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Neural models of spatial perception and the control of movement *)

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In a recent book, Arbib [1] has argued that we must view the brain as "an action-oriented layered somatotopically-organized distributed computer". The paper presents two theories developed within that framework. The first analyzes the spatial component of perception: while the second models the role of the cerebellum in the control of movement. Both models fit the same general methodology: we develop a clear understanding of the function which is to be explained (psychology) and of the units of explanation (neurophysiology) and then build a theory which bridges the gap between the global and local levels of description.

In the model of figure-ground separation [8] which we shall present as applied to stereopsis, the psychological data are provided by the Julesz studies on perception of random dot stereograms; while the neurophysiological data comes from the study of disparity detecting neurons in visual cortex made by such workers as Barlow, Bishop, Blakemore and Pettigrew. The synthesizing model involves interaction of layers of excitatory and one layer of inhibitory neurons.

In the model of cerebellar control of movement [4], the psychological data are provided by the Russian school founded by Bernstein and by Greene's theory of tasks; while the neurophysiological data come not only from such studies as those summarized in Eccles, Ito and Szentágothai in "The Cerebellum as a Neuronal Machine", but also from studies of climbing fiber responses, Oscarsson's mapping studies, and Tsukahara's work on reverberating loops. The model shows how climbing fiber activation can release synergies, pretuned by mossy fiber activation, by freeing reverberatory loops from Purkinje cell inhibition.

1. Introduction

Before developing the neural models of stereopsis and control of movement which form the core of this paper, we wish to set a general framework by

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discussing two "slogans":

1. The brain "is" an

   ACTION-ORIENTED
   LAYERED
   SOMATOTOPICALLY ORGANIZED
   computer.

2. Transformation and Equilibration of Spatio-temporal Input Patterns in Interaction with Innate or Previously Formed Patterns (Internal Models of the World) yield Adaptive Interaction of the Organism with its World.

The term "action-oriented" reminds us that neurons do not simply exist to provide activity that the neurophysiologists can correlate with the animal's stimuli and responses; but rather form part of a complex system for mediating the animal's interaction with its world. Thus we must think of neural activity not in terms of representations of simple laboratory stimuli (such as flashes of light) but rather in terms of representing interaction, unfolding in time, with a complex world of three-dimensional objects. (This would seem to be precisely the point made by the paper of MacKay and Mittelstaedt in this volume. See also the related paper by Didday and Arbib [9]). The term "layered" suggests that rather than jump directly from overall behavior of the animal to the activity of single neurons, we should seek intermediate levels of analysis, and that the spatio-temporal pattern of activity in a layer of neurons - such as the layers of the retina or of the cerebellar cortex - may provide such a level. We all know of the somatotopic maps ("soma" is Greek for body, "topos" for place) of body surface displayed as homunculi on human motorsensory cortex, with regions of body being mapped to regions of cortex, with rough preservation of spatial relationships, though not of metrical ones. The term "somatotopically organized" is to remind us that, even though position on the body does not provide the key coordinate for interpreting activity in most layers of the brain, nonetheless such a type of "map" or""positional code" will help us make sense of what is going on in these layers. (Our first "slogan" is developed at length in "The Metaphorical Brain" [1].)

The second slogan cautions us against any too naive a stimmulus-response or unidirectional-information-flow view of neural activity. As such terms as "afferent control of receptors" and "internal feedback" serve to remind us, there is a continual interaction between different layers. Again, the transformations induced in the layers bear the marks of the animal's history, both as individuals, and as members of the species - and whether we talk of "learning" or of "evolution", we seek to understand processes of adaptation which better fit the organism to interact with its world. However, in this paper we shall not be concerned with learning in neural networks, for we believe it important to understand how the structure of networks can

enable them to subserve complex functions, whether or not the structure will be modified over time.

