SINUS BRADYCARDIA WITH CARDIAC ASYSTOLE

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A case of sinus bradycardia with cardiac asystole was described in this journal (Pearson, 1945). The subsequent history and the post-mortem findings are now recorded.

The patient was a woman aged 52 years who had been under observation since February 1943. Her pulse rate varied between 16 and 30 and her main symptoms were attacks of unconsciousness, resembling Stokes-Adams attacks; these were shown to be due to periods of asystole lasting up to 22 seconds. The shorter periods of asystole were not accompanied by loss of consciousness. At times these attacks occurred with great frequency, and on one occasion fifteen were recorded in 45 minutes.

It was observed that exercise caused a transient increase of pulse rate; atropin 1/25 of a grain intravenously failed to increase the rate to more than 35 a minute. A radiograph of the heart and lungs at this time was entirely normal. She was discharged from hospital in August 1944.

In April 1945, the patient was seen in her home. Her condition had improved slightly and she had only once lost consciousness since January 1945. She had recorded her pulse rate daily since discharge and this varied between 26 and 33. She had been out for short walks during the previous month. Examination showed no alteration in her condition and her blood pressure was recorded as 218/140. At this time she was taking half a grain of thyroid sicca t.i.d. and half a grain of barium chloride t.i.d.

In July she began to complain of pain in the left side of her chest and was confined to bed by her own doctor. On August 8th her husband found her dead in bed. He had seen her half an hour before and at that time her condition gave no cause for anxiety, nor had she complained of feeling ill. It is presumed that she died in a syncopal attack.

Permission was obtained for an autopsy examination which showed the following findings.

The apex of the left lung was attached by recent adhesions to the chest wall and there was an effusion of moderate size at the left base. A mass could be felt at the root of the left lung spreading into the mediastinum. This proved to be a neoplasm which arose in the left upper lobe bronchus. There was very little spread into the lung itself, but a mass could be felt bounded above by the arch of the aorta and extending below to the pericardium. The heart showed some dilatation of the right auricle and ventricle. The pulmonary artery was dilated but the left main branch was considerably narrowed by the mass of growth through which it passed. There was some infiltration of the upper and posterior aspect of the parietal pericardium but the heart itself was not involved. The coronary vessels were normal. There was no evidence of atheroma of the aorta and the heart valves were also normal. The mediastinal tissues were cut by transverse incisions into four blocks about an inch thick. Neoplastic tissue invaded the mediastinum, surrounding some of the structures which normally pass through it and displacing others (Fig. 1 and 2).

I am indebted to Professor H. A. Magnus for the following report on sections of these blocks,

of the sino-auricular node, and of a portion of the left auricular wall where it was in contact with the neoplastic mass.

"Large lantern slide sections taken through the mediastinum at three different levels all show diffuse infiltration by an oat-celled bronchial carcinoma which is, in places, necrotic. The growth has completely surrounded and compressed the main vessels and numerous nerve trunks can be seen embedded in and compressed by growth.

"Sections of the left auricular wall show no evidence of growth. The area of the sino-auricular node was defined in the right auricular wall. From this area the muscle was blocked and interrupted sections were examined until the node was found. This also showed no evidence of involvement by growth." The abdominal organs were examined and no secondary deposits were found. The brain also appeared normal.

FIG. 1.—Section showing carcinoma tissue infiltrating the mediastinum, as seen from above. The constricted left branch of the pulmonary artery passes through the centre of the neoplastic mass and adjacent to it is a gland containing carbon pigment and new growth.

DISCUSSION

Unfortunately the mechanism responsible for the unique disturbances of rhythm in this case remain obscure even after autopsy. Any explanation must account for the persistent slow heart rate, the attacks of asystole, and the failure of atropin to cause a marked increase in pulse rate.

FIG. 2.—Section through the mediastinum at the base of the heart seen from below. The probe enters the dilated pulmonary artery and emerges from the branch entering the right lung. The apex of the left auricle has been cut through and the pericardial sac infiltrated with growth is seen occupying the space between it and the pulmonary artery.

