

DISTRIBUTION OF AFFERENTS FROM THALAMIC ASSOCIATION  
NUCLEI IN THE OCCIPITAL AND PARIETAL CORTEX IN CATS

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The laminar distribution of endings of thalamocortical fibers was studied in the parietal (area 7) and visual cortex (area 17) after ultrasonic destruction of the pulvinar, by the Fink-Heimer method and by electron microscopy. Degenerating fibers and their endings were found in the parietal cortex in all layers, with the greatest concentration in layers III-V. In the visual cortex fibers from the pulvinar terminate chiefly in layer IV. Degenerating fibers terminate on spines and thin branches of dendrites in both the parietal and the visual cortex.

KEY WORDS: synapse architectonics; cerebral cortex; thalamocortical connections

The study of the thalamocortical system of fibers in the projection and association cortex in cats is interesting both for an understanding of the role of individual thalamic nuclei in the organization of neocortical activity and for the study of the principles of the synaptic organization of cortical association and projection areas.

Some workers have found that the pulvinar and other nuclei of the posterior thalamic group project to the suprasylvian gyrus and occipital (visual) cortex [3, 8, 10, 14]. The projection of some thalamic relay and association nuclei to the cortex exhibits a caudo-rostral topographic organization [4].

The object of this investigation was to study the laminar distribution of endings of fibers from the pulvinar of the thalamus in area 7 of the parietal cortex and area 17 of the visual cortex in cats and to determine what types of synaptic contacts they form.

#### EXPERIMENTAL METHOD

Thirty adult cats were used. The pulvinar was destroyed by focused ultrasound, so that there was no risk of degenerative changes in higher structures. The ultrasonic apparatus and method of sonication were described previously [1]. Sonication was carried out transdurally through a burr-hole 10-12 mm in diameter located above the parietal cortex. The parameters of sonication were: frequency 1.98 and 2.76 MHz, intensity 2000 W/cm<sup>2</sup>, duration 0.5 sec. The coordinates of the sonicated structures were determined from the stereotaxic atlas [12]. All operations were carried out under sterile conditions on animals anesthetized with pentobarbital (35 mg/kg). Light-optical and electron-microscopic [6] investigations of cortical areas 7 and 17 and of brain structures along the course of the ultrasonic beam were undertaken 1 h 40 min and 3, 5, and 7 days after sonication. The brain of a normal animal served as the control. Eight cases, which will be described below, were chosen for investigation.

#### EXPERIMENTAL RESULTS

In all the animals studied the greater part of the pulvinar was destroyed, and also the ventrolateral part of the dorsal lateral nucleus. The focus of destruction had clear boundaries formed by a concentration of proliferating glial cells.

On investigation by the Fink-Heimer method terminal degeneration was maximal on the fifth and seventh days after destruction of the thalamic structures, in agreement with the results of electron-microscopic investigation.

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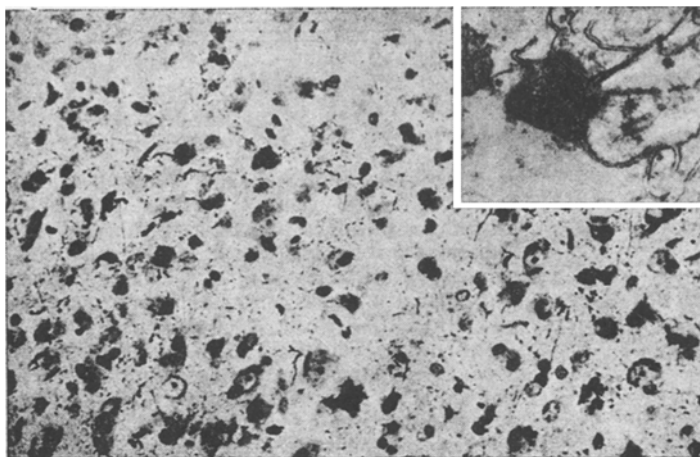


Fig. 1. Degeneration in layer III of the parietal cortex (area 7) on fifth day after destruction of pulvinar. Fink-Heimer method, 400  $\times$ . Electron micrograph of degenerating axo-dendritic synapse of asymmetrical type in layer III of cortex of area 7 on fifth day after destruction of pulvinar shown in top right-hand corner (30,000  $\times$ ).

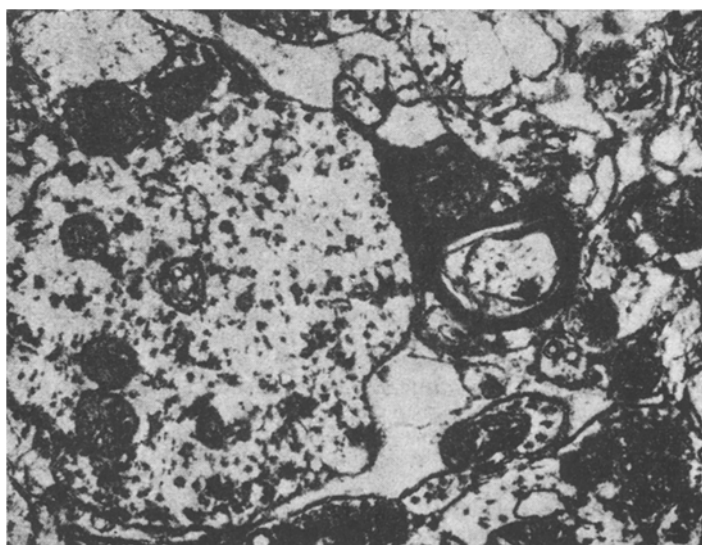


Fig. 2. Degenerating terminal forming axo-dendritic synapse on dendrite of medium caliber in layer IV of parietal cortex (area 7) on seventh day after destruction of pulvinar (25,000  $\times$ ).

