

11482

Non-Induced Cardiopathic Disease in a Rabbit—Electrocardiographic and Pathologic Study.

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The recognition of heart disease in experimental animals would be a great asset in the selection of healthy animals. Miller¹ has shown that spontaneous interstitial myocarditis existed in rabbits. Reference to the electrocardiographic diagnosis of non-induced cardiac disease in laboratory animals was not found in the literature. However, many electrocardiographic studies on induced heart disease have been made.²⁻⁴ Seifried⁵ referred to pathologic studies in rabbits with heart disease.

In a preliminary control electrocardiographic study on 16 three-month-old rabbits, there was a definite variation in the voltage, rhythm and form of the electrical complexes. The electrocardiogram of rabbit No. 7 in this series diverged definitely from the average so that a diagnosis of acute myocardial disease was suggested. This tracing also indicated the possibility that the disease might be localized in the myocardium.

This rabbit was received in a shipment one week previously and no known experiments had been performed on it. General observations showed the animal to be drowsy, inactive, and anorexic. The rectal temperature was 103° F. on the day the electrocardiogram was taken. Inanition continued for 2 days. On the third, the rabbit was found dead in its cage. On autopsy, a gross inspection of the body, lungs and abdominal viscera showed no demonstrable pathology. The epicardium, however, was adherent to the right antero-lateral chest wall. No pericardial effusion was present. By comparison with normal hearts it measured about the same size. The heart was placed in formalin, sectioned and stained with hemotoxylin and eosin for microscopic study.

Figure I. shows electrocardiograms of a control rabbit and of

¹ Miller, C. P., *J. Exp. Med.*, 1924, **40**, 524.

² Agduhr, E., and Stenstrom, N., *The Appearance of the Electrocardiogram in Heart Lesions Produced by Cod Liver Oil Treatment*, Almquist and Wiksells, Uppsala, 1930.

³ Johnston, F. D., Hill, I. G. W., and Wilson, F. N., *Am. Heart J.*, 1935, **10**, 903.

⁴ Wood, F. C., and Wolferth, C. C., *Arch. Int. Med.*, 1933, **51**, 771.

⁵ Seifried, O., *Krankheiten des Kannedens*, Julius Springer, Berlin, 1937.

rabbit No. 7. The control animal exhibited only slight variations in the contours of its electrocardiograms during 4 months of observation. Significant variations in the standard leads (I, II, III) and of the exploring right and left chest leads paired with the indifferent left leg electrode were observed in rabbit No. 7 as compared with the control observations.

