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Viscerosomatic interactions in the thalamic ventral posterolateral nucleus (VPL) of the squirrel monkey

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Abstract

In anesthetized squirrel monkeys single cell recordings were performed using tungsten microelectrodes. The responses of 29 viscerosomatoceptive and somatoceptive VPL neurons to noxious distension of the urinary bladder, the lower esophagus and the distal colon and to innocuous and noxious somatic stimuli were assessed when these stimuli were presented separately or together. Neuronal responses were defined as additive or interactive depending on the relative changes in responses to individual somatic or visceral stimuli, and on their responses during conditioning (somatic and visceral stimuli applied concurrently). In 13 neurons interactions between the somatosensory and visceral inputs could be demonstrated. The dominant interactive effect was inhibition, although facilitatory effects were seen as well (2 of 13). The magnitude or direction of the interactions seemed independent of the location of the somatic and visceral receptive fields. The mean population response of the neurons showing interactions was 4.66 spikes/s to somatic stimulation, and 0.07 spikes/s to visceral stimulation. During conditioning the mean interactive effect was −62% of the calculated additive effect. This implies that overall the somatic responses are halved during a coincident visceral stimulus. In a subgroup of the VPL neurons, which were classified as pure somatic responsive ($n = 14$) due to their unresponsiveness during visceral stimulation alone, a third ($n = 5$) still exhibited visceral convergence during conditioning. The latter neurons, therefore, receive visceral inputs, which function in a purely interactive (modulatory) manner. It is concluded that part of the described effects is due to competition (cross modality suppression) between the visceral and somatic inputs. We further conclude that the suppression of somatic information by noxious visceral stimuli may contribute to a more effective processing of the discriminatory aspects of nociceptive visceral information previously demonstrated in VPL. © 1998 Elsevier Science B.V.

Keywords: Thalamus; Nociception; Visceral sensation; Somatovisceral convergence; DNIC; Descending and ascending inhibition

1. Introduction

In a recent study we demonstrated that most neurons in the squirrel monkey somatosensory thalamus receive convergent somatic and visceral inputs [5], and many of the neurons met the criteria for coding visceral information. In addition, we found indications for a modulatory role of these convergent visceral inputs, because for example most visceral responses were inhibitory, no viscerotopical arrangement was found, and in many cases the viscerosomatic convergence was improper with respect to the spinal cord segmental inputs.

Interactions between the somatic and visceral systems have been demonstrated in various studies at different levels of the CNS in rat (e.g., Refs. [6,10,12,14,15], cat [1,7], and in psychophysical studies [4,8]). One common feature of these interactions is that they are inhibitory, i.e., that either somatic conditioning stimuli suppress responses to visceral stimuli, or vice versa, or that the ongoing activity of neurons is suppressed.

Given the results of our previous study [5] we hypothesized that the same visceral inputs, which are encoded by individual neurons, might modulate the responsiveness of neighboring neurons, i.e., that the somatic and visceral inputs would interact in the lateral thalamus of the squirrel monkey. To test this hypothesis, we performed a second, independent study, where we applied somatic and visceral stimuli separately, like in the previous study, but in addition, we now presented visceral somatic stimuli together,

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and analyzed the observed interactive effects quantitatively.

2. Materials and methods

All surgical procedures, housing and care of the animals were approved by the local Committee for the Humane Use of Animals. Before each experiment anesthesia was induced by ketamine hydrochloride (35 mg/kg i.p.) and maintained with halothane (0.8–1.25%) in a mixture of N_2O/O_2 (33/67%). Blood pressure, expiratory CO_2 concentration, and O_2 saturation were monitored continuously. Data acquisition was stopped when the values were out of the physiological range. The trachea was catheterized, and the animals breathed spontaneously. The body core temperature was kept around 38°C by means of a feed back heating pad. The experiments were performed in four adult, female squirrel monkeys (weight 650–850 g). In three monkeys following a craniotomy a recording chamber was implanted over the right hemisphere of the skull to gain access to the region in and around the lateral thalamus. This procedure was performed under sterile conditions, and the animals were given at least one week to recover. Once or twice every two weeks these animals were deeply anesthetized and 8 h recording sessions were performed. In one monkey a craniotomy was performed over the right hemisphere without implanting a recording chamber. In this case the experiment lasted 28 h after which the animal was given a lethal dose of pentobarbitone.

