

the administration of various iron preparations, it was argued that no iron was absorbed. The weak point of this reasoning is of interest. The intestinal mucuous membrane is the place of excretion for the iron and not the urine, which is capable of removing only a few milligrammes. Iron which gets into the blood-current accumulates in the liver and spleen, these organs, magnet-like, attracting it. They are the warehouse of the body for superabundant iron, which is given off very gradually by these organs, and is removed by the intestinal juice from the body. How do the iron salts which are absorbed act as remedies? Is the chlorotic organism wanting in atoms of iron around which the hæmoglobin molecule can be built up? Every girl developing chlorosis takes up in daily food more iron than the most extravagant estimate demands. But this iron is exclusively contained in nucleo-albumins. There is no doubt that the healthy organism takes up iron from nucleo-albumins and employs it for the manufacture of hæmoglobin. Why does not the chlorotic patient do the same? Is absorption at fault? On the contrary, absorption is excellent. Iron is absorbed but is not used. There is a diminution of the energy of growth in the hæmatopoietic organs (bone marrow), resulting in the non-utilization of the ferruginous nucleo-albumins which are brought by the blood. So far everything is proved. Now for the region of hypothesis. Let us suppose that the iron salts circulating in the blood effect a stimulation of the hæmatopoietic cells of the medulla of bones, and that the result of this stimulus is an amelioration of the constitution of the blood. On the other hand, the nucleo-albumins containing iron which are circulating in the blood exert but a weak stimulus on bone marrow. Experience proves that in the treatment of chlorosis any iron salt is more efficacious than the ferruginous nucleo-albumins, like hæmoglobin, hæmotogin, hæmogallol, etc., which pure theorists have tried to foist on the profession. Arsenic gives good results. Yet success is much greater when combining arsenic and iron. Practical experience proves this is the best plan. At health resorts, where there are carbonated ferruginous springs, resident physicians do not recommend these waters to chlorotic subjects, but advise the mineral waters of Levico or Roncegno, which contain arsenic and iron.

These substances are closely allied chemically, and exert on the cells an effect that is only quantitatively different.—*Medical Record*.

Rheumatic Angina. *Roos (Berl.klin. Woch.)* first relates cases showing the relation between rheumatism and this angina. In the English writings this relation is generally recognised. Rheumatic angina may be a prodromal symptom of acute rheumatism. This latter disease is in all probability an infective process, and the author thinks that the actual cause of the disease may gain access through the faucial mucous membrane. Rheumatic angina is, however, said not to be very common. Pain on swallowing, swelling and redness of the fauces, especially of one or both tonsils, with or without suppuration, the occasional presence of a purulent deposit on the tonsils, and the relatively long duration of the affection are to be noted. Former rheumatism will assist in the diagnosis. This rheumatic angina will probably explain the presence of valvular lesions in some cases in which there is no history of rheumatism. Correct diagnosis is important, as the salicylates rapidly cut short the disease, and perhaps prevent the articular affection. Rheumatic angina cannot at present be distinguished from other forms of angina. Articular rheumatism may appear after the follicular form. It is possible that after certain anginas the infection may involve the joints (polyarthritis anginosa). This polyarthritis after angina sometimes differs from rheumatism by reacting badly to the salicylates, and by its long duration. In this it resembles the polyarthritis seen after scarlet fever and diphtheria. The author thinks that a number of these cases of polyarthritis after angina will resolve themselves into examples of infective processes when the etiology is better known. He refers to cases in which etiologically the disease should be looked upon as pyæmic, and yet no suppuration occurs in the affected joints. Here the micro-organisms may have lost their virulence (modified pyæmia). It appears strange that after suppuration in a tonsil a simple polyarthritis should occur. The author then refers to the researches of Sahli in respect to the causation of rheumatism by the staphylococcus. (*Epitome*, October 22nd, 1892, par. 343). He concludes that either the angina preceding the polyarthritis