

Neural Pathways and Structures in SSAD Theory

The neural pathways and structures of SSAD theory are outlined in this section. Excerpts from the basic paper by Prescott (1970) are presented. The pioneering studies of Heath (1971) with his graphic model of neural pathways and brain structures are illustrated.

<http://www.violence.de/prescott/mp/article.html>.

The comparative anatomy of frontal cortex and thalamofrontal connections provided by Akert (1964) document connections between Medialis Dorsalis (MD) and frontal cortex, specifically, the tripartite structure of MD where *Pars Magnocellularis* thalamic projections defines the frontal orbital cortex

Berman, Berman and Prescott (1974) documented that paleocerebellar decortication but not neocerebellar decortication transformed an adult pathologically violent mother deprived monkey into a peaceful, social and inquisitive monkey. The paleocerebellum has primary connections with the brain stem and limbic system; the neocerebellum with the cerebral neocortex.
<http://www.violence.de/berman/article.html>

Schwarz, Dietrich W.F. and Frederickson, John M. (1970) documented that there are minimal vestibular projection fields in the cerebral neocortex of the rhesus monkey. *Science*, 14 October 1970, Volume 172, p. 280f. <http://www.violence.de/others/sci71ac.html>

Prescott (1992) presents data that support sexual dimorphism in the developing human brain that shows differential coupling of frontal-cerebellar connectivity in male and female brains. These data are based upon NICHD supported research on cranial-facial growth and development, through lateral-skull X-rays, by the Krogman Growth Center, Children's Hospital, Philadelphia, PA. (Solomon Katz, PI and Geoffrey F. Walker, Biometrics Laboratory, University of Michigan).<http://www.violence.de/archive.shtml>

There are statistically significant differences between males and females where females show a greater neuronal interconnectivity between the cerebellum and frontal cortical areas throughout development. Males show no brain maturational frontal-cerebellar connectivity. These findings suggest a more neurointegrative brain in the female than the male; a greater neural integration between cortical and subcortical brain structures; and the observed greater nurturance and peaceful behaviors in the female than the male. The environment plays a major role in the structuring of these relationships and the underlying biology.

Modern MRI and fMRI are needed to confirm these growth pattern differences, their sexual dimorphism and the implications that these findings have for the emotional-social-sexual and mental development of the human male and female and the future of *Homo sapiens*.

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EARLY SOMATOSENSORY DEPRIVATION AS AN ONTOGENETIC PROCESS IN THE ABNORMAL DEVELOPMENT OF THE BRAIN AND BEHAVIOR.

By James W. Prescott, Ph.D.

**From Medical Primatology 1970 (I.E. Goldsmith and J. Moor-Jankowski, Eds.),
pp. 356-375. S. Karger, Basel, New York**

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animal are the marked autonomic changes produced by stimulation of these areas, particularly, the development of sham rage by stimulation of area 14 [51]. More recent evidence has been obtained in implicating the orbital gyri in emotional behaviors in the study of BUTTER *et al.* [11], in which orbital frontal lesions in rhesus produced hyper-reactivity, heightened oral tendencies, and a reduction in aggressive behaviors. As noted above, it is of more than passing interest that sham rage was also produced by cerebellar stimulation [20], which may be suggestive of further support for a possible functional link between the cerebellum and frontal orbital gyri. It should be noted that any cerebellar-orbital gyri linkage that would be mediated by thalamic relay nuclei would necessarily involve the dorsomedial nucleus since ROSE and WOOLSEY [74] have proposed that the frontal granular cortex be defined as that part of the frontal lobe which serves as a projection area for the dorsomedial nucleus. More specifically, it is known that *medialis dorsalis* (MD) consists of three distinct substructures: *Pars paralamellaris*, *Pars parvocellularis*, and *Pars magnocellularis* which project to areas 8, 9, and orbitalis [13 and 14], respectively, within frontal granular cortex [2]. Thus, *Pars magnocellularis* of *medialis dorsalis* (MD) assumes a critical role in any thalamic mediated cerebellar-orbital gyri linkage. Evidence to support this actual anatomical linkage is sparse; however, the study of NIIMI, *et al.* [55] demonstrated terminal degeneration in *medialis dorsalis* subsequent to unilateral lesions of the cerebellar nuclei which is supportive of these speculations. The findings of FOX [30] that *pars magnocellularis* of DM receives pathways from the amygdala and of GUILLERY [35], who traced fibers to it from the midbrain tegmentum and septal region, give greater interpretative significance to HEATH's [41] findings of septal and cerebellar spiking activity in the somatosensory deprived rhesus. It is appropriate to mention the observations of DOW [21] that the extraordinary development of the neocerebellum in the higher primate corresponds specifically to the development of the frontal and temporal association areas and not to the cerebral cortex as a whole, and that neocerebellar projections to the pons in man constitute 1/5 of the whole of the internal capsule (as the fronto- and temporo-pontine fibers). Thus, sufficient neuronanatomical, neurophysiological and behavioral evidence appears to exist to encourage speculation of a cerebellar-orbital frontal-limbic-reticular circuit in the mediation of abnormal emotional behaviors. EVARTS and THACH [28] can be profitably consulted for additional information on cerebrocerebellar relationships.

