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The Electrocardiographic Changes in Anoxemia.

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A sign usually considered to be pathognomonic of coronary thrombosis is the high branching of the "T" wave of the electrocardiogram. It appears soon after the accident of coronary occlusion and reaches its maximum intensity within a few hours; and disappears after a few days. Although the mechanism of the change is unknown, it has been attributed to a number of factors, among which may be mentioned the influence of the current of injury on the normal change of electric potential of the heart,[1] anoxemia of the heart muscle[2] and asphyxia.

The following experiments enable us to draw certain conclusions concerning the mechanism of the change.

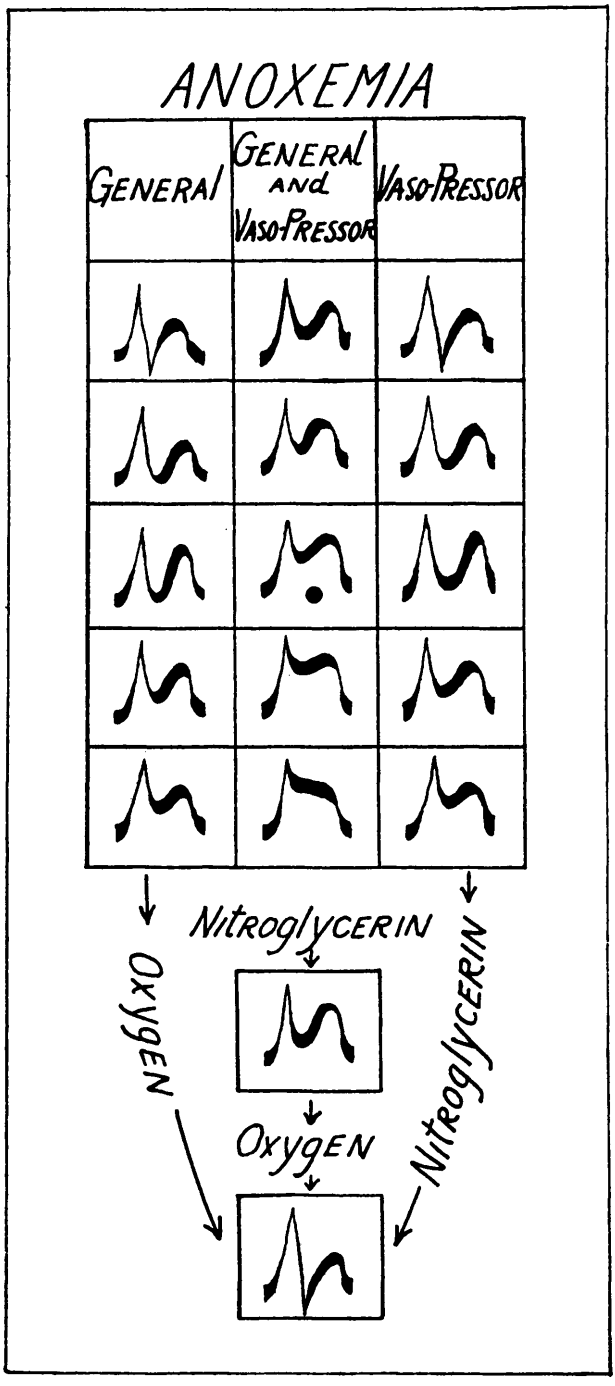
Dogs were anesthetized with amytal and were placed under conditions of anoxemia. To accomplish this, they were connected by means of a tracheal cannula to a large dead space into which the animal rebreathed and from which the carbon dioxide was absorbed with soda lime. They, therefore, breathing air from which the oxygen was gradually reduced, passed into a state of anoxemia, and carried to a point of complete asphyxia at which time the oxygen content of the arterial blood was as low as 20%. When the oxygen saturation of the arterial blood fell below 50% of the normal, the animals showed in their electrocardiogram changes similar to those observed clinically in coronary occlusion. The release of the animal from the anoxemia state caused the electrocardiogram to return to normal.

As was pointed out by Gruber and Kountz,[3] a picture similar to anoxemia was produced by the injection of pitressin in normal unanesthetized animals. They have also shown that one of the primary actions of the drug on the heart was that of coronary constriction. The change in the electrocardiogram produced by the drug could be caused to disappear by vasodilator substances such as sodium nitrite. Therefore, as the action of pitressin is that of coro-

[1] Clark, N. E., and Smith, F. J., *J. Lab. and Clin. Med.*, 1925, xi, 1071.

[2] Katz, L. N., Feil, H. S., and Scott, R. W., *Am. Heart J.*, 1929, v, 77.

[3] Gruber, Chas. M., and Kountz, Wm. B., PROC. SOC. EXP. BIOL. AND MED., 1929, xxvii, 161.


ANOXEMIA
GENERAL
GENERAL AND VASO-PRESSOR
VASO-PRESSOR
NITROGLYCERIN
OXYGEN
OXYGEN
NITROGLYCERIN

nary constriction the result may be explained on the basis of anoxemia of the heart.

A combination of the two experimental procedures, namely the giving of pitressin to anoxemic animals, produces a change similar to that seen in the most extreme changes of coronary occlusion. In the curves under this condition, it was found in the electrocardiogram that the "T" wave branched directly from the downstroke of the "QRS" complex. The electrocardiogram immediately returned to normal if oxygen and vasodilator substances were given.

The change in the electrocardiogram began by a decrease in amplitude of the "T" wave followed by inversion, which occurred at 30% of oxygen unsaturation of the arterial blood. As the anoxemia progressed, however, the "T" wave became upright, increased in height and widened at the base. The widening occurred at the expense of the S.T. interval which became short and was finally obliterated. Further widening of the base of the "T" wave as occurred when pitressin was given to animals in a state of anoxemia, causes the crest of the "T" to fuse with the "QRS" downstroke.

The conclusion is that anoxemia of the heart muscle may be the cause of the pathognomonic sign seen in the electrocardiogram. Anoxemia occurs as a result of shutting off of blood. This electrical phenomena disappears experimentally by relief of the coronary constriction or with the administration of oxygen. Clinically, in coronary thrombosis one might assume that its disappearance is due to the establishment of a collateral circulation.

The study suggests the possibility of a clinical test to determine the efficiency of the coronary circulation.