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 shabby. 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, had 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. The stage of collapse thus resembles the condition present after a severe hæmorrhage, or in surgical shock.

This striking absence of all ordinary signs of heart muscle failure has made investigators question whether, after all, the heart is really involved in the circulatory failure. Romburg and several of his pupils in the Leipsic clinic, notably Pässler, have recently investigated this problem very carefully, and published their results in a series of papers. At the outset they studied the mode of death in animals inoculated with one of the following infections: the pneumococcus, the bacillus pyocyaneus and the bacillus diphtheriæ, and found that death occurred after rapidly developing symptoms of collapse similar in every way to the so-called heart failure in man.

They then repeated the inoculations in a large number of fresh animals, and observed the blood pressure at short intervals. They found that it remained normal during the major part of the illness, only beginning to fall when collapse was impending.

Blood pressure, as we know, is dependent upon four separate factors, which may vary independently of one another:

1st.—The energy of the heart.

2nd.—The peripheral resistance.

3rd.—The elasticity of the arterial walls.

4th.—The volume of the circulating blood.

The last two have little interest for us at the moment. The tone of the vessels regulating to a great extent the peripheral resistance depends upon impulses from the vaso-motor centres. Experiments show conclusively that this vaso-motor tone is an absolute necessity for the maintenance of the circulation, not only in the arteries, but also in the veins. Any grave injury to the vaso motor center is followed by stagnation of the blood in the veins, and eventual heart failure because no blood is returned to it. To determine what factor was the