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THALAMOCORTICAL PROJECTIONS TO THE TEMPORAL AND PARIETAL ASSOCIATION CORTICES IN THE RAT

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SUMMARY

Thalamic projections to the parietal cortex were studied with the Nissl technique and thalamic projections to the temporal cortex were studied with both Nissl and horseradish peroxidase techniques. Results indicated that the parietal cortex receives projections from the lateral thalamic nucleus, pars principalis while the temporal cortex receives projections from the latero-posterior nucleus. It was suggested that the temporal cortex may be homologous to the primate posterior association cortex receiving projections from the pulvinar.

A recent study which examined the literature found considerable uncertainty on the question of the existence of posterior association cortex in the rat [11]. However, based upon anatomical and behavioral research using squirrel (*Sciurus carolinesis*) and tree shrew (*Tupaia glis*), the temporal region receiving projections from the lateroposterior thalamic nucleus (or pulvinar in tree shrew) has been described as association cortex [2,3,6]. Lashley [8] using the Nissl technique demonstrated that a small cortical region designated as the temporal cortex, lying rostrolateral to the striate cortex in the rat receives thalamic projections from the lateral nucleus, pars posterior, while the parietal cortex lying rostromedial to the temporal region receives projections from the lateral nucleus, pars principalis. The goal of this study is to replicate and extend Lashley's [8] anatomical findings concerning the temporal and parietal areas in an attempt to determine whether a basis exists to consider these regions in the rat as anatomically similar to posterior association cortex in the primate.

Twenty-eight hooded rats received ablations intended to approximate either the temporal or parietal cortex as mapped by Lashley (8; see his Figs. 15 and

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18). The temporal region was exposed by drilling the skull with a trephine 2.5 mm in diameter centered 1.5 mm rostral to suture lambda and 6.0 mm lateral to suture sagittalis. Parietal ablations were placed by aspirating the cortical matter exposed by drilling the skull with a 3.5 mm trephine located 1 mm caudal to suture coronalis and 1 mm lateral to suture sagittalis. Following at least 30 days survival, the rats were sacrificed, the brains were removed and they were placed in formalin for a minimum of 7 days. The brains were sectioned coronally at 40 μm and every fifth section was stained with cresyl violet. The sections were then examined under the light microscope for retrograde degeneration, as evidenced by gliosis, loss of cells, chromatolysis and shrinkage, and necrosis.

Seven additional rats received unilateral temporal injections of horseradish peroxidase (HRP; Sigma type VI) using a 10 μl Hamilton syringe mounted on a stereotaxic electrode carrier. As a control, one animal received HRP injections into the left striate cortex. The HRP preparation and processing procedures were adapted from those used in previous studies [5,7]. Three 0.4 μl injections of a 50% HRP solution were placed in the rostral, lateral and medial extents of the temporal cortex. After surviving either 24 or 48 h, the animals were sacrificed and perfused with 50 ml of 6% Dextran dissolved in normal saline followed by fixation with a 1% paraformaldehyde-1.25% glutaraldehyde-4% sucrose solution. The brain was removed and stored for 3 days at 4°C in the fixation solution augmented to a 30% concentration of sucrose. Sections were cut at 60 μm and processed according to the method suggested by Jones and Leavitt [5].

Evidence of retrograde degeneration following parietal lesions was consistently located in the lateral nucleus, pars principalis (A—P 4.8—3.2). This evidence included moderate gliosis and loss of cells in this region similar to that shown in Fig. 1. Larger ablations within the parietal cortex were associated with dense gliosis in this nucleus. In all cases, slight to moderate evidence of gliosis and cell loss was also seen in the posterior thalamic nucleus. Inspection

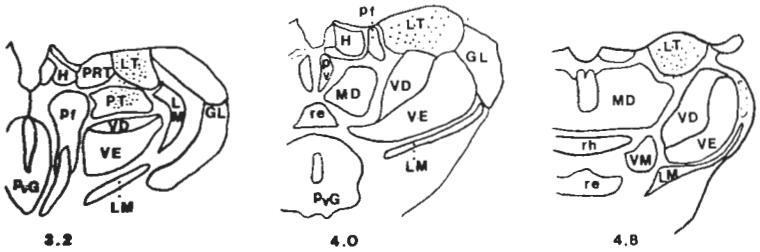

Fig. 1. Thalamic gliosis seen following parietal cortex ablations. Stippling indicates areas of gliosis and cell loss. Abbreviations: GL, lateral geniculate nucleus; H, habenular nuclei; LM, medial lemniscus; LT, lateral thalamic nucleus; MD, mediodorsal thalamic nucleus; pf, parafascicular nucleus; PRT, pretectal nucleus; PT, posterior thalamic nucleus; pv, periventricular nuclei; PvG, periventricular gray; re, reuniens nucleus; VD, ventral thalamic nucleus, pars dorsomedialis; VE, ventral thalamic nucleus; VM, ventral thalamic nucleus, pars medialis. Numbers below each figure indicate A—P level.

