gen content has entered the lipide extract. Cirrhosis affects the lipide content so that it increases above normal but not as high as the normal fasted liver cytoplasm. The phospholipide in the cirrhotic liver decreases, but here also there seems to be a lipide present with a high nitrogen content.

The iodine value of both the normal fasted and the cirrhotic-fasted liver cytoplasms decreased below that of normal. These results are in accord with those of Winter[12] in that he found that after feeding carbon tetrachloride to rats the iodine value of the liver fatty acids decreased from 111 to 100.

Summary and discussion. Chemical changes in the cytoplasm of the mouse liver cell after a short fast and at cirrhosis are reported. These changes occur most dramatically in the lipide fraction during a 24 hour fast resulting in an increase of 3 to 4 fold above normal. This lipide is non-phospholipide in character. Fasted cirrhotic liver differs from the fasted normal liver cytoplasm in that the lipide does not increase so markedly and the phospholipide decreases to about 60% of the normal fasted value. The iodine values of the normal-fasted and the cirrhotic-fasted liver cytoplasm fatty acids decrease below that of the normal values.

In addition to a lipide increase in the fasted-cirrhotic liver cytoplasm there is also a decrease of "ribonucleic acid" and phospholipide in one gram wet weight of the ground fasted cirrhotic liver.

It is possible that there is an interchange of phospholipide during a fast as evidenced by the lipide nitrogen to phosphorus ratio. This interchange would consist of substituting normal phospholipide with a lipide or phospholipide which contains a great deal of nitrogen.

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Electrical Alternation in Experimental Coronary Artery Occlusion.

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The purpose of this preliminary note is to report a high incidence of electrical alternation in experimental coronary artery occlusion.

Methods. In open-chested nembutalized dogs,[1] the left anterior descending coronary artery was occluded for periods of 30 seconds to 32 minutes. Electrocardiographic tracings were made with the exploring electrodes in the cavity of the left ventricle and on the epicardial surface or precordium. Thirty-three experiments were performed on 11 dogs.

Results. Electrical alternation occurred in 8 of 9 dogs (89%) which developed electrical signs of myocardial ischemia[2] following coronary artery occlusion. One dog died of ventricular fibrillation, and one dog failed to develop any electrical signs of ischemia following occlusion for 32 minutes. Electrical alternation occurred about 2 minutes after occlusion, was variable in duration (several seconds to 20 minutes), and disappeared either spontaneously or within 3 seconds to 5 minutes after release of the constriction. Alternation of the ST-T and QRS complexes, alone or together, was observed, the most common being of the ST segment (Fig. 1). There was alternation in the extent of ST elevation in the epicardial leads, and of ST segment elevation or depres-

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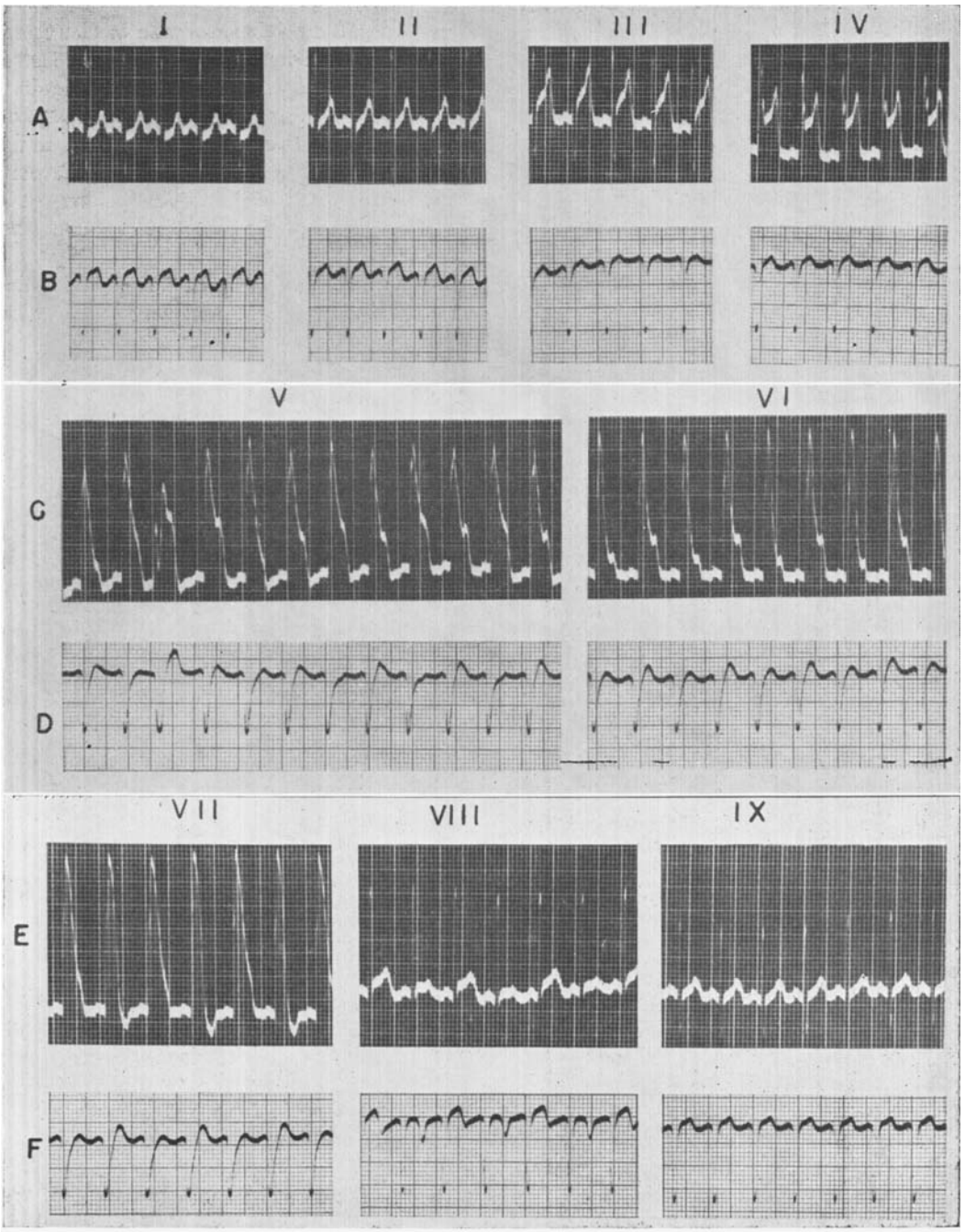

