Excerpts from

The NEURONS and NEURAL SYSTEM: a 21st CENTURY PARADIGM

This material is excerpted from the full β-version of the text. The final printed version will be more concise due to further editing and economical constraints.

A Table of Contents and an index are located at the end of this paper.

A few citations have yet to be defined and are indicated by “xxx.”

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11 Stage 4, Gross Signal Manipulation within the CNS

11.1 Introduction

Baars & Gage published a textbook on Cognition, Brain & Consciousness in 2007. It is largely an introductory text relying more on pictures than detailed discussion and not providing citations to many of the assertions put forward.

Baars & Gage made an astounding statement on page 60, “The great diversity of the neurons in the brain is suggested by Figure 3.4–there are many classes of neurons, neurochemicals, and potential mechanisms of information processing. Our first simplification, therefore, is to focus only on an integrate and fire neurons (see figure 3.3).” The statement is astounding because it eliminates the granular cells of the brain from their considerations. It is conservatively estimated (Section 10.1.2 & 10.1.3) that the granular cells constitute more than 90% of the brain cells in the central nervous system. They have eliminated these from their presentation!! The number of granular cells increases periodically as more investigations using electron-microscopy count granular cells not resolvable using visual microscopes.

Baars & Gage provided a 2nd Edition of their book in 2010 that moved their statement to page 65. The 2nd Edition includes a totally rewritten chapters 8, a major revision to chapter 12 and a new chapter 16 (related to the inferred effect of genes on the neural system based on relatively old interpretations). The preface describes these updates in more detail as necessary because the field is advancing like a “Big Wave” at Waikiki Beach. The book has an added Pull Out section at the front and an extensive Glossary at the rear which are both welcome and important additions. For the first time in a text, the resolution level of MRI techniques have allowed the LGN and the pulvinar to be identified and their relevance discussed (in the rewritten Chapter 8).

The new chapter stresses the two-way communications intrinsic to virtually all thalmo-cortical and trans-cortical commissure via the corpus callosum in line with the assertions of this work. The new Figure 8.10A and B are interesting with regard to the dorsal visual path. Frame A shows the thalmo-cortical paths identified by MRI but frame B relies upon a cartoon to identify the putative cortico-cortical paths within a given hemisphere (even though an associated inset does not justify the cartoon). They then note (page 250), “Keep in mind also that the thalamus is the major input hub for the cortex, and also the major cortex-to-cortex traffic hub. . .However, the basal ganglia operate as a major output hub, for motor control and executive functions.” The subject of two-way communications is also addressed on page 252 relative to vision with “In fact, about 90% of the LGN-
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V1 fibers are ‘running the wrong way’. Above the LGN, everything is a two-way highway. This is a dominant feature of the brain, and it is a great challenge to understand how two-way connections work.” The emphasis was added because of the critical importance of this statement. It is in agreement with the hypothesis of this work.

Sherman & Guillery take a somewhat broader view suggesting that nearly all thalamocortical nerves are two-way and divides them into two (possibly three) distinct categories;

• drivers showing a receptive field similar to that of the original stage 1 sensory neuron.
• modulators showing no characteristic receptive field relatable to the stage 1 sensory neurons
• disrupters (currently speculative and not specifically defined).

These terms have not been widely adopted in the literature. Their nominal circuit configurations are discussed in the next section.

Sherman & Guillery assert the difficulty of defining these circuits or neurons precisely and generally introduce the potential disrupter when some characteristic is not consistently associated with either the drivers or modulators.

11.2 xxxIntroduction

11.3 The major role of the thalamus in the control of neural activity

[xxx review Carpenter & Sutin, pg 538 & 545]
[xxx review Sherman & Guillery, 2000 in vision cabinet in considerable detail.]

In their 1998 paper⁵, Sherman & Guillery make the interesting observation, “One distinct and intriguing possibility is that the major source of a functional drive for corticocortical communication actually goes through the thalamus and derives from cells in layer 5 of one cortical area, which then provide a driver input to relay cells in a higher order thalamic nucleus such as the pulvinar. These thalamic cells then, in turn, send their axons as drivers to layer 4 of another cortical area.” The thalamus, more particularly its shell, the thalamic reticular nucleus (TRN), plays a major command and control role in the animal neural system. Their Conclusion section is carefully constructed. It also includes, “The distinction between drivers and modulators (and disrupters) is important for understanding thalamic relays, and it may prove particularly critical for defining the functional organization of thalamic nuclei where this cannot be studied in terms of readily defined receptive field properties. Possibly, the distinction can be applied much more broadly to the cerebral cortex, as suggested by Crick and Koch, and possibly to other cerebral centers as well.” This observation may become more useful as the character of individual signaling streams are identified. They end with, “The thalamus thus serves as a “gate” not only in the control of information to particular cortical areas about sensory events but also in the control of information passed to other cortical areas from the descending outputs emanating from layer 5.” Their observations are in total agreement with and virtually the essence of CNS signaling in the context of this work.

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⁵Sherman, S. & Guillery, R. (1998) On the actions that one nerve cell can have on another: Distinguishing ‘drivers’ from ‘modulators’ *PNAS USA* vol 95, pp 7121–7126
11.4 Sources of stage 3 connection within the cortex

[xxx use a newer version showing more details]

Figure 11.4.1-1, modified from Hubel, provides a first order description of the connecting paths found between the cerebral cortex and other parts of the cortex. This representation has been expanded over time to show the two-way signaling path associated with each of the paths developed here. See Section xxx).

Figure 11.4.1-1 Major connections between the cerebral cortex and other neural engines. Modified from Hubel, 1988.

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Figure 11.4.1-2 shows a more modern cartoon of the nominal thalamocortical signal paths related to vision provided by Sherman & Guillery, 1998. It stresses the dominant two-way signaling that was unknown to Hubel and colleagues. Note the individual thalamic neurons do not indicate distinct dendritic (non signal inverting) and poditic (signal inverting) inputs to their thalamic neurons which has remained unknown to Sherman & Guillery through 2000. They do not discuss the paths shown except briefly in their caption. The two outermost pairs of neural paths are clearly a two-way signaling pair. The heavier line indicates a “driver” path and the lighter line indicates a “modulator” path. The signal path from the primary afferents (stage 1 sensory receptors through the thalamic relay to cortical area A they define as a first order (FO) relay. The signal path from layer 5 on the left to layer 4 on the right is clearly a stage 3 commissure being relayed through and under the control of the thalamic relay (which is defined in this work as the thalamic reticular nucleus (TRN) rather than more coarsely as the pulvinar in parentheses. They define this path as a higher order (HO) relay. The shorter loop between layers 1-3 on the left to layer 4 on the right is clearly a short distance (less than 2 mm) stage 4 (analog) neuron. It is shown as a combination solid and dashed line to indicate its questionable role as either a driver or modulator. This is the only path that can be associated with the putative dorsal path between individual visual engines delineated as V1, V2, etc.

Baars & Gage have provided an overview (Figure 3.10) attempting to show the similarity in the afferent signal path from the sensory neurons of various modalities to the CNS. Unfortunately, it is rudimentary. As an example, it does not show the well recognized dual nerve paths (tonal and impulse related) found in the auditory modality. Neither does it show the dual nerve paths found in the visual modality (associated with the foveola to the pulvinar and peripheral field to the occipital lobe, both passing through the TRN area first).

11.4.1 Sources of connections from the visual PNS

11.4.2 Sources of connections from the auditory PNS

11.5 Types of stage 3 connections within the cortex

[xxx define fasciculus or whatever it is from Afifi & Bergman]

[xxx see pages 344-347 of Afifi & Bergman Describe as Specific afferent paths (terminating within the brainstem) (terminating within the cerebral cortex)]

Internal association paths (totally within the laminates of the tissue)
Short (u-fiber) paths (generally serving only one gyrus)
association paths (generally between different gyri within same hemisphere)
Commissure (generally between gyri of different hemispheres)

Projecting efferents
  To other cerebral cortex areas (also commissure)
  To deep structures in brain (cortico-spinal) (cortico-thalamic) etc.

**Figure 11.5.1-1** shows one of many interpretations of a figure from Lorento de No.

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**Figure 11.5.1-1** Interconnections of the cerebral cortex. Adapted from Lorento de No.

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**11.6 Reserved**

**11.7 Higher level signal manipulation (information extraction) within the CNS**
The role of major areas of the CNS have been known, on a non-exclusive basis for a long time. The early work of Broca, of Wernicke and others have even identified some areas, at the centimeter square area level, by the functional role they play within either a sensory or motor modality. Investigators are now beginning to uncover specific areas at the few millimeter square area level, and specific neural paths that carry information extracted from the sensory signals delivered to the CNS. The latter are frequently located on a probe and record basis because there remains no way to map and designate neurons at the individual neuron level within the CNS.

11.7.1 The visual modality

11.7.1.1 Lateral geniculate areas associated with angular convergence & accommodation

Carpenter & Sutin have provided the most detailed discussion of the LGN from a morphological and traffic analysis perspectives. However, their work is necessarily condensed in order to provide a textbook, and the condensation may not be adequately supported. Their discussion of the hilus (the point where the LGN buds off of the TRN) is not described with precision. They do not discuss the functional performance of the LGN. They do provide one partial circuit diagram based on traffic analysis. Without delineation of any pia matter boundaries within the folds of the LGN, it is difficult to confirm the fusing of layers they describe. It is clear that layers 1, 4 & 6 typically represent crossed fibers from the contralateral retina. Layers 2, 3 & 5 represent uncrossed fibers from the ipsilateral retina. This ordering places uncrossed fibers from the same retina adjacent to each other in layers 2 & 3.

The role of the LGN is far greater than that of a relay point. Carpenter & Sutin note, “The LGN is not a simple cortical relay nucleus, but a nucleus in which important transformations of visual information occur through physiological processes.” The LGN carries out three main tasks.

1. Converging the images from the two eyes via the Precision Optical System (POS) servo loop.
2. Optimizing the focus of the two eyes via the accommodation subsystem.
3. Merging both the brightness and chrominance images from each lateral half of the visual field into a composite image(s) to be delivered to the visual area of the cerebral cortex (primarily BA 17).

