have been identified and characterized. Apparently, each of these neurons is phenotypically unique. Therefore, it may be possible to compare the molecular composition of the chromosomes of neurons that are developmentally closely related but which have differentiated into functionally unique individuals.

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Rhesus Monkey Vestibular Cortex: A Bimodal Primary Projection Field

Abstract. *Single units in the rhesus monkey* (Macaca mulata) *cortex responded to both vestibular and proprioceptive somatosensory stimuli. This bimodal response characteristic is unlike the modality specificity noted for other primary sensory fields. The vestibular field is located, contrary to previous opinion, within a distinct cytoarchitectonic area outside of area 2.*

A limited projection for the vestibular nerve has been found on the cerebral cortex of the cat (*1, 2*) and the rhesus monkey (*Macaca mulata*) (*3*) with the use of the evoked potential technique while the animals were under deep barbiturate anesthesia. This implies the existence of a primary cortical vestibular field comparable to the primary fields of other afferent systems (somatosensory, auditory, and visual).

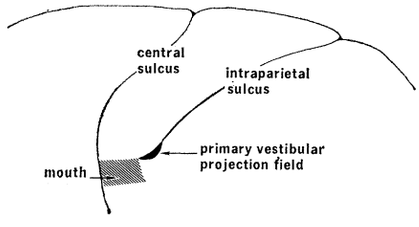

Fig. 1. Rhesus cortical units that responded to vestibular stimulation were located in the black region. Vestibular units were recorded from the depth of the intraparietal sulcus as well. Immediately rostral to this is the primary projection field of the mouth in S1 area 2.

Single units within primary sensory fields have heretofore been found to be strictly modality specific, that is, influenced only by the modality that evoked a slow cortical potential under deep barbiturate anesthesia. This also holds true for all specific relay nuclei. The vestibular system does not appear to fit this scheme of sensory modality specificity, for in the primary relay station, the vestibular nuclei, 80 percent of the units are also influenced by kinesthetic afferents (*4*). Central convergence of these two modalities of "proprioceptive" afferents is apparently essential not only for lower reflex mechanisms but also for the conscious perception of position and movement (*5*). It would be reasonable, therefore, also to expect the primary vestibular cortical field to be an exception to the rule of modality specificity.

The modality specificity of single units within the primary cortical vestibular field in the rhesus monkey (*3*) was qualitatively tested. Single units in the cortex at the lower lip of the distal end of the intraparietal sulcus (Fig. 1) were monitored extracellularly with glass micropipettes (3 to 10 megohms, filled with 2*M* potassium citrate) by the closed-chamber technique in awake monkeys (local anesthesia and Flaxedil). Sensory stimuli included (i) vestibular: bipolar d-c labyrinthine stimulation (0.2 to 1.5 ma) delivered through silver–silver chloride electrodes (round window against bone near posterior semicircular canal); (ii) somatosensory:

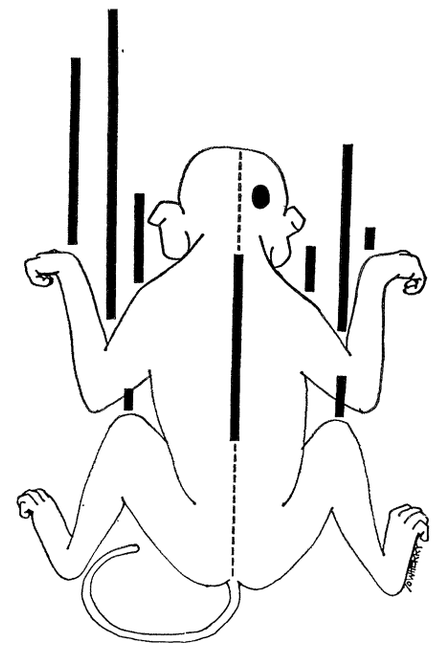

Fig. 2. Joints specifically influencing vestibular units within the primary cortical vestibular projection field. Bar length symbolizes the relative frequency with which the respective joints influenced neuronal activity. The black dot indicates the side of the parietal cortical recording site.

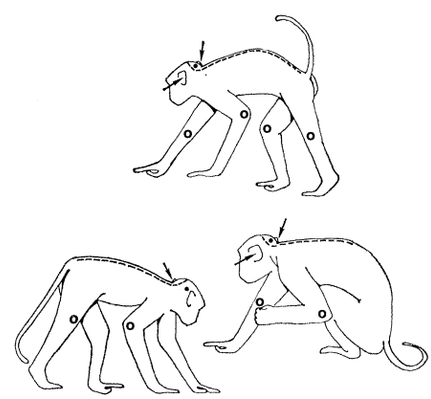

Fig. 3. Three examples of coordinated kinesthetic afferent patterns influencing one cortical unit. Open circles, arrows, and black dots represent, respectively, the effective joint, the labyrinth stimulated, and the cortical recording site. The illustrated joint position produced unit activation, whereas the reciprocal position caused inhibition.

superficial touch and pressure, hair bending, pinching the skin, deep pressure, and joint movement; (iii) auditory: white noise, clicks, and pure tones; and (iv) optic: diffuse illumination intensity changes and movement of contrast patterns in various directions.

