REFLEX CHANGES IN CIRCULATION DURING STIMULATION OF DEFLATION RECEPTORS OF THE LUNGS

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Aspiration of air through the intubation tube from the diaphragmatic lobe of the lung in anesthetized dogs evoked reflex bradycardia and arterial hypotension. The reaction was associated with stimulation of specific deflation receptors in the lung parenchyma and its pathway lay in the vagus nerves.

The character of the cardiovascular response to adequate stimulation of the specific deflation receptors of the lungs is unknown [11]. Some workers consider that this form of stimulation affects only respiration and not the circulation [8]. The writer's previous investigations suggest that in fact certain changes are observed in the circulation under these circumstances [1].

The object of the present investigation was to study this problem more closely.

EXPERIMENTAL METHOD

Dogs (36) were anesthetized with morphine (1-4 mg/kg) and urethane (0.7-1 g/kg). Adequate stimulation of the deflation receptors of the lungs was produced by aspirating air from one (diaphragmatic) lobe of the lung through the intubation tube, which was inserted deep into the bronchus of the tracheotomized animal [5]. Sometimes the stretch receptors of the lungs were stimulated by injecting air through the same intubation tube [2] for comparison. The degree of deflation or stretching of the lobe of the lung was judged from the intensity of the changes in intrapulmonary pressure (in mm Hg).

The lung receptors were paralyzed by production of an intrapulmonary steam burn [3, 7]. The intubation tube was connected to a flask in the bottom of which water was boiled.

Respiration (pneumograph), arterial pressure (mercury manometer), and the heart rate (Fleisch's intervalograph [9]), and sometimes also the venous and intrathoracic pressures (water manometer), were recorded on a smoked drum.

EXPERIMENTAL RESULTS

Deflation of one lobe of the lung (DOLL) evoked generalized arterial hypotension, bradycardia, and bradypnea. The venous and intrathoracic pressures changed only slightly or not at all (Fig. 1). The response was weak especially as regards changes in the arterial pressure. Cardiac changes were somewhat more marked, and were sometimes considerable (Figs. 1 and 2). With an increase in the degree of DOLL, the response became stronger (Fig. 2). In four dogs, slight quickening of the heart rate was observed, with no changes in the arterial pressure, in response to moderate DOLL. However, with an increase in DOLL the typical depressor response appeared in these animals also. In nine dogs, no response whatever occurred to DOLL. This may have been due to increased sensitivity to the anesthesia because after deliberate deepening of the anesthesia with barbiturates, the response to DOLL was always abolished. However, despite the specific differences between the responses described, the typical depressor response to DOLL on the whole was statistically significant (Fig. 2).

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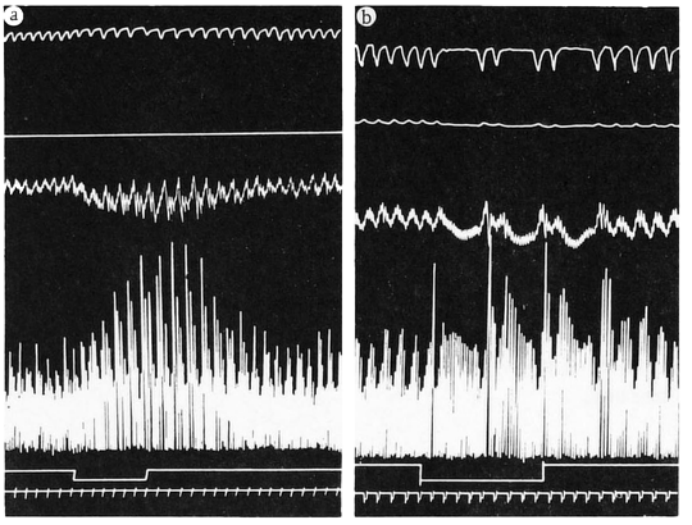

Fig. 1. Response of cardiovascular system to decrease of 20 mm Hg in pressure in diaphragmatic lobe of right lung (a and b denote different types of response). From top to bottom: respiration, venous pressure, arterial pressure, heart rate (each vertical line corresponds to time interval between two heart beats; the longer the lines the slower the heart rate, and vice versa), marker of stimulation and time marker (3 sec).