Based on the following subsections, it is possible to define the major elements of the topology associated with the fundamental tasks of the LGN (and to an extent the PGN) but not the specific interconnection of these elements. Topology supports a variety of correlator interconnection designs. In order to define the complete topology, elements and their interconnection, the work of the Hamori, Pasik et al. team of the last subsection must be extended to determine which neurites of the interneurons synapsing with the arriving retinal axons are additive and which are subtractive.

The optic chiasm leading to the LGN serves a special purpose. The cross-over of sensory information within the visual system is on a field of view basis rather than a sensory organ basis. The two left lateral fields proceed to the right LGN and the two right lateral fields proceed to the left LGN.

Each LGN consists of a series of layers with the stage 3 signals from the retinas mapped onto individual layers. The mapped images are aligned so a specific retinal location appears along a single line penetrating all layers. These signals are interdigitated with the brightness signals from the two eyes applied to layers 1 and 2 (called the magnocellular layers). The signals of the first chrominance are applied to layers 3 and 4. The additional chrominance signals are applied to subsequent layer pairs. The layers of the LGN are not always complete and locations in the retina may not appear in every layer of the LGN. Discontinuities in the individual layers appear at the location of the blind spot in the retina. Carpenter & Sutin provide an autoradiograph of part of the

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right LGN of a monkey showing that layers 1, 4 & 6 receive “crossed” stage 3 signals from the left eye. The uncrossed stage 3 signals from the right eye terminate in layers 2, 3 & 5. The area labeled layer 6 is particularly prominent. It has been conventional to describe only six layers of the LGN. However, a fourth set of layers can be described. Their discussion highlights the incompleteness of the histologically defined layers and the little understood physical fusion of the morphological layers (page 527). They also provide a drawing of the LGN (page 526) showing conceptually the incomplete and asymmetrical nature of the major layers. Their photomicrographs (page 528) show a less idealized structure.

Carpenter & Sutin make an interesting casual statement in the caption on page 526. First they describe the magnocellular layers 1 & 2 as constituting the ventral nucleus. They define the parvocellular layers (above 3) as forming the dorsal nucleus, and note, “Only cells in the dorsal nucleus project fibers to the visual cortex.” They note the opposite conclusion on page 535, “Autoradiographic findings confirm that fibers from area 17 project to all cell layers of the LGN.” These assertions and their scant citations are worthy of careful review, and experimental confirmation. The ventral, or magnocellular layers are believed to support the brightness information about the visual field.

Schneider et al. have provided fMRI studies of the LGN at 3 Tesla. The resolution (voxel; 1.5 x 1.5 x 2 mm) is still too coarse to provide any meaningful data beyond traffic flow from the retina. Chen et al. have performed a similar fMRI study of the LGN at 4 Tesla with similar results.

The difficulties involved in exploiting MRI (especially as the resolution increases) has been developed by Friston. The problems of maintaining the head fixed in space become considerable.

Many older documents suggest no neural interconnections between the lamina of the LGN. de Courten & Garey have described the morphology of the neurons of the LGN more recently in considerable detail. They note, “Most neurons have dendrites restricted to the laminae, but some dendrites cross the borders of both magno- and parvocellular laminae.”

O’Brien, Abel & Olavarria have reported a significant anomaly in the structure of the LGN in three monkeys, *Macaca fascicularis*. As many as five “fingers” emanated from the LGN or the hilus where the LGN normally formed. This anomaly may suggest how the LGN is normally formed.

Mapping of the retina onto the LGN is complicated by the bending of the layers of the LGN in the central coronal slice. The center of each layer is displaced caudally from the two ends of each layer. Efforts to locate the point of fixation and the location of the optic disk on the layers of the LGN have also been controversial. Figure 11.7.1-1 compares the proposals of Kupfer (1962) and Hickey & Guillery (1979). The upper half of the figure shows three views of the same LGN from one subject.

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that they assert is typical in spite of the young age. This may be open to some discussion because of the considerably shorter rostral-caudal dimension than found in Kupfer's data. However, this dimension may be one of the very highly variable parameters of the LGN. The mapping on the left is from the angle shown by the arrow at upper right. Hickey & Guillery identified the location of the optical disk based entirely on the idea that the break in the sixth layer at location 2280 microns is coincident with the optical disk of the retina. They did not assert any line of projection existed from that point to a similar point in other layers. This methodology provides no indication of scale on the LGN surface or the location of the retinal point of fixation. Kupfer provides an entirely different interpretation of the retinal map. By exploring the atrophy of cells in the LGN in an 83-year old subject known to have poor eye sight in one eye since his youth, he presents the images in the lower part of the figure. On the left, a retinogram is shown describing the scotoma in the left eye. The height of the scotoma is approximately six degrees and the width is as much as 12 degrees (based on the macula being 5 degrees in diameter). The left LGN shows the damage to the ipsilateral layers from the left eye based on a coronal slice at 5.6 mm from the rostral pole of the LGN. The right LGN shows the corresponding contralateral damage at the corresponding location resulting from the damage to the left macula. These representations suggest the macular alone is mapped onto virtually the entire surface of the LGN viewed from the dorsolateral angle defined by Hickey & Guillery. In this case, the optic disk is located somewhere among the folds of the LGN unrelated to the nominal projection Hickey & Guillery used, and unrelated to any apparent break in a layer of the LGN. The Kupfer evidence is the significantly stronger of the two. However, Malpeli & Baker have indicated surprise at Kupfer's data relative to other species (page 590). Malpeli & Baker have provided detailed mappings onto the LGN for the monkey, *Macaca mulatta*, that may or may not be similar enough to apply to the human. Carpenter & Sutin side with Hickey & Guillery when they summarize the situation from their perspective, “Early studies suggested that macular fibers projected to central regions of the caudal two-thirds of the LGN, but physiological data reveal that its projection is strictly limited to the caudal pole of the nucleus (page 532).”

A strong need to image the reaction of the individual layers of the LGN to retinal stimulation exists. However, it must be at a voxel resolution on the order of 0.1 mm which remains out of sight using fMRI technology. Such imaging would provide a clear understanding of the complex folding found in the LGN away from the “principle plane.” The term principle plane is used here to describe the coronal section through the LGN showing layers that are all parabolic. This is the plane most often shown in texts.

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Pasik and associates have made extensive histological and electron microscope studies of the LGN of monkey, including destruction of the stage 3 neurons leading to and from areas 17, 18 & 19 of the cerebral cortex. In 1973, they changed their previous interpretation of certain axon-axon synapses to more conventional axon-dendrite synapses. In 1974, they presented initial circuit concepts applicable to the LGN. The 1976 paper gave additional electron microscopic data on the synapses and other mechanisms within the LGN and noted the greater than expected interconnection of the neurons.

Mastronarde made extensive studies of the receptive fields of the neurons within the LGN of cat.

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12Pasik, P. Pasik, T. Hamori, J. & Szentagothai J. (1973) Golgi type ii interneurons in the neuronal circuit of the monkey lateral geniculate nucleus Exp Brain Res vol 17, pp 18-34


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during the 1980’s\textsuperscript{15}. While the exploratory effort was extensive, little effort was made to relate the results to a physiological model supporting the measurements. Vigeland et al. performed interesting follow-on experiments in cats but, lacking a schematic, could only speculate on what they described as triadic neurons\textsuperscript{16}. Their descriptions point toward their “lagged” neurons as being three terminal neurons computing the difference in time between the arrival of two different retinal signals and passing that information to the vergence control system of the auxiliary optical nucleus.

11.7.1.1.1 Correlating and merging the contralateral images

Achieving ocular convergence and resulting image summation is a critical function in carnivores and other animals exhibiting binocular vision. The correlation function used to achieve ocular image convergence is performed twice, at nominal precision in the LGN and at high precision in the PGN.

Carpenter & Sutin (1983) assert on page 529, binocular fusion does not occur in the LGN because retinogeniculate fibers end on different layers. An expansion of their conceptual circuit diagram of page 534 overcomes this difficulty with their concept. The contemporary laboratory work of de Courten & Garey (1982) and of Hickey & Guillery (1979), discussed below, also support the opposite view. de Courten & Garey have used Golgi preparations to show that while the stage 3 neurons terminate in separate layers, the associated stage 4 neurons do interconnect the layers\textsuperscript{17}. They state, “Most neurons have dendrites restricted to the laminae, but some dendrites cross the borders of both magnocellular and parvocellular laminae. Somata are also seen in interlaminar zones with dendrites reaching the adjacent laminae.” An important consideration is the method of folding the cerebral sheet to form the LGN. Unfortunately most investigators did not focus on the location of pia matter (outer inactive surface of the cortex) associated with the individual layer. de Courten & Garey did discuss the use of different stains to achieve their results. Many stage 3 neurons do not have soma within the LGN and may be overlooked if the staining does not elucidate myelinated retinogeniculate stage 3 neurons. de Courten & Garey noted the problem of identifying myelinated axons leaving the layers of the LGN. They also noted the presence of neurons with “beaded” dendrites. These Nodes of Ranvier prior to the soma are also seen in the stage 3 neurons leaving the cochlea in hearing (ref, section xxx) and are indicative of stage 3 (phasic) operations. On page 164, they may also have described the TRN imposing an outer “fibrous” layer on the LGN. “In many brains we observed a large number of fibres running between the circumgeniculate capsule and the LGN, oriented mainly perpendicularly to the plane of the parvocellular laminae.” This layer is said to include long, and sparsely-spiny, cylindrical dendrites, as appropriate for TRN material. They also described “neurons positioned between the laminae. “Other fibres, particularly in the interlaminar zones, but also within laminae, run parallel to the layers (illustrated as B in their figure 17).” [xxx dupl. related to figure below.]

While Carpenter & Sutin exhibit a very orderly camera lucida drawing of a six-layered LGN, their principle reference is Hickey & Guillery. In a study designed to explore the variability of the human LGN, Hickey & Guillery note this variability in a histological study of 57 brains from a variety of

\begin{thebibliography}{10}
  \bibitem{17}de Courten, C. & Garey, L. (1982) Morphology of the neurons in the human lateral geniculate nucleus and their normal development \textit{Exp Brain Res} vol 47, pp159-171
\end{thebibliography}
ages\textsuperscript{18}. They provide a series of sample cross sections (each 40 microns thick) for a single LGN that shows only limited similarity to the image in Carpenter & Sutin. They go on to note in their Abstract, “The laminar arrangement within the human nucleus is surprisingly variable. It is always possible to recognize a small segment with two layers (the monocular segment), one with four layers and one with six layers. Often an \textit{8-layered segment can also be seen.}” In stating the purpose of their effort, they go on, “The laminar arrangement in the brains that have been available to us is far more variable than it is in, for example, cats or monkeys, and in the following account we have attempted to document this variability and to analyze it. They discuss the variability in the size of the layers of the LGN in detail. The alignment of the layers is clearly associated with the stereoptic convergence task. The variable extent of the layers relative to the vertical alignment shown in the following figure appears to relate to the non-binocular areas of vision in the two eyes.

