VAGOTONIC ACTION OF CARBON DIOXIDE

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In milder concentrations carbon dioxide is well known to be a stimulant of the heart and respiration. It was shown by Mathur (2) that the hearts of apparently dead *Ciona intestinalis* could be revived if carbon dioxide was blown into the sea water bathing them. Young *et al* (3) have shown that dogs who are made hypercarbic by making them breathe a mixture of 20% CO_2 and O_2 show an increase in the effects of vagal stimulation on the heart whereas in hypoxic dogs the action of vagus is markedly decreased. Kumar and Srivastava (1) reported that when dogs breathing 20% CO_2, and O_2 mixture developed apnoea and asystole of the heart, and 3 or 4 min. after the development of apnoea, a few gasps appeared shortly and then the heart beat reappeared for some time before the animal finally died. An endeavour has been made to study the mechanism of this terminal reappearance of the respiration and heart beat during hypercarbia.

MATERIALS AND METHODS

Healthy mongrel dogs were obtained from the city Corporation and they were anaesthetised with pentobarbital sodium given in a dose of 30-40 *mg!kg* of the body weight, dissolved in 50 *ml* of normal saline and injected intravenously, slowly. Femoral blood pressure and diaphragmatic respiration from a balloon in between the liver and diaphragm were recorded. Electrocardiogram was intermittently taken in Lead II.

In the first series of five dogs hypercarbia was started by making them breathe a mixture of 20% of CO_2 and 80% O_2 by the Boyle's anaesthesia machine. The expiratory valve was kept open to avoid rebreathing.

In the second series, five dogs were completely atropinised by giving atropine sulphate intravenously at the rate of 0.1 *mg!kg* body weight, before exposing them to hypercarbia. In the third set of another five dogs the region of carotid bifurcation was denervated by dissecting out the adventitia of the common carotid artery and its branches for about 2 cm on either side of bifurcation, and then painting the region with 80 percent phenol in water. These dogs too were exposed to hypercarbia in the similar manner.

RESULTS

In the first series an apnoea occurred on an average 12.30 min. after the start of hypercarbia which was followed by a fall of blood pressure to zero and then an asystole of the heart occur-

red about 3-4 min. after the apnoea. After about 40 seconds to 1 minute of asystole there was the appearance of a few gasps (Fig. 1) in all the dogs followed by the reappearance of the heart beat which was ineffective in raising the blood pressure. The rhythm of the heart was sinus in each case. The heart finally stopped on an average about 10 minutes of the appearance of the gasps due to severe bradycardia. These observations confirmed the results reported earlier (1).

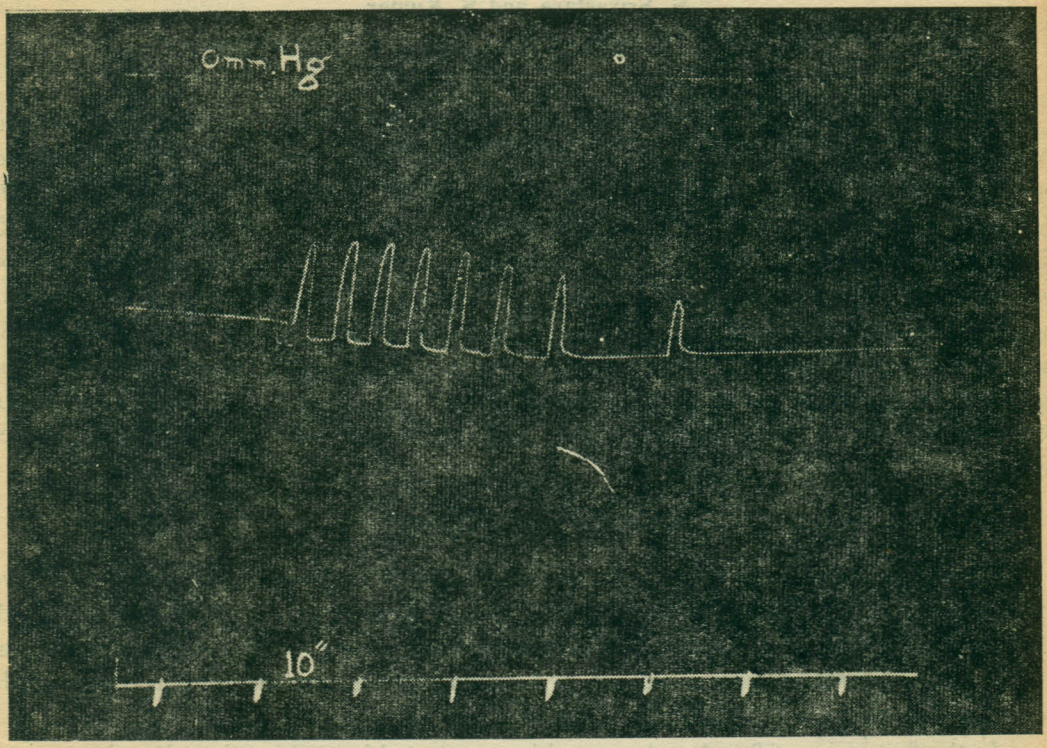

Fig. 1

Dog, showing the appearance of terminal gasps of respiration. The blood pressure is zero throughout.

