

EFFECT OF CAROTID BARORECEPTOR STIMULATION ON CARDIAC ARREST IN HYPOXIC DOGS

SURENDRA KUMAR, SHOBHA SRIVASTAVA, K.K. PANDEY AND D.N. KHANNA

*Department of Physiology,
King George's Medical College, Lucknow*

Summary: Complete denervation of the carotid region on the left side and chemical glomectomy on the right side was performed in healthy mongrel dogs anaesthetised with pentobarbitone. The carotid sinus of the right side was perfused and stimulated by the animals own blood. During mild and moderate hypoxia the baroreceptor stimulation produced bradycardia and hypotension as in normal air breathing animals but during severe hypoxia the response was tachycardia and decrease in hypotension. In one of the dogs ventricular fibrillation occurred when carotid sinus was stimulated during severe hypoxic stress which was followed by respiratory arrest.

These results suggest that either the response from the carotid sinus is altered during extreme hypoxia or that two types of afferents arise from the cartotid sinus one remaining effective till, moderate hypoxia and the other effective in severe hypoxia.

Key words: pressoreceptors carotid sinus hypoxia

INTRODUCTION

Wylie (8) in 1956 suggested that sudden cardiac failure during surgical anaesthesia could be of a reflex origin, and that hypoxia, hypercarbia and direct interference of the heart and great vessels were important underlying causes. However it has been reported that hypoxia per se does cause sudden cardiac failure but only produces marked cardiac irregularities (4, 6). Earlier Sloan (7) had suggested that vagal stimulation at the hilum of the lung under hypoxia could lead to dangerous inhibition of the heart. Vagal tone is said to originate largely from the sino-aortic baroreceptors (3). It is likely therefore that the fatal inhibition of the heart could be of baroreceptor origin under hypoxic stress. In this paper, we report the effect of carotid sinus baroreceptor stimulation under progressive hypoxic stress.

MATERIALS AND METHODS

Twenty three healthy mongrel dogs weighing between 8 and 16 kg were anaesthetised with i.v. pentobarbitone sodium (35 mg/kg). Femoral blood pressure, and respiration from the tracheal cannula were recorded. Hypoxia was produced by letting the animal rebreathe air from a 7 litre spirometer equipped with a soda lime chamber.

Carotid Sinus Preparation: The left side carotid bifurcation region was completely denervated by dissecting out the adventitia for about 2 cm on either side of the bifurcation and then painting it with 70% phenol in normal saline. All branches of the right common and external carotid arteries were ligated as far away from the carotid sinus as possible. The

carotid chemoreceptors on this side were destroyed by the method recommended by Bevan *et al.* (1). Glomectomy was considered to be complete when respiratory response to intra-carotid injection of 1 ml of 1% sodium cyanide disappeared. The animals were heparinised by 1 mg/kg i.v. heparin. A T-cannula was put into the right common carotid artery whose stem was connected to a 50 ml glass reservoir connected to a pump. Clamping the carotid artery distal to the cannula filled the reservoir with blood which could then be pumped under pressure into the carotid bifurcation region after clamping the carotid proximal to the cannula. In this way, pressure in the carotid sinus could be raised to 200 mm Hg while perfusing it with the blood at an oxygen level prevailing in the animal itself.

Effects of carotid sinus stimulation on arterial pressure, ECG and respiration were recorded before hypoxia and under graded hypoxic stress. Hypoxia was considered mild when the blood pressure started rising, moderate when the elevated blood pressure started falling and severe when the fall in blood pressure continued and apnoea developed. Simultaneous blood samples from the femoral artery were collected under liquid paraffin to measure the oxygen content and % saturation of haemoglobin using the techniques described earlier (5).

RESULTS

In animals breathing room air O_2 saturation of haemoglobin was $94 \pm 13\%$. Carotid sinus stimulation always resulted in a significant hypotension ($P < 0.01$) and bradycardia occurred in 65% of the dogs. ECG tracings did not show any change.

TABLE I : Showing the data obtained on stimulation of carotid sinus during different grades of hypoxic stress.

