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EFFECTS OF ACUTE HYPOXIA ON HAEMODYNAMICS IN DOGS

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Background: Almost 50 years ago the proposition that acute hypoxia elicits pulmonary vasoconstriction was made. By now methods have been standardised for exploring the pulmonary and systemic circulation in a systematic & sophisticated way and the introduction of cardiac catheterization had brought the previous remote pulmonary circulation within reach. ***Methods:*** This study was designed to see the effects of acute hypoxia on hemodynamics with the advanced, sophisticated equipment and standardised method. The effects of acute 10 minutes hypoxia were studied on hemodynamics in anaesthetized dogs. ***Results:*** Acute hypoxia increased the Cardiac Output from 1.76 ± 0.43 to 1.91 ± 0.52 L/min, HR from 240 ± 55.58 to 260 ± 63.34 beat/min, Ppa from 2.24 ± 0.61 to 3.39 ± 1.09 kPa, PVR from 9.49 ± 3.33 to 14.30 ± 5.29 kPa . s/L, Psa from 14.53 ± 1.16 to 15.16 ± 1.17 kPa, SVR from 61.50 ± 14.81 to 64.33 ± 15.02 kPa . s/L and CBF from 173.62 ± 81.99 to 192.50 ± 85.40 ml/min, respectively. ***Conclusions:*** The results showed that Acute hypoxia increased all the parameters but the increase in pulmonary vascular resistance was more significant than systemic vascular resistance. This shows that in vivo the hypoxic response of cerebral vessel is dilative, while the hypoxic response of pulmonary vessels as well as that of peripheral systemic vessel is constrictive.

Key Words: Hypoxia, Haemodynamics.

INTRODUCTION

Almost 50 years have elapsed since Von.Euler and Lilijestrand[1] advanced the proposition that acute hypoxia elicits pulmonary vasoconstriction. The idea was not new[2-5] but the report excited considerable interest because it was both propitious & prescient. 1946 was a good year to perceive that pulmonary vasoconstriction response to hypoxia is part of self regulatory mechanism by which pulmonary capillary blood flow is automatically adjusted to alveolar ventilation.

By now methods have been standardised for exploring the pulmonary and systemic circulation in a systematic & sophisticated way and the introduction of cardiac catheterization had brought the previous remote pulmonary circulation within reach.

This study was designed to see the effects of acute hypoxia on hemodynamic with the advanced, sophisticated equipment and standardised method.

MATERIALS AND METHODS

14 domestic dogs of either sex (10-20 kg) were anaesthetised by an IV bolus injection of Na-pentobarbital 30 mg/kg and intermittent doses of 100mg was given intravenously as necessary. The animal was fixed in supine position, intubated with cuffed endotracheal tube and left to breath spontaneously. The body temperature was maintained at 37'C.

The femoral vessels were dissected and exposed. A three way catheter was introduced into the femoral vein and advanced to inferior vena cavae for fluid infusion and injection of normal saline for cardiac out put measurement. A thermistor catheter was introduced into the femoral artery and advanced to the arc of aorta and connected to cardiac output computer (EQ 611V) to measure cardiac out put as well as connected to a transducer to measure mean systemic arterial blood pressure (Psa).

The Jugular vein and carotid artery were exposed and separated, a 5F Swan Ganz catheter was introduced into the external jugular vein and advanced to pulmonary artery to measure mean pulmonary artery pressure (Psa). An electromagnetic flowmeter probe (2 mm) (Nihon Kohdon FM 1200) was placed around the carotid artery to measure the blood flow by which the change in cerebral blood flow (CBF ml/L) was approximately estimated. The ECG lead 2 was recorded continuously to calculate the heart rate (HR beat /min) and to monitor the cardiac function.

After a resting period of one-hour, the pulmonary artery pressure, systemic arterial pressure, cardiac output (CO) and cerebral blood flow were recorded on Nihon Kohden polygraph system before and at the 10^{th} minute of the hypoxic challenge, which was performed by inspiring 10 % O_2, to test hypoxic vascular responses. Three consecutive 10-minute hypoxic challenges were induced in the start. Each hypoxic challenge was followed by a 30-minute recovery under normoxia until the P'pa and Psa became normal. This was to stabilise the hypoxic vascular response. PCO_2, PO_2 and pH were monitored before and after hypoxia with ABL –30 acid base analyser (Copenhagen Denmark)

The parameters were measured before and 10 minute after induction of hypoxia. The hypoxic vascular responses of Pulmonary, Systemic and Cerebral

vessels were expressed as percentage change in pulmonary vascular resistance induced by hypoxia (delta PVR %), percentage change in systemic vascular resistance (delta SVR %) and percentage change in cerebral blood flow (delta CBF %) respectively.

They were calculated as follow:

$$\text{PVR (kPa.s/L)} = \frac{\text{Ppa (kPa)} \times 60}{\text{CO (L.min -1)}}$$

delta PVR = PVR during hypoxia - PVR before hypoxia

$$\text{delta PVR\%} = \frac{\text{delta PVR}}{\text{PVR before Hypoxia}} \times 100\%$$

$$\text{delta SVR\%} = \frac{\text{delta SVR}}{\text{SVR before hypoxia}} \times 100\%$$

$$\text{delta CBF\%} = \frac{\text{delta CBF}}{\text{CBF before hypoxia}} \times 100\ \%$$

The data was analysed by SAS soft ware. The values are expressed as $x + s$. $P < 0.05$ is considered statistically significant.

RESULTS

As shown in the table , 10 minute hypoxia increased the Ppa and PVR significantly from 2.24 ± 0.61 to 3.39 ± 1.09 kPa and 9.49 ± 3.33 to 14.30 ± 5.29 kPa .s L/min respectively. Similarly SVR and CBF values were increased from 61.50 ± 14.81 to 64.33 ± 15.02, 173.62 ± 81.99 to 192.50 ± 85.40 ml/min, respectively. CO and HR were increased from 1.76 ± 0.43 to 1.91 ± 0.52 L/min and 240 ± 55.58 to 260 ± 63.34 beat/min.

Table-1: Effects of 10 minutes hypoxia on hemodynamics in dogs (x + s), (n = 14)

PARAMETERS	CONTROL	HYPOXIA
CO L/min	1.76 ±0.43	1.91 ± 0.52*
HR (b/min)	240 ± 55.58	260 ± 63.34**
Ppa (kPa)	2.24 ± 0.61	3.39 ± 1.09***
PVR (kPa. S/L)	9.49 ± 3.33	14.30 ± 5.29***
Psa (kPa)	14.53 ± 1.16	15.16 ± 1.17**
SVR (kPa.s/L)	61.50 ± 14.81	64.33 ± 15.02**
CBF (ml/min)	173.62 ± 81.99	192.50 ± 85.40**

** $P < 0.05$, ** $P < 0.01$, *** $P < 0.001$, compared with control.*

DISCUSSION

Our results showed that acute hypoxia has significantly increased the pulmonary vascular resistance and cerebral blood flow which are consistent with Jin[6] and Rowell[7]. However the systemic vascular resistance was also increased in dogs. This reveals that in vivo the hypoxic response of cerebral vessel is dilative, while the hypoxic response of pulmonary vessel as well as that of peripheral systemic vessels is constrictive.

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