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on

the effects of blindness and other impairments on early development

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SOMATOSENSORY DEPRIVATION AND ITS RELATIONSHIP TO THE BLIND*

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The objective of this paper is to illustrate how a visual defect can result in deprivation to the somatosensory modality and that behavioral deficits usually attributable to the primary visual sensory deficit may, in fact, be attributable to deprivation of the somatosensory system. It is suggested that this type of confounding may also occur when other sensory channels suffer deprivation. Neurobiological and neurobehavioral principles derived from animal sensory deprivation studies are briefly summarized in order to clarify the meaning of behavioral symptoms occurring in humans who also have been deprived of sensory stimulation during early development.

SOCIAL DEPRIVATION AS SENSORY DEPRIVATION

A major conceptual dilemma characterizes a number of studies on the effects of early experiences upon development. Specifically, studies of infants separated from their mothers have been characterized as "maternal-social deprivation," "perceptual deprivation," and/or "emotional deprivation." In addition to the error of using the term "maternal" rather than "parental" there has been little effort to describe the separation experience in terms of specific sensory processes. Unfortunately, noted authorities in this area of research have used language to describe their experiments in a way which has lead to a conceptual differentiation of *social development* from *neurobiological development* and consequently, the sensory processes that are common to these two disciplines have been neglected.

The failure to conceptualize the studies of "maternal-social" deprivation and isolation rearing within a neurobiological frame-of-reference of *sensory deprivation* can be attributed in part to the orientation of H. F. Harlow [1] who stated:

The viewpoints expressed in this article are those of the author and do not necessarily reflect those of the National Institutes of Health or the U.S. Department of Health, Education and Welfare.

*The original presentation included a showing of films and slides accompanied by informal commentary. The present article is a more structured presentation with more recent data of these ideas and hypotheses that relate somatosensory functioning to blindness.

It should be recognized that this paper is primarily a theoretical and speculative venture to link a number of different issues from several disciplines within a common frame-of-reference. It is acknowledged that certain points of view have limited support from experimental data, however, it is hoped that this conceptual effort may prove heuristic in developing new research approaches to some basic scientific questions that have unusual relevance for human development.

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"The most extreme deprivation condition we have studied is total social isolation (not sensory isolation, only social isolation). The animal has no mother figure of any kind; also it has no opportunity to see, hear, or contact any other animal or even a human being, for during the isolation period it is fed and tested by remote control. Thus these animals have no chance of developing any affectional tie." ([1], p. 154.)

The distinction between social and sensory deprivation was also emphasized by Casler [2] in his review of the effects of "maternal" deprivation where he stated:

"One may agree that it is social rather than perceptual or sensory deprivation that is involved in these cases." ([2], p. 23.)

This point of view, or conceptual schema, has received support from another noted authority in "maternal-social" deprivation, namely, R. A. Spitz [3], who states:

"In conclusion, I call the reader's attention to the terms I have used in dealing with this subject. I have spoken advisedly of *affective* (emotional) deprivation. In recent years a great deal of illuminating and interesting work has been done with animals and humans on the effects of sensory deprivation. . . it should be realized that sensory deprivation and emotional deprivation are not interchangeable concepts. Granted, in the present state of the art, it is practically impossible to inflict the one without involving the other . . . accordingly, I believe that further experimentation and study will be required before we can delineate the nature of the two forms of deprivation and isolate their effects from each other." ([3], pp. 281-284.)

This disassociation of affective (emotional) processes from sensory processes is also evident from Spitz's discussion on infant perception. He states:

"Particularly during the first six months of life, and, to a certain extent even later, the perceptual system, the sensorium of the infant is in a state of transition. It shifts gradually from what I have called coenesthetic reception toward diacritic perception. The sensorium plays a minimal role in coenesthetic reception; instead, perception takes place on the level of deep sensibility and in terms of totalities, in an all-or-none fashion. . . . Furthermore, perception through the sensorium (diacritic perception) does not yet operate; this absence of diacritic perception intensifies coenesthetic 'reception,' since only coenesthetic signals will be received, experienced and become effective." ([3], pp. 140-41.)

Spitz's distinction between coenesthetic reception and diacritic perception is made even more clear by the following:

"It is my opinion, however, that a large proportion of the pathways involved belong to a system of 'sensing' basically different from the system of perception that operates at a later age and with which we are familiar. I have discussed the nature of these two systems and the differences between them elsewhere (Spitz, 1945, [4]) and have called the one present at birth the *coenesthetic* organization. Here, sensing is extensive, primarily visceral, centered in the autonomic nervous system, and manifests itself in the form of emotions. Accordingly, I prefer to speak of this form of 'perception,' which differs so fundamentally from sensory perception, as reception. It is an all-or-none phenomenon operating as a binary system.

In contrast to this stands the later development of what I have called *diacritic organization* where perception takes place through peripheral sense organs and is localized, circumscribed, and intensive; its centers are in the cortex, its manifestations are cognitive processes, among them the conscious thought processes." ([3], p. 44.) (See also Spitz, 1945 and 1946 [4-6].)

It is clear that Spitz views the emotional (affective) processes as primarily a visceral process localized in the autonomic nervous system and unrelated to sensory mechanisms and higher brain processes. This unusual psychoanalytic interpretation of the neurobiology of emotional processes which denies the primacy of sensory mechanisms and higher brain processes in these functions can have only contributed to a misdirection of scientific thought and research on the neurobiology of emotional ("maternal-social") deprivation phenomena.

The continuing distinction between social, emotional, and sensory processes in "maternal" (parental) deprivation phenomena has contributed to a lack of clarification of mediating mechanisms which is reflected in Yarrow's [7] summary of the effects of maternal-social deprivation:

> "We can only speculate on the processes through which the mother comes to acquire special meaning to the child." ([7] p 486.)

This writer advocates a sensory-neuropsychological orientation to the study of parental deprivation and isolation rearing phenomena. Studies reviewed below and elsewhere have led this writer to the conclusion that the somatosensory system (cutaneous, visceral, proprioceptive and vestibular afferents) is the sensory modality that mediate the abnormalities consequent to isolation rearing; and that deprivation to the other sensory modalities during the formative periods of development will not lead to abnormal social-emotional behaviors provided somatosensory stimulation is present.

From the above it would seem appropriate to suggest that it is timely to move from speculation "on the processes through which the mother comes to acquire special meaning to the child" to basic research on the sensory processes in the parent-child relationship.

It is recognized that many investigators have acknowledged the importance, if not the primacy, of physical contact in isolation rearing studies and the Harlows' emphasis on "contact comfort" attests to this recognition [8-10]. Mitchell [11] summarizes this point of view succinctly:

> "Rearing in *social* isolation produces severe behavioral pathology in all primates. The most important source of stimulation that is absent in such a rearing condition is physical contact from another animal involving a complex combination of skin or fur contact clinging, movement, oral contact, and warmth." ([11] p. 243.)

Unfortunately, the phenomena of "physical contact" in primate behavioral studies has been restricted to the conceptualizations of "social" behavior and thus has distracted from a *neurobiological* interpretation which could have led to systematic studies of sensory processes and brain functioning which tragically are still lacking today in the isolation reared primate.

The theoretical orientation outlined above does not deny the importance and relevance of the other sensory modalities in the normal development of social relationships. It is the conviction of this writer, however, that the affective/emotional component of social behavior is mediated by the somatosensory system (near receptors) and not by the visual and auditory systems. These latter modalities (distance receptors) do, however, acquire the capability of triggering emotional behavior. This is accomplished through a complex developmental conditional learning process which involves sensory integrative mechanisms. Whether the sight or sound of the parent (conditional stimuli) triggers positive or negative emotional responses in the child is dependent upon the history of the quality of parent/child somatosensory relationships, namely, pleasure or pain (unconditional stimuli).

It is the belief of this writer that the "psychological" or "psychosocial" environments must be understood in terms of their transformation into sensory experiences that are perceived by the organism. The alternative is to invoke extrasensory perception or non-sensory (spiritual) experiences in the development of social relationships and behaviors.

EFFECTS OF EARLY SENSORY DEPRIVATION

A number of studies have clearly documented that sensory deprivation during formative periods of development can result in failure of the deprived sensory system to develop and function normally; in such cases neuroanatomical, neuroelectrical, and neurochemical abnormalities have been described from the level of sensory receptors through sensory relay nuclei to the cerebral cortex [12-43; 50]. Conversely, enriched sensory stimulation during the formative periods of development can result in increased growth of the sensory system that has been enriched [33-42].

Special attention is drawn to the study of Volkmar and Greenough [38] since they demonstrated significant differences in higher order dendritic branching in Layer V pyramidal neurons in visual cortex between rats reared in groups in a complex environment and litter mates *reared in pairs in normal laboratory cages.* Differences were also found with litter mates reared in single cages (isolation condition). Further, these animals were not assigned to experimental treatments until they were 22 to 25 days of age. Two important conclusions can be drawn from this study:

1. It is clear that an enriched environment when compared to a *normal social control environment* is sufficient to produce significant structural alteration of brain cells. It does not require extreme sensory deprivation to produce these effects upon the brain;

2. These animals did not require differential rearing environments *from birth* to produce structural alterations in the brain. Consequently, neither severe alterations in the sensory environment nor deprivation in very early life are required to produce structural alteration of brain cells.

Greenough, et al. [39] have extended the findings of the above study by demonstrating that rats reared in *complex environments* had significantly more basal dendritic branches in the temporal cortex than those from *littermates reared socially or in isolation.* No significant differences due to rearing were detected in the frontal cortex. They conclude that the effects of environmental complexity on dendritic branching are not restricted to those previously seen in the visual cortex and that the specificity of effects rules out generalized influences that could be expected from hormonal or nutritional differences. Greenough and Volkmar [40] are more explicit on these interpretation of differential rearing effects.

> "The results suggest that the differences in dendritic branching seen after rearing in complex environments are a function of this complexity, rather than of visual deprivation, and indicate a mechanism whereby the effects of the early environment may be expressed in later behavior."([40], p. 491.)

A similar point of view has been expressed by Rosenzweig, et al. [34].

These findings must raise questions about the effects of moderate sensory variations upon brain development in higher mammals, specifically, primates. It can be concluded that the immature mammalian brain at birth and throughout its period of immaturity is dependent upon sensory stimulation for its normal growth, development and function. Sensory stimulation is like a nutrient and just as malnutrition adversely affects the developing brain so does sensory deprivation [35, 36].

Studies of sensory deprivation have revealed not only the above developmental neurobiological principle but also a developmental neurobehavioral principle. Specifically, animals that have been deprived of sensory stimulation during the formative periods of development will as adults engage in stimulus-seeking behaviors, as if to maximize the sensory stimulation that they were deprived of during early development. This behavioral phenomenon has been previously referred to as a "neurophysiological addiction" for sensory stimulation [37]. Based upon Riesen's [13; 15] observations and suggestions that Cannon's Law of Denervation Supersensitivity [44, 45] may be related to environmental sensory deprivation effects, this writer has proposed that denervation supersensitivity is

the neurophysiological mechanism underlying stimulus-seeking behaviors consequent to early sensory deprivation, i.e., partial functional deafferentation [44-47]; Sharpless' review on denervation supersensitivity and its relationships to behavior should also be consulted [48].

Lindsley, et al. [49] have reported that visually deprived monkeys will bar press over a thousand times per hour to receive light stimulation and that they show enhanced electrocortical responsivity to lights *on* and *off*. Similarly, Arnott's* study described elsewhere [37] also reported that cats born in darkness and remaining in a totally deprived visual environment for one year would bar press continuously for light stimulation from eight to twenty hours per day or hold the bar down permanently until fatigue intervened. These animals were tested after being deprived of food for 24 hours and had a choice to bar press for food or light. The studies of Butler [51, 52] and Butler and Alexander [53] also document the increase in visual behaviors subsequent to visual deprivation.

Similar stimulus-seeking behaviors are well substantiated for the somatosensory system. Melzack and Thompson [54] and Melzack and Scott [55] have reported the hyperexcitability behavior of dogs reared in isolation where body contact, touching and movement stimulation from other animals and humans were virtually nonexistent. Riesen [56] has shown that normal environmental stimulation after severe early sensory deprivation of isolation rearing can result in (a) hyperexcitability, (b) hypoexcitability, (c) increased susceptibility to convulsive disorder, and (d) localized motor dysfunction. The instance of hypoexcitability occurred when an animal developed severe response inhibition which resulted in death. The studies of the Harlows' [8-10] and their many students and colleagues have amply documented the self clutching, rocking behaviors, thumb, toe, and penis sucking and cage "swirling" in isolation-reared monkeys. This writer has interpreted these behaviors as a consequence of somatosensory deprivation which induces dysfunctional somesthetic and vestibular-cerebellar activity. Cannon's Law of Denervation Supersensitivity might be a relevant explanatory neural mechanism for these dysfunctions. Thus, stimulus-seeking behaviors have been documented in another sensory modality (the somatosensory) as a consequence of deprivation in that sensory system and additional examples have been given elsewhere [35, 36; 57, 58].

*Personal communication.

VESTIBULAR-CEREBELLAR MECHANISMS IN ROCKING AND HYPERACTIVITY SYNDROMES

It would appear helpful to illustrate how Cannon's Law of Denervation Supersensitivity might account for the perseverative rocking behaviors of isolation reared monkeys and institutionalized children. Under these social-environmental conditions it is proposed that neither infant monkeys nor children receive sufficient body contact and movement by being picked up, handled, and carried, as evidenced by their movement stereotypes. Consequently, both somesthetic (touch) and vestibular (movement) sensory receptors and their projections to other brain structures do not receive sufficient sensory stimulation for normal development and function. If Cannon's Law of Denervation Supersensitivity applies to the CNS, then the sensory projection fields in the cerebral and cerebellar cortex, and other neural structures receiving somatosensory afferents would become supersensitive, hyperactive, and hyperreactive in function. It is suggested that these conditions may result in an excessive output from cerebellar cortex through the Purkinje axons which are exclusively inhibitory in nature. These cerebellar cortical efferents impinge directly upon a variety of brain structures including the deep cerebellar nuclei [59]. The cerebellar cortical efferents could directly inhibit those brain structures involved in the transmission of neural impulses associated with rocking. Consequently, the rocking behaviors would continue since the appropriate brain structure(s) would not receive the sensory stimulation generated by the rocking behaviors and the presumed "instruction" to "stop rocking." In other words, the rocking behaviors are nonfunctional and so rocking continues.

If the above theoretical neuronal model to account for perseverative rocking and movement behaviors has any validity, then the following procedures should bring about termination of the rocking and movement stereotypes. One strategy involved the neurosurgical removal of the presumed abnormal cerebellar cortex (cerebellar decortication) which would eliminate the cerebellar cortical inhibition of the vestibular nuclei and other brain structures to which it projects. Berman [60, 61], and Berman, et al. [62] performed a series of cerebellar decortications in rocking isolation-reared infant monkeys and violent isolation-reared adult monkeys. In the few animals that were operated upon, a dramatic reduction in rocking time occurred without observable motor impairments except for one animal (the nature of this specific deficit has yet to be determined). Also, violent behaviors in adult animals receiving a midline vermis decortication were eliminated. The adult animals receiving a lateral hemispheric decortication continued to behave violently [62]. These preliminary neurosurgical studies provide some support for the role of the cerebellum in mediating rocking behaviors and impulsive, violent-aggressive behaviors. These findings illustrate how little we know about the neurophysiological mechanisms that mediate perserverative rocking behaviors.

