Recordings, behaviour and models related to corticothalamic feedback

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In this paper, we review recent work on aspects of corticothalamic interactions in the auditory and in the visual systems. There are gross similarities in the arrangements of these systems, but considerable contrasts in the processing computations and in the effects of corticothalamic feedback.

Keywords: corticothalamic feedback; pitch discrimination; auditory; visual; medial geniculate body; lateral geniculate nucleus

1. INTRODUCTION

Primary sensory cortex receives input about external stimulus events largely through the corresponding thalamic nucleus. Both cortex and thalamus show an orderly map structure with respect to various physical parameters that define the stimulus, providing a spatial order to the afferent (feed-forward) flow. An enormous amount of material exists about the responses of individual cortical and thalamic neurons to the appropriate stimuli, and this has served as the basis for trying to understand the successive representations and transformations of the sensory information. However, the system is made much more complex by the massive corticothalamic feedback pathways originating in layer VI of the cortex. The feedback pathways are generally far more massive than the direct afferent path (for the visual system; Erisir *et al.* 1997; Van Horn *et al.* 2000), and are also distributed with the same map as the feed-forward system. The contribution of feedback pathways to the analysis process is not clearly understood either at the primary sensory cortex, nor at and between any two communicating cortical areas. In sensory thalamocortical circuitry, the feedback, despite its numerical superiority compared with the feed-forward system, seems to have mostly modulatory functions. For example, receptive-field structures of LGN neurons are much more like those of ganglion cells in the retina than those of V1 cortical neurons. Due to the tight feedback loop between cortex and thalamus it has proven difficult to define the role of each level in analysis of sensory information and in behaviour. In this paper, we will examine the effects of acute cortical inactivation in auditory and visual systems particularly with respect to the implications about interactions between thalamus and cortex.

2. AUDITORY CORTEX AND HEARING

There is an extensive literature starting from the late 1950s (Butler *et al.* 1957; Diamond & Neff 1957; Gold-

berg *et al.* 1958; Kelly 1970; reviews in Pickles 1982; Heffner & Heffner 1990; Buser & Imbert 1992) that examined the effects of auditory cortex ablation on sound-based behaviour. In a wide variety of animals it was found that such animals could easily be retrained after the operation to perform both detection and discrimination (pitch, amplitude) tasks. There were some higher-order defects, for example in the discrimination of tone sequences ('melodies') and in some spatial tasks like walking towards a brief source of sound. In later work it was possible to demonstrate that such defects had pitch specificities determined by which portions of the cortical frequency map had been ablated or spared (Jenkins & Merzenich 1984). However, the general interpretation of this body of data (which rapidly became dogma in the field) was that auditory cortex is not necessary for the basic simple auditory tasks although, with some species differences, it is probably involved in 'higher-order' processing needed for more complex auditory behaviour.

However, we note that all such ablation experiments have a long time constant. Recovery from operation and the subsequent retraining can consume weeks, a timescale that is known to be adequate for considerable plastic modifications of system organization. Modern tools have allowed revisiting the problem using short-term and reversible inactivation of auditory cortex, thus avoiding the possibility that reorganization is taking place. Behavioural experiments during local cortical inactivation in the mustached bat (*Pteronotus parnelli*) have been performed in the Suga laboratory (Riquimaroux *et al.* 1991, 1992), and showed impairment appropriate to the specialized echo analysis function of the particular cortical area. Prior to our work, to our knowledge, no similar studies on less specialized auditory systems have been reported. We worked only with wistar rats (*Rattus norvegicus albinus*), and the results possibly may not generalize to other creatures.

Rats were trained to make a pitch discrimination (Talwar & Gerstein 1998, 1999). For each test session the stimulus consisted of a long sequence of (500 ms on, 500 ms off) tones of a single pitch. Randomly at intervals of 5–20 s in the sequence a tone of slightly different pitch was presented, and the rat had to discriminate this and

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respond by entry into a nose-poke within 2 s after the onset of the Δf tone. Successful performance was rewarded with a portion of water; false alarms or misses were not punished. (All animal procedures followed NIH and University rules and were supervised by the veterinarian staff.) Rats easily learned this task, and were soon able to deliver a threshold Δf of 5–6% that gradually improved as training continued for many weeks (i.e. there was some degree of perceptual learning which did not generalize to another base stimulus frequency). Data were analysed using the quantity A' of signal detection theory, a function of hits and false alarms (Green & Swets 1966; Grier 1971; Talwar & Gerstein 1998, 1999). In the tasks described here the rats were presented either with a Δf of 8% (the difficult task) or a Δf of 20% (the easy task), and a session consisted of 10 presentations of each Δf . At the onset of a session, the rat demonstrated detection of the acoustic task by approaching the nose-poke, and by giving some false alarms.

