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.

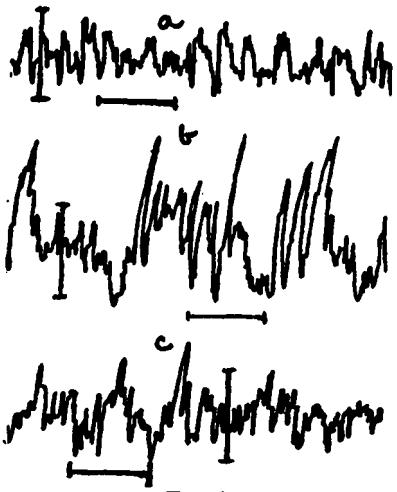

FIG. 1.

a. Normal brain.
b. Avitaminous brain.
c. Same brain 2 hours after intramuscular injection of 2.5 mg thiamine.
Time—1 sec. Amplitude—100μV.

orally, or both. At the conclusion of the experiment, the scalp was sewn together. Despite the absence of aseptic technique no sepsis resulted and repeated records could be obtained from one bird.

Normal brain potentials (Fig. 1a). A predominant 10-25 per sec. rhythm of 10-30μV is usually superimposed on one of 2-5 per sec. and 80-100μV. A 35-50 per sec. rhythm of less than 10μV is often superimposed on both of the preceding ones. Occasional "spike-like" potentials of 100μV and more interrupt the slower waves. The several frequencies are not very regular, individually or in their sequence.

Potentials from the avitaminous brain (Fig. 1b) are more regular than in the normal. The fastest rhythm is unchanged or decreased, the slower ones are increased in amplitude by as much as 100%, and more and larger spikes appear.

Administration of vitamin B_1 does not influence potentials of the normal brain. In avitaminous pigeons, deficiency symptoms disappear within 2 hours of its injection, paralleled by a reversion of potentials toward normal (Fig. 1c). Brain potential records were controlled for muscle, eye and other stray potentials.

Although a definite change in brain potentials can be observed when the normal pigeon becomes avitaminous and this is reversed by thiamine administration, it is too variable in detail to permit

quantitative analysis. O'Brien and Peters[3] showed that oxygen consumption is moderately reduced in the avitaminous brain coupled with impairment of carbohydrate metabolism. This parallelism to insulin shock is further shown by the changes in brain potentials recorded here and, under insulin, by Dubner and Gerard,[4] Himwich *et al.*,[5] and others.

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Further Experience With the Use of Thrombin as a Hemostatic Agent.*

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We have recently reported animal experiments[1] on the use of purified thrombin[2] as a hemostatic agent. We are now able to obtain thrombin solutions free of bacteria, and with minor loss of activity, by use of fritted glass filters.[3] We wish to report additional toxicity experiments; also preliminary experience with the use of thrombin in a small group of human cases.

Toxicity Experiments. As pointed out previously,[1] thrombin, applied to operative surfaces, is non-toxic. Numerous other toxicity experiments with thrombin have since been performed, and a small group of these is presented below.

(1) Intramuscular administration. An injection of 6000 units into the thigh muscles of an adult rat produced no local thrombosis, and no clinical evidence of toxicity. Blood samples drawn several

3 O'Brien, J. R., and Peters, R. A., *J. Physiol.*, 1935, **85**, 454.

4 Dubner, H., and Gerard, R. W., *J. Neurophysiol.*, 1939, **2**, 142.

5 Himwich, H. E., Hadidian, Z., Fazekas, J. F., and Hoaglund, H., *Am. J. Physiol.*, 1939, **125**, 578.

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1 Seegers, W. H., Warner, E. D., Brinkhous, K. M., and Smith, H. P., *Science*, 1939, **89**, 86.

2 Seegers, W. H., Brinkhous, K. M., Smith, H. P., and Warner, E. D., *J. Biol. Chem.*, 1938, **126**, 91.

3 Morton, Harry E., and Czarnetzky, E. J., *J. Bact.*, 1937, **34**, 461.