Paroxysmal Cardiac Arrest

Report of a Case with Simultaneous Atrio-ventricular Standstill Presumably Due to Carcinoma of the Lung and Responding to Radiation Therapy

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INTRODUCTION

In spite of occasional surgical cures, carcinoma of the lung usually casts the physician in the role of comforter and alleviator of distressing symptoms. This case report will illustrate the tragic plight of a young man with inoperable carcinoma of the lung who developed frequent attacks of paroxysmal cardiac arrest with Adams-Stokes attacks resembling status epilepticus.

CASE REPORT

A 37-year-old, married, white, machine tool inspector entered the White River Junction Veterans Administration Hospital on October 22, 1959 because of an abnormal routine chest x-ray film finding. He had no complaint. Eight years prior to admission, while in the Navy, at age 29, a spontaneous left-sided pneumothorax was treated by poudrage. His mother and brother were thought to have had tuberculosis at one time. Accordingly, he had been x-rayed every three months by the Vermont Health Department. His last x-ray film prior to admission showed a large left hilar mass which was not present three months previously. He had smoked one and one-half packs of cigarettes a day since age 14. He denied any symptom whatsoever, and his initial physical examination was unremarkable.

One of three sputum samples showed positive cytologic evidence of malignancy. Eight prescalene lymph nodes showed no evidence of tumor. Bronchoscopic examination showed some fixation of the left tracheobronchial angle, but the orifices appeared normal.

Thoracotomy disclosed massive tumor involvement of the left hilum with multiple pleural and pulmonary metastases—biopsy of which showed undifferentiated carcinoma of large-cell type. Resection was impossible. Postoperatively, he received nitrogen mustard treatment and was sent home on November 25, 1959, still with essentially no symptom.

A month later, a mass was palpable in the left supraclavicular fossa. At about this time, he began to note brief syncopal attacks. These momentary losses of consciousness were the cause of several falls and bruises. They lasted less than a minute and seemed to be precipitated by exertion or straining at stool. His wife noted his pulse was unobtainable during these episodes. Because these attacks increased in frequency to about 20 a day, he sought readmission on February 1, 1960. During the course of a physical examination on this day, three episodes were observed by the house officer, who noted unconsciousness lasting about 30 seconds and during which there was a long period of asystole followed by a slow regular rate 45/min. Pressure on the 3 x 3 cm. hard, left cervical mass did not precipitate an attack. Indirect laryngoscopy revealed paralysis of the left vocal cord.

Chest x-ray examination showed further widening of the superior mediastinum. An electrocardiogram showed sinus bradycardia with ectopic auricular beats. Prolonged simultaneous atrio-ventricular standstill lasting up to 15 seconds was documented as the cause of Adams-Stokes attacks (Fig. 1). Frequent doses of subcutaneous atropine and subsequently oral belladonna had no effect on the rate. Oral ephedrine, isoproterenol (Isuprel) linguets in large doses, and barbiturate sedation were equally ineffective alone or in combination with atropine. The pulse rate hovered at 30-40/min. between attacks of unconsciousness, which now became associated with generalized convulsive movements. By February 22, the episodes were very frequent and had so completely unnerved him that he screamed with terror, as dizziness and tinnitus heralded the onset of another attack—now 10-15 times an hour. Drastic measures such as large doses of intravenous atropine (2.0 mg.) and intravenous epinephrine (0.2 mg.), application of an external cardiac pacemaker, and left vagal nerve block were of no avail. That the sinoatrial pacemaker of the heart muscle itself was involved with tumor seemed plausible.

X-ray therapy to the mediastinum was given over a four-week period through two portals totaling 4000 r. There followed quite dramatic cessation of the Adams-Stokes attacks (Fig. 2). There was no episode of standstill from March
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The 12 lead electrocardiogram was normal at time of thoracotomy, November 9, 1959, but showed sinus bradycardia, premature auricular contractions and prominent U waves on February 26, 1960. Lead V1 shows part of a typical episode of atrio-ventricular standstill, which lasted over 10 seconds and was accompanied by convulsions and unconsciousness.

30 until his death on June 30. For at least three months, therefore, a remarkable amelioration of symptoms occurred.

He was able to spend the last few weeks at home before returning with fatal pneumonitis. Necropsy revealed oat-cell carcinoma originating in the upper left lobe of the lung with metastases to the hilum, liver, spine, left adrenal, pancreas, and carotid sheaths (compressing it in the area of bifurcation). The heart and pericardium were normal grossly and microscopically, although careful search for tumor in the muscle fibers near the sino-atrial node was made.

DISCUSSION

Since Adams and Stokes called attention to the syndrome which now bears their names (in 1827 and 1846 respectively), many clinical observations have been made. A number of cardiac mechanisms have been postulated. Electrocardiograms have substantiated a variety of cardiac arrhythmias associated with the episodes. These include prolonged ventricular tachycardia, flutter, fibrillation, or asystole. Usually there is an associated heart-block.

These arrhythmias in turn have been attributed to a number of reflex mechanisms, some arising in the carotid sinus, or from pressure on the vagus nerve due to neoplasm, or esophageal-diverticulum. Other attacks have been associated with straining at stool or digital irritation of the anal sphincter. Exaggerated vagal tone frequently has been blocked with atropine to relieve attacks. In other cases, the development of complete atrio-ventricular block and its associated idioventricular rhythm has permanently halted multiple previous neurogenic bouts of simultaneous auricular and ventricular standstill. Simultaneous atrio-ventricular standstill is extremely rare and usually follows a prolonged episode of ventricular arrest. Also unusual in this case was the absence of ventricular escape beats at any time suggesting probable vagal suppression of the usual lower cardiac centers capable of automaticity.

In the present case of simultaneous atrio-ventricular standstill, all pharmacologic efforts at relief failed. The attacks were thought to be due to increased vagal effect or decreased sympathetic effect or both. In an effort to increase rhythmic, sympathetic effect at the sino-atrial node, large doses of sympathomimetic drugs were given with no effect. Similarly of no avail were efforts to block any vagal overtones with enormous doses of atropine.

The gratifying response to radiation therapy was accompanied by pronounced shrinkage of the mediastinal metastases suggesting that the mediastinal tumor mass was responsible for a reflex neurogenic effect by pressure or infiltration of the nerves. The fact that there was extensive infiltration of the carotid sheath at nec-
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Paroxysms no cardiac arrest or syncope.

Because of the terrifying nature of these multiple convulsive episodes of asystole and the demoralizing effects they had not only on the patient, but also upon his relatives and the professional staff, the complete cessation following x-ray therapy to the mediastinum is considered an important enough observation to share.

Paraffin sections and histologic study of the heart, however, may substantiate this. Complete absence of any direct cardiac involvement by tumor was of interest. There is also the possibility that the tumor elaborated some humoral agent which indirectly altered the optimal cardiac milieu. However, the electrolytes, when measured were unremarkable, and the shape of the arrhythmic electrocardiogram did not differ much from the previously normal one.

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References