The second procedure involved intense vestibular stimulation of severely hyperactive mentally retarded children who engaged in perserverative circling and pacing behaviors. These children were the patients of Dr. William Nyhan and were diagnosed as having the Delange syndrome. Given the above outline of the principles underlying stimulus-seeking behaviors and the corresponding presumption of central neural inhibition of sensory impulses associated with that activity, it was felt that sufficiently intense sensory stimulation could "override" the central inhibition of sensory input. Under these conditions, the sensory impulses would be transmitted to the cerebellum and other brain structures and a decrease of the circling-movement stereotypes would be expected. Accordingly, children were placed in an ordinary office chair and whirled as fast as possible. The two children who received less than two minutes of this intense rotational stimulation were completely quieted and one child yawned and became sleepy. It became clear that stimulant drugs were not necessary to control hyperactivity in these children and suggests that other hyperactive children may also be similarly benefited. It is of interest that one child showed no post-rotatory nystagmus and the other child showed a minimal and very short post-rotatory nystagmus. This finding may reflect neuropathology of vestibular cerebellar mechanisms and other brain structures involved in nystagmus activity in these children. It also suggests that neuropsychological nystagmus assessment in hyperactive children might prove to be diagnostically useful, as it has proven to be elsewhere [63]. This study has been reported previously by Nyhan [64] and indicates the potential of sensory stimulation techniques in the treatment of the sensory-handicapped or sensory-dysfunctional child. These findings are consistent with the theoretical concept of central neural inhibition of sensory input as the mediating mechanism in some movement stereotypes. It is recognized that rigorous and systematic evaluation of this neural-behavioral model is required.

The studies of Mason [65] and Berkson [66, 67] also document that movement stereotypes, and rocking stereotypes in particular, are a consequence of somatosensory-vestibular deprivation. Mason reared isolated infant monkeys on a moving surrogate mother and demonstrated that these animals did not develop rocking behaviors, as did monkeys reared under conditions that were identical except for the fact that the surrogate mother did not move. This study was dramatically illustrated in the film "Rock-A-Bye Baby" [68]. The studies of Berkson reported in this conference and elsewhere [66, 67] indicate that infant monkeys blinded at birth but reared with their mothers and peers do not develop abnormal rocking behaviors. These results support the point of view that visual sensory deprivation alone does not lead to the development of rocking behaviors. This conclusion is also supported by the findings of Adelson and Fraiberg reported in this conference and elsewhere [69, 70] that congenitally blind children do not develop emotional

movement stereotypes provided that they receive sufficient somatosensory stimulation. These findings are also vividly portrayed in the film "Rock-A-Bye Baby" [68]. Riesen [14] has provided additional data that emotional disturbances result from somatosensory deprivation and not from visual deprivation in his studies on the effects of isolation rearing in cats. He states:

> "Multisensory deprivation has a much more pronounced effect on the motivational systems of our animals than has the restriction of vision alone. It was not difficult to keep our animals healthy and emotionally capable of learning new behavior if we made sure that they were not deprived in more than the visual sensory area. They were socially dependent upon litter mates and later upon cage mates and human contacts, as mediated tactually and through hearing. A number of animals with which our precautions were insufficient, including both cats and monkeys, were permitted a greater degree of isolation and monotony. These animals proved to be untestable. They were typically either withdrawn or hyperactive to the point of uncontrollability when brought into a complex environment. These observations are not surprising when viewed in relation to the work of the Harlows." (p. 297.)

IMMOBILIZATION AS VESTIBULAR AND CUTANEOUS DEPRIVATION

Additional clinical data on the effects of somatosensory deprivation on children have been provided by Freedman [71], and Friedman et al. [72]. They demonstrated that immobilization of infants and children with plaster casts, traction, Dennis-Brown splints, and other restraint procedures resulted in emotional disturbances of depression mixed with hyperactivity and outbursts of violence. Sibinga and Friedman [73] also reported delays in language development in children with a history of immobilization.

Sibinga has also observed gastric ulcerations in children who have been extensively immobilized subsequent to severe burn injuries or CNS surgery and that a lesser degree of immobilization frequently resulted in chronic idiopathic diarrhea. Sibinga* has suggested that immobilization or extreme inactivity associated with burn injuries or CNS surgery may be an important contributing factor to the development of Curlin's and Cushing's ulcer. It should be noted that Cushing stressed the importance of nervous influences in the pathogenesis of ulcers and drew attention to the relatively high incidence of acute gastric ulcers after intracranial operations some of which involved cerebellar surgery [74]. In this context the finding by Wolfe [75] that lesions in the posterior vermis of the cat cerebellum produces gastric ulcerations is notable.

Ader [76] has summarized an extensive body of literature that documents the induction of ulcers in rodents by immobilization where the development of acute lesions is related to the degree of restraint and the duration of restraint. Although it is not possible to review, herein, what Ader [76] has already reviewed it seems cogent to mention a few of the more relevant findings from immoblization studies as they relate to somatosensory-cerebellar mechanisms. Specifically:

1. Animals that were immobilized during their activity period developed ulcers whereas animals who were immobilized during their quiescent period of activity did not develop ulcers [p. 17].
2. The degree of ulceration is greater in rats isolated in individual cages than rats who were housed in groups (Martindale, et al., [77]; Essman and Frisone [78]).
3. Differential housing effects were masked by a stress (change in housing conditions) 24 hours prior to immobilization. Incidence of ulcers was increased in the mice that experienced a change in housing (Essman, et al. [79]).
4. Rats who were group housed from weaning until approximately 80 days of age and then subjected

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*Personal communication.

to 48 hours of immobilization were more susceptible to ulcers than rats housed individually from weaning (Stern, et al. [80]).

5. Prematurely weaned rats (15 days of age) were more susceptible to ulcers than animals weaned at 21 days (Ader, et al. [81]).

6. *Individually-housed* rats who were *handled* for 10 minutes daily for three weeks beginning at weaning (21 days) developed less hemorrhagic damage to the gastrointestinal system than did nonhandled controls when subjected to 48 hours of immobilization at maturity (Weininger, [82]).

7. *Group-housed* rats that had been *handled* 5 or 10 minutes per day were more resistant to gastric lesions than a non-handled group (Winokur, et al., [83]).

8. Rats subjected to *daily handling* throughout the pre-weaning period developed less gastric ulcers than rats subjected to electric shock stimulation after 18 hours of immobilization. This effect held for both group-housed and individually-housed animals (Ader, [84]).

9. Rats subjected to a stress of a shocking cage, and who had entered the "stage of exhaustion from stress" which was characterized by drowsiness, passively rolling on the cage floor, and finally acute rigidity were invariably found to have gastric ulcers (Levrat and Lambert, [85]).

10. Lesions which are experimentally produced in the glandular portion of the rat's stomach are microscopically and histologically indistinguishable regardless of the specific stimulus conditions used to induce the lesion. This suggests that there may be some common pathway or mediating mechanism responsible for the development of such lesions [76] (p. 33).

The above findings leave little doubt that immobilization and isolation procedures (which by definition involve vestibular and cutaneous deprivation) result in gastric ulcerations. The observations that individually housed animals are generally more susceptible to gastric lesions than group housed animals (differential somatosensory stimulation) and that *handling* confers a degree of protection from immobilization ulcers provides further evidence of a primary role of somatosensory deprivation induced by immobilization in gastric ulceration. These findings in the context of Wolfe's report that cerebellar lesions result in gastric ulceration provides compelling evidence for a possible cerebellar mediation of immobilization ulcers. Unfortunately, a direct test of this hypothesis has yet to be conducted where cerebellar studies in immobilized animals are clearly indicated.

Relevant to this suggestion are the findings of Eichelman and Thoa [86] that rats immobilized for 2 hours daily over a period of four weeks exhibited elevated levels of shock-induced fighting immediately after immobilization and after four weeks of recovery. Brain tyrosine hydroxylase (the rate-limiting enzyme in the synthesis of norepinehprine-NE) in the hypothalamus was significantly ($p < 0.01$) elevated in both the stressed and recovered groups. Hypothalamic NE was also elevated in pooled samples from the stressed rats when compared with controls. Unfortunately, the cerebellum was not specifically analyzed.

The vestibular-cerebellar hypothesis would also predict that gentle rocking during the immobilization period should act like "handling" in reducing gastric ulcers. Additionally, it is well known that the floccular-nodular lobe of the cerebellum mediates nausea and other symptoms of motion sickness since ablation of that structure prevents the development of motion sickness. The role of the cerebellum in regulating gastrointestinal activity is a supportable hypothesis.

The findings of Schapiro, et al. [87] are related to the above issues. They reported that auditory and vestibular deprivation produced by bilateral labyrinthectomy resulted in a marked inhibition of the acid output and pepsin concentration in canines consequent to insulin hypoglycemia and histamine injections. Although no behavioral studies were carried out they noted that these dogs became friendlier as the study

progressed even though they were individually caged. Unfortunately, no information was given on how much handling these dogs received in the course of this study. It is known that petting and handling reduces irritability and heart rate in dogs [88]. It should also be noted that Schapiro, et al. [87] reported similar gastric secretory responses in canines subjected to bilateral ablation of the eyes and that these effects were not significantly different from bilateral labyrinthectomy. They concluded that "interference with any of the sensory modalities was capable of almost complete suppression of gastric secretion (p. 518). In a review of relevant literature, Schapiro, et al. [87] cited the studies of: (a) Ebstein [89] in which damage to the labyrinths and the internal ear of rabbits resulted in gastric erosions; (b) Le Heux and Kleing [90] in which labyrinth extirpation in cats resulted in a decreased barium transit time; and (c) that deafness in man is associated with diarrhea, constipation and a lack of tone in the intestinal wall. These authors also comment that they have never seen a deaf peptic ulcer patient nor found any literature reports dealing with deaf peptic ulcer patients [87, 91].

Schapiro's et al. study is cited to illustrate that sensory deprivation does have an effect upon gastric function, although evidence for a specific modality effect is lacking in this report. Additionally, the opposite effects between complete sensory deafferentation (bilateral labyrinthectomy and enucleation) and partial functional deafferentation (immobilization) upon gastric function (hypo and hyper gastric activity, respectively) needs clarification. It is beyond the scope of this paper to develop such a clarification, although it can be suggested that bilateral labyrinthectomy which would destroy cells in the floccular-nodular lobe through the mechanism of transneuronal degeneration could mimic, in part, a floccular-nodular lobe ablation, and thus result in a common effect, namely, hypogastric activity. Since the visual system is integrated with vestibular-cerebellar processes, it is natural to raise questions concerning the effect of absence of visual input upon vestibular-cerebellar functioning [92-103].

The recent demonstration of visual afferents into the flocculus of the cerebellum [98, 99], where lesions of the flocculus markedly reduced visual suppression of caloric nystagmus, optokinetic after-nystagmus, positional alcohol nystagmus, and spontaneous nystagmus [104] suggest that nystagmus activity in the blind might be profitably studied. It would also be of interest to determine whether there is altered gastro-intestinal activity and susceptibility to motion sickness in the blind as compared to the sighted. Whether the absence of visual input into the flocculus of the cerebellum would have consequences for any of these functions is not known. It would seem worthwhile to explore the possibility of whether hypogastric activity consequent to enucleation might involve visual floccular-nodular mechanisms. Such studies would have to carefully distinguish between the nodular and floccular structures of the vestibular cerebellum since Cohen and Takemori [104] found that nodulus lesions did not affect visual suppression of nystagmus activity (see Figs. 19 and 22). Dow [100] in his discussion of the Cohen and Takemori study has emphasized the regulatory function of the cerebellum in its links to vision. It is this regulatory function of the cerebellum which is being stressed in this paper and it will be returned to later.

If it is reasonable to assume that immobilization processes adversely affect cerebellar functioning, then it is tempting to speculate whether other symptoms reported to be associated with immobilization might also involve cerebellar mechanisms. Thus, the finding of Sibinga and Friedman [73] reported above that delays in language development were found in children with a history of immobilization naturally raises an inquiry as to the possible role of the cerebellum in language and reading processes. The frequent association of hyperactivity syndromes with learning, reading, and language disorders lends support to this line of speculation. It is hardly necessary to point out that speech and language are highly developed motor skills requiring exquisite control and regulation

of motor sequences. Needless to say, regulation of motor sequences is one of the primary functions of the cerebellum. The suggestion that the cerebellum may be involved in language and reading processes and in their disorders is consistent with theoretical models of the cerebellum developed by Marr [105]; Blomfield and Marr [106] and Albus [107, 108] to account for memory processes and the perception of patterns. Based upon the known anatomy and physiology of the cerebellum, Albus [107, 108] has postulated that the cerebellum is structurally and functionally equivalent to a modification of the classical perception pattern-classification device. He has suggested that "the mossy-fiber granule cell Golgi cell input network performs an expansion recording that enhances the pattern-discrimination capacity and learning speed of the cerebellar Purkinje response cells" (p. 25). He has further suggested that the parallel fiber synapses of the dendritic spines of Purkinje cells, basket cells, and stellate cells are all specifically variable in response to climbing fiber activity and that variability is the mechanism of pattern storage. Albus also argues that in order for the learning process to be stable, pattern storage must be accomplished principally by weakening synaptic weights rather than by strengthening them. It is recognized that the suggestion that the cerebellum may be involved in language and reading processes requires rigorous evaluation and that the data to support this hypothesis is tenuous and unsatisfactory. Although a more detailed outline of cerebellar anatomy and function is presented later, it should be noted that the only two neural inputs into the cerebellum are the climbing fiber, and mossy fiber systems and that the only output from cerebellar *cortex* is the Purkinje axon system which is exclusively inhibitory in function. The only other output from the cerebellum. . . the deep nuclei. . . is exclusively excitatory in function.

The above studies suggest that the problems of the blind are not necessarily confined to the visual sensory system and that many problems of the visually deprived may well involve concomitant somatosensory deprivation and associated vestibular-cerebellar mechanisms. For example, Fraiberg* has noted that it is not infrequent for blind infants to sleep or remain untouched and unhandled for as much as 14 to 18 hours per day. It is not surprising that special efforts must be made to stimulate the other sensory systems, particularly the somatosensory system in blind infants and children.

It would be remiss not to mention in this section on immobilization the findings of Zubek et al. [110] who immobilized adult faculty subjects for periods up to 24 hours (mean = 12.9 hours; S.D. = 6.6 hours). Only 8 of their 40 subjects were able to endure immobilization for the prescribed 24 hours. Vision and hearing was not interfered with and social interaction with the experimentors was maintained. Head movements were prevented with a head-holder. These investigators reported that 85 percent found immobilization stressful and that 75 percent stated they would not repeat the experience in a week's time. The severe restriction of a kinesthetic (proprioceptive and vestibular) activity *alone* resulted in their showing more "intellectual inefficiency, bizarre thoughts, exaggerated emotional reactions, time distortions, changes in body image, unusual bodily sensations and various physical discomforts, than did the recumbent controls" (p. 128). Specifically, subjects felt "that some part of their body was disconnected or did not belong to the rest of the body, that they were melting or merging into their surroundings, and that at times they felt like a different person." (p. 126). Reports included "whole body floating or revolving in space, arms or legs rising, whole body feels as heavy 'as a ton of bricks'; and various distortions of body properties such as one limb being much shorter than another" (p. 126). Psychosomatic complaints were also frequent and included "periodic aches and pains, numbness, *dizziness*, physical discomfort, chills, perspiration, weakness, strong desire to scratch parts of the body and difficulty in sleeping. These physical symptoms, together with the emotional changes reported earlier, were responsible for almost all of the early terminations of the experiments prior to the prescribed

*Personal communication.

24 hours" (pp. 126-7). Additional data are summarized elsewhere [111, 112].

It is apparent from the above that severe somatosensory deprivation in *adults* for less than an average of 13 continuous hours has profound effects upon the integrity of intellective, emotional, and somatic functions. Disintegration of integrative functioning would seem to aptly describe the above effects of somatosensory deprivation. It would seem appropriate to inquire as to the effects of "insufficient" somatosensory stimulation during critical periods of development, particularly upon maturation of integrative functioning. What constitutes "sufficient" or "insufficient" somatosensory stimulation for the developing infant appears to be an issue of extraordinary importance and major research efforts should be directed to that question.