Needless to say, the above neuronal model is only one of a number of

Physiologic Basis of Emotional Expression: Evoked Potential and Mirror Focus Studies in Rhesus Monkeys¹

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Data are reported which demonstrate functional connections between certain sensory relay nuclei (postero ventro lateral thalamus and deep nuclei of the cerebellum) and brain sites (primarily the septal region and hippocampus) where physiologic activity has previously been shown to be correlated with emotional expression. Using rhesus monkeys prepared with electrodes into relevant specific deep brain sites and over the cortex, two procedures were performed: (i) To demonstrate anatomic-physiologic connections between various nuclear sites, potentials were evoked by applying stimuli between leads of an implanted electrode while recordings were obtained between leads of an electrode at another site; (ii) To demonstrate the nature of the functional interrelation within the sites which the evoked potential studies had shown to be connected, cobalt was implanted into one nuclear mass and sequential electroencephalograms were obtained to record the spread of epileptiform activity from the implanted site through other deep brain sites. By these techniques, the fastigius nucleus of the cerebellum and the postero ventro lateral thalamus were shown not only to be directly connected to each other, but to have direct back

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and forth connections with the septal region and hippocampus. Indirect (polysynaptic) connections were also shown to exist between these sensory nuclei and other sites, including the mesencephalic reticulum, hypothalamus, and orbital cortex, identified in pathways for emotional expression. These physiologic findings support previously reported experimental data and clinical observations which suggest a close functional relation between sensory input and behavioral phenomena (feelings, emotion, and awareness).

INTRODUCTION

In studies conducted in human subjects, rhesus monkeys, and cats during the past 20 years in the Tulane laboratories, designed to help clarify physiologic correlates of behavior, we identified the septal region (Heath, 1954a) and its outflow through the medial forebrain bundle to the interpeduncular nuclei, the hippocampi, and the amygdala as key regions of the integrated brain in expression of emotions and feelings and in awareness. Briefly summarized, our principal observations were as follows:

1. The septal region, the medial forebrain bundle, and the region of interpeduncular nuclei constituted a pleasure and alerting system. When their septal regions were stimulated, patients prepared with deep and surface electrodes became alerted and reported feelings of profound pleasure (Heath *et al.*, 1954a, 1968; Heath, 1964a, 1964b). In contrast, when, during manifestations of psychotic signs and symptoms, patients had reduced levels of awareness and were dysphoric, electroencephalographic (EEG) abnormalities in the form of spikes and slow waves were consistently recorded from the septal region.

2. Ablation of the septal region in animals lowered levels of awareness and produced catatonic signs and impairment of emotional expression (Heath, 1954b).

3. Correlations were observed between activity in sites closely interconnected with the septal region—hippocampus and amygdala—and emotional expression (Heath and Gallant, 1964). With intense emotion, either painful (emergency states of fear and rage) or extremely pleasurable (states of joy or pleasurable recall or anticipation), changes, in the form of high-amplitude spindling, were consistently reflected in recordings from these sites. When feelings of pleasure prevailed, the spindling was noted predominantly in the septal region and at sites in the amygdala. Electrical stimulation of the amygdala and the hippocampus consistently induced intense emergency emotions (fear or rage). Similar aversive or painful emergency responses were observed when stimuli were directed to the midline (periventricular) hypothalamus and to the midline (periaqueductal) mesencephalic tegmentum. Stimulation of many other brain sites, including the basal ganglia, yielded neutral responses, that is, neither aversive nor pleasurable.

Against the background of these extensive data, reports by Harlow and

The fastigius nucleus is conventionally considered part of the oldest cerebellum integrally associated with vestibular activity and without significant influence on higher centers. Our studies suggest that, through influences on structures associated with expression of emotions, feelings, and levels of awareness, vestibular and kinesthetic stimuli, as well as other somatosensory inputs (via specific relay nuclei), profoundly affect the function of the higher nervous system and behavior.