FIG. 1.

Electrical alternation following coronary artery ligation. Rows A, C, and E are epicardial records, and B, D, and F intracavitary records from left ventricle. Column 1 is control. Columns 2, 3, 4, 5, 6, 7, and 8 are 25, 55, 105, 243, 253, and 293 seconds after occlusion of left anterior descending artery. Note T wave changes precede ST deviation. Alternation is more marked after appearance of a premature ventricular beat (Column 5). ST-T alternation in Columns 5, 6, and 8. T wave alternates in direction in cavity lead in Column 8. Column 9 shows recovery 7 minutes after release of occlusion.

sion in intracavitary leads. Alternation of the QRS complex showed variation of less than 10% in QRS areas between large and small beats. Areas of the ST-T complexes varied on an average of 40% between beats.

Discussion. Fundamentally, the factor underlying all forms of alternation is a marked prolongation of the refractory phase of some part of the heart leading to alternating localized block.[3,4] In Wiggers' laboratory, Orias[5] and Tennant[6] showed that masses of cardiac fibers are deleted from contracting as a result of ischemia following coronary artery occlusion. Green[7] demonstrated that this deletion may occur alternately, *viz.*, mechanical alternation occurred after experimental coronary artery occlusion with the area of ischemia bulging alternately, and producing an alternation of aortic pressure. Similarly, we may assume that electrical alternation of injury effects seen in our experiments is on the same basis, *i.e.*, a failure of certain fractions of ischemic myocardium to respond on alternate beats.

Alternation was predominantly of the ST-T complex, probably because injury currents (of rest and of activity)[1] persist longer than the transient initial changes of depolarization and repolarization in early acute myocardial ischemia.[2] Alternation in the direction of the T wave implies alternation in the epicardial-endocardial order of repolarization, while alternation of the magnitude of the T wave implies alternation of the rate or intensity but not of the direction of repolarization.[8]

Previous temporary occlusions predisposed the myocardium to the production of alternation. The etiological significance of ventricular premature beats remains unsettled since alternation occurred with or without preceding ventricular premature beats. Heart rate was not significantly increased, so that electrical alternation cannot be explained on this basis.[9]

Summary. Electrical alternation developed in 8 of 9 dogs (89%) which survived coronary artery occlusion and showed electrocardiographic signs of myocardial ischemia. Electrical alternation occurred within 2 to 3 minutes after occlusion and was transient. Repeated temporary occlusions predisposed to the development of electrical alternation.

Alternation was predominantly of the ST-T complex, although less marked alternation of the QRS complex and of the T wave also occurred.

It is postulated that in our experiments, electrical alternation of injury effects is due to the failure of certain fractions of ischemic myocardium to respond on alternate beats.

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