SENSORY SUBSTITUTION

The use of alternate sensory systems, particularly, the somatosensory, in developing "visual perceptions" in the blind is described by Bach-y-Rita in this publication. An important element in the ability of the blind to perceive objects from pattern stimulation of the skin is the subject's control of the video camera which provides the image that is converted to tactile stimuli. Thus, somatosensory, specifically, proprioceptive cues become critical in "tactile-visual" perceptions, particularly when these "perceptions" are externalized in space and not referenced to the specific skin surface being stimulated and where "transfer" of these perceptions occur, i.e., it makes little difference (within limits) what skin surface is stimulated. Bach-y-Rita elaborates on these issues in his paper here and elsewhere [113]. These observations assume greater significance in the context of the findings of Held and Hein [114]; Hein and Held [115]; Held [116, 117], and Held and Bauer [118]. These investigators have provided strong support for the role of motor functions in perception. For example, animals reared under conditions which deprived them of visual information of limb movement were characterized by defects in visually guided behavior. Similar defects were also found when animals were passively moved around in an environment that would otherwise permit the development of normal vision. Thus, the relative role of *active movement* versus *passive movement* becomes critical in certain issues of visual perception. It is of more than passing interest that the role of movement in the "tactile-visual" perception of the blind is as relevant for them as it is for the "normally" sighted.

Given the above it seems natural to speculate on the possible role of the cerebellum in sensory substitution phenomena. Snider [101, 119] has reviewed the role of the cerebellum in regulating afferent input and Cangiano, et al. [120] have shown that the excitability of the fast conducting cutaneous afferents can be increased by 180 to 200 percent when the contralateral fastigial nucleus in the cat cerebellum is repetitively stimulated. It is beyond the scope of this paper to further elaborate on the neural mechanisms of sensory substitution and these issues have been treated elsewhere (Bach-y-Rita [113]; Sterling, et al. [121].

Additional evidence that somatosensory experiences are crucial for the blind infant has been provided by Adelson and Fraiberg in this publication where they report that *delays* in mobility and locomotion in the blind infant could be decreased when auditory-*tactile* hand experiences were provided so that sound alone could serve as an effective cue for forward progression. This becomes particularly important since these authors also point out that sound does not begin to function as a cue to the presence of an out-of-reach object until late in the first year. Given this limitation of auditory cues it would seem desirable to utilize more extensively somatosensory cues and olfactory cues in the localization of objects in space. Thus, different objects could have different "smells" (perfume, spices, etc.) to facilitate the blind child's localization of the objects in space. Since visual and auditory sensory functions are developed later than other sensory functions [122, 123] it would seem possible to train the blind infant in spatial-temporal perceptions much earlier with additional sensory cues

rather than with auditory cues alone. The combination of olfactory, somatosensory (tactile and vestibular) with auditory cues might facilitate the development of intrasensory organization and functions in the blind.

It should be emphasized that the blind infant's inability to reach out to some stimulus object is not just a consequence of being unable to see the object. Helbrugge's study, illustrated in the film "Rock-A-Bye Baby," demonstrated that a sighted institutionalized child could not reach out and localize a rattle at six months of age. This child did not smile or show any signs of positive affect. This was corrected after a one-to-one maternal caretaker relationship was established. It was also notable that the lack of emotional expression of infants playing and responding to objects in Selma Fraiberg's film was associated with a failure to localize objects in space at 10 months of age. At 11 months of age the child crawled across the room and *smiled* when he retrieved the object. It is possible that the "smiling" response associated with object retrieval in these infants might reflect a behavioral sign of "meaning acquisition." Whatever the preferred interpretation for these events, it should be recognized that affective-emotional and psychomotor processes (somatosensory functioning) play an all-important role in intellective-cognitive development of all infants and that cognition is pleasurable [124].

In this context it is relevant to emphasize the findings of Adleson and Fraiberg in this report and elsewhere [69, 70] that *postural* test items which reflect neuromuscular maturation in control of head and trunk are not significantly different between the blind and sighted but that *mobility* items which normally follow each postural achievement were considerably *delayed* in the blind. It was also noted that the onset of self-initiated mobility was related to the child's ability to reach out and attain an object presented by sound cue alone. This phenomenon does not occur until the last quarter of the first year. It should also be noted that the cerebellum has a major role in the *initiation* of movement [125, 126].

INSTITUTIONALIZATION AS SENSORY DEPRIVATION

It would seem helpful, however, to pursue further other factors that contribute to psychomotor retardation. The findings of Helbrugge in "Rock-A-Bye Baby" have been cited previously. It is well established that institutionalization and orphanage rearing of *sighted* children results in low developmental quotients and delays in the appearance of locomotor abilities [3, 4; 6, 7; 127-136]. It is particularly relevant to note the rearing conditions in the Paraskevopoulos and Hunt [132] study which included:

a. an orphanage with an infant-caretaker ratio of 10:1,

b. an orphanage with an infant-caretaker ratio of 3:1, and

c. traditional home family rearing.

Marked and significant differences in development were obtained between these groups with the greater deficits occurring in the infant-caretaker ratio of 10:1. Substantial differences in somatosensory stimulation (touching, holding, and carrying) may have characterized these groups.

Unfortunately, quantitative data on the extent and duration of specific forms of sensory stimulation are lacking in this study and in most studies of this kind. It is usually presumed that a lower infant-caretaker ratio will result in increased physical contact and handling between infants and caretakers. This assumption, however, is not always warranted and systematic quantitative data on the specific nature of sensory stimulation should be a requisite for future studies of this kind.

Dennis [136] describes the profound deprivation of sensory stimulation in his study, *Children of the Creche*. He noted that "babies from birth to about one year spent most of their time in bassinets (Fig. 19). During most of this year they were fed by bottle in their cribs, and were almost never taken from their cribs except for daily bathing and change of clothing" (p. 13). In addition there was profound cognitive (visual and auditory) deprivation.

The infant-caretaker ratio was about 10:1 and most of the caretakers had themselves been reared in the Creche as foundlings during their first six years. The mean caretaker IQ was 57. Dennis [136] also described the paucity of language where there was little verbal communication between child and caretaker and virtually none between the children. *Children of the Creche* must be read to appreciate the degree of experiential deprivation imposed upon the infants and children. Dennis [136] summarizes: "It seems correct to say that there was no respect in which the experiences, either sensory or motor, of the Creche foundlings were equal to those of even the poorest family-reared child in Beirut" (p. 23).

The consequences of the above deprivation were severe, e.g., no foundling under one year of age could sit alone, or creep, or scoot and at two years of age many could not walk. At one year of age both boys and girls had a mean behavioral quotient of about 50 which they retained as long as they remained in the Creche. Legalization of adoption permitted them to assess the effects of a foster home upon development. They reported that Creche foundlings adopted *within* the first two years of life attained an IQ of approximately 100 at age four. Children who were adopted after the age of two showed increases in their test scores but retained the absolute deficiency in mental age which they had when they left the Creche. Dennis [136] concludes that intervention before two years can reverse environmental retardation whereas intervention after two years does not reverse the impairment which he considers to be relatively permanent. Specifically, "those adopted between ages 1-0 and 1-11 had gained more in IQ in approximately two and one half years than those adopted one year later had gained in almost seven years" (p. 90). He suggested that age two may be a critical period for complete recovery from the effects of experiential deprivation upon intelligence.

It is of interest that Geber [137] found that African infants appear to lose their accelerated developmental standing if they continue to be raised in the traditional environment after two years and Masse's [109] summary also indicates that precosity disappears by age two, as reported by Warren [138].

The effects of deprivation in *The Creche*, however, were not confined to intelligence. Dennis [136] states: "While the test scores have received the greatest emphasis in this report, the data, fewer in quantity, on school progress, social, marital, and vocational adjustment, and psychiatric referrals are probably of equal importance. It has been shown that the two kinds of data are closely related. Those who were in experientially deprived institutions throughout childhood were not only of low intelligence but also evinced more signs of social maladjustment and personality disorder than did the adoptees" (p. 107).

Although it is clear from Dennis' description of experiential deprivation that multiple sensory deprivation was involved, attention must be given to the profound somatosensory deprivation that the *Children of the Creche* experienced. It would be of interest to determine whether modern techniques in quantitative electrophysiology, specifically, nystagmus and assessment of brain hemispheric relationships utilizing cortical evoked potentials obtained from the different sensory modalities might not provide discriminative information on the relative impairment of the different sensory systems. Such a follow-up study would be invaluable for developmental theory and rehabilitation practices.

This writer is intrigued by the two-year critical period which Dennis' data appears to identify and it would seem heuristic to inquire whether this could be supported by a parallel critical period in brain development. The observation made later in this paper that postnatal neuronal development of the human cerebellum continues to about two years of age is suggestive of one possibility. It would seem timely to explore in some systematic manner the occurrence of behavioral developmental landmarks with landmarks of brain structure development and function as they may relate to the differential rates of sensory modality development.

PRECOCIOUS MOTOR DEVELOPMENT

Additional evidence that may be interpreted as supporting the singular importance of somatosensory deprivation in contributing to developmental deficits is the study of Geber [137] who reported precocious motor development in African infants when contrasted to caucasian infants. The observation that precocity is greater for infants from traditional or lower class families than for westernized or middle class families suggests that social factors need to be considered in accounting for this precocity [133, 134; 137]. In a recent study on African infant precocity, Leiderman, et al. [134] found that Kikuya infants surpassed United States test performance on 38 mental test items and 20 motor test items. The Kikuya lagged behind U.S. test performance on 7 mental test items and 2 motor test items.

In suggesting reasons for these differences, Leiderman, et al. [134] stated:

> "It should also be noted, however, that maternal caretaking within the Kikuya community involves more *physical contact* with the infant in the first six months of life, and therefore might also account for these differences (additional data on maternal behavior will be presented elsewhere)." (Italics mine, p. 248.)

Additionally, in a multiple regression analysis of Bayley test scores with demographic factors, the number of individuals over 40 years of age in the household was the only factor significantly correlating with motor test scores [134]. This suggests that additional infant caretaking (greater somatosensory stimulation) may have been provided by those over 40 years of age which would have contributed to infant motor development which is consistent with the greater maternal-infant physical contact observed in the Kikuya by the authors. This interpretation is further strengthened by their findings that infants from a household with two or more individuals aged 41-60 scored *highest* on motor test scores compared to those infants from households which had no adults over the age of 41 years. Infants from households without adults over age 40 scored lowest on motor test scores. It is apparent that the hypothesis that adults over 40 years of age provide additional somatosensory stimulation to the infant can only be resolved by direct observation.

Warren [138], recently reviewed a number of studies on African infant precocity and concluded that it has not been satisfactorily demonstrated. There is some basis, however, to question his conclusion. Warren [138] reviewed 14 studies in which 12 supported precocity and two did not. Most of the studies were rejected because of flaws of design, analysis, and reporting. The studies of Kilbride, et al. [139], Theunissen [140], Falmagne [141], and Liddicoat and Koza [142] were considered satisfactory. The studies of Theunissen and Falmagne found no general differences between blacks and whites and the studies of Kilbride, et al. and Liddicoat and Koza found evidence in support of African infant precocity. It should be noted that in no instance did any study report white infant precocity. It is evident that Warren [138] gives greater weight to the studies of Theunissen and Falmagne than to the studies of Kilbride, et al. and Liddicoat and Koza.

Warren [138], however, has made the following observation:

> "The new Bayley test, however, has been very carefully standardized and the most compelling evidence for African precocity based on comparison with test norms comes, therefore, from Kilbride and Goldberg, using the motor scale only and from Lusk and Lewis [143] who used both scales but with only 10 subjects of various ages. The real problem of course, relevant to all research except that of Theunissen and Falmagne, is that comparisons were made only with available Western norms and not with samples of European babies taken by the same investigator. A further point that may be important is that most of those who report African precocity

against norms made observations in the infants' own homes and then compared performance with normative data collected in clinic situations" (p. 360).

With respect to differences by social level, however, Warren [138] states: "For once, the results, sparse as they are, show consistency. Infants from homes of a low social level are initially more advanced than elite babies" (p. 363). He concludes: "It can hardly be claimed that we have here a definite phenomena. However, there is a somewhat surprising indication that infants from poor African homes develop more rapidly at first than those from elite homes" (p. 364).

It is the interpretation of this writer and others that this social class effect may well be due to the westernization of child rearing practices in the higher social classes. The lower social class maintains ethnic customs that involve greater physical handling and carrying of the infant. As Lusk and Lewis [143] comment: "Infants of acculturated families tended to be kept in cribs much of the time. . . they were held and carried much less often than the village infants and fed on schedule rather than on demand" (p. 59). Thus, African social class differences in infant development may well be directly a function of variation in early sensory stimulation, specifically somatosensory stimulation. This hypothesis, however, requires rigorous evaluation.

Warren [138], in discussing these issues stated:

"The clear prima facie reason for expecting uniform African development is a racial-genetic hypothesis. . . . No other hypothesis would plausibly suggest uniform African development across the continent. Uniformity of infant environments is too much to ask" (p. 364).

This writer would like to suggest that this above conclusion by Warren needs clarification. Specifically, this writer has evolved a somatosensory theory of development which predicts that cultures characterized by high infant physical affection (higher body touching, contact and carrying) would be characterized by low physical violence, and conversely that cultures characterized by low infant physical affection (low body touching, contact, and carrying) would be characterized by high physical violence. This relationship was verified by consulting Textor [144] which summarizes a wealth of cross-cultural data. It was found that the coded scale of *Infant Physical Affection* developed by Barry, Bacan, and Child was significantly related to the coded scale of "Killing, Torturing or Mutilating the Enemy" developed by Slater and described in Textor [144]. This relationship was significant at the $p = 0.004$ level which correctly classified 36 of the 49 cultures in the sample. These results are summarized in Table 10 below.

The Barry, Bacon and Child scale included the following items, rated from 1 to 7:

"Display of affection toward the infant: to what extent is he held, fondled, caressed, played with?" ([145], p. 293).

This writer analyzed a more recent coded scale on infant physical contact developed by Barry and Paxson [146] which was also found to be significantly related to Slater's coded scale of physical violence ($p = 0.03$, $N = 43$). The Barry and Paxson scale was defined as follows:

"Bodily Contact:

This measures the proportion of the day when the baby is held or carried by any caretakers. Two stages, an early period; a later infancy are defined as (a) the first few months after birth; and (b) when the baby first crawls, at approximately nine months of age.

1. Limited to routine and precautionary care
2. Only occasionally
3. Up to half of the time
4. More than half of the time
5. Almost constantly."

In brief, there is evidence of systematic relationships between specific infant environments and later adult behavioral characteristics.

TABLE 10

Infant Physical Affection/Adult Physical Violence

High Infant Physical Affection Low Adult Physical Violence	Low Infant Physical Affection High Adult Physical Violence	High Infant Physical Affection High Adult Physical Violence	Low Infant Physical Affection Low Adult Physical Violence
Andamanese	Alorese	Cheyenne	Ainu
Arapesh	Aranda	Chir-Apache	Ganda
Balinese	Araucanians	Crow	Kwakiutl
Chagga	Ashanti	Jivaro	Lepcha
Chenchu	Aymara	Kurtatchi	Pukapuka
Chuckchee	Azande	Zuni	Samoans
Cuna	Comanche		Tanala
Hano	Fon		
Lau	Kaska		
Lesu	Marquesans		
Maori	Masai		
Murngin	Navaho		
Nuer	Ojibwa		
Papago	Thonga		
Siriono			
Tallensi			
Tikopia			
Timbira			
Trobriand			
Wogeo			
Woleaians			
Yahgan			
	N = 49	PHI = 0.41	
	XSQ = 8.38	% = 73	
	P = 0.004		

THE GUATEMALAN STUDY

The above cross-cultural data has direct relevance to a study reported by Kagan and Klein [147] on early infant development in remote villages of Guatemala. In their comparative study of infant cognitive development they found that "the rural Guatemalan infants were retarded with respect to activation of hypotheses, alertness, and onset of stranger anxiety and object permanence, (however) the preadolescents' performance on the tests of perceptual analysis, perceptual inference, and recall and recognition memory were comparable to American middle-class norms" (p. 957). These authors conclude that "Infant retardation seems to be partially reversible and cognitive development during the early years more resilient than had been supposed" (p. 957). This concept of "resiliency" or "recovery of function" has significant implications for social policies that attempt to correct the adverse consequences of "impoverished" environments upon human development. For example, "these data, together with the poor predictive relation between scores on infant developmental tests and later assessments of intellectual functioning, strengthen the conclusion that environmentally produced retardation during the first year or two of life appears to be reversible" (p. 959). And, "If the first (early) environment does not permit the full actualization of psychological competencies, the child will function below his ability as long as he remains in that

context. But if he is transferred to an environment that presents greater variety and requires more accommodations, he seems more capable of exploiting that experience and repairing the damage wrought by the first environment than some theorists have implied" (p. 960).

