SEGMENTAL AND SUPRASEGMENTAL PROTECTIVE MECHANISMS AT THE THORACIC LEVEL OF THE SPINAL CORD

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Lesions of the pleura inhibit intercostal reflexes on the ipsilateral side. Inhibition of motoneurons at the thoracic level of the spinal cord is effected through postsynaptic mechanisms. Transection of the spinal cord at the level C7-T1 produces a greater increase in intercostal reflexes on the side of the lesion than on the unaffected side. Consequently, besides segmental inhibitory mechanisms, suprasegmental mechanisms also participate in the protective reflex at the thoracic level.

The object of this investigation was to study the nervous mechanism of the protective respiratory reflex: limitation of respiratory movements of the chest wall on the side of a lesion of the parietal pleura accompanying pleuropneumonia, pleurisy, pneumothorax, or wounds of the chest.

To study the relationship between segmental and suprasegmental mechanisms in this phenomenon, intercostal polysynaptic reflexes were investigated before and after transection of the spinal cord (at the C7-T1 level) in animals with a unilateral pleural lesion.

EXPERIMENTAL

Experiments were carried out on cats weighing 2.5-5 kg. A lesion of the pleura was produced by single or repeated injections of 2-5 ml hot water (80-90°) into the pleural cavity by means of a syringe. Intercostal polysynaptic reflexes were investigated by Downman's method [3]. The central ends of the divided 6th, 7th, or 8th intercostal nerves were stimulated with single square pulses 5-10 times above the threshold strength and 0.5 msec in duration. Responses were recorded from the central ends of divided neighboring intercostal nerves (5th, 6th, or 7th, respectively). For stimulating and recording responses, the central ends of the divided intercostal nerves were placed on bipolar platinum electrodes, with interelectrode distance 0.5 cm. Nerves and electrodes were insulated from the surrounding tissues by means of cellophane packs. The spinal cord was divided at the level C7-T1. The surface of the spinal cord at the point of division was irrigated with 1-2% procaine solution 10-15 min before transection, without opening the dura. To investigate the postsynaptic mechanisms of inhibition, the animals received an intravenous injection of strychnine (0.05-0.5 mg/kg). General anesthesia with chloralose and urethane (40 and 200 mg/kg and 20 and 100 mg/kg, respectively) was used. The animal was placed on an automatically controlled heated table, the surface temperature of which varied from 36 to 40°. Exposed parts of the nerves and tissues were irrigated with warm mineral oil. The areas of the polysynaptic responses, expressed as percentages relative to the initial background, were compared. At each time interval not less than 3-5 single responses were recorded and their arithmetic mean value determined. Each series of the investigation included 10 experiments. The criterion of significance was calculated by Wilcoxon's method.

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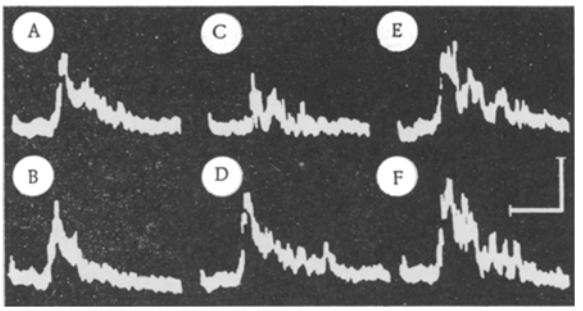

Fig. 1. Polysynaptic reflexes recorded in the 7th intercostal nerve in response to single electrical stimulation of ipsilateral 8th intercostal nerve. A, C, E) Side of injury; B, D, F) intact side; A, B) before unilateral injury to pleura; C, D) after unilateral injury to pleura; E, F) intravenous injection of strychnine nitrate increases reflexes on both sides, but more especially on the injured side (E) compared with amplitude of reflexes immediately before injection of strychnine (C, D); Calibration: voltage 100 μV, time 10 msec.

