CHAPTER 8
POSTURAL STABILITY AND LENGTH-TENSION REGULATION

8.1. Separate Postural and Movement Systems

Two distinct types of adaptive circuits have thus far been invoked to ballistically move the eyes to target positions. The muscle linearization network (MLN) compensates for the nonlinearity of the oculomotor plant, whereas the retinotopic command network (RCN) and vector command network (VCN) use visual error signals to alter the direction and length of saccades. Neither of these mechanisms suffices to maintain the position of the eye during gaze. This is because both types of movement circuits are used to calibrate the proper direction and length of a saccade, but not the tensions needed to maintain gaze after a saccade is over. As Section 1.12 noted, unequal lengths of agonist and antagonist muscles must coexist with equal tensions in these muscles to maintain gaze after the saccade is over.

Figure 7.4 provides a more precise understanding of this design issue. In Figure 7.4, the medium lead burster (MLB) population v_6 that excites the motoneuron (MN) cells v_{10} of an agonist muscle also completely inhibits the MN cells v_9 of the antagonist muscle. In particular, the inhibition of the antagonist MN by the agonist MLB prevents the antagonist tonic (T) cells from reading-out their signals to the antagonist muscle during the saccade. This push-pull arrangement prevents the antagonist muscle from unnecessarily slowing down during a rapid agonist contraction. In particular, the force exerted by the antagonist muscle on the eyeball is much reduced during a saccade.

By contrast, as soon as the saccade is over, the MLB inhibition is removed from the antagonist MN cells, and the full impact of T cell output is felt at the antagonist muscle. The balance of tensions between agonist and antagonist muscles consequently changes. In order to prevent the eye from drifting away from its target position after the saccade is over, an additional circuit is needed to compensate for any imbalances that may exist in agonist-antagonist tensions. This circuit must be able to do its work without altering the muscle lengths that were attained by a saccade. Thus movements and postures must be regulated by different control systems in order to preserve the lengths achieved by movement, yet compensate for the tension changes that occur during posture.

The postural system, no less than the movement system, must be capable of learning. The intrinsic tension characteristics of the eye muscles may change through time. The degree of tension imbalance at each combination of agonist-antagonist muscle lengths may also differ, and could not be effectively counterbalanced by a prewired mechanism.

8.2. Tension Equalization Network

Figure 8.1 describes a network capable of learning to generate equal agonist-antagonist tensions without undermining the length characteristics of the movement system. This circuit is called a *Tension Equalization Network* (TEN). The goal of the TEN is to prevent post-saccadic drifts from occurring. It does so by using motions of visual cues with respect to the retina as error signals after a saccade is over. Visual motions with respect to the retina can also occur when the eyes are moving due to active movement commands. In order not to confuse these two types of motion, gating mechanisms are needed which enable learning to occur only when the eye is not in an active movement mode. Another type of gating must also occur within the TEN. Read-out of postural signals from the conditioned pathways of the TEN must also not be allowed except in the postural mode. If equalizing tensions were imposed upon agonist and antagonist muscles in the movement mode, then movements would be impaired. It remains to discuss whether the gating mechanism which shuts off learning except in the postural mode is the same gating mechanism which shuts off output signals except in the postural mode. This would, of course, be the most parsimonious solution.

8.3. Design of the Tension Equalization Network

The TEN shares many of the design features that are used by the MLN and the EPUN. It also shares design features with networks capable of vestibulo-ocular reflex (VOR) adaptation. As in the MLN and the EPUN, the TEN spatially encodes T cell activity patterns at an eye position map (EPM). The EPM, in turn, sends conditionable pathways to the adaptive gain (AG) stage.

