CHAPTER 4
COMPARING TARGET POSITION WITH PRESENT POSITION: NEURAL VECTORS

4.1. Reconciling Visually Reactive and Intentional Computations

In Chapter 1, we outlined several basic reasons why the positions of light on the retina need to be recoded as target positions in a head coordinate map. The calibration of intermodality movement signals, the attentional selection of a target position for an intentional movement, and the encoding of sequences of predictive saccades can all be carried out using a target position map. In Chapter 3, we described how retinotopically encoded light positions can be used to correct visually reactive saccades. In this chapter, we begin to show how these two types of processing requirements, which on the surface may seem to be contradictory, can be reconciled. Indeed, we will argue that both types of processing are essential. The visually reactive system provides a way to learn accurate saccades. The attentional, intentional, and predictive systems can then make use of the movement parameters that were learned in the visually reactive mode. We will argue in Chapter 11 that the learned parameters of the visually reactive system provide a foundation without which the attentional, intentional, and predictive systems could never learn to operate well. This claim does not imply that accurate intentional movements cannot be made in an adult organism after large parts of the visually reactive system are extirpated. The suggested nature of the dependence between these two systems is more subtle than that, and is based upon an analysis of neuronal development rather than upon performance characteristics of an adult network.

In this chapter, we will focus on a core problem in the construction of the intentional system: the design of the *Head-Muscle Interface*, or HMI (Section 1.6). The HMI mediates between target position computations and retinotopic computations. It does so by comparing target positions with present positions to compute vector differences in motor coordinates. By describing first our solution of this problem, our solutions of a number of other problems become easier to motivate, such as how to design an invariant target position map, how to transform vector differences from motor coordinates into retinotopic coordinates, and how to attentionally modulate which movement commands will finally be expressed in observable saccades. These problems become easier to understand because we can then see how to constrain their solutions to be compatible with input-output constraints that are imposed by HMI design.

4.2. Experimental Evidence for Vector Inputs to the Superior Colliculus

Before describing our formal solution to this problem, we review some

relevant experimental data. A number of experiments on saccadic performance have suggested that vector differences are somehow computed by the saccadic control system (Hallett and Lightstone, 1976; Mays and Sparks, 1980; Schiller and Sandell, 1983; Zee *et al.*, 1976). All of these experiments test how the saccadic system responds to two or more inputs that are delivered to the saccadic system in rapid succession. The experiments of Mays and Sparks (1980) are particularly notable.

Mays and Sparks (1980) have ingeniously tested whether the nervous system computes transformations analogous to vector operations by recording from quasi-visual (QV) cells of the superior colliculus (SC). The SC is a structure that receives inputs from the retina and the visual cortex, among other neural regions. The SC plays an important role in generating saccades, but this role is not easy to characterize despite the facts that the SC is a phylogenetically old structure that receives inputs directly from the retina.

The Mays and Sparks (1980) experiments illustrate the subtlety of SC architecture. The SC is divided into a succession of functionally distinct stages. The superficial layer reacts rather directly to retinotopically coded information. The final stages react to information that is attentionally modulated and encoded in motor coordinates (Huerta and Harting, 1984; Schiller and Stryker, 1972). The QV cells from which Mays and Sparks (1980) recorded lie between these processing extremes.

Mays and Sparks isolated QV cells that fired before the eye saccaded from position O to a light flash at position C (Figure 4.1). They then studied how these cells responded to a light at position B followed by direct electrical stimulation of the superior colliculus. The direct electrical stimulation caused the eye to move from O to A before it could saccade to the light at position B. Both positions A and B fall within the right hemifield of the retina while the eye fixates position O. By contrast, a light to position C excited the left retinal hemifield while the eye fixated position O. Consequently, if the QV cell that fired before the motion O→C encoded retinal position, then this cell should not fire before the eye saccaded from A to B. Note, however, that the vectors $\vec{OC}$ and $\vec{AB}$ encode the same direction and length of motion. Thus if the QV cell encoded vector differences, then it should fire both before the eye moved from O to C and before the eye moved from A to B. This is, in fact, what Mays and Sparks (190) observed. Schiller and Sandell (1983) have replicated these findings, in addition to showing that the frontal eye fields can also compute a similar compensatory "vector". These data suggest that vector differences between a target position map and an eye position map influence the firing of QV cells in the superior colliculus, as well as a subset of cells in the frontal eye fields. In an attempt to localize the compensatory "vector" mechanism, Schiller and Sandell (1983) performed a bilateral ablation of the frontal eye field or superior colliculus. Monkeys could still accurately compensate for ocular perturbations caused by electrical stimulation. This suggests that neither the frontal eye field nor the superior colliculus alone is uniquely responsible for corrective saccade computa-

tion. In contrast, even normal monkeys could not accurately compensate for ocular perturbations caused by stimulation of the motoneurons within the abducens nucleus.

4.3. Adaptive Inhibitory Efference Copy in Motor Control

Mays and Sparks (1980) described this vector operation as one that compares an expected position with an actual position of the eye. We use the terms target position and head coordinate map instead of expected position. We prefer this terminology because there are several senses in which expectations are computed within the nervous system. The present computation differs both qualitatively and quantitatively from the types of sensory expectation that help to complete ambiguous visual forms, as in perceptual switches between ambiguous figures (Grossberg, 1980); to regulate motivational decisions using templates formed by patterns of conditioned reinforcer signals (Grossberg, 1982b); to focus attention on motivationally valued cues (Grossberg, 1982b); or to control phonemic restoration, word superiority effects, and priming of lexical decisions during processing of speech or printed text (Grossberg, 1985c; Grossberg and Stone, 1985a). In all these cases, input patterns that match the expected pattern are selectively amplified (Section 2.6). In the present instance, an eye position that matches the target position causes an attenuation, rather than an enhancement, of activity, due to the property that the difference of two equal vectors is zero. A suppressive effect of a target controlled motor template on feedback signals has also been reported in the electric eel (Bell, 1981), where an efference copy exists that is opposite in sign to the efferent input from ampullary electroreceptors. This efference copy is adaptive, as is the motor template that exists in our model network. Taken together, these results suggest that many sensory expectancies, when matched, cause selective pattern amplification, whereas many motor commands, when matched, cause selective pattern attenuation.

Another reason for refining the language used to describe "expected position" is to explain how the "expected position" is computed. To compare a target position that is computed in head coordinates with a present eye position that is computed in agonist-antagonist motor coordinates, it is necessary to first convert both types of information into the same coordinate system (Section 1.6). We show how a learning process converts target positions in head coordinates into target positions in motor coordinates. This transformation is designed so that differences between target position motor coordinates and present position motor coordinates can be used to generate the correct "vector difference" command in retinotopic coordinates (Section 1.11). All of these computations use dynamical neural network operations rather than algebraic vector calculus operations. We hereby explicate the vector metaphor and develop terminology that includes the distinctions necessary to do so. These nonlinear neural network computations provide an alternative to the linear tensor computations of Pellionisz and Llinás (1980, 1982).