The following controlled stimuli were applied to characterize lateral thalamic neurons exhibiting ongoing activity: air puff (pico-spritzer), skin displacement (Chubbuck), innocuous and noxious mechanical pressure (two different hemostats), and in a few instances noxious heat (feed back controlled thermo-probe). In the three monkeys with an implanted recording chamber the distal colon and the lower esophagus were catheterized with double-lumen balloon catheters. In the fourth monkey, in addition, the urinary bladder was catheterized through the urethra with a double-lumen catheter without a balloon. The organs were distended via one of the tubes and the intraluminal pressure was measured through the other tube. In order to investigate interactions between the somatic and visceral systems, somatic and visceral stimuli were applied separately and simultaneously (for details see below).

Extracellular single cell recordings using tungsten electrodes (tip diameter: 1 μm; impedance at 1 kHz: 1–1.5 MΩ) were performed. During the experiment the neuronal activity was window discriminated, displayed as peristimulus-time histograms (PSTHs), and correlated with the various stimuli applied. For the quantitative analysis, the recorded neuronal activity was reanalyzed off-line with respect to spike shape and amplitude to make sure that only single cell activity was correlated with the different combinations of stimuli. For this commercially available hardware and software (DataWave Technologies, Colorado) was used. A response had to occur at least two times, before a neuron was considered responsive. To be considered a response, changes in neuronal activity had to be at least $\pm 30\%$ and statistically significant (t-test; $p \leq 0.05$).

2.1. Definition of interaction

To determine quantitatively the interrelationship between the somatic and visceral inputs of a given neuron its ongoing activity (generally for 20 to 60 s) before each stimulation was subtracted from both the responses to visceral (ΔV) and somatic (ΔS) stimulation alone. Both rates were added ($\Delta V + \Delta S$; expected rate or additive effect) and compared to the change in discharge rate during the simultaneous application of visceral and somatic stimuli (ΔVS; conditioning effect). An ideal test consisted of two runs: (1) 30–60 s ongoing activity, somatic stimulation alone (to calculate ΔS), ongoing activity, followed by visceral stimulation alone (to calculate ΔV). (2) Ongoing activity, somatic stimulation alone, simultaneous visceral and somatic stimulation (to calculate ΔVS), somatic stimulation alone. Between runs at least 5 min had to pass. Each test was performed at least twice. If there was a significant difference between the expected additive effect ($\Delta V + \Delta S$, which assumes no cross interference between the somatic and visceral channels accessing the neuron, i.e., two independent channels), and the conditioning effect (ΔVS), it was concluded that the two inputs interact. Significant difference (interaction) was defined as:

$$I(V,S) = 100 \times \left[\Delta VS - (\Delta V + \Delta S)\right] / |(\Delta V + \Delta S)|$$

to be larger than $\pm 30\%$. When $I(V,S)$ was negative ($\leq -30\%$), the interaction was defined to be inhibitory, i.e., co-activation of both inputs led to a lower discharge rate than expected from activating each input separately (competitive suppression), and when the difference was positive ($\geq +30\%$), it was defined as a facilitatory interaction (cross facilitation). It should be noted that, given the definition of $I(V,S)$, when $\Delta V = 0$ or when $\Delta VS = 0$ one cannot imply a lack of visceral inputs or a lack of viscero-somatic interaction. When $\Delta V = 0$, there may still be visceral inputs that modulate the somatic responses, i.e., the visceral inputs are purely interactive. When $\Delta VS = 0$, it only implies that the conditioned (or combined) response is zero, which may or may not be equal to the additive responses.

