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·Original Article·

Effects of pentobarbital anesthesia on nociceptive processing in the medial and lateral pain pathways in rats

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Abstract: Objective To investigate the effects of pentobarbital anesthesia on nociceptive processing in the medial and lateral pain pathways. **Methods** Laser stimulation was employed to evoke nociceptive responses in rats under awake or anesthetic conditions. Pain-related neuronal activities were simultaneously recorded from the primary somatosensory cortex (SI), ventral posterolateral thalamus (VPL), anterior cingulate cortex (ACC), and medial dorsal thalamus (MD) with 4 eight-wire microelectrode arrays. **Results** Compared with the awake state, pentobarbital anesthesia significantly suppressed the neural activities induced by noxious laser stimulation. Meanwhile, the pain-evoked changes in the neuronal correlations between cortex and thalamus were suppressed in both medial and lateral pain pathways. In addition, the spontaneous firing rates in all the 4 areas were altered (including inhibition and excitation) under the condition of anesthesia. **Conclusion** The nociceptive processing in the brain can be dramatically changed by anesthesia, which indicates that there are considerable differences in the brain activities between awake and anesthetized states. It is better to employ awake animals for recording neural activity when investigating the sensory coding mechanisms, especially pain coding, in order to obtain data that precisely reflect the physiological state.

Keywords: anesthesia; laser; multiple single-unit recording

1 Introduction

Pain is a multidimensional phenomenon. It is composed of sensory-discriminative and affective-motivational components, which are processed by parallel neural systems[1]. The "lateral pain system", including lateral thalamic nuclei and somatosensory cortex, is thought to mainly transmit the information of the sensory features of pain stimuli, such as stimulus location, duration, intensity, and quality. On the other hand, the "medial pain system" including the medial thalamic nuclei and cingulate cortex, has been proposed to mediate the affective-motivational aspects of pain[2-4].

It is well-known that anesthetics can strongly modulate functional properties of cortical neurons[5,6]. Pentobarbital is one of the commonly used hypnotics/anesthetics. It can potentiate the effects of γ-aminobutyric acid (GABA) through acting at its own receptor site on the GABA/receptor ionophore complex[7-9]. However, its effects on nociceptive responses have not been clearly defined. Also, the effects of pentobarbital anesthesia on pain processing in medial and lateral pain pathways are still unknown.

Currently, many studies on pain have been conducted, using anesthetized rats as subjects. However, discrepant results may be obtained, due to the different states (awake or anesthetized) of animals. An fMRI study has elucidated a

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ketamine-mediated nociceptive map and strong activations in the frontal subcortical regions[10]. Shaw *et al.* have demonstrated that pentobarbital could strongly modify various components of noxious laser-evoked potentials[11]. It has been reported that the neurons within the medial and the lateral pain pathways are activated by cutaneous noxious laser stimulation in behaving rats. To clarify the influence of anesthetics on nociceptive processing in the brain, single units across the parallel pain pathways were recorded in pentobarbital-anesthetized rats with brief laser stimulation on the rat hind paws.

2 Materials and methods

2.1 Animals Four adult male Sprague-Dawley rats (300-350 g) were housed individually under a reversed light/dark cycle (light off from 7:00 AM to 7:00 PM) for 7 d, with access to food and water *ad libitum*. All the experiments were performed in accordance with the Institutional Animal Care and Use Committee of Chinese Academy of Sciences.

2.2 Surgery Prior to chronic implantation of microelectrode array, initial anesthesia was administered by ketamine injection (100 mg/kg, i.p.). Supplementary doses (1/3 of the original dose) of ketamine were given when needed, to maintain a proper anesthetic depth during surgery. Rats were mounted on a stereotaxic apparatus (David Kopf Instruments, Tujunga, CA), and 4 arrays of eight stainless steel Teflon-insulated microwires (50 μm in diameter; Biographics, Winston Salem, NC) were slowly lowered into the following target areas: (1) primary somatosensory cortex (SI), -1.0 mm posterior to Bregma (A), 2.0 mm lateral to the midline (L), and 2.0 mm to the skull surface (V); (2) anterior cingulate cortex (ACC), 3.2 mmA, 0.8 mm L, and 2.8 mm V; (3) medial dorsal thalamus (MD), -2.3 mm A, 0.8 mm L, and 5.5 mm V; (4) ventral posterolateral thalamus (VPL), -3.0 mm A, 3.0 mm L, and 6.0 mm V, according to the Rats Atlas of Paxinos and Watson[12]. Six stainless steel screws were driven into the skull to serve as anchors for cementing the microwires in place after implantation. Before surgery, animals all received penicillin injection (16 000 U, i.m.) to prevent infection, and after surgery, they were allowed to recover for one week before recording sessions commenced.