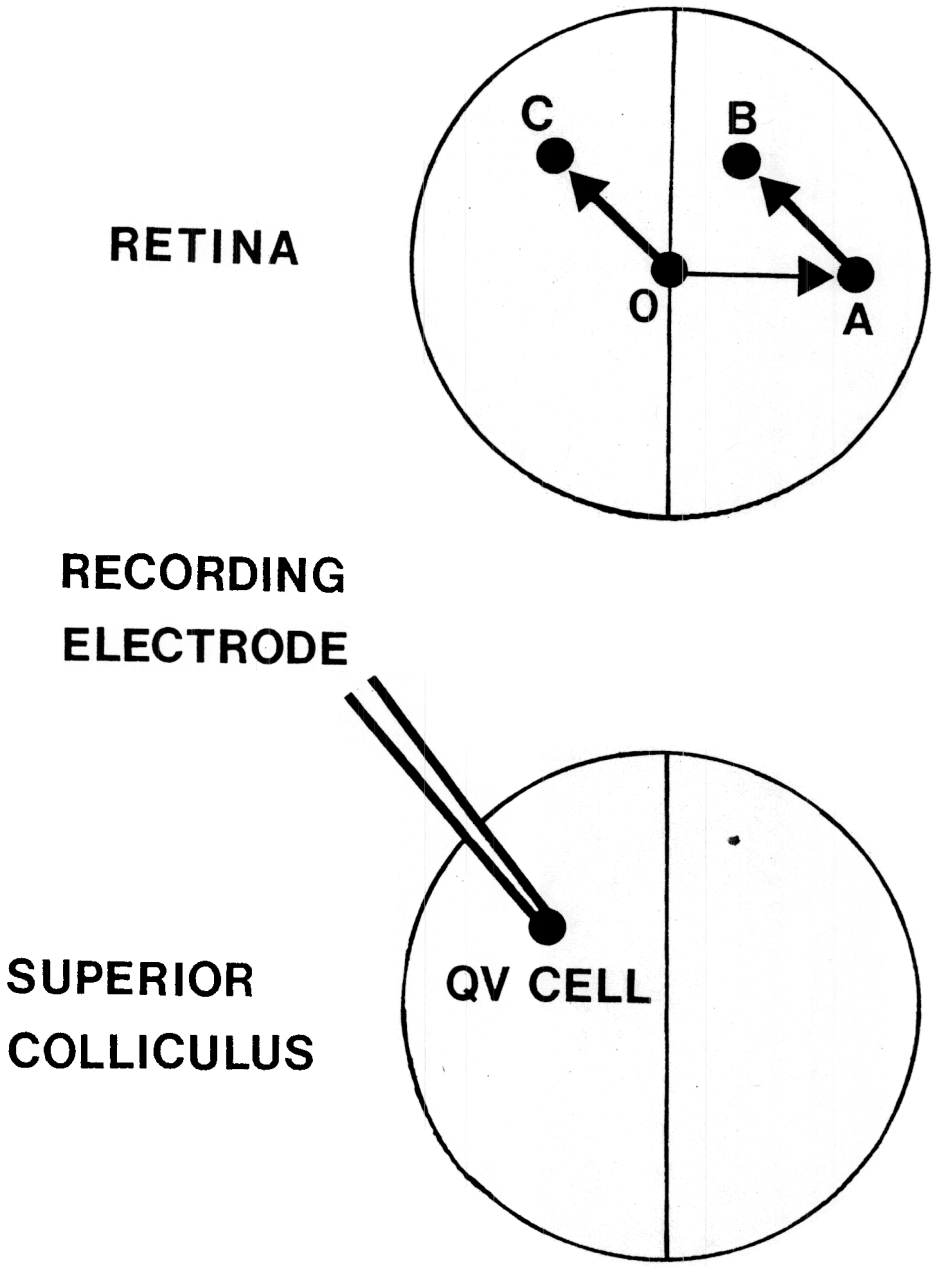

Figure 4.1. The Mays and Sparks (1980) paradigm. Motions from O to C and from A to B represent the same vector length and direction. A quasi-visual (QV) cell that fires before a $\overrightarrow{OC}$ saccade also fires before a $\overrightarrow{AB}$ saccade.

4.4. Multistage Elaboration of a Vector Map

Mays and Sparks (1981) interpreted their data in the manner depicted in Figure 4.2, which represents the vector model of Zee *et al.* (1976). In Figure 4.2, eye position and retinal position are first combined by a vector addition process before a vector subtraction process compensates for eye position. The vector subtraction process is delayed in time relative to the vector addition process. This property is needed because the vector addition that computes the target position of the light uses the position of the eye before the eye saccaded to the electrical stimulus. The vector subtraction that converts the target position of the light into a vector command uses the eye position after this saccade occurs. Then the vector command generates a second saccade to the correct target position of the light. It is not clear from Figure 4.2 why such a long delay should exist between the first and second registrations of eye position or how the nervous system computes a vector difference operation. Figure 4.3 describes a macrocircuit that suggests a resolution of the time delay problem. Variations on the stages in Figure 4.3 may occur in different species.

Stage 1 in Figure 4.3 is computed in retinal coordinates. A retinal light that excites a region of stage 1 transfers this excitation to stage 2, at which retinally induced signals and initial eye position signals are joined to compute a target position map in head coordinates. In monkeys, this target position map is computed outside the SC (Schiller and Koerner, 1971). We suggest that these computations take place in a circuit within the parietal cortex (Goldberg, 1980; Hyvärinen, 1982; Lynch, 1980; Motter and Mountcastle, 1981; Mountcastle, Anderson, and Motter, 1981; Sakata, Shibutani, and Kawano, 1980) and/or the internal medullary lamina of the thalamus (Schlag and Schlag-Rey, 1981; Schlag, Schlag-Rey, Peck, and Joseph, 1980; Schlag-Rey and Schlag, 1983). A target position map has, however, been found alongside a retintotopic map in the SC of cats (Guitton, Crommelinck, and Roucoux, 1980; Peck, Schlag-Rey, and Schlag, 1980). Thus the location of the target position map may vary from species to species. Behaviors due to unilateral ablation of the parietal cortex are consistent with the hypothesis that parietal cortex includes a target position map in egocentric coordinates. Such an ablation leads to hemifield neglect, or a lack of goal-oriented behaviors to one side (Motter and Mountcastle, 1981).

Stage 3 computes a vector map by combining signals from the target position map with signals that register present eye position. Stage 3 is the HMI. This vector map relays its signals to stage 4, which is interpreted to be a retinotopic map within the SC. These SC cells are identified with the quasi-visual (QV) cells in the intermediate layers of the SC (Mays and Sparks, 1980). Then the retinotopic map projects to stage 5, which is interpreted as the deep layers of the SC.

This anatomical interpretation of the stages that occur within the SC is compatible with the results of Schiller and Koerner (1971). These authors found retinally coded cells throughout the depth of the monkey SC. Although all of the cells in the model SC are "retinally coded", some

of them should be interpreted as retinotopically recoded vectors, as the data of Mays and Sparks suggest.

In this macrocircuit, competition between visually reactive, intermodality, and intentional sources of saccadic commands is assumed to occur within the target position maps of stage 2. The projection from stage 2 to stage 3 (the HMI) is adaptive, as we will show in Section 4.6. It transforms target positions from head coordinates into motor coordinates. The projection from stage 3 to stage 4 is also adaptive. It transforms vectors from motor coordinates into retinotopic coordinates. This adaptive process enables the HMI vectors to make use of visually reactive movement commands that can be corrected using second light visual error signals (Chapter 3). Competitive interactions within stage 4 are used to help choose the map locations that will engage in this learning process (Chapter 11). Thus competitive interactions are assumed to occur within at least two stages of the macrocircuit: stages 2 and 4. Both of these competitive stages play a role in choosing between different sources of saccadic commands. In the experiments of Mays and Sparks (1980), the electrode input and the HMI vector take precedence over the direct light-activated retinal signal.

The hypothesis that a direct visually reactive, retinotopically encoded pathway exists suggests that total ablation of all target position maps that contribute to saccadic commands may disinhibit the direct retinotopic pathway. The data of Schiller, Sandell, and Maunsell (1984) on express saccades in the monkey are consistent with this hypothesis. Such a test may be confounded by secondary effects if destruction of a target position map also alters the gating of cell responsiveness in the deeper layers of the SC (Hikosaka and Wurtz, 1983) in the manner described within the next section. Chapter 11 describes in greater detail the processing stages and the types of behavioral effects that lesions of these stages would be expected to have.

