cerebellar lesions impair the ability of animals to make these gain changes. Since cerebellar lesions also disrupt the ability of the animal to compensate for initial eye position (Ritchie 1976, Optican & Robinson 1980), the mechanism responsible for the adaptive adjustment of saccadic parameters could also be responsible for compensating for initial eye position.

The saccadic command signals found in the SC may need to be supplemented by signals compensating for orbital position. Although not studied in detail, neither the saccade-related discharge of collicular neurons (Wurtz & Goldberg 1972a, Sparks et al 1976) nor the discharge of MLBs in the PPRF (Keller 1974, van Gisbergen et al 1981) appears to depend upon initial eye position. As noted above, stimulation-induced saccades do not fully compensate for initial eye position (Segraves & Goldberg 1984, **D. L.** Sparks, unpublished observations). The effects of original eye position upon the trajectory of the stimulation-induced movement, however, are small. Moreover, the saccades produced by collicular stimulation are comparable to natural saccades in their amplitude-velocity characteristics, and the eyes maintain their new position after a stimulation-induced saccade. This suggests that the basic motoneuron recruitment mechanism is intact, but there is a failure to provide a small adjustment for initial position.

A neural circuit involving neurons in the nucleus reticularis tegmenti pontis (NRTP) and cerebellar vermis may be associated with the generation of a compensatory signal. The region of the NRTP containing cells with saccade-related activity receives extensive projections from the intermediate layers of the SC (Harting 1977) and sends most, if not all, of its efferent projections to the cerebellum (Brodal & Jansen 1946, Gerrits & Voogd 1986, 1987). Cells in the NRTP with saccade-related activity have movement fields similar to those described for cells in the SC (Crandall & Keller 1985), but, more importantly, the vigor of the burst of NRTP cells differs depending upon the origin of the movement (Crandall & Keller 1985). Also, neurons in the cerebellar vermis display eye-position-dependent variations in saccade-related bursts (McElligott & Gochin 1986), and the direction and size of saccades evoked by cerebellar stimulation are dependent upon initial eye position (McElligott & Keller 1984).

Coordinates of Premotor Signals

BRAINSTEM SIGNALS A large body of evidence based upon recording, microstimulation, anatomical, and lesion data (see Raphan & Cohen 1978, Fuchs et al 1985 for reviews) indicates that signals responsible for the horizontal component of saccadic eye movements are generated in the

PPRF. Similarly, neurons in two regions of the rostral mesencephalic reticular formation (the INC and the riMLF) are involved in coordinating the vertical recti and oblique muscles during vertical saccades (Buttner et al 1977a,b, Buttner-Ennever 1977, Buttner-Ennever & Buttner 1978, King & Fuchs 1979, King et al 1981).

Not only must separate horizontal and vertical saccadic signals be formed, these signals must be coordinated during oblique saccades. Oblique saccades usually have relatively straight (rather than curved) trajectories (Figure IA) because the onsets of the horizontal and vertical pulses are synchronized and the durations of the horizontal and vertical displacements are approximately equal (Guitton & Mandl 1980, Evinger et al 1981, King et al 1986). When the amplitudes of the horizontal and vertical components of an oblique saccade are unequal, the duration of the smaller component is greater than the duration of a pure horizontal or vertical saccade of the same amplitude (Figure 1 B). The increased duration of the minor component of oblique saccades is neurally mediated; abducens neurons (King et al 1986) and midbrain MLBs (King & Fuchs 1979) display a decrease in average firing rate and an increase in burst duration under these conditions. This implies that signals controlling the velocity of the horizontal and vertical components of oblique saccades are based upon the amplitude of the total movement.