The error signals which these pathways sample at the AG stage are neither the error signals from the outflow-inflow interface (OII) of the MLN, nor the second light error signals of the EPUN. The TEN needs to use error signals that can measure the amount of post-saccadic drift. At first broach, one might think that an OII-activated error signal might be suitable, because it could use inflow signals to register motions of the eye muscles with respect to outflow commands. This possibility seems less tenable when one considers the physiological mechanisms whereby inflow signals are generated. If Golgi tendon organs were used as the inflow source, the mechanism would fail totally both because the tendons are insensitive to passive stretch and because tension-regulating feedback from the TEN would confound whatever length-predictive information the tendons did deliver. Length-based inflow signals could also be altered by tension-regulating feedback from the TEN.

We suggest that visual error signals are used by the TEN. These error signals must be capable of correcting post-saccadic drifts even in the absence of moving visual targets. Hence we suppose that directionally tuned error signals are activated by motions of the whole visual field. The accessory optic system is capable of generating error signals of this type

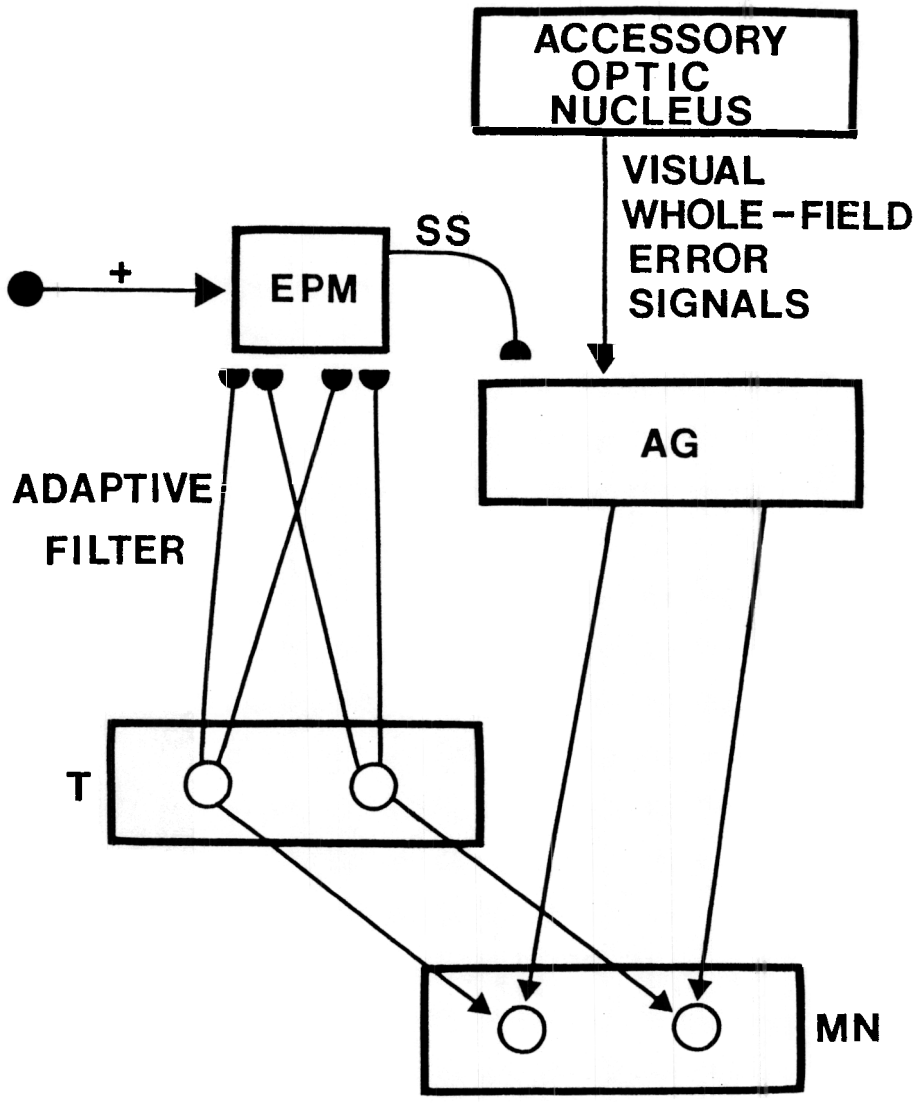

Figure 8.1. Circuit diagram of a tension equalization network (TEN): This network prevents post-saccadic drift from occurring during gaze. It adaptively balances agonist-antagonist tensions even though the agonist-antagonist muscle lengths are unequal. As in the muscle linearization network (MLN) of Figure 5.2., an eye position map (EPM) is the source of sampling signals to the adaptive gain (AG) stage. As in the circuit for vestibulo-ocular reflex (VOR) adaptation of Figure 8.3, whole-field visual error signals are used, rather than second light error signals. Abbreviations: T = tonic cells, MN = motoneurons.

(Simpson, Soodak, and Hess, 1979). Directionally-tuned whole-field visual error signals can, for example, be generated by a network whose cells fire only if they receive convergent inputs from a sufficiently large number of cells tuned to a similar direction. We assume that a whole-field visual motion in a direction opposite to a prescribed muscle's direction of contraction causes the strength of the conditioned pathway to the AG strip of that muscle to increase and the strength of the conditioned pathway to the AG strip of the antagonist muscle to decrease (Figure 8.2).

As in the MLN, the AG stage sends pathways directly to the MN cells. The pathway

