Her highest temperature was 101°F; her pulse varied from 128 to 148; the cardiac dulness was normal. There were no adventitious sounds within heart or lungs. The patient re-acted fairly well to antitoxin, the laryngeal stridor disappearing within twelve hours. On the afternoon of the third day of the disease, however, the patient suddenly collapsed, the pupils became widely dilated, the heart action very rapid, the sounds indistinct. The breathing remained regular until death took place about twenty minutes after the beginning of the collapse. Microscopic examination of the heart showed absolutely nothing pathological in that organ. The liver, the lungs, and the spleen were normal beyond a certain amount of injection.

Case II.—G. W., male aged 19 years, was admitted to the hospital on the third day of a very severe attack of scarlet fever. His temperature while in the hospital varied from 103° to 105° F, his pulse from 140 to a rate uncountable. The notes on admission stated that he was a well nourished muscular adult with anxious facial expression; his pupils were equal and active; his conjunctivae much injected; the lips abnormally pale. A most intense dusky red rash covered the body. The breath sounds throughout both lungs were normal but the respiration was of a sighing character. The pulse was rapid, of small volume and low tension, and very shallow. Cardiac dulness was normal in extent, but the heart sounds were feeble. No adventitious sounds were present; he responded feebly to a normal saline injection and collapsed a few hours afterwards, during the administration of a second. The autopsy findings as to the cause of death were most unsatisfactory. Nothing definite could be found either macroscopically or microscopically leading to a fatal result.

If questioned as to the exact pathology of these suddenly fatal cases, the general answer would be that the heart muscle, as the result of the action of certain toxins, has undergone degenerative or inflammatory changes which so impaired its functional ability that it was unable to meet the demand made upon its ventricles by some slight extra strain. Such an explanation is plausible, but what does the post mortem show? A striking contrast between the empty heart chambers of such a death and the engorged auricles and right ventricle of true heart failure. Microscopically we find only a slight change in the muscle cells; rarely any significant inflammatory lesions. Looking back on the clinical history of such a case we note also the absence of all ordinary signs of a failing heart. No œdema, no venous stasis, no cyanosis, but extreme prostration; a blanched cool skin, and a rapid ineffectual heart beat.