A direct extension of Robinson's model assuming independent pulse/step controllers for the horizontal and vertical components of saccades cannot account for the fact that visually guided oblique saccades are usually straight rather than curved. Even if the onsets of the pulses gen-

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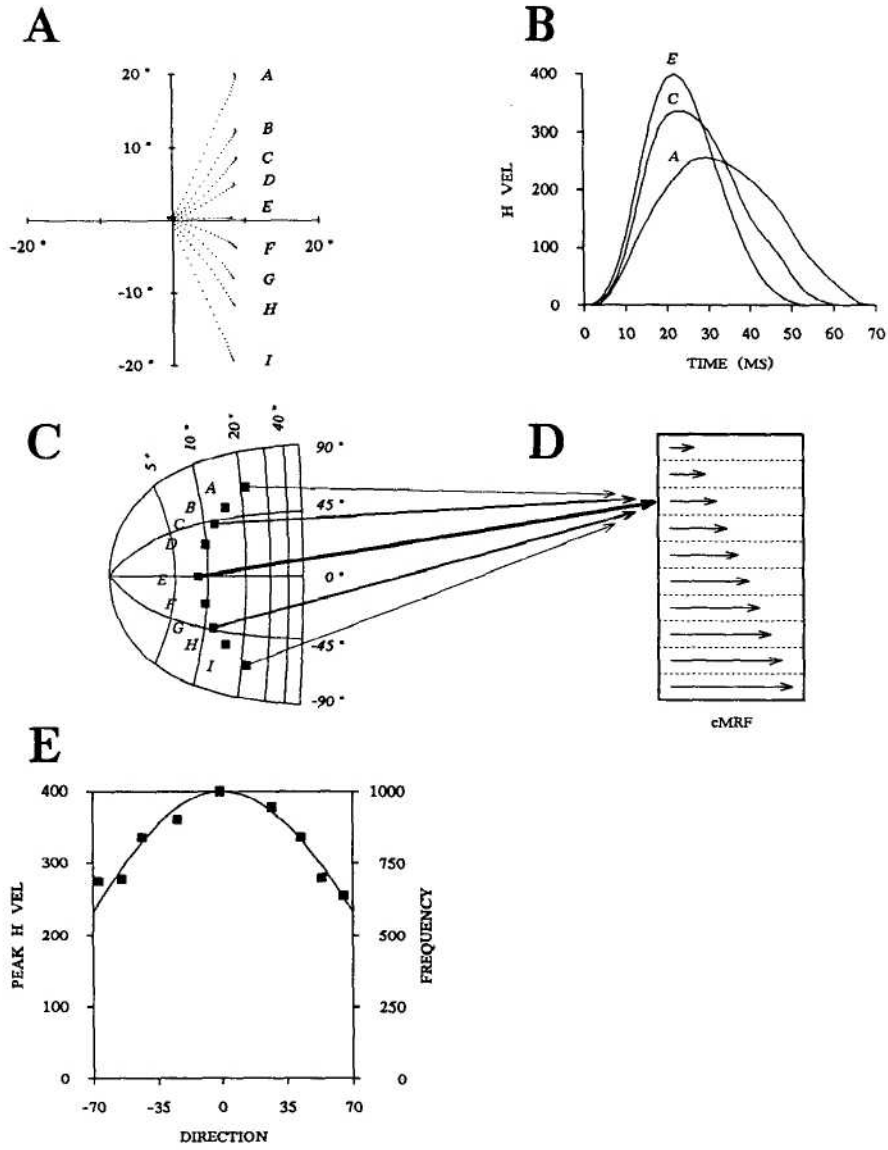
Figure 1 Examples of nine saccades all having 8° rightward horizontal components but differing in the magnitude and/or direction of the vertical component. B: The velocity profiles associated with three of the saccades illustrated in A. Note the reduction in peak velocity and the increase in duration of the horizontal component of movements having larger vertical components. C: The locations of collicular neurons discharging maximally before each of the nine saccades illustrated in A. D: It is hypothesized that by appropriate synaptic weighting, some tectorecipient neurons in the central mesencephalic reticular formation (cMRF) would display a frequency of firing proportional to the velocity of the horizontal (or vertical) component of oblique saccades. Neurons discharging maximally before saccades with small vertical components (e.g. E) provide large excitatory drive, whereas those discharging maximally before saccades with larger vertical components would provide proportionally smaller excitatory drive. E: A plot *(filled squares)* of the peak velocity of the horizontal component of the nine saccades shown in A as a function of the direction of the saccade (0 = straight right). The firing rate *(solid line)* of a hypothetical neuron receiving inputs from all collicular neurons discharging maximally before saccades with an 8° horizontal component is shown. The strength of synaptic input is weighted according to the cosine of the angle of the movement. (From Sparks 1990).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erated by the horizontal and vertical controllers were synchronized via pause cells, the offsets could be asynchronous and result in curved saccades. To prevent this, the velocity of the minor component must be reduced by lowering the discharge frequency of its MLBs so that the durations of the two components are also equal (King et al 1986). Several workers (Tweed & Vilis 1985, van Gisbergen et al 1985, van Gisbergen 1988, Grossman & Robinson 1988, Scudder 1988) have proposed modifications of Robinson's model to account for the coordination of the horizontal and vertical components of oblique saccades. These are considered in more detail below.