In short, we wish to understand how to analyze layers of the brain in dynamic interaction with one another being modulated by sensory input and yielding, via the musculature, activity in the world. Rather than tackle this problem for one complete sensorimotor system, we devote this paper to two neural models: on emphasizing the processing of sensory input to determine *where* in space are the objects with which the animal is to interact - a model of one of the mechanisms of stereopsis [8]; and one emphasizing the role of the cerebellum and its environs in balancing the flexions and extensions which constitute the synergies of movement [4]. In both cases, we have constructed theories which attempt to bridge the gap between overall function (what one might call Psychology) and the sparse unit data of neuroanatomy and single-cell neurophysiology.

We start to break down overall system function by making plausible hypotheses about the parcellation of subfunctions between regions of the brain; look at each subsystem in terms of the spatiotemporal processing in its layers; while - working up as well as down - our views of a layer's processing are constrained by available knowledge of the interconnections and interactions of its neurons.

2. Stereopsis

A point light source will stimulate a small region on both retinas of an animal (such as cat, monkey or man) with forward-facing eyes. If the point is at the focal point of the two eyes, or on the Vieth-Müller Horopter Circle [1]) defined by the focal point and the two lenses, the regions on the two retinas will be similarly located, as for F_L and F_R, or for B_L and B_R, in Figure 1. However, as we move away from the Horopter circle, the disparity between the coordinates of the two retinal activities changes. Thus while each retina provides only a two-dimensional map of the visual world; the two retinae between them provide information from which can be reconstructed the three-dimensional location of all unoccluded points in visual space. We indidicate this in Figure 1, where the right retina cannot distinguish A, B or C ($A_R = B_R = C_R$), and where the left retina can distinguish them but cannot determine where they lie along their ray. The two retinae can actually locate them on the ray: (A_L, A_R) fixes A, (B_L, B_R) fixes B, and (C_L, C_R) fixes C.

There are two problems:

The first is that our observation that the two retinae contain enough information to determine the three-dimensional location of a point in no way implies that there exists a neural mechanism to use that information. How-

[1]) For the purpose of our model, the subtlety of the optics may be ignored, and we can in fact view the horopter as an actual circle in the cross-section of Figure 1.

ever, work by Bishop [20], Barlow, Blakemore and Pettigrew [2] seems to have unearthed such a mechanism [2]): briefly, they find cells in visual cortex which not only respond best to a given orientation of a line stimulus, but do so with a response which is sharply tuned to the disparity of the effect of the stimulus upon the two retinae.

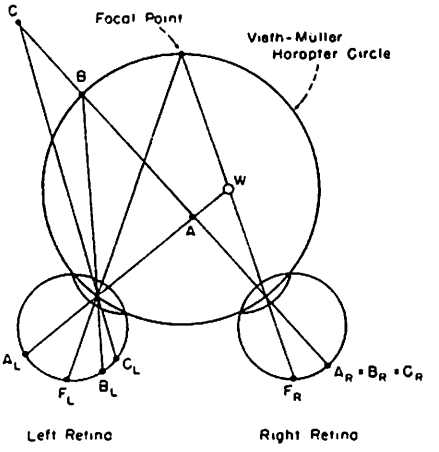

Fig. 1:
The Notion of Disparity

The second problem is that information is given about three-dimensional location of points only when the corresponding points of activity on the retinae have been correctly paired. If the only stimuli activated in Figure 1 were at the focal point and at A, then A can only be accurately located if A_L is paired with A_R - were A_L to be paired with F_R, the system would "perceive" an "imaginary" stimulus at W. The main thrust of the model presented below will be to suggest how disparity-detecting neurons might be connected to restrict ambiguities resulting from false correlations between pairs of retinal stimulation. But before giving the details, let us examine some psychological data which define the overall function of the model.