$$T \text{ cell} \rightarrow EPM \rightarrow AG \rightarrow MN \tag{8.1}$$

can influence muscle tensions without altering the tonic cell outputs that determine muscle length. In order for the TEN to work well, the conditioned pathways to these MN cells must be active only in the postural mode, and learning must be possible only in the postural mode. Both of these requirements can be achieved by a single gating signal that nonspecifically inhibits the TEN somewhere between the T cells and the AG stage in Figure 8.1. The cells which give rise to such an inhibitory gating action must be active only during a saccade. Burst cells could therefore be the sources of this inhibitory gating signal.

The TEN accomplishes its function in the following way. During a saccade, its conditioned pathways to the MN cells are inactivated by gating signals. After the burst cells shut off, these conditioned pathways are disinhibited, as the T cells register the outflow signal pattern corresponding to where the eyes should be. If the eyes then begin to drift in their orbits, these outflow signals do not change. Hence they provide a stable source of sampling signals to the AG stage. Each distinguishable T cell outflow pattern can control a different conditionable pathway (Figure 8.2) to the AG stage. Each such pathway can learn to control conditioned gains of all relevant muscles at the AG stage. These gains change as a result of learning in a way that tends to prevent future drifts when the eye ends up in the same, or a similar, outflow position. In short, the TEN is indifferent to how the eye got to where it is at the end of a saccade. Wherever that might be, the TEN uses its outflow-activated sampling pathways to hold it there.

It is important to realize that the TEN is designed to accomplish this goal without interfering with the functioning of other adaptive circuits. For example, the RM and EPM sampling maps of the EPUN have already determined an outflow pattern across the T cells before the TEN is allowed to turn on after a saccade terminates. The MLN has also achieved an approximately linear muscle plant response via the MN cells before the TEN is allowed to turn on. By the time the TEN turns on, the T cell pattern that activates its EPM has already been determined, and the positions of the eyes in the head have also been determined. It remains only for the TEN to resist passive eye movements after the active movement signals shut off, and to do so via the MN cells so as not to disturb the other computations that got the eyes to wherever they might be. Thus,