2.3 Electrophysiological recording To record the neuronal activities, rats were placed in a plastic chamber (44 × 44 × 44 cm^3) in a quiet room at (22 ± 1) °C and allowed unrestricted movement during the entire recording session. Neural spike recording started after the rats became adapted to the experimental environment. Extracellular signals were collected by the chronically implanted microwire assemblies that were connected to a preamplifier via a head stage plug and 2 lightweight cables. The outputs of the preamplifier were filtered (0.5 and 5 kHz, 6 dB cut-off) and sent to a multichannel spike-sorting device (Biographics, Inc.) for online signal processing. Waveforms were discriminated individually by setting multiple time-voltage windows using a PC-based software Magnet (Biographics, Inc.). The time stamps of these waveforms were then stored on a personal computer for off-line analysis. Graphical capture of waveforms, interspike interval histograms and autocorrelograms were used to validate the on-line sorting of single unit. Spike train activity was analyzed with the PC-based programs Stranger (Biographics, Inc.) and NeuroExplorer (Plexon, Dallas, TX).

2.4 Laser stimulation The stimulus was generated from a CO_2 laser stimulator (Institute of Optical Precise Engineering and Physics, Chinese Academy of Sciences, China) in a single pulse mode, at the wavelength of 10.6 μm, with a beam diameter of 2.5 mm and a pulse duration of 20 ms. CO_2 laser beam projection was guided by a helium laser producing a red light spot. Laser pulses with increasing intensities (120, 130, 140, 150, 160 and 180 mJ) were applied to the plantar surface of the hind paw contralateral to the microwire implantation.

2.5 Experimental procedures At the beginning of the experiments, the rat was awake and free-moving, and 40 trials of 140 mJ laser stimulation were delivered to the hind paw when the rat was quiet and showed no voluntary motor activity. Here the intensity of 140 mJ was determined to be noxious according to the observation of obvious withdrawal of the hind paw and the previous study in our lab[13]. After that, the rat was anesthetized with sodium pentobarbital (50mg/kg, i.p.) and was monitored by respiratory rate (50-80 breaths/min), eyelid reflex, pinch withdrawal and vibrissal movement, to maintain the stage of medium anesthesia[14]. Supplemental injection of sodium pentobarbital was adopted

when the first clear sign of vibrissal movement was observed. Stimuli of increasing intensities were applied on the same paw in the state of anesthesia. Each recording block consisted of about 40 trials with the same energy level.To minimize the tissue damage, sensitization and habituation, the stimuli were randomly applied to a local skin area and the interstimulus interval was no less than 30 s.

2.6 Data analysis The neuronal firing rate was quantified for each neuron using peri-stimulus time histograms (PSTHs). The bin size was 0.01 s for the computation of PSTHs. Bin counts for each trial were calculated using the analysis program NeuroExplorer (Plexon, Dallas, TX) and the results were exported to Matlab (The MathWorks, Inc.) in spreadsheet form. Neural responses to noxious stimulation were evaluated using a sliding window averaging technique, in which a 0.1-s time window was slid through the entire period of a trial at 0.01-s step. The bin counts of each window were compared with those of a preset 1-s control window 1 s before the stimulation event by Student's *t*-test. The differences were considered significant only when it reached a significance level of $P < 0.005$ in three consecutive steps, thus achieving a global significance of $P < 0.05$. Non-parametric chi-square test was used to determine the significant differences of numbers of neurons across responses between anesthesia *vs* awake sessions. Units that showed significantly increased activities after laser stimuli were defined as excitatory. To compare the neural responses between different sessions, the neuronal firing rates were transferred into *Z*-scores using MatLab program: $Z = (X - M)/S$, where X represented the actual firing rate obtained from PSTH, and M and S indicated the mean and the standard deviation of the baseline discharging (-1– 0 s), respectively. The firing rates for all neurons were normalized and arranged into a spreadsheet. A clustering analysis (*K*-means, SPSS) was performed to classify neuronal responses depending on the similarities in patterns of excitation or inhibition around stimulation events.

