injury or from the amputation. Among the causes of necræmia, German pathologists mention violent convulsions, overwhelming emotions, the shock from an amputation, a stroke of lightning, and even a severe exhausting labor. The shock then, was quite sufficient to account for the death of the blood. This being admitted, we can readily understand how gas may be generated by the decomposing blood, and thus account for its accumulation in the bloodvessels. It is not so easy, however, to account for the occurence of piarhæmia.

We are all aware of the physiological piarhæmia, the result of digestion, pregnancy, lactation, and hybernation. About two hours after the ingestion of ailment the serum is found to be turbid, opalescent, and semi-opaque, a transitory condition which is due to the absorption of the fatty matters of the food formed into an emulsion by the pancreatic juice, and absorbed as such in the duodenum. The microscope shows this condition to be due to the presence of a large number of fat globules and of molecular granules of albumen. According to Christison, the passage of the chyle into the blood renders the serum turbid; this turbidity lasting until the insoluble fatty matters, oleine, stearine, and margarine, enter into combination with the free soda of the blood, and become converted into oleic, stearic, and margaric acids. That the case under consideration was not a case of *physiological piarhæmia* is evident from the fact of the patient having taken very little food for some time, as well as from the absence of the peculiar lactescent appearance of the serum usual in such cases. There is, however, a *pathological piarhæmia*, the result of certain diseases. It has been noted in diabetes, chronic alcoholism, dropsy, jaundice, nephritis, hepatitis, pneumonia, and especially Bright's disease.

Various explanations have been given of the occurrence of fatty blood in disease. Dr. Babington regards piarhæmia as a fatty degeneration of the albumen of the blood. Robitansky thinks it is often due to fatty degeneration of the colorless corpuscles, which are previously formed in excess, so that it is to be regarded as a modification of leucocythemia; but he also admits the direct introduction of fat into the blood, and the liberation of combined fat contained in it to be possible causes. Virchow regards it as dependant upon the non-combustion of fat and its consequent accumulation in the blood; while he considers the presence of molecular albumen to be only a secondary phenomenon, the slow saponification of the excess of fat abstracting from the albumen of the blood the alkali required to keep the latter in solution. These explanations are all plausible, and may each be applicable in some instances; but in the case before us, the microscopical examination would not warrant us in supposing that