After performance had stabilized, two small (3 mm in diameter) stainless steel chambers were implanted over left and right auditory cortex, leaving an intact dura in the bone opening. Electrical connection to the chambers allowed monitoring of local electroencephalogram and evoked response activity. A few days after implantation and recovery there was normal performance in the pitch discrimination task described in the previous paragraph. At this point we made use of muscimol (a γ -aminobutyric acid agonist) to shut down the auditory cortices during the behavioural task (Talwar *et al.* 2001). A typical run consisted of the following. After a baseline session, an appropriate muscimol dose was instilled into both cortical chambers, and a spaced series of additional sessions was begun.

As shown in figure 1 for a typical rat, as the muscimol diffused into cortex, the rat became profoundly deaf and stopped responding to the onset of a tone sequence in a new session. In this condition the rat continued to explore and otherwise behave as during a normal inter-session period. This failure of detection could not be compensated by making the tones louder, as indicated by the $\times$ symbols in the upper panel of figure 1. (However, we note that the rat would still show an auditory startle response to a sharp loud sound. It is known that pathways from lower auditory centres directly to the reticular formation are involved.) Many hours later, as the cortical inactivation caused by the muscimol slowly faded, there was a return of detection (i.e. the rat would move to and prepare to enter the nose-poke as the tone sequence came on, but could not discriminate even the 20% Δf , and produced only completely random responses). Later, there was gradual return of discrimination performance, first at the 20% Δf level, then finally at the 8% level. Electrical recordings of evoked responses from the auditory cortex followed a similar time course, with rapid reduction to zero and a gradual recovery, demonstrating that the auditory cortices were indeed inactivated (not shown here, but see Talwar *et al.* 2001). Similar results were obtained in a group of four additional rats.

These experiments indicate that auditory cortex is essential for performance of even basic auditory tasks of detection and pitch discrimination, although the nature of its involvement is of course not determined. The obser-

Figure 1. Effect of muscimol inactivation of both auditory cortices on rat auditory performance. The abscissa is the test session time relative to the muscimol application at 0 h. The second time point is 15–30 min after this application. The ordinate is the tone intensities in dB SPL and the frequency discrimination performance (A') when sound detection occurred. Black circles, tone intensities that were detected; crosses, tone intensities that were not detected; white circles, easy frequency discrimination; pluses, difficult frequency discrimination. (Adapted from Talwar *et al.* (2001).)

vations in the earlier ablation literature could be explained as resulting from slow plasticity and reorganization of the system so as to eliminate such dependence of performance on intact auditory cortex.

A possible caveat in our experiment is that we have not directly demonstrated that the rat is capable of some other sensory detection or discrimination task during the muscimol inactivation of auditory cortex. However, indirect evidence abounds. The rats continued to explore, sniff, scratch, chew, etc., during all phases of the experiments, showing that the motor system was intact. This also demonstrated that the somatosensory, olfactory and visual inputs were intact and were correctly interpreted and acted upon, showing that cognitive performance was intact. The persistence of the startle reaction to loud sounds demonstrated that the peripheral auditory system and lower nuclei were operational. During early phases of the recovery, as the detection of loud tones became possible, the rat responded to tone sequence onset in every session, but performed randomly in terms of discrimination of the Δf ; this shows that motivation and auditory cognition were not affected. As the recovery progressed there was a return of discrimination at the 20% level (but not yet at the 8% level); this also demonstrates normal auditory cognitive function despite a partial sensory deficit. Finally, the rats never huddled in a corner with unkempt fur, showing that there was no general malaise from the muscimol.

3. AUDITORY THALAMUS (MEDIAL GENICULATE) ACTIVITY DURING CORTICAL MANIPULATION

The early cortical ablation literature apparently demonstrates that without cortex, the lower nuclei, IC and MGB are capable of producing normal detection and pitch discrimination performance. (The pathways from these nuclei to the rest of cortex that would have to be involved in the behavioural response are not clear.) Thus, it is possible that the essential computations are always performed at the thalamic or tectal level, and that auditory cortex merely serves some necessary supportive role. The acute removal of the corticothalamic or corticotectal feedback would impact the computations or even silence the lower nuclei, but with sufficient time for adaptation, the IC or MGB could return to normal functioning.