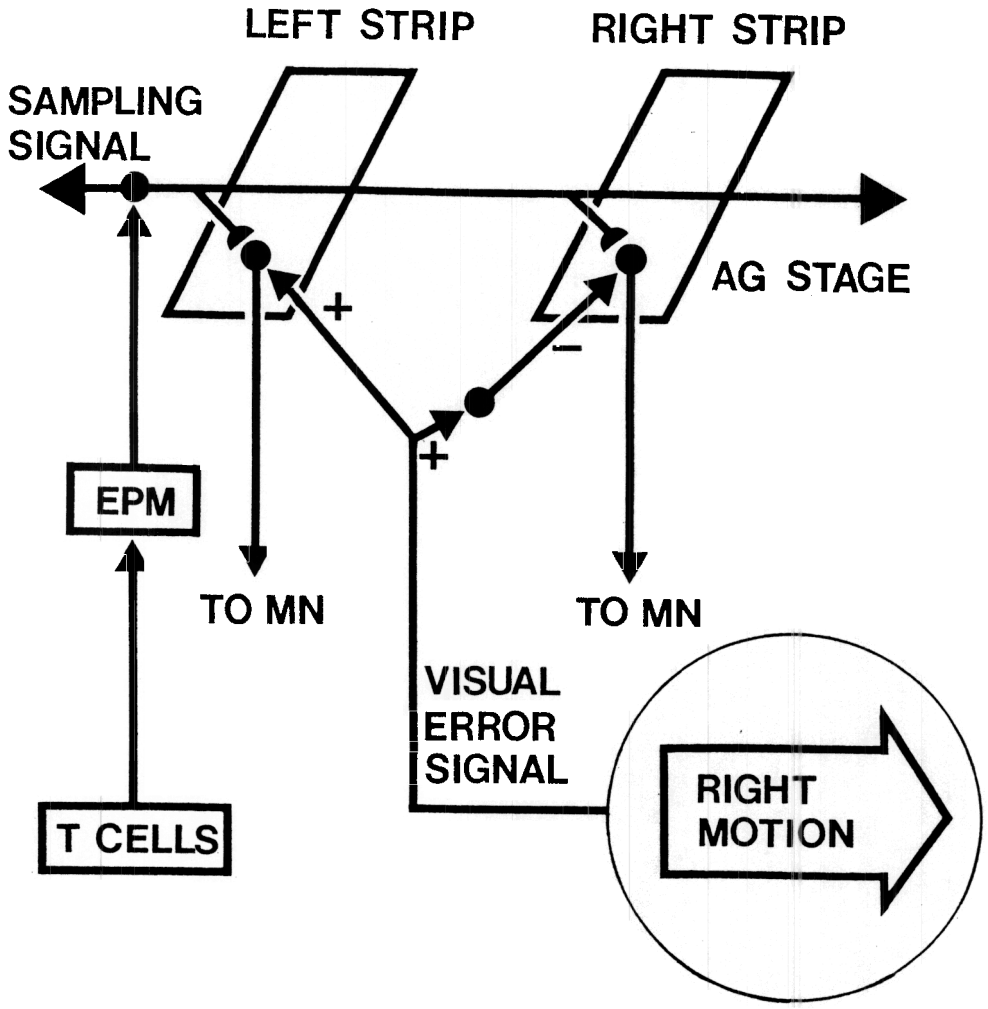

Figure 8.2. Influence of whole-field visual error signals on the adaptive gain (AG) stage: A whole-field motion to the right on the visual field causes the conditioned gain in the left motor strip to increase and in the right motor strip to decrease. Alternatively, competition between the agonist-antagonist strips could cause the net gain of the right motor strip to decrease, as in Figure 3.6b.

at times when the TEN is turned on, the signals from the MN cells to the eye muscles explicitly encode the tension requirements that hold the eyes in place, but implicitly encode both length and tension information that prevent the eyes from moving from wherever they were intended to be.

8.4. Adaptive Step Gain and Pulse Gain: Correcting Post-Saccadic Drift

Several laboratories, notably the laboratory of D.A. Robinson, have reported data that are compatible with the existence of separate cerebellar movement and gain subsystems, notably with properties of the TEN circuit as a model of gain control during gaze. The cerebellar direct response cells that Ron and Robinson (1973) discovered may be included in TEN circuits as well as in MLN circuits. In addition to the cerebellar direct response cells, which elicit saccades with the remarkably short latency of 5–9 msec., Ron and Robinson (1973) have reported the existence of cerebellar cells that elicit saccades with a much longer latency (12–26 msec.). These latter signals have properties compatible with their action as conditioned movement signals to the SG. Hepp, Henn, Jaeger, and Waespe (1981) have also reported saccade-related burst-tonic mossy fibers in the vermis having properties compatible with an SG action.