On page 244, they assert, “The 6-layered region of the lateral geniculate nucleus may thus represent the basic mechanism dealing with central vision. However, it is worth pointing out that a 6-layered nucleus \textit{per se} is a neuroanatomical myth. The 6-layered portion represents less than half of the nucleus, and one could make a stronger case for treating the nucleus as basically 4-layered (Kaas et al., ’72, and see footnote 2, p. 222). The split into eight layers must be considered of dubious functional significance.” Gray’s Anatomy, 40\textsuperscript{th} Edition makes a passing comment concerning additional layers\textsuperscript{19}, “Most ventrally an additional superficial, or S, lamina is recognized.”

In the context of this work (see also \textbf{Sections 15.6.6 \& 15.6.7} of “Processes\textsuperscript{20}”), the dubious nature of the 8 layers to a histologist can be replaced by the obvious requirement for 8 layers by a physiologist. \textbf{Figure 11.7.1-2} shows a nominal 8 layer LGN from case 71 of de Courten & Garey. They found two cases of 8-layer LGN’s in a group of 12 female monkeys (\textit{Macaca fascicularis}). The first two layers are related to the brightness information of the R-channel propagated to the LGN by the magnocellular pathway. The remaining six layers represent the three chrominance difference channels (O–, P– & Q–) of the parvocellular pathway (Section xxx). It is tempting to suggest the layers originate at the lower right corner where they attach to the tissue of the TRN.

Hillery & Guillery focus on the cross section of the individual layers as well as the overall volume of the LGN without considering how they could be formed from a flat cortical sheet.

Historical practice has been to slice the LGN into thin layers nominally perpendicular to the layers of the LGN. However, the complex folding of the LGN after its initial formation at a hilus of the TRN tissue results in very complex patterns similar to the topographical maps of the western Pennsylvania coal fields where much of the rock formations have been tilted upward from the horizontal. When stained, individual neurons look much like the individual seams of coal pointing upward.

\textbf{Figure 11.7.1-2} Nissl-stained coronal section of LGN of a monkey (case 71). Eight LGN layers can be counted in some regions of the nucleus. Bar: 1 mm. From O’Brien et al., 1997.

\textsuperscript{18}Hickey, T. & Guillery, R. (1979) Variability of Laminar Patterns in the Human Lateral Geniculate Nucleus \textit{J Comp Neur} vol 183: 221-246.

\textsuperscript{19}Standring, S. ed. (2008) Gray’s Anatomy, 40\textsuperscript{th} Ed. NY: Elsevier pg 314

It has become conventional to employ cresyl violet or the Nissl-stain as the stains of choice in LGN histology because they illuminate the location of the soma of individual cells so well. Unfortunately, these stains do not define the paths of myelinated neurons or of the pia matter forming the outer surface of individual cerebral tissue. The use of a Weigert stain to illuminate the myelinated neurons and a stain capable of highlighting the O\textsuperscript{th} layer (the pia matter) of the cerebral tissue would lead to a much better understanding of the morphogenesis of the LGN.

This work has examined several different means by which one or more folds in the cerebral cortical sheet could form the LGN. One method is by a set of opercula forming fingers extending from the tissue of the TRN. The two magnocellular layers (#1 & 2) would be formed by one operculum of the TRN, and the parvocellular layers would be formed in pairs by additional opercula of the TRN. In this arrangement, the white space between the layers would be the channel by which stage 3 neurons from the retinas reach their target location, and by which correlation neurons access each of the pair of neurons in order to form output signals for processing as described below. Figure 11.7.1-3 overlays the above figure of O’Brien et al. to illustrate this candidate. Four opercula are shown emanating from a hilus at the lower right, although at some points the pia matter boundary (shown dashed) of the operculum goes out of the plane of the slice. The approach is compatible with the observations of fingers emanating from an LGN in figure 1b of O’Brien et al. In this case, the fingers would emanate from the TRN and ultimately form the LGN.

**Figure 11.7.1-3** A candidate morphology of the LGN emanating from four opercula at lower right. Each operculum forms two layers of the LGN as shown. The white space between the layers within one operculum supports stage 3 and stage 4 neural paths. Bar: 1 mm. See text. Modified from O’Brien et al., 1997.
There are a variety of other ways that the LGN could be formed from an operculum of the same tissue forming the TRN, the thin layer of tissue enclosing a majority of the thalamus. Figure 11.7.1-4 shows a candidate folding from a single hilus. Note how pairs of surfaces are formed with their active neural surfaces facing each other. The dashed line separates the neural layers of the operculum (not the laminates of the layers) and is defined as the interlaminar zone (IL). This IL should be recognized as different from the space between opercula.

[xxx add words relative to layers and destinations. Use layer labels from Carpenter & Sutin.]
[xxx add words about projections on the layers forming straight lines and the fact that part of the layers are striated.]
Figure 11.7.1-4 Candidate, & idealized, formation of the LGN from a flat neural sheet. The process begins with an operculum originating at a single hilus of the thalamic reticular nucleus. Stage 3 neurons from the retina enter through the hilus and travel along the dashed line until reaching their appropriate location. Exiting stage 3 neurons follow the reverse course before diverging to their target locations. See text.
The subject of where the point of fixation is on each layer and how much of each layer LGN is devoted to the foveola remains open to discussion. Malpeli and Baker\(^{21}\) mapped the visual field representation in the lateral geniculate nucleus of the macaque electrophysiologically and on the basis of their results took issue with Kupfer's ('62) interpretation\(^{22}\) that in the human the fovea is represented in approximately the posterior two-thirds of the nucleus. In the macaque the foveal representation is much smaller than this, and if one accepts that in the human nucleus the optic disc is represented about one third of the distance from the rostral tip, then it is necessary to conclude that in the human nucleus, too, the foveal representation is likely to occupy less of the nucleus than suggested by Kupfer ('62). This work suggests the area of the foveola is largely irrelevant because the major task of extracting detailed information from the image on the foveola occurs in the PGN, not the LGN or visual cortex.

de Courten & Garey studied the detailed interconnections between the neurons of the LGN of the human. “One of our aims was to determine whether differences exist between neurons in magnocellular and parvocellular laminae. Apart from the finding of mainly larger cells in layers 1 and 2, and the absence in them of some relatively rare neurons, no major differences were detected in the neuronal composition or architecture of these compared with the parvocellular laminae.”

Figure 11.7.1-5 shows a summary of their findings where L1 and L2 represent a pair of layers beginning with an odd number. L3 represents the first layer of the next pair. Their findings were well stated. “The majority of neurons have dendrites which remain within the lamina in which the cell body is situated, but some have wide-ranging dendritic ramifications in other laminae. Trans-laminar dendritic trees have been seen linking layers receiving axons from the same and from different eyes. For example, not only are dendrites seen between adjacent magnocellular (the commonest) or parvocellular laminae (linking opposite eyes), but also between magnocellular lamina 2 and parvocellular 3 (linking the same eye laminae). However, no dendrites have been seen spreading across more than two laminae. The relatively infrequent neurons in the interlaminar zones seem to always send their dendrites into the adjacent laminae.”


They define the character of their neurons in detail. In the context of this work, the interlaminar bitufted neuron is clearly a bistratified differing neuron in position to send a myelinated axon down the space between layers and through the hilus to other locations in the brain. While they describe the histological form of their neurons in detail, they do not discuss their physiological role.

de Courten & Garey stress their observation of dendrites (A' & E' as examples) passing out of the confines of the pair of layers into other layers, and particularly between the parvocellular layer 3 and the magnocellular layer 2.

While de Courten & Garey found little difference between prenatal, natal and adult LGN’s, the use of a stain identifying myelin would probably surface significant differences related to the axons within the operculum trench separating the layers.

de Courten & Garey also discuss the “circumgeniculate capsule” surrounding the LGN. In section xxx, this capsule is defined functionally as a portion of the TRN.

de Courten & Garey also discuss the physiological impact of inadequate LGN development. “In the monkey fully mature dendritic arborizations appear generally during the second postnatal month, a time when spatial resolution of LGN cells is increasing and experimental visual deprivation begins to lose its effects. In man visual acuity increases rapidly between birth and six months, reaching adult levels soon after, and he is particularly sensitive to visual deprivation at around this time.”
The Hickey & Guillery paper of 1981 largely parallels that of de Courten & Garey. They appear to modify their position relative to their 1979 paper. From their abstract, “Many magnocellular dendrites cross freely into adjacent layers; whereas the short dendrites of restricted cells rarely cross from one layer to another, the dendrites of extended cells may either be confined to a single layer (intralaminar) or may have a translaminar distribution across two or even three layers. Small cells, with dendrites entirely confined to an interlaminar region, have been seen in the magnocellular division of the nucleus on either side of layer 2.” They assert, “When necessary, the identification of laminar borders was facilitated by counterstaining the sections with cresyl violet.” It is difficult to judge how well the interopercula space (IO) and interlaminar zone (IL) borders were identified from the paper. They noted, “Relatively few axons are stained and the majority of them do not show extensive terminal arborizations. Possibly, this failure of the Golgi method may be related to the fact that many of the axons are relatively thick, and thus myelinated. None of the axons can reasonably be compared to retinogeniculate axons of other species, nor are there any that can be regarded as recurrent collaterals of projection neurons. Some processes that may represent locally ramifying axons or axoniform dendrites have been seen in the parvocellular layers and these are described in a subsequent section.” They clearly identify stage 3 projection neurons, “The beaded appearance characterizes all parts of these translaminar axons, suggesting that synaptic contacts may be established in more than one layer, and also in the interlaminar zones.” They make special note of “Extended” Cells in the Magnocellular Layers. “These, characteristically, have longer and straighter dendrites radiating out from the cell body. Occasionally the dendrites radiate in all directions from a centrally placed perikaryon, but more commonly they occupy only one sector of a sphere having the perikaryon at its center. Sometimes the sector includes more than a hemisphere and sometimes it is narrow.” These appear to be involved in correlation tasks based on this work. Many of their dendrites could not be fully tracked because they were longer than their microtome slices were thick (“400 microns or more in length.”). They also observed beaded dendrites (like those stage 3 neurons in the cochlea of hearing. “The characteristic feature of these cells, apart from the relatively small perikaryon, is the beaded, lobulated, generally axoniform appearance of many of the dendritic processes. Mostly these cells look as though the terminal segments of some, but never of all, of the dendrites have taken on the branching patterns and modes of termination of axons.” Their summary discussion on page 571 is very useful to the student of the LGN.

