indirectly responsible for the disintegration of the corpuscles, it will be observed that atebrin and plasmochin did not precipitate the attack, whereas quinine succeeded in bringing it about on two occasions out of three. It would appear, therefore, that the hæmoglobinuria is not dependent on quinine alone, but some other factors also play a part in the production of this condition. What these factors are remains to be worked out. Many of the cases of black-water fever met with in Bengal and Assam are of this nature and it is possible that the substitution of atebrin for quinine in the treatment of malaria will bring about a reduction in the occurrences of such cases.

A CASE OF PAROXYSMAL TACHYCARDIA AND ITS SEQUEL

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The attack.—S. L., Hindu male, aged 23, was admitted to hospital on 7th March, 1934, with the complaint of continuous palpitation of four days' duration. He gave no history of any previous disease other than occasional fever; he denied venereal infection. He had been subjected to recent severe mental strain, as in the Bihar earthquake in January his house came down and all his family except himself were killed.

Severe and continuous palpitation started while he was in the train four days previously; the discomfort was increasing, but there was no pain, no œdema anywhere, and no subjective dyspnœa; the patient was very cyanosed.

The pulse was 180 per minute, perfectly regular, and of normal volume and tension. The cardiac impulse was diffuse, and the apex beat 1 inch outside the nipple line; there was a systolic thrill and the impulse was forcible; a soft systolic murmur was audible in the mitral and tricuspid areas, and the pulmonary second sound was accentuated. Beyond his colour, which was almost blue, and some basal pulmonary congestion, there were no other positive findings.

The Wassermann test was negative.

After a day's observation, during which he was on glucose and insulin, 5 units, the pulse remaining steady between 170 and 180, he was given intravenously one injection of strophanthin 1/125th grain, and the next day three of 1/100th grain with no effect. He was then put on 5 gr. doses of quinidine thrice daily, and after the sixth dose the pulse rate fell suddenly to 90, and remained at that rate thereafter. His colour became normal, and he left hospital relieved on 23rd March. The paroxysm had lasted for about ten days.

The sequel.—On 24th September, 1934, the patient was re-admitted for pain in the left back, fever, and dyspnœa, all of some 12 days' duration. He was found to have a left pleural effusion of clear fluid, some of which was removed and replaced by air on two occasions. Very shortly he developed a pericardial rub and then an effusion; this also was aspirated, and tubercle bacilli were demonstrated in it. There were no intra-pulmonary signs. The patient ran a continued fever of low degree, and became progressively worse until his death on the 7th December, 1934.

Post-mortem findings

Lungs: Right.—Slightly emphysematous. Right side of the pericardium was adherent to the lung on the right side. The adhesions were recent. The pleural cavity contained four ounces of fluid.

Left.—Covered with thickened pleura and collapsed at its base, where a sac had formed by the collection of some fluid in between the two layers of the thickened pleura. A chain of enlarged tuberculous glands was found in the left side of the neck. This chain had continued on to the anterior mediastinum, connecting up with the enlarged tracheo-bronchial chain of glands, which were found adherent to the pericardium and also to the left auricle whose walls were practically fixed to the posterior mediastinum, probably due to a previous adhesive mediastinitis, tuberculous in origin.

Heart.—The heart with its pericardial covering was very much enlarged—the greatest dimensions (after opening the pericardial sac and withdrawing the fluid) were 8 inches by 7 inches. The sac contained 8 ounces of dirty fluid, in which flakes of fibrin were seen floating. The inner side of the parietal pericardium was studded with tubercles, while it was adherent to the right lung with recent adhesions, but on the left side it had completely fused with the pleura. No adhesions of old standing were found in between the two layers of the pericardium. The visceral pericardium was covered with flakes of fibrin and was very rough. It could be compared to a 'bread and butter heart' where butter was applied more profusely and irregularly. The heart as a whole was displaced to the right and at the same time slightly rotated to the left, so that the enlarged right ventricle had formed the apex, while the left ventricle was much smaller and formed the posterior surface of the heart. The left auricle was so adherent to the posterior mediastinum, that it could not be approached from outside. The mitral valve did not show any stenosis; the tricuspid admitted the tips of more than three fingers. The right auricle was enlarged. The blood vessels arising from the heart were covered with fibrin, but no actual strangulation by fibrous tissue was noticed around them.

Liver, spleen and kidneys.—Showed marked chronic venous congestion.

Morbid histology

Pericardium.—Showed uniform fibrosis with islets of small round cells here and there. Caseous foci were present at different places with some attempt at giant-cell formation.

Heart muscle.—Showed no fatty changes. Brown atrophy present in its early form.

Lymph gland.—Tuberculous.

Comments.—It is interesting that nine months before his death no signs of the infection from which he died were found in this patient. At that time it seemed probable that an emotional factor was the prime cause of the paroxysmal attack. This having ceased, there was no evidence of disease of the heart, a normal electrocardiogram being obtained. It is difficult to believe that, if a tuberculous focus had been the exciting cause, the normal rhythm would have been restored so easily.