The administration of oxygen, 60 to 70 per cent in inspired air, to dogs with ectopic ventricular tachycardia resulting from myocardial infarction produced no consistent changes in ectopic frequency, but the blood pressure was elevated during oxygen administration in every trial.

The production of myocardial infarction in dogs' hearts by experimental coronary occlusion utilizing a standard technic results in the development of ventricular tachycardia in almost all cases. Typically the tachycardia begins within four and one-half to eight hours after occlusion, and persists for two to four days if untreated. Certain other findings in these animals are elevated temperature for one to three days, hypotension in some animals, dyspnea in a small percentage of them, and electrocardiographic patterns typical of myocardial infarction when not obscured by the ventricular tachycardia.

Using such animals, experiments have been performed to determine the effects of oxygen, 60 to 70 per cent in inspired air, upon the arrhythmia, and upon the arterial pressure.

Technics

Thirteen tests were performed in eight dogs. For surgery, the dogs were anesthetized with pentobarbital sodium, 30 mg. per kilogram. Using artificial respiration and aseptic surgical technics, the anterior descending artery was dissected free enough to pass ligatures under it at the level of the distal edge of the left auricular appendage, and was ligated there in two stages. A partial occlusion was produced first by tying one ligature snugly, but not tightly, around the artery, together with a 20-gauge hypodermic needle and withdrawing the needle. The second ligature was tied tightly around the artery after an interval of 30 minutes. By this method, losses by ventricular fibrillation during the immediate danger period of the first 10 minutes which follow abrupt occlusion were avoided. The chest was closed, and the animal given fluids and other routine postoperative care, including morphine if needed.

On the following morning, 16 to 20 hours after occlusion and after the animal had completely recovered from anesthesia, a number of control electrocardiograms and simultaneous blood pressure records were made and then testing was begun.

The arrangement for the administration of oxygen to dogs is illustrated in figure 1. The dog's head was placed in a bell jar of appropriate size. Oxygen tubes were attached to the small end of the bell, and the large end was fitted by a rubber collar which was drawn snugly about the dog's neck by a piece of twine. A regulated flow of the gas entered the respiratory apparatus through tube A. Tubes B and C were connected with a motor circulator type basal metabolism machine which served as an oxygen reservoir and removed carbon dioxide. There was a considerable leak of gas by the rubber collar around the dog's neck. The rate of flow of gas was such that the oxygen content of frequent samples remained between 60 and 70 per cent and the carbon dioxide content less than 0.2 per cent. Samples of air for analysis were taken from inside the bell by needle through the rubber dam.

Arterial blood samples for oxygen determinations were drawn (a) before each oxygen test, (b) near the end of the period of administration of oxygen, and (c) 3/4 to 1 hour after the animal was removed from the apparatus. Analyses were made by the Van Slyke and Scholander technics. The dog, sedated by prior administration of morphine or Demerol, usually slept through the test.
RESULTS

Figures 2 and 3 illustrate typical ectopic ventricular tachycardias and blood pressures on the first postocclusion day and the results obtained upon administration of oxygen.

In the experiment represented in figure 2 the ectopic rate, measured from long records, did not change significantly during oxygen administration, ranging from 180 to 190 during a period of four hours of observations before, during, and after the oxygen test. The duration of the period of respiration of oxygen in the bell was one hour. The record in figure 2B was made just before termination of this period. The blood pressure rose during respiration of oxygen at high tension and declined again after the return to air (Fig. 2C). The systolic pressure varied considerably due to the arrhythmias. The diastolic pressure was steadier, and showed a rise from a control level averaging about 85 mm. Hg to a plateau of about 105 during oxygen administration and declined to about 65 or 70 after return to air.

Figure 3 is from another animal. In this test there was a diminution of ectopic rate from 190 to 150 during the period in the oxygen bell. After the return to air the ectopic rate remained between 150 and 170. The diminution in ectopic frequency was an exceptional response and is regarded as fortuitous.

The mean blood pressure ranged between 60 and 70 mm. Hg during most of the control period in this case, and exhibited an increase to range between 90 and 110 mm. during the oxygen administration. After return to air the pressure leveled off between 70 and 85. The dog slept through the entire test and for some time afterward.

During 13 oxygen administration tests in eight dogs, the ectopic rate was unchanged in nine, increased in two, and decreased in two.

