EMG Responses in Humans during Painful Heterosegmentary Stimulation

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The amplitude-temporal parameters of the nociceptive flexor reflex evoked in the upper and lower extremities by painful heterosegmentary electrical stimulation were studied in healthy volunteers. This reflex was detected bilaterally in the muscles of the upper and lower extremities independently of the site of painful stimulation. The maximum amplitude of the reflex was observed in the case, when the segmentary muscle innervation and the application site of painful stimulation coincided. The least latency of the nociceptive flexor reflex was observed after painful stimulation of the ear lobe. It was concluded that nociceptive flexor reflex is not an elementary polysynaptic spinal reaction, but involves also some supraspinal centers.

Key Words: *pain; nociceptive flexor reflex*

The nociceptive flexor reflex (NFR) is a typical defensive reflex, which is widely used in clinical practice [1,5,7,10] and experimental studies [10,13,14] for quantitative assessment of pain reactions. In common practice, NFR evoked by electrical stimulation of *n. suralis* or plantar surface of the foot is recorded [1, 11,15]. Sometimes NFR is recorded during painful stimulation of the fingers [2]. NFR is manifested by two components on the electromyogram (EMG): RII and RIII responses [1,6,11,15]. The latency of RII response is 40-60 msec. This component is associated with activation of thick low-threshold A_β-fibers, while RIII response appears with a 90-130-msec latency after electrical stimulation surpassing the excitation threshold for A_δ-fibers [1,9,15]. It is believed that NFR is a polysynaptic reflex closed up at the spinal level [11]. At the same time, NFR can be induced by heterosegmentary stimulation [4,10]. This fact is explained by the existence of the propriospinal system in the spinal cord responsible for ascending and descending intersegmentary neuronal interaction. However, spinal locomotion is a functional part of the motor system, which is controlled by the supraspinal motor centers [12]. Therefore, heterosegmentary responses can be explained by involvement of the supraspinal centers into NFR. Our aim was to evaluate the amplitude-temporal characteristics of NFR in the ipsi- and contralateral muscles of the upper and lower extremities during heterosegmentary painful stimulation.

MATERIALS AND METHODS

Twelve healthy male volunteers (age 35-54 years) were studied in compliance to International Association of Study of Pain (IASP). The volunteers were adapted to a comfortable reclined posture. Pain stimulation was performed with single rectangular electrical pulses (0.2 msec duration, 35-40 mA amplitude, 0.5 Hz repetition rate), applied to the ear lobe, the 1st and 2nd phalanges of the index and long fingers, and the phalanges of the I and II toes. NFR was recorded in *m. tibialis anterior*, *m. extensor carpi radialis longus*, and

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m. thenar via the bipolar surface electrodes (5 mm diameter) during weak tonic back flexion of both feet or simultaneous weak flexion of the hand with thumb adduction. EMG was amplified in an TESY-VIII electrophysiological system within the frequency band of 20-20,000 Hz. The tonic strength developed by the muscles was maintained at a constant level of 100-150 μV by visual control of the EMG oscillations. The amplified EMG was digitized and fed to a computer, which averaged it by modulus (averaging of the initial integrated EMG). The original software recorded the evoked EMG with a repetition rate of 0.8 msec and averaged 150-200 records. Origin 3 software was used to analyze the temporal and amplitude NFR parameters. The results were analyzed statistically using Student's *t* test and the non-parametrical Wilcoxon's test.

RESULTS

Electrical stimulation of the ear lobe, fingers, and toes evoked EMG responses in the muscles only when the strength of the stimulus was perceived as painful. When

