THE DUAL NATURE OF RESPIRATORY JAW REFLEX

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Opening of the mouth sets up two extra-pulmonary respiratory reflexes. The first consists of an increase in amplitude of breathing, operative under conditions requiring greater respiratory activity; and the second consists of an increase in the rate of breathing, operative under conditions of raised body temperature.

Mathur and Chowdhuri (1952) described a reflex mechanism, consisting of an increase in respiration rate on opening the mouth, operative in dogs only when their body temperature is raised. This was then confirmed by Kumar (1953) who stated that in majority of the dogs this mechanism starts operating between 40 - 41°C body temperature, and then quickly disappears beyond 42°C. Kumar and Sinha (1961) again confirmed these findings and further observed that as the respiration rate increases on opening the mouth its amplitude is also reduced. They named this reflex on this account as the 'accelerator' reflex. Sharma and Sharma (1958) on the other hand observed an increase in respiration rate on opening the mouth in dogs without increasing their body temperature, but whose respiratory activity was increased by inducing hydrothorax or by inhaling 7 per cent carbon dioxide and oxygen mixture. They argued that the factor conditioning the appearance of this reflex is the greater demand of respiratory activity rather than the rise in body temperature beyond a critical level. It was thought desirable, therefore, to further investigate the nature of this reflex.

METHODS

These experiments were done on 21 healthy mongrel dogs weighing between 9·0 - 12·5 kg. and anaesthetised with intravenous chloralose (80 mg/kg). They were divided into two groups. In the first, the body temparature was kept constant and the respiration rate was increased (i) by producing pulmonary embolism in 5 dogs by intravenous injection of mashed potatoe; and (ii) by producing unilateral hydrothorax in 11 dogs by slow intrapleural injection of warm normal saline through a primary artificial pneumothorax needle. Their respiration was recorded by introducing and inflating a toy balloon in between the diaphragm and the liver. In the second group of 5 dogs, the degree of pulmonary ventilation was kept constant by giving artifi-

cial respiration at a fixed rate and tidal volume. Their chest was opened by removing portions of third to seventh ribs bilaterally in such a way that a mid line thoraco-abdominal window was made and through this the movements of the diaphragm were recorded. The degree of pulmonary ventilation was so adjusted that spontaneous diaphragmatic movements just appeared. The body temperature of these animals was raised by applying radiant heat from all round the body and covering the animal in a jacket. Deep rectal temperature was recorded in all dogs by a Centigrade thermometer.

RESULTS

The initial rectal temperature of the dogs subjected to pulmonary embolism varied between 37—38°C, and their rate of respiration varied between 10—21 per min. On opening their mouth no change in the rate or depth of respiration was found to occur. On intravenous injection of mashed potatoe, though the body temperature remained unaltered, the respiration rate increased several folds, and on opening the mouth any further acceleration of respiration rate was absent (Fig. 1).

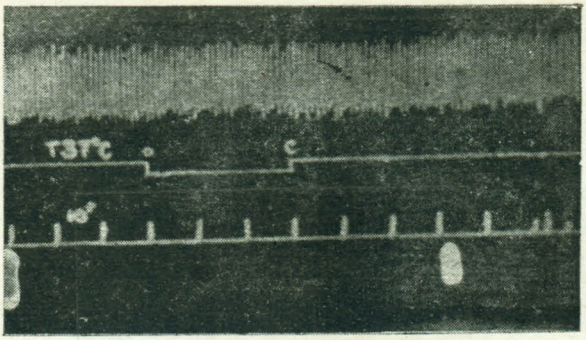

Fig. 1. The initial rate of respiration was 18/min. It increased to 54/min on producing pulmonary embolism.

In the dogs subjected to artificial pneumothorax the initial rectal temperature varied between 36·2—38·5°C, and their initial respiration rate ranged between 8—21 per min. Initially, on opening the mouth no further acceleration in respiration rate occured in these dogs also. As normal saline was injected into the pleural cavity the rate of respiration gradually increased. On opening the mouth at this stage although no increase in rate occured, but there occured a remarkable increase in the amplitude of breathing (Fig. 2) which returned back to the original depth on closing the month.

In 4 dogs after the above mentioned observations were confirmed the body temperature was raised and it was observed that at raised body tempe-

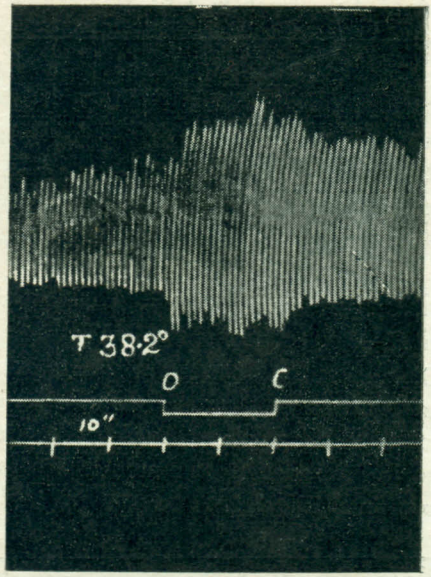

Fig. 2.

The initial respiration rate of 12/min increased to 54/min. On opening the mouth (O) the amplitude of respiration increased although the rate remained unaltered.