Although not intended by the authors the above statements could be readily interpreted by social planners that the early environment is not all that important for human development and that, therefore, major environmental enrichment programs for "poor" infants and children are not really necessary. For these reasons serious attention must be given to the Kagan and Klein study, particularly with respect to a) whether early *cognitive* deficits have actually been satisfactorily demonstrated during infancy; b) what actually has been "reversed" to support the concept of "resiliency" and "recovery of functions"; c) the criterion of "average" performance: mediocrity vs. superior performance, i.e. excellence of achievement; and d) whether the tests employed at the developmental age of final testing (11-12 years) would permit the assessment of the effects of these early environments upon "ultimate" development, i.e. "excellence" of achievement. This is to suggest that average or mediocre functioning may not be affected by certain early deprivation but superior functioning may be seriously affected.

The beginnings of this particular study by Kagan and Klein was influenced by their observations of Guatemalan infants and children:

> "During the first 10-12 months, the San Marcos infant spends most of his life in the small, dark interior of his windowless hut. . . . The infant is rarely spoken to or played with, and the only available objects for play, besides his own clothing or his mother's body, are oranges, ears of corn, and pieces of wood or clay. These infants are distinguished from American infants of the same age by their extreme motoric passivity, fearfulness, minimal smiling, and, above all, extraordinary quietness. A few with pale cheeks and vacant stares had the quality of tiny ghosts and resembled the description of the institutionalized infants that Spitz called marasmic. Many would not orient to a taped source of speech, not smile or babble to vocal overtures, and hesitated over a minute before reaching for an attractive toy" (pp. 949-950).
>
> "We saw listless, silent, apathetic infants; passive, quiet, timid 3-year-olds; but active, gay, intellectually competent 11-year-olds. Since there is no reason to believe that living conditions in this village have changed during the last century, it is likely that the alert 11-year-olds were, a decade earlier, listless, vacant-staring infants. That observation has forced us to question the strong form of continuity assumption in a serious way" (pp. 947-948).

The above clinical description of Guatemalan infants, which categorizes them as similar to Spitz's institutionalized infants, carries with it all the developmental implications that are known about institutionalized children. It is well established that extreme emotional disturbances occur in children deprived of human physical contact (hospitalization and institutionalization) and that these emotional disturbances are intimately linked to poor intellectual development. Kagan and Klein assumed that similar behaviors warranted the postulating of similar etiologies and developmental outcomes. However, in their own report they cite evidence that cannot support their equating of Guatemalan infants with Spitzian institutionalized infants:

> "The infants cried very little because the slightest irritability led the mother to nurse her child at once. Nursing was the single, universal therapeutic treatment for all infant distress, be it caused by fear, cold, hunger, or cramps" (p. 950).

Clearly, the maternal closeness and prompt and rich physical affection given the Guatemalan infant by its mother precludes any comparison with Spitz's maternally-socially deprived infants. Thus, the observations of "fearfulness," "minimal smiling" and "extraordinary quietness" must be accounted for differently

and interpreted differently. It is suggested that Kagan and Klein committed the classical error of interpreting a natural cultural difference in emotional expressivity (Guatemalan vs. American) as evidence for a "deficit" on part of the non-American culture. American infants are expected to be smiling, extroverted, assertive and reactive to their environment. Guatemalan infants are somber, introverted, quiet and relatively non-reactive to their environment. The interpretation of the Guatemalan infant behaviors as "marasmic," i.e. as abnormal appears to be a major diagnostic error. This has lead to an interpretation of task performance differences as reflecting deficits in cognitive abilities when it is more likely that such differences can be attributed to emotional and motivational factors.

The lack of familiarity with complex visual and auditory stimuli from "western" culture tests and "the unstimulating environment of the dirt hut" are more likely explanatory mechanisms than the mechanisms of "maternal-social" deprivation associated with institutionalization. Consequently, questions can be raised as to the precise nature of the "retardation" observed. If novelty and fear reactions and "motivational" variables are contributing sources of variance to "retardation" then questions must also be raised as to which "functions" have been "recovered." Recall that the San Marcos infants were characterized by their "extreme motoric passivity, *fearfulness, minimal smiling*, and above all, extraordinary quietness. . . . Many would not orient to a taped source of speech, *not smile* or babble to vocal overtures and *hesitated over a minute* before reaching for an attractive toy" (italics mine) (pp. 949-950). Additionally, the close presence of white strangers who smile a lot and speak a strange language could be expected to enhance their "fearfulness" in a test situation. Body language differences would appear to be substantial with unpredictable consequences upon child behavior. Unfortunately systematic data on the emotional state of children during testing was not recorded. It could be argued that it was the emotional-motivational "deficits" that were overcome ("recovered") with increasing experience of a novel environment rather than cognitive deficits per se. The phenomena described by Kagan and Klein [147] are more reminiscent of Fuller's [149] description of "emergence shock" in dogs required to confront a novel complex multisensory stimulating environment after a period of rearing in a sensorily restricted environment. Similarly, Harlow described the necessity of testing his isolation reared monkeys "in isolation" if the learning capabilities of the animals were to be assessed. It was found that testing these animals in a novel social environment was so emotionally disruptive as to prevent the assessment of learning; and that enriched monkeys tested in the home-cage environment performed significantly better than enriched monkeys tested in novel-cage environment (Gluck, et al., [150]).

In this context it is not unreasonable to suggest that San Marcos children who typically spend most of their day within a 500 yard radius of their homes and who are taken with their mother to a "special laboratory equipped with a chair and a stage that simulated the setting in the Harvard laboratories" might experience some negative emotional states that could interfere with "cognitive performance." This point of view receives some support from their finding that on a test of recall memory for familiar objects common to village life (animals, kitchen utensils, clothing) no significant differences were found between Guatemalan and American children for both age groups (7 and 11 year olds), whereas tests with strange materials (Harvard Test Apparatus) yielded significant differences.

Kagan and Klein's [147] interpretation of Harlow's data that isolation reared monkeys "could, if the experimenter were patient, solve the complex learning problems normally administered to feral-born monkeys," (p. 959) erroneously leaves the impression that isolation rearing has no lasting effects upon cognitive functioning and/or that appropriate environmental intervention can fully reverse any deficits observed: "The prolonged isolation did not destroy their cognitive competence (Harlow, Schiltz and Harlow, 1969 [151]," [147] p. 959). On the contrary,

Harlow, et al [152] reported that "socially enriched preadolescents and adolescents, as contrasted with the socially isolated adolescents and controls, proved to be superior at the 0.001 significance level on our most complex problem--oddity-learning set" (p. 543). Additionally, Gluck, et al. [150] reported that "monkeys reared in enriched environments were superior to partially isolated controls on the complex oddity-tasks but not on two-choice discriminative or delayed response problems" (p. 598). The partial isolates stabilized at approximately 65 percent correct responding whereas the enriched animals stabilized at approximately 80 percent correct responding. These authors conclude:

> "We have suggested elsewhere (Gluck and Harlow, 1971 [153]; Harlow et al., 1971 [152]) that differential rearing has effects on learning performance only indirectly, by either adversely affecting adaptation of deprived subjects to the testing procedures, thus biasing their test scores in a negative direction, or by providing enriched subjects with specific learning experiences which transfer to the criterion test. The present data force a significant change in stance with regard to this position.
>
> First, prior to the presentation of any problems, all present subjects were tediously adapted to all facets of the testing procedure according to rigid criteria. This insured that no pretest group performance differences existed to confound that which followed. Second, since the enriched monkeys demonstrated learning superiority only on the oddity tasks and not generally across all tests, the specific transfer hypothesis does not presently seem tenable. The identification of the nature of these observed differences is left as the problem for future research." --([150] p. 603)

Further, Davenport and Rogers [154-156] found significant differences between deprived and feral reared chimpanzees on two-choice discriminations and delayed-response tasks as well as oddity problems.

Davenport, et al. [157] tested six adult chimpanzees that had been reared for the first two years of life in restricted laboratory environments and were found to be significantly inferior in cognitive skills to eight wild born control subjects, as assessed by *Transfer Index* testing. These authors concluded:

> "The persistence of cognitive deficits in the restrictedly reared chimpanzees, even after 12 years of environmental enrichment, prolonged testing, and group maintenance, is interpreted to mean that deficits so incurred are not readily corrected, at least in the chimpanzee. . . . These results, in conjunction with those reported recently by Harlow, et al. (1971 [152]) with rhesus subjects, also should serve to reinforce the concerns of those who believe that long-term, perhaps life-long cognitive deficits may be induced in human children who do not have the benefit of enriched, stimulating experiences during infancy and the preschool years" (pp. 346-47).

The above data strongly question the "resiliency and recovery of function" of the San Marcos children, as suggested by Kagan and Klein [147]. This could be due to several factors:

a. insufficient cognitive task complexity at age 11 years (assuming a cognitive deficit exists);

b. cognitive differences obtained at earlier ages could be attributable to confounding emotional/motivational variables and not to any real cognitive deficit per se;

c. lack of cognitive differences at later ages (11-12 years) may reflect:

   1. increasing familiarity with a complex and changing environment with concomitant reduction in emotional/motivational variables which interfered with earlier cognitive task performance; and

   2. the absence of a presumed absolute infant retardation

in the first place since the above animal studies do show lasting cognitive deficits consequent to an early impoverished environment.

It should be pointed out that the chimpanzee data are probably more relevant to human infant data since the chimpanzee is closer phylogenetically to man than the monkey. This is reflected in the prolonged period of infant helplessness in the chimpanzee and man when compared to the monkey. Issue must also be taken with Kagan and Klein's [147] interpretation of the "monkey therapy" studies of Suomi and Harlow [160, 161] which are reviewed later in this paper. Briefly, the "monkey therapies" are effective when initiated during *infancy* and involve *primarily physical contact* variables. Therapies initiated later in life and which do not involve physical contact have not reversed the social and emotional abnormalities [158-162]. Since the San Marcos infants were never deprived of physical contact (in fact they received an enriched amount of physical contact), it is difficult to appreciate the relevance of these animal studies to the Guatemalan infant environment and the assumption that the San Marcos infants were deprived in the Spitz and Harlow sense. Both short-term and long-term effects of partial and total social isolation are well described [163-169; 285, 286] and these studies also question the "recovery of function" thesis of Kagan and Klein [147]. It is recognized however, that the San Marcos infants had a restricted visual and auditory environment in contrast to infants from a western culture. This lack of experience with a highly stimulating, complex and ever-changing environment that infants from a western culture experience throughout the early developmental years undoubtedly contributed to their passivity, quietness, and introverted personality characteristics. It is apparent that terminology such as "vacant stare," "tiny ghosts," "institutionalized infants," and "marasmic" denote a different psychological condition than that denoted by such terms as "passivity" and "quietness." Perhaps it is not too extreme to suggest that Kagan and Klein committed a cultural error by judging the behavior of another culture (passive, quiet, contemplative, introverted) in terms of the behavior of one's own culture (aggressive, active, highly verbal, extroverted). It is questionable whether behaviors of western cultures are more desirable, let alone "superior" to the behaviors of non-western cultures. For example, the excessive emphasis upon "cognitive" functioning to the neglect of "emotional/affective" functioning in western cultures is a case in point and it appears that this cultural bias has influenced the Guatemalan study, as reported by Kagan and Klein [147].

The recent findings of Epstein [170, 171] are particularly relevant here since he demonstrated that humans exhibit growth spurts in brain weight and skull circumference around the ages of 7, 11, and 15 years; and mind growth spurts during the intervals 2-4; 6-8; 10-12; and 14-16 years. He has some evidence that spurts in learning capacity exist and that these correlate with the brain-mind growth spurts. Further, Gottlieb, et al. [172] in their reviews of a variety of animal deprivation studies conclude that if animals are deprived through the final or second brain growth spurts they may not have a normal final brain growth spurt. They extend these findings to the human condition and conclude that if a child is deprived during one or all of the first three brain/mind growth spurts that this would result in an abnormal final brain/mind growth spurt. The implications for human development are enormous since, as Epstein and his co-workers point out, the final brain growth spurt may be correlated with abstract or conceptual thinking. The characteristic concrete-bound or non-abstractive thinking of deprived children was cited as supportive of this interpretation (Jensen, [173]). The implications of the above for the Guatemalan study [147] are self-evident. The oldest children tested by Kagan and Klein were 11-12 years, i.e. during the third brain/mind growth period. It would be necessary to test these children at 17 years of age with sufficiently complex tasks (comparable to the oddity tasks which were failed by deprived monkeys and chimpanzees) to determine the validity of the Kagan/Klein hypothesis.

Significant differences between the experimental and control groups at age 17 would essentially nullify the "recovery" arguments advanced by Kagan and Klein and would provide evidence in support of Epstein's hypothesis that early deprivations during the first three brain/mind growth periods affects the development of the final brain/mind growth spurt which is associated with higher complex cognitive functioning. Failure to find differences would raise questions about the nature of the early "deprivations" and leave unresolved the issue of "recovery." Clearly, the retesting of the Kagan-Klein sample at age 17 should be a high priority objective. The utilization of quantitative electrophysiological assessments *during* cognitive task performance would contribute invaluable information upon brain-behavior functions as they relate to the Kagan-Klein and Epstein hypotheses.

In summary, if emotional and motivational factors have contributed to deficiencies in cognitive performance during earlier testing of the San Marcos children, then the assumption of "cognitive retardation" must be questioned as well as "cognitive recovery." Otherwise the implication (not intended by the authors) that early deficits can be overcome without intervention could be extrapolated to justify a national social policy previously suggested by a "benign neglect" doctrine. It hardly needs to be emphasized that cognitive *excellence* in contrast to cognitive *mediocrity* is highly unlikely to be realized from a Guatemalan village environment nor from many American urban environments as we know them.

Additionally, emotional and affective excellence is as important as cognitive excellence for they continually interact throughout development, and in the final analysis, cognitive behavior cannot be fully understood in the absence of knowledge of its affective components.

COGNITIVE-AFFECTIVE INTERACTIONS

The interplay between "cognitive" and "affective" processes can be approached by focusing upon the sensory modalities involved in their functioning as the following study by Pedersen, Yarrow and Rubenstein [174, 175] illustrates: they examined mother-infant interactions in terms of the sensory modality used for communication. These "sensory-interactions" were related to scores of mental and psychomotor development which were obtained from the Bayley Scales of Infant Development plus four additional measures derived from the Bayley, namely:

a. social responsiveness;

b. goal orientation;

c. secondary circular reactions (perseverative play); and

d. object permanence.

The results indicated that *kinesthetic* stimulation was the most important sensory modality variable since it was significantly correlated with all six dependent variables where three of these six correlations were significant at the one-percent level. Thus, vestibular stimulations (physical holding and carrying) were significantly related to the infant's mental and psychomotor developmental status as well as to the specific social and cognitive-motivational variables. Particularly interesting was the finding that passive and active somesthetic (tactile) stimulation were significantly related to only two of the dependent variables, viz., *goal orientation* and *secondary circular reaction*. Strikingly, visual and auditory stimulation were significantly related to only one dependent variable, viz., *social responsiveness*.

The above study clearly supports the prepotency of somatosensory stimulation, particularly vestibular stimulation, in infant development and provides additional evidence for the primacy of the near receptors over the distance receptors for *infant* motor-mental and emotional-social development [35, 36; 57; 134; 174, 175]. The distance receptors assume increased significance in later development. The above data assumes greater significance for Adelson and Fraiberg's study where they noted that "*physical intimacy* united with the sound of the parent's voice provided the first union of tactile-auditory experience for the children" which influenced the delays in mobility and locomotion (italics mine). The findings of

Casler [135]; White and Castle [176]; Klaus, et al. [177]; Kennel et al. [178]; Korner and Thomas [179]; Neal [180]; Woodcock [181]; Rapoport [182]; Ayers [183]; Barnard [184]; and Ainsworth [185] are consistent with this point of view.