Normal stereograms are made by photographing a scene with 2 cameras, with relative position roughly that of a human's two eyes. When a human views the resultant stereogram - with each eye viewing only the photograph made by the corresponding camera - he can usually fuse the two images to see the scene in depth. Julesz has invented the ingenious technique of random-dot stereograms to show, inter alia, that this depth perception can arise even in the absence of the cues provided by monocular perception of familiar objects. The technique is caricatured (using a 5 × 4 grid, rather than a grid of 100 × 100 or even finer texture) in Figure 2. The slide for the left eye is prepared by simply filling in, completely at random, 50% of the squares of an array. The slide for the right eye is prepared by transforming the first slide by shifting sections of the original

2) However, MacKay, in discussion at the conference, suggested that recent work of Hubel casts some doubt on their findings.

pattern some small distance (without changing the pattern within the section) and otherwise leaving the overall pattern unchanged. In Figure 2, we have simply taken one 2 × 3 rectangle and shifted it one square left [3]), filling in at random the 2 squares thus left blank. Of course, this array is not dense enough to yield interesting effects, but with Julesz's actual arrays, one slide presented to each eye, subjects start by perceiving visual "noise" but eventually come to perceive the "noise" as played out on surfaces at differing distances in space corresponding to the differing disparities of the noise patterns which constitute them.

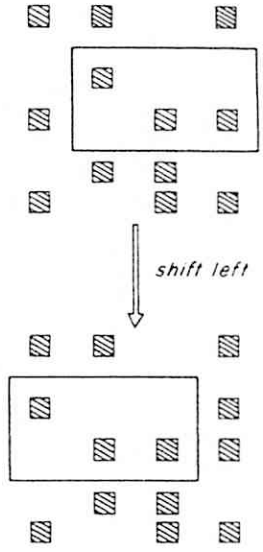

Fig. 2:
Julesz Stereograms. The brain can correlate random subarrays of varying disparity. Time is required; surfaces „suddenly" appear.

Note well that both stimuli of the stereogram pair are random patterns. Interesting information is only contained in the correlations between the two - the fact that substantial regions of one slide are identical, save for their location, with regions of the other slide. Then the visual system is able to detect these correlations. If the correlations involve many regions of differing disparities, the subject may take seconds to perceive so complex a stereogram - during which time the subjective reports will be of periods in which no change is perceived followed by the sudden emergence of yet another surface from the undifferentiated noise.

To clarify the ambiguity of disparity in Julesz stereograms, let us turn from the rectangular arrays of Figure 2 to the linear arrays of Figure 3. The top line shows the 21 randomly generated 0's and 1's which constitute the "left eye input", while the second line is the "right eye input" obtained by displacing bits 7 through 13 two places left (so that the bit at i position

[3]) The outlining of the rectangle is only given for the convenience of the reader - it would not be shown on the actual stereogram pair presented to a subject.

goes to position i-2 for $7 \leqq i \leqq 13$) while the bits at position 12 and 13 thus left vacant are filled in at random (in this case, the new bits equal the old bits - an event with probability 1/4), with all other bits left unchanged. Then in the remaining 5 lines of the figure we show a disparity array, with the i^{th} bit of the disparity of line D being a 1 if and only if the ith bit of the "right eye input" equals the (i + d)th bit of the left-eye input.

The disparity array of Figure 3 suggests the stripped-down caricature of visual cortex which we shall use for our model (which is a simplification of the Dev [8] model of figure-ground separation). Rather than mimic a columnar organization, we segregate our mock cortex into layers, with the initial activity of a cell in position i of layer d corresponding to the presence or absence of a match for the activity of cell i of the right "retina" and cell i + d of the left retina. (This positioning of the elements aids our conceptualization. It is not the positioning of neurons that should be subject to experimental test, but rather the relationships that we shall posit between them.) As we see in Figure 3, the initial activity in these layers not only signals the "true" correlations (A signals the central "surface"; B and D signal the "background"), but we also see "spurious signals" (the clumps of activity at C and E in addition to the scattered 1's, resulting from the probability of 1/2 that a random pair of bits will agree) which obscure the "true" correlations.