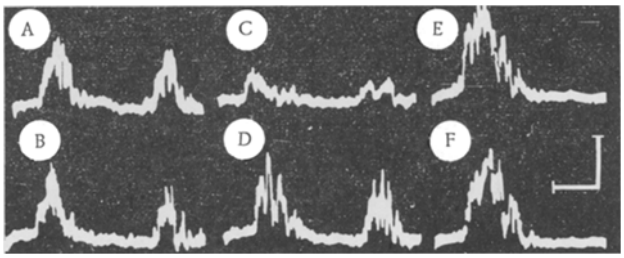

Fig. 2. Polysynaptic reflexes recorded in 7th intercostal nerve in response to single electrical stimulation of ipsilateral 8th intercostal nerve. A, C, E) Affected side; B, D, F) unaffected side; A, B) before unilateral injury of pleura; C, D) 30 min after unilateral injury of pleura; E, F) transection at level C7 increases reflexes from both sides, but more so on affected side (E) compared with amplitude of reflexes immediately before transection of spinal cord (C, D); calibration of voltage 100 μV, of time 10 msec.

EXPERIMENTAL RESULTS

According to data in the literature [3, 7], maximal stimulation of the central end of the divided intercostal nerve produced only polysynaptic responses in the neighboring intercostal nerve. No monosynaptic reflexes were recorded under these conditions. The latent period of the polysynaptic reflexes varied from 4 to 8 msec, their maximal duration usually varied from 15 to 20 msec, and their maximal amplitude from 200 to 1000 μV. The threshold of stimulation largely depended on the depth of anesthesia and varied from 100 to 500 mV. Definite irradiation of these polysynaptic reflexes from one side of the spinal cord to the other was observed. The amplitude of the contralateral responses was 30-40% less, and their latent period 2-2.5 msec longer than those of the ipsilateral polysynaptic reflexes.

In some experiments these polysynaptic reflexes (with a latent period of 4-8 msec) were followed by polysynaptic discharges with a latent period of 25-30 msec. The characteristics of these late reflexes corresponded to late polysynaptic responses at the lumbar level whose centers lie in the mesencephalic reticular formation [1, 6]. Late polysynaptic reflexes were more variable than segmental. Sometimes late polysynaptic reflexes of thoracic segments of the spinal cord appeared in response to the same thresholds of stimulation as segmental polysynaptic reflexes, sometimes with lower thresholds.

Impulses from the affected pleura caused a decrease in amplitude of the segmental intercostal polysynaptic responses evoked by an afferent test discharge on the side of the lesion (on the average by 55.2%, $P < 0.01$), and an increase in amplitude of these responses evoked by a test discharge from the unaffected side (on the average by 19.4%; $P < 0.05$).

To determine the mechanism of inhibition of the intercostal polysynaptic reflexes by a lesion of the pleura, the animals were injected with subconvulsant doses of strychnine, which block inhibitory synapses [5]. Injection of strychnine led to an increase in responses from both sides, but on the side of the lesion (compared with the magnitude of the responses before injection of strychnine) this increase averaged 74.5% ($P < 0.01$) more than the contralateral side (Fig. 1). It thus follows that on the side of the lesion, activity of inhibitory synapses was much greater than on the unaffected side.

As several workers have shown [2-4], high transection of the spinal cord increases intercostal-intercostal reflexes, thus indicating that normally these reflexes are under supraspinal inhibition. A similar increase in amplitude of intercostal reflexes took place after transection of the spinal cord in animals with a lesion of the pleura. The spinal cord was divided at the level C7-T1 between 30 min and 1 h after unilateral injury of the pleura, at a time when the inhibition of polysynaptic reflexes on the side of injury was clearly marked. Immediately after transection of the spinal cord the spino-bulbo-spinal reflexes disappeared, segmental polysynaptic responses were sharply increased on both sides, but on the side of injury this increase, compared with the amplitude of the responses immediately before transection, was much greater (Fig. 2) than on the contralateral side (on the average by 125%, $P < 0.01$). It follows from these results that, besides segmental mechanisms of inhibition, supraspinal inhibitory mechanisms also participate in the organization of the protective respiratory reflex.

The difference between the degree of increase in polysynaptic reflexes on the two sides was reduced 20-30 min after transection of the spinal cord, and stimulation of the central ends of the divided intercostal nerves produced inhibition of intercostal polysynaptic responses both on the side of the lesion and on the unaffected side. This is a very interesting fact, because it shows that the weakening of respiratory movements of the chest is confined to the side of lesion because of descending influences of the central nervous system. Because of the physiological features distinguishing the thoracic division of the spinal cord, it could easily spread also to the healthy side.

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