ACTIVITY OF RESPIRATORY NEURONS IN THE ABSENCE OF RESPIRATORY MOVEMENTS

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After administration of an apnea-producing dose of lysthenon (succinylcholine bromide), spikes continue to appear in neurons of the bulbar respiratory center, but with an increase in the duration of volleys of spikes, number of spikes, mean frequency per volley, and duration of intervals between volleys. The distribution of intervals between spikes in the volley is undisturbed. Artificial respiration quickly shortened the duration of the volleys and reduced the number of spikes per volley. When artificial respiration was stopped, the apneic picture of unit volley activity was repeated.

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After the cessation of respiratory movements, rhythmic volleys of spikes continue to pass from the respiratory center to the diaphragm [2, 10]. Recent investigations have shown that with the arrest of respiratory movements as a result of administration of muscle relaxants, spike activity of certain groups of respiratory neurons remains intact [3, 4-9]. The duration of the volleys and the number of spikes per volley increase considerably under these circumstances.

It has been suggested that these changes are the result of interruption of the flow of afferent impulses from stretch receptors of the lungs and respiratory muscles after cessation of respiratory movements. On this basis, it can be postulated that the forced restoration of respiratory movements (artificial respiration) must restore the normal character of spike activity of the respiratory neurons through resumption of the periodic arrival of afferent impulses as a result of stretching of the lungs and chest wall.

The object of the present investigation was to make a quantitative analysis of the responses of various groups of neurons in the respiratory center (RC) after the cessation and forced resumption of respiratory movements in order to establish the role of periodic afferent impulses in the formation of the respiratory cycle.

EXPERIMENTAL METHOD

Experiments were carried out on cats anesthetized with nembutal (35-40 mg/kg). Spike activity of neurons in the bulbar RC was recorded extracellularly by means of glass microelectrodes (1-3 μ; 2.5 M KCl). Respiratory movments were interrupted for short periods by intravenous injection of lysthenon (0.4-0.6 mg/kg). The duration of volleys of impulses in various phases of respiration, the number of spikes, the mean frequency of spike discharge, the duration of intervals between volleys, and the distribution of intervals between spikes in the volley were analyzed. Activity of 19 neurons (11 inspiratory and 8 expiratory) was recorded.

EXPERIMENTAL RESULTS

After injection of an apnea-producing dose of lysthenon the volleys of impulses continued to appear in respiratory neurons (Figs. 1A and 2B, C), although substantial quantitative changes in activity took place.

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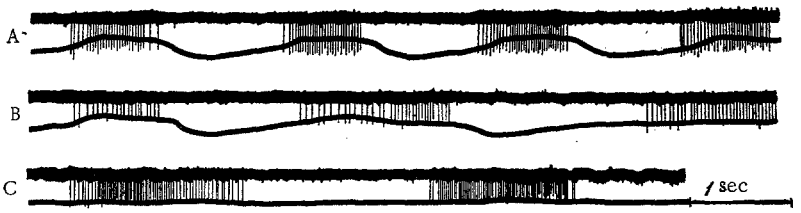

Fig. 1. Changes in activity of inspiratory neuron after abolition of respiratory movements by lysthenon. A) Normal respiration; B) changes in respiration and neuronal activity after injection of lysthenon; C) respiratory movements absent. Top recording shows unit activity, bottom shows respiratory movements (inspiration–expiration).

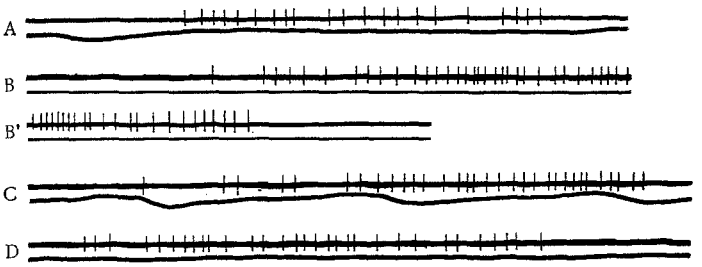

Fig. 2. Changes in activity of expiratory neuron after abolition of respiratory movements by lysthenon and during artificial respiration. A) Normal respiration; B) respiratory movements absent; C) artificial respiration; D) resumption of normal respiration. Time marker 1 sec. Legend as in Fig. 1.

As the action of lysthenon developed, the respiratory cycle was gradually lengthened and the amplitude of respiration decreased until the respiratory movements ceased completely (Fig. 1B). After the cessation of respiratory movements, the number of spikes increased by 1.67 ± 0.71 times, and the duration of the volley of spikes discharged by inspiratory neurons increased by a somewhat smaller degree (by 1.42 ± 0.4 times), as a result of which the mean spike frequency per volley increased very slightly (by 1.19 ± 0.4 times). The interval between volleys of inspiratory neurons was lengthened on the average by 1.21 ± 0.17 times, corresponding to an increase in duration of volleys of the expiratory neurons (by 1.35 ± 0.37 times). During apnea for 40-100 sec, the observed parameters of activity of the inspiratory neurons showed no essential change.

Application of artificial respiration quickly shortened the duration of the volleys and reduced the number of spikes per volley, but these parameters did not reach their initial values (Fig. 2C). After artificial respiration had been stopped, the apneic character of spike activity of the neurons was resumed.

Simultaneously with the arrest of respiration,the number of spikes per volley of the expiratory neurons was increased on the average by 1.55 ± 0.36 times, and the duration of the volleys (by 1.35 ± 0.37 times) and the mean frequency of spikes per volley (by 1.27 ± 0.32 times) also increased. The duration of intervals between volleys showed only a slight increase (by 1.06 ± 0.34 times).

These changes were observed in several groups of neurons differering both in the distribution of spikes in the volley and in the relationship between volleys and the phase of respiration. Characteristically, the typical distribution of spikes in the volley associated with different groups of neurons was not disturbed after the cessation of respiratory movements (Fig. 1C). The original form of activity of the respiratory neurons was completely restored as soon as the action of lysthenon ceased (Fig. 2D).

The results indicate that the ability of respiratory neurons to discharge with regular volleys, and also the character of distribution of spikes in the volleys, are independent of the rhythms of arrival of afferent impulses indicating the degree of stretching of the lungs and respiratory muscles. The neurons retain their

inherent rhythms of volley activity, with the characteristic configuration of spikes, despite the cessation of periodic reflex influences. Consequently, the ability of respiratory neurons to generate rhythmic volleys of spikes is a property of the internal organization of the RC and is independent of the periodic component of the afferent flow, which merely determines the quantitative parameters of volley activity of the respiratory neurons.

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