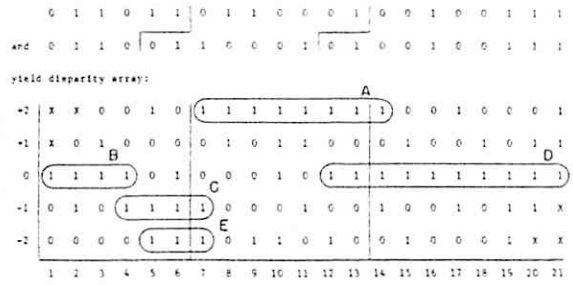

Fig. 3: The Problem of Resolving Ambiguity
Conceptualization: „Layers" of cells (they are really in „columns"), one for each gross disparity.
Principle: Minimize the Number of Connected Regions.
Possible Solution: Moderate local cross-excitation within layers; increasing inhibition between layers as difference in disparity increases

Can we, then, interconnect the "neurons" of our "disparity array" in such a way that the spurious correlations are suppressed? We might imagine (but only as a crude first approximation) the resultant array of activity as then providing suitable input for a higher-level pattern-recognition device which can in some sense recognize the three-dimensional object whose visible surfaces have been so clearly represented in the brain.

Our aim then is to divide the "visual field" (in this case indexed by the numbers 1 through n = 21) into a number k of regions $[1, x_1), [x_1, x_2), \ldots, [x_{k-1}, n]$ such that in each region only cells of one level of the disparity array are active. In putting conditions upon this division, we are guided by the plausible hypothesis that our visual world is made up of relatively few connected regions. Our "optimization principle" will then be to attempt to choose $x_1, x_2, \ldots, x_{k-1}$ in such a way as to minimize k while at the same time accounting for "most" of the original activity in the array. While we do not have a precise functional scheme which we have minimized, we do have a plausible interconnection scheme which yields qualitatively appropriate behavior of the disparity array:

The essential idea is given by the rule that there be moderate local cross-excitation within a layer; and inhibition between layers which increases as the difference in disparity increases. Let then $x_{di}(t)$ represent the activity of the cell in position i of layer d at time t; and let h(j) and k(j) be functions of the form indicated by Figure 4. Then the change of activity of a cell is given in our model by the equation (to avoid end conditions we may regard the disparity array as being wrapped on a torus)

$$(1) \qquad x_{di}(t+1) = \sum_{d'} \sum_{i'} h(d-d')\, k(i-i')\, x_{d'i'}(t) + x_{di}(t_0)$$

where it is understood that the sum "saturates" at 0 and at 1.

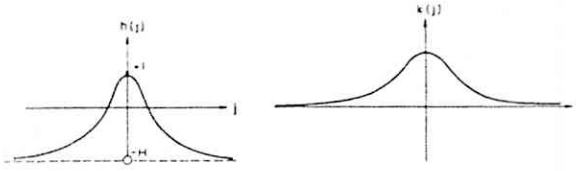

Fig. 4:
Interaction Coefficients

What this scheme does is allow a clump of activity cells in one layer to "gang up" on cells with scattered activity in the same region but in other layers, while at the same time recruiting moderately active cells which are nearby in their own layer. The system then tends to a condition in which the activity is clearly separated into "regions", with each region having its own unique disparity (layer of activity). (In a more finely textured model than this, one might also see gradations of disparity corresponding to sloping surfaces.) In other words, such a scheme resolves ambiguity of disparity information through suppression of scattered activity, thus permitting activity related to only one disparity at any one location. Moreover, the dynamics of the model does represent the Julesz phenomenon of a noise stereogram taking some time to be perceived, with each new surface being

perceived rather abruptly. This is simulated in the model by the fact that, once a sufficient number of clumps achieve high activity, the recruiting effect fills in the gaps between the clumps to form a good approximation to its final extent.

Before closing our discussion of this pleasingly simple model for one of the mechanisms involved in stereopsis, we should note that equation (1) can be rewritten in a fashion which suggests a plausible scheme of neural interconnection.