Hickey & Guillery (1979) provided a definition of lines of projection. “the lines of projection are represented within the human nucleus by the perikaryal elongation and by the tendency of cells to line up in rows” nominally perpendicular to the individual layer. In 1981, they provided a different definition that extends the lines of projection to include multiple layers. “Lines roughly perpendicular to the layers (the lines of projection of Bishop et al., '62; Sanderson, '71) represent points in the visual field, while lines parallel to the layers represent lines in the visual field.” They do not provide substantiation that the points of the various layers are congruent in the retina. They concur with the general view, “In the parvocellular layers of the human nucleus the lines of projection can be defined by rows of perikarya (Hickey and Guillery, '79; Hitchcock and Hickey, '80) and the perikarya themselves are elongated in the direction of the rows. Correspondingly, the dominant dendritic orientation is parallel to these rows.” While this definition can be applied to the coronal slices of the central portion of the LGN, it is not clear how they apply to the rostral portions of the LGN. Based on figures 1 & 4 of their paper, Hickey & Guillery note in 1979, “We do not fully understand how the lines of projection are organized in the rostral parts of the nucleus, where the layers pass through each other in complex patterns, but presumably the lines are significantly disrupted.” Their 1981 discussion of the contents of the IL can not be relied upon based on their statement that they were unable to stain myelinated axons. Their discussion of the parvocellular layers suggest an involvement in stereopsis. “Alternatively, one can treat the number as indicating the degree to which binocular interlaminar zones, and thus the corticogeniculate feedback, may play a role in the binocular interactions related to a particular part of the visual field. Since the several parvocellular layers are architectonically rather uniform, the case for a separation of functions within the individual parvocellular layers is weak (see, however, Schiller and Malpeli, '78) and the second
interpretation merits consideration.” They go on, “Alternatively, one can treat the number as indicating the degree to which binocular interlaminar zones, and thus the corticogeniculate feedback, may play a role in the binocular interactions related to a particular part of the visual field. Since the several parvocellular layers are architectonically rather uniform, the case for a separation of functions within the individual parvocellular layers is weak (see, however, Schiller and Malpeli, ’78) and the second interpretation merits consideration.”

Kupfer has indicated the extent of the projection of the macular area of the retina onto the human LGN24. His figure 6 illustrates the difficulty of applying the lines of projection concept to the LGN. Only the slice representing the coronal slice at 6.3 mm from the rostral pole is consistent with the concept of lines of limited curvature passing through multiple layers. He shows that the five degree diameter macula projects onto virtually the entire surface of the coronal slice at 6.3 mm.

The function of correlating and merging the contralateral images into a single image can not be envisons on a single neural path basis. The process involves comparing an array of retinotopic signals from each contralateral image and minimizing the difference between them by repointing one eye. The procedure is similar to comparing the data bits in two computer shift registers and shifting the data in one register until a minimal difference is found between them.

It is suggested the purpose of a more complete equivalent circuit is critical to the convergence function of the LGN. Each LGN receives two copies of one lateral half of the field of vision. These copies are acquired from slightly different angles of view and from two laterally spaced separated sensors. The LGN is responsible for achieving an optimum summation of these two copies into one composite copy for transmission primarily to one hemisphere of area 17 of the cerebral cortex. In the process of comparing these two copies, it also extracts pointing signals for return to the precision optical system (POS) for optimizing the pointing of the eyes. Simultaneously, it calculates the nominal range to the most prominent object in the field of view. This signal is sent back to the diencephalon for forwarding to the accommodation motor system of the eyes.

Carpenter & Sutin’s circuit diagram of the LGN on page 534 appears over constrained. The circuit shows multiple dendritic paths from the Golgi type II neuron to the axon of the primary retinal afferent. While this situation is possible as a method of reducing the impedance of the dendritic path, it is not appropriate in a fundamental path analysis. It also shows the axon of the Golgi type II neuron reverting back to the synapse between its own dendrite and the primary retinal afferent. This situation is unlikely as it fosters extraneous oscillations in the circuit. They also incorporate a closed loop between the Golgi type I (LGN relay neuron) and Golgi type II neurons, that is prone to oscillation, without suitable justification. They did note the presence of glomeruli in the LGN. Morphologically identified glomeruli are functionally diode matrices. The functional organization of glomeruli have been studied more thoroughly with respect to the olfactory modality (Section xxx).

Figure 11.7.1-6 shows the proposed expanded circuit diagram for one fundamental path. The fundamental stage 3 signaling paths from corresponding locations on each retina are brought to their corresponding locations within the LGN. The pedicles of each stage 3 neuron act as decoders and deliver an analog voltage value to their respective glomeruli (neuropil of other authors). The dendrites of the vertical and the horizontal data registers sense the analog signals from multiple glomeruli. The vertical and horizontal convergence comparators compare the values in the two registers and generate an analog voltage which is passed to the appropriate orthogonal axis of the precision optical system (POS) servomechanism. The algorithm used in these comparators has not been determined. The POS operates to generate an optimum signal level at the comparator input.

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At this condition, the oculars are optimally converged and the signals from the two contralateral signal paths can be summed optimally and passed to the stage 3 neuron passing the signal primarily to area 17 of the cerebral cortex.

Thus, the comparators provide outputs to control the convergence of the eye (possibly in push-pull format) and a separate output to control the accommodation of the eyes. Simultaneously, the comparators provide a gating signal to the geniculofugal Golgi type I commissure indicating when the signal it is receiving from the retinas are adequate to propagate to the cerebral cortex.
Figure 11.7.1-6 The fundamental visual circuit path through the LGN. This path is replicated for the brightness and each chrominance channel. Neural paths proceeding from the LGN to the visual cortex carry “merged” images from the left and right fields of the two eyes. See text. Compare to page 534 of Carpenter & Sutin, 1983.
The specific role of the corticofugal commissure of Golgi type I is not known explicitly at this time. It may indicate whether the cerebral cortex has received an adequately merged image from the two visual fields supplied to the LGN.
11.7.1.1.2 Time correlograms of LGN signals

11.7.1.1.3 Cellular-level synaptic mapping within the LGN’s

Pasik et al. have performed histological experiments on the LGN of monkeys following lesioning of areas 17, 18 & 19 of the cerebral cortex. “The purpose of the present investigation was an attempt to identify the participation of Golgi type II cells in the synapses with retinal afferents by removing the relay neurons through retrograde degeneration after excision of the entire visual cortex in the monkey.” “Total excision of areas 17--18--19 in the monkey leads to disappearance of relay cells and corticogeniculate axon terminals in the LGN (lateral geniculate nucleus). The few remaining neurons can be safely considered as Golgi type II cells.” “Light microscopic examination of Golgi series from adult normal monkeys revealed two types of interneurons in the LGN, both having extremely thin axons.” “Thence, the role of the Golgi type II interneuron could be interpreted at least in part as lateral inhibition.” Lateral inhibition is associated with the poditic neurites of differing neurons in this work.

Hamori et al. have provided detailed information about the synaptic junctions and related tissue within the LGN of the Macaca mulatta monkey. The techniques used and the results obtained are similar to those of Vardi et al. for the photoreceptors of the retina. They noted the common association of two dendritic structures with each axon arriving from the optic nerve (based on their planar electron micrographs). They define this as a triadic combination even though they stress the frequent inclusion of a second axon arriving from the cerebral cortex. The electron micrographs showed both the proposed synaptic area as well as multiple areas exhibiting significant electrical charge. These later areas are proposed to be associated with the electrostenolytic process in this work. They also noted the presence of interneurons that have traditionally been assumed to be inhibitory neurons (differecing circuits).

Figure 11.7.1-7 shows their figure 1. Both frames of the figure show regions of high electrical charge density along the axonal and neuritic lemma of the neurons. This enhanced charge density is frequently asymmetrical with respect to the two lemma of two neurons in close association. It is proposed this high charge density is typically associated with the electrostenolytic supply of energy to the neuron from the glutamate–GABA reaction and is largely unrelated to synaptic activity. Note the presence of multiple asymmetric areas of high charge density in the left Dp of frame a and the extended area of high charge density along the right edge of the Id in frame b. These are probably not related to synaptic activity.

Hamori et al. have addressed the overarching role of their junctions, primarily at the conceptual level. They note, “The functional significance of the synaptic triad in the LGN has been variously interpreted.” and “However, there is little correlation between morphologic findings and neurophysiologic data on presynaptic ‘inhibition.’” Additional information, including the electrical condition of the proposed synapse is necessary to support such an assessment.

Hamori et al. addressed the interconnections as involving Golgi type I, type II and type III (Guillery) neurons without discussing the signaling role of these types. They conclude, “It is obviously impossible to decide on these options on purely anatomical grounds.” They do focus on the Golgi type II neurons as being abundant in their preparations of both LGN and MGN tissue of the monkey.


Guillery’s designation of a type III Golgi neuron has not withstood the test of time. Their findings concerning Golgi type II neurons is consistent with their role as unmyelinated differencing neurons with both dendritic and poditic arborizations. “This suggests that the I-cells of the sensory relay nuclei are chiefly of an inhibitory character. Such a conclusion is supported by a great host of physiological observations on the inhibitory role of Golgi type II interneurons in other areas of the central nervous system.” The state of myelination of the neurons in their figures was not addressed.

They assert, apparently appropriately, “The present investigation attempts to concentrate on the issue of the 'triadic' combinations mentioned above. Although the material upon which these considerations are based derives from the lateral geniculate nucleus (LGN) of the monkey, it must be emphasized that most of the findings and discussion apply with minor modifications to the LGN of other mammals, to other extrinsic thalamic masses (medial geniculate, ventralis posterior lateralis), and, surprisingly, even to some intrinsic nuclei of the thalamus as well.”