4.5. Attention Modulation in Parietal Cortex and Inhibitory Gating of SC Signals: The Delay in Vector Subtraction

Given that many, possibly conflicting, sources of saccadic commands exist in the several target position maps, a mechanism is needed to decide which of these commands will elicit a saccade. Attentional factors obviously influence this choice. We assume that attentional processing occurs at stage 2, which is interpreted to be a neocortical region, such as the posterior parietal cortex, or a subcortical region that is intimately connected with neocortex (Mountcastle, Anderson, and Motter, 1981; Wurtz, Goldberg, and Robinson, 1982).

We can now physically interpret the formal delay of vector subtraction in Figure 4.2 by noting that the neocortical attentional computation which is hypothesized to occur in stage 2 of Figure 4.3 takes time. Thus the eye position information that is used to compute target positions at stage 2 is earlier eye position information than is used to compute vectors at stage 3. Given that the attentional processing within stage 2 is relatively

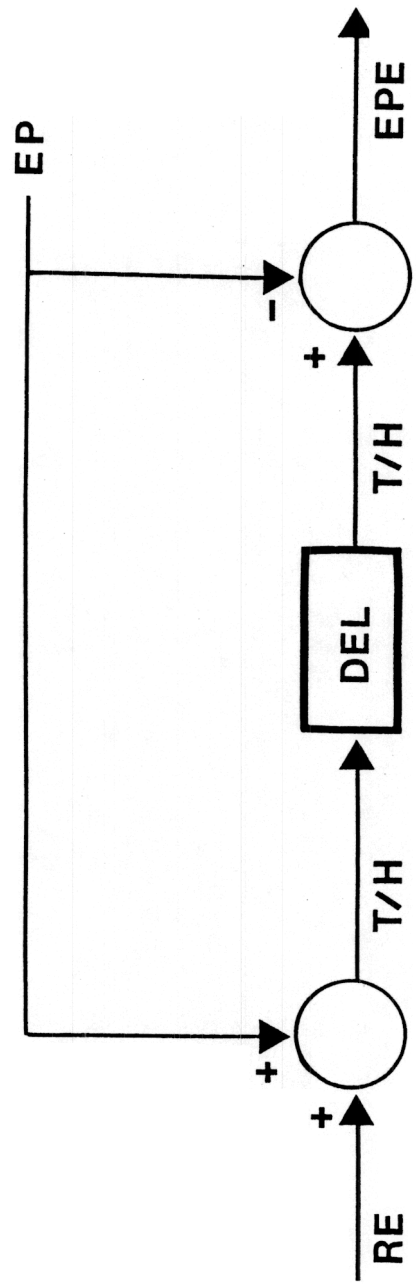

Figure 4.2. The Zee *et al.* (1976) vector model. See the text for a description. The abbreviations mean: RE = retinal error signal; EP = eye position signal; T/H = position of the target with respect to the head; DEL = delay; EPE = eye position error signal.

slow, we are confronted with a new design problem. The direct light-reactive retinotopic pathway between stages 1→4→5 does not necessarily pass through stage 2, or in any case could be expected to react quickly to retinal lights (Chapter 11). Somehow this light-reactive pathway must be prevented from automatically eliciting a saccade until the outcome of the attentional competition in stage 2 is completed. Otherwise visually reactive saccades would always preempt the occurrence of other types of saccades. The excitability of the direct light-reactive retinotopic pathway must therefore be modulated by attentional factors. Given our suggestion that the formal stages 4 and 5 have analogs within the SC, this argument implies that a surprisingly long delay should occur between activation of the superficial and deep layers of the SC. Such a delay between activation of the superficial and deep SC layers does, in fact, occur.

Schiller and Stryker (1972) and Sparks (1978) have shown that the delay between activation of the deep SC layers and saccade initiation is approximately 20 msec. The delay between onset of the visual stimulus and activation of the superficial SC layers is approximately 50–60 msec. For a typical saccade, it therefore takes approximately 120 msec. for activation of the superficial SC layers to reach the deep SC layers (Sparks and Mays, 1981). This is a surprisingly long time, but it provides a physical basis for the delay postulated in Figure 4.2.

The hypothesis that this delay is due to attentional interactions within cortical target position maps of stage 2 can be used to clarify the results of Mays and Sparks (1980). In particular, the target position of light B (Figure 4.1) can be computed at stage 2 shortly after the light occurs using pre-saccadic eye position signals. Ordinarily, the delay within stage 2 is used to select the particular target whose head coordinates will be used as a basis for a saccade. In the Mays and Sparks experiment, this delay enables the eye position signals that obtain after the electrode-induced saccade occurs to be used in computing the vector command at stage 3 that gives rise to the second saccade.

It remains to say how the direct visually reactive retinotopic pathway is prevented from activating the SC deep layers much sooner than 120 msec. after the SC superficial layers are activated. Hikosaka and Wurtz (1983) have shown that the excitability of the deeper layers of the SC is gated by inhibitory signals from the substantia nigra (SN). Disinhibition of the SN is one factor that enables the deep layers of the SC to generate saccadic commands. Inhibitory gating by the SN of the SC may be jointly controlled both by visually reactive and intentional mechanisms. Hikosaka and Wurtz (1983) have, for example, studied monkeys who were rewarded if they could delay their saccades to a light source until after the light was turned off. Although the light itself did cause some SN cells to be inhibited, the monkeys successfully performed the task. We will describe this type of gating mechanism in greater detail when we discuss attentional processing in Chapters 10 and 11.