To compare the tempo-spatial neuronal response pattern between awake and anesthesia conditions, discharges of each neuron were counted in 100-s bins with a time range from 50 min pre- to 150 min post-pentobarbital application. Firing rates in each bin were transferred into *Z*-scores, and clustering analysis was performed.

Cross-correlation histograms were created by the same computer analysis software. One neuron within a given region was selected as the reference neuron, and all the neurons from another region were defined as partner neurons for the cross-correlograms. The time of occurrence of spikes from the reference neuron was set at 0 s and the partner neuron's firing 0.5 s before and after each reference neuron's spike was plotted using 5-ms bin size. The significance level of the cross-correlogram was tested using 95% confidence intervals. Data falling into the 10 s period around laser stimulation (5 s before and 5 s after stimulation) were calculated separately. Non-parametric chi-square test was used to determine the significant differences of numbers of neuron pairs between anesthetic *vs* awake sessions.

2.7 Histology After the termination of the experiment, rats received an overdose of ketamine. Recording sites were marked by electrophoretically deposited iron (10-20 μA DC current, 10-20 s duration, anode at the electrode) at the tips of selected wires. Animals were then perfused with 4% paraformaldehyde. The brains were post-fixed in a solution containing 5% potassium ferricyanide and 4% paraformaldehyde for several days. Coronal sections (40 μm) were cut through SI, ACC and the thalamus. Recording sites were determined under a light microscope. The iron deposits were easily identified as blue dots.

3 Results

3.1 Laser-evoked nociceptive responses A total number of 131 neurons were recorded from the 4 rats (30 SI, 36 ACC, 36 VPL and 29 MD). Fig. 1 showed the neuronal responses of cortex and thalamus arranged in clusters according to the temporal sequence. As shown in Fig. 1, the 4 areas were all strongly activated by noxious laser stimulation under awake condition. Out of the 36 units recorded from the ACC, 23 (63.9%) showed significant excitatory responses to noxious laser stimulation. In the MD thalamus, 22 of 29 (75.9%) units were activated. In SI cortex, 86.7% of units displayed increased alterations in the firing rate. In addition, 61.1% of the recorded units increased the spike activity in VPL thalamus. Compared