Optican and Robinson (1980) have reported cerebellar data that more directly support the existence of separate movement and gaze cerebellar pathways. They studied the effect of discrete cerebellar lesions upon the movement (pulse) and gaze (step) components of saccadic movements. Lesions of the vermis, paravermis (lobes IV–IX) and the fastigial nuclei abolished adaptive control of the pulse component, but not of the step component. Lesions of the flocculus abolished adaptive control of the step component.

Optican and Robinson (1980) also operated on the medial and lateral recti of one eye. They showed that saccade lengths and post-saccadic drift in the weakened eye were corrected only when the eye was exposed to visual experience. This fact is compatible with the use of visual error signals to recalibrate both saccade length and post-saccadic drift. Our theory suggests, however, that different visual cues are used to calibrate the pulse and step gains; namely, localized second light error signals and whole-field visual motions, respectively. This prediction may be testable by ablating the accessory optic nucleus instead of the flocculus, and testing whether the pulse gain recovers from an eye muscle operation, whereas the step gain does not. Optican and Robinson (1980) also commented (p.1071): "The fact that the time constant of the postsaccadic drift was about 40 ms presents a puzzle. This is much shorter than the dominant time constant of the orbital mechanics, which has a mean value in rhesus monkeys of about 200 ms." This property is explained in our theory by the fact that the muscle gain during post-saccadic drift is modulated by active signals from the TEN, not only by passive muscle properties. Thus cutting the pathways subserving the gain control signals can cause saccadic overshoots or undershoots to occur even if the muscles are undamaged. This fact

is consistent with the observation of Optican and Robinson (1980) that cerebellectomy caused both saccadic overshoots and post-saccadic drifts.

8.5. Relationship to the Vestibulo-Ocular Reflex

We have suggested that directionally-sensitive whole-field visual motions generate the error signals of the TEN, and that the accessory optic nucleus may be a source of these error signals to the cerebellum. It has been suggested that the accessory optic nucleus also generates the whole-field error signals that are used to adaptively calibrate the VOR (Simpson, Soodak, and Hess, 1979). Figure 8.3 summarizes a VOR model which maximizes the functional homology with the TEN circuit. As in the TEN, a whole-field visual error signal in the VOR circuit acts to change conditioned gains in order to prevent slippage of visual cues across the retina. Within the TEN, the EPM that samples these error signals is activated by T cells. Within the VOR circuit, the EPM that samples these error signals is activated by the vestibular canals (Ito, 1984). In both the TEN and VOR circuits, different head positions can control different synergies of conditioned gains capable of preventing motions of the eyes relative to the visual world. Also in both circuits, a whole-field visual drift towards the right strengthens the eye motion to the left. In the TEN, the strengthened eye motion to the left prevents post-saccadic drift to the right during posture. In the VOR circuit, the strengthened eye motion to the left prevents relative motion of the visual field to the right during a head movement.

The functional homology between the TEN and the VOR circuits calls attention to the possibility that sampling signals within the VOR circuit may be gated at some stage between the vestibular canals and the cerebellum. In the TEN, this gate is opened only in the postural mode. Such a posture-dependent gating action would also be useful in the VOR circuit. This can be seen by noting that a saccade may accompany a head movement. The head movement can activate the VOR sampling pathways as the saccade may generate visual whole-field motions. Saccades thus generate whole-field error signals at times when the VOR circuit is already correctly calibrated. A posture-dependent gating action can prevent saccade-generated whole-field motions from erroneously altering the VOR gains. In species where the VOR circuit can compensate for head movements during saccades, this gating action is likely to occur at the synaptic knobs of the sampling signal pathway within the AG stage. That is, a posture-dependent nonspecific gating input is likely to occur at the parallel fiber-Purkinje cell synapse of the cerebellum. At such a locus, the posture-dependent gate can prevent learning from occurring except in the postural state, but could nonetheless allow read-out of the conditioned gains to occur even during saccades.