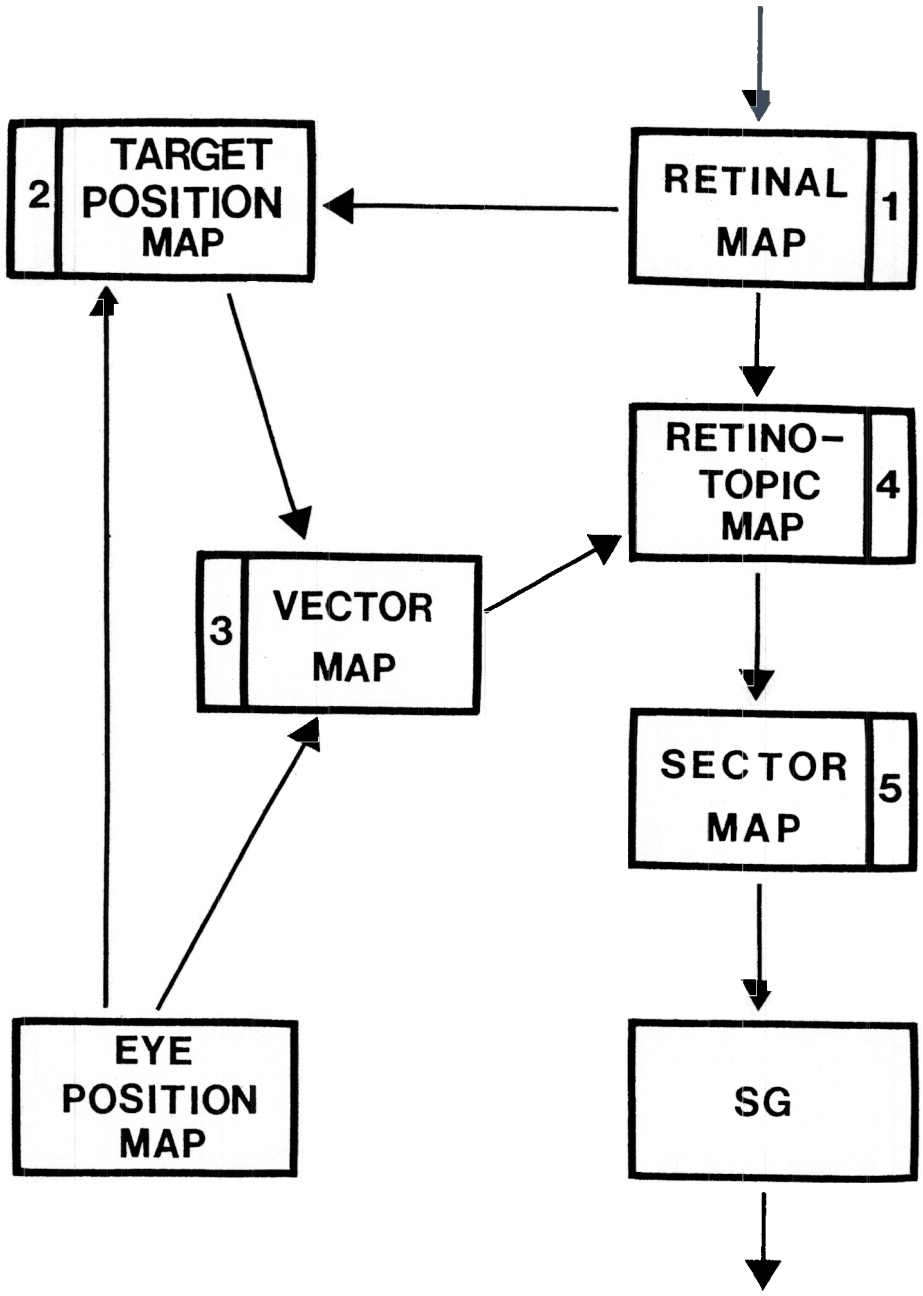

Figure 4.3. The functional stages and coordinate systems needed to neurally compute vector additions and subtractions. The delay between registering the target positions of competing sensory signals at stage 2 and reading the chosen target position into stage 3 is assumed to explain the formal delay in Figure 4.2.

4.6. Stages in the Adaptive Neural Computation of a Vector Difference

This section outlines how the computation of vector differences can be accomplished by self-calibrating neural mechanisms. We call a network capable of carrying out such a computation Vector Command Network, or VCN. A visually-reactive network is, by contrast, called a Retinotopic Command Network, or RCN. The following four problems are solved by a VCN.

A. *Head-to-Muscle Coordinate Transform*

To compare a target position computed in head coordinates with an eye position computed in agonist-antagonist motor coordinates, the target position is transformed into motor coordinates. The transforming mechanism that we use works even if the target position map, or TPM, possesses a complex internal structure. The head coordinates of different target positions can even be randomly distributed across the TPM without causing any calibration difficulties. This property may be important *in vivo* since, in the parietal lobes, functionally different types of cortical columns seem to be mixed together into a nontopographic cortical array (Lynch, 1980; Mountcastle, 1957, 1978).

The TPM needs to contain loci corresponding to a large number of different egocentric positions, whereas only six pairs of agonist-antagonist muscles move the two eyes (Section 1.5). In order to map head coordinates into muscle coordinates, each target position in the TPM sends sampling signals to motor representations of all the agonist-antagonist muscle positions at the head-muscle interface (HMI). A massive convergence of pathways must therefore occur from the TPM onto each muscle representation in the HMI.

One way to provide space for so many converging pathways is to represent each muscle within a large region of cells, to let each target position in the TPM project to a small subset of these cells, and to distribute the eye position signal in parallel across the entire region using many axon collaterals. In this type of anatomical realization, a potentially confusing mixture of cellular response profiles would present itself to a physiologist's electrodes. Signals due to both lights and eye positions would change rapidly from cell to cell due to target position inputs computed in head coordinates. These signals would intermix with signals due to eye positions that change slowly from cell to cell. The next paragraph explains why these influences would appear and disappear through time in a complex way, depending upon where and whether lights flash on and off and are differentially attended through time. These properties may be one reason why Motter and Mountcastle (1981, p.7) have written that "no consistent set of rules has evolved for naming the classes of neurons with different properties that can be identified in the parietal cortex in waking monkeys" and have classified as "unidentified cells" 31% of all the neurons that they studied. Perhaps, use of the new 24 channel microelectrode (Kuperstein and Eichenbaum, 1985) in parietal cortex will show how neurons can be part of collective neural group properties.

B. *Present Eye Position Signals: Corollary Discharges*

Outflow signals that move and help to hold the eye muscles in position are assumed to be the source of eye position signals (Section 1.9). This assumption is supported by data of Guthrie, Porter, and Sparks (1983), who showed that vector compensation still occured in the Mays and Sparks paradigm after all inflow signals from the eye muscles were surgically eliminated. Since these outflow signals are computed in agonist-antagonist coordinates, they can be directly relayed to the HMI. We identify the sources of these outflow signals with the tonic cells in the peripontine reticular formation (Keller, 1981) or in the vestibular nucleus (Fuchs and Kimm, 1975; Keller and Kamath, 1975; Miles, 1974).

C. *Simultaneous Calibration of the Head-to-Muscle Transform and of the Vector Difference between Target Position and Eye Position*

The HMI transforms target positions from head coordinates into motor coordinates in order to compute vector differences of target positions and present positions in motor coordinates. We will show how the HMI accomplishes both tasks using the same network equations.

First consider the mechanism which learns the transformation of target position from head coordinates into motor coordinates. Suppose that a cell population within the TPM encodes the target position by being activated before the saccade begins. Let the activity of this population be stored in STM until after the saccade is over. One might legitimately ask how any saccade whatsoever can be generated before the transformation within the HMI is learned. If the visually reactive Retinotopic Command Network (RCN) that we described in Chapter 3 did not exist, this concern would be a valid one. As it is, we consider a developmental stage before the HMI is calibrated when saccades can be unconditionally generated and corrected by visual error signals within the visually reactive RCN.

The active population within the TPM sends sampling signals over its conditionable pathways to the HMI. The HMI also receives corollary discharge signals that encode eye position as time goes on. These corollary discharge signals provide the eye position data that the conditionable pathways will learn. Not all eye positions are, however, the correct ones to learn. For example, before the saccade occurs, the corollary discharge signals encode initial eye position. The correct eye position to learn is not the initial eye position. Rather, it is the intended eye position.

This simple observation leads to our first major conclusion about HMI design. The conditionable pathways from the TPM target position to the HMI can learn only *after* a saccade is over. This property raises an important question. If the TPM target position is stored in STM throughout the saccade, then it can emit sampling signals along its conditionable pathways throughout the saccade. How can these sampling signals be prevented from encoding all the eye positions that are attained before and during the saccade? How can these sampling signals be caused to encode only the eye position that is attained after the saccade? We conclude that a gating signal exists which is capable of modulating the learning that occurs within the LTM traces of active conditionable pathways. Learning

is prevented except when this gating signal is on. The gating signal turns on only after a saccade is over. A similar gating signal was needed to prevent the learning of intermodality circular reactions (Section 1.3).

If these formal constraints can be achieved, then a target position stored within the TPM can learn the eye position that is attained by the subsequent saccade. How does the HMI know whether this final eye position is the "expected", or intended, eye position, namely the eye position which corresponds to the target position that is coded within the TPM? The answer is, quite simply, that the HMI does not possess this information. The HMI transformation succeeds in learning the expected eye position only because the visually reactive RCN can learn to generate correct saccades. Thus, as a result of the visual error correction that takes place within the visually reactive *retinotopic* system, the HMI can learn accurate transformations of target positions into *motor* coordinates.