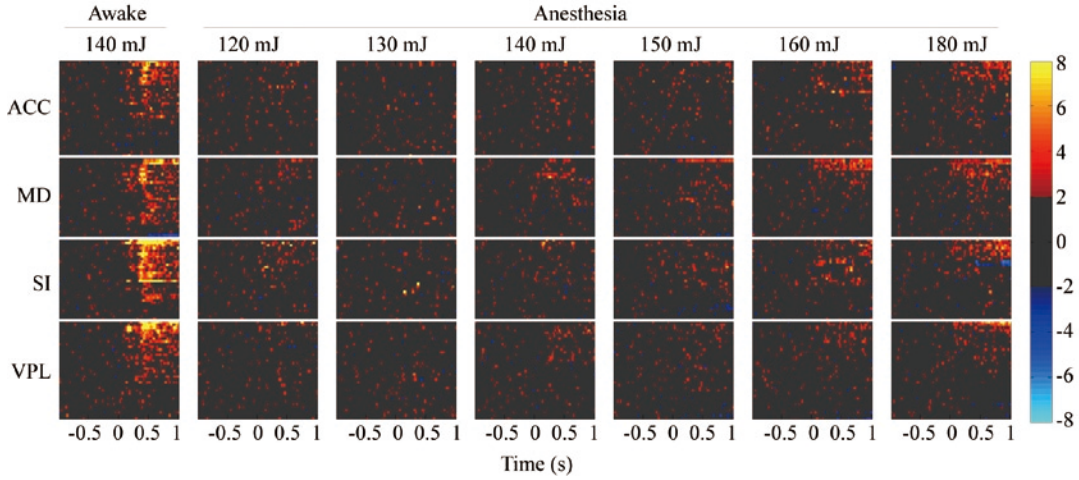

Fig. 1 Neuronal firing patterns in response to the laser stimulation at increasing intensities before and after anesthetic administration. Under the awake condition, all the 4 areas were strongly activated by the noxious laser stimuli. Following pentobarbital injection, the relative response magnitude with stimulus intensity of 120-180 mJ was much weaker and the number of responsive neurons was much smaller than that in the awake state. Cluster plots depicted neuronal activities from 1 s pre-stimuli to 1 s post-stimuli. The firing rate of each neuron (indicated with a line in the image) was normalized to an average of 0 and a standard deviation of 1 to display relative changes (light yellow for the highest frequency and light blue for the lowest). Time = 0 s on the transverse axis corresponded to the onset of stimulation.

to the awake condition, the proportion of excitatory neurons in all the 4 areas was dramatically decreased following pentobarbital treatment. The relative response magnitude with stimulus intensity of 120-180 mJ was much weaker and the number of responsive neurons was much smaller than that in the awake state. Note the difference in the discharging frequency between awake and anesthesia states in Fig. 1. The laser intensity of 120-150 mJ almost induced no changes in the neuronal activity under the condition of anesthesia. Take the intensity of 140 mJ as an example, only 2.8% of units in ACC and 3.4% of units in MD were activated. In SI and VPL, the number of excitatory neurons was zero. Rasters and peristimulus histograms depicting the average firing rate and typical excitatory response were illustrated in Fig. 2.

3.2 Changes in the temporal coding patterns To compare the difference in neuronal spontaneous activity between awake and anesthesia states, continuous recording was performed from 50 min pre- to 150 min post-pentobarbital

Table 1. Comparison of correlated neuronal activity between awake and anesthesia conditions

	Total pairs of neurons	Awake	Anesthesia					
			120 mJ	130 mJ	140 mJ	150 mJ	160 mJ	180 mJ
SI-VP correlations								
Number	268	64	20	24	33	36	34	33
Percentage	-	23.9%	7.5%***	9.0%***	12.3%**	13.4%**	12.7%**	12.3%**
ACC-MD correlations								
Number	258	62	30	39	40	23	29	11
Percentage	-	24.0%	11.6%***	15.1%*	15.5%*	8.9%***	11.2%***	4.3%***

$^{*}P < 0.05$, $^{**}P < 0.01$, $^{***}P < 0.001$, compared with the awake state. Data were analyzed with Chi-square test.