	Blood pressure (mmHg)		Heart rate min.		Change in heart rate on C.S.S. (% of dogs)			Change in % saturation of Hb. on C.S.S. (% of dogs)		
	Before C.S.S.	During C.S.S.	Before C.S.S.	During C.S.S.	No change.	Increase.	Decrease.	Regular	Irregular	Mean
Normal air breathing	132 ± 11	102 ± 18	198 ± 23	182 ± 11	35	0	65	100	0	94 ± 13
Mild hypoxia	149 ± 17	119 ± 20	196 ± 36	186 ± 32	33	11	56	95	5	63 ± 12
Moderate hypoxia	125 ± 19	109 ± 24	196 ± 48	162 ± 44	41	14	45	77	23	32 ± 10
Severe hypoxia	102 ± 23	94 ± 27	91 ± 39	146 ± 65	32	32	36	50	50	19 ± 14

C.S.S. = Carotid sinus stimulation.

In mildly hypoxic animals when blood oxygen was 63% the ECG remained normal except in one dog which showed a sinus irregularity. Carotid sinus stimulation again resulted in a significant hypotension ($P < 0.01$). Bradycardia was observed in 56% and tachycardia in 11% of the dogs. No ECG change was observed on baroreceptor stimulation.

In moderate hypoxia as the blood oxygen fell to 32% cardiac irregularities appeared in 23% of the dogs. On carotid sinus stimulation a hypotension resulted which was still significant ($P \leq 0.05$). Tachycardia was now observed in 14%. The ECG changes of hypoxia were not altered by baroreceptor stimulation.

In severe hypoxia when the % saturation of Hb fell to 19%, ECG changes by way of irregular sinus rhythm, disappearance of P waves, partial heart block and ST segment deflections appeared in 50% of the dogs. In one dog ventricular fibrillation was observed. On carotid sinus stimulation hypotension was observed, but it was insignificant ($P > 0.05$). In one dog the response was actually reversed to a hypertensive one (Fig. 3). Bradycardia was observed in 36% and tachycardia (Fig. 1) in 32% of the dogs. This tachycardia was now significant

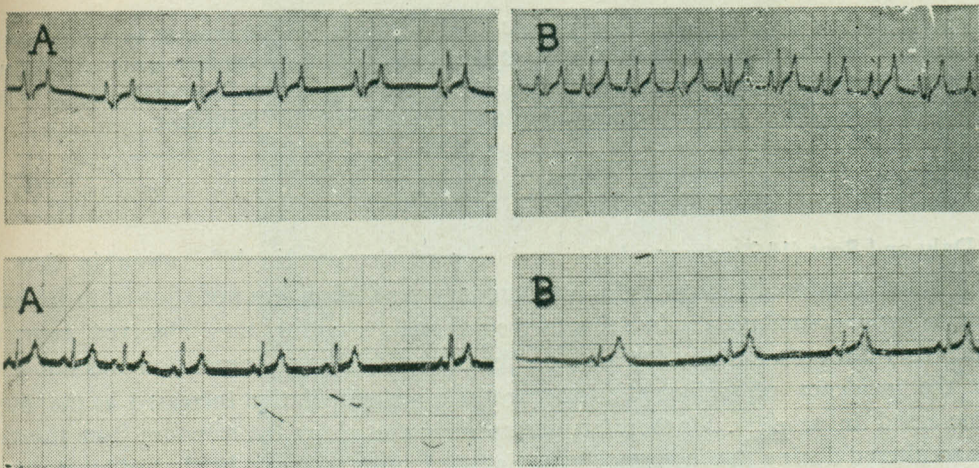


Fig. 1: Upper record: Dog 18; Severe hypoxia, lead II electrocardiogram: A just before, and B, during carotid sinus stimulation. Note an increase in heart rate from 83 to 150 per minute.

Lower record: Dog 8; Severe hypoxia, lead II electrocardiogram. A, just before, and B, during carotid sinus stimulation. Note that irregular and rapid sinus rhythm becomes regular and slow.

($P < 0.01$). On baroreceptor stimulation, in two of the dogs in whom irregular sinus rhythm had set in, the rhythm became regular (Fig. 1) and in one of the dogs where the P waves had disappeared, they suddenly reappeared in large numbers though they were ineffective in exciting the ventricles. In the dog which developed ventricular fibrillation the respiration continued for about a minute after cardiac arrest (Fig. 2).