We decree that the neurons of the disparity array all be excitatory. We now introduce a layer of inhibitory interneurons, the ith of which has activity at time t given by the simple equation

$$y_i(t) = \sum_d x_{di}(t).$$

Let us now pick a constant H such that $\bar{h}(j) = H + h(j) \geqq 0$ for all j. We may then rewrite (1) in the form

$$x_{di}(t+1) = \sum_{d'} \sum_{i'} (\bar{h}(d-d') - H)\, k(i-i')\, x_{d'i'}(t) + x_{di}(t_0) \tag{2}$$

so that

$$x_{di}(t-1) = \left\{ \sum_{d'} \sum_{i'} \bar{h}(d-d')\, k(i-i')\, x_{d'i'}(t) \right\} - \sum_{i'} l(i-i')\, y_{i'}(t) + x_{di}(t_0) \tag{3}$$

where $l(i-i') = Hk(i-i')$. Thus (3) shows that our model may be given structural expression in a form in which

the x_{di} are all excitatory, with excitation decreasing with distance, the excitation being appropriately counteracted by inhibition from single layer of inhibitory interneurons.

The resultant architecture of several layers of excitatory neurons, each layer interacting with the others through a single layer of inhibitory neurons, lends a simple and novel framework for experiments involving adaptation effects between feature detectors, such as in the McCollough effect.

It is interesting that the two properties of suppression and of recruitment occur as two separate phenomena. Activity in the inhibitory layer is the first to stabilize at its new value. Thus, further changes of one disparity array do not directly affect the others.

1. Because of the increased inhibition, regions of low input reduce their activity even further. Any scattered high input has little effect because of the decrease in surrounding activity. This is the suppression effect.
2. The disparity layer with high input increases its activity drastically (a linear model shows an exponential time course for this increase). Scattered regions of low input achieve much higher activity than they would alone because of the high excitatory input from neighbors. Cracks

and gaps do not get fully plugged but they come close to it. The spread of such a plugging or filling-in effect is limited by the spatial extent of the excitatory interaction, i.e., high activity does not spread like a wave across a wide gap. Rather, every clump of high activity can raise activity in a surrounding region, and, if these regions are sufficiently close to each other, the gap is plugged. (Such a wave-like spread can be achieved by nonlinear systems under suitable conditions. System parameters must be adjusted so that the wave stops at the desired boundary.)

3. Control of Movement

In describing the movement of a horse, we do not normally talk of the independent contraction of hundreds of thousands of muscle fibers, but instead specify the gait - a walk trot or canter, say - and the speed at which it is moving. This suggests a more general strategy: to describe the control of movement in terms of selecting one of a relatively short list of modes of activity, and then, within each mode specifying the few parameters required to tune the movement. This strategy seems to have been borne out by the experiments of the Russian school [4]) of Bernstein [3], Gel'fand [11], Shik [22, 23] and Orlovskii [17, 18], who suggest that movement be analyzed in terms of *synergies* in the sense of relatively standard patterns of coordinated activity in some ensemble of muscles: the dynamics of muscles acting in concert. We shall thus view the problem of motor control in terms of the sequencing and coordination of synergies, rather than try to directly analyze the control of the vast number of degrees of freedom offered by the independent activity of all the motor units. We have, to use the language of Greene [12], to get the system "into the right ballpark", and then to tune activity within that ballpark - the dual problems of *activation* and *tuning*. It is to a model of neural circuitry for the activation and fine tuning of synergies that we now turn. We shall suggest that such circuitry is provided by the cerebellar cortex in interaction with the cerebellar nuclei and various brainstem motor nuclei. As is well-known, the only output of the cerebellar cortex is provided by the *Purkinje cells*, which provide inhibitory input to the cerebellar nuclei. The Purkinje cell has two input systems. One input is via a single *climbing fiber* which ramifies and synapses all over the Purkinje cell's dendritic tree (one climbing fiber to a Purkinje cell). The resultant synaptic interaction is so strong that, when its climbing fiber is fired, a Purkinje cell responds with a sharp burst of 4 or 5 spikes, known as the *climbing fiber response* (cfr). Many authors have thought that the "secret" of the climbing fiber is this sharp series of bursts, but we shall suggest below that the true role of the climbing fiber input is to provide the suppression

[4]) These experiments are still viewed with scepticism by many Western neuroscientists; but we believe the general pattern of these findings is correct; and that they offer a major new paradigm for the analysis of movement.