3. Results

Twenty-nine VPL neurons were tested for modulatory effects of visceral inputs on somatic responses. In 13 of these neurons significant interactions between the somatosensory and the visceral systems could be demon-

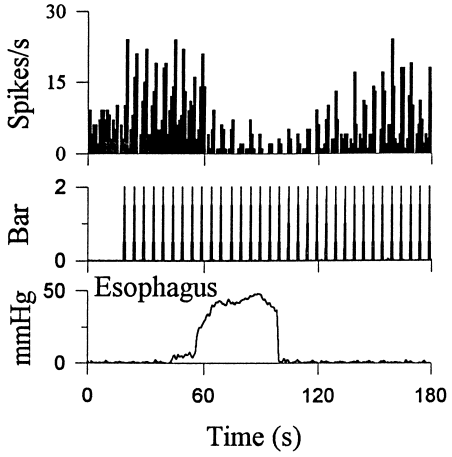

Fig. 1. PSTH (bin width 1 s; upper panel) of the inhibitory interaction of responses to air puff applied to the fur of the wrist (middle panel) and esophagus distension (lower panel) of a VPL neuron.

strated. Fig. 1 is an example of the responses of a neuron with significant viscerosomatic interactions. The unit had an ongoing activity of 6.3 spikes/s and increased its activity to 11.1 spikes/s due to hair movement by air puff applied to the lateral wrist ($\Delta S = +4.8$ spikes/s). Distension of the lower esophagus alone decreased the activity to 0.2 spikes/s ($\Delta V = -6.1$ spikes/s), and the same somatic and visceral stimuli presented together elicited a decrease in neuronal activity to 1.6 spikes/s (mean rate during maximal esophagus distension; $\Delta VS = -4.7$ spikes/s). Thus, the difference between the expected and the observed discharge rate during conditioning [$\Delta VS - (\Delta V + \Delta S)$] was -3.4 spikes/s (inhibitory interaction), the strength of the interaction was $I(V,S) = -262\%$. Therefore, the simultaneous activation of visceral and somatic channels decreased (negative I value) the response of this neuron by 262% of its summed response to each stimulus modality.

A second example of a viscerosomatic interaction, this time between responses to air puff and colon distension is shown in Fig. 2. In contrast to the neuron described above, colon distension ($\Delta V = -0.06$ spikes/s) alone had no statistically significant effect on the unit's ongoing activity (Fig. 2B). However, the responses to air puff ($\Delta S = 6.01$ spikes/s) were decreased during distension of the distal colon ($\Delta VS = 0.17$ spikes/s). Thus, the visceral input was only apparent during conditioning, and $I(V,S) = -121\%$. Therefore, visceral inputs of this neuron had only modulatory effects on the somatic responses during conditioning.

In the 29 VPL neurons, the following interactions were tested: colon distension and air puff or skin displacement (LTs): $n = 25$; showing interaction: $n = 9$; esophagus distension and LTs: $n = 19$; showing interaction: $n = 3$; bladder distension and LTs: $n = 7$; showing interaction: $n = 0$. The colon and bladder responses were combined to represent the incidence of interaction when the lower body viscera were tested. Chi square test of incidence of interaction for lower vs. upper body viscera resulted in borderline significance ($p < 0.09$), i.e., colon responses had a higher incidence of interactions, implying that interaction incidence may be organ specific.

In the 13 neurons exhibiting interactions six were tested only with colon distension, and two with only esophagus distension, all showed inhibitory interactions. Five were tested with both colon and esophagus distension, three had inhibitory interactions for both organs, one had inhibitory colon interaction but facilitatory esophagus interaction, and one had facilitatory interactions from both esophagus and colon, as well as trials where the interaction from colon were inhibitory. Thus, the dominant interaction effect was inhibitory for the different combinations of viscera tested.

