chemical properties as the albuminous substance which enters so largely into the composition of red corpuscles. When treated with chemical re-agents, though they may produce no appreciable effect on its chemical composition, nevertheless it loses its peculiar power of causing serous fluids to coagulate. Hence, though there is great reason to believe that the fibroplastic globulin which exists in the serum does really come from the red corpuscles, the globulin which is obtained in large quantities from these bodies by the use of powerful re-agents has no coagulating effect on the pericardial or other serous fluids.

Though globulin is so unsusceptible of change when in solution, it may be dried at a low temperature and kept in the form of powder for many months without losing its coagulating power. This globulin added, under proper conditions, to serous effusion, is a coagulator of that effusion, giving rise to the development of fibrin in it. This it does by its interaction with a substance contained in the serous effusion, which can be extracted by itself, and then plays just the same part towards a solution of globulin as globulin does towards its solution. This substance, fibrinogen, is exceedingly like globulin, and may be thrown down from serous exudation by carbonic acid, just as globulin may be precipitated from the serum of the blood. Thus it would clearly appear that the coagulation of the blood and the formation of fibrin are caused primarily by the interaction of these two substances, viz., globulin and fibrinogen—the globulin existing in the serum of the blood and some other tissues, whilst the fibrin often exists in the plasma of the blood, lymph and chyle.

The preliminary considerations of these physiological conditions of coagulation of the blood may possibly enable us the better to appreciate the pathological conditions antecedent to the formation of fibrinous concretions. If an excess of alkali or the presence of acids possess the power of destroying the coagulating properties of globulin, and, consequently, of arresting the formation of fibrinous concretions, what a valuable fact is discovered!

In discussing fibrinous concretions in the heart and large vessels, it may not unreasonably be asked, why not follow the nomenclature of Virchow, and speak of thrombus and embolus. The reason is simply that fibrinous concretions is an accurate description of the actual condition found on post mortem examination, and does not commit me to the etiology of the clot. Whereas thrombus, *i. e.*, a clot, and the process thrombosis, by which the clot becomes clotted, and embolus, a projected coagulum detached from the walls or valves of vessels and carried into the circulation, does imply a theory and knowledge of the formation of these concretions. The records of concretions in the heart, arteries and veins are but of recent date, nevertheless, they are exceedingly interesting and instructive. To the busy practitioner as well as to the physiologist and pathologist, do these conditions demand the most searching investigation. The practical significance of this condition is at once recognised when we associate it with the various diseased conditions of the different organs in the body. Nor is this all, certain local conditions of pressure and irritation have resulted in the formation of concretions in the vessels, and they, in turn, have become the chief factors in the production of pyæmia, or gangrene, or even softening of the brain. Dr. Aitkin says, he has never traced a case of phlebitis or pyæmia without discovering that the affection essentially begins by a real coagulation of the blood at some fixed point. When this beginning is discovered it is exceedingly significant as pointing to some source of local irritation, which, by simple disturbance of the flow of blood in some way, determines the formation of a clot. M. Ribes describes several cases illustrative of the formation of clots. In one case chilblains was the starting point, clots formed in the veins, proceeded to the superior veni cava, into the right auricle and ventricle, and thus causing death.

There are, doubtless, many proximate causes tending to the formation of clots in the veins, and that they occur much more frequently in our practice than is generally supposed. The more attention is directed to this subject, the more obvious will be the common cause of sudden death revealed in cases of wounds, fractures and operations.

The conditions favorable to the formation of clots in vessels are to be found where veins open upon fœtid ulcers or where noxious gases are readily absorbed. These conditions tend to alter the fluidity of the blood. The poisoning of the blood by septic matter has long been recog-