Brain Research 1661 (2017) 49–66

Contents lists available at ScienceDirect

Brain Research

journal homepage: www.elsevier.com/locate/bres

Research report

Robust interactions between the effects of auditory and cutaneous electrical stimulations on cell activities in the thalamic reticular nucleus

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ARTICLE INFO

Article history:
Received 18 October 2016
Received in revised form 1 February 2017
Accepted 7 February 2017
Available online 12 February 2017

Keywords:
Auditory system
Juxta-cellular labeling
Rat
Somatosensory system
Thalamic reticular nucleus
Unit discharge

ABSTRACT

The thalamic reticular nucleus (TRN), a cluster of GABAergic cells, is thought to regulate bottom-up and top-down streams of sensory processing in the loop circuitry between the thalamus and cortex. Provided that sensory inputs of different modalities interact in the TRN, the TRN could contribute to fast and flexible cross-modal modulation of attention and perception that incessantly takes place in our everyday life. Indeed, diverse subthreshold interactions of auditory and visual inputs have been revealed in TRN cells (Kimura, 2014). To determine whether such sensory interaction could extend across modalities as a universal neural mechanism, the present study examined TRN cell activities elicited by auditory and cutaneous electrical stimulations in anesthetized rats. Juxta-cellular recording and labeling techniques were used. Recordings were obtained from 129 cells. Auditory or somatosensory responses were modulated by subthreshold electrical stimulation or sound (noise burst) in the majority of recordings (77 of 85 auditory and 13 of 15 somatosensory cells). Additionally, 22 bimodal cells and seven cells that responded only to combined stimulation were recognized. Suppression was predominant in modulation that was observed in both early and repeatedly evoked late responses. Combined stimulation also induced de novo cell activities. Further, response latency and burst spiking were modulated. Axonal projections of cells showing modulation terminated in first- or higher-order thalamic nuclei. Nine auditory cells projected to somatosensory thalamic nuclei. These results suggest that the TRN could regulate sensory processing in the loop circuitry between the thalamus and cortex through the sensory interaction pervasive across modalities.

1. Introduction

The thalamic reticular nucleus (TRN), a cluster of GABAergic cells located at the junction of bottom-up and top-down streams of sensory processing in the loop circuitry between the thalamus and cortex (Pinault, 2004; Halassa and Acsády, 2016), could play a crucial role in pre-attentive and/or attentive modulation of sensory processing through its inhibitory projections to thalamic nuclei (Crick, 1984; McAlonan et al., 2008; Wimmer et al., 2015). A recent study (Kimura, 2014) has revealed diverse subthreshold interactions of auditory and visual inputs in TRN cells projecting to first- and higher-order thalamic nuclei that, receiving major excitatory inputs from the periphery and cortex, relay information primarily to the primary and higher cortical areas (Guillery and Sherman, 2002). This sensory interaction, potentially affecting thalamic and consequently cortical cell activities (Halassa et al., 2011; Willis et al., 2015), could be a neural substrate for fast cross-modal alterations of sensory responses recognized in the primary and non-primary sensory areas in the cortex (Foxe and Schroeder, 2005; Driver and Noesselt, 2008; Brang et al., 2015), which may not be fully attributable to corticocortical connections (Rockland and Ojima, 2003; Budinger et al., 2006; Iurilli et al., 2012). The sensory interaction may also reorganize the temporal structures of sensory processing because the TRN is an important neural structure for oscillatory activation of the loop circuitry (Steriade et al., 1987; Pinault, 2003). In view of the projection from the prefrontal cortex to the TRN (Zikopoulos and Barbas, 2006), it is postulated that the TRN subserves cross-modal gain and/or gating control of sensory information (Crabtree and Isaac, 2002; McAlonan et al., 2006; Yu et al., 2009) on the basis of synaptic weights of sensory cues and cognitive functions that likely interact in the TRN. Then, the question arises of whether the sensory interaction of the TRN

Abbreviations: MG, medial geniculate nucleus; MGD, dorsal division of medial geniculate nucleus; MGM, medial division of medial geniculate nucleus; MGV, ventral division of medial geniculate nucleus; Po, posterior nucleus; SG, suprageniculate nucleus; TRN, thalamic reticular nucleus; VPL, ventral posterior lateral nucleus; VPM, ventral posterior medial nucleus.

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http://dx.doi.org/10.1016/j.brainres.2017.02.005
0006-8993/

could extend across other modalities to compose a universal mechanism for cross-modal facilitation and/or competition of attention and perception that incessantly takes place in our everyday life filled with various sensory cues of different modalities.