Pasik, Pasik & Hamori have provided additional data and discussion related to the synaptic junctions of the neuropil found in the *pars dorsalis* (LGNd) of the lateral geniculate nucleus:

![Figure 11.7.1-7](Figure 11.7.1-7) Electron micrograph of LGN tissue of monkey. Note areas of high electric charge density along the lemma of cells. Complex is described as a type 1 triad. a: glomerular complex with retinal axon terminal (R) in the center, presynaptic to a P-cell dendritic protrusion (Dp) and to an I-cell dendrite (Id) containing flattened or pleomorphic synaptic vesicles. Note also the presence of an axon terminal of cortical origin (Co) and of an unclassified axon ending (A) in the periphery of the glomerulus. b: synaptic triad from a glomerular complex showing the same connectivity as in (a). A spinous process (Sp) emerging from the P-cell dendritic protrusion is postsynaptic to both R and Id profiles, R being also presynaptic to Id. Scales: 1 micron except where indicated. From Hamori et al., 1974.

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geniculate nucleus\textsuperscript{28}. Most of their information is of a statistical nature based on a totally morphological model and measurements from 8 monkeys. “The most striking finding derived from quantitative electron microscopy of the LGNd neuropil in the monkey is the richness of connections between interneurons, a feature not suspected heretofore. Approximately 40 % of the extent of presynaptic membranes present in I-cell profiles is used to contact other I-cell elements through axodendritic and dendrodendritic synapses, in addition to some somatodendritic and dendrosomatic combinations which have not been included in the quantification. This connectivity suggests the existence of an interneuron network which gains in directional complexity when the possible existence of reciprocal synapses, as described in the rat (Lieberman and Webster, 1973), is also considered.”

None of the above papers has made an effort to relate the location of their tissue to the recognized layers of the LGN shown in the previous section. The P-cells of this team correspond to the Type I Golgi neurons of the above figure modified from Carpenter & Sutin. The I-cells correspond to one of the circuit elements in the Golgi type II correlator shown in that figure.

The pairing of neuritic structures of I-cells with each axon do suggest the neurites come from different structures involved in summing the signals from the two eyes into a common output. Alternately, the summing could be accomplished by their P-cells with differencing performed by the I-cells as required to accomplish convergence of the two eyes.

11.7.1.1.3 Achieving optimal focus of the visual field EMPTY

Once the oculars are converged, the signal level at the glomeruli can be maximized by passing the signal summed for propagation to the cerebral cortex to the accommodation servomechanism. This signal is used to vary the focal length of the lens of each ocular until a second level of maximum signal strength is achieved.

The operation of the convergence and accommodation servomechanism are coupled and are responsible for the difficulty of a subject seeing a small object (like an airplane) against a uniform background (the sky) until either proper convergence or proper accommodation is achieved.

11.7.1.1.4 Merging the brightness & chrominance information EMPTY

11.7.1.2 Perigeniculate areas associated information extraction EMPTY

The proposed correlation process is the same as that proposed by Rolls except the junctions between the horizontal and vertical rows consist of synapses acting as electrolytic diodes\textsuperscript{29}.


Research throughout the 20th Century, and without the benefit of non-invasive imaging available today led to the creation of many cartoons relating to the visual modality involving signal paths between several mappable areas such as V1, V2, V3 etc. These cartoons generally predicted traffic paths leading from one of these mappable areas to the next higher level as a matter of logic.

**11.7.1.3 Word serial/bit parallel signal projection within stage 4**

Barbas & Zikopoulos have reported “distinct clusters” of neurons trafficking between various stage 4 and stage 5 engines (page 73), particularly the executive regions of the prefrontal cortex and the thalamus. These observations strongly suggest signaling using multiple parallel neurons carrying information in a word serial/bit parallel format. Such signaling is likely between the PGN and the saliency map of stage 4.

**11.7.1.4 The question of dorsal & ventral paths in information extraction**

The new Figure 8.10A and B in the 2nd edition of Baars & Gage (2010) are interesting with regard to the question of dorsal versus ventral visual path. Frame A shows the thalmo-cortical paths identified by MRI but frame B relies upon a cartoon to identify the putative cortico-cortical paths within a given hemisphere (even though an associated inset does not justify the cartoon). They then note (page 250), “Keep in mind also that the thalamus is the major input hub for the cortex, and also the major cortex-to-cortex traffic hub. However, the basal ganglia operate as a major output hub, for motor control and executive functions.” The subject of two-way communications is also addressed on page 252 relative to vision with “In fact, about 90% of the LGN-V1 fibers are running the wrong way. Above the LGN, everything is a two-way highway. This is a dominant feature of the brain, and it is a great challenge to understand how two-way connections work.” The emphasis was added because of the critical importance of this statement. It is in agreement with the hypothesis of this work.

**Figure 11.7.1-8** from the recent 2nd edition of Baars & Gage (pages 250-252) is an interpretation of a semantic discussion in the philosophical paper by Shipp. It shows their recent thinking in this regard and is in alignment with this work as cited above and in Section 11.1. Shipp’s paper is addressed in detail in Section 11.8.xxx.

**11.7.2 Signal flow within the visual modality**

Peters & Payne produced useful numerical data relating to the primary visual cortex in cat30. Scannell, et al. have recently developed a method of digesting large volumes of trace data and producing very detailed statistical tables31. Mastronarde has provided some early data comparing signals at different points within the visual modality and presenting cross-correlograms to aid interpretation of the data. Usrey and colleagues provided some very useful traffic analysis within the cranium (retina to LGN to occipital lobe) of the cat requiring the use of a variety of recently introduced tools such as multiple probe arrays. They also interpreted their data via cross-correlograms indicating the time delay related to the signals traveling along different signaling paths.

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30Peters, A. & Payne, B. (1993) Numerical relationships between geniculocortical afferents and pyramidal cell modules in cat primary visual cortex *Cerebral Cortex* vol 3(1) pp 69-78

31Scannell, J. Burns, G. Et al. (1999) The connectional organization of the cortico-thalamic system of the cat *Cerebral Cortex* vol 9, pp 277-299
The 2004 work of Izhikevich et al.\textsuperscript{32} is largely a mathematical modeling activity based primarily on cartoons rather than documented waveforms.

Jin et al. have introduced the use of large scale multiple sensing probes to the study of the LGN\textsuperscript{33}. However, their lack of addressing the variability of the delay between a light stimulus and the resulting generator potential at the pedicle of a photoreceptor places their analysis, and specifically their conclusions, in jeopardy of falsification. Their introduction is largely speculative, even though citing multiple sources (but without quotations). Their signals from OFF cells within the LGN may be the result of significant signal processing in this stage 4 engine following the signal processing of stage 2 within the retina itself. Their recorded signals may apply to signals prepared for passing to the precision optical system (POS) introduced in Section 11.7.1 and not to the visual cortex. Their paper followed that of Yeh et al.\textsuperscript{34}. Yeh et al. provided a broader range of data but still did not discuss whether the cats eyes were both used and/or whether they were converged although it was noted the eyes were dilated and their nictating lenses moved aside. No signals were recorded from the retinas and the signals recorded at the LGN were largely of the center-surround type suggesting they were the result of significant undocumented stage 2 signal processing within the stage 2 retina. They did not describe the chromatic aspects of their center-surround signals. Yeh et al. did note that much of their data was acquired within <5 degrees from the point of fixation and all was acquired from within 10 degrees from the point of fixation. Yeh et al. described the character of the X and Y neural pathways more completely than Jin et al. “Whereas most X retinal afferents project to a single dLGN layer (i.e., layer A), and virtually each X dLGN afferent projects to a single cortical area (area 17), Y retinal afferents can diverge into two dLGN layers (i.e., A and C) and Y dLGN afferents can project to multiple cortical areas (for review see Sherman 1985\textsuperscript{35}).” Sherman’s laboratory was a wellspring of information on the signal traffic flow to and from the cat LGN but provided no electrophysiological characterization for these signals. Yeh et al. showed a brief cross section of the cat LGN but referred to Sanderson for orientation of the neural paths at the LGN. The properties of their layers A, A1 and C were not defined. Sanderson did not appear to be concerned with the chromatic aspect of the signals recorded in their A and C layers of the dorsal LGN of cats.\textsuperscript{36,37} Neither Jin et al., Yeh et al. or Sanderson et al. distinguished between the magnocellular and parvocellular layers of the LGN.

A major problem in discussing the above and following sources of data is their universal failure to follow the Scientific Method and identify a null hypothesis that they are trying to confirm or falsify. Without such a null hypothesis, they are unable to describe their hypothesis using a schematic of the portion of the neural system of interest. Section 11.7.1 shows a schematic with at least four distinct output streams associated with the LGN, several of which do not lead to the visual cortex. Much of


\textsuperscript{36} Sanderson, K. (1971) The projection of the visual field to the lateral geniculate and medial interlaminar nuclei in the cat J Comp Neurol vol 143(1), pp 101–117

the data mentioned above and annotated below applies to one or more of these paths.

11.7.2.1 LGN experiments of Mastronarde & colleagues

Mastronarde has provided excellent data on the relationship between neurons in the LGN sharing a visual field with stage 3 ganglion neurons within the retina of the cat\(^{38,39}\). It should become obvious that Mastronarde only explored one retina of each of his cats. As a result, he could not demonstrate the image merging properties of the LGN or the generation of the coarse convergence signals that are the responsibility of the LGN’s. He did allude to the perigeniculate nuclei in passing, the nuclei responsible for generating precision convergence signals derived from foveola data.

Mastronarde stated in Part I (1987a, page 359) of the series of papers, “The retinal electrode was introduced through the side of the left eye and positioned as described elsewhere (16). The LGN electrode was initially placed near A6, L9, usually in the right LGN.” He also used a complicated protocol that conflated many features, such as the center-surround properties of his retinal ganglion cells. While the protocol complicates his analysis considerably, a reinterpretation of his resorted data can aid in confirming the operating methodology shown in the above figure and in material in the sister work of this author\(^{40}\) concerning the role of the LGN in vergence within the visual modality.

**Figure 11.7.2-1** shows the cross-correlogram between two neural types in the LGN. They are comparing the time of occurrence of the action potential created by a ganglion neuron driving the optic nerve and an action potential being generated within a specific layer of the LGN.