From the above discussion, one can begin to understand how a vector difference can be computed by the HMI. Learning an eye position within the HMI occurs only after a saccade is over. Before a saccade begins, an active target position that is stored at the TPM can read-out the motor coordinates which it learned on previous occasions. These are the motor coordinates of the *target* position, not of the eye's *present* position before the saccade begins.

Thus before the saccade begins, information about target position and present position are simultaneously available within the HMI. There is no danger that the conditionable pathways will forget their learned target position by relearning the present eye position, because learning occurs only after a saccade is over, not before it begins. This fact guarantees the stability of memory before the saccade begins, but it does not yet explain how the HMI can compute a vector difference of target position and present position before a saccade begins. How this occurs can be better seen by considering the form of the learning process that occurs after a saccade is over.

A corollary discharge reads eye position into the HMI in the form of a pattern of excitatory inputs. At times when learning can occur, the conditionable pathways from the TPM continue to learn until their signals can match the corollary discharge signals. Then learning stops. Thus the conditionable pathways from the TPM to the HMI are *inhibitory* pathways (Figure 4.4). These inhibitory pathways carry the "adaptive inhibitory efference copy" of the HMI (Section 4.2). When the excitatory corollary discharges equal the inhibitory conditionable TPM→HMI signals, learning stops.

Before a saccade occurs, the active target position within the TPM reads its motor coordinates into the HMI as a pattern of inhibitory signals. The corollary discharge reads its present position into the HMI as a pattern of excitatory signals. The sum of these inhibitory and excitatory signal patterns represents a vector difference of target position and present position in motor coordinates.

D. *Visually-Mediated Gating of Vector Outputs*

Since the eyes always assume some position, corollary discharges are tonically received by the HMI. Since these eye position signals are excitatory, the HMI is always active, even if no target position is read into the HMI from the TPM. Indeed, when the TPM is inactive, no inhibitory signals whatsoever are sent to the HMI. Given that the HMI is always active when the TPM is inactive, we are led to ask: what prevents the HMI from persistently generating output motor commands, and thereby eliciting series of saccades, in the absence of either visually reactive or intentional saccadic commands? Clearly, something is missing from the HMI design.

To fill this gap, we assume that the output from the HMI is multiplicatively gated to zero except when some TPM population is actively reading-out a target position to the HMI. Thus, whereas the HMI is always activated by corollary discharges, it can only generate output signals at times when a vector difference between a target position and a present position is being computed at the HMI.

The TPM can only read-out target positions at times when visual or other input sources can activate target position populations within the TPM. Thus visual signals play two distinct roles in the VCN: a specific role and a nonspecific role. Their specific role is to provide information concerning which target positions to activate within the TPM. Their nonspecific role is to enable outputs from the HMI to be released. In Sections 4.9–4.11, we will explain some paradoxical data using the simple fact that visual inputs subserve both specific and nonspecific functions within the VCN.

The reader can now appreciate what a forbidding task it would be for a neurophysiologist to characterize the function of a network like the HMI without first having a good theory of how it works. Visual signals and positional signals get mixed together to compute target positions within the TPM, only to be recoded as positional signals in motor coordinates within the HMI. Thus positional signals are encoded in two different ways within the HMI by the inhibitory TPM inputs. Superimposed on these inhibitory positional signals are excitatory positional signals due to corollary discharges. Modulating this mixture of excitatory and inhibitory positional signals are two types of nonspecific gating signals. Visual inputs influence one of these gating signals, but not the other. Thus visual signals influence the network in two different ways and positional signals influence the network in three different ways. All of these interactions occur rapidly in time, albeit at different phases of the saccadic cycle. Superimposed upon these rapid signalling events are signals which can change slowly due to learning. Although the learning takes place in motor coordinates, it is modulated by a gating signal that is computed in head coordinates. The HMI is a neurophysiologist's nightmare, despite the intuitive simplicity of its functional design.

Another summary of HMI dynamics can be given which hints at its functional role as part of a larger processing scheme. Two different types

of gating signals are needed to regulate HMI dynamics. One type of gating signal is turned on before a saccade begins, and is used to regulate saccadic performance. The other type of gating signal is turned on after a saccade ends, and is used to regulate saccadic learning. When their properties are described in this way, these properties suggest the conclusion that these two types of gating signals are controlled by complementary movement and postural subsystems that help to regulate the saccadic rhythm (Chapters 9 and 11).

4.7. Modulators of Head-to-Muscle Coordinate Learning

The hypothesis that a gating signal regulates learning within the HMI leads to several experimental predictions. Such a gating signal is also called a Now Print signal in the neural modelling literature (Grossberg, 1982a). The cells which control this Now Print signal may either be transiently activated after a saccade ends (rebound cells) or may be turned on except during saccades (pause cells). Cells are known to exist that fire after a visually evoked saccade terminates, such as the refixation neurons found by Motter and Mountcastle (1981). Pause cells are also well-known saccade system components (Keller, 1981; Robinson, 1975; Schlag-Rey and Schlag, 1983). In order for pause cells to work well as sources of Now Print signals, the saccade must begin shortly after the HMI is activated by the TPM. Otherwise, the TPM target position would be able to sample the initial eye position, as well as the terminal eye position, for a significant amount of time. A critical parameter in ruling out a pause cell as a possible generator of a Now Print signal is the relative amount of time after the saccade ends as compared to before the saccade begins that the TPM command and the pause cell are simultaneously active. This ratio must be large in order for a pause cell to be an effective Now Print signal source.

A stronger test of whether a particular refixation neuron or pause cell is controlling a Now Print signal to the HMI can also be made. Suppose that such a cell could be excited on a series of saccadic trials by an electrode before each saccade occurs from a fixed target position. Then the head-to-muscle transform should gradually encode the initial eye position. Consequently, over successive learning trials, saccades in response to the fixed target position should become progressively smaller.

Gating signals of the type that are needed within the HMI have been reported in invertebrate sensory-motor learning circuits by Hawkins, Abrams, Carew, and Kandel (1983) and Walters and Bryne (1983). In these studies, the gating signal is mediated by a presynaptic Ca^{++} current that modulates the chemical transmitter system in the conditionable pathway. When a candidate HMI circuit is isolated, the possibility that a Ca^{++} current carries the Now Print signal can be tested using the same methods that have been developed in the invertebrate preparations.