Thus, it is important to assess the activities in IC and MGB during acute cortical manipulation. Such experiments have been performed in bats at the Suga laboratory using localized cortical application of either lidocaine or glutamate (Yan & Suga 1996; Zhang *et al.* 1997; Suga *et al.* 1997). Those experiments demonstrated that the corticofugal projection was excitatory to the thalamic map point (i.e. to the MGB or IC point with the same frequency tuning as the cortical point), and inhibitory to adjacent thalamic points. Thus, local cortical manipulation could in effect move the tuning curve structure of MGB or IC neurons along the frequency axis. However, although local cortical lidocaine could reduce MGB or IC neural response strength by *ca.* 40%, these lower neurons were never silenced.

Our laboratory has made related observations by monitoring MGB activity in the rat during the application of muscimol to auditory cortex. These were acute experiments under light ketamine–xylazine anaesthesia. MGB activity and response magnitude to auditory stimuli were occasionally slightly reduced as the cortical muscimol took effect, but there was never a silencing of MGB activity (nine neurons in four experiments). A typical time course for a tone-evoked response of a single MGB neuron together with the diminution of the cortical-evoked response under these experimental conditions is shown in figure 2. As the cortical-evoked response gradually disappears during the muscimol inactivation, the MGB neuron continues a robust response to the 5 kHz stimulus tone burst to which it is tuned. The indicated spike counts are each the sum over presentations of the 5 kHz tone during 20 tone burst sequences (pseudo-random frequency order). The muscimol effects on the evoked potential that are shown in figure 2 take place more rapidly than in the behavioural experiments in Talwar *et al.* (2001). This is because the acute experiment shown in figure 2 had a freshly exposed and thin dura over the auditory cortex, whereas in the chronic behavioural experiments the dura had considerably thickened with new growth, thus increasing the diffusion time for the muscimol to penetrate the cortex.

The simplest explanation of our observed behavioural deafness during cortical muscimol would be that the abrupt removal of corticofugal influence on MGB acts somewhat like spinal shock after acute spinal section. This model would indicate that the lower neurons are silenced by the lack of tonic excitatory input from cortex, so that

Figure 2. The effect of muscimol inactivation of both auditory cortices on a rat MGB neuron. The black squares show spike counts over 20 presentations of a 30 ms 5 kHz 70 dB SPL tone presented during a pseudo-random frequency sequence, 1 tone per 1.5 s. The small figures below each black square show the average cortical evoked response recorded at the steel well used to apply the muscimol. The calibration bars are 20 ms and 0.2 mV.

MGB no longer carries out its appointed calculations. This simple model is obviously incorrect given the kind of data shown in figure 2. Cortex must play a more complicated role than simply as a source of tonic excitation that permits MGB to function. We have not made comparable observations of IC neurons during cortical muscimol, but it seems likely from the Suga laboratory data that a similar result would be obtained.

Thus, in normal conditions, auditory cortex plays an essential computational role in even the simplest auditory tasks. The division of such computations between cortex and the lower centres remains unclear. The old auditory cortical ablation literature demonstrates, however, that given sufficient time, the system can reorganize in such a way that it can do without the cortical contribution.

4. THE ROLE OF LGN IN ORIENTATION TUNING; THE INFLUENCE OF CORTEX

We turn now to the visual system and the interactions of V1 cortex with LGN. Specifically, we examine these interactions for their role in producing orientation tuning of cortical neurons. The Sillito laboratory has for some time been recording from close (1–4°) pairs of LGN neurons in the cat (*Felis catus*) during systematic variation of drifting sine wave grating orientation, and more recently has included muscimol application to V1 cortex in the experimental protocol (Jones *et al.* 2000; Andolina *et al.* 2002).

