SINO-AURICULAR BLOCK ASSOCIATED WITH INTRA-THORACIC NEW GROWTHS

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In 1930 Fishberg discussing the clinical syndromes arising from tumours of the heart observed that such tumours were usually asymptomatic: even when symptoms were present, diagnosis before necropsy was never definite. Seven years later eight examples of various cardiac arrhythmias associated with intrathoracic new growths were reported by Van Nieuwenhuizen and Kamerling (1937). Discussing cardiac arrhythmias due to bronchial carcinoma in 1944, Pearson found an incidence of arrhythmias of 10.6 per cent in his series of 66 patients. The diagnosis was proved by necropsy in 51 of these patients, and invasion of the atrium had occurred in each of the patients with an arrhythmia and in one other—an incidence of 12 per cent.

Most writers on the subject (Fishberg, 1930; Yater, 1931; Van Nieuwenhuizen and Kamerling, 1937; Scott and Garvin, 1942; Rusby and Thompson, 1943; Pearson, 1944; and Robertson and Russell, 1950) have stressed the importance of such arrhythmias chiefly as an occasional presenting symptom of a growth or as an indication of its spread to the heart and hence inoperability. In 1937, however, Van Nieuwenhuizen and Kamerling, having found in some of their patients evidence of sympathetic stimulation, suggested that a cardiac arrhythmia might result from irritation of the cardiac branches of the vagosympathetic nerves.

Two patients, each with sino-auricular block due to irritation of the vagus nerve by growth, are now described.

CASE RECORDS

Case 1. The patient, a man of 55 years, was first seen by his medical attendant complaining of hoarseness which persisted until his death a year later. Previously he had been a fit man apart from winter bronchitis.

During August, 1951, he developed nausea and flatulence and became afraid to eat. In the first week of January, 1952, he had a seizure while at work. Similar attacks occurred from then onwards—as many as twenty in a day. On admission to hospital in February, 1952, he was found to have lost two stones in weight. He had a hoarse voice from a complete abductor paralysis of the left vocal cord. The heart rate was 70 a minute and the rhythm regular. The rate increased on exercise to 90–100 a minute but was not slowed by carotid sinus pressure. The heart size, impulse, and sounds were normal. The blood pressure was 110/90 mm. Hg in each arm. Clinically the nervous, respiratory, and gastro-intestinal systems appeared to be normal.

His major seizures occurred at any time. The patient found that over-breathing precipitated the attacks which were preceded by nausea and retrosternal tightness and followed by loss of consciousness. In the attacks, the upper limbs showed clonic movements and consciousness was lost for two to three minutes during which there was occasional incontinence of urine. The heart first slowed and then stopped for about ten to twenty seconds before returning to normal rate and rhythm. During the attacks, auricular pulsations were never seen in the neck veins, nor were heart sounds to be heard. With the return of consciousness, the head and neck became flushed but otherwise no sequelæ were observed. Both memory and behaviour were unaffected.

Investigations. Fluoroscopy revealed a mass in the superior mediastinum which did not pulsate but which obstructed the œsophagus at the level of the carina. During one examination, the patient lost

consciousness and complete standstill and dilatation of the heart was observed for about ten seconds. Tomograms confirmed the presence of a mass extending from the hilum of the left lung in front of and above the aortic arch.

Electrocardiographic records during attacks showed initial slowing followed by complete standstill of the heart. Recovery was initiated by ventricular escape, followed by an auricular rhythm from an ectopic focus with inversion of the P wave. Restoration of the normal rate and rhythm occurred at varying times: in the record shown (Fig. 1) after a standstill of twenty-one seconds. Electrocardiograms between attacks were normal. An electroencephalogram during an attack was probably normal allowing for superimposed muscle waves. Both the Wasserman and Kahn reactions were negative. At bronchoscopy and œsophagoscopy, no abnormality was detected.

FIG. 1.—Case 1. Cardiogram taken during an attack, showing sino-auricular block. All records are lead II and not as marked.

Course. The syncopal attacks were seen on many occasions and always followed the same pattern. As the electrocardiographic findings pointed to sino-auricular block as the cause, he was given atropine 1/100 gr. sublingually four-hourly. The attacks rapidly diminished in number and ceased after eight days. Soon afterwards, however, two small subcutaneous nodules were noticed in the chest wall. These were excised and found to be metastatic deposits of an anaplastic carcinoma.

Two weeks later, the patient developed a cough with purulent sputum, fever, and a palpable pleural rub in the left axilla. Radiographs of the chest revealed extensive infiltration of both upper lobes. A few days later the seizures recurred, together with stridor, cyanosis of the head, and engorgement of the neck veins. Despite the use of atropine, he died in an attack on March 30, 1952.

Necropsy. There was a carcinoma of the right anterior basal bronchus. The hilar lymph nodes were enlarged, being replaced by growth, much of which was necrotic. The left vagus nerve entered these nodes, but its course and that of its recurrent branch could not be traced. The pericardium and heart together with its conducting system were intact and the brain was normal.

Histology. The tumour was an oat-celled bronchial carcinoma infiltrating the hilar lymph nodes and both upper lobes. Sections of the hilar mass showed several nerves surrounded by the growth and the left vagus nerve was seen to be infiltrated by carcinoma cells. Six centimetres above the site of the compression and invasion by the tumour, the left vagus nerve showed no evidence of Wallerian degeneration.

Case 2. This patient who attended King's College Hospital in 1921 had had radiotherapy for a lymphosarcoma affecting the cervical lymph nodes. He then developed seizures resembling Morgagni-Adams-Stokes' attacks. They occurred without warning and even during sleep. The patient would attempt to get out of bed and then would become still. In attacks, no pulse was felt and no heart sounds heard for about half a minute. Then movements of the upper limbs with stertorous breathing occurred. On recovery the patient appeared dazed but the pulse was slow and regular in rhythm. No evidence of heart block either clinically or electrocardiographically was found between seizures. An electrocardiogram was not obtained during a seizure. Tincture of belladonna diminished the frequency of the attacks but the patient eventually died during one three months later.