These physiologic data could provide an explanation for the behavioral data, derived from studies in animals and humans, which show that sensory deprivation induces severe alterations in emotion and awareness, as well as for the clinical observations that psychotic behavior with profound impairment of emotional expression is associated with significant impairment of sensory perception.

ACKNOWLEDGMENTS

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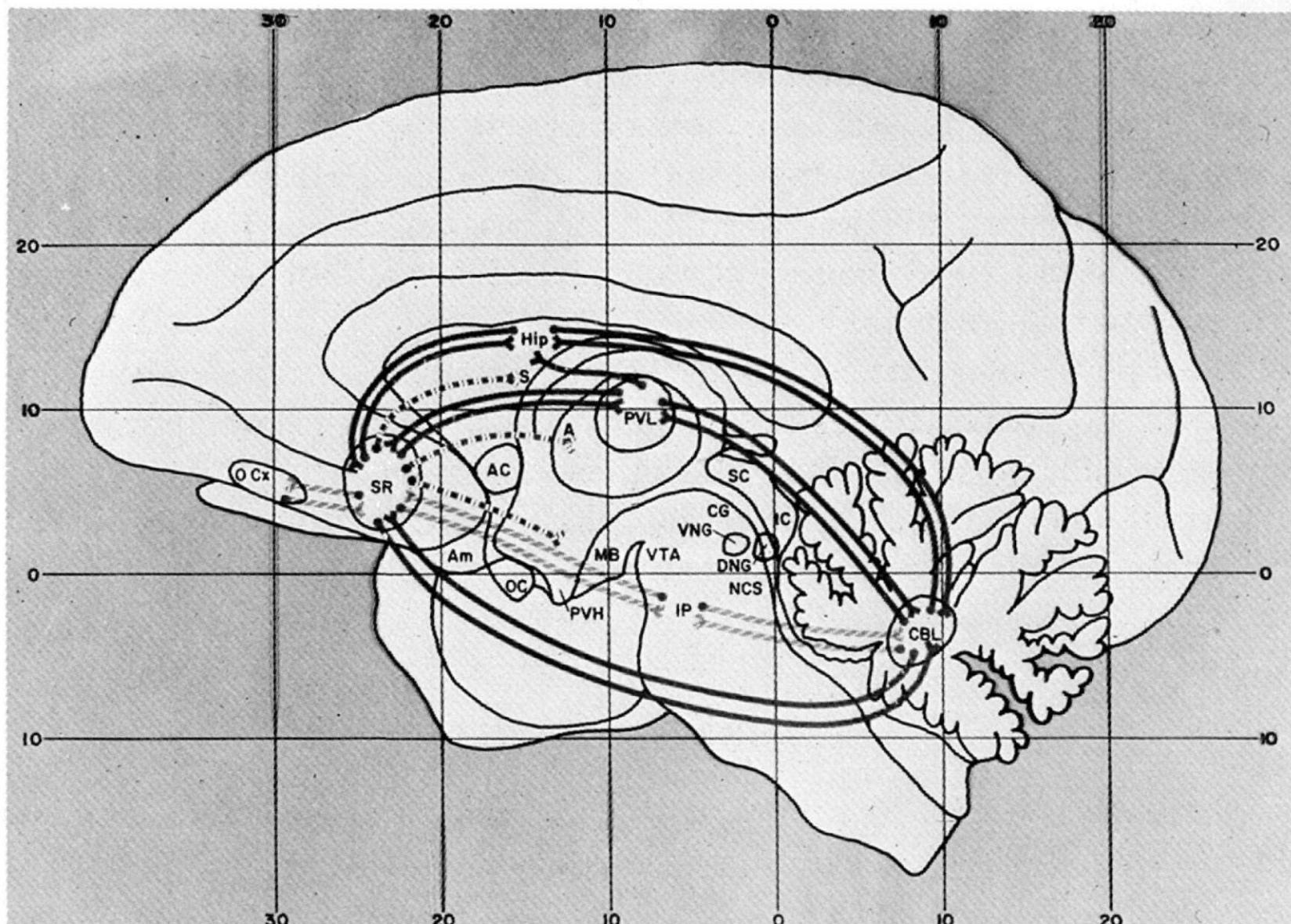
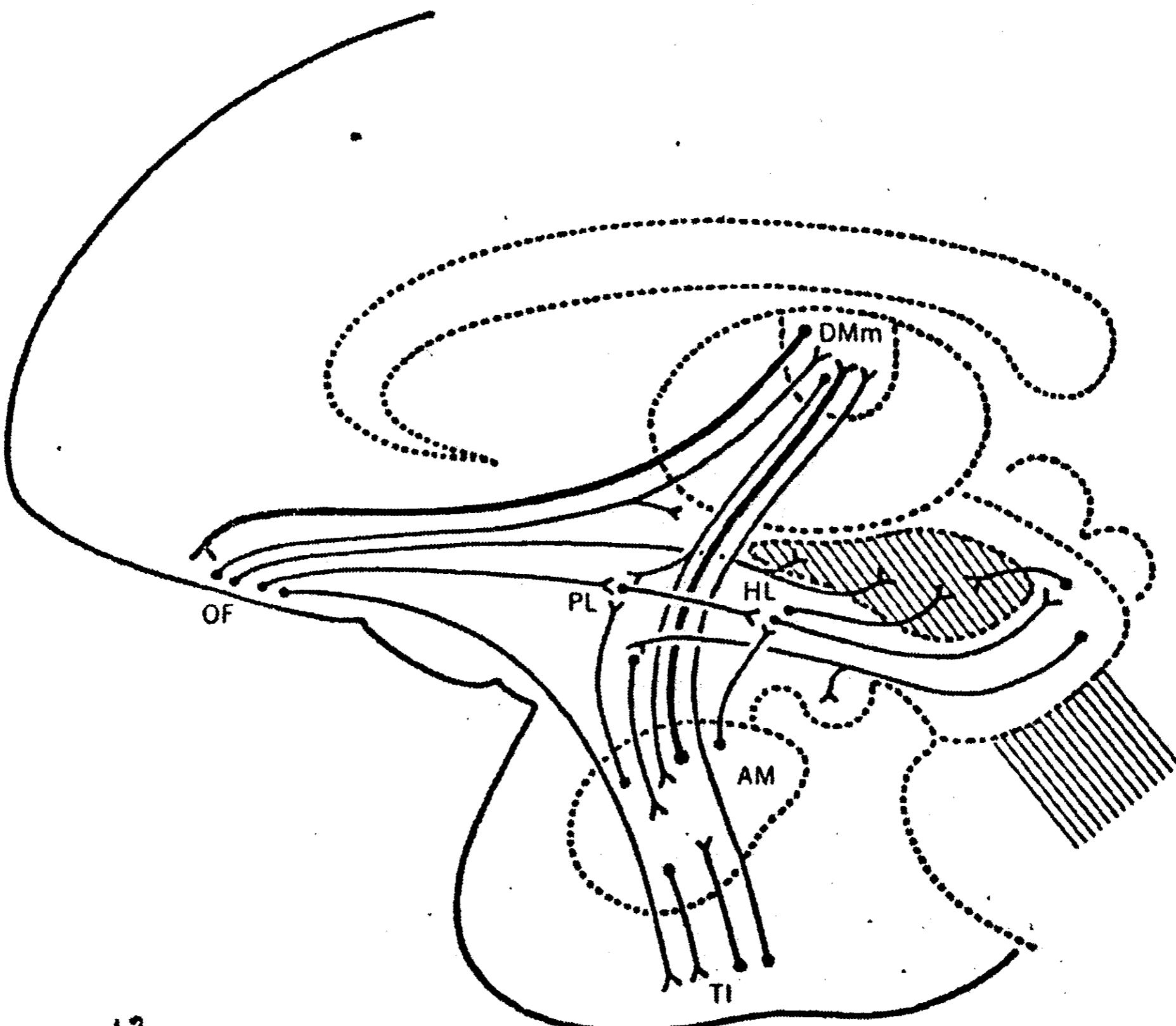