REQUIRED TRANSFORMATIONS OF COLLICULAR SIGNALS The discharge of collicular neurons is related to the direction and amplitude of eye displacement, and is not uniquely related to the amplitude of either the horizontal or the vertical component of the movement. Thus, separate signals of horizontal and vertical motor error must be extracted from the anatomical map of motor error found in the SC. How and where are the separate signals needed by the burst generators formed? Several possibilities exist. Three are discussed in more detail below.

First, each collicular neuron could synapse (with appropriate weighting) directly upon the horizontal and vertical MLBs in the pons and midbrain. In this case, an intermediate stage in which signals of horizontal and vertical motor error are explicitly extracted would not exist. Since MLBs in the pons are not activated with monosynaptic latencies by collicular stimulation (Raybourn & Keller 1977), the available evidence does not support this possibility.

A second possibility is that separate signals of the horizontal and vertical motor error are present in populations of cells intermediate between the SC and horizontal and vertical MLBs. Most recent revisions of Robinson's model (Keller 1981, Tweed & Vilis 1985, Scudder 1988) assume that a signal of horizontal motor error is formed by selective convergence of axons of collicular neurons upon LLBNs, the cells assumed to provide excitatory drive to horizontal MLBs. Direct projections from the SC to the contralateral PPRF have been demonstrated anatomically (Harting 1977) and electrophysiologically (Raybourn & Keller 1977, Keller 1979). Raybourn & Keller (1977) reported that all LLBNs in their sample received short-latency excitatory input from the SC and that the latency of the response was in the monosynaptic range for approximately one third of the sampled neurons. Moreover, each LLBN was driven by stimulating electrodes placed in both rostral and caudal areas of the SC, evidence for convergence of signals thus originating from widespread regions of the SC.

Although anatomical evidence is consistent with the hypothesis that

collicular signals converge upon LLBNs, there is no anatomical evidence that the LLBNs provide an input to MLBs. Moreover, it is not clear that LLBNs provide a signal of horizontal motor error. LLBNs display a relatively long period of irregular low-frequency activity as a "prelude" to an intense burst of firing that precedes saccade onset by 10-11 msec (Luschei & Fuchs 1972, Keller 1974, Henn & Cohen 1979, Hepp & Henn 1983). Because most investigators have not plotted the movement fields of LLBNs, there is no unequivocal evidence that the discharge of LLBNs is related only to the horizontal component of oblique saccades. Van Gisbergen and colleagues (1981) reported that information about saccade size, saccade duration, or saccade velocity is not coded in any obvious way by the discharge patterns of individual LLBNs. Their sample of LLBNs often fired well before saccade onset and long after saccade offset. During the saccade, the burst rate varied little with saccade size or eye velocity. They concluded that the signal recorded from single LLBNs would need to be combined with signals from other sources to provide an input that was the basis for the strict relationship between burst parameters and eye velocity observed in MLBs. Clearly, additional studies are needed, designed explicitly to examine the functional properties of LLBNs, as these neurons play a prominent role in current models of the saccadic system.