Twenty-two of 29 neurons were characterized with somatic and visceral stimulation alone as well as with viscerosomatic conditioning. In the other seven only the conditioning and somatic stimulation alone were performed. In the latter group only the magnitude of ΔVS could be determined. In regard to separate somatic and visceral stimulation eight (of 22) neurons were classifiable as somatoviscerocceptive, and 14 as somatoceptive. In the somatoviscerocceptive group all showed interactions, and in the somatoceptive group (i.e., neurons which did not respond to visceral stimulation alone) five showed interactions. Thus, in five neurons the visceral input was only apparent when somatic and visceral stimuli were applied simultaneously. With respect to somatic stimulation six of the 29 neurons tested were somatic nociceptive, all of them

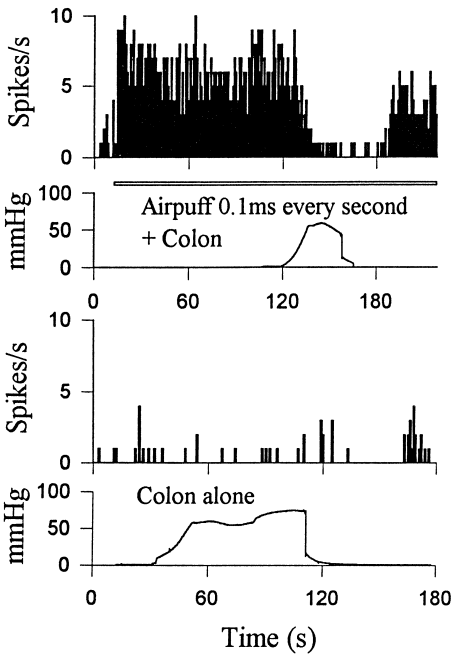

Fig. 2. (A) PSTH (bin width 1 s) of the interaction of responses to air puff stimulation (total duration of repeatedly applied single air puffs [0.1 s duration; every 1 s] indicated by the horizontal bar) and colon distension (intraluminal pressure changes shown in the lower traces of both graphs) of a VPL neuron. (B) discharge rate of the same unit during colon distension alone.

Table 1
Relationship between interaction types and the location of the somatic receptive fields

Interaction type	Upper body som. RF	Lower body som. RF
Col −	2	4*
Col − /Eso −	0	3
Col − /Eso +	0	1
Col ± /Eso +	0	1
Eso +	1	0
Eso −	1	0
Sum	4	9

Abbreviations: Col, colon distension; Eso, esophagus distension; som. RF, somatic receptive field; −, inhibitory; +, excitatory; ±, mixed excitatory/inhibitory.
The asterisk indicates one neuron, which was excited by visceral stimulation alone, but was inhibited during conditioning.

exhibiting interactions for somatic LT type stimuli. Only two of the six somatic nociceptive neurons were tested for viscerosomatic interaction with noxious thermal somatic stimulation.

There was no significant difference between hetero- vs. peri/homo-segmental somatic receptive fields and the direction (i.e., excitatory or inhibitory effects) of the interactive effects (paired t-test, $p = 0.26$; Table 1). In one of 8 cases the response to visceral stimulation alone was in the opposite direction as the interaction, i.e., colon stimulation alone was excitatory, somatic LT stimulation was also excitatory, but the interaction was inhibitory. The latter is an important observation, because it is the only case where the interaction indicates that simultaneous activation of both channels decreases the visceral responses as well as the somatic ones.