Conversely, this emphasis upon the primacy of near receptors for infant development is at variance with the distance receptor school of thought which gives primacy to visual and auditory sensory processes for the motor-mental and social-emotional development of the infant. This latter point of view is reflected in the writings of Walters and Parke [186]; Rhinegold [187]; and Bowlby [188] to mention a few.

The study of Rapoport [182] requires special comment since normal attachment behaviors and emotional and intellectual development were reported in a boy characterized by congenital insensitivity to pain with loss of tactile sensitivity and temperature sensations. Vision and hearing were normal and kinesthetic sensation was intact. Verbal intelligence was normal but motor development was delayed. Maternal attachment was reported to be markedly warm. (Rocking and head banging was the only deviant behavior noted.) Rapoport [182] concluded from this clinical case study that "auditory, visual, and kinesthetic stimuli may be sufficient in the absence of tactile stimulation for initiation and maintenance of attachment behavior" (p. 67).

In evaluating the above conclusion it is necessary to recall that visual sensory deprivation in animals and children does not lead to disturbed social-emotional behaviors. Additionally, the study of Bowyer and Gillies [189] reported that partially and severely deaf children had no greater incidence of maladjustment than children found in an unselected school population. Specifically, they found no significant differences between the partially and severely deaf on a variety of social and emotional adjustment measures. It is of some interest that significant differences in "adjustment" were reported between two schools for the partially deaf and that these differences were "connected with the encouragement of *free movement* and spontaneous conversation in the classroom in schools" (p. 307, italics mine). Similarly, Williams [190] reported that in a group of 51 maladjusted deaf children the psychiatric disorders encountered were similar to those found in hearing children and those handicapped in other ways. Specifically, those children who were characterized with anti-social disorders were noted to have suffered from severely disturbed home backgrounds. It is of more than passing relevance to note the observations of Ling and Ling in these proceedings that mothers of hearing impaired children use much less body contact with their children than did a contrast group of mothers of normal children.

It appears to be necessary to distinguish between somesthetic-cutaneous and kinesthetic-vestibular stimulation in the evaluations of the effects of body contact and movement. The role of kinesthetic-vestibular and kinesthetic-proprioceptive (muscle, joints, tendons) stimulation in contrast to somesthetic-cutaneous stimulation in the Rapoport [182] study is a case in point. The studies of Decarie [191] and Roskies [192] upon thalidomide-induced congenital malformation, where there is abnormality or absence of the limbs (phocomelia), are relevant to the Rapoport case. Decarie [191] found no relationship between the severity of malformations and psychosocial development and observed that the severity of the child's handicap did not seem to be the most significant factor in the behavioral development of thalidomide children. Rather, prolonged and repeated hospitalization, placement in foster homes and particular family circumstances were cited as significant contributing factors to aberrant behavioral development. For example, Roskies [192] reports one mother's comment on the effects of hospitalization upon her thalidomide child: "Because of her hospitalizations, I toilet trained X three times and taught her to walk four times. I don't think I could begin again" (p. 222). As Roskies [192] points out, the above factors, plus the complexity and multiplicity of anomalies produced by thalidomide makes it almost impossible to rank order thalidomide children in terms of severity of impairment and thus makes it difficult to attribute deficits of behavioral

development to phocomelia alone. Unfortunately, it is beyond the scope of this paper to treat more systematically the rich data base obtained from studies of thalidomide damaged children or to attempt to more specifically relate phocomelia, as a sensory deficit, to behavioral developmental processes. It is of interest to note, however, the failure to find EEG abnormalities in thalidomide children by Pampiglione [193], which provides further evidence that deficiency of kinesthetic-proprioceptive stimulation is not as serious for normal development as might be assumed. The emphasis upon movement, i.e., kinesthetic-vestibular stimulation as essential for normal behavioral development is given further support by the study of Siegel [194] who reported marked improvement in the social-emotional behavior of autistic children with movement therapy. Similarly Schopler [195] in his review of the role of receptor processes in autism gave emphasis to sensory deprivation of the near receptors and the efficacy of therapy of stimulation to these receptors (touch and movement). Finally, it should be noted that Korner and Thoman [179], cited earlier, concluded from their study that "vestibular stimulation which attends most caretaking activities may be more crucial than contact for certain aspects of early human development" (p. 67).

The above analyses of selected experimental and clinical data on the effects of sensory deprivation have led to the conclusion that kinesthetic-*vestibular* sensory stimulation is of primary importance for motor-mental and social-emotional development. This writer is not aware of any studies that report congenital vestibular neuropathy in humans nor surgically induced vestibular sensory loss (partial or complete vestibular deafferentation) in animals reared in a normal maternal-social environment. I have previously suggested this study to critically test and evaluate the role of vestibular stimulation and deprivation upon developmental processes [35].

THE SENSORY BASES FOR ATTACHMENT

It would appear helpful to emphasize more clearly the differences between the various sensory systems and their role in social-emotional development, particularly, the acquisition and measurement of attachment behaviors. Bowlby [188] has summarized his view on the relative roles of the different sensory receptors as follows:

> "Both in these experiments and in the everyday settings the social stimulation reported as being effective in promoting attachment behaviour comprises a mixture of visual, auditory, and tactile, and usually kinaesthetic and olfactory stimulation as well. Questions thus posed are: which, if any, of these modes of interaction are indispensable for attachment to develop, and which are the most powerful for this purpose?
>
> In discussions of the subject two trends are noticeable. In much of the earlier literature, which assumed that attachment developed as a result of a child's being fed, emphasis was placed on tactile, and particularly oral, stimulation. More recently, this supposition has been challenged, especially by those such as Rheingold (1961) [187] and Walters and Parke (1965) [186] *whose theoretical position is similar to that adopted here.* These workers emphasize that, from quite early weeks, an infant's eyes and ears are active in mediating social interchange, and they call in question the special role hitherto attributed to tactile and kinaesthetic stimulation. Not only smiling and babbling, but eye-to-eye contact also seems to play a very special part in developing a bond between infant and mother (Robson, 1967), [196]." (italics mine.)
>
> "At first reading it might be thought that the view that visual rather than tactile and kinaesthetic stimuli are prepotent receives support also from a study of the development of attachment

in infants who are averse to being cuddled, reported by Schaffer and Emerson (1964b) [197]. Such a conclusion would, however, hardly be warranted" ([188] p. 319), [197-198].

"The truth is that data do not yet exist for answering the questions posed. That the distance receptors play a far more important part than they have hitherto been accorded seems indubitable, but this is far from concluding that tactile and kinaesthetic receptors are unimportant. On the contrary, when an infant is much distressed, bodily contact seems vital, whether it is in soothing a crying infant during his early months or, a little later, in comforting him when he is frightened. The wisest position to take at present is that in all likelihood all modes of social interaction play a major role, but that, thanks to considerable redundancy in the organisation of attachment behaviour, a shortfall in one mode can, perhaps within wide limits, be made good through some other mode. A plethora of alternative means by which the requirements of survival can be met is, it is known, very common in the animal kingdom." ([188] p. 321.)

Although Bowlby gives primary emphasis to the distance receptors his ambivalence on the subject is still evident and is illustrated in Figure 1 which is the cover of Bowlby's text *Attachment and Loss*, Vol. 1, *Attachment*, which emphasizes the near receptors in the maternal-infant relationship. This writer is in fundamental disagreement with Bowlby's viewpoint that the sensory systems are relatively equipotential in their functional compensatory abilities. Specifically, the distance receptors cannot compensate for loss of physical affection and somatic pleasure consequent to somatosensory deprivation. Additionally, Bach-y-Rita has demonstrated in these proceedings and elsewhere [113] that the skin senses can be utilized to induce visual perceptions whereas the converse is not true, namely, retinal stimulation cannot induce skin perception.

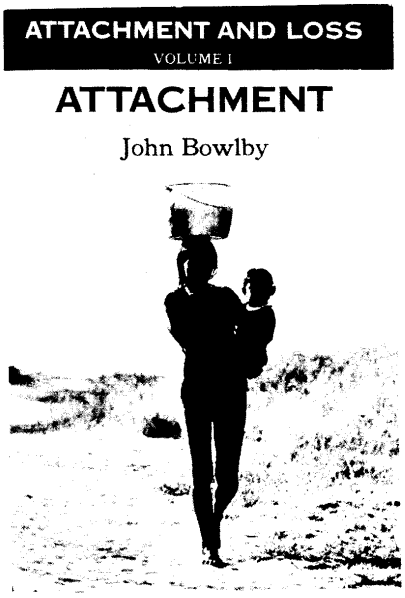

Figure 1. Cover. From J. Bowlby, *Attachment and Loss* (86)

It needs to be emphasized that sensory deprivation to the near receptors in contrast to sensory deprivation to the distance receptors have fundamentally different consequences to the developing organism. For example, Mason [65] demonstrated that infant monkeys reared in isolation with mechanical *moving surrogate* mothers did not develop movement stereotypes which were developed by infant monkeys reared in isolation with a *stationary surrogate* mother. Neal [180] demonstrated that mechanical rocking of human prematures facilitated their health status and maturation of sensory-motor functions.

Sackett [158] in his review of the factors contributing to abnormal behaviors and thus amelioration in the partial isolate concluded:

"The presence of early visual and auditory contact is not therefore sufficient to produce adequate social development in partial isolates. . . thus visual

and auditory exposure to a social stimulus is not sufficient for permanent attachment formation but seemingly must be accompanied by actual physical contact as a crucial stimulus dimension." (p. 120)

In another review by Sackett [159] concerning the effectiveness of various therapies he concluded:

"The PI monkeys in these studies all received social and nonsocial experience after the first year of life. The persistence of behavioral deficits, even after extensive 'therapy' experience suggests that *rearing without physical peer contact* produced animals unable to adapt adequately to many social and non-social situations." ([159], p. 33) (italics mine.)

Harlow, Suomi and their coworkers [152; 160-162; 169] have recently described successful therapy with monkeys reared in total social isolation for the first six months of life which is known to consistently produce severe deficits in virtually every aspect of social behavior. In the above studies it was pointed out that:

a. young isolates exposed to equal-age normal peers achieved only limited recovery of simple social responses;

b. some mothers reared in isolation eventually exhibited acceptable maternal behavior when forced to accept infant contact over a period of months but showed aversion for the contact;

c. isolate infants exposed to surrogates were able to develop crude interactive patterns among themselves; and

d. six month old social isolates exposed to three month old normal monkeys achieved essentially complete social recovery for all situations tested.

In the above studies it was emphasized that normal equal-age peers would frequently attack the partial isolate while the three month old monkey had not yet developed aggressive behavior patterns and would cling to the older six month old. It was concluded:

"The role of *gentle physical contact* and model-serving exhibited by the therapists was likely of paramount importance for isolate recovery" (Suomi, et al. [160]). (italics mine.)

The above brief overview of some primate isolation rearing studies with other data not reviewed here but elsewhere (Prescott, [35, 36]) has compelled this writer to conclude that of all the sensory systems involved in primate social relationships the somatosensory system is the crucial sensory system for the development of normal social emotional behaviors; its deprivation during the formative periods of development is responsible for the variety of abnormal social emotional behaviors observed consequent to "social" isolation; and effective "therapy" for their behavioral disorders must include somatosensory stimulation of a pleasurable-affective nature.

A plea is made to reconceptualize the "social" deprivation studies as "sensory" deprivation studies and that concerted efforts be established to examine the neurobiology of brain structure and function in animals so deprived. This reconceptualization should give greater validity to extrapolating non-human primate behavior to human primate behavior as discussed in more detail elsewhere (Harlow and McKinney [199]). Finally, a major task remains to determine how much somatosensory deprivation in the human infant is sufficient to induce abnormalities of behavior or conversely, how much physical affection is required by the human infant and child to ensure its normal social and emotional development. These issues are explored elsewhere (Prescott, [36]).

Another example of this issue are the Schaffer and Emerson [197-198] studies of "cuddlers" versus "non-cuddlers." It should be noted that differences between these infants do not reflect differences between near-receptor and distance-receptor functioning. Rather, they reflect intra-somatosensory differences, namely, the "cuddlers" are *somesthetic* types and the "non-cuddlers" are *kinesthetic* types. The non-cuddlers are quieted by *movement* stimulation, e.g., being

thrown up and down in the air, wheeled in prams, swung in swings, etc. [57; 197, 198]. It has been suggested previously that cuddlers may reflect, in part, mothers who are active during pregnancy; and conversely non-cuddlers may reflect, in part, mothers who are relatively inactive during pregnancy [57]. This suggestion, however, requires evaluation. Thus, the Schaffer and Emerson [197, 198] studies can be interpreted to give primacy to somatosensory-stimulation variables in the mother-infant relationship.

The issues of attachment vs. dependency have been dealt with extensively elsewhere [200-204]. It is beyond the scope of this paper to elaborate much further on the relevance of specific sensory systems to the attachment/dependency controversy. Suffice it to say that this writer has previously suggested that a major difficulty in distinguishing between attachment and dependency behaviors resulted from using the behavior or *responses* of the child as differentiating criteria [57]. Within the context of the somatosensory theory of socialization, it was proposed that attachment behaviors result from somatosensory stimulation and dependency behaviors result from somatosensory deprivation [57]. Thus, the distinction between attachment and dependency derives from the nature of the *sensory* environment experienced during the formative periods of development and not from the behavioral response repertoire of the child. Further, a psychophysiological assessment of "dependency" and "attachment" was suggested [46; 57] which was based upon the observation that impaired habituation is a consequence of sensory deprivation during the formative periods of development [46; 57]. Thus, it would be expected that "dependent" children would habituate less readily to somatosensory (tactile and vestibular) stimulation than would "attached" children; and that the form or rate of habituation to visual and auditory stimulation would be different from somatosensory stimulation in the "dependent" child whereas no such differences would be expected to be obtained in the "attached" child. Systematic differences should also be found in nystagmus activity consequent to caloric, rotational and optokinetic stimulation. These psychophysiological assessment procedures are of primary relevance to the study of crosscultural differences in child rearing practices, particularly where large differences in somatosensory stimulation of infants and children are observed [46; 57; 36; 205, 206]. A more complete rationale for these suggestions have been provided elsewhere and it has been emphasized herein to further support the differential importance between near-receptor and distance-receptor functioning for developmental social behaviors [35, 36; 57]. This is particularly relevant for understanding the unique problems associated with the sexual behaviors of the blind (Gillman and Gordon [207]; Prescott [208]).*

CEREBELLAR MECHANISMS OF BEHAVIOR

In concluding this paper it would seem appropriate to summarize briefly the neurophysiological properties of the vestibular-cerebellar structures and their relevance, as a regulatory system, to the issues discussed above.

First: The cerebellum is one of the most immature mammalian brain structures at birth and therefore is highly vulnerable to a variety of early postnatal insults, e.g. insufficient sensory stimulation for normal growth, development and function; malnutrition; neonatal anoxia; hyperthermia; viral infections; environmental toxins; etc. (Prescott [35, 36]). Specifically, Altman [209] and Howard [211] have reported upon the postnatal growth of the rodent cerebellum where the latter author has indicated a 580 percent postnatal increase of cerebellar DNA and a 25 percent postnatal increase of cerebral DNA from day 2 to 15 which represents the percent growth of those structures during that postnatal period. Howard, et al. [210] have studied the increase of DNA in cerebrum and cerebellum during development of the human fetus and demonstrated an *exponential* increase in total cerebellar DNA that was not found for cerebrum. Since it is unlikely that an exponentially increasing rate of cell division would be abruptly terminated, these results suggest the presence of a significant amount of *postnatal cell division* of

*Personal communication.

neuronal precursors in the human cerebellum in contrast to the cerebrum. It should be noted that DNA reflects the actual number of nuclei where the cells are diploid. Although Purkinje cells are uniformly tetraploid by six months after birth in the human and since there are approximately 1000 granule cells to each Purkinje cell, the errors of estimate introduced by the tetraploid Purkinje cells are considered minimal given the number of granule cells in total cerebellum (10^{10}) [59]. The studies of Ellis [212] and Raaf and Kernohan [213] report gradual increases in the thickness of the extefnal granular layer in human cerebellum to 10 to 20 months of age. Meyers [214] provides data on the percentage of growth of the cerebellar molecular layer from six months of fetal life until two years after birth. These data indicate that cerebellar cell development is not complete until age two. Winick, et al. [215, 216] have described the effects of severe malnutrition upon cell development in cerebrum and cerebellum in children and found that reduction of cell division by malnutrition appears to level off at about 12 months for cerebral cortex and at about 16 months for the cerebellum. Lapham* has expressed the point of view that the morphological development of the cerebellum spans the first two years of life and that physiological maturation continues for several years in the human.