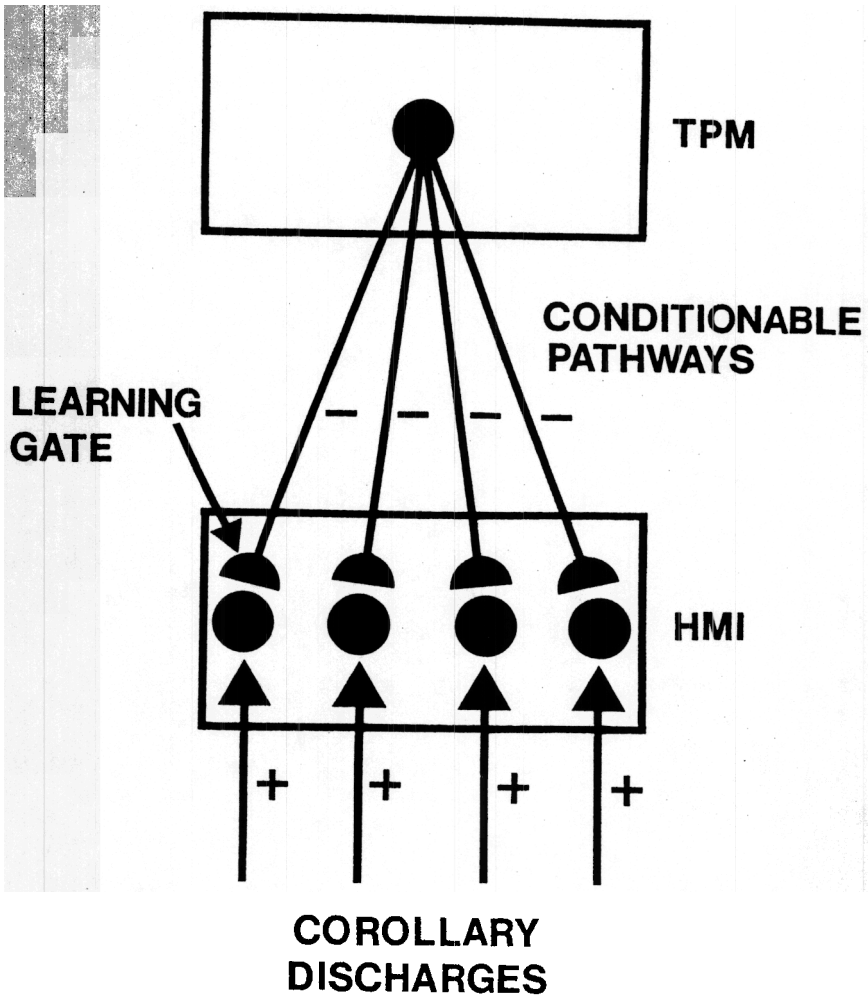

Figure 4.4. Recoding of a target position into muscle coordinates at a head-muscle interface, or HMI: The conditioned pathways learn an "adaptive inhibitory efference copy" from the corollary discharges that they are allowed to sample when the learning gate is active. When the efference copy equals the sampled corollary discharges, learning ceases.

4.8. Mathematical Design of the Head-Muscle Interface

The HMI circuit is a modified version of a motor expectancy learning circuit that was proposed by Grossberg (1972a, Figure 6). Denote by I_j the excitatory corollary discharge signal that represents the eye position corresponding to the jth muscle. Denote by $-S_i$ the ith inhibitory sampling signal that is released by the ith target position within the TPM. Let z_{ij} denote the LTM trace that exists at the synapse of the inhibitory pathway from the ith TPM target position to the jth HMI muscle representation. As in the design of the AG stage in Chapter 3, each LTM trace is assumed to control the rate of transmitter production in its synapse. Each LTM trace z_{ij} is assumed to possess the following properties (Grossberg, 1964, 1968, 1982a):

1. Trace z_{ij} computes a time-average of the product of the ith sampling signal S_i with the jth potential x_j of the HMI whenever x_j is suprathreshold ($x_j > 0$). Otherwise, no learning occurs.

2. Trace z_{ij} multiplicatively gates the signal S_i before it can influence x_j. The net inhibitory signal that influences x_j due to S_i is thus $-S_i z_{ij}$.

3. Potential x_j reacts additively to the sum of all conditionable inhibitory signals $-S_i z_{ij}$ from the TPM and the jth excitatory corollary discharge I_j at times when it is sensitized by a gating signal from the TPM.

4. The learning rate is gated to zero by a presynaptic gating, or Now Print, signal P that is switched on after a saccade terminates.

Hypotheses (1)–(4) are instantiated by the following differential equations for the time rates of change $\frac{d}{dt}x_j$ and $\frac{d}{dt}z_{ij}$ of each potential x_j and LTM trace z_{ij}, respectively:

$$\frac{d}{dt}x_j = -Ax_j + G(\sum_{i=1}^{n} S_i)(-\sum_{i=1}^{n} S_i z_{ij} + I_j) \tag{4.1}$$

and

$$\frac{d}{dt}z_{ij} = P\{-Bz_{ij} + S_i[x_j]^+\}, \tag{4.2}$$

where

$$[x_j]^+ = \begin{cases} x_j & \text{if } x_j > 0 \\ 0 & \text{if } x_j < 0 \end{cases} \tag{4.3}$$

and the gating function $G(\sum_{i=1}^{n} S_i)$ is an increasing function of $\sum_{i=1}^{n} S_i$ that vanishes when all $S_i = 0$. The suprathreshold activity pattern

$$V = ([x_1]^+, [x_2]^+, [x_3]^+, [x_4]^+, [x_5]^+, [x_6]^+ \tag{4.4}$$

represents the instantaneous vector difference between target position and eye position in muscle coordinates for a single eye. We now summarize

how equations (4.1)–(4.4) carry out the operations that were outlined in Section 4.6.

Suppose that, due to attentional processing within the TPM, at most one target position sampling signal $S_i > 0$ at any time. Without loss of generality, we can suppose that the gating function

$$G(\sum_{i=1}^{n} S_i) = \begin{cases} 1 & \text{if } \sum_{i=1}^{n} S_i > 0 \\ 0 & \text{if } \sum_{i=1}^{n} S_i = 0. \end{cases} \tag{4.5}$$

At times when no TPM target position is active, equation (4.1) reduces to the equation

$$\frac{d}{dt}x_j = -Ax_j, \tag{4.6}$$

which implies that the potential x_j rapidly decays to its passive equilibrium value, zero. In particular, no sustained output signals can be generated from the HMI at these times.

By contrast, consider times when some target position, say the ith one, is active within the TPM. At times when $S_i > 0$, equation (4.1) reduces to the equation

$$\frac{d}{dt}x_j = -Ax_j - S_i z_{ij} + I_j. \tag{4.7}$$

The LTM trace is assumed to change slowly relative to the fluctuation rate of the STM trace x_j. Hence we can assume that x_j is always in an approximate equilibrium relative to the slow time scale of z_{ij}. At equilibrium, $\frac{d}{dt}x_j = 0$. Then (4.7) implies that x_j approximately satisfies the equation

$$x_j = \frac{1}{A}(I_j - S_i z_{ij}). \tag{4.8}$$

The forgetting rate B of z_{ij} in (4.2) is also assumed to be slow relative to the learning rate $S_i[x_j]^+$. Consequently, the rate of change of z_{ij} approximately satisfies the equation

$$\frac{d}{dt}z_{ij} = PS_i[x_j]^+. \tag{4.9}$$

Equations (4.8) and (4.9) together imply that

$$\frac{d}{dt}z_{ij} = \frac{PS_i}{A}[I_j - S_i z_{ij}]^+. \tag{4.10}$$

By equation (4.10), the LTM trace changes only at times when the gating signal P, the sampling signal S_i, and the position difference term $[I_j - S_i z_{ij}]^+$ are *all* positive. Moreover, z_{ij} can increase due only to these factors. All decreases of z_{ij} are due to the very slow forgetting term

$-BPz_{ij}$ in (4.2), which can be completely ignored on the time scale of a learning trial.

Suppose that, at the onset of learning, z_{ij} is small, possibly even zero. Also suppose that, when S_i is activated across learning trials, S_i maintains a temporally stable value due to the competitive feedback interactions that store signals in STM within the TPM (Section 2.6). Finally suppose that the gating signal P becomes positive only after a saccade is over. At such times, I_j encodes the corollary discharge (present eye position) corresponding to the jth extraocular muscle, $j = 1, 2, \ldots, 6$. Due to learning within the visually reactive RCN (Chapter 3), at times when $S_i > 0$ the present eye position $(I_1, I_2, \ldots, I_6)$ gradually converges to the eye position $(I_{i1}, I_{i2}, \ldots, I_{i6})$ corresponding to the target position that activated S_i.