In two somatic nociceptive neurons esophagus and colon distension was tested against noxious heat. The different interactions between visceral and somatic inputs observed in these two units are shown in Fig. 3B and C. The neurons were recorded simultaneously and differentiated by spike shape and height (Fig. 3A). The ongoing activity of both neurons did not change during colon or esophagus distension alone (i.e., ΔV was zero, not shown). In both neurons the responses to noxious heat applied to digit 5 of

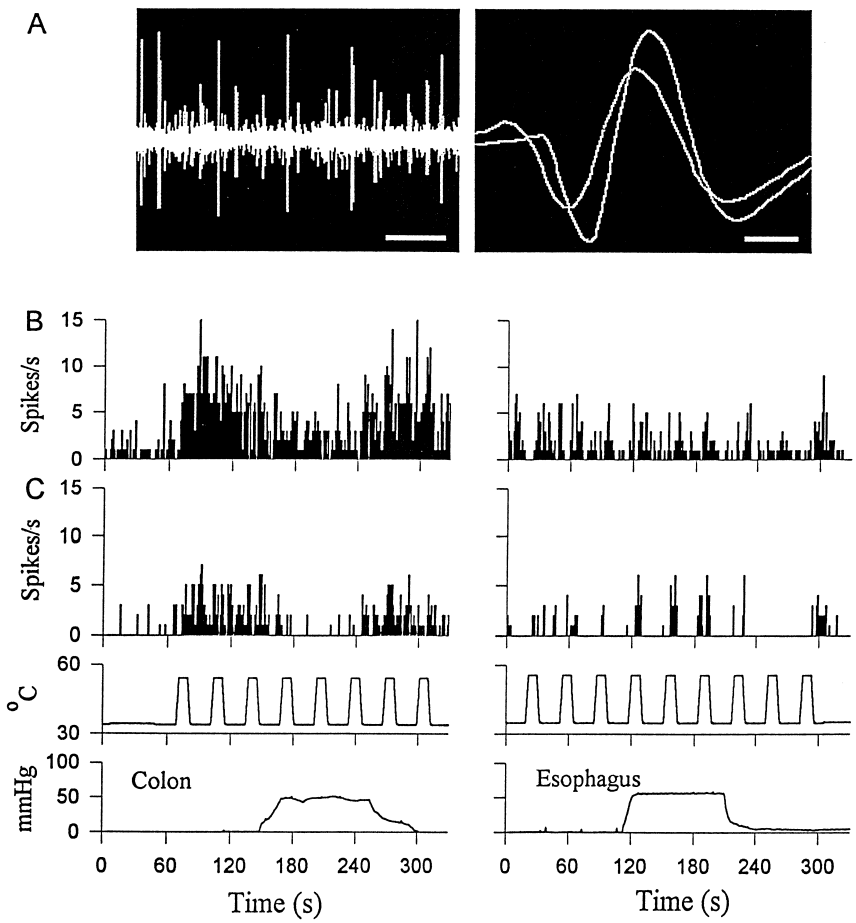

Fig. 3. Interaction of responses of two neighboring VPL neurons to noxious heat applied to digit 5 of the foot shown as original registration and PSTHs (A, B, C; bin width 1 s). (A) On the left side a photograph of an original registration taken from the screen of an oscilloscope (bar length is 1 s) is shown. On the right side the differences in shape and amplitude of the two discriminated spikes are shown (bar length 0.2 ms). The shape and intensities of the noxious heat pulses (52°C; duration 10 s; interstimulus interval 10 s) are shown in the curves immediately underneath the PSTHs. The curves in the lowest panels show the intraluminal pressure changes within the distal colon and the lower esophagus.

the foot were inhibited by noxious colon distension (left A, B graphs, inhibitory interaction $I(\text{Colon},S) = -79\%$ (A) and -100% (B). Noxious distension of the esophagus, however, had no significant effect on the responses to somatic stimulation in the first unit (A, right graph), but had a facilitatory (i.e., positive multiplicative) effect in the second neuron, $I(\text{Esophagus},S) = 213\%$ (B, right graph). Thus, these two neighboring neurons both received inhibitory interactive colon inputs, one did not receive any esophagus inputs, while the other received facilitatory esophagus inputs. These visceral effects were only modulatory, because visceral stimulation alone had no effect.