Second: The cerebellar cortex has perhaps the highest cell density of any neural structure of the brain where estimates of the density of granule cells are from 2.5 to 7 million cells per cubic millimeter. The total number of granule cells in the whole cerebellum are estimated at 10^{10} [59]. It would appear that such cell density and complexity would involve more than the regulation of motor functions. Models of the cerebellum involving pattern perception and memory processes are dependent, in part, upon this high cell density [105-108].

Third: It has been previously proposed that the cerebellum may be a master regulatory system for sensory-emotional-motor processes [35; 57] and that the organization of sensory projection fields within cerebellar cortex suggests a neurophysiological basis for the behavioral distinction of near-receptor and distance-receptor functioning. Figures **2** and **3** present a highly schematized representation of the gross morphology of the mammalian cerebellum and Figure **4** presents the specific sensory projection fields upon the cerebellar cortex for auditory, visual and somatosensory inputs. As can be seen, the sensory projection fields of somatosensory inputs (near-receptors) are different from and relatively independent of the visual-auditory sensory projection fields. Further, the projection fields of visual and auditory inputs (distance receptors) to the cerebellar cortex are co-extensive and overlap; the projection fields of somatosensory inputs (near receptors) are different from and relatively independent of the projection fields of visual-auditory afferents to the cerebellar cortex. Figure **5** presents the vestibular sensory projection fields upon cerebellar cortex and it can be seen that these sensory projection fields are completely different from the projection fields for the other sensory modalities except for a small overlap in the lingular of the anterior lobe. The cerebellum may well process sensory information from the distance receptors and near receptors as distinct functional units which would seem to provide a neurophysiological basis for the behavioral distinction between near receptor and distance receptor functioning in developmental processes. It is of interest to contrast the organization of sensory projection fields within the reticular formation to that of cerebellar cortex. Groves, et al. [218] reported a distinct and differentiated localization with some overlap of visual and auditory projection fields within the reticular formation but found that cells responsive to tactile stimulation were evenly distributed throughout the reticular formation which were somatotopically organized. The anterior, middle and posterior thirds of the body surface projected, respectively, to the anterior, middle and posterior portions of the reticular formation. Unfortunately, vestibular afferents were not studied. It should also be noted that the cerebellum does not mature uniformly and that the differentional maturation of specific regions within the cerebellum has important implications for development which cannot be elaborated

*Personal communication.

GROSS MORPHOLOGY OF MAMMALIAN CEREBELLUM

HIGHLY SCHEMATIZED REPRESENTATION

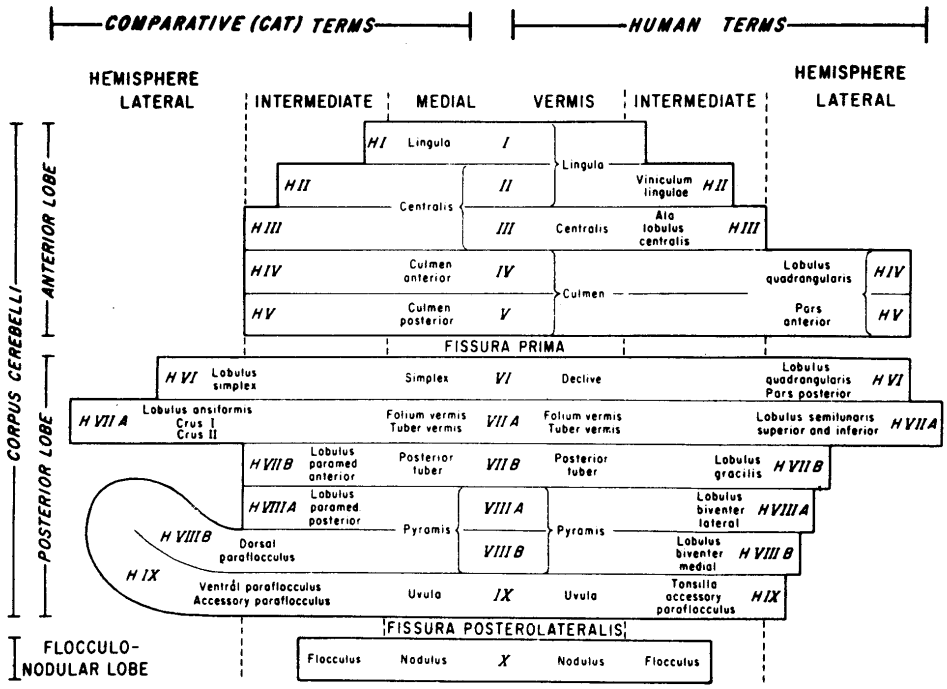

Figure **2.** Cerebellar terminology is presented in a schematic diagram. Those terms frequently applied to the lobular pattern of lower animals as exemplified in the cat are shown on the left, and the terms applied to the human cerebellum are shown on the right. In each category the scheme of numerical designations proposed by Larsell is included. Please note that while lobule II can be readily homologized in all species including man, it is a part of the lingula of human terminology and a part of what has been called the lobulus centralis in subprimate animals. Also note that in the anterior lobe in the human (right) the lateral lobules H IV and H V extend well into the hemisphere, but not in the cat (left). The longitudinal divisions of the lobules of the posterior lobe with respect to the medial, intermediate, and lateral zones are less well defined at the present time than for the anterior lobe; but there is strong evidence in the cat for considering the paramedian lobules, H VIIB and H VIIIA, as intermediate, and some evidence for considering the paraflocculus, H VIIIB and H IX, as lateral. The left half of the diagram differs from the right and shows the paraflocculus, which is large in many mammals, particularly water-living species, and is nonexistent in man. [Dow]

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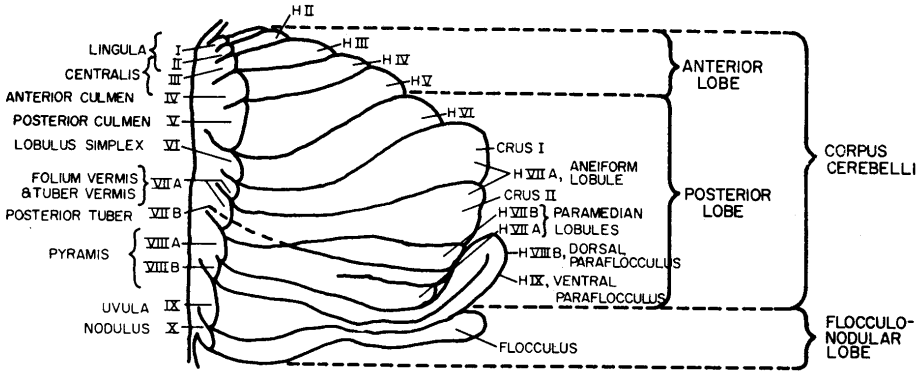

Figure **3.** Generalized structure of the mammalian cerebellum, showing the vermal and lateral folial patterns [modified from Dow, 1961]. For the nomenclatural details, see Figure 24.

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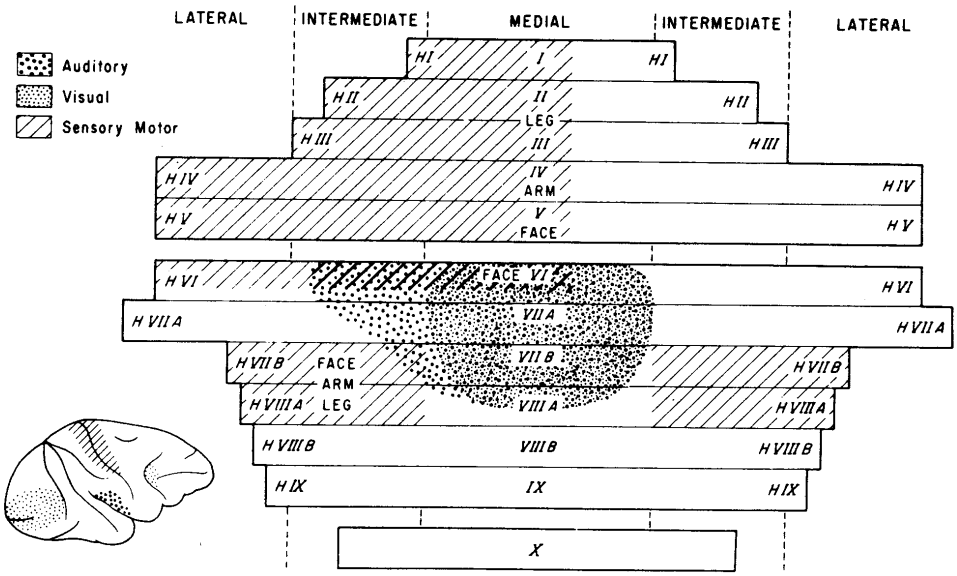

Figure **4.** Diagram of projection areas of specific cerebral cortical regions onto the cerebellar cortex of the monkey [Schematized from Snider and Eldred, 1952, Fig. 4, p. 35].

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upon herein [219]. Figures **6** to **8** illustrate some of the unique relationships of specific cerebellar cortical brain regions to other brain structures.

With respect to *input* and *output* characteristics of cerebellar cortex it should be noted that the only *inputs* to cerebellar cortex from outside the cerebellum are the *mossy* and *climbing* fibers and the only *output* from cerebellar *cortex* is the inhibitory *Purkinje cell axons*. Braintenberg [220, 221] has emphasized this simplicity of input and output as one of the unique features of the cerebellar cortex.

Multiple independent inputs of information have been considered to be a functional characteristic that enhances accuracy and reliability of a regulatory control system. It is with that principle in mind that the organization of sensory inputs into various brain structures should be considered in attempts to relate structure to function. Input characteristics, however, are only one feature of a regulatory control mechanism. Output characteristics and interactions between inputs and outputs are also essential elements of a regulatory control system and these are described below with respect to cerebellar functioning.

Fourth: The *output* functions of the cerebellum are characterized by the nature of neural activity, i.e., excitatory and inhibitory neural processes and not by sensory modality processes. This cerebellar characteristic is in marked contrast to that of cerebral cortex. There are only two output systems from the cerebellum, namely, (a) cerebellar cortex; and (b) the deep cerebellar nuclei. Specifically, the efferent projections from cerebellar *cortex* (Purkinje system) are exclusively *inhibitory* in nature; and the efferent projections from the deep cerebellar nuclei are exclusively *excitatory* in nature. The cerebellar

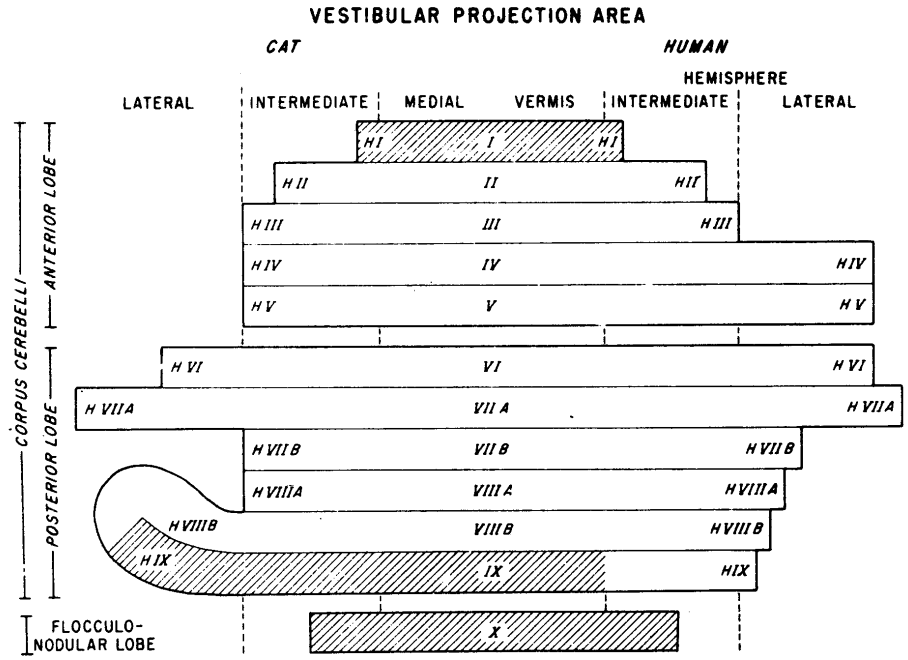

Figure **5.** The vestibular projection areas on the cerebellar cortex are shown by cross hatching. Direct fibers from the labyrinth terminate ipsilaterally while secondary fibers from the vestibular nuclei are distributed bilaterally. [Dow]

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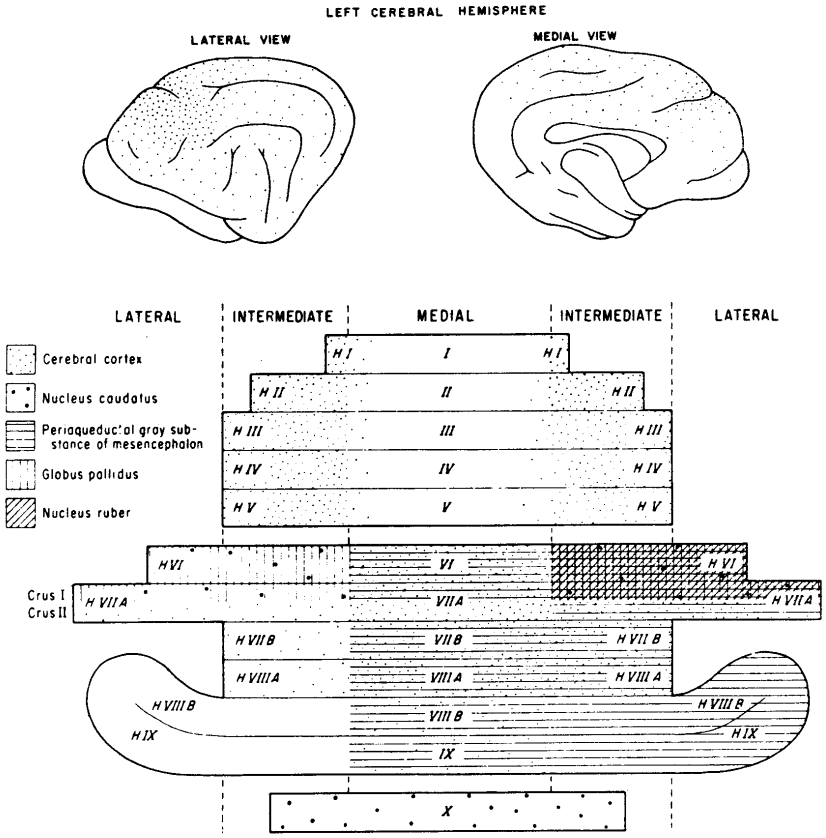

Figure **6.** Diagram representing the ultimate termination of afferents to the cat cerebellum from specific rostral areas of the left side of the brain via specific parts of the inferior olive. The origins of the projections, i.e., cerebral cortex, caudate nucleus, globus pallidus, periaqueductal gray substance of the midbrain, and red nucleus, are shown by various types of shading. Above the schematic diagram of the cat cerebellum the left cerebral hemisphere is shown; the more dense stippling differentiates the sensorimotor area and its cerebellar connections. The unshaded medial area of the anterior lobe receives olivary afferents from the accessory part of the inferior olive that has its afferent connections from the spinal cord. [Schematized from Walberg, 1956, Fig. 23, p. 134.]

