COAL-GAS POISONING AND CARDIAC SEQUELÆ

BY

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The comparative infrequency of coal-gas poisoning may lead those who are not familiar with the subject to deal perfunctorily with victims, and to regard active treatment as unnecessary.

The majority of coal-gas poisoning victims in the South-Eastern region of Scotland are admitted to Edinburgh Royal Infirmary, treatment having been started en route. From a study of 272 such cases occurring during the last decade, only 0·7 per cent were found to show major cardiac sequelæ. The following case is presented, therefore, as a rarity, and substantiates the foregoing tenets.

Case Notes

Mr. F. S., aged 53 years, led a strenuous outdoor life on a small island, and had never been ill in his life, until he was overcome by petrol exhaust gas whilst attending to his motor-boat engine. After an interval of about one hour, he was discovered and removed to bed, but received no treatment. Gradually, after 36 hours, he regained consciousness, and thereafter his recovery was slow, retarded by vomiting and severe nausea. He noted that his chest and left arm were peppered with petechiæ, which rapidly disappeared. Five days after this episode, he felt no obvious ill-effects, and returned to his work, but while attempting to find the leak in the exhaust-pipe that caused the previous mishap, he was again rendered unconscious. He was discovered 20 minutes later, and regained consciousness in three hours. He experienced no untoward effects, apart from nausea, vomiting, and loss of appetite, and he was able to carry on with heavy manual work on the following day.

Fourteen days after the primary gassing, while working, he developed angina of effort, with radiation of the pain into his neck and down to his left wrist. Symptoms recurred on exertion, and were accompanied by a gripping sensation in his throat. He slept well and did not complain of breathlessness or swelling of the ankles.

These anginal attacks increased in frequency and severity so rapidly that seven days later, when coming for a medical examination, his tolerance was reduced to two hundred yards on the flat. Rest still relieved the pain, but he was now developing a concomitant breathlessness.

The family history was free from disease, and gave no specific evidence of heart disease or premature deaths.

On examination, he was a ruddy-complexioned, alert man of 53 years, with no evidence of cyanosis, venous congestion, or peripheral œdema. Examination of the chest revealed no abnormality, the heart being of normal size, in normal rhythm, and without murmurs. The blood pressure was 135/85. Crepitations were present at the left lung base. No abnormality was elicited in any other system.

Examination of the urine showed it to be free from albumen and sugar. The Wassermann reaction was negative. The hæmoglobin was 97 per cent and the red blood corpuscle count 4·7 million. The B.S.R. was 8 mm./hr. (Westergren). X-ray revealed nothing more than a little prominence of the aortic arch. The lung fields were normal. The electrocardiogram showed sinus bradycardia at 56 a minute, a P–R interval of 0·16 sec., no axis deviation, and T waves inverted in lead I, and upright in leads II and III. In the unipolar leads, aVR and aVL were similar, with inverted P and T waves, and Q as the sole initial deflection. In aVF there was a tiny Q, tall R and an upright T wave.

V1 to V3 were similar with small R, deep S, and upright T waves. The transitional zone was between V4 and V5. In V5 there was a tall R, small S, marked S–T depression and sharply diphasic T wave. In V6, R was the sole initial deflection, measuring 30 mm. There was slight S–T depression and inverted T wave.

It was evident that he was suffering from myocardial ischæmia. We are justified in considering the two anoxial episodes as the precipitating factors, as effort pain commenced in a previously fit man so soon after such a severe constitutional upset.

He was sent home and advised to have complete rest in bed. Twenty-four hours later, while in bed, he developed severe præcordial pain and suddenly died. No autopsy was performed, and death was presumed to be due to coronary thrombosis.

Commentary

The major constituent of exhaust gas is carbon monoxide, which has the property of uniting with hæmoglobin when it comes into contact with blood to form carboxyhæmoglobin, exactly replacing the oxygen volume for volume and thus rendering the blood unavailable for oxygen carriage. Apart from carbon monoxide, exhaust gas also contains a varying percentage of hydrocarbons, of which benzol is a known tissue poison, and it was noted by Winslow (1927) that when it was present in petrol exhaust fumes, the toxicity of the gas, in comparison with coal gas, was increased. The important factor in the above case, however, is anoxia, for gassing was rapid and in a confined space. The effect of the necrotoxins is negligible, under these circumstances, compared with the effects of asphyxia.

Haggard (1921) observed that acute oxygen deficiency produced a function impairment of A-V conduction and Steinmann (1937) confirmed this, giving the frequency of electrocardiographic changes found in patients suffering from coal-gas poisoning as an example. In anoxia, it is generally observed that the most specialized tissue suffers first and, therefore, any damage to the heart would affect the A-V node, S-A node, and bundle of His. Experimental asphyxias produced in cats by Lewis *et. al.* (1914) confirmed this premise, and the results in these animal experiments were found applicable to man by Greene and Gilbert (1921), who were of the opinion that any disturbance below the branching of the bundle of His was permanent. Colvin (1928), however, describes a case of coal-gas poisoning where cardiographic changes suggested damage below the bundle of His in a healthy young man; this apparent damage rapidly and completely disappeared. In the pre-critical stage of gassing, there is shortening of the P–R interval, and in the post-critical stage a tendency for the A-V node to supplant the S-A node as pacemaker, and slowing of the heart rate follows. In the case reported, there was no evidence of impairment of the conduction mechanism, but the cardiograph shows changes in V5 in the form of the S–T segments that one would interpret as being due to a myocardial ischæmia, the form of V6 suggesting left ventricular hypertrophy.

Pathological changes in the heart following coal-gas poisoning were first described by Klebs in 1865, when he reported punctiform and diffuse hæmorrhages into the pericardium, including the tips of the papillary muscles. Gurich (1926) reported similar post-mortem findings in four previously healthy people who had died from coal-gas poisoning: there were hæmorrhagic necrotic foci, with surrounding leucocytic infiltrations, most marked in the septum and papillary muscles of the left ventricle. Similar changes have been noted by Fishberg (1940). Unfortunately, no necropsy was carried out in this case.

Treatment

There is no specific treatment for these patients who show impairment of cardiac function following a gassing episode, but in all cases of gassing, immediate treatment is important.

Rescue workers in mines, abmulance drivers, etc., are taught to remove the victim from the noxious atmosphere, to keep them absolutely at rest, and to combat shock in order that the metabolism and oxygen consumption be reduced to a minimum. A mixture of 93 per cent oxygen and 7 per cent carbon dioxide should be inhaled; if this is not available, artificial respiration is an alternative; carbon monoxide will be eliminated as quickly as possible, and in the victims suspected of having sustained cardiac damage, such treatment is imperative to permit normal conductivity, restricting irreversible processes to a minimum.

Tables compiled from patients admitted to the Royal Infirmary, Edinburgh, suffering from coal-gas poisoning, show that unconsciousness does not occur until the blood level of carbon monoxide

reaches 40 per cent or over, death occurring at a level of 55 per cent or over. In the case reported, the patient's carboxyhæmoglobin lay between 40 and 50 per cent. It is known that the rate of elimination of carbon monoxide from the blood in a given period after exposure varies directly with the initial saturation and it appears the same for the same range of saturation. Thus, in the case quoted, with a carboxyhæmoglobin figure of 40 to 50 per cent, saturation, it would have taken at least ten hours for the carbon monoxide to have been eliminated completely following recovery of consciousness (Sayers and Yant, 1923). Had active treatment been available, this period of partial anoxia (in all, 48 hours) would have been reduced to two hours.

Summary

A case of petrol-exhaust gas poisoning is reported, the victim dying from coronary thrombosis.

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