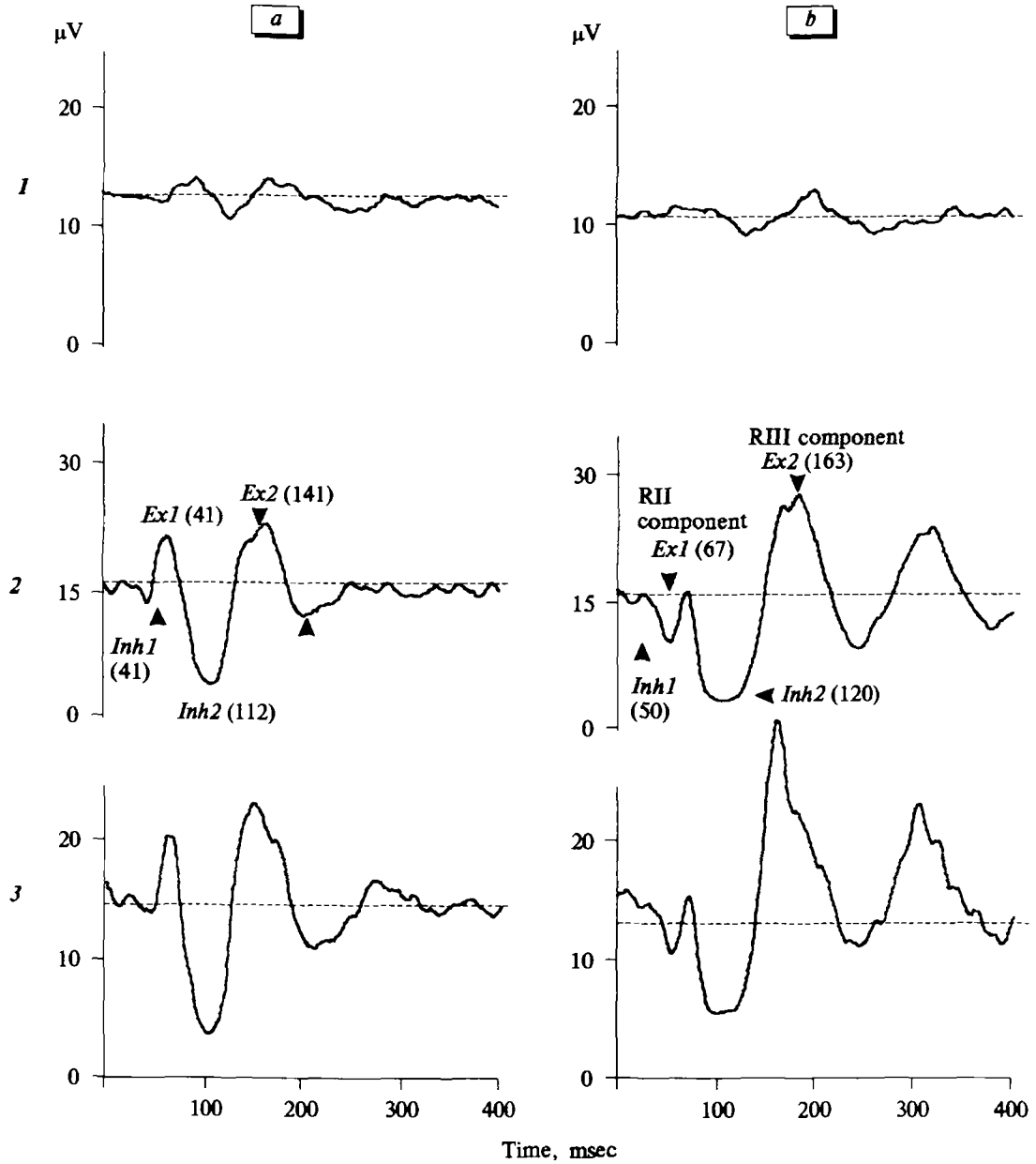

Fig. 1. EMG responses *m. thenar* (*a*) and *m. tibialis anterior* (*b*) during tactile (*1*), weak (*2*), and strong (*3*) painful electrical stimulation of the index finger (*a*) and toes (*b*). Here and in Figs. 2 and 3: E1 and I1 indicate the excitatory and inhibitory phases of RII component, E2 and I2 mark the excitatory and inhibitory phases of RIII components, correspondingly; numbers in brackets show latency (msec) of the inhibitory and excitatory phases. Ordinates: EMG amplitude.