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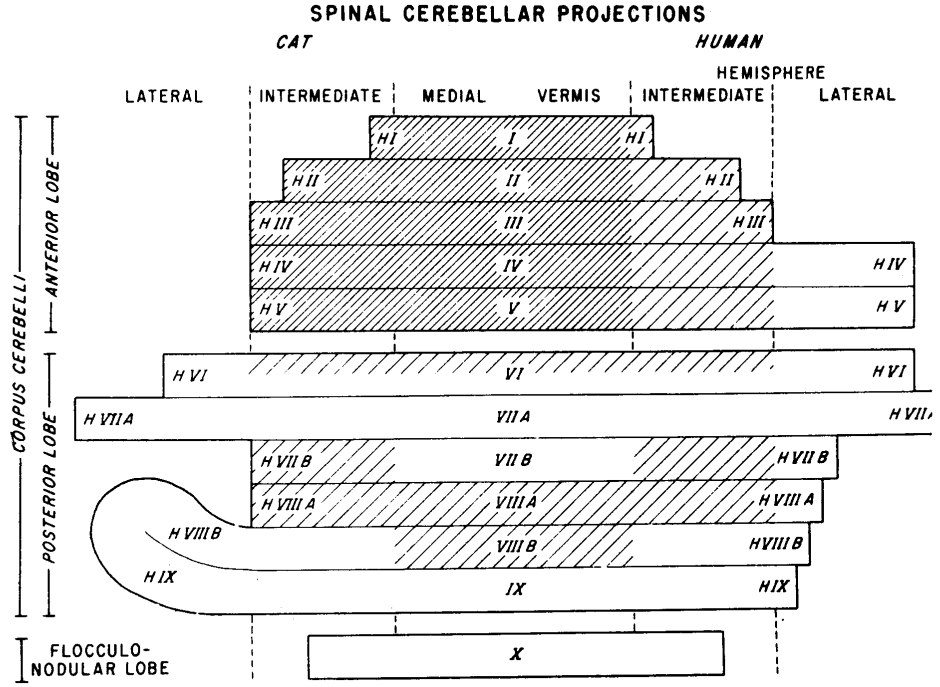

Figure 7. Spinocerebellar projection on the cerebellar cortex. The spacing of the lines indicates the density of the connections. The connections to the anterior lobe are almost entirely ipsilateral, i.e., fibers from one side of the body go to the same side of the anterior lobe. The projection to the paramedian lobules, however, is bilateral. For details, see Oscarsson, 1965. There is some evidence that the projection in primates is relatively denser medially and is encroached upon by pontine projection laterally (Dow, 1942b). This difference between subprimates and primates is shown by differences in density of shading on the left and right halves of the diagram. [Dow]

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cortical Purkinje axons project to the deep cerebellar nuclei and to other brain structures. Thus, the neural excitatory output from the deep cerebellar nuclei is modulated by Purkinje *inhibitory* efferents from cerebellar cortex. This differentiated output characteristic of cerebellar activity should also contribute to the enhancement of accuracy and reliability of cerebellar functioning in its role as a regulatory control system. It is, of course, recognized that the cerebellum is subjected to a variety of neuronal influences from other brain structures but it is beyond the scope of this paper to describe these relationships which have been more adequately presented elsewhere [59; 222, 223].

Fifth: The cerebellum has a *structural horizontal* organization and a *functional longitudinal* organization. Figures to 2 are 10 illustrative of this organization. Figures 9 and 10 illustrate more vividly the longitudinal organization of cerebellar function which is of particular interest for the proposal that the cerebellum may be a master regulatory system for sensory-motor behaviors. As can be seen from Figures 26 and 27 there is a distinct

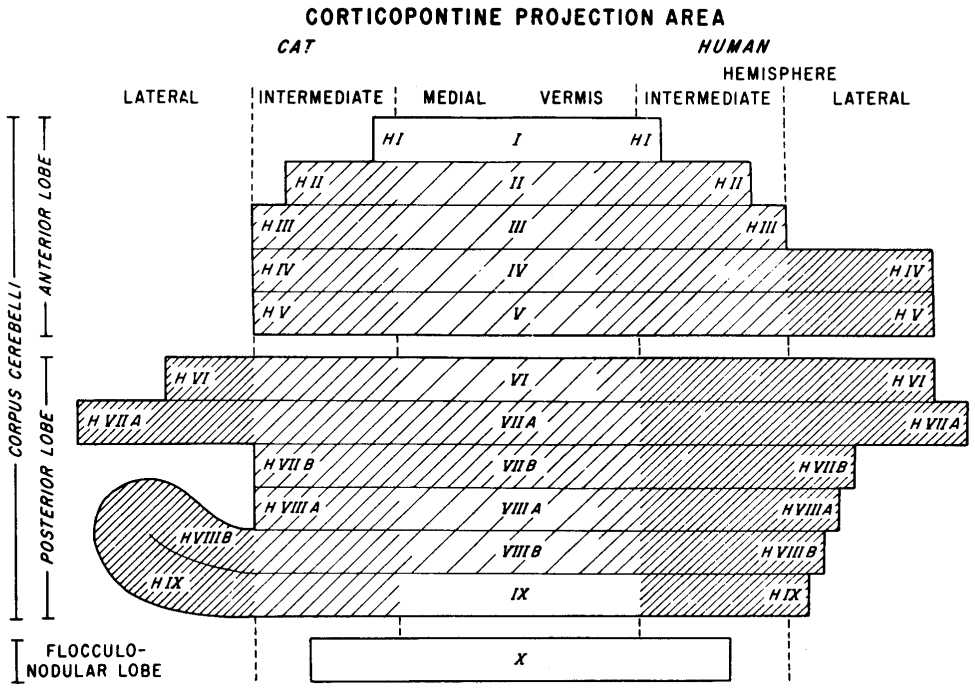

Figure **8.** Corticopontine projection area on the cerebellar cortex. The spacing of the lines indicates the density of the connections. Note that in the right-hand side of the diagram which represents the primate cerebellum the corticoponto-cerebellar connections have great density in the anterior lobe and extend further medially than in subprimate animals shown on the left-hand side. Lobule H VIII B and H IX (paraflocculus) is enormous in aquatic forms and receives its mossy fiber input exclusively from the pontine nuclei. [Dow]

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relatively non-overlapping relationship between the output of the cerebellar cortex to the three deep cerebellar nuclei. The midline or vermal area of cerebellar cortex projects to the *fastigial* nuclei; the intermediate or paravermis area of the cerebellar cortex projects to the *interpositus* nuclei; and the hemispheric or lateral areas of the cerebellar cortex projects to the *dentate* nuclei. As can be seen in Figure **6** there are highly specific projections of the cerebellar cortical-deep nuclei relationships to other brain structures that are involved in motor functions. The importance of the above interrelationships for the point of view being presented herein is that the output of the *fastigial* nuclei project primarily to old brain structures, i.e. it represents paleocerebellar functions. The main relationships of the *fastigial* nucleus are with the vestibular nuclei and the pontine and medullary reticular formation. The output of the interpositus and dentate nuclei projects primarily to new brain structures, i.e. they represent primarily neocerebellar functions. The main relationships of the *interpositus* and *dentate* nuclei are with the thalamic and red nuclei and cerebral cortex [59]. More extensive treatment on cerebral-cerebellar interrelationships

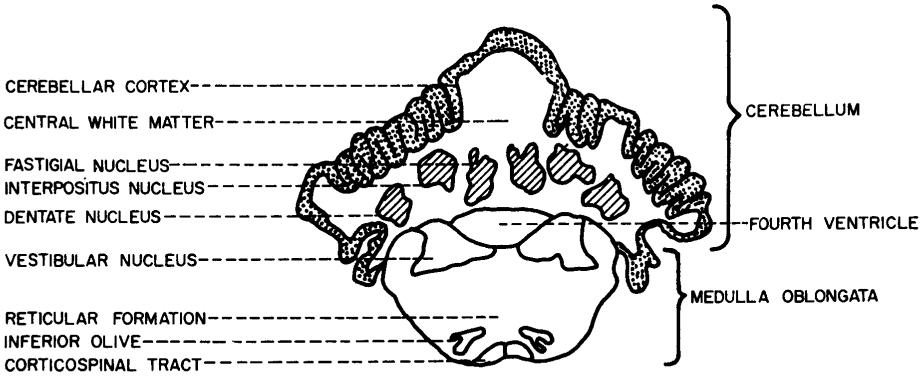

Figure **9.** Cross section through cat cerebellum and brainstem [adapted from Sprague and Chambers, 1954]. "Dentate nucleus" is given here for comparative purposes. The correct name for the structure in subprimates is "lateral nucleus."

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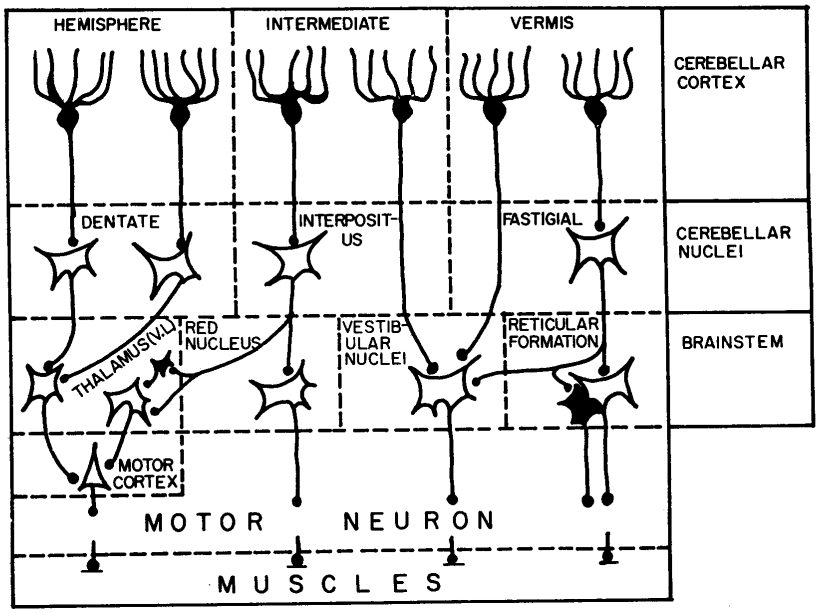

Figure **10.** Pattern of outflow from the cerebellar cortex. Black neurons are inhibitory, white are excitatory. The other neurons intervening between those in the brainstem and the motor neurons are not shown. [Figure adapted from Ito, W. S.]

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can be found in Evarts and Thach who describe these interconnections in the initiation of motor movement [125, 126].

It needs to be emphasized that cerebellar cortical-cerebellar nuclei relationships are organized according to phylogenetic-evolutionary principles and not in terms of sensory function. Thus, the regulation of behavior through cerebellar functions is mediated not only by the way in which sensory input is organized within cerebellar cortex but also by the way in which cerebellar cortex is organized phylogenetically. Consequently, it is suggested that the cerebellum may be a central processor for the transformation of sensory information into a format that permits a phylogenetic interpretation of that sensory information. The enormous development of the cerebellar hemispheres and their special relationship to cerebral neocortex in the higher primates and some ocean mammals, e.g. the bottlenose dolphin (Tursiops truncatus) in contrast to the lower mammals suggests an evolutionary significance for the cerebellum that has yet to be explained [224-231]. These issues seem not unrelated to Lilly's [224, 225] studies on non-human intelligence and language in the dolphin and MacLean's concept of the triune brain [228]. The findings of Gardner and Gardner [232, 233]; Premack [234, 235] and Rumbaugh, et al. [236, 237] that one of the higher primates (chimpanzee-Pan) can learn rudimentary language and the studies of Lilly [224, 225] which indicates that humanoid conversations between the bottlenose dolphin (*Tursiops truncatus* and *homo sapiens* may be possible, acquire an added dimension when taken within the context of the theories of Marr and Blomfield and of Albus [105-108]. Specifically, these writers propose that the cerebellum may be involved in pattern perception and memory processes which suggest that the extensive development of the cerebellar lateral hemispheres and their special connections to cerebral neocortex in these species (*homo sapiens*, *Pan*, *Tursiops truncatus*) may be related to their language abilities.

This theoretical orientation has some support in the clinical findings of Frank and Levinson [238] who have reported that:

> "Of 115 consecutive dyslexic children selected and referred for psychiatric evaluation on the basis of their poor or refractory response to reading instruction, 112 (97%) children showed evidence of a cerebellar-vestibular dysfunction. This cerebellar-vestibular dysfunction manifested itself in the dyslexic children by: positive Rombergs, difficulty in tandem walking, articulatory speech disorders, dysdiadochokinesis, hypotonia, and various dysmetric or past-pointing disturbances during finger-to-nose, heel-to-toe, writing, drawing, as well as during ocular fixation and scanning testing (Dow and Moruzzi, 1958 [239]), ([238], p. 690).

The speculative nature of these suggestions are recognized and it is beyond the scope of this paper to extend these concepts any further. However, they seemed worthy of mention because of their implications for heuristic thinking about the evolutionary significance of cerebellar development and functioning in behavior.

It must be recognized that the above is a highly abbreviated and skeletal outline of the complexity of the brain structure relationships associated with cerebellar functioning. However, in its gross outlines there appears to be an evolutionary or phylogenetic organization of the cerebellum that is related to behavioral patterns and capacities associated with old brain and new brain functions. These features may contribute to the functional role of the cerebellum as a master regulatory system of sensory-emotional-motor processes [35, 36; 57].

Sixth: The cerebellum is also involved in the regulation of autonomic functions which, of course, are directly related to emotional processes. It is beyond the scope of this paper to review these and other interrelationships and the interested reader can consult Dow and Moruzzi [239]; Fulton [240]; Fox and Snider [241]; Adey and Tokizane [242]; Snider [119; 279]; Hockman, et al. [217]; Doba and Reis [243, 244]; Miura and Reis [245, 246] and Moruzzi [223] for this information. Evidence linking cerebellar function to limbic system activity

has been provided elsewhere [35] and based upon these theoretical speculations, Heath and Harper [247-251] have confirmed cerebellar-limbic-frontal orbital linkages which have been previously proposed to be involved in maternal-social deprivation and isolation-reared behaviors [35]. Further, Reis, et al. [252] have demonstrated that predatory attack, grooming and consumatory behavior in the cat can be evoked by electrical stimulation of the cerebellar rostral fastigial nucleus.

The review of Karamyan [227] reported that cerebellar ablations in mammals produced marked disturbances in sensory, motor, autonomic, and emotional behaviors, specifically enhanced sensitivity to touch and pain with increased scratching and licking reflexes, ulcerations in various parts of the body, loss of hair, various gastrointestinal disturbances, and impairment or abolishment of conditional reflex activity which provides further support for a broader view of cerebellar functioning. Berman, et al. [62] have reviewed additional studies on cerebellar mediation of emotional behaviors (pleasure and aggression) and have described the reduction of violent behaviors in isolation reared monkeys consequent to selected cerebellar lesions.

Peters and Monjan [253] have reported taming effects (marked pleasure reactions [kneading and purring] and stroking around the experimenter's legs) in cats following cerebellar vermis lesions. Similar taming effects were found in squirrel monkeys consequent to cerebellar vermis lesions where it was possible to approach and stroke them without difficulty and no fighting or markedly aggressive behavior could be seen after surgery. These authors, however, could not provide any theoretical rationale for their observed results.