Fig. 11. Diagram of principal pathways of emotional expression based on Tulane studies. Those pathways considered most significant on the basis of our studies are shown as solid lines. (This diagram is not intended to show all pathways involved in emotional expression.) A, anterior thalamic nucleus; Am, amygdaloid nucleus; AC, anterior commissure; CBL, deep nuclei of the cerebellum, indicating fastigius and dentate; CG, central grey substance; DNG, dorsal tegmental nucleus of Guden; Hip, hippocampus; IC, inferior colliculus; IP, interpeduncular nucleus; MB, mammillary bodies; NCS, nucleus centralis superior; OC, optic chiasma; O Cx, orbital cortex; PVH, paraventricular hypothalamus; PVL, postero ventro lateral thalamus; S, septal nuclei; SC, superior colliculus; SR, septal region.

Recently, Snider (1950; 1967) comprehensively reviewed the subject of cerebellar relationships to higher centers. The relationship between the cerebellum and the postero ventro lateral thalamus has long been established (Crosby *et al.*, 1962), but its origin was identified not in the fastigius nuclei but elsewhere in the cerebellum. It is possible that after stimulation of the cerebellar cortex, the impulse passed through one or more synapses into the fastigius nucleus before exciting the thalamus. Such a route might account for the longer latency period that Snider noted. Whiteside and Snider (1953) reported connections to the hypothalamus of short latency. Anand and associates (1959) presented the first extensive evidence of paleocerebellar projections to parts of the limbic system. They did not report latencies after simulation of the cerebellum, and particularly of the flocculonodular lobe, to demonstrate responses over the surface of the hippocampal gyrus, orbital gyrus, and in the amygdala and hypothalamus. They did not stimulate deep cerebellar nuclei or record from the septal region or hippocampus proper.



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Figure Diagram illustrating connections between amygdaloid complex (AM), medial part of dorsomedial thalamic nucleus (DMm), caudal orbitofrontal cortex (OF), and ventral regions of temporal lobe (TI). All except the last-mentioned project directly to the lateral preopticohypothalamic region (PL and HL), which in turn has efferent connections with the mesencephalic reticular formation. From *Brain*, 1962. (Courtesy of the Macmillan Company, New York.)

(From Akert, 1964)

Akert, K. (1964). Comparative anatomy of frontal cortex and thalamofrontal connections. In: Warren and Akert, *The Frontal Granular Cortex and Behavior*. McGraw-Hill, New York).