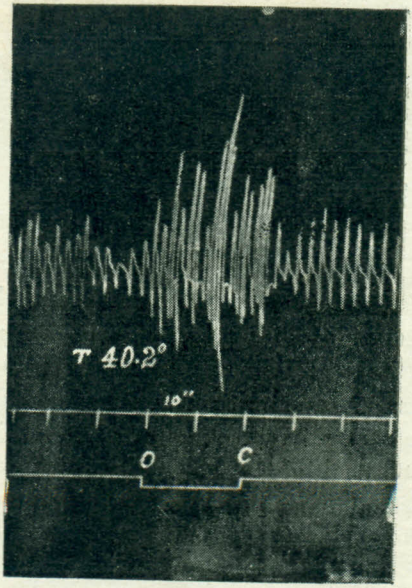

Fig. 3

rature on opening the mouth in addition to an increase in depth there also occurred an increase in rate of respiration. The depth of respiration however, became irregular (Fig. 3).

The initial body temperature and the rate of diaphragmatic excursions in dogs ventilated at a uniform rate, varied between 36·4—38·0°C and 12—28 per min respectively. Any further change in rate or depth of respiration was not observed on opening the mouth in any of these dogs at this range of temperature (Fig. 4). When their body temperature was raised the rate of

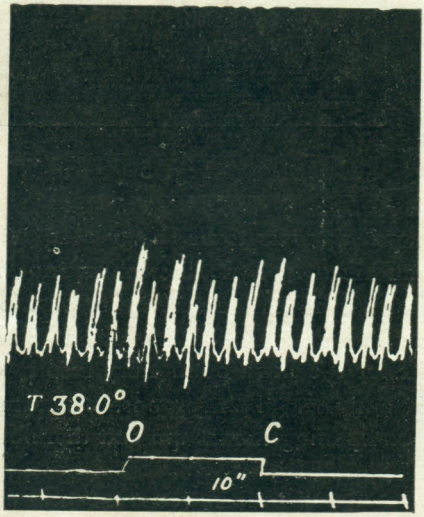

Fig. 4

respiration remarkably increased, and it further increased on opening the mouth (Fig. 5). There was, however, no change in the amplitude of breathing. As the temperature of the animal approximated 42°C the respiration rate reached to its maximum and no further acceleration occured on opening the mouth. On further application of heat, although the body temperature did not rise beyond 42°C, but the closed mouth rate of respiration decreased, and the reflex acceleration in respiration on opening the mouth reappeared.

DISCUSSION

The results of the present study point to opening of the mouth acting as a trigger for two different reflex mechanisms affecting the respiration of the dogs. The first of these results in increase in amplitude of breathing, and the second resulting in an increase in the rate of breathing. The factor condi-

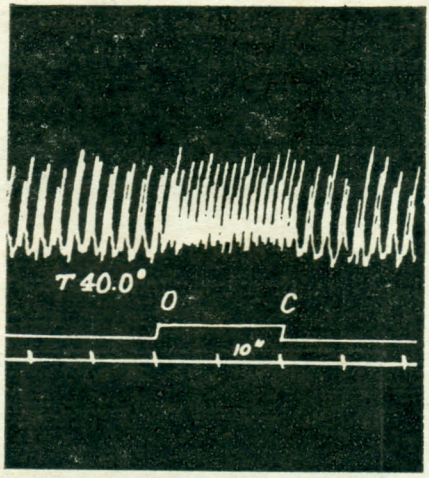

Fig. 5

tioning the first response appears to be the greater respiratory needs of the animal which are produced by hydrothorax but not by pulmonary embolism though both of them result in an increase in respiration rate. It, therefore, does not confirm the observations of Sharma and Sharma (1958) who stated that the pre-requisite for the extra-pulmonary respiratory jaw reflex is an increase in respiratory activity. This response which results in an increase in depth of breathing on opening the mouth can therefore be called the 'augmentator' reflex.

When the rate of pulmonary ventilation is kept constant and the body temperature raised, the rate of spontaneous diaphragmatic excursions, which indicates the rate of respiration, gradually increases. This further increases on opening the mouth and returns back to the original on closing it, provided the body temperature is between 40—42°C. Since the pulmonary ventilation was kept constant, this response appears to be affected by the degree of body temperature alone. Kumar and Sinha (1961) reported that opening of the mouth under similar conditions in intact dogs results in an increase in the rate and a decrease in the amplitude of breathing. This type of shallow breathing on opening the mouth, they argued, results in a greater to and fro movement of the dead space air and a consequent better heat loss, without necessarily affecting the alveolar air. In the present study although opening of the mouth resulted in an increase in rate of respiration, but the depth remained almost unaffected. This is because in a dog being artificially ventilated the degree of pulmonary ventilation has perhaps to be slightly less to

produce spontaneous diaphragmatic excursions. This would, therefore, set up both the 'augmentator' and the 'accelerator' reflexes resulting in an increase in rate of respiration without affecting its depth. It further stands to reason that in dogs at raised body temperatures there exist two mechanisms for heat loss. The first is an increase in respiration rate, and the second is a further reflex acceleration in rate of respiration on opening the mouth. As the body temperature approaches 42°C the rate of respiration reaches to its maximum and no further increase occurs on opening the mouth. With the further application of heat the temperature regulating mechanism starts failing and the rate of respiration falls below the maximum. Under such conditions the accelerator reflex reappears. When the temperature regulating mechanism starts failing the reflex mechanism of heat loss though last to appear is also the last to disappear.

When hydrothorax was produced and the body temperature raised, i.e., both the above mentioned conditioning factors were present, opening of the mouth sets in both the 'augmentator' and the 'accelerator' reflexes. This, therefore, results in an irregular respiration.

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