In the second series of dogs which were atropinised and then made hypercarbic, an apnoea occurred about 20 min. after the start of hypercarbia, and about 12 min. after the apnoea, gasps similar to the first series appeared though a temporary asystole had not occurred and the heart was showing a normal sinus rhythm (Fig. 2). Final stoppage of the heart occurred about 20 min. of the apnoea due to a progressive bradycardia but the rhythm of the heart was sinus upto the end except in the very terminal stage.

In the third series of carotid denervated dogs, findings similar to those of the atropinised dogs were observed except that the heart showed some irregularities (Fig. 3) before the heart finally stopped.

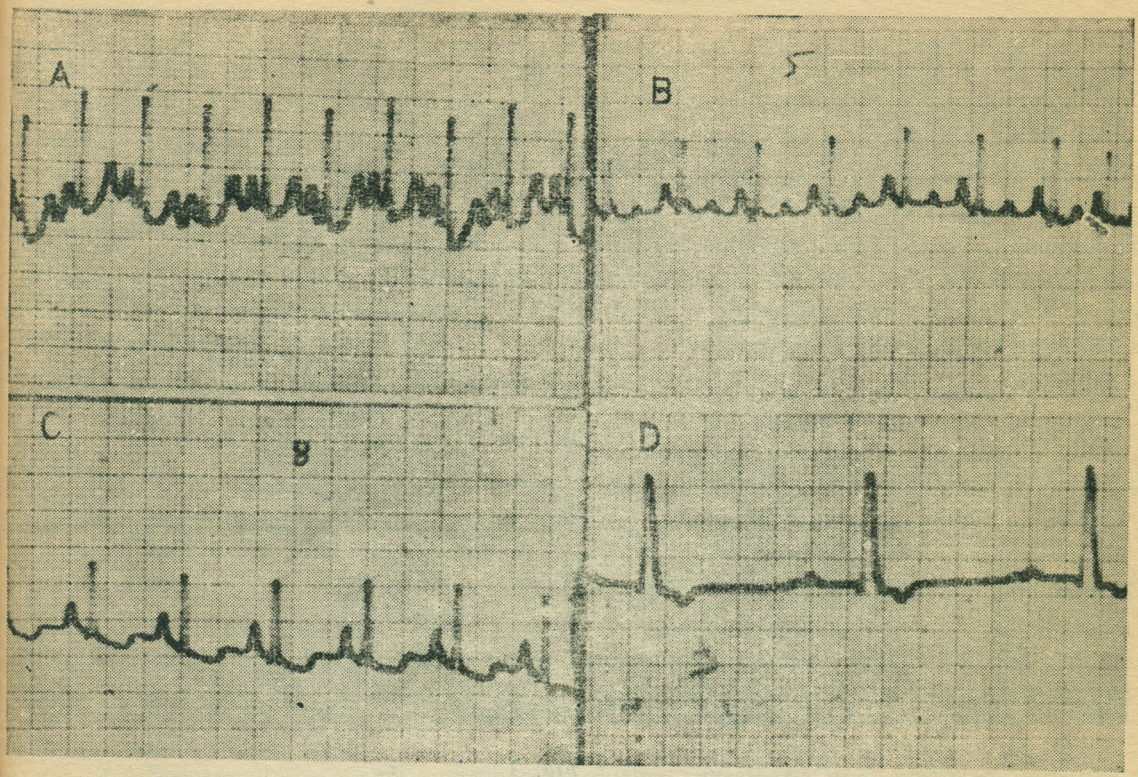

Fig. 2

Dog completely atropinised. ECG Lead II (A) under resting, and (B), (C) and (D) under progressive hypercarbia. Note gradual lengthening of PR interval and inversion of T wave, with a regular sinus rhythm throughout.

DISCUSSION

The appearance of respiratory gasps 3-4 minutes after the appearance of apnoea suggests that carbondioxide acts once again as a respiratory stimulant before it finally becomes a protoplasmic poison. The fact that heart beats reappeared just after the appearance of respiratory gasps is suggestive of irradiation of inhibitory impulses from the respiratory centre to the vagal centre. This is comparable to the irradiation of impulses from the respiratory to the cardiac centre resulting in an increase in heart rate during inspiration in cases showing sinus arrythmia.

