safe. Had one been able to demonstrate anaesthetic areas especially in the mammary region, evidences of Tactile anaesthesia or delayed anaesthesia in regard to pain. It evidently cannot be peripheral neuritis. As to the abdominal pain, microscopic examination of the water should be made. I don't remember seeing a case of bladder crisis, but I saw a case of laryngeal crisis, and during an attack so severe that the house surgeon did tracheotomy, but without relieving the condition.

Dr. Nichols—I couldn't see any other disease that would fit, except tabes.

Dr. Rorke noted the sensation in the ends of the fingers beindelayed and blunt, but couldn't find anything wrong with the sensations in the chest. Blood in the urine would rather shut off the gastric crisis.

Dr. Hughes—In my opinion it is a case of Locomotor Ataxia. The attacks were Renal Colic, though Dr. Bond failed to demonstrate a stone by his Skiagram, of course the question of a Renal Crisis has to be considered, but in my opinion it is not upheld.

Dr. Munroe showed case, male, 26 years, former health good, till two years ago when patient had severe attack of inflammatory rheumatism. In bed five weeks. Since has felt feeling around the heart, especially on exertion, palpitation and disturbance. Not incapacitated from work not entailing exertion. Well marked mitral murmur, of a peculiar character with high pitch and intensity.

Dr. Gardner—The heart is displaced outwards and to the left. The peculiar character of the murmur is a high pitch, ringing sound, which is transitory in character, occuring about the end of systole.

Dr. Manchester though the murmurs were mitral, and faint, but did not get the peculiar murmurs, while Dr. Nichols said he heard the peculiar sound referred to by Dr. Munroe, and it seemed to be at the end of systole. He thought the lesion was mitral and regurgitant.

Dr. Mackay presented a cardiac case and asked Dr. Rorke to elicit the history. Patient 47, had several attacks of inflammatory rheumatism the first, 19 years ago, affected the heart, when patient was kept in bed for six months, three months of which a pillow was not allowed. The upper valve was affected, and for two years after, the slightest exertion would cause collapse, and patient was frequently laid up during those two years. Carried brandy continuously, to which he was often forced to resort. The second attack was not so severe as the first, but affected the heart and also the joints, but the third attack did not affect the joints. Other members of the family are all healthy. Patient attributed it to getting into a damp bed in winter, while occupied as commercial traveller. His limbs, from knee down, would get purple during an attack. Pulse was remarkably slow, 38, or as low as 32 when patient was in bed, and very rarely reaches 50.

At present, the heart is considerably enlarged, and is down one inch, down to about the sixth intercostal space, a little outside the nipple line. No diastolic murmur could be found, and slight thrill, to the left of sternum. He thought it a case of aortic stenosis. The heart is not so enlarged, nor the apex hardly as variable and is heaving the left side to the chest to the extent you would expect in any degree of aortic insufficiency. Owing to the enlargement of the heart one can say it is not an accidental murmur. A second sound could be heard, which seems the closure or re-action of the aortic valve to the blood pressure. Cannot say whether slowness of heart's action is due to involvement of heart muscle or not; he doesn't seem to have any anginoid pains which would explain that.