Electrocardiographic and Biochemical Study in Hemorrhagic Shock in Dogs Treated with Hyperbaric Oxygenation

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The standard Fine preparation for producing irreversible hemorrhagic shock in dogs was used in our laboratory and found to be too severe for assessing various methods of therapy. A modification of the Fine shock preparation was developed, and our observations of the effects of hyperbaric oxygenation (OHP) on the electrocardiographic and biochemical changes and survival rate of such a preparation constitute the present report.

Methods

Seventy-three medium-sized mongrel dogs, average weight 11 Kg., were used in this study. Thirty-five dogs (group A) comprised the control shock group breathing air at atmospheric pressure. Thirty-eight dogs comprised the pressure oxygen breathing group. Of the latter, 25 dogs (group B) were subjected to hemorrhagic shock as in group A and treated with OHP, and 13 dogs (group C) were pressurized with OHP without prior bleeding. All dogs were fasted and received 1.5 mg./Kg. body weight of morphine sulfate intramuscularly. The femoral vessels were used for bleeding and monitoring of arterial pressure. Aqueous heparin in a dose of 2 mg./Kg. body weight was administered intravenously. Groups A and B were bled to a mean arterial blood pressure of 30 mm. Hg. This pressure was stabilized for 30 minutes while both groups breathed room air. The shed blood averaged 46 ml./Kg. in group A, and 49.7 ml./Kg. in group B. Group A remained in shock for a total of two and a half hours, after which the shed blood was reinfused intravenously. Dogs in group B were disconnected from the blood reservoir and introduced into an oxygen pressure chamber where, with oxygen gas, a gauge pressure of 30 lbs./sq. in. (3 atmospheres absolute pressure) was obtained for a total shock time equivalent to that in group A. Reinfusion of the shed blood was then performed. Group C was also pressurized to 3 atmospheres absolute pressure with oxygen for two and a half hours without bleeding. After decompression, a bilateral cervical vagotomy was performed. On the following day, these dogs were again subjected to 3 atmospheres pressure in oxygen.

In a few dogs, samples of mixed venous blood were obtained via a cardiac catheter introduced through the jugular vein. Electrocardiograms and rectal temperatures were monitored throughout all procedures. In groups B and C, arterial, venous, and mixed venous blood samples were drawn before hemorrhage, in shock prior to compression, immediately after decompression, and one hour after reinfusion. In group C, arterial blood samples were drawn during the compression period, in addition to the above samples. Blood was examined for pH, CO₂ content, hematocrit, lactate, pyruvate, and oxygen saturation. Plasma bicarbonate and pCO₂ were calculated from the Singer-Hastings nomogram.

Results

Electrocardiographic Changes

Standard and augmented unipolar limb lead electrocardiograms were recorded throughout the experiments.

In group C, the most constant change observed in the electrocardiogram was in the cardiac rate. No change was observed between the baseline rate recorded outside the tank and that inside the tank before compression. However, bradycardia appeared as the pressure increased. The decrease in cardiac rate was inversely proportional to the pressure in the tank although its extent was variable among several animals. The baseline average cardiac rate of this group was 80 per minute. This dropped to 71 beats per minute at a pressure of 2 atmospheres, and 52 beats per minute after one hour’s compression (fig. 1). On decompression, the average cardiac rate was
observed to be 55 per minute at 2 atmospheres, and 69 per minute at the end of decompression (out of the tank). In all animals, sinus arrhythmia appeared and persisted throughout the experiment. This arrhythmia might have been induced by morphine narcosis since it appeared in the baseline electrocardiograms. In some tracings, inversion of the T wave and depression of the S-T segment were noted (fig. 2). These changes became more pronounced with increase in pressure and duration of exposure, but receded during the decompression period. In the vagotomized dogs, the sinus arrhythmia which had appeared previously under morphine disappeared, and the cardiac rate became approximately twice the original rate. There were no significant changes in either rate, rhythm, or wave configuration of the electrocardiograms of these vagotomized dogs during exposure to OHP. In one dog, nodal beats appeared at a pressure of 3 atmospheres.

In group B, subjected to hemorrhagic shock and then treated with OHP, a persistent sinus tachycardia developed after bleeding and increased during compression. This trend was reversed by decompression and reinfusion of the shed blood. Figure 1 demonstrates an average increase in cardiac rate from 55 to 159 beats per minute after bleeding. A further increase to 250 beats per minute was observed upon compression to 2 atmospheres, with stabilization of the cardiac rate at 223 beats per minute at 3 atmospheres. A gradual decrease occurred on decompression and reinfusion.

In this group, sinus arrhythmia was also frequently present after morphine. There was a change in the configuration of the P wave after bleeding, with a marked decrease in the amplitude of the QRS complex. During exposure to OHP, a depression of the S-T segment was constantly observed, the T wave becoming tall and sharp (fig. 3). Atrial fibrillation and atrioventricular dissociation were occasionally seen. One dog demonstrated a severe depression of the S-T segment a few minutes after decompression; this depression improved with reinfusion of blood. Three dogs developed ventricular tachycardia followed by ventricular fibrillation and death one hour after compression at 3 atmospheres absolute. Nearly all changes in the configuration and amplitude of the various complexes of the electrocardiogram reverted toward normal after decompression and reinfusion.

Biochemical Changes

The biochemical changes are summarized in figure 4. The oxygen saturation shows a definite increase in the arterial blood in groups B and C. The mixed venous blood was not measured in either group in the chamber; however, it was moderately increased immediately after decompression in the hemorrhagic shock group treated with OHP. The arterial pCO₂ was observed to drop in both groups after exposure to OHP. The mixed venous pCO₂ increased at the end of decompression in the shock group treated with OHP, whereas an opposite change was seen in the control group. The lactate levels showed a marked increase in both groups during exposure to OHP, with reversal to normal values after decompression.

