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Effect of Anoxemia upon Electrocardiogram of Cats after Coronary Ligation.

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Ligation of the left branch of the left anterior descending coronary artery in cats results in typical "coronary" electrocardiographic records. These abnormal tracings frequently disappear in 2 to 3 weeks despite the persistence of grossly demonstrable infarcts at autopsy. Following the suggestion of Levy, Bruenn, and Russell[1] that the electrocardiographic changes which anoxemia induces in patients with coronary disease, may be of diagnostic value, these cats were subjected to mild anoxemia. When normal cats, anesthetized with pentobarbital, were made to breathe 10% oxygen in nitrogen for 20 minutes, the electrocardiographic changes induced were slight, and unlike those following coronary ligation.

Immediately after coronary ligation, the electrocardiograms of 7 of the 8 cats studied showed deviation of the RS-T segment in 19 of 21 leads. The electrocardiogram of the eighth cat was normal, but subsequently showed RS-T segment deviation. The T-waves were generally increased in amplitude. At this time, the induction of mild anoxemia exaggerated the RS-T deviations in 13 of the 19 leads, and elicited RS-T deviations in the electrocardiogram of the one cat in which they were lacking postoperatively. T-wave changes were slight and variable. The induction of the anoxemia was repeated at weekly intervals thereafter and continued to exaggerate the RS-T deviations, or to cause their reappearance.

Meanwhile, the RS-T segments of the electrocardiograms of the non-anoxemic cats approached the iso-electric line, and the electrocardiograms of 6 of the 8 cats returned to normal within from 12 to 29 days. One of the 2 cats whose electrocardiograms continued to show "coronary" changes was sacrificed 14 days postoperatively because of impending exitus from "snuffles." The other continued to show electrocardiographic changes until it was sacrificed, 50 days postoperatively. When the electrocardiograms had returned to normal, the induction of anoxemia resulted in the

[1] Levy, R. L., Bruenn, H. G., and Russell, N. G., Jr., *Am. J. Med. Sci.*, 1939, **197**, 241.

reappearance of "coronary" complexes in every case. Thirteen of 18 leads showed deviation of the RS-T complex.

After experimental coronary ligation, the resulting infarct is composed of a central area of ischemic tissue, surrounded by a relatively narrow periphery of edematous, anemic, but still viable myocardium. Since it was shown that the electrocardiograms of such cats frequently resume normal contours in the presence of a fibrotic area, it is suggested that the electrocardiographic changes which had been present were due to local myocardial anoxia and not to the mere presence of a mass of scar tissue. If it is assumed that the electrocardiograms return to normal when the local anoxia has fallen below a given threshold value, the induction of mild anoxemia would cause the reappearance of "coronary" changes. By raising the level of local anoxia above the threshold, its effects upon the electrocardiogram would become manifest. This was actually accomplished.

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Avitaminosis B_1 and Pigeon Brain Potentials.

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Since vitamin B_1 lack disturbs the normal carbohydrate metabolism of brain, it was desirable to follow its influence on brain potentials. Pigeons were standardized on a stock diet for 30 days, then on a diet of polished rice and salt.[1] Although deficient in other nutritional elements, only vitamin B_1 lack could play a rôle within the time limits, for typical beri-beri symptoms (opisthotonus) developed in 30-40 days. A small portion of the calvarium over the occipital portion of one cerebral hemisphere was removed under nembutal (30 mg/kg, sufficient to relax the rigid muscles) and the exposed brain kept moist and warm with Ringer. Potentials were led off (different electrode on exposed brain; indifferent on bone over opposite hemisphere) with Ag-AgCl wick electrodes,[2] amplified and recorded with a crystograph. When desired, 2-5 mg of crystalline vitamin B_1 were injected intramuscularly, administered

1 Kinnersley, H. W., Peters, R. A., and Reader, V., *Biochem. J.*, 1928, **22**, 276.
2 Libet, B., and Gerard, R. W., *J. Neurophysiol.*, 1939, **2**, 153.