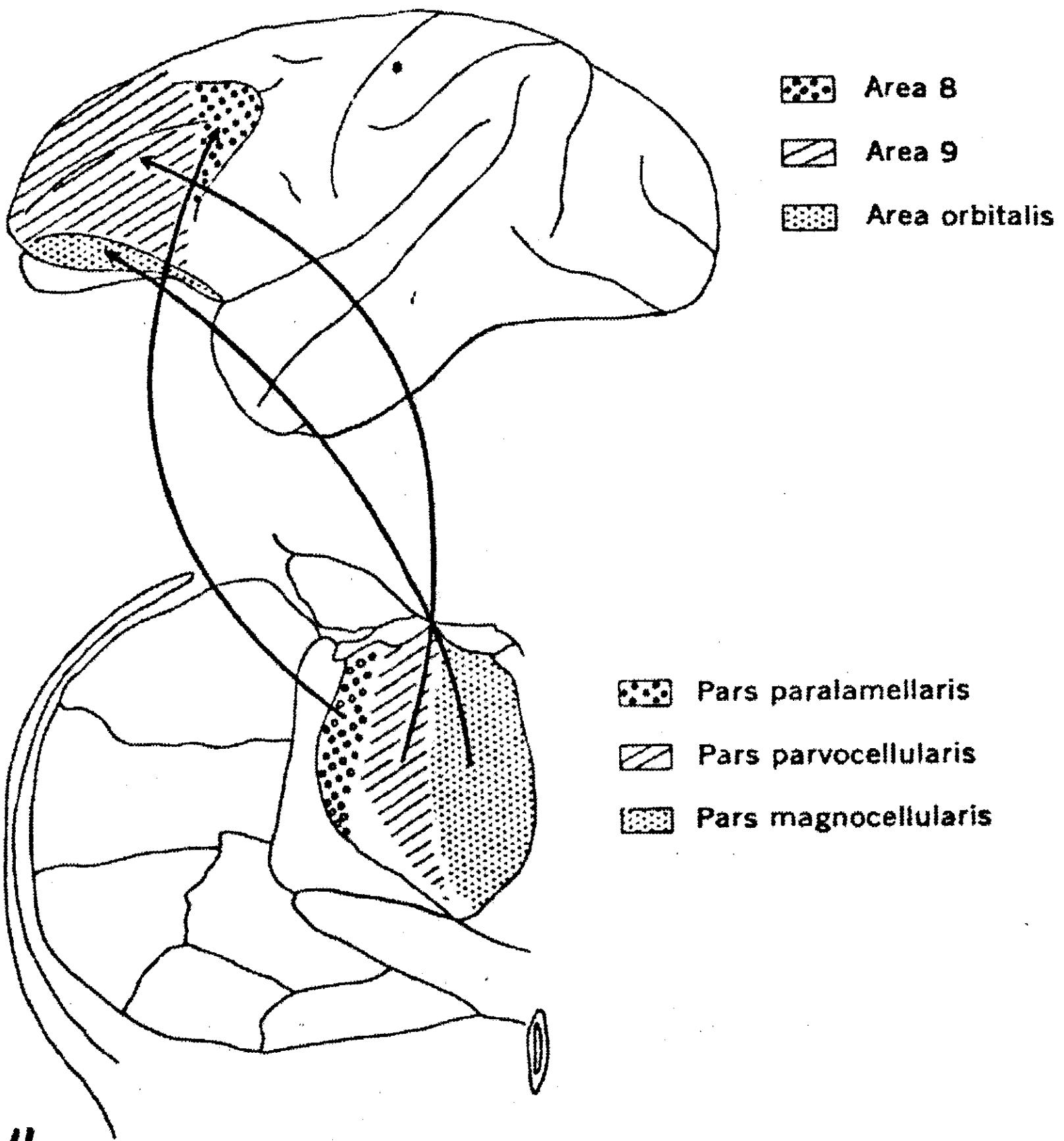
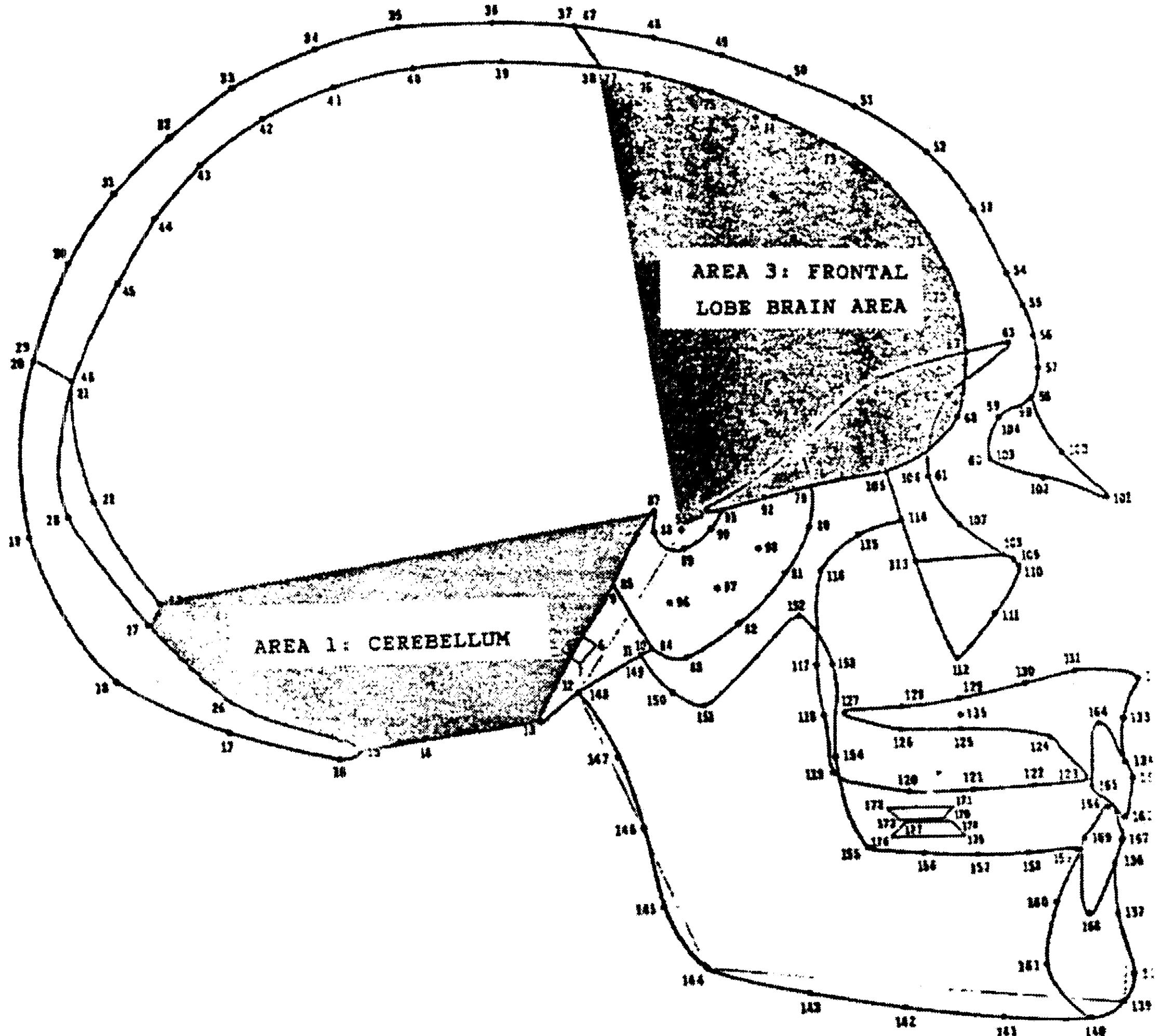
