pyramidal system as the most striking symptoms, developed in this patient after a latency of approximately one week.

CASE 3.—A 30 year old stoker, who had been in good health and had had no signs of epilepsy or other nerve diseases, was found in a narrow room, where, as proved by an investigation after the accident, there was a 0.023 per cent content of hydrogen sulfide in the atmosphere, due to leakage from a drainpipe. It could be established that the patient had been in that room for at least 20 minutes. When he was found, he had cramps in his right arm and froth about his mouth and was unconscious. Breathing was shallow and slow; the pulse, regular, with about 60 beats per minute. As soon as he had been taken out of the room, the cramps subsided and he became highly restless. After a while, however, he lay in profound sleep for a few hours. Data concerning pupil reactions, etc., are lacking.

He was unconscious during the first examination after his arrival at the hospital, but he had no other nerve symptoms. He showed signs of a circulatory crisis, with a blood pressure of 80/50 and a pulse rate of 60. The heart sounds and the electrocardiogram were normal. The day after his arrival the patient suddenly went into shock and recovered only after heavy stimulation. However, the systolic blood pressure did not rise above 100 until after three days despite persistent stimulation. He left the hospital six days after his arrival, fully recovered and with a blood pressure of 120/80. He was under observation over a period of two years with no recurrence of pertinent symptoms.

This case differs from the others because the time of exposure was considerably longer (more than 20 minutes) and the concentration of hydrogen sulfide in air lower (about 0.025 per cent). It also differs in the clinical picture, which shows initial cramps, possibly of an epileptic type, and a collapse of the circulatory system. There were, however, no signs of damage of the brain after the acute symptoms had subsided.

The difference may possibly be explained in agreement with Haggard's experimental observation that hydrogen sulfide in low concentrations has a stimulating effect and in high concentrations a paralytic effect on the central nervous system. In the cases with short but intense exposure hydrogen sulfide has a toxic-depressive effect on the central nervous system (respiration center, basal ganglions, etc.) with more or less permanent damage as a result. No signs of general anoxia developed in these cases. In the case with low but lengthy exposure, however, hydrogen sulfide primarily has a stimulating effect on the respiratory tract, with accompanying acapnia, apnea and asphyxia, which are decreased only after the carbon dioxide tension has risen again. This is repeated as long as the patient remains in the gas-containing atmosphere. The result will be a protracted hypoxia with peripheral circulatory collapse.

Sequelae have occurred even after acute poisoning without unconsciousness. In 15 cases the conditions of exposure, the first acute symptoms and the after-course have been such that the diagnosis "sequelae after acute hydrogen sulfide poisoning" has seemed justified. The majority, as in the first two cases described below, have had a history of repeated acute intoxications; after each, neurasthenic troubles have occurred with increased susceptibility to gas. All have had their work within the area *A* to *E* (chart 1) or in the refinery.

Four cases will be described below as examples, the first of which (case 4) represents the most common type. Case 5 is unique in that the patient at first refused to interrupt his work at the place exposed to gas and so came to serve as an