nized, but it is still questionable whether the influence of poisonous matter on the coagulability of the blood in the living vessels has received that care and attention which its importance demands.

A coagulum once formed in the vessels may either undergo a complete fibrinous organization and become like connective tissue, or it may undergo, as was shown by Mr. Gulliver, a change of structure; the central layers may become puriform in character and chiefly composed of granules. These clots may, and often do, soften, break up, and are again carried into the circulation, and thus give rise to pyœmia. It thus becomes manifest that clots, however small and insignificant, in the peripheral veins, may lead to very serious if not fatal results. The size of the capillaries they have to pass through determines the locality of the secondary deposits and abscesses. The capillaries in the liver are well known to be larger than in the lungs, hence metastatic abscesses would be produced in the lungs, because the capillaries of the liver allow the debris to pass through them, and the capillaries of the lungs arresting the progress of the morbific product. This is considered by Virchow as so certain and conclusive, that he is inclined to regard all cases of metastatic abscesses of the lungs as of embolic origin. In cases of puerperal fever followed by metastatic abscesses in the lungs, he invariably discovered thrombi in the pelvic vessels. The connection formed in the veins and those found in the heart and large vessels must, from the preceding remarks, become apparent. It may be useful, for the purpose of discussion, to classify fibrinous clots into venous and arterial, and to note those clots that are associated with cardiac disease, vascular disease, or both; also those clots that are not associated with organic disease of the heart or vessels, but which appear to depend for their formation on certain antecedent pathological conditions of the blood.

1st.—The formation of fibrinous concretions associated with organic disease of the heart is easily understood, in those cases where the disease of the mitral or aortic valves interferes with the circulation of the blood, thus forming a mechanical obstruction and consequent coagulation. On this physiological process we depend when we employ pressure for the cure of aneurism. Again the detachments of fibrinous growths from the valves of the heart is a frequent cause of arterial embolism. The atheromatous condition of the vessels, as it roughens the inner coat and obstructs the circulation, has a tendency to separate the fibrin from the serum of the blood, and the result is a fibrinous clot.

2nd.—Venous concretions. The formation of these concretions on the right side of the heart are frequently associated with fatty degeneration of the heart: the action of the heart is enfeebled, and consequently the circulation of the blood is retarded, a clot is formed. Clots are generally formed near some diseased portion of the arterial walls; there may be either atheromata or local inflammatory action, irritation or constriction of a vessel; but, when once formed, the clot may become detached and carried onwards in the circulation to some part of the vessel perfectly healthy. When a clot is thus discovered it was evidently not formed *in situ*.

The next group of cases in which I have found fibrinous concretions are those in which I failed, after most careful examination, to discover any organic disease of the heart or blood vessels. Consequently, I surmise that their formation was due to some antecedent pathological conditions of the blood. These cases suggest some very valuable and practical thoughts on the treatment of disease. The subjects of fibrinous concretions not associated with heart disease, nor disease of the vessels, nor traumatic injuries, were all children. The following cases have come under my observation, and a record of them may not be devoid of interest to the busy practitioner.

Case 1.—A. E., the child of Mrs. E., aged 18 months, was attacked with scarlet fever, malignant sore throat, nephritis, dropsy, albumen in urine, and casts. The case went on and was apparently doing well, except that it continued weakly and was having a tedious convalescence. There was nothing marked or unusual in the condition of the child until five weeks after the attack, when the child was suddenly taken ill about two o'clock in the morning. The father, an intelligent man, noticed that his child's heart was beating violently, and that it became suddenly pale and in a cold sweat, with twitching of the arms and legs. In a word, it was in convulsions, and died before my arrival. Now, what was the cause of death? The ready answer would be convulsions after and depen-