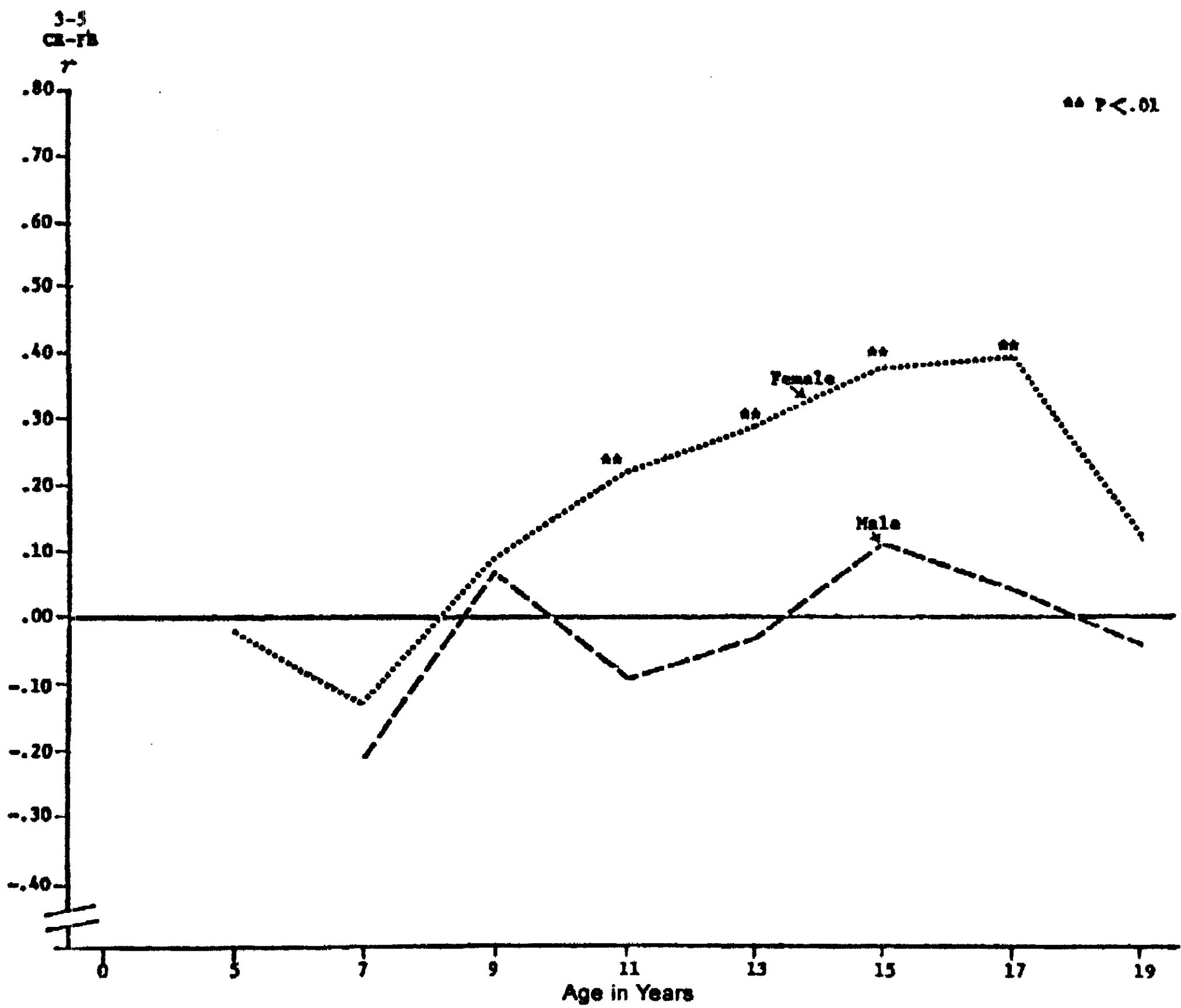


