

that the drug may occasionally induce abnormal impulse formation in the ventricular muscle, but satisfactory proof that it does so is as yet wanting.

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Changes in the electrocardiogram following the arsphenamine treatment of cardiac and aortic syphilis.

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We have recently observed conspicuous changes in the electrocardiogram following the administration of arsphenamine in four cases of cardiac and aortic syphilis.

A patient with syphilitic myocarditis and complete right bundle branch block developed an abnormal idioventricular rhythm following the administration of two-tenths gram of arsphenamine and died a few days later. For several weeks preceding the treatment his condition had been stationary.

Two patients with syphilitic aortitis, but without definite signs of cardiac syphilis, and with practically normal electrocardiograms, developed diphasic complexes suggesting incomplete bundle branch block, following intensive arsphenamine therapy. In one of these patients the T-wave changes gradually disappeared but the QRS changes persisted. The other patient could not be followed. Similar but less conspicuous changes occurred in a third patient with syphilitic aortitis who showed great enlargement of the heart and left ventricular preponderance before treatment.

These observations indicate that the administration of arsenamine in cases of cardiac syphilis may sometimes be followed by myocardial changes of an undesirable kind. The slow development and persistence of the electrocardiographic changes suggest that they are not due to a local Herxheimer reaction, although this possibility cannot be excluded. The rapid destruction of the luetic lesions and their replacement by scar tissue with

injury to the intraventricular conducting system, directly or through interference with the coronary circulation, is a possible cause.

Similar treatment produced no changes in the electrocardiograms of five other patients with aortic syphilis. In twenty patients with primary and secondary lues intensive arsphenamine treatment produced no electrocardiographic abnormalities. A study of about sixty patients with primary and secondary lues has convinced us that reliable clinical or electrocardiographic evidence of involvement of the heart or aorta during this stage of the disease is decidedly rare.

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Factors influencing distribution of potential differences, produced by heart-beat, at surface of body.

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In order to understand the influence of the position of the electrocardiographic electrodes upon the form of the electrocardiogram it is necessary to have some idea of the laws which govern the flow of electric currents in solid conductors.

When a constant difference of potential is produced in a thin conducting sheet of infinite extent, the potential (V_1) of any point of that sheet is determined according to the following equation: $v_1 = \frac{Q}{2\pi dk} \log_e \frac{r_1}{r_2}$ in which Q is the quantity of electricity flowing in unit time, d the thickness of the sheet, k the conductivity of the material of which the sheet is composed, and r_1 and r_2 the distances of the point from the sink and the source respectively. In dealing with the difference in potential between two points at a given instant the expression $\frac{Q}{2\pi dk}$ may be replaced by k . Then

¹ Electrokinetics, *Ency. Brit.*, 11th Ed., ix, 216.