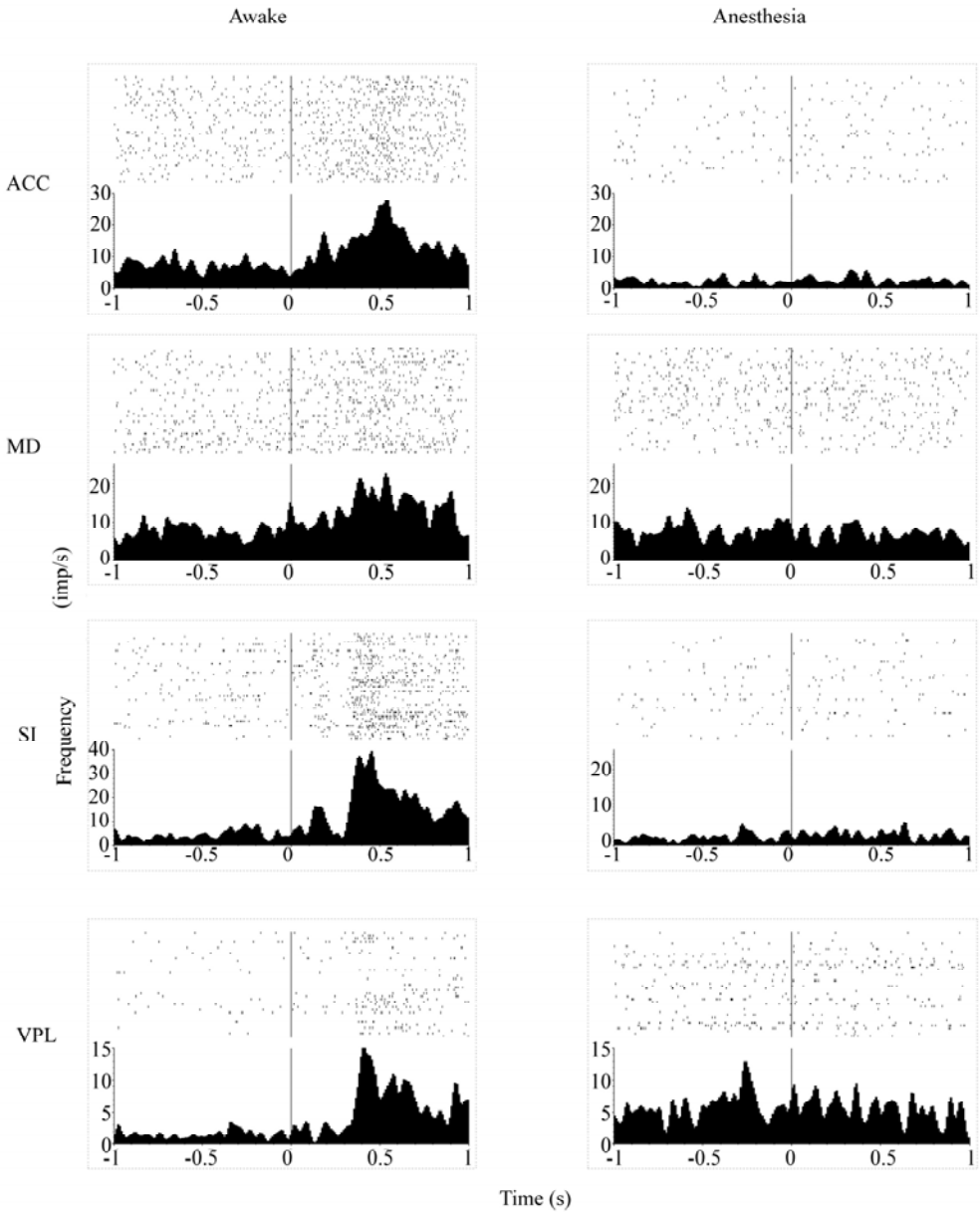

Fig. 2 Typical neuronal responses before (left column) and after (right column) anesthesia in the medial (ACC, MD) and the lateral (SI, VPL) pain pathways. The laser-evoked responses were significantly reduced in all of the 4 brain areas following pentobarbital anesthesia.

administration. The cluster analysis revealed the changes of spontaneous activity pattern of the thalamo-cortical neurons after pentobarbital anesthesia. In general, of the 131 neurons recorded from all the 4 areas, 12 (9.2%) and 23 (17.6%) exhibited persistent excitatory and inhibitory responses, respectively, following pentobarbital injection (Fig. 3A). Sorting neurons in each brain area according to the clusters revealed that neurons with strong inhibition were mainly located in SI. By contrast, most of the neurons with long-lasting activation were located in VPL (Fig. 3B). Examples of time histograms for each cluster were illustrated in Fig. 3C.

3.3 Changes in the neuronal cross-correlations Pairwise cross-correlations were calculated for simultaneously recorded neurons. Correlated activity was found between neu-