These comparisons between the TEN and the VOR circuits suggest that both circuits may sample error signals that are registered within the same part of the cerebellum. The cerebellar lesion experiments of Optican and Robinson (1980) are compatible with the hypothesis that the flocculus may be the cerebellar region where these whole-field error signals

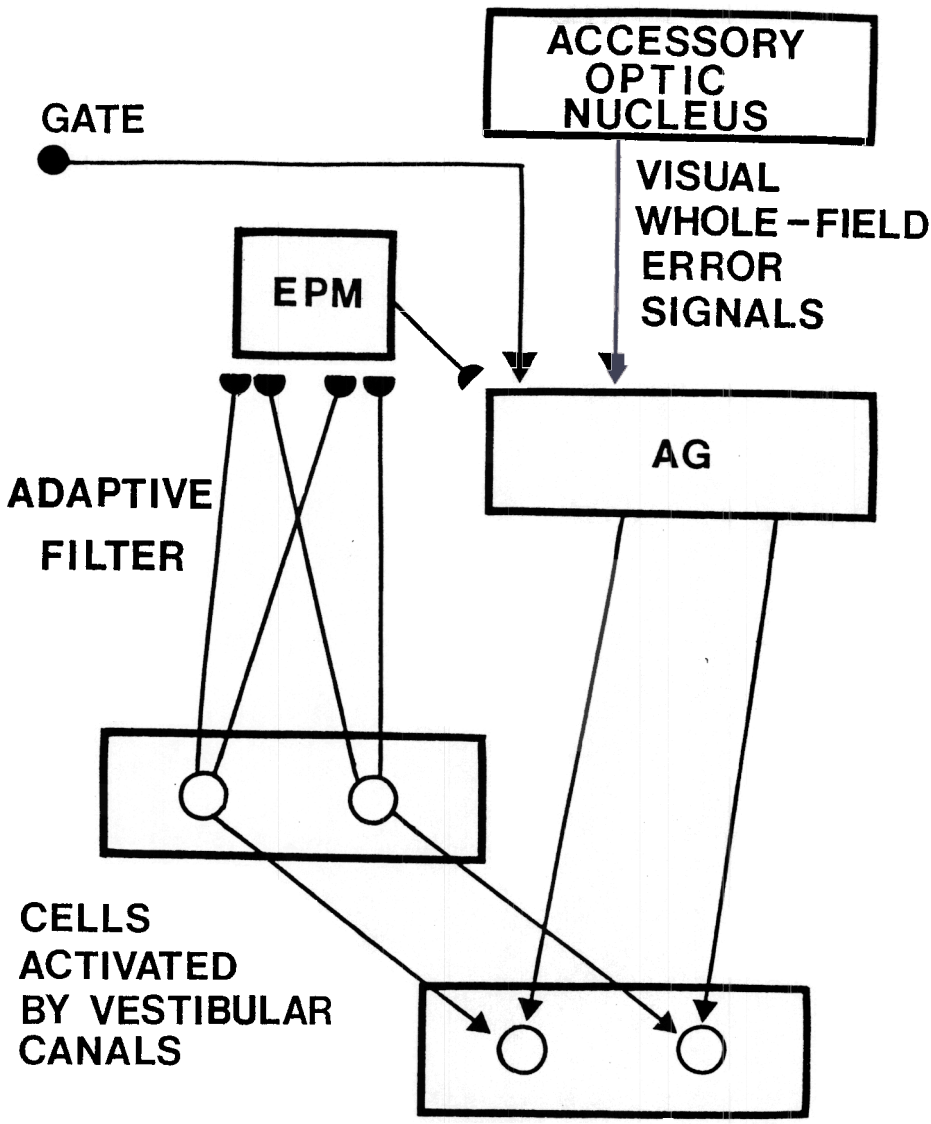

Figure 8.3. Circuit diagram for vestibulo-ocular reflex adaptation: This circuit is intended to suggest possible homologs with the circuit of Figure 8.1 that adaptively prevents post-saccadic drift.

are sampled by both circuits. This hypothesis is further strengthened by the results of Ito *et al.* (1974), who showed that lesions of the flocculus also impair VOR adaptation.