The original caption was;

“Bin width is 2 ms in the main graphs and 0.1 ms in the insets. Dashed lines indicate the LGN cell’s base-line firing rate. The correlograms in this and other figures are from maintained activity unless stated otherwise. A: correlogram between OFF-center Xs-cell and its excitatory retinal data.”


\(^{39}\)Mastronarde, D. (1987b) Two classes of single-input x-cells in cat lateral geniculate nucleus. II. Retinal inputs and the generation of receptive-field properties *J Neurophysiol* vol 57(2), pp 381-413

input. The range of the inset is 0-600 spikes/s. In these legends, the following set of numbers will be given for each correlogram: eccentricity of the cell pair in degrees, separation between receptive-field centers in spacings, and, inside parentheses, the number of spikes from the ganglion cell and the LGN cell. 2°, 0 spacings, (7,742, 2,420). B: correlogram between OFF XL-cell and its excitatory retinal input. Inset range is 0-100 spikes/s; 2°, 0.8 spacings, (10,325,2,425)."

The insets provide additional data on the absolute delay between the action potential of the ganglion neuron exciting the LGN and the related action potential generated by the selected LGN neuron. Bins are 2 ms wide in the main graphs and 0.1 ms in the insets. The shape of the histogram in inset A is precisely that of a single action potential because of the way the data was processed. The shape of the histogram in B is somewhat less ideal. They both indicate a delay of only marginally less than 5 ms between the ganglion action potentials and the action potentials generated within the LGN. As Mastronarde indicates, many of his recordings are from extracellular probes. They may be contaminated by signals energy from nearby neurons.

Unfortunately, the particular role of the LGN neuron in the context of the responsibility of the LGN, based on the earlier discussions, cannot be determined precisely from the Mastronarde papers. He asserted, “The LGN electrode was initially placed near A6, L9, usually in the right LGN. Subsequent electrode tracks were placed so as to record from cells whose inputs could be recorded most easily by the retinal electrode; thus most cells most easily by the retinalelectrode; thus most cells (80%) had eccentricities between 3 and 8°.” The locations, A6, L9 were not described further in the paper. The papers suggest such information may be in their citation, Dubin & Cleland, 197741.

As Mastronarde noted, “1. The retinal inputs to cells in the cat’s lateral geniculate nucleus (LGN) were directly recorded to study the basis for the properties of two classes of LGN X-cells: Xs (single) and XL (lagged). The presence of excitatory or inhibitory input to an LGN cell from a particular simultaneously recorded ganglion cell was assessed with cross-correlograms during unstimulated activity.” This protocol is entirely different from that assigned to the experiments by Baars & Gage and Sherman & Guillery (Section 18.3.1.1).

Mastronarde defined his XS and XL cells; xxx

“2. Because neighboring ganglion cells do not fire independently, features in a retinogeniculate correlogram can arise in two ways that must be distinguished I) by a direct effect of the ganglion cell on the LGN cell, or 2) by correlated firing between that ganglion cell and some other ganglion cell that is an excitatory or inhibitory input to the LGN cell. It was possible to determine the origin of correlogram features because features indicating a retinogeniculate effect were distinctly different in timing and strength from features arising solely from correlated firing in the retina.”

“3. The characteristic feature in a correlogram between an LGN cell and an excitatory retinal input was a sharp peak in LGN cell firing rate at the appropriate latency after the firing of the ganglion cell. The characteristic feature for an inhibitory input was a dip in LGN cell firing rate after the firing of the ganglion cell. Typically, this dip lasted 10-40 ms and was followed by a prolonged enhancement in LGN cell firing rate, which may reflect a postinhibitory rebound.”

“4. XS-cells had a single retinal X input (e.g., from a single retina) whose excitatory effect caused most of the LGN cell’s spikes during stimulated and unstimulated activity. There was no conclusive evidence that any XS-cell received excitatory retinal input from either Y-cells or other X-cells of the same center sign. There was usually evidence for inhibition of XS-cells by retinal X-cells of opposite center sign, with receptive fields highly overlapping that of the XS-cell, but rarely evidence for inhibition by Y-cells.

5. X\textsubscript{L}-cells also had only a single excitatory input, but this X input had a relatively weak effect that caused only a minority of the LGN cell's spikes, typically 17% during maintained activity and 29% during visual stimulation. The input's excitatory effect was immediately followed by strong inhibition of the X\textsubscript{L}-cell. X\textsubscript{L}-cells were also inhibited by retinal X-cells of the same center sign that were adjacent (nearest neighbors) to the excitatory input. The strength and latency of both of these inhibitory effects indicate that the inhibition was disynaptic."

Based on the hypothesis of this work, the local neurons of the LGN's should produce two distinctly different signals;

- one set of “error” signals aiding in the stereoscopic vergence of the two-eyed by taking a difference between the signals from the equivalent retinal position of the two eyes. The quality of this error signal may be enhanced by the center-surround character of the inputs from the particular ganglion cells employed.

- one set of signals representing the merged signals from the same hemi-fields of the two retinas following the canceling out of any gross vergence errors in the preceding step. These signals are passed to the occipital lobe in connection with the awareness and alarm modes of visual modality operation, the primary responsibility of the LGN/occipital lobe couple.

The papers of Mastronarde can be re-analyzed for support or falsification of the model presented here. However, the complexity of Mastronarde's protocols, and the lack of any attempt to apply signals from the two retina of the same cat when the eyes were near vergence makes much of the data lack adequate specificity. The difference between the shapes of the main histograms and the shapes of the inset histograms suggest the processing used to obtain them employed different parameters, and probably insufficient number of data cycles to insure statistically relevant histograms. The mean background spike rate of the X\textsubscript{S} cells relative to the peak amplitude would suggest they are summing neurons. The mean background rate of the X\textsubscript{L} cells relative to the peak amplitude, combined with the asymmetry of the histograms, would suggest they are differencing neurons. The response of the X\textsubscript{L} cells would appear much more symmetrical about the background mean if the vertical scale was plotted logarithmically to correspond to the method of encoding employed in the neural system.

Mastronarde's papers built on the much earlier foundation laid by Bishop et al. The Bishop et al papers are discussed in Section 2.6.2.3 of this work. The Bishop papers are best explained in terms of the three-terminal neuron of this work. They can not be explained using the two-terminal neuron and/or the chemical theory of the neuron.

Many investigators have followed Mastronarde. Bair et al\textsuperscript{42} provided considerable data on the macaque monkey relating to signal timing in the photoreceptors, the LGN and the visual cortex. Their method of data collection involved the use of sine wave gratings rather than pulses of light, making the interpretation of their data more difficult. It also leaves many of their assertions without clear and strong support.

Saul & Feidler\textsuperscript{43} have explored the X-class of neurons in the LGN and cortex of kittens. The scope of their experiments can be ascertained from the following, “The lagged/nonlagged distinction arises at the level of the LGN. Retinal ganglion cells of both X and Y types project to neighboring lagged and nonlagged geniculate neurons (Mastronarde, 1987a,b; Humphrey and Weller, 1988a,b; Mastronarde et al., 1991; Hartveit, 1992). Nonlagged cells relay their retinal input to the cortex, often with strengthened surrounds and more transient firing (Hubel and Wiesel, 1961; So and Shapley, 1981; Mastronarde, 1992; Mukherjee and Kaplan, 1995; Usrey et al., 1999; Rowe and

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Fischer, 2001). Lagged cells, in contrast, transform their input via intrageniculate triadic circuits that invert part of the retinal signal to produce feedforward inhibition (Mastronarde, 1987b; Humphrey and Weller, 1988b). Their focus was on “direction selectivity (DS) and timing. DS depends on timing differences within the receptive field. The recognition that DS is a function of the receptive field suggests the neurons they studied were at least indirectly associated with the vergence subsystem of the eyes.

Vigeland et al. have provided additional data supporting the role of their triadic neurons in the formation of vergence signals in cats. Their triadic neurons appear to be analogous to the Golgi type II correlator of [Figure 11.7.1-6]. The following quotation describes their experiments, “Here, using intracellular recording in vivo, we report on the synaptic events underlying the initial spike suppression and leading inhibition in the responses of XL cells to a same-sign contrast stimulus (bright spot for an on-center cell) limited to the receptive field (RF) center.”

The Golgi type II comparators determine the difference in arrival times between the leading pulses of action potentials groups from two photoreceptors aligned to the same point in the external field of view when the eyes are properly converged. When the eyes are not adequately converged, these comparators first generate a boxcar waveform indicative of the difference in time between these initial pulses. Via a lookup table, an action potential is generated with a delay indicating the magnitude of the vergence error. After comparing similar delays generated by other pairs of LGN comparators, the precision optical system (POS) commands the orbital muscles to converge the two eyes. Vigeland et al. corroborate the work of Mastronarde, “XL cells are abundant in the LGN accounting for up to 40% of the relay X-cell population (Mastronarde, 1987a).” Their “blockage” of XL neuron action pharmacologically is consistent with the role of glutamic acid and GABA in powering their X-class neurons.”

Vigeland et al. also hint at additional neurons playing a role in the overall Golgi type II comparators, “Early electron microscopic studies of thalamic relay nuclei including the LGN identified a unique synaptic architecture where the principle afferents make excitatory contacts onto dendrites of relay neurons and also dendritic processes of local inhibitory neurons (Famiglietti and Peters, 1972; Hamos et al., 1985). The latter in turn make inhibitory contacts on the same relay cell dendrites receiving the direct afferent excitation. These triadic synapses are formed on grape-like appendages of the relay cell dendrite and the entire structure is enclosed within a glial sheath. In the LGN, Humphrey and Weller (1988a) found these grape-like appendages to be present at many branch points within the dendritic trees of all XL cells but relatively few XN cells. This circuitry predicts a feedforward inhibition tightly locked (1 ms) to optic tract single inputs, which has been identified in 34% of mouse LGN neurons in vitro and shown to control spike number and increase spike precision (Blitz and Regehr, 2005).” Their paper is worthy of additional study in the context of this work.

11.7.2.2 LGN experiments of Usrey & colleagues

Usrey, Reid and colleagues have provided data similar to that from Mastronarde but based on entirely different protocols and more sophisticated instrumentation.