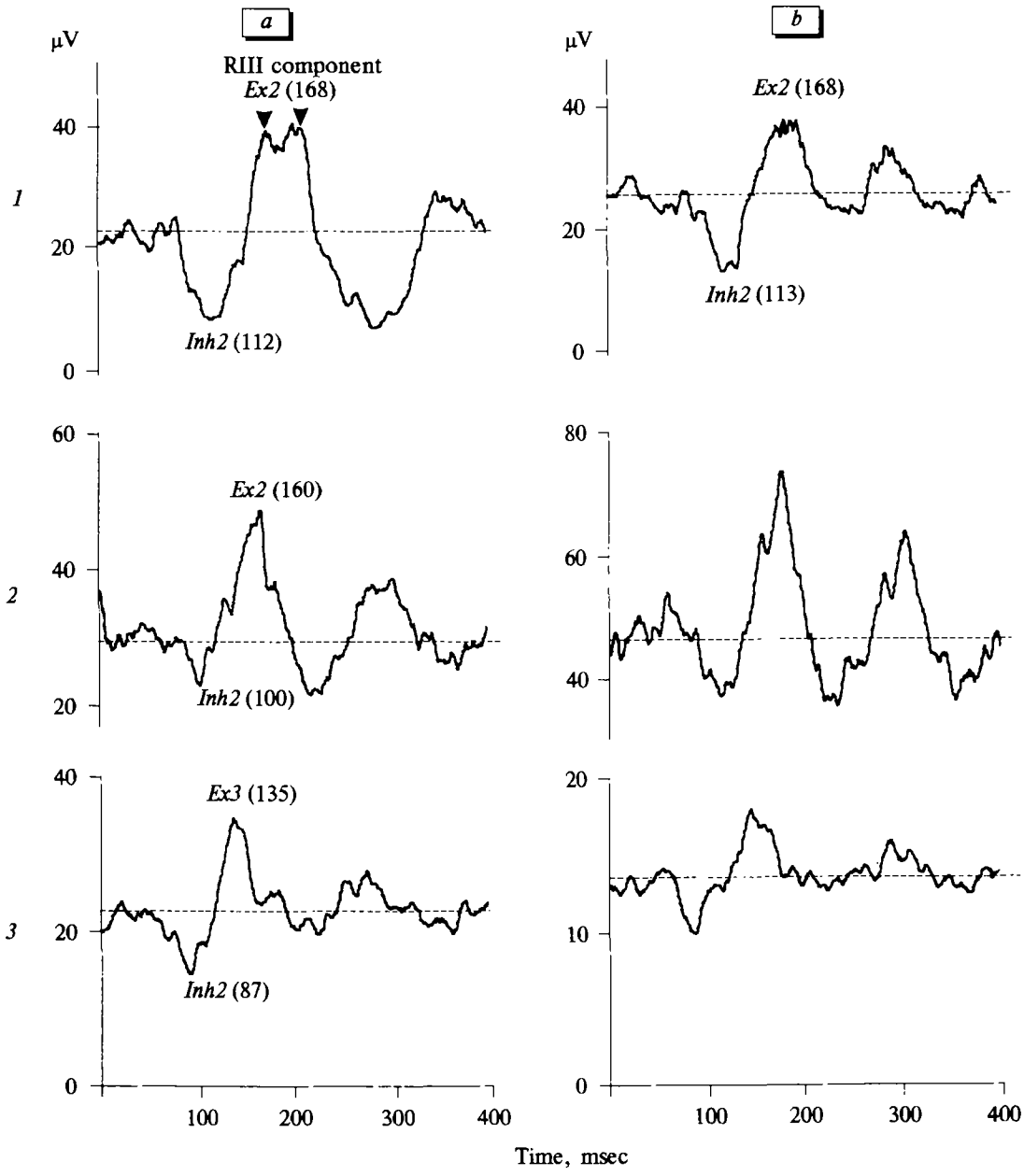

Fig. 2. EMG responses in ipsi- (*a*) and contralateral (*b*) *m. tibialis anterior* during painful electrical stimulation of the toes (*1*), fingers (*2*), and ear lobe (*3*).