The idea here is that each close pair of LGN neurons should project strongly to those V1 neurons at the same visual field position whose orientation preference is along the line joining the centres of the LGN neuron receptive fields. Each of the individual neurons in the pair will of course be active for any orientation of the stimulus grating. But the maximum influence on the cortical target cell

Figure 3. Quantities used to calculate predictions of the geometric model for near-synchronous firing of two LGN neurons. LGN neuron receptive fields are at a and b, with an approaching rectangular stimulus bar below. See § 5.

should come when both neurons are firing simultaneously, thus producing a high rate of near-synchronous spikes (Alonso *et al.* 1996; Usrey & Reid 1999; Roy & Alloway 2001). This condition would be produced by a grating that simultaneously excites both LGN neurons. Let us call this grating orientation the ‘reference angle’. As the grating angle is changed away from this angle, the maximum firing of each of the two LGN neurons will occur with a time offset. Therefore, there will be less near-synchronous firing, and the influence of the two LGN neurons on their target V1 neuron will be reduced.

The raw, uncorrected cross correlogram is a simple measure that quantifies the temporal relationship of the firing rate changes imposed by the stimulus. There will be a single peak in the correlogram that will be centred when the stimulus grating is at the reference angle. This peak will move systematically to either side of the centre as the grating orientation is changed and the firing rate increase of one LGN neuron is delayed relative to the other. For any grating orientation, the near-synchronous firing of the LGN neurons (to within 5–10 ms) that will have the greatest influence on the target V1 neuron is measured by the bin counts near the centre of the correlogram, irrespective of the location of the peak. As grating orientation is systematically changed and the peak moves through the origin, these central bin counts form a tuning curve for the putative contribution of these LGN neurons to the V1 target neuron, with a maximum at the reference angle. An appropriate single numerical descriptor of such a tuning curve is its HWHH. (Note that the cross-correlation measure here is being used only to quantify timings of firing rate modulations. This allows us to obtain the amount of near-synchronous firing that would be seen by the V1 target neuron and to form the tuning curve of this near-synchronous firing. Presumably the V1 neuron would be driven most effectively by this near-synchronous input, and its tuning curve for orientation would be proportional to the tuning curve of that input. Only the raw cross correlation is used for this measurement. The shift or shuffle predictors that are usually used to separate correlation caused by shared stimulus and neuronal interaction are irrelevant here since the V1 neuron could not compute them and would be influenced only by the amount of total correlation.)

5. THE GEOMETRIC MODEL TUNING CURVE

It is instructive to examine a purely geometric model of the experiments described in § 4. The objective is to calcu-

late an expected tuning curve of near-synchronous firing that can be compared with the experimentally obtained values. Figure 3 shows the geometric model. The LGN neurons have simplified receptive fields of radius r_1 and r_2 (no surrounds) with $r_1 < r_2$ and centres labelled a and b. The distance between the receptive-field centres is d (the line segment ab) and the grating stimulus is presented at an angle θ relative to the reference angle. We use a square stimulus grating with bar width w moving at velocity v . The line segment oa is x and the distance between the leading edge of the stimulus bar and the edge of the receptive field at b is y .

Making use of the similar triangles oae and obf we can quickly find that

$$y = d \sin \theta + r_1 - r_2. \quad (5.1)$$

We make the approximation that near-synchronous firing of the two neurons is proportional to the time that any part of the stimulus bar is in both receptive fields. The front edge of the bar reaches the receptive field at b after it has moved a distance y from that shown in figure 3. The rear edge of the bar leaves the receptive field at a after the bar has moved the distance $w + 2r_1$. Thus, the overlap time that some part of the bar illuminates both fields is

$$OT = \frac{w + 2r_1 - y}{v} = \frac{w + r_1 + r_2 - d \sin \theta}{v}. \quad (5.2)$$

However, note that there is a critical angle (when the stimulus bar is simultaneously tangent to both receptive fields with $y = 0$) below which the overlap time is constant and independent of angle:

$$OT_{\max} = \frac{w + 2r_1}{v} \quad \text{for } r_1 < r_2. \quad (5.3)$$

To get the HWHH of the tuning curve we must evaluate equation (5.2) when it is equal to one half of the maximum value represented by equation (5.3). Solving for that angle we obtain

$$\theta_{\text{hwhh}} = \arcsin \left[\frac{w + 2r_2}{2d} \right] \quad \text{for } r_1 < r_2. \quad (5.4)$$

The dashed curve labelled ‘geometry’ on figure 4 plots this HWHH angle as a function of d , the distance between receptive-field centres, and for values of w and r that are typical for the experiments. Note that the distance is normalized to the average receptive-field radius.