Peters, Bleek and Monjan [254] also reported that reactivity to low level shock (below 200 μa) was significantly altered by cerebellar vermis lesions but not by cerebellar hemispheric lesions. These differential findings are consistent with those reported by Berman [60, 61] and Berman, et al. [62] where marked reduction in aggressive behaviors consequent to cerebellar vermis lesions but not cerebellar hemispheric lesions were reported. The additional findings of Peters, et al. [254] of differential responding to low level and high level shock (above 200 μa) where no differences between the groups for high level shock was reported needs to be placed in the context of Sackett's [159] findings that isolation reared rhesus monkeys would avoid water tubes with low current levels (hyperreactivity) which normal animals would tolerate; yet maintain contact with the water tube that had high current levels, even when tissue damage would occur (impaired pain perception) which normal animals would avoid. These findings need to be clarified since this writer has previously implicated cerebellar functioning in mediating hyperreactivity to tactile stimulation and impaired pain perception in the isolation reared syndrome. The conclusion of Peters, et al. [254] that the findings of Sprague and Chambers [255], where midline lesions of the cerebellum reduced reactivity to painful stimuli, could not be supported, seems premature in the context of other findings. Specifically, Siegel and Wepsic [256] reported a 350 to 400% elevation in nociceptive thresholds to painful tail shocks in monkeys when cerebellar areas whose efferents traverse the brachium conjunctivum were stimulated (paleocerebellar system). These effects were not obtained when cerebellar areas related to the brachium restiformis were stimulated. These authors noted that this differential effect was "not surprising as the brachium conjunctivum carries impulses to the reticular formation (RF) of the mesencephalon, pons, and medulla which in turn affect non-specific thalamic as well as brain stem and spinal cord sensory areas" (p. 192). These findings are particularly consonant with this writer's theoretical neural model that hyperexcitability of cerebellar cortex induced by partial functional deafferentation (isolation rearing) according to Cannon's *Law of Denervation Supersensitivity* results in an excessive outflow of cerebellar cortical efferent activity which inhibits those brain structures associated with somesthetic afferent activity [35; 46]. This model is similar to that advanced by Siegel and Wepsic [256] only the *mechanism* by which cerebellar cortex is activated is different. In addition, the neural

model of this writer interprets the waxing and waning of the hyperexcitable neurons (Ward, [257]) as a possible mechanism to account for the aversion of hyperreactivity to touch *and* the impaired pain perception observed in the isolation reared animal [35]. In this context, the cerebellum should be considered in therapeutic efforts to regulate and control pain (and pleasure) experiences. Additionally, Ward [257] observed that anatomical studies indicated that the hyperexcitable neurons (in the epileptic focus) are "characterized by a striking loss of dendritic spines" (p. 379). This of course, is a classic effect of early sensory deprivation and provides further support for Cannon's *Law of Denervation Supersensitivity* at the central nervous system level [44-46; 48; 258].

Equally significant is Ward's [257] observation that "cells in the epileptic focus are difficult to evoke into activity by afferent synaptic input"--and that this reduction of input to cortical neurons may play a role in the genesis of the hyperactive state that underlies epilepsy" (p. 329). If we substitute the terms behavioral hyperactivity, hyperreactivity and impulse dyscontrol which includes aggression for "epilepsy" then we have an even greater congruence of theory, data and neural mediating processes. Thus, similar neural processes may be involved in different neurofunctional systems which relate to epilepsy and other kinds of behavioral dyscontrol syndromes. It also suggests that an enriched and superthreshold afferent input into cells in the epileptic focus might reduce if not eliminate epileptic seizure discharges where the source of disorder of the epileptic focus resides elsewhere in the nervous system. This possibility has been dramatically demonstrated by Tassinari [259] where he suppressed focal spikes by somatosensory stimuli in an 11 year old boy. This patient had a right hemiparesis that followed a traumatic delivery with subarachnoid haemorrhage that resulted in seizures at 8 years of age in the left fronto-parietal area with spreading to the midline regions. During wakefulness the seizures where characterized by bi-or triphasic spikes with an amplitude of 100-150 μv which recurred rhythmically at 3-4 Hz. The response of the patient to sensory stimuli is best described by Tassinari [259]:

> "(a) Effective stimuli: the continuous rhythmic spikes were suppressed by passive and voluntary movements of flexion or extension of the right foot, such as during the Babinski manoeuvre. Suppression of the spikes by such stimuli--which will be referred to as *effective stimuli*--was constant, reproducible at will, and persisted without habituation. The effective stimuli blocked the spikes regardless of anticipation. In all instances, the spikes reappeared immediately after the end of any form of stimulation. Contemplation of movement only did not suppress the spikes.
>
> (b) Ineffective stimuli: tactile stimuli and passive and voluntary movements involving regions other than the right foot did not modify the spike activity. Visual and acoustic stimuli, mental activity (calculation, reading) and situations of emotional stress did not modify the spike activity." (pp. 574-575)

It is also of interest that the stimuli effective during wakefulness also suppressed spike activity during rapid eye movement (REM) stages of sleep but were ineffective during stages 2, 3 and 4.

Tassinari [259] interpreted the spike rhythm in his patient as a result of a cortical lesion resulting from partial deafferentation. He noted that:

> "Decreased somatosensory input by posterior chordotomy facilitates the synchronization of the cortical activity in the corresponding regions of sensory projections (Anderson, 1962 [260]), and may even produce frank epileptiform activity in the thalamic (VPL nuclei) and cortical (somatosensory) regions (Bava, et al., 1966 [261])." (p. 577)

His discussion of this phenomenon is particularly relevant:

"The electrical activity in chronic partially isolated somatosensory cortex is characterized by: (a) the appearance of paroxysmal bursts of slow waves, which are progressively replaced by spike discharges (Grafstein and Sastry, 1957 [262]; Echlin and Battista, 1963 [263]); (b) by high voltage evoked potentials in response to local electrical stimulation or to afferent stimuli applied to peripheral nerves (Burns, 1950 [264]; Echlin and Battista, 1961 [265]); (c) by the occurrence of subclinical seizures, spontaneous or induced by repetitive stimulation of peripheral nerves (Echlin and Battista, 1961 [265]). Similar features were also found in this case.

The importance of the passage of time in determining the continuity and rhythmic of the spikes is stressed since these patterns appeared 10 years after birth. The rhythmicity of the spikes could result from a progressive increase in the excitability of a partially isolated area, or from a change in the degree and quality of the afferent input "driving" the hyperexcitable neurons into rhythmic activity. It was found by Burns (1950 [264]) and by Echlin and Battista [1961 [263] and 1963 [265] that "the spontaneous" activity in the partially isolated cortex (somatosensory area) was largely dependent on the afferent input to this area" (p. 577).

It is also of interest that Tassinari [259] interpreted the failure of the effective somatosensory stimuli to suppress spike discharges during sleep stages 2, 3 and 4 as a consequence of additional functional deafferentation associated with slow stages of sleep; and that the failure of generalized tonic contractions to suppress spike activity might be attributable to cerebellar inhibitory efferent activity resulting from seizure discharge stimulation of the cerebellum.

Although Tassinari [259] reported the failure of "mental activity" (representing right foot stimulation) to reduce spike discharges it should be noted that Sterman and Friar [266] were able to suppress epileptic seizures following sensorimotor EEG feedback training.

Studies reviewed earlier in this chapter that enriched somatosensory stimulation can reverse behavioral abnormalities of somatosensory deprived (isolation reared) animals are consistent with Tassinari's [259] findings. The findings of Nyhan [64] reviewed earlier herein are particularly cogent since rotational-vestibular stimulation of nine additional DeLange children resulted in a prompt initiation of smiling, laughter and joy. Prior to this vestibular rotational stimulation these DeLange children were characterized by a bland, affectless emotional state. This writer interpreted these additional findings as further support for the role of the vestibular-cerebellar neuroaxis in regulating emotional behaviors [267].

Additional evidence related to this cerebellar hypothesis is Moruzzi's [223] demonstration and description of the diphasic functioning of the cerebellum:

"The stimulation of the interior of the cerebellum in the diencephalic cat is followed by responses which are strikingly different from those observed by Magoun, Hare and Ranson [35, 268] in their decerebrate preparations. They have found that faradization of midline structures evokes a decrease of ipsilateral rigidity, followed at the end of the stimulation by a powerful extensor rebound. This diphasic character of the paleocerebellar response is still observed in Bard's preparation, but the sphere of influence is now no more restricted to the postural tonus. Inhibition and rebound facilitation of autonomic and somatic manifestations of sham rage clearly dominate the picture, pushing into the background the well known postural responses of the mesencephalic cat.

Of course, inhibition of diencephalic activity is observed only if the cerebellar stimulation is timed to occur during an outburst of sham rage; and

then a complete suppression of the infuriated behaviour is seldom observed, but a clear decrease of manifestations of anger, in both somatic and autonomic spheres, is quite a common observation. The outbursts of sham rage are, instead, enormously increased during the rebound period. Of course, the rebound facilitating responses are much more striking, if the stimulation is timed to occur during an interval of placidity. Then no response is observed during the stimulation, but a typical outburst of sham rage is evoked by the rebound response. Of course, it is quite easy to provoke manifestations of anger with sensory stimulations of any kind, but the appearance of sham rage just at, and only at, the very end of the stimulation is a peculiar characteristic, so far as I know, of cerebellar responses.

Curiously enough, the best responses have not been obtained by stimulating the anterior lobe, but the faradization of midline structures behind the *fissura prima*, chiefly in the region of Ingvar's *Lobulus Medius Medianus*, has proved to be more effective. The cerebellar nature of these responses is proved: 1. by their typical diphasic character; 2. by the fact that the cerebellar threshold is lower than for motor cortex; and 3. by the observation of silent areas only 2 mm before or behind strongly active spots." (pp. 85-86)

This biphasic characteristic of cerebellar functioning has a number of implications for a variety of physiological and behavioral functions where wide swings of *hypo* and *hyper* excitability are observed. Specifically, such events could lead to excessive inhibition of cardiovascular and respiratory functions with death as the ultimate consequence. Such a possibility has been previously suggested by this writer as a mechanism underlying the sudden infant death syndrome [269].

Fulton [240] described the importance of the study of Nulsen, et al. [270] which reported upon the importance of *frequency* of cerebellar stimulation in producing inhibitory or facilitatory effects upon motor activity (knee jerk):

"In dog, monkey, and chimpanzee it was disclosed that increasing frequency of cerebellar stimulation caused facilitation rather than inhibition of a cortically induced contraction. The distribution of such facilitatory foci was identical with that of the inhibitory obtained at low frequencies. Inhibition and facilitation could thus be obtained from identical points depending upon the rate at which the point was stimulated. (Below 100 Hz: Inhibition; Above 180 Hz: Facilitation) Nulsen and his collaborators then asked themselves whether this meant that identical pathways were giving opposed effects in accordance with their rate of discharge, or whether there might be two channels of outflow from the cerebellum, one of which would give inhibition, the other facilitation.

To answer this question they sought to destroy individual channels of egress from the cerebellum. When the dentate nucleus was destroyed, particularly its dorsomedial portion, it was found that only facilitation could then be obtained from the anterior cerebellum irrespective of the rate at which a given focus was stimulated. Thus they concluded that the dentate nuclei were primarily responsible for conducting inhibitory effects from the anterior cerebellum. In keeping with this they found that direct stimulation of the uninjured dentate nucleus evoked pure inhibition at all frequencies.

They observed furthermore that when the fastigial nuclei were destroyed, only inhibition could be obtained at all frequencies; similarly, when the uninjured fastigial nuclei were stimulated, facilitation was obtained at all frequencies. In tracing these effects through the cerebellar peduncles and into the brain stem they found that both facilitation and inhibition are mediated through

the bulbar reticular formation, the inhibitory effects passing to the suppressor region of the reticular formation as defined by Magoun and Rhines, the excitatory effects being mediated by its facilitatory areas.

Nulsen points out that the same cerebellar focus which receives a tactile stimulus from a localized cutaneous area will upon electrical stimulation modify motor activity in the same region of the body. Depending upon the frequency of stimulus, either inhibition or facilitation can be evoked from the given focus. The evidence would suggest that the pathways for the two effects diverge and travel through different cerebellar nuclei to the suppressor and facilitatory areas of the reticular formation. (pp. 126-128)

This classic study of Nulsen, et al. [270] has assumed greater significance in the context of the studies of Cooper and Snider [271] that low frequency stimulation (8-12 H_2) of the cerebellum inhibits seizure discharges in monkey and man whereas high frequency stimulation (100-300 H_2) facilitates the abnormal seizures. These issues are far from resolved, however, and Cooper, et al. [231] should be consulted for additional information.

Ball, et al. [272] also reported that cerebellar stimulation produced a variety of behaviors that are seen in the isolation reared animal, e.g. compulsive biting (self-mutilation in the isolate); compulsive licking of forepaws and hindpaws (compulsive oral-genital and toe stimulation in the isolate); and stereotypical circling behaviors seen also in the isolate [274]. Berntson, et al. [273] have also reported complex social behaviors (eating and grooming) consequent to cerebellar stimulation in the cat.

It should be noted that virtually all studies of cerebellar stimulation and lesions which produced marked changes in social-emotional behaviors involved primarily midline (paleo) cerebellar structures with little or no effects when lateral (neo) cerebellar structures were studied. Thus, it would appear that the fastigial nuclei-paleo cerebellar complex is of greater significance for these kinds of behaviors than the dentate nuclei-neo cerebellar complex which would appear to be more related to language and cognitive behaviors. These recent findings amply support this writer's earlier proposal that the cerebellum is involved in complex social-emotional behaviors; that it may well serve as a master regulatory mechanism for sensory-emotional and motor processes; and that it is intimately involved in many of the behavioral and emotional abnormalities of isolation reared animals including disorders of habituation; and in infants and children deprived of somesthetic and vestibular stimulation [35].

A case for an increasing role of the cerebellum in the behaviors of the blind seems plausible. The study of Blass, et al. [275] which demonstrated that congenitally blind subjects with a prevalence of finger-to-hand movements showed significantly greater language skill at encoding complex sentences which portray descriptions of patterned interrelationships among experiences is supportive of this possibility. Their finding that congenitally blind subjects who have a predominance of continuous body touching (touching other parts of the body as objects) exhibited less skillful language products is of considerable theoretical interest within the context of sensory deprivation and stimuli-seeking behaviors as manifestations of dysfunctional states. These authors argue that there is a central role of motor activity in ongoing thought construction and that body movement is a necessary pre-condition for linguistic representation in the blind. The precise role that the cerebellum may have in these processes, particularly for the blind, has yet to be determined.

In concluding this review it would be remiss not to draw the reader's attention to the theories of Bovard [276, 277] which are conceptually very similar to this writer's theoretical orientation.

The above brief outline of the role of the cerebellum in the

processing of sensory information [103; 119], particularly, somatosensory afferents [278]; its influences upon autonomic functions [279]; its interactions with limbic system structures [241; 247-251; 280]; its classic role in the initiation and regulation of motor functions [281, 282; 125, 126]; and its relationship to cerebral mechanisms]283; 125]; its developmental immaturity [209, 210]; and its relationship to problems in clinical medicine [239] has, hopefully, provided a basis for implicating cerebellar mechanisms in a wide variety of developmental behaviors, heretofore not recognized. The suggestion that the cerebellum may be a master regulatory system for sensory-emotional-motor processes [35, 36; 58] is not too far removed from Eccles, et al. [222] conception of the cerebellum as a neuronal machine:

> "The immense computational machinery of the cerebellum with a neuronal population that may exceed that of the rest of the nervous system gives rise to the concept that the cerebellar cortex is not simply a fixed computing device, but that it contains in its structure the neuronal connexions developed in relationship to learned skills. We have to envisage that the cerebellum plays a major role in the performance of all skilled actions and hence that it can learn from experience so that its performance to any given input is conditioned by this 'remembered experience.' As yet, of course, we have no knowledge of the structural and functional changes that form the basis of this learned response." (p. 314)

> "In attempting to gain insight into the way in which its neuronal structure can function as a computing machine it is essential to be guided by the insights that can be achieved by communication theorists and cyberneticists who have devoted themselves to a detailed study of cerebellar structure and function. We are confident that the enlightened discourse between such theorists on the one hand and neurobiologists on the other will lead to the development of revolutionary hypotheses of the way in which the cerebellum functions as a neuronal machine; and it can be predicted that these hypotheses will lead to revolutionary developments in experimental investigation." (p. 315)

In addition to communication theory, cybernetics and neurobiology, it would seem timely and appropriate to add developmental psychology and that, hopefully, this addition will contribute to the "revolutionary developments in experimental investigation," which will be necessary if human behavior and its phylogenetic and ontogenetic antecedents are to be understood.

It is to these ends that these efforts will contribute to the realization of Fulton's [240] perceptive insight that:

> "Motor skills, however, do not make the man, for there is a great reservoir of function concerned with the emotional life of human beings which, little by little, is being brought into the sphere of physiological analysis." (p. 130)

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