Figure 11.7.2-2 shows the scope of the experiments of Usrey et al. Their text reviews the results of many investigators and their bibliography is extensive. Their work focuses on the in-vivo signals from action potential generating neurons of stage 3. They assert there are about 100,000 X cells (the majority of the ganglion neurons, with smaller receptive fields) and 5000 Y cells (a class of large ganglion neurons, consisting of less than 5% of the total population) in the cat retina. The location of the cells interrogated in their experiments were typically “off-center” or located in the peripheral field of the cat.

An earlier paper describes their experimental configuration\textsuperscript{45}. They used pulses of light, typically 0.3° x 0.3° as projected onto the cat retina as a source to map the sensitivity of a particular ganglion neuron in the retina and determine its angular receptive field. They repeated the mapping on a neuron of the LGN believed to share a common receptive field and then repeated the process again for a neuron in the occipital lobe of the cerebral cortex. In general, their neurons reflected a center-surround receptive field as shown in the individual local perimetry frames and described in their text. By measuring the temporal waveforms at these locations and performing cross-correlations between the waveforms, they established the histograms shown. The paper does not indicate the difference between or location of the identified LGN areas. They only provided one larger scale histogram (Magno LGN to Layer 4C of the occipital lobe) in the cited paper but it did not apply to any of the histograms shown in this figure. In the large scale histogram, they noted, “The narrow peak (0.7 msec full width at half maximum) displaced 2.4 msec to the right of zero indicates that the geniculate neuron provided monosynaptic input to the cortical cell.”

They did not incorporate any block diagram or schematic in their presentation. Neither did they provide precise descriptions of where their various neurons were morphologically located. This leaves many questions open, such as were the action potentials recorded at the LGN due to the magnocellular neurons arriving from the optic nerve or were they due to stage 3 neurons leaving the LGN for the occipital lobe. While providing significant amounts of information, they do use a variety of terms lacking formal definitions ex., “monosynaptic connection.” Since their paper contained a broad discussion of convergence versus divergence in the signaling channels, it is not clear how this term was used routinely. They did note that only 12 out of 205 neural pairs used in the study exhibited this feature. They do discuss the roles of chemical versus electrolytic (Gap junction) synapses. However, this is only in the context of the conceptual time delays involved in signal transmission not synaptic delay itself.

\textbf{Figure 11.7.2-2} Divergence/convergence in the retino-geniculo-cortical pathway of the cat. The experimental protocol and supporting text does not provide sufficient detail about the signal paths (See text) even though their original caption is quite long (See original paper). From Usrey & reid, 1999.

In a related paper, they also provided Figure 11.7.2-3 showing the temporal similarities of the pulse patterns acquired in vivo within a single channel over a period of time (typically recording 300 action potential patterns resulting from individual stimulations of the retina. They concluded the paper with a short “Methods” section but included no block diagram or schematic of the neural configuration under test. The random arrangement of the spikes to the left and right of the center column in c suggests pulses due to background noise at the input to the LGN from other than the identified retinal ganglion neuron. On the other hand, an experienced investigator with a firmer model of the modality sees some non-random content in the individual rows of spike patterns. The center column shows the pulses generated in response to the identified retinal ganglion neuron when the retina was visually stimulated. The typical ganglion to LGN delay was tightly grouped around 4.8 ms as shown by the correlogram, d.

Frame e reports on a paired spike experiment. Only paired light pulses that caused the generation of paired ganglion spikes were used. Comparing frame c and e suggests the test conditions have changed. The LGN pulses to the left of zero time suggests the identified LGN neuron was responding less robustly to the source of extraneous signals than in c. Frame e also suggests the initial pulse in response to the first retinal ganglion pulse was less effective than in c. However, the initial pulse may have primed the LGN neuron (made it more sensitive to the second ganglion pulse) as shown by the consistency of the second column of pulses at nominally 14 ms after the 1st retinal spike in frame e and the height of the histogram in f. Their calculations of the efficacy of the pulses from the ganglion neurons in frames e & f suggests that when they stimulated the retina using a pair of light flashes spaced 10 ± 0.4 ms apart after a dead time of at least 20.0 ms, the first pulse was significantly less effective than the second.

Combining the two papers, the stage 3 signal
projection time from a specific retinal ganglia to the LGN in the cat is not over 4.8 msec. The comparable time for the signal to traverse from the LGN to layer 4C of the occipital lobe is not over 2.4 msec. The physical distances involved were not given.

Additional information can be extracted from these two papers when an adequate block diagram or schematic is associated with the collected data.

11.7.2.3 PGN experiments of Dubin et al.

Dubin & Cleland have investigated the perigeniculate nucleus of the cat in considerable detail even though “The emphasis of these experiments was on determining the sources of visual input to LGN inter-neurons.” They provided an extensive discussion but without benefit of a block diagram or schematic to guide their analyses47. The paper contains a very extensive set of citations along with a Table 1 comparing the characteristics of cat LGN neurons reported by different investigators. It also gives some coordinates with the head “oriented in standard stereotaxic planes48 and semantic labels for a few otherwise undefined layers of the LGN. The paper is worthy of additional study with reference to the previous section on the cat LGN. Three of their conclusions are worthy of direct quotation here;

“1. Two groups of interneurons that are involved in the organization of the lateral geniculate nucleus (LGN) are described. The cell bodies of one group lie within the LGN; these units are referred to as intrageniculate. The cell bodies of the other group are found immediately above the LGN at its border with the perigeniculate nucleus; these units are referred to as perigeniculate.

2. Intrageniculate interneurons have center-surround receptive fields that resemble those of relay (principal) cells. They can be subdivided into brisk or sluggish and sustained or transient categories. They are stimulated trans-synaptically from the visual cortex and have a characteristic variation in the latency of their spike response to such stimulation both at threshold and for suprathreshold stimuli. The pathway for this stimulation appears to be via cortical efferents to the LGN. Intrageniculate interneurons receive direct, monosynaptic retinal inputs, as determined by recording simultaneously from such interneurons and from the ganglion cells which provide excitatory input to them. Similar to relay cells, they are shown to have one or two major ganglion cell inputs.

3. Perigeniculate interneurons are generally binocularly innervated and give on-off responses to small spot stimuli throughout their receptive field. They respond well to rapid movement of large targets. They respond to electrical stimulation of the retina with a spike latency that falls between that of brisk transient and brisk sustained relay cells. This latency is one synaptic delay longer than that of brisk transient relay cell activation and suggests that they are excited by axon collaterals of these relay cells. Electrical stimulation of the visual cortex is also consistent with this model; the latency of the response of perigeniculate interneurons is approximately one synaptic delay longer than the latency of the response of brisk transient relay cells.”

Elements of these assertions are inconsistent with the block diagram and schematic provided in the section below (Section 11.7.2.4). They actually softened their fourth conclusion when discussing the stimulation of the PGN neurons by signals returning to the diencephalon from the occipital cortex, they conclude;

“The delay of approximately 1 ms that separates the brisk transient relay cell and perigeniculate interneuron responses shown in Fig. 5 is not enough time for even the fastest axons to deliver a signal to the cortex, cross a synapse, and initiate a spike back to the LGN.”


Their discussion included the following description of the PGN neurons (page 423):

“Perigeniculate intemeurons

These cells are clearly distinguished from relay cells both by their receptive fields and by their anatomical position. Such units in the cat with binocular, nonconcentric, on-off receptive fields have been noted in passing by numerous workers. Except for one recent report that placed all such cells above layer A-a point we now confirm—no prior study had paid careful attention to the position of these cells relative to the geniculate laminae. It is interesting to note that in those studies where the data presented allow cell position to be plausibly assumed, the on-off cells reported almost all lie just above layer A. This region comprises the perigeniculate nucleus. Our present findings, and the evidence of Sanderson that the retinotopic mapping of the laminated LGN extends continuously and predictably into and through the perigeniculate region, suggest a more integral relationship between these two regions than may previously have been supposed. These comments are in agreement with the morphological map of Section xxx.

11.7.2.4 A block diagram/schematic supporting Usrey and Mastronarde papers

Figure 11.7.2-4 provides a block diagram of stages 0 through 4 of the visual modality relative to the experiments of Usrey and associates discussed above. Stage 0 is included because the amplitude profile of the light pulses used in the experiments may play a role. Even the difference between a mechanical and an electronic shutter forming the light pulses can be relevant. Clearly the state of adaptation of the stage 1 photoreceptors is relevant. The character of any stage 2 signal processing related to the configuration of the bipolar (summing) neurons and the amacrine and horizontal (differencing) neurons is also important as is any stage 2 signal processing associated with the analog inputs to the stage 3 ganglion (encoding) neurons. The latter frequently involves bandpass filtering (shown as #2 F in the figure) that may be different for the paths following the summing and differencing inputs. Finally, the actual code used by the encoding neurons affects the detailed structure of the actual action potential pulses generated.
Figure 11.7.2-4 Block diagram/schematic underlying the Usrey model. Bottom; the overall block diagram of the first five (including stage 0) stages of the visual modality applicable to the Usrey experiments. The stage 3 circuits between the LGN and the occipital lobe and within the CNS are not shown in detail. Top; The coarse stage 3 signal projection circuit associated with the peripheral retina between the stage 2 signal manipulation, and the stage 4 LGN is shown in expanded form. Inset; cross-correlogram of probe P2 relative to probe P1 on a logarithmic rate scale. See text.

The stage 2 signal manipulation is different for the peripheral retina and the foveola of the retina as illustrated in stages 3 and 4. Usrey and colleagues were clearly interrogating the coarse channel associated with the LGN and the peripheral retina. Attempting to interrogate the fine channel is much more difficult protocol-wise. The receptive fields for the stage 3 ganglion neurons and the associated stage 4 perigeniculate neurons are much smaller and the related neurons within the stage 4 occipital lobe may not be locatable. As noted above, Dubin & Cleland documented this separate path in considerable detail in 1977. Cleland and colleagues presented a large amount of information about the retinal to LGN/PGN of the cat during the 1960-70 time period, including comparisons between different investigators\(^4\). However the data was very early and without benefit of any block diagram/schematics. They struggled with minor movements of the cats eyes (~1/4 degree) even when believed to be well sutured to a support ring. No notice was taken of the much smaller angular tremor of the normal eye. They did use the term S potential but did not describe its origin.