6. COMPARISON TO THE NETWORK MODELS AND EXPERIMENTS

The geometry curve in figure 4 is hyperbolic in shape and, for the two LGN neurons being considered here, states that the width of the near-synchronous firing tuning curve for orientation depends strongly on the distance between the receptive fields of the neurons. This seems intuitively satisfactory: for close receptive fields, near-synchronous firing from the two LGN neurons should occur over a wide range of stimulus orientations, and would lead to a wide orientation tuning curve. Conversely, when the two LGN neurons are far apart, their near-synchronous firing would occur only for a narrow range of stimulus orientations and would produce a relatively narrow orientation tuning curve.

Figure 4. The HWHH of the orientation tuning curve for near-synchronous firing of two LGN neurons shown as a function of distance between the receptive-field centres. The distance is normalized to the average receptive-field radius. The dashed line is the result from the geometric model of figure 2. The solid line is the network model result when the feedback from the cortical layer is turned off. The dot-dashed line is the full network model result, i.e. with cortical feedback to LGN. Although not shown, experimental points fall near the lower curve (Andolina *et al.* 2002). See § 6.

However, this result does not agree at all with the experimental data (Jones *et al.* 2000; Andolina *et al.* 2002, not shown). It turns out that the width of the observed orientation tuning curves for pairs of LGN neurons is essentially constant in the inter-field distance range of 1–10°. This discrepancy between experimental values and the simple geometric model indicates that feedback from the cortex to LGN, which was not considered in the model, might be involved.

Accordingly, we have studied a more complicated, network-based model consisting of cells with one or two compartment elements (Kirkland *et al.* 2002). The network consisted of three layers, each with a 24×24 array of neurons, representing the retina, LGN and cortex. All projections were topographic. The projection from retina to LGN was 1-to-1, and the synaptic strengths of these excitatory connections were very strong. Projections from the LGN layer to the cortical layer consisted of a symmetrical 3×3 excitatory patch without orientation tuning properties. Thus, in effect each cortical neuron represented a hypercolumn. Feedback from the cortical layer to the LGN consisted of a probabilistic 3×3 excitatory centre, surrounded by an annulus of probabilistic inhibitory connections reaching out into a 7×7 region. Only the cortical layer had lateral connectivity; this was also modelled, for each cortical element, as a narrow surrounding ring of excitatory connections and a wider annulus of inhibitory connections. (Such rules of local connectivity have previously been shown to produce network stability over a wide range of conditions (Xing & Gerstein 1996a–c; Kirkland & Gerstein 1998), and are in agreement with the dual intracellular measurements by Thomson & Deuchars (1994).) No inhibitory interneurons were explicitly modelled, but the connections that were inhibitory had longer delays to mimic the extra processing time. All simulated cells were spiking neurons with thresholds for production

of action potentials and suitable refractory periods. The LGN neurons had two compartments: the soma received the retinal excitation and inhibitory feedback from the cortex, and the dendrite received relatively weak feedback excitation (Wilson *et al.* 1984). Synaptic currents were α functions (a product of an exponential and a linear function), with a longer time constant for inhibitory than for excitatory events. A later version of the model was modified to incorporate the elliptical asymmetry (along the preferred orientation axis) of the cortical cells' projection to the LGN, reported by Murphy *et al.* (1999). This additional feature proved to be critical (Kirkland *et al.* 2002), thus indicating an important role for the elliptical corticogeniculate projections noted by Murphy *et al.* (1999).

HWHHs of orientation tuning curves as a function of LGN receptive-field distance obtained from the complete network model are shown in the lower curve of figure 4. The error bars are standard deviations over many runs of the model network. This curve is much flatter than the geometry curve as a function of LGN receptive-field distance, and is a quite good approximation of the experimental data (Andolina *et al.* 2002, not shown). If in the network model we turn off the feedback connection from the cortical layer to the LGN layer, we obtain the upper curve in figure 4. The HWHH of the orientation tuning curves now depends on LGN RF distance in same way as the values produced by the purely geometric calculation, i.e. a strongly hyperbolic function. The same phenomenon is observed in recent experiments with LGN neuron pairs (Jones *et al.* 2000; Andolina *et al.* 2002, not shown) when muscimol is used to inactivate V1 cortex.