3.1. Population response

The mean change in discharge rate to somatic stimulation alone (ΔS) of all 13 neurons with interactions was 4.66 ± 6.06 spikes/s, that to visceral stimulation alone (ΔV) was 0.07 ± 3.2 spikes/s, and that to conditioning (ΔVS) was 1.79 ± 6.03 spikes/s, resulting in a population $I(V,S) = -62\%$. Thus, considering the population of neurons that showed interactions between somatic and visceral inputs, visceral stimulation alone showed a minimal change in population firing rate (although nearly half of the individual neurons had visceral responses), but significantly decreased the population response to the somatic input during conditioning. Facilitatory interactive effects were seen in two units only, where $I(V,S)$ ranged from 213% to 844%. All other interactions were inhibitory, ranging from -38% to -590%.

3.2. Distension intensities for interactive effects

Visceral activation thresholds were around 30 ± 3 mm Hg for seven neurons (five to colon, and two to esophagus distension), while in two neurons the thresholds were around 55 mm Hg. The first group corresponds to visceral wide dynamic range type neurons (WDRv), and the second group to visceral nociceptive specific (NSv) type neurons [5]. In four neurons the conditioning effects were observed at distension intensities corresponding to their threshold for visceral responses alone, while in five neurons the conditioning effects were observed at 20 mm Hg above visceral threshold. Thus, in at least half the neurons with interactions the interactive effects were observed at visceral distension intensities below the noxious range (see Ref. [5]).

4. Discussion

The results show that the responses to visceral stimulation in 13 out of 29 VPL neurons were modulated by convergent conditioning somatic inputs, that is the somatic and visceral systems interacted. In a subset of VPL neurons, characterized as purely somatic according to separate somatic and visceral stimulation, the visceral inputs were only revealed during conditioning (i.e., simultaneous somatic and visceral stimulation), because visceral stimulation alone had no effect on the units' ongoing activity. As demonstrated in our earlier study in the squirrel monkey [5], at least 85% of the VPL neurons have convergent viscerosomatic inputs. The fact that in the present study for some of the neurons visceral inputs could only be demonstrated by conditioning, suggests that the proportion of visceroceptive neurons could be even higher.

These neurons most likely subserve two different tasks under different conditions, i.e., firstly, coding visceral stimulus intensity and duration, and thus most certainly playing a role in the discriminative aspects of visceral nociception, and secondly, suppressing somatic responses when somatic and visceral stimuli are applied together, to facilitate visceral information processing.

The recording sites were not verified histologically because they were not marked with electrolytic lesions to minimize brain damage in surviving animals. Most likely, however, we investigated the same population of lateral thalamic neurons as described in our previous studies where extensive histological analysis was performed [3,5], because the somatic and visceral responses, receptive field sizes and sequences were not distinguishable from the VPL neurons investigated in the previous studies. In the present study only a limited range of intensities and kinds of stimulation (mainly innocuous intensities in case of somatic stimulation and mainly noxious visceral stimulation) was applied; therefore, a variety of other possible interactions remain to be studied.

4.1. Supraspinal interactions

The dominant viscerosomatic interactive effect was inhibitory. This finding is in agreement with two other studies performed at the thalamic [1] and the cortical level [7] in the cat. Both studies used electrical stimulation of somatic and visceral nerves. Airapetyan et al. [1] stimulated the radial (somatic) and splanchnic (visceral) nerves and recorded from VPL neurons. They observed that both somatic responses conditioned by visceral stimulation, and vice versa, were suppressed.

Chernigovskiy et al. [7], using the method of evoked potentials, demonstrated interactions between the somatic and visceral systems in the second somatosensory cortex (SII) and in the parietal cortex of the cat. They found also that both systems had inhibitory effects on each other, and that irrespective of which system was activated first, the responses to stimulation of the second system were suppressed. Thus both systems have been shown to be able to suppress the other one at the thalamic and cortical levels.