Survival Rate

Of the 35 dogs which constituted group A, five died of cardiorespiratory arrest early in the experiment, before reinfusion of the shed blood, and were excluded. Twenty-three died
within 24 hours, and two died two days after the procedure. Only five were long-term survivors, giving a survival rate of 17 per cent.

In group B, six dogs were excluded for the same reason as those in group A, five died within 48 hours, and 14 survived 48 hours and longer, giving a survival rate of 74 per cent.

Discussion

In 1878, Bert observed a lowering in the pulse rate during exposure to 3 atmospheres of air pressure. In 1921, Dautrebande and Haldane reported an increase in the bradycardia as the oxygen pressure was raised to 2 atmospheres. This was explained as a possible protective mechanism against excessive changes in respiratory metabolism. Whitehorn and Bean found a similar bradycardia in decerebrate dogs, which progressed to as low as one-half to one-third of the precompression value. Behnke et al. observed a consistent bradycardia in man breathing oxygen at pressures of 1 to 4 atmospheres. At 4 atmospheres, the bradycardia might terminate in syncope or in an increased pulse rate followed by convulsive seizure. Taylor found a 50 per cent decrease in the cardiac rate of rats anesthetized with chloralose or urethane and subjected to oxygen at 6 atmospheres.

Our experiments confirm the above observations, although only morphine narcosis was utilized. Various explanations have been given for this bradycardia. Whitehorn and Bean demonstrated the dependence of this bradycardia on the intact vagi; tachycardia was not noted after vagotomy, but was replaced by a delayed bradycardia, which occurred three to four hours after exposure. They also demonstrated a similar effect on the cardiac rate by exposures to high CO₂ as well as low oxygen tensions, and concluded that the bradycardia is initially due to a predominant vagal activity, but later to a more direct mechanism compatible with an increased CO₂ content of the tissues. Our data in the control group, as well as those presented by Lamberts et al., however, do not confirm this assumption, since no increase in the pCO₂ of the arterial blood or tissues was observed. Besides, if this bradycardia were secondary to CO₂ retention, we
should have observed it in the hemorrhagic shock group treated with OHP; instead, tachycardia occurred.

As to the effects of OHP on the heart, Whitehorn and Bean⁴ demonstrated, in experiments on frog hearts, an alteration in the pacemaker, in conductivity, and in contractility. In dogs, they showed prolongation of the P-R interval amounting to 200 to 300 per cent of the precompression value, frequently progressing to atrioventricular block. There was also alteration of the conformation of the electrocardiogram, although it was not so consistent as were the alterations of the cardiac rate and conduction. A diminution of the amplitude of the P wave occurred most prominently at the height of OHP effect. The QRS complex was not significantly altered except terminally. The T wave was increased in both duration and amplitude in about 50 per cent of the experiments. Atrial fibrillation appeared in two experiments only. Conduction disturbances were also reported by Taylor.⁶ Amorim⁸ demonstrated lengthening of the P-R interval in 11 patients subjected to 2 and 3 atmospheric pressures, with minor variations in P and T waves. Right-axis deviation and complete atrioventricular block were also observed.

The electrocardiographic changes seen after hemorrhagic shock and OHP, i.e., S-T elevation or depression and T-wave inversion (upright or tent-shaped), were reported to occur after hemorrhagic shock alone.⁹ They denote disturbances of myocardial function secondary to myocardial damage. It is interesting to note that Whitehorn and Bean⁴ ascribed cardiac failure after exposure to OHP to failure of the pacemaker, the conductive mechanisms, and probably also the mechanism of contraction, but not to ventricular fibrillation. In group B, subjected to hemorrhagic shock and OHP, three dogs developed ventricular tachycardia followed by ventricular fibrillation and death in the chamber. Despite these electrocardiographic changes, a significant increase in the survival rate was demonstrated in hemorrhagic shock treated with OHP. This is ascribed to the improved tissue oxygenation provided by the increased amount of oxygen in physical solution which is made available to the tissues, thus bypassing the deficient oxygen-carrying hemoglobin system in this state of shock.¹⁰

Summary

A modification of the standard Fine preparation for hemorrhagic shock yielding an 83 per cent mortality rate was developed. Hyperbaric oxygenation (OHP) at 3 atmospheres absolute was found to decrease the mortality rate significantly, to 26 per cent. Electrocardiographic changes induced by OHP were bradycardia and sinus arrhythmia, which were abolished by vagotomy. Tachycardia, depression of the S-T segment, with changes in the configuration of the T wave that indicated myocardial damage, was observed in shock dogs treated with OHP. All changes improved after decompression and reinfusion of shed blood. The improved survival rate is attributed to better oxygenation of the hypoxic tis-

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HEMORRHAGIC SHOCK & OHP

PCO₂ mm Hg.

INTERPOLATED VALUES
IN O₂ CHAMBER

TIME IN HRS. 0 1 2 3 4

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HEMOCRIT

INTERPOLATED VALUES
IN O₂ CHAMBER

TIME IN HRS. 0 1 2 3 4

CONTROL OHP

PCO₂ mm Hg.

INTERPOLATED VALUES
IN O₂ CHAMBER

TIME IN HRS. 0 1 2 3 4

CONTROL OHP

HEMOCRIT

INTERPOLATED VALUES
IN O₂ CHAMBER

TIME IN HRS. 0 1 2 3 4

Figure 4

Graphs showing changes after control OHP and hemorrhagic shock and OHP in: top left, PCO₂ and hematocrit; top right, CO₂ content and pH; bottom left, bicarbonate and O₂ saturation; bottom right, lactic acid in hemorrhagic shock and OHP.

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sues by the increased physically dissolved oxygen.

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