Thus, cortical feedback to thalamus appears both experimentally and in models to be crucially involved in narrowing the orientation tuning curves for near-synchronous firing of LGN neuron pairs. The effects are particularly evident at small distances between the LGN receptive fields.

A somewhat unexpected aspect of the LGN near-synchronous firing contribution to cortical orientation tuning is seen when the width of the LGN tuning curves is compared with that of a typical V1 neuron. LGN HWHH values are *ca.* 5° with intact cortex both experimentally (Andolina *et al.* 2002, not shown) and with the network model (figure 4). However, V1 neurons have orientation tuning curves with *ca.* 15° HWHH. At first glance it would seem that although the feedback from cortex to LGN apparently sharpens the available orientation input to cortex, this is somehow lost in the LGN to cortex projection. One possible explanation of this contradiction could be that many LGN neurons actually contribute to a particular V1 target neuron. Receptive fields of LGN neurons are smaller than that of a V1 neuron, so that contributing LGN pairs could in fact represent a variety of reference angles, thus effectively broadening the V1 orientation tuning. Unfortunately this simple explanation does not hold up. Data on the number and size of LGN receptive fields that project to a given V1 neuron are available (Reid & Alonso 1995; Alonso *et al.* 1996; Usrey *et al.* 2000). Adding together an appropriate array of LGN orientation tuning curves either using experimental values (Andolina *et al.* 2002) or in a theoretical study at our laboratory (G. L. Gerstein, unpublished work) does not significantly

broaden the predicted cortical orientation tuning curve beyond that of a single LGN neuron pair. The apparent loss of orientation information between LGN and cortex remains unexplained.

7. CONCLUSION

In this review, we have examined certain aspects of the thalamocortical relationship in the auditory system and in the visual system. In the auditory system we have demonstrated, in reassessment of earlier literature, that a functioning cortex is essential for the fulfilment of even simple auditory tasks like detection or pitch discrimination. That earlier literature showed that rats and cats with a wide ablation of auditory cortex (and a long recovery and retraining period) were capable of most auditory tasks, mediated presumably by the intact thalamic and tectal auditory nuclei. However, during the acute and temporary cortical inactivation produced by local application of muscimol, we have observed a complete absence of response to a familiar and well trained auditory task. With time, as the muscimol wore off, first detection and then pitch discrimination gradually returned. It also turns out that auditory thalamic neurons in the MGB are only slightly affected, and certainly not silenced, during the acute removal of feedback from the muscimol-inactivated cortex. Much more work is necessary to unravel the normal division of computational tasks between cortex and MGB and IC, and to trace the reorganization that allows auditory task behaviour after chronic cortical ablation.

In the visual system we have focused on the contribution that LGN neurons could make to V1 cortical orientation tuning. In particular, we examined the properties of near-synchronous firing in LGN neuron pairs as stimulus orientation changed. Such near-synchronous firing should have a large influence on the target V1 neuron. A purely geometric model calculation for the tuning curve width of near-synchronous firing showed a strong hyperbolic dependence on LGN neuron spacing. This was not observed in experimental studies by the Sillito laboratory, and it required a three-layer neural network with an asymmetrical corticogeniculate projection to produce results like the experiments. In both the experiments and network model, turning off the cortex (V1 muscimol for the experiments, disconnection in the model) resulted in a good match to the predictions of the geometric model. This implies that cortical feedback to the LGN is instrumental in narrowing the orientation tuning signal projected to cortex. Further work is necessary to clarify the overall discrepancy in the orientation tuning width between single cortical neurons (wide) and the LGN neuron pairs (narrow) that project to them. An experimentally based superposition of the contributions from many LGN pairs to a single V1 receptive field does not account for the width discrepancy. No behavioural assessment of cortical inactivation comparable with that in the auditory work is available for the visual system.

In both these sensory systems the corticothalamic feedback has profound effects on the overall computation. The parsing of computational tasks between cortex and thalamus in the normal state, and delineating the shifts or reorganizations during acute or chronic cortical inactivation remain an urgent goal. It is unfortunate indeed that

anatomy precludes turning off the corticothalamic feedback pathway alone while maintaining the thalamocortical afferent flow.

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GLOSSARY

- HWHH: half width at half height
 IC: inferior colliculus
 LGN: lateral geniculate nucleus
 MGB: medial geniculate body
 SPL: sound pressure level; units are dB relative to the average sound threshold intensity in humans
 V1: primary visual cortex