of Purkinje cell activity for as much as 100 msec which has been found (Murphy and Sabah [16]) to follow the cfr. The other input system is via the mossy fibers, which activate granule cells whose axons rise up into the layer of Purkinje cell dendrites (which are flat, with the planes of all their dendritic trees parallel to one another) to form T's, whose cross-bars run parallel to one another at right angles to the planes of the Purkinje dendritic trees. Some 200,000 parallel fibers run through a single Purkinje tree in man, with perhaps 1 in 5 synapsing as it passes through. (The mossy fibers also synapse upon Golgi cells, and the parallel fibers (granule T-bars) also synapse upon basket and stellate cells, but we shall ignore these inhibitory interneurons in our initial model, and instead study only the basic cerebellar circuit (Llinás, [15]) of mossy and climbing fibers, granule cells and Purkinje cells which is common to the cerebellar cortices of all vertebrates from frog to man.)

As a "working example" we have sought to outline cerebellar function in locomotion of the high decerebrate cat as described by Shik and Orlovskii (Shik, et al. [22, 23]). The locomotory "algorithm" has been shown to be available even in the spinal cat in both Sherrington's [21] classical work and modern (Forssberg and Grillner [10]) studies: which supports other work on the segmental programming of stepping (Szekely [24]). Thus, we must expect a purely modulatory role for the cerebellum in locomotory synergic programming. The nature of this modulation - and how it is transmitted to the musculature - is now becoming known. In particular, the red and Deiters nuclei, driven principally by the cerebellum in the decerebrate cat, are found to have activity modulated in accordance with contraction of flexors and extensors. However, this modulation is impressed upon already substantial levels of "background" activity in both nuclei, which is present whether or not the associated musculature is currently active. It is as though nuclear contributions - and, hence, cerebellar influences - are directed or "switched" into the muscles only at appropriate times. Indeed, Orlovskii [18] has provided compelling evidence that, during locomotory performances, Deiters nucleus can influence extensors (and red nucleus the flexors) only when that particular musculature is being actively employed. The existence of similar "Switching" phenomena (which appears to be a spinal process) were discussed, relative to peripheral inputs to the spinal reflexes ("die Umkehr") long ago by Sherrington, [21].

Figure 5 illustrates schematically a hierarchical control architecture capturing the features of "synergy generators" and lower-level output switching. We surmise that the locomotory algorithm (resident spinally, with reticular intervention) functions as an "executive" selecting which of several cerebellar synergy generators shall be actively employed in muscular modulation via output switching. The generators themselves (whose outputs appear in such brainstem nuclei as the red and Deiters) operate continually. Their outputs remain "tuned" to environ-

mental perturbations by feedback from lower centers and the periphery; much of this feedback is known to exert its influence via the cerebellum (Orlovskii, [17]. So, with these considerations and many others, how might it be that cerebellar synergic generators are constructed, and what are their operational properties?

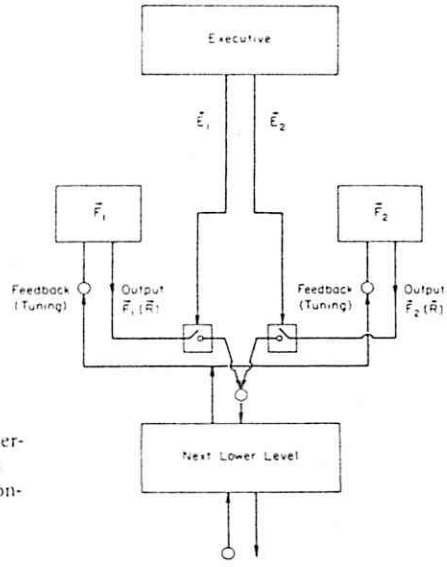

Fig. 5:
Hierarchical scheme for locomotory control: Synergic function generators (F_1 and F_2) have outputs selectively switched into lower centers, but are continuously tuned by feedback