Recent experiments suggest that neurons in the central midbrain reticular formation (cMRF) could be involved in extracting information about the horizontal component of oblique saccades. Neurons in the intermediate and deeper layers of the SC project to the cMRF (Cohen et al 1986) and, in turn, neurons in the cMRF project to regions of the pontine reticular formation involved in the control of horizontal saccades (Cohen & Buttner-Ennever 1984). Cells in the cMRF burst before contralateral saccades having horizontal components, and microstimulation in this area produces only horizontal saccades (Waitzman 1982). The amplitude of the stimulation-induced horizontal movement depends upon the depth of the stimulating electrode. Small amplitude movements are evoked by stimulation dorsally, and progressively larger movements are evoked as the electrode is moved ventrally (Waitzman 1982, Cohen et al 1985).

Based upon the findings of Cohen and colleagues (Cohen et al 1985), Sparks (1986) proposed a mechanism for extracting a signal of horizontal motor error from the anatomically coded signal observed in the SC. He suggested that the axons of all the collicular neurons residing along an isoDH curve (same change in horizontal position) converged upon neurons at a specific depth in the cMRF (see Figure 1D). Neurons at that depth would discharge maximally before any saccade having a particular horizontal component, regardless of the vertical component of the movement. Cohen & Buttner-Ennever (1984) observed differential retrograde labeling in rostral and caudal areas of the SC following injection of anatomical

tracer material into dorsal and ventral cMRF, but their data cannot be used to support this hypothesis because they looked for patterns of label in the SC related to the overall amplitude of the saccade, not the amplitude of the horizontal component. Other tectorecipient neurons are hypothesized to extract information about the vertical component of oblique saccades.

Van Gisbergen and colleagues (1985) suggested a third possibility for how signals of horizontal and vertical motor error are extracted from the collicular signals. They argue that a signal of the velocity of the combined movement (vectorial velocity) is computed first and later decomposed into separate velocity signals for the horizontal and vertical components (component velocity). These authors hypothesize that the vectorial pulse generator is composed of an array of LLBNs, each of which codes eye velocity in a certain direction. In Scudder's model of the saccadic system (Scudder 1988), LLBNs act as integrators of topographically coded inputs from the SC. This model predicts that the rate of increase of activity of LLBNs will be proportional to the size of the component in the on-direction of the cell. Unfortunately, available data do not permit an assessment of these hypotheses, but earlier studies failed to identify LLBNs displaying a relationship between the firing rates and saccadic velocity (van Gisbergen et al 1981).

The activity of some MLBs could be related to the vectorial velocity of oblique saccades. There are several reports (Luschei & Fuchs 1972, Henn & Cohen 1976, Keller 1977, Strassman et al 1986a,b, Nelson et al 1988) that some MLBs discharge maximally before oblique saccades.

The possibility that the activity of saccade-related burst neurons in the SC encodes vectorial velocity must also be considered. The SC would be a good candidate to coordinate oblique saccades, since information about both the horizontal and the vertical components is present and there appears to be some control of vectorial velocity as well. The velocity of saccades is reduced after collicular ablations (Wurtz & Goldberg 1972b, Schiller et al 1980) or reversible deactivation of collicular neurons (Hikosaka & Wurtz 1985, Lee et al 1988). Preliminary data (see above) are consistent with the hypothesis that the frequency of firing of neurons in the active population is related to the velocity of the saccade along a particular trajectory (vectorial velocity). What is needed is a mechanism by which component (horizontal or vertical) velocity can be scaled according to saccadic direction. A possible mechanism is illustrated in Figure 1C,D. To extend the earlier suggestion of Sparks (1986), neurons encoding the velocity of the horizontal (or vertical) component of oblique saccades by their frequency of firing could be formed by allowing these cells to receive excitatory synaptic inputs from all members of an isoAH curve,

but scaling the strength of the excitation by the location of the cell along the curve. For example, those members of the 8° isoAH curve that are located rostrally and near the midline (cells associated with 8° horizontal movements having small vertical components) would provide greater excitatory drive than members located caudally (i.e. cells associated with 8° horizontal movements and large vertical components).

