Progress while in Ward.—The oedema and enlargement of liver and spleen disappeared entirely and the cyanosis was less marked, but on the other hand his gait remained the same and he left the hospital on June 30th without any improvement in the Raynaud phenomena.

Since leaving the hospital the patient did not improve and Dr. Gibson, who saw him with Dr. Menzies, his family doctor, reports that while the oedema had reappeared, possibly in great measure due to his cardiac dilatation, he had much more pain in hands and feet and more definite evidence of the development of a neuritis in the nerves of the affected limbs. Dr. Gibson noted that there was marked dropped-foot, feebleness of all the muscles of the arms and legs and loss of tendon reflexes. The final stage was reached when the patient became the victim of cardiac failure, with general anasarca, ascites and hydrothorax and soon succumbed.

There was no gangrene in Case 1 and therefore the vascular and nerve changes cannot be referred to a secondary cause. Whether Raynaud would have accepted any cases with neuritis has been many times discussed. There is no satisfactory reason for separating such cases and it seems highly probable that neuritis, if not extremely definite may be missed by the worker who is not experienced in interpreting nerve sections unless some method such as the Marchi is used.

The nerves were fixed in formaline and Muller's fluid cut in paraffin and celloidin and stained by haematoxylin and benzo-purpurin and other reagent.

The cord sections were cut in paraffin and stained for nerve cells as well as for nerve fibres and vessels. Toluidin blue and polychrome blue and eosin were used for the nerve cells.

I am indebted, as already indicated, to Dr. Gibson for permission to publish these cases, and also to the resident physicians, whose clinical records of the cases I have used. Dr. Dalmahoy Allan is responsible for the notes of Case 1 and Dr. R. W. Johnstone for Case 2.