4.2. Spinal interactions

The studies cited above and our present report of interactions between the visceral and somatic systems are

partially in agreement with numerous other investigations, which show that remote noxious, but not innocuous somatic and visceral stimuli inhibit nociceptive and non-nociceptive responses in spinal dorsal horn neurons (e.g., Refs. [6,10–13,15,17]).

4.2.1. DNIC

Le Bars et al. [10] investigated a specific effect, which they termed 'diffuse noxious inhibitory control' (DNIC), where remote (i.e., heterosegmental) noxious, but not innocuous stimuli suppress the ongoing activity and responses of wide dynamic range (WDR) neurons (convergent neurons; Ref. [10]) in the spinal dorsal horn. Prolonged inhibitions, termed post effects, were also consistently observed during DNIC. Nociceptive specific, non-nociceptive and proprioceptive neurons were not affected by DNIC. These inhibitory controls disappeared when the animals were spinalized [12]. Thus, this effect is different from homosegmental inhibitory effects described by others (e.g., by Ref. [9]), where in addition innocuous somatic stimuli can suppress neuronal activity.

Although DNIC has not been studied in the monkey thalamus, our findings speak against the view that the inhibitory interactive effects observed were primarily due to DNIC. Firstly, there were no observable differences between the direction of the interaction caused by esophagus or colon distension and the location of the somatic receptive field on the upper or the lower body, respectively. Secondly, although the dominant conditioning effect was inhibition, facilitatory effects were also observed (also seen in the rat spinal cord by others [14,15], see below). Furthermore, due to the experimental paradigm, interactions were observed in neurons with somatic low threshold response properties, irrespective of the location of somatic or visceral receptive fields, i.e., neither the stimulus modality (noxious), nor location (remote) conditions are necessary to observe thalamic interactions. Therefore, it seems that viscerosomatic interactive inhibitions are more generalized in the thalamus than in the spinal cord. Thus, although DNIC type mechanisms at the level of the spinal cord certainly must be reflected in the interactions we observed in the thalamus, circuitry other than DNIC must contribute to the thalamic viscerosomatic interactions.

4.2.2. Nocigenic inhibition

This view is supported by Ness and Gebhart [14,15], who investigated a comparable, but more general phenomenon, which they proposed to be termed 'nocigenic inhibition'. This was defined as ''...the inhibition of neuronal, behavioral, or reflex responses to a nociceptive test stimulus produced by another nociceptive stimulus, and the term applies to segmental and intraspinal (i.e., propriospinal) as well as supraspinal levels of interaction''. Thus, the definition of the diffuse inhibitory effects includes DNIC, but is kept more general than the phenomenon of diffuse and remote inhibition defined by Le Bars et al. [10]. DNIC and nocigenic inhibition partially overlap, but are not the same.

There are similarities and differences between the present study and those by Ness and Gebhart [14,15] in the rat spinal cord. The studies show that the dominant viscerosomatic interactive effect is inhibitory, and that a small number of neurons exhibit excitatory interactions. However, in the rat spinal cord more remote stimuli (= heterosegmental: 106 of 129 trigeminal–cervical neurons) seem to be more effective in inhibiting test stimuli than less remote conditioning stimuli (perisegmental: 59 of 100 L3–L5 neurons). This tendency was not seen in the monkey thalamus, although the numbers in our study are small. Differences between spinal cord and thalamus interaction properties can readily be explained by the variety of convergent inputs on lateral thalamic neurons (e.g., see Ref. [5]).