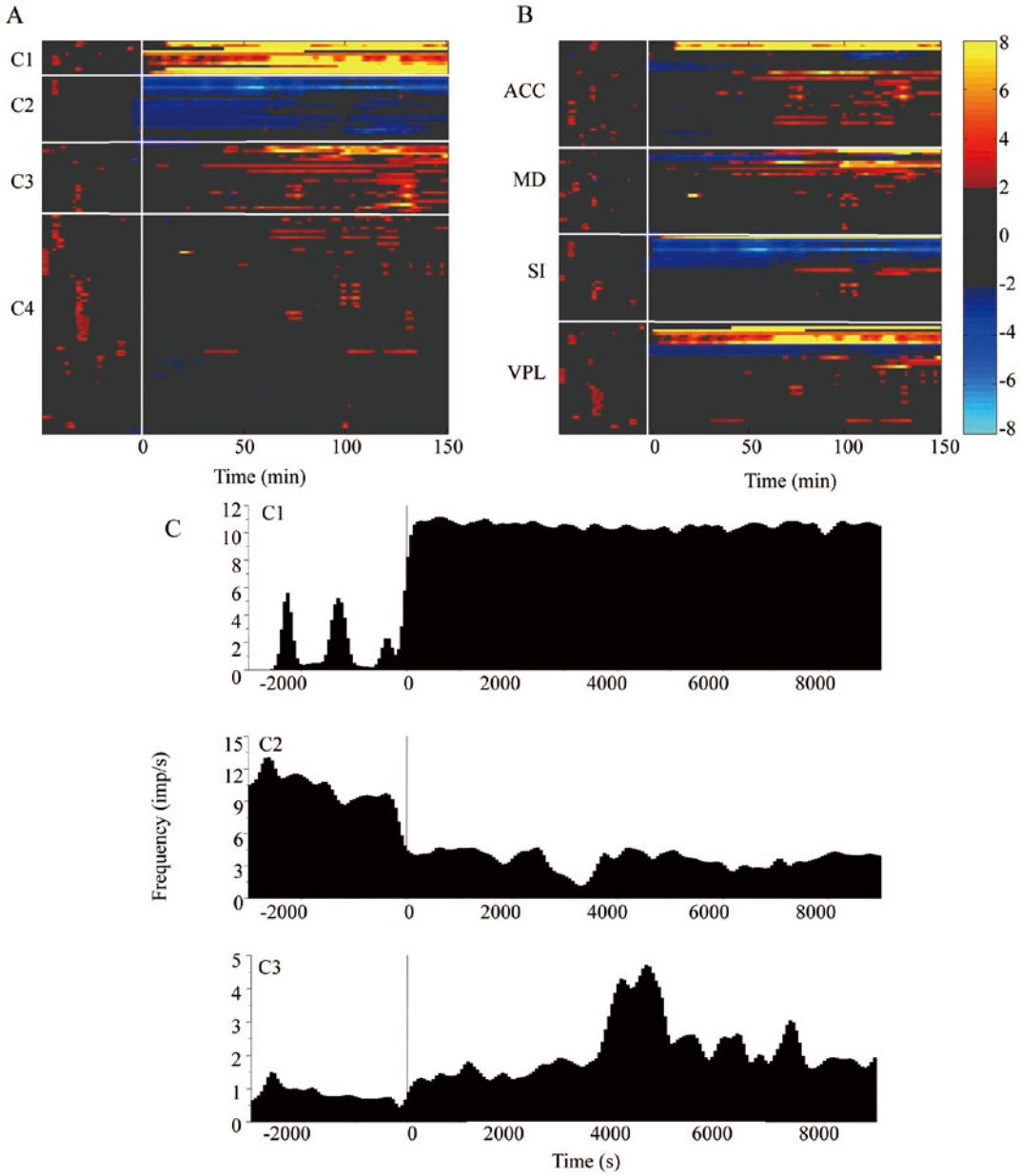

Fig. 3 Temporal distribution pattern of spontaneous neuronal activity before and after anesthesia. Zero indicated the onset of pentobarbital injection. Each line of the image in A and B represented normalized activity of one neuron. A: The cluster analysis revealed that the cortical and the thalamic areas contained units with 4 clusters (C1-C4) of coding patterns in response to the noxious laser stimulation. B: The temporal coding patterns in ACC, MD, SI and VP were significantly changed by pentobarbital injection. C: Examples of rate histograms for clusters C1-C3.

ronal pairs in both medial and lateral pain pathways. As shown in Table 1, the peri-stimulation correlations within the lateral pathway were significantly deceased following anesthesia as compared to that at awake state. The correlation between ACC and MD was also reduced after anesthesia.

3.4 Histology The location of microwires was revealed by the iron deposits at the tips of selected microwires. As shown in Fig. 4, in the cingulate cortex, most of the iron deposits were found in the anterior areas; in the somatosensory cortex, most of the recording tips were in the hind limb region; in thalamus, tips were mainly located in the mediodorsal and ventroposterior part, as indicated in reference[12].