the stimuli was painless, EMG responses in the muscles were not recorded (Fig. 1, *1*). Increasing the strength of stimulating current to a subjective pain threshold was accompanied by appearance of EMG responses in the muscles (Fig. 1, *2*). Further increase of stimulation current was assessed by patients as a pain of moderate intensity, the amplitude of EMG responses increased (Fig. 1, *3*). Independently of the site of stimulation, painful electrical stimuli evoked bilateral EMG responses in the muscles of the upper and lower extremities. The patterns of EMG responses consisted of alternating inhibitory and excitatory phases (Figs. 1, *2*; 2, *1*; 3, *1*). Despite the similarity of EMG responses in *m. tibialis anterior*, *m. extensor carpi radialis,* and *m. thenar* during painful transcutaneous stimulation of the ear lobe, fingers, and toes, their amplitude and temporal parameters were different. The amplitudes of the inhibitory and excitatory waves attained the maximum in the cases, when painful stimulation was applied to the zone of segmentary innervation of the corresponding muscle (Figs. 2, *1*; 3, *1*). The latency of the inhibitory and excitatory phases gradually decreased in the following series of the stimulation sites: ear lobe>fingers>toes (Figs. 2 and 3). In the other words, the latency of RIII component decreased when the site of stimulation approached the

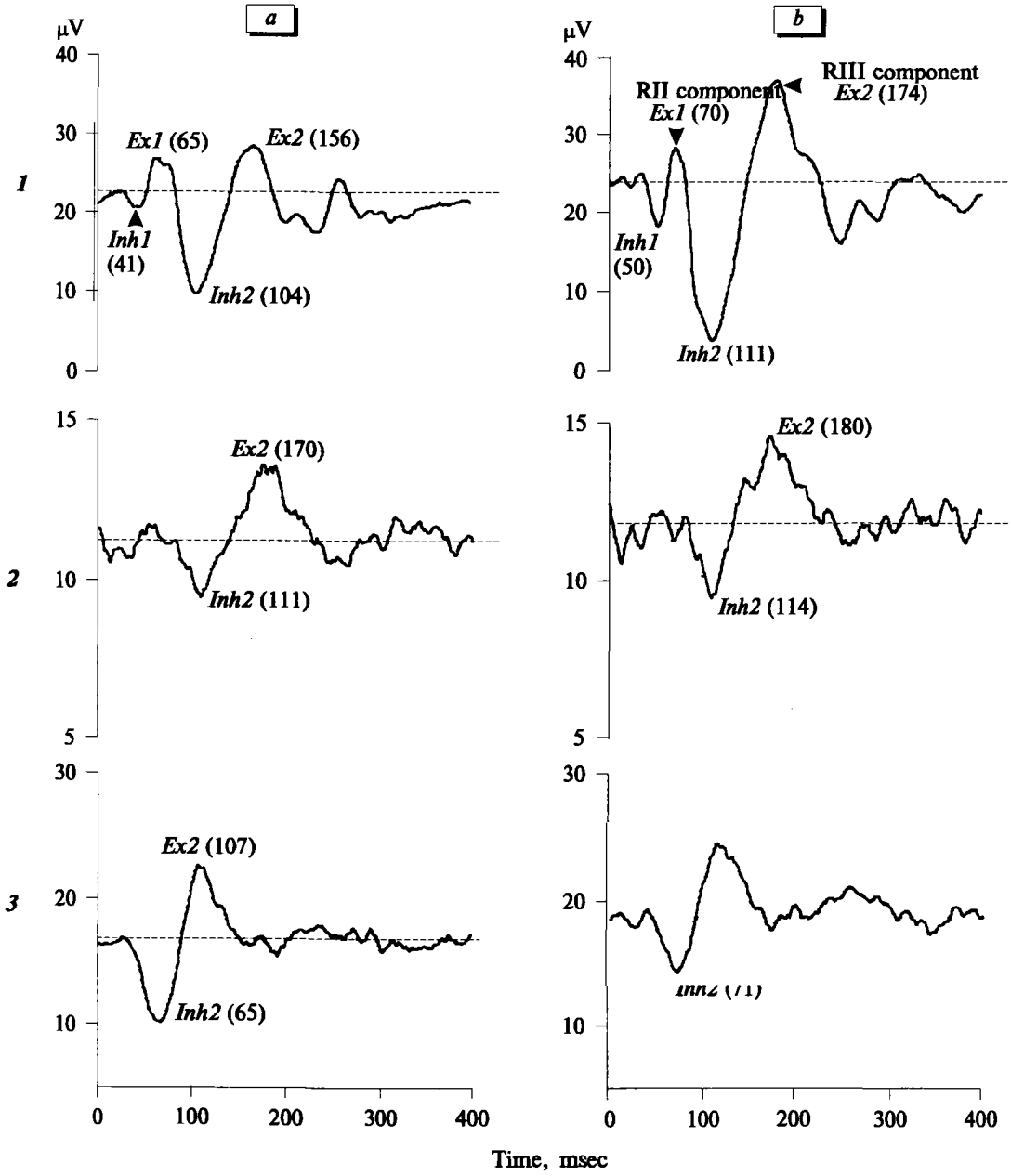

Fig. 3. EMG responses in *m. thenar* (*a*) and *m. extensor carpi radialis* (*b*) during painful electrical stimulation of the fingers (*1*), toes (*2*), and ear lobe (*3*).

head (Table 1). NFR evoked in *m. tibialis anterior* by painful stimulation of the ear lobe had shorter latency than NFR evoked in the same muscle by finger or toe stimulation ($p<0.05$). Therefore, homotopic NFR was preceded by the heterosegmentary NFR. This is possible only if NFR is closed in the supraspinal structures of the CNS. Moreover, the appearance of RIII component in the muscles of the upper and lower extremities during painful stimulation of the ear lobe attests to involvement of cerebral structures in the NFR.