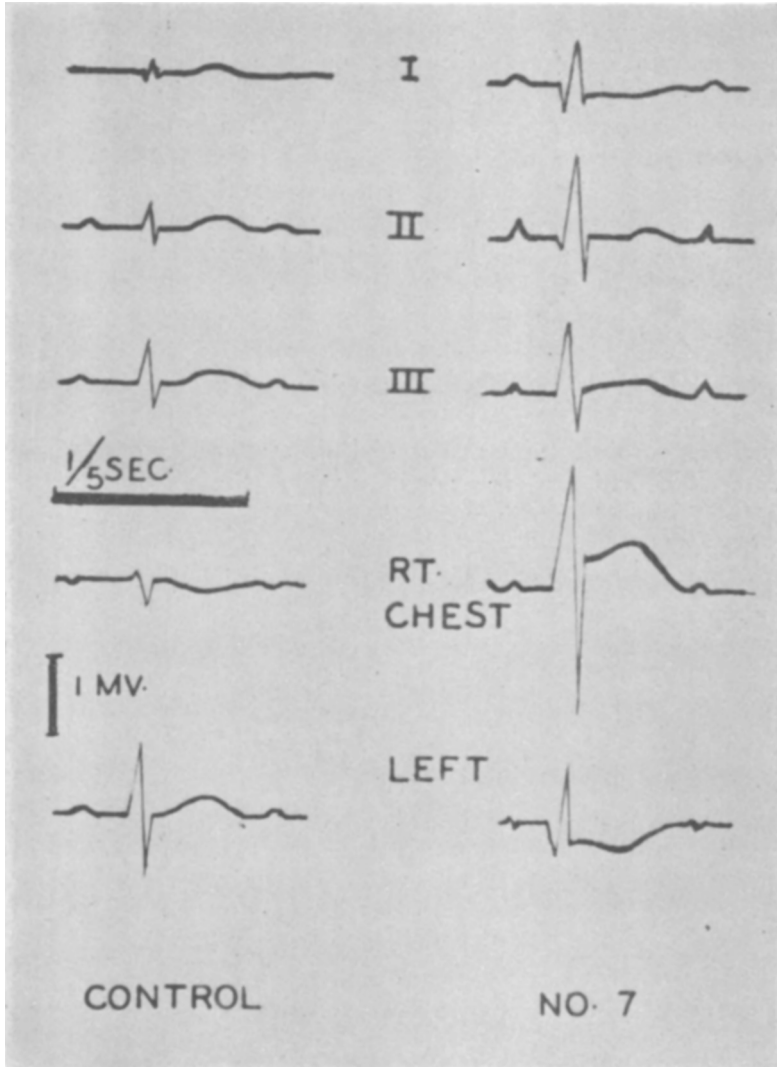


FIG. 1.

Diagram of a normal (control) and the abnormal rabbit electrocardiograms (rabbit No. 7) taken in all leads at normal sensitivity and adapted to the new terminology for the exploratory leads.

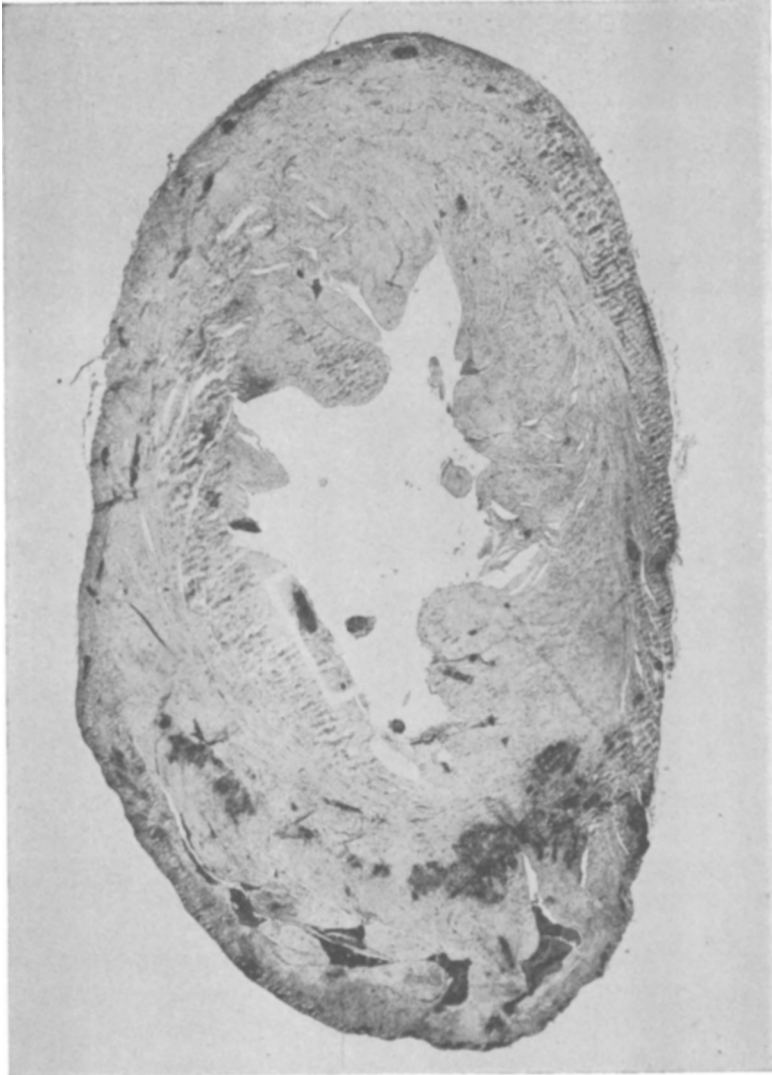


PLATE 1.

A section through rabbit No. 7 myocardium, approximately midway between the apex and the auricular junction. Low power magnification.

The chief objective differences between these graphs were defined by describing the deviations in the abnormal electrocardiogram of rabbit No. 7. The presence of the Q_1 of 2.8 mm, the depressed $RS-T_1$ of 1.7 mm, the inverted T_1 of 1.7 mm, the elevated $RS-T_3$ of 1.4 mm were probable deviations from the normals, but the T_2 and T_3 in the standard leads were upright and not definitely

abnormal. The right chest exploring lead showed no evidence of a Q wave, but a markedly elevated RS-T segment, associated with an upright T wave. The left chest exploring lead showed a deep Q wave and a markedly depressed RS-T segment associated with an inverted T wave. These RS-T deviations are definitely abnormal. The voltages of the chief QRS deflections in all the leads appeared greater than those observed in normal rabbits. In general, the rate of 300 per minute was faster than the average of normal rabbits studied.