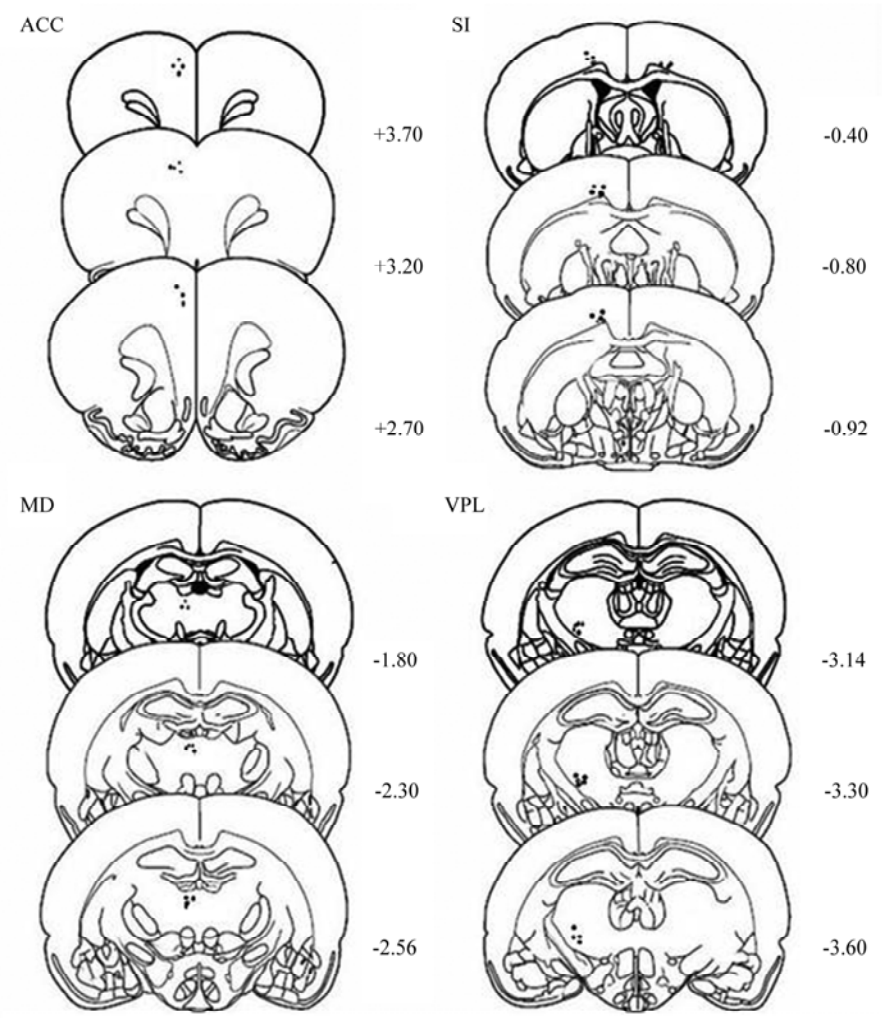

Fig. 4 A schematic drawing indicating the locations of recording sites in ACC, SI, MD and VPL. The black dots labeled the position of iron deposits as the tips of selected microwires.

4 Discussion

The present study investigated the effect of pentobarbital anesthesia on nociceptive processing in rats' medial and lateral pain pathways. Data demonstrate that the increased neuronal activities in ACC, MD, SI, and VPL induced by noxious stimulation could be significantly suppressed by pentobarbital anesthesia. The depressive effects included the decrease in the neuronal response magnitude, the reduction in the fraction of responsive neurons, and the shortening of response duration. Meanwhile, pentobarbital anesthesia could suppress the pain-evoked changes in the correlation between cortex and thalamus in both medial and lateral pain pathways.

Sodium pentobarbital is a short-lived barbiturate that is used as an anesthetic and euthanasia agent in animals, and serves as a sedative hypnotic clinically[15,16]. Shaw *et al.* have demonstrated that anesthesia could disturb the brain activity during pain processing, by recording the mechanical- and laser-evoked potentials in rat primary somatosensory cortex during pentobarbital anesthesia. They found that the major cortical components observed in the wakeful condition would disappear after pentobarbital administration[11]. Chapin *et al.*

investigated the effect of anesthesia on different latency components of cutaneous sensory responses of single units in the SI cortex of rats. The results showed that anesthesia slightly depressed the short-latency component exhibiting highly sensory properties but strongly inhibited the longer-latency components that exhibited relatively "nonspecific" properties[6]. Furthermore, some studies have reported that only in awake monkeys, a negative cortical component could be elicited by the cutaneous stimulation of hand[17,18]. These studies together with our results suggest that anesthesia can influence the sensory processing in brain and thus change the neural response pattern to environmental stimuli.