By (4.10), z_{ij} changes only when $S_i > 0$ and $P > 0$. When $S_i > 0$ and $P > 0$, (4.10) implies that $S_i z_{ij}$ approaches I_j as learning proceeds. Since each I_j converges to I_{ij} on learning trials when $S_i > 0$, $S_i z_{ij}$ converges to I_{ij} on these trials. The slow forgetting rate B prevents $S_i z_{ij}$ from getting stuck at I_j values that may occur before I_j converges to I_{ij}.

This argument shows that the gated signal $S_i z_{ij}$ approaches I_{ij} for *all* $j = 1, 2, \ldots, 6$. Thus activating the target position corresponding to S_i reads-out the signal pattern $(S_i z_{i1}, S_i z_{i2}, \ldots, S_i z_{i6})$ into the HMI. Due to learning, this signal pattern approaches the target position $(I_{i1}, I_{i2}, \ldots, I_{i6})$. In all, the head coordinates of S_i have learned to read their target position, expressed in agonist-antagonist muscle coordinates, into the HMI.

After learning occurs, suppose that $S_i > 0$ at a time when the eye is at the present eye position $(I_1, I_2, \ldots, I_6)$. By (4.8),

$$x_j = \frac{1}{A}(I_j - I_{ij}) \tag{4.11}$$

at these times. Equation (4.11) computes the vector difference of the present eye position and the target position, expressed in muscle coordinates. The HMI output signals are

$$V = ([x_1]^+, [x_2]^+, \ldots, [x_6]^+). \tag{4.12}$$

In pattern V, if an agonist muscle representation has a positive value, its antagonist muscle representation has a negative value, and conversely. Thus at most three of the six entries in V are positive at any time. These entries completely determine which vector difference is being computed.

4.9. Muscle Linearization and Retinotopic Recoding

As it stands, the HMI circuit clarifies some issues and raises others. The HMI design shows how target positions can be recoded into motor

coordinates so that vector differences which automatically compensate for present position can be computed (Section 1.7). The HMI design also shows how the visually reactive system prevents an infinite regress from occurring: although learning within the HMI can only associate *final* eye position in motor coordinates with a TPM target position, this eye position approaches the *target* eye position due to learning within the visually reactive system.

Two major problems still need to be solved in order for the HMI to work well: linearization of the muscle response to outflow signals, and transformation of the HMI output patterns V in (4.12) from muscle coordinates into retinotopic coordinates.

A. *Linearization of Muscle Response*

The need to linearize the muscle response to outflow signals can be seen by considering equation (4.11). Each potential x_j computes the difference of present eye position I_j and target position I_{ij} in muscle coordinates. Both I_j and I_{ij} are derived from outflow signals to the muscle plant. Unless the muscle plant contracts as a linear function of these outflow signals, neither I_j nor I_{ij} provides a reliable index of where the eye is actually pointing at any time. Thus a circuit is needed which can linearize the muscle plant's response to outflow signals, despite the fact that the muscle plant is nonlinear (Section 1.10).

This argument can be made more vividly by noting that the output V of the HMI is based upon a vector *difference* of target position and present position. *Infinitely* many choices of these positions can generate the *same* vector difference. The function of each fixed vector difference is to encode a determinate distance and direction that the eye must move to foveate a light. If the many individual target positions and present positions that lead to a fixed vector difference do not accurately reflect where the eye actually is or intends to go, then the vector difference itself cannot encode how the eye must move to foveate a light. A single vector difference could then be generated by combinations of target positions and present positions that do not represent the same distance and direction of motion between the actual present eye position and the actual position of a light on the retina.

These considerations strongly suggest that the muscle response is linearized by a separate learning circuit. In Chapter 3, we considered many possible ways whereby the saccadic control system could, in principle, compensate for muscle plant nonlinearity. The design of the HMI suggests a particular scheme. Given that a separate circuit linearizes the muscle response, the simulation described in Figure 3.12B indicates that a retinotopic sampling map may be sufficient to control the LTM traces which are tuned by second light error signals at the AG stage.

This conclusion does not imply that adaptive compensation for initial eye position is no longer needed. Indeed, such compensation occurs within the circuit that linearizes the muscle response (Chapter 5). However, this circuit delivers its conditioned signals to a processing stage that occurs

after, rather than before, the saccade generator (SG). Only a retinotopic sampling map is needed, for purposes of correcting individual saccades, to deliver conditionable signals before the SG stage. Despite this fact, there are other reasons why converging conditionable pathways from more than one sampling map are needed at a stage prior to the SG stage, as we will see in Chapter 9.

B. *Retinotopic Recoding*

The HMI transforms the multimodal target position map of the TPM into a much simpler unimodal motor map. However, muscle coordinates are the wrong coordinates from which to generate saccadic commands. Such commands need to be generated in retinotopic coordinates, so that they can benefit from second light visual error signals (Chapter 3). We are hereby led to appreciate more fully the simple but subtle deviousness of sensory-motor systems. Visual signals in retinotopic coordinates are recoded as target positions in head coordinates, then recoded as target positions in muscle coordinates, so that they can be recoded as vector differences in muscle coordinates, only to be recoded into retinotopic coordinates once again. The circle from vision to motor coordinates and back to vision is hereby closed.

How can a vector difference in muscle coordinates be recoded into retinotopic coordinates? Are these two types of information dimensionally compatible? Another subtlety is explicated by the answer: although a target position in muscle coordinates is dimensionally incompatible with retinotopic coordinates, a vector difference in muscle coordinates is dimensionally compatible with retinotopic coordinates. This is true because of the way in which a vector difference is computed. In order to compute a target position, initial eye position is added onto the retinal position of the light. In order to compute a vector difference, initial eye position is subtracted from target position. The addition-then-subtraction of initial eye position from retinal position shows that the vector difference is retinotopically consistent. Of course, this description ignores all the coordinate transformations and time delays that make these transformations functionally meaningful. Just adding and subtracting initial eye position seems meaningless, even absurd, outside of this functional context. However, this description shows that the vector differences that are computed in muscle coordinates within the HMI can, in principle, be mapped back into retinotopic coordinates. We suggest how this is done in Chapter 11.

We can now explain why we have often used the term "retinotopic" coordinates instead of "retinal" coordinates. We use the term "retinal" coordinates only to describe the frame in which lights on the retina are registered. The more general term "retinotopic" coordinates is used to describe any coordinate system, including vector differences, that can be mapped in a one-to-one way on retinal coordinates.

Before considering our solutions to the muscle linearization and retinotopic recoding problems, we describe some saccadic data that are clarified by properties of HMI dynamics.

4.10. Saccade Staircases and Automatic Compensation for Present Position

Schiller and Stryker (1972) have shown that a sustained electrode input to the monkey superior colliculus (SC) causes a staircase, or succession, of saccades of equal amplitude. In particular, Schiller and Stryker (1972) showed differences in evoked saccades due to electrical stimulation of superior colliculus and abducens oculomotor nucleus in the monkey. In the abducens nucleus, saccade amplitude was proportional to stimulation duration. In the deeper layers of the superior colliculus, stimulation longer than about 25 ms evoked a saccade whose amplitude was determined by collicular location. When stimulation duration exceeded 150 ms, two saccades were evoked with an intervening fixation. With prolonged stimulation a staircase of identical saccades was produced. The current threshold for generating such a staircase decreases by a factor of 20–100 as the electrode moves from the superficial layers to the deep layers of the SC. We will therefore focus on how such a staircase can be elicited from the deep layers of the SC.

Hikosaka and Wurtz (1983) have also generated saccade staircases from the SC. They accomplished this by injecting bicuculline, a GABA antagonist, into the SC. This manipulation, which decays over a time course of minutes, was made to directly test their hypothesis "that SN [substantia nigra] cells exert tonic inhibition on SC cells and that the pause in SN cell discharge before saccadic eye movements allowed the burst of activity in the SC cells. If this hypothesis were correct, application of GABA agonists and antagonists in the SC should clearly affect the initiation of saccades" (p.368), and this is what Hikosaka and Wurtz confirmed.