In area 7 terminal degeneration was found in all layers including layer I, but the greatest density of degeneration was in layers III-V of the cortex. In area 17 degenerating terminals and fibers were located chiefly in layers IV-V, but a few degenerating fibers and their endings were found at the levels of cortical layers II, III, and VI. The density of terminal degeneration in the parietal cortex was higher than in the occipital cortex, also in agreement with the results of the electron-microscopic investigation (Fig. 1).

On electron-microscopic investigation the initial reaction of terminal degeneration of the degenerating fibers consisted of swelling of the synaptic vesicles, increased osmiophilia of the matrix of the ending, and destruction of mitochondria. This reaction then changed into "dark" degeneration — the form of direct degeneration most widespread in the CNS.

In area 7 degenerating terminals were distributed throughout the depth of the cortex with their greatest concentration in layers III-V. In area 17 degenerating endings were localized mainly in layer IV, but single terminals also were found in layers II-VI. Endings of fibers from the thalamic association nuclei in both projection and association areas of the cortex are presynaptic and form synapses of asymmetrical type [10] (see Fig. 1).



Fig. 3. Degenerating axo-spinous synapse in layer IV of occipital cortex (area 17) on seventh day after destruction of pulvinar (50,000  $\times$ ).

Most degenerating terminals in area 7 had contacts with dendritic spines containing a typical and well-marked spinous apparatus, and also with thin branches of the dendrites. A few degenerating terminals formed axo-dendritic synapses on the trunks of dendrites of medium caliber (Fig. 2). As a rule these dendrites contained mitochondria and numerous microtubules. Axo-dendritic synapses formed by degenerating terminals on the trunks of the dendrites of medium caliber were found in layers III and IV.

In area 17 the degenerating terminals formed axo-dendritic synapses on thin branches and spines of dendrites (Fig. 3) and no degenerating endings were found on the large dendritic trunks. These findings correspond to the ultrastructural features of the synaptic organization of the parietal and occipital cortex of normal cats, characterized by predominance of axo-dendritic synapses on dendrites of various calibers, by contrast with other forms of interneuronal contacts [5, 7].

On light-optical and electron-microscopic investigation of the brain tissue along the course of the ultrasonic beam no degenerative changes were found either in the cortex or in the underlying subcortical structures.

The evidence thus shows that endings of fibers from the thalamic association nuclei were located in the parietal association cortex throughout its depth, with the highest concentrations in layers III, IV, and V, i.e., the distribution was diffuse in character.

Many workers have pointed to the "nonspecific" diffuse character of distribution of the various afferent systems in the parietal cortex (afferents from thalamic relay nuclei and thalamic association nuclei and also callosal fibers) [2, 12-14].

It can be tentatively suggested that the principle of diffuse distribution of the various afferent systems of fibers in the parietal cortex may be an important principle of the organization of its activity.

In the projection cortex (area 17) the character of distribution of endings of the thalamocortical fibers from the pulvinar was closer to the "specific" type of distribution of afferents than in the parietal cortex. These observations agree with the results of many morphological and electrophysiological investigations in which most ascending specific projections were found to be to layer IV and sublayer III<sub>3</sub> of the projection cortex.

According to the results of the present investigation, both light-optical and electron-microscopic, the density of the degenerating terminals in the association cortex was considerably higher than in the visual cortex. The presence of a well-marked representation of the thalamic association nuclei in the parietal association cortex is evidence of the important role of the nuclei in the integrative activity of this region of the neocortex.

Endings of fibers from the thalamic association nuclei in the projection and association cortex, like the endings of most thalamic afferents in the neocortex, form mainly axo-dendritic synapses of asymmetrical types on the spines and thin branches of the dendrites. This emphasizes the fact that the different thalamocortical

inputs share common principles of synaptic organization. Many investigations have shown that most contacts in the cerebral cortex are axo-dendritic synapses; it is these which are affected by destruction of the sub-cortical formations, whereas the axo-somatic and axo-axonal contacts in the cortex remain intact. The special features of the localization of degenerating endings on different portions of the dendrites of neurons in the parietal and occipital cortex are evidence in support of the writers' previous hypothesis concerning the mechanisms of afferent synthesis and integration at the synaptic level [4, 5].

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#### ROLE OF THE LUNG MACROPHAGES IN REGULATION OF THE QUANTITY OF ALVEOLAR SURFACTANT

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Fixation of the rat lung by perfusion through the pulmonary artery prevents the flushing of the macrophages into the lumen of the alveoli and maintains their natural distribution in the hypophase of the alveolar extracellular lining, beneath the film of surfactant. Surfactant synthesis is intensified in the large alveolocytes of the remaining lung 5-7 days after left-sided pneumonectomy, the quantity of tubular myelin in the hypophase of the hypertrophied alveoli is increased, and the surface tension of the lung washings falls. The number of alveolar macrophages is more than doubled in this period. The alveolar macrophages utilize the "excess" of surfactant (tubular myelin) in the hypertrophied lungs and so participate in the regulation of the surface tension of the alveoli.

KEY WORDS: alveolar macrophages; left-sided pneumonectomy; surfactant

The role of the lung macrophages in the utilization of the components of the extracellular lining of the alveoli was first suggested by Macklin [11], who observed the close contact between these cells and the polysaccharide covering of the alveolar epithelium. Later, after fixation of the lung by perfusion through the pul-

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