Besides, the spontaneous activities of thalamocortical neurons during awake and anesthesia states were recorded (Fig. 3). Significant inhibition was observed in some neurons after anesthesia. Given the fact that the pentobarbital could activate GABAergic receptors which are widely distributed in the brain and mediate inhibitory effects[19], there is no doubt that the neural activities are reduced after pentobarbital administration. However, in the present study, a small proportion of neurons became excited after pentobarbital administration, mostly located in VPL. This suggests that some silent VPL neurons in awake state are "aroused" by anesthesia. It is widely accepted that neurons in cortical layer VI give rise to excitatory feedback to the thalamus. The corticothalamic feedback axons terminate on the reticular nucleus (RTN) in addition to the relay neurons and interneurons in thalamic nuclei. Since the RTN neurons have inhibitory projections to the thalamic relay neurons[20], the corticothalamic feedback may inhibit the relay neurons through RTN and decrease the spontaneous neuronal activity. Thus, it is reasonable that the cortical inactivity by anesthesia leads to disinhibition of the thalamic relay neurons, thereby enhancing the spontaneous neuronal activity. Some studies also support that the thalamic neurons display spontaneous bursting activity and do not obviously respond to peripheral stimuli after anesthesia[21,22].

In the present as well as our previous studies, correlated neuronal activity has been found within the medial and lateral pain pathways, suggesting that the pain processing is definitely a result of synchronized activities across different neuronal circuits. It is noteworthy that a decreased neuronal correlation has been observed within each pain pathway following anesthesia. Thus, anesthesia may disturb the functional connection in the pain-signaling pathways, thereby preventing the nociceptive transmission and integration in the spinal and supraspinal regions.

At the single-neuron level, it is usually difficult to determine whether a given response is related to particular function, especially for a neuron in the brain and when the animal is anesthetized. We consider the response nociceptive because (1) the intensities of laser stimulation we employed evoke clear flexor reflex in awake animals; (2) the neurons we recorded are from thalamocortical pathways processing pain and other somatosensory signals; (3) no other concurrent stimulation from any sensory modal was applied; and (4) the neuronal response show clear time-lock with our stimulation.

In summary, the present study shows that the noxious laser-evoked neuronal responses in the cortical and the thalamic areas are dramatically changed after anesthesia. This suggests that it is better to employ awake animals for recording neural activity when investigating the sensory coding mechanisms, especially pain coding, in order to obtain data that precisely reflect the physiological state.

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戊巴比妥钠麻醉对大鼠痛觉加工内外侧通路信息处理的影响

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摘要：目的 探讨麻醉对大鼠痛觉加工内外侧通路信息处理的影响。**方法** 利用清醒动物神经细胞群单位放电多通道同步记录技术，在大鼠的初级躯体感觉皮层(SI)、丘脑腹后外侧核(VPL)、前扣带皮层(ACC)以及丘脑背内侧核(MD)埋置电极，给予对侧足底伤害性激光刺激，观察大鼠清醒状态下以及戊巴比妥钠麻醉状态下由激光刺激引发的各脑区神经活动的变化。**结果** 与清醒状态相比，戊巴比妥钠麻醉显著降低了伤害性激光刺激所引发的四个脑区神经元活动的增强，同时也抑制了由疼痛引起的内、外侧通路上脑区之间的同步电活动。另外，各脑区的自发放电频率也因麻醉而发生显著改变，包括抑制和增强两种情况。**结论** 麻醉能显著改变疼痛相关的神经活动，表明大脑活动在麻醉与清醒状态下有着很大差别。该结果提示，在研究感觉神经编码尤其是痛觉编码时，最好使用清醒动物记录神经活动，以获得能真正反映生理状态的实验数据。

关键词： 麻醉；激光；多通道记录