Kinematic Constraints

BRAINSTEM SIGNALS The implications of the rules of rotational kinematics were considered by Tweed & Vilis (1987) for models of the VOR and saccade generation. Current models of the VOR require a neural integrator to transform signals corresponding to eye velocity into signals of eye position. Models of the saccadic system use the same integrator to construct an eye position signal from the output of saccade-related burst neurons in the pons and midbrain. Tweed & Vilis (1987) argued that one-dimensional and two-dimensional saccadic models that subtract current eye position from desired eye position to generate signals cannot be easily extended to handle three-dimensional rotations. They proposed an alternative model implemented with quaternions in which angular velocity was multiplied by position feedback before integration. In their computer simulations, however, both the subtractive and multiplicative models performed well for saccades in the oculomotor range. Moreover, the major failure of the subtractive model discussed in their paper occurred when gaze shifts of 180° were attempted, a highly unusual circumstance.

REQUIRED TRANSFORMATIONS OF COLLICULAR SIGNALS Robinson's model (1975b) of the saccadic system assumes that saccade targets are specified in terms of the final position of the eye in the orbit but does not indicate how these signals are generated. Revisions of Robinson's model, designed to accept collicular commands for changes in eye position, do not address the question of how Donders' law is implemented. Recently, Tweed & Vilis (1990) outlined a model of the saccadic system based upon a revival of Westheimer's (1957) use of quaternions for describing the rotations of the eye. In this model, a signal of the desired gaze vector is coded **in** head coordinates upstream from the SC. The gaze vector signal is passed through a Listing's law operator and the output is combined with a signal of current eye position to determine the site of collicular activation. The site of activation in the SC controls the rotation of the eye in three dimensions rather than, as usually assumed, in two. Listing's law operator, a constraint on craniotopic eye position, is placed upstream from the SC so that the operation occurs at a level at which saccades are still coded in craniotopic coordinates. Clearly, this model is shaped by the

concern of devising a scheme by which Listing's law can be obeyed but, as noted above, it is possible that Listing's law could be approximated by simpler operations in which the torsional component is not computed explicitly.

This issue needs to be resolved. The computations to be performed by the saccadic system are quite different, depending upon which model is correct. According to a simple scheme in which commands are organized in horizontal and vertical coordinates without an explicit computation of the torsional component of a saccade, the topographic map of the SC specifies the amplitude and direction of the saccade. The collicular signal is decomposed into horizontal and vertical commands and routed to the appropriate prenuclear bursters. Just as the SC produces a "standard" command for a saccade of a given direction and amplitude, the prenuclear bursters would have the same output for a given saccade, regardless of initial eye position. Information about eye position would be needed only for the motoneuron recruitment mechanism, which is used by all conjugate subsystems.

According to the Tweed & Vilis model (1990), or other models in which there is an explicit torsion signal, identical saccades must result in the activation of different subsets of prenuclear bursters, depending on initial eye position. For example, the behavior of bursters during a 10° upward saccade started with the eyes 20° to the left must differ from their behavior when the 10° upward saccade begins with the eyes 20° to the right. Otherwise, the post-saccadic eye position would display torsion that would violate Listing's law. A Listing's law transformation predicts that the output of the prenuclear burst neurons should vary for identical saccades as the eyes assume different tertiary positions. Although no obvious differences in burst output have been noted (Keller 1974, van Gisbergen et al 1981), this has not been systematically studied. Furthermore, the magnitude of the expected differences is unknown and would be difficult to compute.

