days and a half. Characteristic anæmic infarcts were found occupying the anterior part of the septum, and that part of the anterior wall of the left ventricle which adjoins the interventricular septum.

W. T. Porter* has an interesting series of experiments on dogs on closure of the coronary arteries, and comes to the conclusion that the "frequency of arrest is in proportion to the size of the artery ligated."

In the human heart blocking of one of the branches of the coronary arteries by an embolus or by thrombosis may cause sudden death, and the frequency would be in proportion to the size of the vessel affected, other things being equal. In thrombosis the rate of closure would have an influence. That sudden death can occur is of special importance in medico-legal cases, as it happens rather frequently from this cause.

Should sudden death not occur we have the condition known as anæmic necrosis or white infarct as a result. The affected areas, when recent, are firm and appear only as dull, yellowish discolorations of the heart muscle; after a time they become softened and friable and assume a yellowish-white tint. They are generally irregular in outline and are at first raised above the surface, although later they may be retracted. The pericardium over it may be covered with a grayish membrane, and if the area extends to the endocardium, we may find thrombi adhering to the heart wall corresponding to the area affected.

A red or hæmorrhagic infarct is present when an extravasation of blood takes place from the capillaries, and the appearance and subsequent processes differ only to the extent of the extravasation.

Microscopically the muscle cells are fragmented and granular in appearance, the nuclei and striæ disappear and sometimes there is some formation of oil globules. The tissue is soft and easily torn.

Should this condition affect a considerable portion or the entire thickness of the wall, the result might be a rupture of the heart and consequent, more or less sudden, death.

When this process of anæmic necrosis has proceeded a certain length and death does not ensue, we have a process of repair set up. This consists of the removal of the detritus followed by increase in connective tissue and the gradual formation of the condition known as fibrous myocarditis. Here the original anæmic infarct is converted into fibrous tissue with contractions of this tissue, showing its position. The muscular elements are never regenerated.

Fibrous myocarditis may thus result by the gradual transforma-

*Journal of Experimental Medicine, January, 1896.