There are three obvious ways by which the bronchial neoplasm might affect the heart's rhythm, (1) by infiltration of the heart muscle or specialized tissues such as the sino-auricular node, (2) by interference with the vagal or sympathetic nerves supplying the heart, or (3) by toxic effects due to the neoplasm. Finally, the association between cardiac irregularity and bronchial neoplasm in any given case may be an accidental one.

A number of articles have in recent years been written concerning cardiac arrhythmia in association with bronchial neoplasm. Twenty-eight cases have been recorded (Fishberg, 1930; Lechleitner, 1935; Auerback *et al.*, 1936; Formijne and Zuidema, 1937; Scott and Garvin, 1942; J. E. S. Pearson, 1944; Van Niewenhuizer and Kamerling, 1937).

In twenty-one, auricular fibrillation, usually paroxysmal in nature, occurred and in three of these paroxysmal flutter was also recorded; one of the remaining cases had attacks of paroxysmal flutter. Of the remaining six, one had a constant tachycardia varying between 136 and 154 (Pearson, 1944), four had simple paroxysmal tachycardia (Formijne and Zuidema, 1937; Pearson, 1944; Lechleitner, 1935, two cases), and one probably had attacks of paroxysmal ventricular tachycardia (Van Niewenhuizer and Kamerling, 1937). Post-mortem findings were recorded in twenty cases. In four only the heart and pericardium were intact, and in three the pericardium

alone was affected. The remaining thirteen showed infiltration of one or both auricles. In all these cases there was some form of tachycardia, often paroxysmal, and in the majority this could most reasonably be explained by infiltration of the heart muscle with growth. There is no resemblance between these cases and the one that forms the subject of this communication. Careful examination of the heart, including microscopy of the left auricle where it approximated to the growth, and of the sino-auricular node itself failed to reveal any evidence of direct neoplastic infiltration.

There is little doubt that many of the nerves passing to and from the cardiac plexus were subjected to considerable interference and disorganization by the neoplastic mass in the mediastinum. The superficial part of the plexus lies beneath the arch of the aorta and in front of the right pulmonary artery; it is formed by the superior cardiac branches of the left sympathetic trunk and the lower of the superior cardiac branches of the left vagus. The deep part lies in front of the bifurcation of the trachea above the point of division of the pulmonary artery and behind the aortic arch; it is supplied by sympathetic nerves from the cervical ganglion and cardiac branches of the vagus and recurrent nerves. This region between the base of the heart and the arch of the aorta was extensively infiltrated and many isolated bundles of nerve fibres were seen passing through it.

It is not easy to see how stimulation or paralysis of nerves forming the cardiac plexus could account for the changes in rhythm recorded here. Stimulation of the vagus as it passed through the growth might theoretically cause slowing and even asystole, but offers no explanation of the failure of atropin to increase the heart rate to normal. In animals it is in any case impossible to maintain vagus inhibition for any length of time and escape from vagal control constantly occurs.* Finally, it is doubtful if neoplastic infiltration can ever give rise to stimulation of nerve fibres in the neighbourhood of the growth. Complete paralysis of all vagal and sympathetic nerve fibres supplying the cardiac plexus, an unlikely possibility, would almost certainly lead to an increased heart rate and simultaneous stimulation of these nerves in animals leads to paroxysmal tachycardia and auricular fibrillation (Van Nieuwenhuizer).

There is no evidence in favour of toxic damage to the sino-auricular node: if this were the correct explanation, similar changes of rhythm would be expected to occur more commonly. Finally, the association between the bronchial carcinoma and the cardiac dysfunction may be coincidental. To believe that this is so still leaves the problem of the cardiac arrhythmia unsolved.

CONCLUSION

The autopsy findings in a case of sinus bradycardia with cardiac asystole are described. Possible explanations that might account for the disturbance of rhythm are discussed.

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* Personal communication from Prof. W. R. Spurrell.