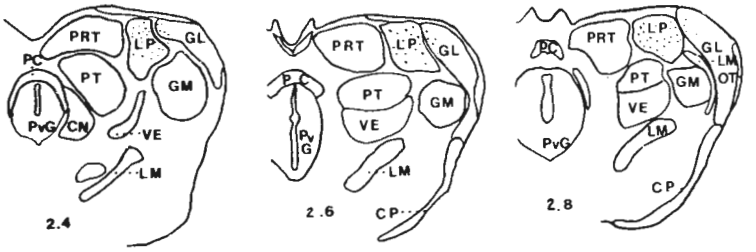

Fig. 2. Thalamic gliosis seen following temporal cortex ablations. Stippling indicates areas of gliosis and cell loss. HRP uptake was also seen in the areas shown as stippled. Abbreviations not included in Fig. 1: CN, cuneiform nucleus; CP, crus cerebri; GM, medial geniculate; OT, optic tract; PC, posterior commissure.

of brains following temporal ablations indicated that gliosis and cell loss was most consistently located within the lateral nucleus, pars posterior (latero-posterior; A—P, 3.0—2.4). Due to the irregular shape of the temporal region, some striate cortex was incidentally ablated in many cases which produced evidence of degeneration in the lateral geniculate nucleus. However, there were several cases in which no striate cortex was damaged and in which moderate gliosis and cell loss was seen only in the lateroposterior nucleus as shown in Fig. 2.

The criterion for establishing retrograde transport of HRP was adopted from Jones and Leavitt [5] and therefore, only neurons showing black or dark brown granularity filling the soma and some proximal dendrites but excluding the nucleus were accepted. Fig. 3 illustrates a single cell in the lateroposterior nucleus which showed retrograde transport following unilateral temporal injections of the HRP solution. False positive reaction products were frequently seen in regions of dense myelination (e.g. corpus callosum, optic tract, internal capsule) as well as endothelial and other vascular cells, but these were not considered as evidence of transport. While the control striate injection indicated retrograde transport to the ipsilateral lateral geniculate nucleus only, temporal injections evinced transport to the population of neurons comprising the lateroposterior thalamic nucleus similar to that shown in Fig. 4. Neurons showing transport of HRP were dense in the rostral portions of the latero-posterior nucleus (A—P, 3.0—2.6) with only scattered HRP cells being evident in the more caudal regions of this nucleus. Scattered HRP-positive cells were also identified in the lateral geniculate nucleus in all cases, perhaps due to diffusion of the HRP solution into the striate cortex.

The results of these studies indicate that the two subdivisions of the lateral nucleus, pars principalis and pars posterior, are distinguishable based upon the cortical regions to which they project. That retrograde degeneration was located in pars principalis and the posterior thalamic nucleus following parietal lesions replicates previous degeneration studies [1,8] and findings with the HRP technique [5]. Perhaps of greater interest is the finding that the temporal region mapped by Lashley [8] receives projections from pars posterior. Since

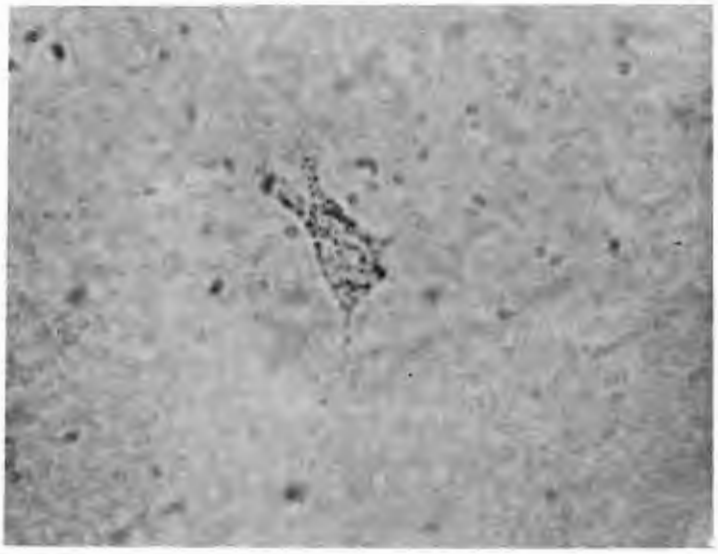

Fig. 3. A neuron in the lateroposterior thalamic nucleus with granularity filling the cell body indicative of retrograde transport of HRP. × 1000.

Fig. 4. Retrograde transport throughout the lateroposterior thalamic nucleus. × 100.

the lateroposterior nucleus has been suggested as homologous to at least a portion of the primate pulvinar [1,4,6,8], it is appropriate to suggest that the cortical recipients of projections from this nucleus may represent a region homologous to primate posterior association cortex. This suggestion is parallel to that of Leonard [9] who cautiously proposed that two cortical areas in the rat which received projections from the mediodorsal thalamic nucleus might be, in part, homologous to primate prefrontal cortex.

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