NEW LIMITATION CHANGE

TO
Approved for public release, distribution unlimited

FROM
Distribution authorized to U.S. Gov’t. agencies and their contractors; Administrative/Operational Use; MAR 1964. Other requests shall be referred to Department of the Army, Fort Detrick, Attn: Technical Release Branch/TID, Frederick, MD 21701.

AUTHORITY
Fort Detrick/AMXFD ltr dtd 9 Feb 1972
DDC AVAILABILITY NOTICE

Reproduction of this publication in whole or in part is prohibited. However, DDC is authorized to reproduce the publication for United States Government purposes.

STATEMENT #2 UNCLASSIFIED

This document is subject to special export controls and each transmittal to foreign governments or foreign nationals may be made only with prior approval of Dept. of Army, Fort Detrick, ATTN: Technical Releas Branch/ TID. Frederick, Maryland 21701

DEPARTMENT OF THE ARMY
Fort Detrick
Frederick, Maryland
ELECTROCARDIOGRAPHIC FINDINGS IN GUINEA PIGS INFECTED WITH B. ANTHRACIS


It is known that during the course of anthrax the myocardium undergoes degenerative alterations of a fundamental kind, while the mechanism whereby B. anthracis brings about the death of the animal is still in dispute. In view of this fact electrocardiograms have been made in an attempt to reveal and follow up the initiation of the degenerative process in the myocardium and to determine whether such research could supply useful information to show whether any of the theories advanced to explain the cause of death in anthrax might be valid (details are given in the full, unabridged text of this paper).

To this end, 12 guinea pigs were infected with anthrax by subcutaneous inoculation with 1 ml. of a suspension of spores in dilutions of 1:200,000; 1:100,000 and 1:50,000. At the same time, two guinea pigs were inoculated in the same manner with 1 ml. each of sterile physiological solution as control animals.

The animals were given electrocardiograms before infection and after infection at 12-hour intervals until death, using a Galileo Model G6 electrocardiograph having a tape speed of 50 cm. per second and calibrated for 2 cm. deflection equal to one millivolt.

The electrocardiograms of each animal obtained after infection were compared with those taken before infection in order to evaluate more accurately those alterations which could be observed.
The results obtained show that the anthrax induced experimentally in our animals did not change the rhythm, the frequency, the duration of R-R interval, and the shape of ventricular complexes. On the other hand it did bring about an alteration of the atrial P wave with a marked increase in voltage and amplitude as well as abnormal S-T end phase and T phase in the electrocardiogram consisting in a considerable accentuation of the T waves and various deflections in the S-T segment. The chief abnormality is that of the P-wave since it appeared both large in amplitude and frequently (in 75% of the animals).

The frequency of appearance of these abnormalities was in no way related to any differences in the concentration of the suspension used for inoculation.

The data obtained by us lend themselves to interesting conjecture since although the changes in the terminal portion of the trace, their differences in amplitude, time of appearance and duration can be attributed to degenerative changes in the myocardium already pointed out by various authors and confirmed by us by examination of the tissues of the guinea pigs used in the experiments, these changes probably being due to the action of a toxic factor at the level of the myocardium and a concomitant presence of necrotic foci; the alteration in the atrial wave, when compared against the tissue findings, appears to be the expression of pulmonary congestion with overloading of the lesser or pulmonary circulation with a plethora or excess of blood in the veins, manifesting itself chiefly in the condition of the atria, brings one to recognize in a cardiac-respiratory insufficiency the possible cause of death in experimentally-induced anthrax infection.

Enzymatic or toxic factors of various types may play a part or be the principal cause of such an insufficiency, consequently such hypotheses would seem to us to remain valid, while the absence of any other type of abnormality in our cardiograms lead us to reject those theories based on occlusive embolisms, acute hypoxemia, serious disturbances of the autonomic nervous system or of salt metabolism, etc.

(With presentation of tables, electrocardiographs and microphotographs of tissues).

- END -

2 Best Available Copy