The existence of staircases *per se* will be explained in Section 7.6, after we have analysed how inputs to the saccade generator (SG) are normally updated after each saccade. In this section, we will comment upon two issues that are related to saccade staircases: Since saccade staircases can *sometimes occur* in response to a sustained input to the SG, then what prevents them from *always* occurring in response to any sustained command to the SG? Since each saccade in a saccade staircase starts out at a different initial position of the eyes, and since the same electrode input amplitude causes all of these saccades, why doesn't the size of successive saccades change due to the nonlinearity of the muscle plant, as it may in some species?

The main issue raised by the first question derives its interest from a comparison of the Mays and Sparks (1980) data with the Schiller and Stryker (1972) data. In both experiments, an electrode input to the SC was used. In the Mays and Sparks (1980) paradigm, our theory claims that the saccadic control system compensated for the movement caused by the electrode input by updating the present eye position input to the HMI, and computing the vector difference of the updated present eye position with the target position of the light. The vector difference then generated the command whereby the second saccade foveated the light.

Given that eye position is updated at the HMI after a saccade, and that

present position signals are excitatory, why doesn't *every* saccade cause its own saccade staircase by updating present position at the HMI and thereby generating another saccade? Thus the Mays and Sparks (1980) and Schiller and Stryker (1972) experiments, which seem to be unrelated when described in lay language, raise serious design issues when their implications are considered on the mechanistic level.

In our theory, this question is answered as follows. The TPM cannot activate a target position solely in response to eye position signals. A combination of visual signals and eye position signals is needed. The HMI, in turn, can generate output signals only at times when the TPM is actively reading a target position into the HMI. Thus, whereas eye position inputs to the HMI are updated by electrode-induced saccades in the Schiller and Stryker paradigm, these signals cannot elicit outputs from the HMI.

By contrast, an attended light input can activate a target position within the TPM. When the saccade caused by this light is over, the updated present position input to the HMI typically equals the target position input to the HMI. The target position and present position then cancel each other by vector subtraction. Thus although the HMI is capable of generating an output in this case, no saccade staircase is generated because the output equals zero.

These conclusions depend critically upon the idea that visually-dependent TPM activation controls the output gate of the HMI. Other authors have also realized that some sort of output gating is needed to control automatic compensation for present position. These authors have not, however, chosen the gating source that we require. For example, in his important review of arm movement control, Hyvärinen (1982, p.1115) wrote: "Joint neurons in area 5 discharge more vigorously during active than passive movements.. .and arm-projection and hand-manipulation neurons probably receive sensory signals from joints, muscles, and skin during active movements. It appears likely that the sensory activity in these neurons is 'gated' by inputs related to the preparation of the motor act...in the form of corollary discharge from motor structures". Hyvärinen thus suggested that the corollary discharges, not visually modulated target position commands, are the source of the gating signals. If this were so in the HMI, saccade staircases would relentlessly occur in the dark due to the tonic nature of the corollary discharges. We venture to say that a similar catastrophe would occur if corollary discharges gated the HMI that automatically compensates for present position in the arm control system.

The requirements imposed by HMI design also provide a simple answer to the question: Why are all the saccades in the saccade staircase equal in size, despite the nonlinear nature of the muscle plant? Our answer derives from the hypothesis that the muscle plant response is linearized by a separate adaptive circuit, so that the corollary discharges to the HMI have behavioral meaning. Equal electrode inputs generate equal movements from this linearized plant. In Chapter 5, we will state a prediction capable of testing this explanation.

We also note the report of Hikosaka and Wurtz (1983) that saccade oscillations can occur in their paradigm. In every case, a visual fixation point existed in their experiments. These oscillations may be due to saccades elicited by the fixation point that alternate with the drug-induced saccades. Such an oscillation could occur when a visually elicited activation is strong enough to competitively inhibit the drug-induced activation. Alternatively, the mechanism whereby saccades return to the head-in-center position may be at work. These alternatives can experimentally be differentiated by studying monkeys in the dark or in front of a homogeneous fixation screen.

4.11. Corrective Saccades in the Dark: An Outflow Interpretation

Shebilske (1977) has collected data which he used to argue for the existence of inflow signals from the eye muscles to the saccadic command system. He analysed the saccades of human subjects in response to briefly flashed lights that were turned off before the end of the saccades. Shebilske discovered that on trials wherein the first saccade to the light was incorrect, a second corrective saccade was directed towards the target with high probability. Since the light was no longer on, Shebilske argued that an internal representation of the position of the target in space was stored in STM. Shebilske (1977, p.46) also assumed that "outflow encodes the internal eye position." This assumption led him to conclude that the present eye position signals which are used to generate a corrective saccade must be inflow signals. Otherwise, since intended position and outflow are the same, there would be no basis for computing the discrepancy between present eye position and intended eye position.

An alternative explanation that uses outflow signals to provide present eye position information can be given for these data using properties of the HMI. Let the TPM store a target position in STM which reads-out a movement command to the HMI. Suppose that the many processing steps leading from the TPM through the HMI towards the cells that establish the new position of the eyes are susceptible to occasional encoding errors due to internal system noise. Also suppose that the signals from these outflow cells to eye muscles are almost always correctly interpreted. In other words, whereas Shebilske (1977, p.46) claims that "outflow encodes the intended eye position," we suggest that outflow does not always encode the intended eye position.

Suppose that after the first saccade occurs, the incorrect outflow signal, which has been obeyed by the muscles, is also used to generate a corollary discharge to the HMI. Since the muscles obeyed this outflow signal, it provides a veridical estimate of eye position after the saccade terminates. When the vector difference of target position and corollary discharge is computed at the HMI, a new command is generated which is capable of eliciting a corrective saccade in the dark.

Shebilske (1977, pp.45–46) reviews two test conditions to confirm his inflow hypothesis. Both test conditions are compatible with the above

outflow interpretation.

The main point of this discussion is to suggest that the Shebilske (1977) data are compatible with the same mechanism that we have used to explain the Mays and Sparks (1980) data. Recently, Guthrie, Porter, and Sparks (1983) have shown that vector compensation occurs in the Mays and Sparks paradigm after surgical elimination of all inflow signals from the eye muscles. In the light of our conceptual bridge between the two experimental paradigms, these recent data provide further support for an outflow interpretation of Shebilske's results. Evidence for outflow but not inflow has also been found using loaded contact lens (Skavenski, Haddad, and Steinman, 1972). They performed experiments in which they either fixed inflow signals or fixed outflow signals and tested for perceived monocular visual direction. In the first experiment, a suction contact lens was put on an eye and one of three loads was attached to the lens. The subject fixated a target straight ahead with a load on. In order to maintain this fixation, the subject had to increase the amount of outflow. Measurements of perceived direction were made by having the subject move a test target to the perceived straight ahead. The mean shift of perceived direction increased monotonically with increasing loads on the contact lens. Since loading the eye does not affect inflow, it was concluded that perceived direction is computed using outflow.

In the second experiment, a red target was exposed to the left eye while the loaded right eye was exposed to brief flashes of a white target. The subject was asked to match the direction of the white target to the red target. Here it was assumed that because the two eyes are yoked, the left eye maintained a constant outflow for both eyes, since it was exposed to a constant target. The right eye rotated passively under load, thereby varying inflow. There was no change in the mean perceived direction of the red target when the amount of passive rotation was subtracted from the measurements to account for shifts in the retinal locus. Again it was concluded that inflow does not contribute to perceived visual direction.