Cross-modal sensory interactions between auditory and somatosensory modalities, which are well exemplified by human psychological phenomena (Foxe, 2009; Occelli et al., 2011), have been revealed in subcortical and cortical cell activities (Kayser et al., 2005; Lakatos et al., 2007; Wu et al., 2015). These are assumed to provide a neural basis for auditory-somatosensory interaction to the TRN. A previous study, however, has detected only a few bimodal cells activated by auditory and somatosensory stimulations in the TRN (Shosaku and Sumitomo, 1983). On the other hand, the convergence of auditory and somatosensory afferents to the TRN (Jones, 1975; Kimura et al., 2012) and the mutual connectivity of TRN cells (Landisman et al., 2002; Deleuze and Huguenard, 2006; Lee et al., 2014) raise the possibility of subthreshold interactions between auditory and somatosensory modalities like those observed between auditory and visual modalities. To address the above question, the present study focused on possible interactions of the effects of auditory and cutaneous electrical stimulations on single TRN cell activities. The results indicated not only robust subthreshold interactions but also the presence of bimodal cells at an unexpectedly high incidence, suggesting that the sensory interaction is pervasive across sensory modalities inside the TRN.

2. Results

Data analysis was carried out on recordings obtained from 129 cells. There were 85 auditory cells responsive only to noise burst stimuli, 15 somatosensory cells responsive only to electrical stimuli to the hindpaw, 22 bimodal cells that responded to both noise burst and electrical stimuli, and seven cells that showed sensory responses only when noise burst and electrical stimuli were combined (Table 1). Labeling of the cell body was observed in 56 auditory, eight somatosensory, 10 bimodal cells and two cells that responded only to combined stimulation. Labeling of the axonal projection and terminal field was recognized in 39 auditory, six somatosensory and five bimodal cells.

Electrical stimuli to the hindpaw or noise burst stimuli, which did not elicit unit discharges, i.e., subthreshold somatosensory or auditory inputs, modulated auditory or somatosensory responses (unit discharges) in the majority of cells (77 out of 85 auditory cells and 13 out of 15 somatosensory cells) (Table 1). Additionally, auditory-somatosensory bimodal cells were found to comprise a substantially larger percentage of recorded cells (22 out of 129) as compared to auditory-visual bimodal cells (2 out of 137) recognized in the previous study (Kimura, 2014). Unit discharges were modulated in both early responses evoked within 100 ms (55 out of 64 auditory cells and 12 out of 14 somatosensory cells showing early responses) and repeatedly evoked late (>100 ms) responses that were observed up to the end of recording (67 out of 79 auditory cells and 11 out of 11 somatosensory cells showing late responses). Six auditory and four somatosensory cells exhibited only early responses. The majority of cells exhibited late responses with (58 auditory and 10 somatosensory cells) and without (21 auditory and one somatosensory cells) preceding early responses. As such, sensory response patterns were diverse across cells and alterations of sensory responses were further diverse upon early and repeatedly evoked late responses in a given cell with respect to response magnitude, latency and burst spiking as represented by the cases shown in Figs. 1–3 and 6. Despite the overall complexities of response and modulation, alterations in response magnitude and burst spiking were primarily suppression (Tables 1, 3 and Figs. 4, 5). Response latency alterations were bidirectional in terms of the mean and standard deviation (jitter) of latencies (Table 2). These alterations of sensory responses took place in two types of cells that projected to first- (the ventral division of the medial geniculate nucleus (MGV) and the ventroposterior lateral nucleus (VPL)) or higher-order (the dorsal and medial divisions of the medial geniculate nucleus (MGD and MGM respectively); the suprageniculate nucleus (SG); and the posterior nucleus (Po)) thalamic nuclei.

2.1. Representative alterations of auditory responses by electrical stimulation of the hindpaw

Electrical stimuli to the hindpaw were given before (Fig. 1) or after (Fig. 2) early auditory responses. In either timing electrical stimuli suppressed auditory responses except in a small subset of cases where electrical stimuli facilitated late responses and/or induced de novo cell activities as early and/or late responses (Table 1, Figs. 4A and 5A). Electrical stimuli before early auditory responses resulted in drastic suppression of the early responses (Fig. 1A and B). They subsequently exerted differential influences on late auditory responses in a cell that sent an axonal projection to the MGV (Fig. 1A) or continued to suppress repeatedly evoked late responses in the other case (Fig. 1B). Electrical stimuli also modulated late responses or induced de novo cell activities as late responses without affecting preceding early (Fig. 1C) and/or late auditory responses. Further, late auditory responses were modulated in cells that had no early responses as shown in Fig. 1D. Electrical stimuli also modulated response latency and/or burst spiking in both early (magnified raster and graph in Fig. 1B) and late auditory responses (magnified raster and graph in Fig. 1C and D) regardless of the presence or absence of significant alterations in response magnitude (Tables 2 and 3).

Table 1
Number of cells showing modulation.

Modality		Auditory			Somatosensory		
Response		Early	Late	Total	Early	Late	Total
		64	79	85	14	11	15
Response magnitude	Increase	0	3	3	2	0	2
	Decrease	20	40	49	6	7	8
	De novo	5	13	18	1	2	3
	Total	25	45	57	8	9	12
Latency	Mean	10	21	29	8	3	8
	SD	33	28	49	5	3	7
	Total	38	39	58	8	5	9
Burst	Total	15	22	32	5	5	7
Modulation	Total	55	68	77	12	11	13

SD, standard deviation.