We hypothesize a neuronal device which not only can be "programmed" to define various synergies when its output is spinally switched into the musculature, but also can temporarily "store" such programs while they are in use and tune them in accord with peripheral factors; we could term this a programmable "reflex". An architecture suited to this task is to be found in the arrangement of Figure 6, which is an anatomical template of circuitry ubiquitous in cerebellar transactions. That is, specific labels could be given to, say, the "brainstem output nucleus" (e.g., red or Deiters), "reticular nucleus" (reticularis tegmenti pontis, paramedian, etc.), and so forth. From this architecture we gather that cerebellar output is derived fundamentally from a clash - in the cerebellar nuclei - between cerebellar cortical inhibition on one hand, and explosively excitatory drive got from reticulo-cerebellar "reverberatory loops" on the other. These latter have recently been the subject of intense study (Ito, [13]; Brodal, et al., [6, 7], particularly by Tsukahara, [25], whose physiological work has demonstrated the possibility of intense reverberation following the removal of Purkunje inhibition).

The output of a cerebellar synergy generator is expressed as a spatio-temporal neuronal activity pattern in an output nucleus - or consequently a cerebellar nucleus. We hypothesize that the excitatory "kernel" of such

patterns is created through topically precise climbing fiber activity; the mechanism involves their direct cerebellar nuclear activation coupled with the suppression of target Purkinje cell activity in the cortex via the so-called "inactivation response". Once the essence of a synergic program is installed in corticonuclear interactions via climbing fiber intervention, the underlying reverberatory excitation helps to retain or "store" it, as we desired. At the same time, this activity is transmitted to the cerebellar cortex on mossy fibers, eventually altering the inhibitory pattern in the nuclear regions surrounding the active locus. The synergic program is thus "sculpted" spatially in a way dependent upon the elaborate geometry of cerebellar cortex and corticonuclear projections. Mossy inputs of various types tune the resultant program to the demands of the periphery; and the program is spinally "read-out" at intervals, as described above.

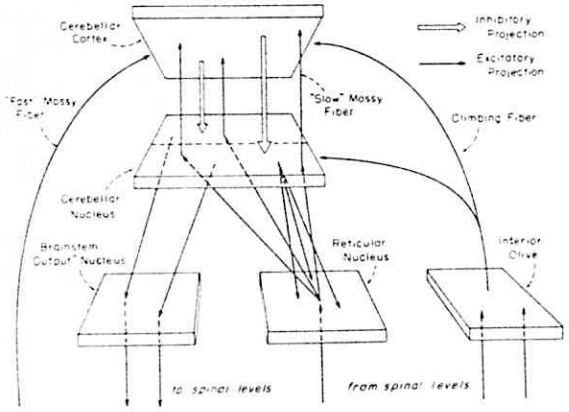

Fig. 6:
Schematic of fundamental cerebellar synergy-generating architecture

Testing of our various hypotheses has required computer simulation of the neuronal apparatus of Figure 6. We have attempted to hew closely to recent anatomical and physiological findings in this work. Figures 7 - 9 illustrate several anatomical templates so developed for specific elements of the cerebellar synergy generator (see Boylls, [4] for details); special attention has been paid the geometrical relations which have so intrigued investigators for many years, using the simplified "basic cerebellar circuit" in the cerebellar cortex.

Simulation results have corroborated our conjecture that cerebellar-related circuitry could support the short-term storage of synergic "programs" initiated (and periodically refreshed) by climbing fiber activity. Figure 10 suggests a typical nuclear activation pattern so introduced. Attention to geometrical facts has allowed us further to predict that the agonists of a cerebellar synergy will be "represented" along a sagittal strip of the cere-

bellar cortex, while its antagonists will lie orthogonally to that strip (in the mediolateral plane). Applications of this formula to the cortical topography of the anterior lobe, as developed by Voogt, [26] and Oscarsson, [19] permit us to identify particular cortical regions as associated with equally particular types of hindlimb-forelimb, flexor-extensor synergic groupings. These lead to conclusions which are experimentally testable (via lesion studies with standard techniques). This theory seeks what to organize into a coherent whole a substantial amount of what is known of cerebellum. Its distinguishing feature is that this organization emerges from a most reasonable view of how motor control is achieved. Interested readers may wish to consult Boylls, [4] for further details.