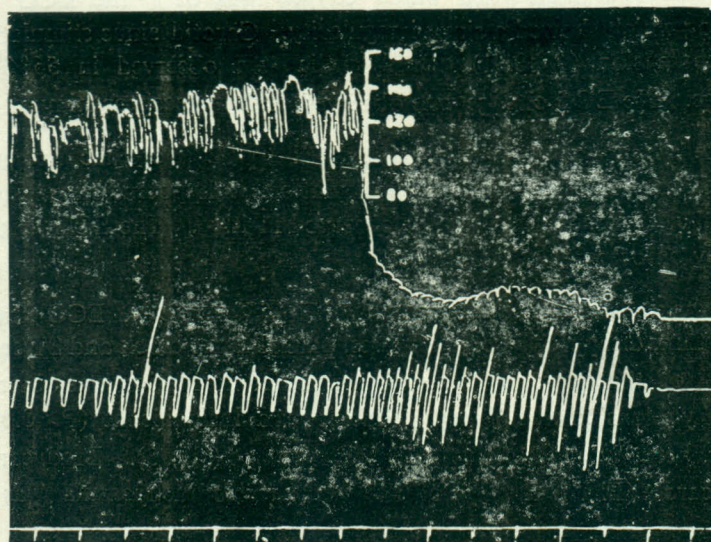


Fig. 2: Dog 13; Severe hypoxia. From below upwards time markings every 10 seconds, respiration and blood pressure. Note that after sudden fall of blood pressure to zero the respiration continues for about one minute. The small fluctuations in blood pressure observed terminally are artefacts produced by the respiratory lever. The ECG taken during this period showed ventricular fibrillation.

DISCUSSION

Daly and Scot (1962) observed that perfusion of the carotid bifurcation in dogs with hypoxic blood produced bradycardia if pulmonary ventilation was not allowed to increase in response to hypoxic excitation. In a few of their animals however, they observed tachycardia for which they did not offer any explanation. In this study though respiration was not controlled, yet the incidence of tachycardia on carotid sinus stimulation increased as the hypoxic stress increased in severity. It therefore seems likely that either the response from the carotid sinus is so altered during the extreme hypoxia that instead of bradycardia, it produces tachycardia, or that two types of afferents arise from the carotid sinus one remaining effective upto mild or at the most moderate hypoxia which on stimulation produces bradycardia, and the other which are excited during extreme hypoxia and produce tachycardia. The latter explanation seems to be plausible because during severe hypoxia when irregular cardiac rhythm had set in, stimulation of the carotid sinus changed the irregular sinus rhythm into regular one in two dogs and in one it produced a barrage of P waves which, of course, were ineffective in exciting the ventricle possibly due to a severe A-V block.

In the present study hypoxia was allowed to progress till death occurred. Obviously such a degree of hypoxic stress would effect the heart and blood vessels as well as the central

nervous system. Whether the change in baroreceptor sensitivity is due to the baroreceptor themselves or secondary to the changes in the above mentioned systems needs to be explored. In all except one dog respiratory failure occurred earlier than the cardiac failure. Similar mode of death in intact anaesthetised dogs exposed to hypoxia has been reported earlier (4). In one dog the ventricle first went into fibrillation and the respiration continued for one minute before it also stopped (Fig. 2). The primary cardiac arrest as the mode of death is recognised by the anaesthetists during surgical practice. Kumar and Srivastava (5) observed similar course of death in coronary artery ligated dogs showing evidence of myocardial infarction when exposed to hypoxia. This could be due to deprivation of some type of tonic influence from the carotid chemoreceptors, or due to an aberrant reflex hypoxic excitation from the carotid baroreceptors. Although the former reason cannot be ruled out, the latter appears more probable because in one of the dogs carotid sinus stimulation during severe hypoxia reversed the usual hypotensive response to a hypertensive one.

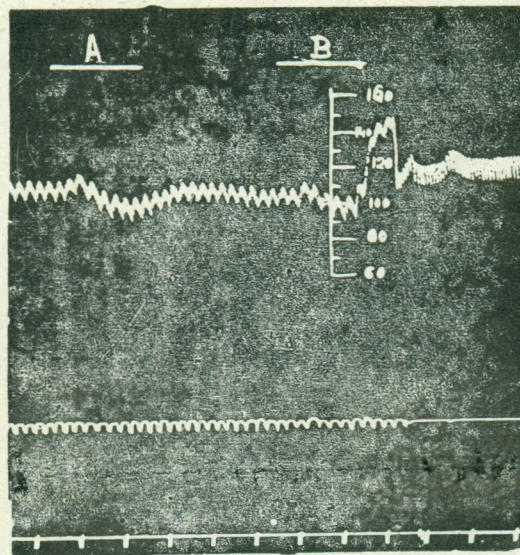


Fig. 3: Dog 9; Severe hypoxia. From below upwards time marking every 10 seconds, respiration and blood pressure. Note that carotid sinus stimulation at A produced a hypotensive response, but soon afterwards when apnoea set in, sinus stimulation produced a hypertensive response, B.

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