Seventy-four units were thoroughly tested. All units responded, but none exclusively to vestibular stimulation. There were no responses to either auditory or optic stimuli. Vestibular units fitted two groups—group 1, those responding to both vestibular and kinesthetic (joint) stimuli; and group 2, those responding to vestibular stimulation and deep muscle pressure.

Group 1 units (convergence of vestibular and kinesthetic afferents) responded to vestibular polarization with different patterns of adaptation: (i) slow adaptation following a short phasic activation peak (dynamic transient), (ii) phasic on-activation followed by phasic off-inhibition or vice versa, and (iii) tonic inhibition with rebound activation.

The activity of these units was invariably influenced by joint movement and the response was always direction specific, that is, activation in one direction and inhibition in the other. When a joint was moved from its resting position toward the activation direction and held there, most frequently unit activation peaked during movement (dynamic transient). In the new static position, the firing frequency (slowly adapting) was higher than that for the resting joint position. Proximal and forelimb joints were more frequently effective than distal and hindlimb joints (Fig. 2). Contralateral joints were more influential than ipsilateral joints were. Often neighboring joints activated the same unit. One unit was seldom influenced by many joints; however, when this did occur, the activation pattern appeared to mimic a moment in a coordinated movement (Fig. 3).

Group 2 units were influenced by vestibular polarization and deep muscle pressure. Effective muscle groups included the proximal flexors and extensors of the contralateral fore- and hindlimbs. Joint rotation also affected these units, but only when the effector muscle was attached to that joint. When the joint was held in a fixed position, pulling the tendon of the effector muscle also produced a response. The exact whereabouts of the afferents producing this effect remains uncertain. It is interesting that A1 muscle afferents have been found to project to the region of the cat's vestibular cortex (*1*).

Our results show that the primary vestibular cortex is not modality specific as classically described for the somatosensory, auditory, and visual systems. If we assume, however, that the cortex will only reflect the sensory specificity, which might be expected for differentiated conscious perception, then one should not expect modality-specific vestibular input. In contrast to the auditory, visual, and somatosensory systems, the perception of position requires integration of at least two different sensory modalities: the vestibular (head position) and the kinesthetic (joint position). Functionally, the afferent input to the primary vestibular projection field may be considered to be as "specific" for conscious orientation as the differentiated, strictly modality-specific inputs of other primary fields are for hearing, vision, and somatosensation.

It has been stated that the rhesus cortical vestibular field is probably located in area 2 of the somatosensory cortex as defined by Vogt and Vogt (*6*). If this were correct, one would expect units immediately rostral to the vestibular field to be affected by stimulation of body regions somatotopically similar to those influential in the vestibular field, since the S1 cortex is organized somatotopically in segmental strips across areas 3, 1, and 2 (*7*). However, single units immediately rostral to the vestibular field were responsive exclusively to tactile stimuli on small receptive fields in the mouth region and were strictly modality specific (*8*). Furthermore, the microelectrode tracts in the vestibular field were located in a cytoarchitectonic area not corresponding to that described for area 2 (*6, 9*), or PC (*10*). In fact, a thorough study of serial sections (Klüver-Barrera stain) of the brains of two rhesus monkeys, one sectioned horizontally and the other coronally, demonstrated that the vestibular field (Fig. 1) does not correspond to the other neighboring cytoarchitectonic areas of 5, 7, and 19 (*6, 9*) or PEm, PF, and OA (*10*).

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Norepinephrine: Reversal of Anorexia in Rats with Lateral Hypothalamic Damage

Abstract. *Injection of norepinephrine in the lateral ventricles of rats recovering from lateral hypothalamic anorexia caused immediate feeding and, frequently, overeating. Intraventricular administration of the α-noradrenergic blocker, phentolamine, suppressed feeding in both normal rats and rats that had recovered from lateral hypothalamic lesions. Feeding is reinforced by ascending medial forebrain bundle fibers that form α-noradrenergic synapses in the hypothalamus and forebrain. Damage to these fibers suppresses feeding by reducing noradrenergic transmission and, hence, the rewarding value of food. Recovery of feeding after hypothalamic lesions coincides with the recovery of noradrenergic reward function.*

Feeding is facilitated by neurons in the lateral hypothalamus (*1*). Electrical stimulation of this area induces feeding (*2*), whereas bilateral damage stops feeding and even causes death by starvation (*3*). On the chemical side, there is considerable evidence that norepinephrine is a transmitter in the feeding system. Injection of norepinephrine directly into the hypothalamus or limbic forebrain causes satiated rats to eat, whereas adrenergic blocking agents suppress feeding and antagonize the facilitating effects of noreprinephrine (*4*).