8.6. Cerebellar Functional Heterogeneity

Our analyses of circuits such as the RCN, VCN, MLN, EPUN, and TEN suggest that at least three different types of error signals are registered within the cerebellum: second light visual error signals, OII-activated error signals, and visual whole-field error signals. These results thus provide an evolutionary rationale for subdividing the cerebellum into regions which are functionally specialized to receive different types of error signals. Our analyses also suggest how all of these error signals can take advantage of a common delivery system by climbing fibers feeding into a fractured somatotopy, a hemifield competition, or both (Chapter 3). Several different learning circuits can sample error signals to each of these functionally distinct regions, much as both the TEN and VOR circuits have been hypothesized to sample the flocculus. Each of these different learning circuits maps RMs or EPMs onto the cerebellar topography. Hence multiple copies of several different type of sampling maps need to be superimposed upon maps of several different types of error signals, just to move the eyes and hold them in place (Figure 8.4).

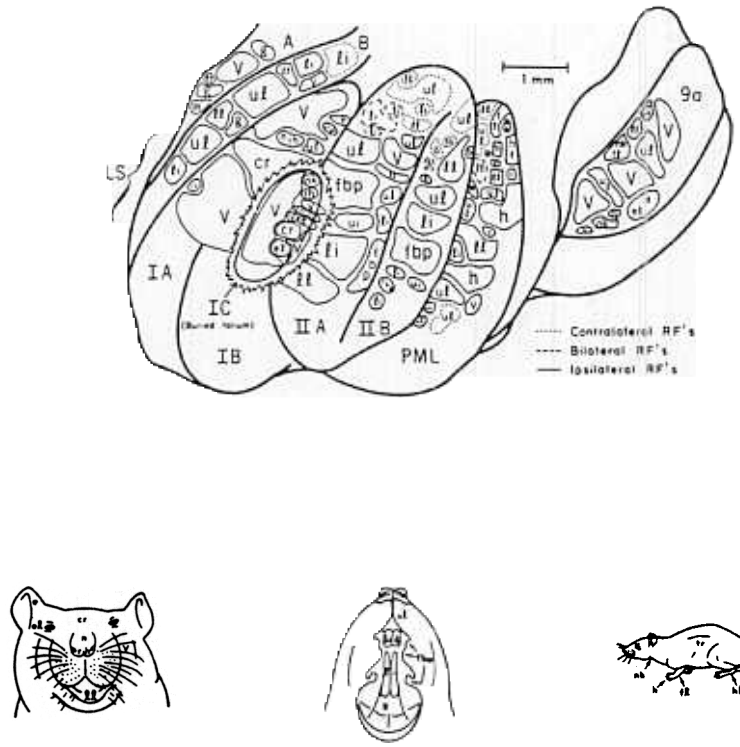

Figure 8.4. Multiple somatotopic representations in cerebellar cortex: Tactile mossy fiber projections to the granule cell layer of the cerebellar cortex are arranged in a patch-organized mosaic as shown. Letters within each patch indicate the body region that projects to that patch. Abbreviations: LS = lobulus simplex, IA and IB = the two surface folia of crus I, IC = the buried folium of crus I revealed in the "cutaway" of the overlying crus I and II, IIA and IIB = the two folia of crus II, PML = paramedian lobule, 9a = uvular folium IXa. From Bower *et al.*, *Brain and Behavioral Evolution*, **18**, 1981, 1–18. Reprinted with permission.