The absence of specific changes in pressure in the large veins or in the thorax in response to DOLL is indirect evidence that the blood flow in the venae cavae and in the pulmonary circulation was unchanged. It was therefore suggested that the depressor response arises through a reflex from the lung parenchyma, in the same way as the corresponding respiratory response [5]. This suggestion was confirmed by the fact that the response was easily blocked by transient burning of the investigated lobe of the lung.

Afferent fibers of the corresponding reflex arc evidently traveled in the vagus nerves, for the response disappeared completely after cold blocking (9-10°) of these nerves and after bilaterial vagotomy. The cardiac response disappeared after injection of atropine, but the fall of arterial pressure remained. Consequently, it can be postulated that efferent fibers to the heart run in the vagus nerves, and those to the vessels run in the sympathetic vasomotor nerves.

Stretching of one lobe of the lung (SOLL) also evokes a reflex depressor response and inhibition of respiration [2]. Possibly, therefore, under DOLL conditions, compensatory stretching of the opposite lung develops and this may be the cause of the reflex. However, this hypothesis was not confirmed experimentally. Contralateral vagotomy, for example, neither blocked nor weakened the response but, on the contrary, strengthened it. After ipsilateral vagotomy the response either ceased completely or became considerably weaker. The effect of ipsilateral burning of the lobe of the lung, described above, agreed with these results.

The possibility is not ruled out that during DOLL stretch receptors of the investigated lung also are stimulated and that this is the cause of the reflex. However, tests showed that the depressor responses to DOLL and SOLL were most probably due to completely different mechanisms. For example, the response to DOLL usually increased gradually in strength during the period of stimulation, and this effect continued for a short time after stimulation also (Fig. 1). During SOLL no such effect was observed. Repetition of DOLL at short time intervals (0.5-1 min) usually caused gradual weakening and, eventually, total cessation of the response. The response recovered after 5-10 min. Repetition of SOLL had no effect on the character of the response. Following application of a cold block (9-10°) to the vagus nerves the response only to DOLL was completely abolished, while that to SOLL remained unchanged. The same effect was obtained by burning the investigated lobe of the lung: the response to DOLL was blocked while that to SOLL remained.

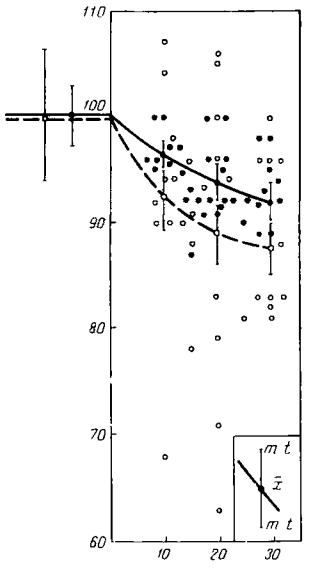

Fig. 2. Relationship between response of cardiovascular system to degree of deflation of diaphragmatic lobe of lung. Arithmetic mean data: continuous line shows arterial pressure, broken line shows heart rate. Results of individual experiments: filled circles denote arterial pressure, unfilled circles denote heart rate. Abscissa, decrease in intralobar pressure (in mm Hg); ordinate, changes in indices (in percent of initial value).

It can therefore be concluded that the depressor response to DOLL is evoked by stimulation of specific deflation receptors of the lungs. They are evidently located in the lung parenchyma because no significant disturbances of the blood flow in the pulmonary circulation were observed during DOLL, and the corresponding response was easily blocked by an intrapulmonary burn.

Slowing of the heart beat during deflation of the lungs, when produced by stopping artificial respiration in curarized dogs, has also been observed by Nikiforovskii [6], and during clinical stimulation of deflation receptors of the lungs by Paintal [10]. In both these experiments the bradycardia was possibly due to the same reflex as that described in the present paper. However, as reported elsewhere [4], the writer discovered that bradycardia arising after stopping artificial respiration in curarized dogs developed mainly through stimulation of sino-aortic receptors [4]. According to Widdicombe [11], it has not yet been finally established that Paintal was in fact studying specific deflation receptors of the lungs.

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