4.3. Psychophysically studied interactions

In a psychophysical investigation involving nine healthy humans it was demonstrated that not innocuous, but noxious gastric distension is capable of inhibiting a nociceptive spinal flexor reflex (RIII) in a graded manner [4]. This clearly is in agreement with the phenomenon of DNIC. However, that there are in addition more general mechanisms active in awake humans other than DNIC has also been demonstrated in another psychophysical study [8]. Here the perceptions of eight healthy subjects elicited by gastric and duodenal distension were investigated. After determining the thresholds for discomfort, low (just perceivable) and high (non-painful), transcutaneous electrical nerve stimulation (100 Hz, 100 μs) was used as the somatic conditioning stimulus. The authors demonstrated that well perceivable, but non-painful remote stimulation of somatic nerves suppressed the perception of unpleasant visceral sensations, because the thresholds for perception of gut distension were increased. Thus, in healthy humans unpleasant visceral sensations are suppressed by innocuous somatic stimuli. This effect, which is clinically well known as counter-irritation [18], has been studied in humans experimentally with respect to somatosomatic nociceptive interactions in numerous investigations and will not be discussed here (for review and references see Refs. [14,15]).

4.4. Quantitative measure of interaction

In the present study interaction was defined by calculating the difference between the expected activation, i.e., the sum of the number of action potentials during somatic stimulation alone and during visceral stimulation alone, and during conditioning, i.e., when both stimuli were applied at the same time. The ongoing activity before each stimulation was subtracted to quantify only response related spikes. With this approach the strength and direction

of the interaction is indicative of the properties of the underlying synaptic circuitry. This linear approach to calculate interactions is simplistic. However, the approach is useful because we assume that the somatic and visceral inputs reach the neuron via independent channels (or codes). The fact that somatic and visceral information is conveyed in the CNS generally by convergent viscerosomatic neurons, does not contradict this assumption, because in principal the same pathways can be used to transmit information about different kinds of stimuli (touch, pressure, pinch, heat, cold, etc., like in polymodal neurons, or somatic and visceral information, like in viscerosomatic convergent neurons). We assume that the activation of both channels will result in a summation of EPSPs and IPSPs in single cells, and thus result in action potentials, which reflects the sum of the two independent inputs. For CA1 pyramidal neurons it is known that synapses in various parts of the dendritic tree are nearly equally effective in this regard. Andersen [2] wrote: ''EPSPs produced by neighboring synapses sum linearly, both with each other and with hyperpolarizing, inhibitory potentials''. Shadlen and Newsome [16] state that cortical EPSPs sum linearly too. Given these results we assume that thalamic inputs sum linearly as well. Thus, if the number of spikes during conditioning is different from the sum of the spikes during separate somatic and visceral stimulation, there must have been cross talk between the two channels before the combined information reached the unit. However, there are also arguments for non-linear (in the sense of non-additive) mechanisms involved in the generation of spikes.

In the earlier studies cited above interactions between different input channels were quantified by the percent change in either ongoing activity or evoked responses (steady state; Refs. [10,12,14,15]), where the ongoing activity or response to a test stimulus was defined as 100%. In contrast, we defined the additive effect (i.e., $\Delta V + \Delta S$) as 100%, because this approach indicates differences as well as demonstrates latent visceral inputs onto neurons, in which the ongoing activity is not affected by the visceral (test) stimulus ($\Delta V = 0$).

4.5. Populational interactions

The psychophysical data reported by Coffin et al. [8] are in good agreement with the physiological data reported here, and indicate that part of the effects we see is due to cross modality suppression, i.e., a competition between visceral and somatic inputs.

The fact that there exists a sub-population of neurons with no changes in neuronal activity to visceral stimulation alone, but with reduced somatic responses during concurrent visceral stimulation imply the existence of a circuitry to amplify the less frequent and less dominant visceral inputs. This is in agreement with the population response of the neurons exhibiting interactions in the present study. Here visceral stimulation alone had no significant effect, but the excitatory responses to somatic stimulation were suppressed, when at the same time visceral inputs were present. This interaction circuitry would function in increasing the signal to noise ratio for processing visceral information. In the behaving organism the thalamus is constantly bombarded by somatic inputs and less by visceral, especially noxious visceral inputs. Thus, the interaction circuitry provides a mechanism for amplifying the less frequent inputs.

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