Dogs exposed to hypercarbia after preliminary atropinisation did not show a temporary asystole although apnoea developed and was followed by respiratory gasps. This confirmed the suggestion that the temporary asystole was due to an increase in vagal tone and not to a peripheral action on the myocardium. That the origin of this enhanced vagal tone was from the baroreceptors was shown in the third series of dogs in whom bilateral denervation of the region

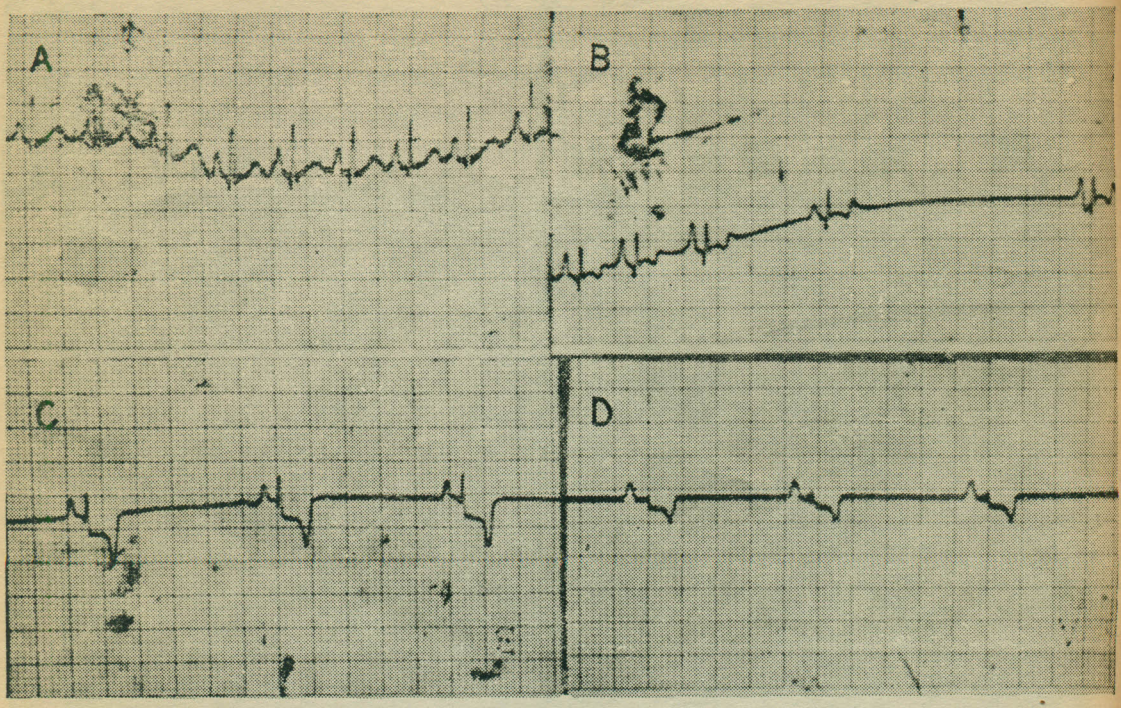

Fig. 3

Dog, bilateral carotid denervation, ECG Lead II (A) under resting and (B), (C) & (D) under progressive hypercarbia. Note inversion of T wave, slight lengthening of PR interval and depression in ST segment, with sinus rhythm.

of the carotid bifurcation was done. In this series of dogs also the period of asystole before the terminal gasps was not observed because the origin of vagal tone was abolished. Whether carbon dioxide sensitized the baroreceptors only, or the carotid chemoreceptors too had a role to play could not, however, be ascertained as selective denervation of the chemoreceptors was not done. Occasional irregularities in the cardiac rhythm that were now observed in this series could be due to reflexes from the aortic region which was intact.

Young *et al* (3) observed vagal inhibition of the heart to be more effective in dogs breathing 20% carbon dioxide. The present study is suggestive of an increase in vagal tone under similar conditions of hypercarbia. This would explain the bradycardia preceding cardiac arrest observed by Ziegler (4) in cases of congenital heart disease under going surgical correction. Hypercarbia frequently occurs during open chest surgery and if during surgical manouvers vagal stimulation also occurs then cardiac arrest may be precipitated. Complete atropinization of the individual shall protect the heart both against the enhanced vagal tone as also from any accidental vagal stimulation.

SUMMARY

The terminal action of 20% CO_2 inhalation in dogs is the development of a sudden apnoea followed 2-4 minutes later by a temporary asystole of the heart. After forty seconds to a minute of this asystole, there is an appearance of a few gasps followed by a reappearance of the heart beat which is ineffectives in raising the blood pressure. In completely atropinised dogs or in dogs with bilateral carotid denervation no such temporary asystole was observed although gasps of respiration appeared. This temporary asystole appears to be a vagotonic action of CO_2 the mechanism of which is probably reflex. Reappearance of heart beat a little after the appearance of gasps is suggestive of irradiation of impulses from the respiratory centre to the vagal centre, thereby reducing the vagal tone.

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