tion, and it is mainly upon this feature that any question of differential diagnosis rests.

Concerning the clinical picture of the two maladies, we find in each an identical classification into types; in both a chronic form is described and in both a more acute, characterized often by the presence of irregular fever, early enlargement of glands, early onset of hæmorrhages and a rapidly progressive lethal termination. Such is Ebstein's (3) case of acute Hodgkin's disease, in which, except for the condition of the blood, all the essential features of the case were identical with those of an acute leukæmia. Such, too, appears to be the reason why authors occasionally speak of a lymphatic Hodgkin's disease where a lymphocytic leucocytosis occurs of too moderate a degree to warrant the application of the term leukæmia.

In the more chronic forms of these two diseases there is often a different mode of onset of the lymphoid enlargements, inasmuch as in Hodgkin's disease it is the glands, while in leukæmia it is the spleen that is first affected. Yet such a distinction is far from being absolute. Cases of Hodgkin's disease have been observed in which the spleen alone has been affected, while other lymphoid structures remained quite normal, and we have seen one such case in the hospital here in which, from general physical conditions as well as from examination of the blood, it was impossible to make any other diagnosis than that of a splenic form of Hodgkin's disease.

Whether or not we are justified in considering the two diseases as distinct from any other clinical conditions apart from the blood examination we are unable to say. Eichhorst (4) suggests that an essential difference between the two diseases may be observed in the urine; that in leukæmia there is an excess of uric acid, while in Hodgkin's disease he has never been able to discover any such condition. Considering, however, the frequency with which diseased blood conditions are attended with superabundance of uric acid in the urine, it would seem that an altered metabolism which induces the leucocytosis might likewise explain the presence of excessive uric acid in the urine, and that the condition may be associated in some way with the altered blood condition. That such a process does indeed occur has been proved experimentally by Kühnau (5) who has shown that an excessive leucocytosis is invariably associated with an increased production of uric acid. In this case, then, such a