It is widely known that the motor system in humans provides three level of the motor control: the spinal cord, the descending systems of the brain stem, and the motor cortex [3,12]. These systems operate both hierarchically and in parallel, and they modulate (directly or indirectly) activity of spinal interneurons and motor neurons [3,12]. Thus organization of the motor system makes it possible to observe NFR in spinal patients [11]. It seems that NFR involves both segmentary and supraspinal systems of the motor control. It is also confirmed by the data on the multilevel morphological and functional structure of the nociceptive and antinociceptive systems controlling pain sensitivity and nocifensive reactions [8]. It is established that activity of nociceptive neurons in the dorsal horns is determined not only by the peripheral input, but also

TABLE 1. Latency (msec) of Inhibitory (I) and Excitatory (E) Phases in RIII Components in *M. thenar* and *M. tibialis anterior* during Heterosegmentary Painful Stimulation ($M \pm m$)

Stimulation Site	*M. thenar*		*M. tibialis anterior*	
	Inh	Ex	Inh	Ex
Ear lobe	68.0±3.2*	106.0±7.8*	84.0±4.4*	136.0±8.5*
Finger	94.0±4.8	148.0±8.4	96.0±6.8	161.0±8.6
Toe	106.0±6.9	181.0±9.2	112.0±7.3	184.0±10.8

Note. *$p<0.05$ compared to finger stimulation.

by the supraspinal cerebral structures inhibiting these neurons via the system of dorsolateral and ventrolateral funiculi [8,10,14]. Therefore, the amplitude-temporal characteristics of NFR depend on activity of the suprasegmentary structures. It was directly confirmed by experiments comparing the peculiarities of NFR changes during long-term pain in intact and spinal rats [10,14]. In contrast to spinal rats, preserved supraspinal mechanism of pain control in intact rats antagonized the increase in NFR amplitude during long-term painful stimulation.

Our data suggest that NFR is not a mere polysynaptic spinal reaction. It is rather a process, which develops under the control of the suprasegmentary nociceptive structures. This approach widens the diagnostic perspectives of NFR recording and makes it possible to use it in clinical practice for objective assessment of the pain syndrome not only during homotopic, but also during heterosegmentary pain stimulation.

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