Probe P1 is shown in both frames as interrogating the output (pulse stream) of the identified retinal ganglion neuron near its soma. Probe P2 is shown in both frames as interrogating the output of the ganglion neuron near its pedicle (at least a few millimeters distant). Usrey also interrogates a

neuron at the occipital lobe (P4) that he interprets as directly associated with the neurons probed by P1 and P2. However, no evidence could be found as to whether his intermediate probe was in fact at either the input to the LGN (P2) or at the output of the LGN (P3). The LGN is a complex structure with the primary role of comparing the neural signals received from the two retinas in order to achieve registration, coarse stereoscopic vision via the LGN/occipital lobe couple and precise stereoscopic vision via the PGN/pulvinar couple. Under the most minimal assumption, there may be 100 neurons processing the signals interrogated by P2 before generating a new stage 3 pulse stream that could be interrogated by P3. The number could be closer to 10,000 based on neuron density in the LGN (Section 11.7.1). The higher number would cause a significant time delay between the signals at probes P2 and P3. Based on the time delays in the Usrey papers noted above, it appears at least likely that his probe data corresponded to probe P3 (the output of the LGN) rather than P2. Usrey offered no information concerning which output channels of the LGN might be under interrogation by his intermediate probe or probe P4. The chrominance channels generally reflect lower signaling bandwidth (although not lower pulse bandwidth) than do the luminance channels. This can impact the details related to the cross-correlograms belonging to these individual channels.

Usrey recognized the “code” used within the stage 3 pulse circuits was not a simple rate code. They speculated on page 386, “Thus there is a partial transition from a rate code to a synchronous population code.” One year later on page 436, they develop their understanding of the coding more clearly. However, they do not arrive at a definitive position on the type of code actually used (See Section xxx). The code is logarithmically based and results in histograms (see inset at the top of the figure) that are much more symmetrical about the quiescent pulse rates than typically shown. The character of the data shown in figures 1 and 4 of the 1998 paper strongly suggests the channel they interrogated was a bipolar signaling channel with quiescent spike rates of between 8 and 24 pulses /sec depending on the state of adaptation of their ensembles of retinal sensory receptors. Frames 1d and 1f of the 1998 paper suggests their histograms were approximately symmetrical about these quiescent values (but the scales of the frames make certainty difficult). If correct, additional analysis is needed at to the character of their center-surround characterization of their identified neurons.

**11.7.3 Infero-temporal areas related to recognition of faces, etc.**

A holy grail of neural research has always been to explain how someone recognizes a specific individual among a group of similar individuals, the “that is my grandma” visual phenomenon.

Haxby et al. have provided a 2005 discussion of feature recognition in relation to locations along the “ventral path” of the visual modality using magnetic imaging

Figure 11.7.2-5 A brain model for visual attention from Shipp based on the recent position of Baars & Gage. It stresses the two-way paths between the visual engines and the thalamus rather than direct connections along either a dorsal or ventral path. See text. An interpretation of Shipp, 2005 by Baars & Gage, 2010.
techniques. They were seeking to move from the historical and coarse region identification (areas of about 10 square centimeters) to smaller areas (1 square centimeter areas). Their equipment and protocol does not lead to isolation of individual neurons, or (probably) individual neural engines.

Romero et al. have recently provided a review of the role of the inferotemporal cortex (Section 10.9.2) in identifying faces and faces with different orientations. While their bibliography is large, they did not provide citations in the captions of their figures. None of the authors of this review have published extensively in this area so further research is required to determine the source of their figures. Most of their figures report on the use of statistical methods to demonstrate the individual neuron they probed were reliably distinguishing between two marginally different images presented to a monkey. Their data uses an analysis of variants technique, (ANOVA). They did not describe the particular technique they employed, nor the details of their test protocol, but relied upon a value of p < 0.05 to represent a successful differentiation between two image presentations. They did not provide detailed coordinates of the locations of the neurons they employed in their experiments.

11.7.4 Hearing

11.7.5 Areas merging visual and hearing information

King provided an extensive report on his electro-physiological measurements of the superior colliculus in the guinea pig and ferret. He subdivided the superior colliculus of gross anatomy into multiple areas labeled the functional superior colliculus, the medial geniculate nucleus and an area labeled reticular formation which may represent the perigeniculate nucleus (PGN) of this work. Most of his work focuses on the auditory portion of the SC. King did not provide any block diagram, schematic or model of his auditory modality. Thus, it is not clear whether his data refers to afferent signal paths or efferent signal paths. He asserts that the visual and auditory spaces are mapped in different ways with the visual system being retinotopic and the auditory space being inertially based. He also suggests the mapping in the superior colliculus is two-dimensional. He makes no reference to any broader saliency map located at a more central region of the CNS. While his assertions may be appropriate when only studying the auditory modality related to source location, they are too parochial to define the overall neural system in these areas.

King provides extensive data that can be mined more effectively if it was associated with a block diagram of the complete neural system. His assertion of a two-dimensional map within the SC may be due to his failure to consider any three-dimensional requirements and/or commands. As noted elsewhere in this work, the saliency map itself is three-dimensional but also highly abstract. The three-dimensional information in the saliency map is drawn from and supports the vergence, version and focus controls of the ocular systems of vision. These signals are furnished to the eyes without being passed to or through the superior colliculus as well documented in the vision literature [xxx my PBV material. ].

11.7.9 Taste and olfaction EMPTY

[xxx See Taste, olfactory, and food texture processing in the brain,


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and the control of food intake
Edmund T. Rolls 2005 Physiology & Behaviour vol 85, pp 45-56 on the web ] rolls suggests the putative olfactory area roughly straddles areas 11l and 13l

[xxx See chapters 5 & 6 in Zald & Rauch Figure 6.1 shows orbitofrontal cortex with olfactory in human

11.7.9.2 Stage 4 engines of gustation EMPTY

11.7.9.3 Stage 4 engines of olfaction EMPTY

[xxx see section 8.6.7.5 on stage 4 in olfaction
[xxx piriform cortex ]

11.8 Merging of philosophical and physiological perspectives

When studying the literature, it is important to differentiate between those authors with a philosophical perspective (generally not relying upon or citing the detailed empirical literature) and those with a physiological perspective (who do rely upon the empirical literature). Excellent work has been done by both groups. However, those with a physiological perspective can be much more specific in terminology and mechanisms.

11.8.1 The 2005 philosophical paper of Shipp

In 2005, Shipp presented a masterful philosophical paper in an appropriately named journal53. The work focused on the nominal chordate neural system but leaned on data from the macaque monkeys. As noted above, Shipp does not focus on detailed definitions of his terms or on the detailed nature of the objects of his discussion. As an example, the character of the elements creating his granular and agranular layers are not defined beyond the definitions provided by Brodmann in the 1909 period. The terms have only a cartographic meaning in his paper although on a global basis, they are also associated with the presumed signaling traffic between areas of the cortex. Shipp frequently marries terms in the absence of specific definitions (ex., motor/agranular, sensory/granular, F3/SMA and F6/pre-SMA) and leaves terms undefined until obscure locations near the end of the paper (ex., SMA is defined in an endnote not clearly called out in the text). His labeling of the premotor cortex as MD is unusual as his inclusion of this label within his description of the thalamus of figure 8. The designation VA appeared for the first and only time in the caption to figure 8 without any formal definition. The designation Vla only appeared in footnote 5 where it was equated to VLo that appeared only in the citation to figure 8. VLo was equated to the term used by Olszewski with no citation.

He does define a dysgranular area of the cerebral cortex as “(having only an incipient, poorly developed layer 4)” within a specific area. The inference is that an agranular area lacks a layer 4 and a granular area includes a layer 4. The functional significance of these statements are not developed.

In his Figure 1, he has expanded upon an early schematic of the “somato-motor hierarchy” traced

back to Felleman & Van Essen (1991) and more recently expanded by Burton & Sinclair in 1996. The
figure is unusual in that it indicates neither an initial input(s) and final output(s) or the direction
of signal flow between approximately 18 boxes carrying a variety of Brodmann area labels (along
with some areas not identified by Brodmann in the insula and parahippocampal areas).

Because of these tendencies, Shipp has arrived at a number of paradoxes (ex., on page 799, he asserts
the location of the motor area of the cerebral cortex would be expected to be closer to the sensory
areas than the premotor area). This simple concept ignores completely the role of the stage 5
(cognition) in determining what instructions are passed to the premotor areas of stage 6 for
implementation before being elaborated into individual muscle commands within the motor areas
of stage 6. It also ignores the role of the reflex paths between the sensory modalities and the
implementation (motor) modalities.

Shipp treats the concepts of feedback in a very general way relating to message handling rather than
neural signal processing (ex., The key idea is that feedback acts to select certain ascending signals
in preference to others, culminating in a (temporary) steady-state resonance, in which the feedback
and forward activity is mutually reinforcing, over several hierarchical levels.”). While satisfying from
a philosophical perspective, it largely eliminates the opportunity to employ lookup tables (including
stored imagery) as a mechanism leading to recognition of objects in the total absence of any feedback
mechanism regardless of how it is defined.

He is 9 pages into a 17 page text before he mentions the cerebellum even obliquely, suggesting “the
interaction of these alternative pathways is not well understood.” The titles of his sections 6(c) and
6(d) end with a series of ellipses indicating incompleteness or speculation.

[xxx prepare to copy this figure to other sections of my work.]

Shipp did provide Figure 11.8.2-1 from McFarland & Haber54 of 2002 that is useful, although its
terminology varies from that commonly found in physiological material. Most significantly the figure
contains the label thalamus when the text and caption describe this entity as the thalamic reticular
nucleus (TRN). He discusses this figure in the context of cortico-thalamo-cortical paths rather than
thalmo-cortico-thalamic paths more indicative of the actual flow of signals (where the LGN and MGN
are considered part of the thalamus itself for this discussion rather than the more encompassing
diencephalon). This work will suggest additional crucial signal paths in the context of this figure
(See Section xxx).

54McFarland, N. & Haber, S. (2002) Thalamic relay nuclei of the basal ganglia form both reciprocal and
nonreciprocal cortical connections, linking multiple frontal cortical areas. J Neurosci vol 22(18), pp 8117–8132
Figure 11.8.2-1 Schematic for information flow between thalamic reticular nucleus and PFC. Colored gradients in boxes indicate the functional transitions from limbic to motor cortical areas. Each thalamic nucleus has strong reciprocal connections with a restricted region of cortex, but also receives nonreciprocal projections from a higher cortical station. The terms “cognitive” and “executive” are used in a different context than in this work. See original source for abbreviations and terminology. From McFarland & Haber, 2002.
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