shaped, neutrophilic leucocytes, 1 per cent segmented nucleated leucocytes, 55.4 per cent lymphocytes, 2 per cent mononuclears, 0.4 per cent basophiles, and 1.1 per cent atypical cells. The oxydase reaction was positive in all cells. However, the cells diagnosed as lymphocytes were probably oxydase negative myelocytes. The individual died with a diagnosis of micromyeloblastic leukemia. The autopsy revealed an atrophy of the spleen with a few scattered myeloid cells in the pulp, a liver without any myeloid infiltrations, which, on the other hand, existed in the kidney and chorioid plexus. The bone marrow contained many plasma cells, micromyeloblasts, promyelocytes and myelocytes, foam cells of plasma cell origin, and scanty erythroblasts. The clinical diagnosis of leukemia was modified on the basis of the post-mortem findings. This condition, a striking illustration of the great difficulties which may be encountered in the differentiation between a leukemia and a leukemoid reaction after occupational exposure to roentgen-rays, was interpreted as the result of a severe injury to the bone marrow with attempted myeloid repair and scanty myeloid heterotopic metaplasia.

It is fortunate that the observations made by Stewart on four chemists employed in studios in Pennsylvania and suffering from radium poisoning provide additional evidence, supporting the actinic origin of such conditions and showing the potential leukemic outcome of these reactions. Two of the four chemists developed, as the result of a long prolonged occupational exposure to radioactive substances, cutaneous malignancies, which in one case was complicated by a severe radiation anemia of a pernicious type. A third chemist displayed a blood disorder which exhibited at varying times a tendency to an anemic type or a leukemic variety. This individual was apparently in a transitional stage, in which the ultimate outcome of the hematic occupational injury was still in the balance. Such a final turn had been made in the fourth chemist belonging to this group. He was an individual, 39 years old, who had been exposed to radioactive material from 1912 to 1925, and who had been engaged in the manufacture of luminous dial paint until 1927. After having passed through a stage of leukopenia, he died from a myeloblastic leukemia.

The apparent rarity of such intermediary blood reactions is in sharp contrast to the considerable number of leukemias reported, for which causative relations were claimed concerning an occupational exposure to the actinic agents. There are, so far, twenty-one such cases on record, eighteen of which occurred in males and three in females (v. Jagič, Schwarz and v. Siebenrock; Carman and Miller; Davey and Whitby; Evans and Roberts; Weil and Lacassagne; Aubertin; Haagensen; Nielsen; Beclère; Loewy; Vaquez; Laubry and Marchal; Teleky; Weitz; and Stewart). The evidence offered in support of the alleged etiological connection with roentgen-rays and radioactive substances, respectively, is in a considerable number of these cases quite defective. In three of the four cases recorded by v. Jagič, Schwarz and v. Siebenrock, the