In order to conform to Listing's law, the transformation from the SC coding scheme to that of the prenuclear bursters would be much more complex than the simple two-dimensional scheme which ignored Listing's law. Consider two alternatives. The Listing's law operation might occur downstream of the SC. This would require that the output of the SC be re-routed to the prenuclear bursters, depending upon absolute eye position at the time of the onset of the saccade. Alternatively, the Listing's law operation might be upstream of the SC. This view, favored by Tweed & Vilis (1990) suggests that the SC encodes torsion as well as the horizontal and vertical components of the saccade. Further investigation might uncover a third (torsion) dimension of the collicular map. The Tweed &

Vilis model predicts that microstimulation of a single site in the SC will generate the same three-dimensional eye rotation regardless of the original position of the eye. Thus, eye rotations evoked by collicular stimulation should usually end up in final positions violating Donders' law. If saccades are specified by using only horizontal and vertical coordinates, however, the ocular torsion observed after stimulation-induced changes in eye position should fall within normal limits, regardless of the original eye position.

A more fundamental concern is whether Listing's law indeed imposes constraints on the signal transformations involving the SC. As we have noted, Listing's law is obeyed only approximately (Ferman et al 1987a,b). Furthermore, the analysis of extraocular muscle interaction by Robinson (1975a) implies that Listing's law may be approximated if the commands to the extraocular muscles were simply organized in horizontal and vertical coordinates. Finally, the recent report by Miller (1989) emphasizes that the geometry of the eyeball and muscles is complex and can have profound and unexpected effects on the signal transformations. Thus, it may be premature to speculate on the neuronal Listing's law operator before it is established, experimentally, that one is needed.

SUMMARY AND CONCLUSIONS

Chronic unit recording experiments conducted over the past two decades have identified many functional classes of. neurons with saccade-related activity that reside in a host of brainstem nuclei. Older models of the saccadic system were based upon the properties of only a few of these functional types of neurons. They described the putative flow of signals through the brainstem circuitry and specified some, but not all, of the signal transformations to be performed. How the necessary computations were performed by neurons was not always explicit.

Recent experiments investigating the neural control of saccadic eye movements and modifications of the original models are designed to fill in the details of the broad sketch of saccadic circuitry originally available. This review suggests one strategy for proceeding with this effort. Saccadic command signals observed in the SC require transformations to interface with the burst generators and motoneuron pools innervating the extraocular muscles. Specifying the signal transformations required for this interface should facilitate the design of experiments directed toward an understanding of the functional properties of cells located in nuclei intervening between the SC and the pulse/step circuitry, subsets of neurons that often have no role in models of the saccadic system. **In** this review, we hypothesize that neurons residing in various tectorecipient brainstem

nuclei participate in one or more of the required signal transformations. The pathway from SC to cMRF and PPRF may be involved in the extraction of information about the amplitude and/or velocity of the horizontal component of oblique saccades. The pathway from SC to NRTP and cerebellar vermis may act selectively to generate signals compensating for the presaccadic orbital position. Finally, the activity of LLBNs and MLBs discharging maximally before oblique saccades may form the basis of computations required to match component velocity with overall saccade direction and amplitude. Although the data supporting these speculations are meager at present, such conjectures do form the basis of working hypotheses that can be tested experimentally.

We also considered the implications of kinematic constraints, especially Donders' and Listing's laws, for future investigations. Tweed & Vilis (1987, 1990) proposed models specifically designed to handle these constraints. In their models, eye position is represented on four oculomotor channels: three coding the vector components of eye position, and one carrying a signal inversely related to gaze eccentricity and torsion. Yet, other evidence suggest that simpler computations may suffice for the implementation of laws that are only approximately obeyed. Indeed, the question is whether the laws reflect "a special effort and programming by the nervous system, or are just an adventitious consequence of the mechanics of the peripheral oculomotor plant" (Ferman et al 1987b) when movements are specified in a two-dimensional coordinate system.

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