It may be added that it is possible, by making certain assumptions, as in the case of CO_2 discussed by Osterhout and Dorcas[4] to account for the relation between the inside and outside concentrations of dye on the basis of Donnan equilibrium. But in that case we might expect the rate of penetration to increase as the percent of dissociation increases, which is contrary to observation.

The high temperature coefficient (about 4.8) indicates that the dye does not pass in by simple diffusion but combines chemically with some constituent of the protoplasm on its way into the vacuole: or if it passes in by simple diffusion the process is complicated by other factors.

It is evident that the accumulation of dye may be explained on the basis of dissociation as set forth in the present paper or on other grounds as described in previous papers. Which of these explanations is correct may remain for the present an open question.

2919

Successive changes in the electrocardiogram following acute coronary artery occlusion.

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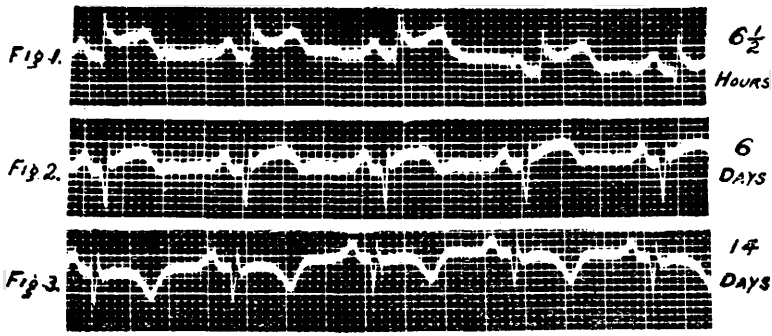
By the use of the portable electrocardiograph[1] it has been possible to secure a series of electrocardiograms in four patients soon after the onset of symptoms diagnostic of coronary artery occlusion. As none of these patients died, there was no opportunity to confirm the diagnosis by a pathological examination of the coronary arteries, but the diagnosis is justified by considerable experience in similar cases in which one or two characteristic electrocardiograms were obtained, which also presented typical symptoms and associated phenomena, and in which necropsy con-

[4] Osterhout, W. J. V., and Dorcas, M. J., *J. Gen. Physiol.*, 1925-26, ix, 255.

[1] Mann, H., PROC. SOC. EXP. BIOL. AND MED., 1925, xxiii, 19.

firmed the diagnosis of acute coronary artery closure. The subsequent course in these four patients was also consistent with the original diagnosis.

In general, the electrocardiographic changes may be divided into two stages. The first stage, which was seen as early as 6½ hours after the onset of original pain, consists of a well-defined deviation from the normal electrocardiogram, namely, the R-T transition, is abnormally elevated above the base line. In other words, after the completion of the R wave, the curve fails to return to the base line until the completion of the T wave. This is illustrated in Fig. 1. Indications of the electrocardiographic



Electrocardiograms taken shortly after acute coronary occlusion. The upper electrocardiogram shows lead 2 as recorded 6½ hours after the acute onset of symptoms. This curve exhibits the characteristics of the first stage. The middle electrocardiogram shows lead 2, six days after the onset. The lower curve shows lead 2 fourteen days after the onset. This curve shows the characteristic features of the second stage.

second stage appeared as early as 32 hours after the onset of clinical symptoms, but the change was not fully developed until the lapse of at least 12 days in one instance, and of 2 or more weeks in the others. The second stage (illustrated in Fig. 3) consists of a change in the T wave. The T wave is more definitely separated from R, due to the fact that the R-T transition approaches the base line; the T wave may be inverted, and assumes a characteristic form; *i. e.*, the first limb is curved, the apex peaked and the second limb is rather straight. This we have called a "coveplane" T. Pardee[2] has described one case in which the electrocardiograms presented these two stages. After an interval of

[2] Pardee, H. E. B., *Arch. Int. Med.*, 1920, xxvi, 244.

6-8 weeks from the onset of the attack, the T wave may lose its characteristic shape and approach the normal. From our experience in other cases of coronary artery occlusion, we know that the cove-plane T may persist for years. On the other hand, we have seen no example of long duration of the changes of the first stage, *i. e.*, marked elevation of the R-T transition. The general similarity of the successive changes in the four cases cannot be regarded as accidental. Therefore, it may be said that the early electrocardiographic changes associated with acute coronary artery occlusion are first seen in the R-T interval, and then in the T wave itself.

2920

Lactic acid of normal and pathological spinal fluids.

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In 1917 Levinson[1] observed that spinal fluids from cases of epidemic meningitis immediately after withdrawal had a subnormal pH. This was in some instances further decreased when the fluids were permitted to stand at room temperature unstoppered. However, normal and tuberculous meningitic fluids when freshly drawn showed similar figures for pH, and the pH increased on standing, particularly in the tuberculous fluids. Levinson believed that the lowering of the pH of the spinal fluid in meningococcus meningitis was due to the accumulation of lactic acid in the fluid, but he presented no data to substantiate this explanation.

In the present communication, data are presented for the sugar and lactic acid of the spinal fluid of normals, miscellaneous pathological conditions and meningitis. Clausen's method was employed for the determination of the lactic acid, and the Folin-Wu procedure for the sugar.

Specimens of spinal fluid obtained from five normal adults, after a night's fast and rest, showed lactic acid concentration from 8 to 15 mg. per 100 cc. In twenty-one miscellaneous non-menin-

[1] Levinson, A., *J. Infect. Dis.*, 1917, xxi, 556.