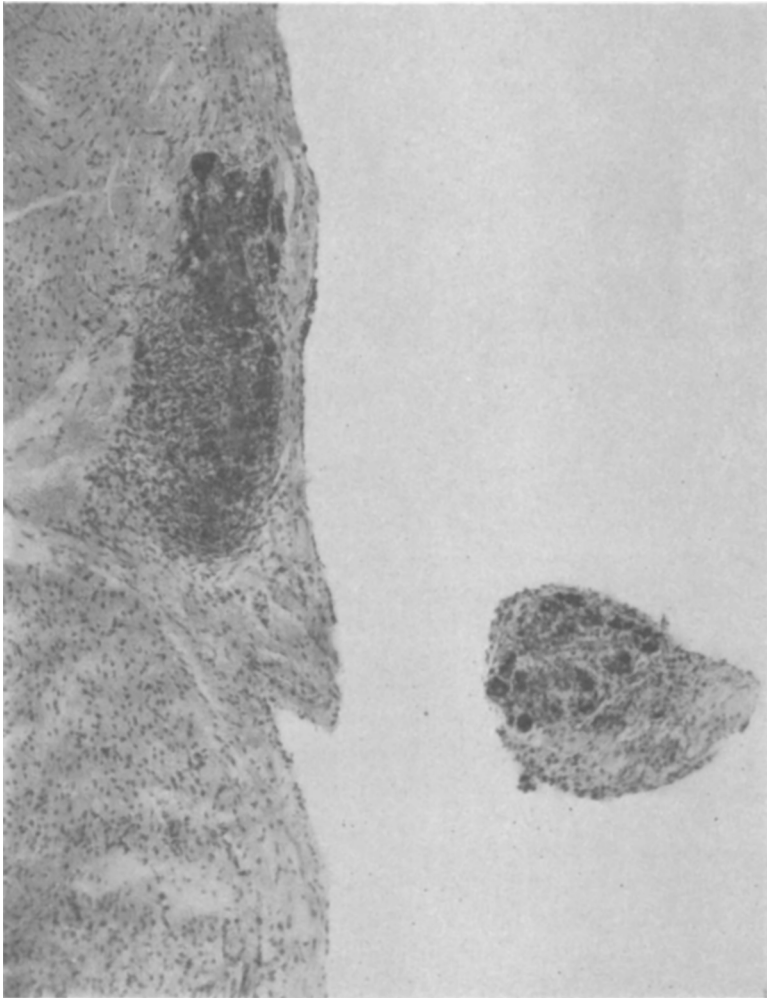


PLATE 2.

Magnification of Plate 1 section in the marked inset showing scattered areas of normal, necrotic, and necrotic tissue beneath the endocardium and of the papillary muscle.

Microscopic sections through different levels of the myocardium of a normal rabbit showed the muscle bundles sharply outlined and the nuclei well stained. No evidence of degeneration or inflammation was present. Sections through the myocardium of rabbit No. 7 showed (Plates 1 and 2) areas of necrosis everywhere, chiefly affecting the muscle and largely involving the right ventricle and interventricular septum. In the left ventricle the areas of necrosis were chiefly beneath the endocardium. The papillary muscles were prominently affected. Slight periarterial infiltration was present and areas of necrosis were seen in both the auricular walls. The anterolateral ventricular epicardium showed a hyaline fat necrosis. The pathological diagnosis was degeneration and necrosis of the myocardium.

Summary. The incidence of non-induced cardiopathic disease among laboratory animals may greatly alter the prognosis, course and reaction to given control or experimental conditions. Disease of the myocardium as confirmed by pathologic studies may greatly alter the electrocardiogram in the rabbit. The changes found suggesting a localized lesion of the ventricle by the electrocardiogram were not supported by pathologic studies. Since bacteriologic studies were not done, no conclusive evidence as to the etiology of the myocarditis was suggested.

11483

Variability of Action on Heart Rate Compared with Metabolic Effect of Various Thyroid Preparations.

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In our previous publication¹ we have shown that certain thyroid preparations fed to thyroidectomized rats exert a stimulating action on the heart rate which varied from one product to the other in its relation to the corresponding metabolic increase obtained. While in 2 U.S.P. thyroid preparations the cardiac effect prevailed, thyroxine and thyroid globulin proved to be of low action on the heart if given at a dose to produce an equal metabolic response. It was shown furthermore, that alkaline hydrolysis of thyroid globulin

¹ Meyer, A. E., and Yost, M., *Endocrinology*, 1939, **24**, 806.