Figure 11 Cytoarchitectural map showing projections from zones of medialis dorsalis to subregions within frontal granular cortex.
(From Akert, 1964)

Akert, K. (1964). Comparative anatomy of frontal cortex and thalamofrontal connections. In: Warren and Akert, *The Frontal Granular Cortex and Behavior*. McGraw-Hill, New York)



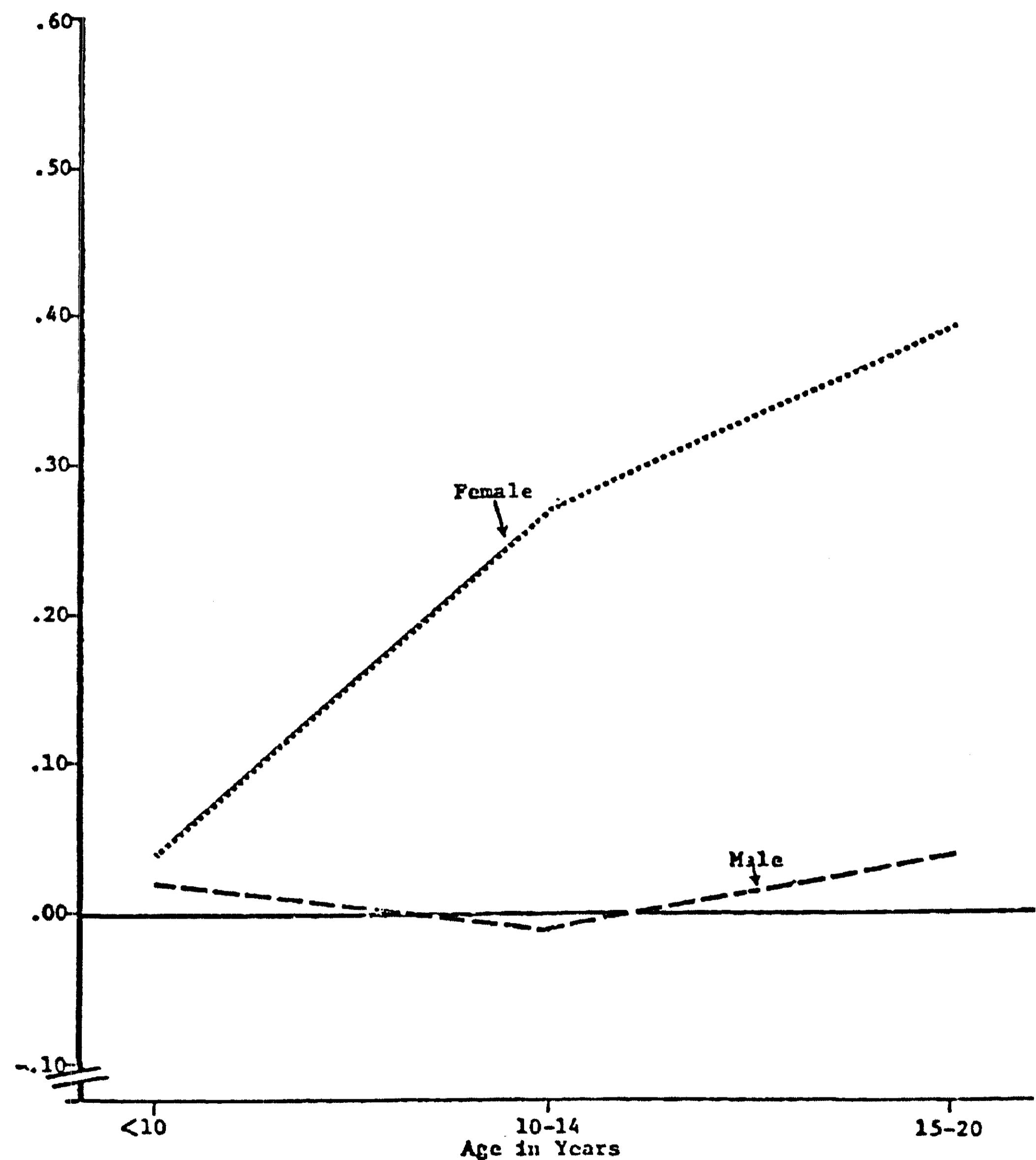
**CEREBELLAR - FRONTAL LOBE BRAIN GROWTH AREA CORRELATIONS
FOR MALES AND FEMALES FROM AGES <10 TO 20 YEARS**



MALE- N	27	74	119	123	94	43	17
FEMALE- N	37	97	160	154	124	66	18

CEREBELLAR - FRONTAL LOBE BRAIN GROWTH AREA CORRELATIONS
FOR MALES AND FEMALES FROM AGES <10 TO 20 YEARS

Correlation
Coefficient



Male-N 101
Female-N 139

300
386

96
136