Necropsy. A mass of growth extended from the left jugular foramen to the apex of the lung. Into this mass, the left vagus nerve entered. On the right side of the neck, a further mass was present to which the right vagus nerve was adherent. The nerve, however, was not invaded. There was a left pleural effusion whilst the left lung contained an indefinite mass of growth. Both lower lobes showed bronchopneumonia. The pericardium, heart, aorta, and brain were normal.

Histology. Sections of the tumour were reported as showing a lymphosarcoma but unfortunately were not available for study.

DISCUSSION

Intrathoracic new growths may cause cardiac arrhythmias in two ways—directly by invasion of the myocardium or the conducting tissue or both, and indirectly by affecting the vagosympathetic pathways to the heart. The former is much the commoner. Pearson (1948) in a review of malignant metastases in the myocardium, described 148 cases with bronchial carcinoma, 98 of which were proved by necropsy. Seven of these had been reported previously (Pearson, 1944). Invasion of the heart occurred in 17 (11.5%) and in approximately two-thirds of these an arrhythmia occurred. This was said to be due to invasion of one or both atria by growth. Reviewing 28 examples of cardiac arrhythmias associated with bronchial carcinoma of which 20 came to necropsy, Pearson (1950) found only four with an intact pericardium and heart, and three with only the pericardium affected. The growth had invaded one or both atria in all the others.

Although extensive infiltration of the mediastinum by growth is common, cardiac arrhythmias that can be attributed to an effect upon the vagus and sympathetic nerves are rare. Bruce Pearson (1945, 1950) reported a patient with bradycardia and attacks of cardiac asystole in which atropine had no effect. At necropsy, the vagi were enmeshed in mediastinal growth from a carcinoma of the bronchus. The obvious explanation is that the arrhythmia was due to vagal stimulation by growth but this is most unusual and in any case atropine did not control the arrhythmia.

Carcinoma of the bronchus, by virtue of its frequency and position, is the commonest intrathoracic tumour to cause cardiac arrhythmias. Other intrathoracic new growths are only occasionally responsible. Pearson (1944, 1948) mentioned one instance of mediastinal lymphosarcoma leading to auricular fibrillation while Van Nieuwenhuizen and Kamerling (1937) recorded another of cancer of the œsophagus with paroxysmal sinus tachycardia.

Of the cardiac arrhythmias described, auricular fibrillation is by far the commonest. Thus Van Nieuwenhuizen and Kamerling (1937) found in their patients that six had auricular fibrillation, one sinus tachycardia, and the other paroxysmal ventricular tachycardia. Three of these patients had evidence of sympathetic irritation in a dilated pupil on the same side as the tumour, and one also had exophthalmos. Pearson (1944) in his series reported four patients showing paroxysmal auricular fibrillation, one paroxysmal auricular fibrillation and flutter, one paroxysmal premature systoles, and one a persistent regular tachycardia.

Few examples have been reported of sino-auricular block in any of its forms (Cowan, 1939) and there is little doubt that they comprise the rarest types of arrhythmia due to growths in the thorax. A patient who had a variety of arrhythmias was reported by Mahaim and Gander (1947). Premature ventricular systoles and auriculo-ventricular nodal rhythm were recorded and in addition to these, the patient had seizures in which the heart stopped for five to ten seconds. These seizures

were controlled by belladonna. Later auricular flutter appeared and the seizures ceased. At necropsy, there was transpericardial spread of growth into the right atrium and destruction of the sino-auricular node but the auriculo-ventricular node and bundle were intact. The authors suggested that irritation by growth of the cardiac branches of the vagus nerve as they coursed over the surface of the heart was responsible for the attacks of cardiac asystole. Later destruction of these nerves by growth and spread to the right atrium was held responsible for the cessation of these attacks and the appearance of auricular flutter in which the ventricular rate was 140–150 a minute. Hence there was neither clinical nor pathological evidence of destruction of the A-V node and bundle. In 1945 Mahaim thought that stimulation of the vagus nerve by growth might well cause attacks of A-V nodal rhythm, but he could not find any recorded examples in which the sino-auricular node was intact, nor do such cases appear to have been recorded since then.

In both patients described in this paper, the left vagus nerve was enmeshed in growth and during the latter part of their illnesses, both patients suffered recurrent attacks of syncope due to sino-auricular block. In Case 1, the attacks were preceded by sinus bradycardia and recovery was initiated by ventricular escape. A similar march of events occurred in Case 2. In both patients, atropine prevented these attacks. It may thus be concluded that the attacks of sino-auricular block were due to vagal stimulation and that involvement of the left vagus nerve in the tumour mass was the determining cause. If this explanation in these cases is correct, then they are examples of a little recognized mechanism.

The evident rarity of sino-auricular block arising in such a way raises two further problems. One is the reason for this rarity in view of the frequency with which malignant tumours in the mediastinum encircle the nerve trunks. The other is to account for the intermittent nature of the attacks. Perhaps the former depends upon the early destruction of the functional activity of the nerve by growth and the latter upon an enhanced susceptibility to physiological fatigue. In any case, escape from vagus control will occur owing to a rise in venous pressure stimulating the cardio-accelerator mechanism (McDowall, 1926).

SUMMARY

Two patients are described with recurrent syncope due to stimulation of the left vagus nerve by malignant tumours. Atropine prevented the attacks for a time but both patients eventually died in an attack.

Sino-auricular block is one of the rarest of the cardiac arrhythmias complicating intrathoracic tumours.

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