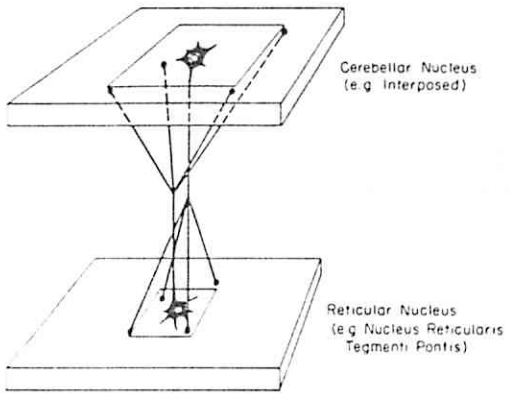

Fig. 7:
Anatomical template of cerebello-reticular reverberatory loop

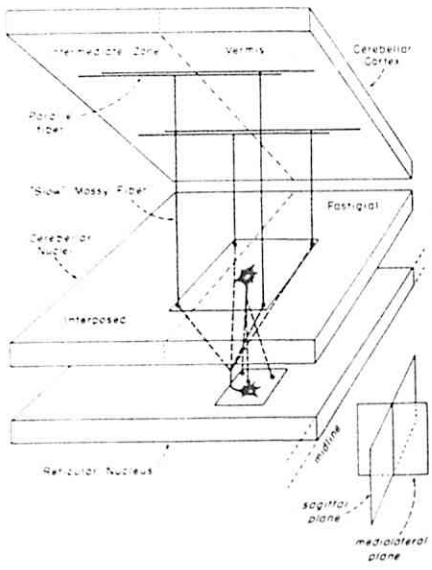

Fig. 8:
Template of "slow" mossy fiber input to cerebellar corex

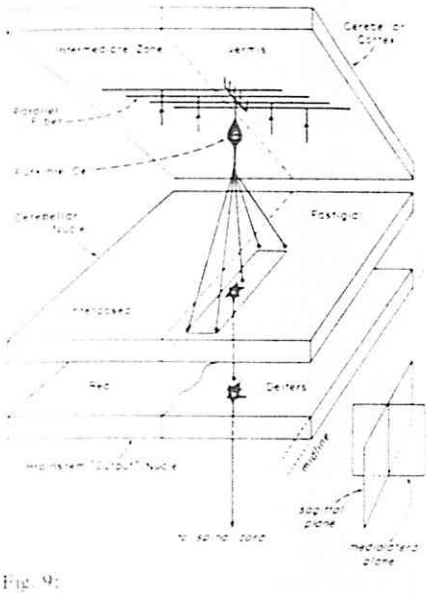

Fig. 9:
Template of cortico-nuclear projection and cerebellar outflow

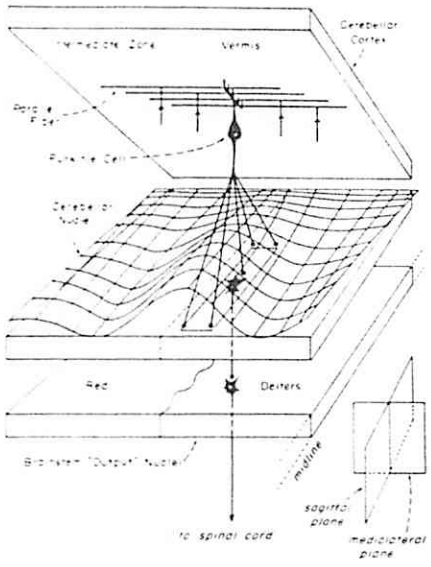

Fig. 10:
Activation pattern "stored" in cerebellar neuronal interactions via climbing fiber activity and which represents a "program" for a particular synergic function when read-out into the musculature at the spinal level

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