Blood pressures were recorded in six of the dogs. During the six tests the blood pressure rose during every trial without exception. The amount of the rise varied between 15 and 40 mm. Hg, averaging 27.

The administration of oxygen increased the average oxygen content of arterial blood from 15.9 to 18.7 cc. per 100 cc. of blood. Analysis of samples taken 30 minutes to 1 hour after termination of oxygen administration showed a decline from the elevated level during administration to an average of 15.8. Percentage of saturation was not determined but oxygen content in the control samples averaged 85 percent of that in the samples taken during oxygen administration. The variation was from 80.0 to 92.5 percent.
DISCUSSION

No data which directly explain the rises of blood pressure upon respiration of oxygen at high tension are at hand. The pulse pressure records do not contain evidence of increased systolic discharge and there was no consistent change in heart rate. It is inferred that the increase in blood pressure is due to vasoconstriction. An item of possible evidence, applicable to these experimental blood pressure changes is found in the observation by Mathison that when the artificial respiration to a spinal curarized animal is stopped, the blood pressure declines and the intestinal loop in a plethysmograph dilates for about 30 seconds before strong vasoconstriction and rise in blood pressure begin. The vasodilatation was shown to be local in origin and the vasoconstriction that supervened when the degree of asphyxia increased to a sufficient degree was due to reflex action. It is probable that the local effects of oxygen lack and asphyxia upon blood vessels are dilator in nature throughout the circulatory system. It has been demonstrated in the coronary and pulmonary vessels. If the hypoxic threshold for local vasodilatation is lower than that for reflex vasoconstriction as indicated by Mathison, then it appears possible that the degree of asphyxia found in the dog with coronary occlusion, sedated by morphine or Demerol, is within the vasodilator range, and that the administration of oxygen removes this cause of vasodilatation and thereby produces an increase in blood pressure. Other studies that included measurement of blood pressure during various degrees of oxygen lack have not recorded reduced blood pressure in mild hypoxia, but diastolic gradients in the records of Sands and DeGraff are indicative of vasodilatation. It may be significant that the animal in the present series with the lowest mean pressure prior to the administration of oxygen showed the greatest increase during the period of breathing oxygen.

Katz and coworkers found that oxygen administration did not change the blood pressure of normal men, produced increases in three tests in cardiac patients, had no effect in two others and that a fall occurred in one. Small and inconstant changes in blood pressure upon administration of oxygen at high pressure to normal subjects has been reported by others.

The finding that the administration of oxygen after the development of ventricular tachycardia had no consistent effect on the arrhythmia probably indicates that lack of oxygen is not an immediate excitant of ectopic impulses in the tachycardia that develops with myocardial infarction. It has been pointed out that the delayed ventricular tachycardia that develops following coronary occlusion in the animal has its onset after a latency that approximates the duration of ischemia that is required to produce microscopic evidence of necrosis. Because of this and other considerations, it has tentatively been assumed that the most important excitatory factors are due to necrosis, and probably to substances liberated during the breakdown of myocardial tissue.

From these experiments it can be concluded that the administration of oxygen at high tension after myocardial infarction is established will not terminate nor significantly reduce the frequency of ventricular tachycardias resulting from this cause. The administration of oxygen may be of considerable benefit, however, by elevating the blood pressure and by increasing the oxygen tension of the blood perfusing the tissues. Levy has stated that the therapeutic use of oxygen in myocardial infarction is responsible for the saving of life in some instances, and listed the criteria which indicate the need for it. These criteria include signs of oxygen deficit and hypotension. Barach and Levy found that 60 per cent oxygen could be given indefinitely without harm and that 70 per cent could be used for several hours at a time.

SUMMARY

The administration of oxygen, 60 to 70 per cent in inspired air, to dogs with ventricular tachycardia resulting from myocardial infarction had no consistent effect upon the ectopic activity, but it elevated the arterial
pressure in every case. Possible mechanisms of these results are discussed.

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Oxygen Administration upon Ventricular Tachycardia and Blood Pressure in Animals with Acute Myocardial Infarction

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_Circ Res._ 1953;1:83-86
doi: 10.1161/01.RES.1.1.83

_Circulation Research_ is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
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Print ISSN: 0009-7330. Online ISSN: 1524-4571

The online version of this article, along with updated information and services, is located on the World Wide Web at:
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