nation and interpreted the tumor as a malignant endothelioma. It was Teilum's, who, by comparing the morphology of the neoplasm with that of the rat placenta, named it endodermal sinus tumor. Other authors prefer the equivalent designation of "yolk sac carcinoma." Both names stress the fact that these lesions show differentiation towards extraembryonic yolk sac endoderm and mesoblastic tissues, as found in the rat placental structures known as endodermal sinuses of Duval. To the best of our knowledge, only seven cases of endodermal sinus tumor of the mediastinum have been reported in the world literature. All of these have occurred in the anterior mediastinum in male patients, predominantly during the second, third, and fourth decades of life. All of the patients died within a maximum of six months from the time of onset of symptoms. None of those cases was examined ultrastructurally. Only two ultrastructural studies of human ovarian endodermal sinus tumor have been published (three cases, only available in summary form).

Our light microscopic examination of this neoplasm showed the characteristic histologic features of the endodermal sinus tumor, as described by Teilum and others. The absence of tumor or scarring in the patient's testis rules out the possibility of an occult primary testicular tumor with metastases to the anterior mediastinum.

The electron microscopic features observed in our case are similar to those reported in the three previously mentioned cases of ovarian endodermal sinus tumor. A striking ultrastructural similarity was noted between our tumor and the yolk sac of the rat, guinea pig, and man, and also murine yolk sac carcinoma. The basement lamina-like material which we found accumulated between and sometimes apparently within many of the neoplastic cells was similar to the substance found in Reichert's membrane of the mouse embryo. This type of material has been identified as basement membrane by Silverberg et al by means of immunohistochemical studies. We do not know the exact significance of the variable periodicity of the collagen fibrils found in the neoplasm.

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Dual Atrioventricular Conduction during an Acute Inferior Myocardial Infarction

Electrocardiographic Evidence

Richard W. Asinger, M.D., and Morrison Hodges, M.D.

A 49-year-old man had electrocardiographic evidence of a dual atrioventricular conduction system which occurred spontaneously during the course of an inferior myocardial infarction. Retrograde concealed conduction involving both conduction systems is proposed as the mechanism to explain the varying patterns of atrioventricular conduction seen.

Moe and associates* provided evidence of dual atrioventricular conduction systems in the dog in 1956. The introduction of His bundle electrographic studies combined with the premature atrial stimulus

*From the Cardiology Sections, Departments of Medicine, Hennepin County Medical Center, and the University of Minnesota, Minneapolis.

Reprint requests: Dr. Asinger, Hennepin County Medical Center, Minneapolis 55415

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DUAL AV CONDUCTION DURING INFARCTION

Figure 1. Routine sequential 12-lead ECG taken on morning of third day of hospitalization, demonstrating evolving inferior myocardial infarction. Limb leads and precordial leads V1 to V6 demonstrate normal sinus rhythm with rate of 95 beats per minute and P-R interval of 0.29 second. Precordial leads V5 to V6 also demonstrate normal sinus rhythm with same rate of 95 beats per minute but with P-R interval of 0.16 second.

CASE REPORT

A 49-year-old man was admitted to Hennepin County Medical Center on Jan 3, 1975, with a two-hour history of severe crushing substernal chest pain associated with diaphoresis, nausea, and lightheadedness. His initial rhythm was complete heart block, and the patient was given 0.5 mg of atropine intravenously, which was followed by restoration of normal sinus rhythm. Physical examination demonstrated a fourth heart sound and fine bilateral basilar rales. A chest x-ray film was normal.

Serial electrocardiograms and levels of cardiac enzymes were consistent with an acute inferior myocardial infarction. The patient's hospital course was uneventful, except for pericarditis and the following conduction disturbance.

Rhythm Analysis

The patient remained in normal sinus rhythm with a normal P-R interval for the first two days of hospitalization. An ECG taken on the third day of hospitalization demonstrated varying atrioventricular conduction (Fig 1). There is an abrupt change in the P-R interval from lead V1 to lead V6, without a change in sinus rate, suggesting a dual atrioventricular conduction system. During the following 15 hours the patient frequently changed from one P-R interval to the other.

The rhythm shown in Figure 2 is an example of a change from normal to prolonged P-R interval. The first three atrial beats conduct to the ventricle via the fast conduction system (Fig 2, a), with normal P-R intervals. We propose that these atrial impulses also enter the slow conduction pathway (Fig 2, b), but do not conduct to the ventricles. There are at least two reasons for this failure of conduction. First, depolarization from the slow pathway could reach the final common pathway when the final pathway is still effectively refractory (eg, Fig 2, atrial beat 2). Secondly, retrograde concealed conduction could occur in the slow pathway via the fast pathway and result in collision of antegrade and retrograde depolarizations (eg, Fig 2, atrial beat 1). The third atrial beat in Figure 2 is also conducted to the ventricle via the fast pathway, with conduction via the slow pathway blocked in the final common pathway. We propose that retrograde conduction of the fast pathway via the slow pathway occurs. The fourth atrial depolarization is blocked in the fast pathway because of the retrograde concealed conduction from the previous beat. Atrioventricular conduction of the fourth atrial beat then proceeds via the slow pathway, with a prolonged P-R interval. Retrograde depolarization of the fast pathway again occurs, allowing the mechanism to be repetitive with continuation of sinus rhythm with prolonged atrioventricular conduction via the slow pathway.

Figure 3 demonstrates changes from normal to prolonged and prolonged to normal P-R intervals. The first two atrial beats are conducted via the fast pathway. The interval from the peak of the P wave to the following QRS complex (PPR interval) of the second beat is slightly increased when compared to the first beat. This increase is presumed to represent the effect of retrograde concealed conduction in the fast pathway of the first beat. Retrograde concealed conduction of the fast pathway from the slow pathway is also presumed to occur in the second beat. The third atrial beat is blocked in the fast pathway, and, hence, atrioventricular conduction proceeds by the slow pathway with its prolonged P-R interval. Atrioventricular conduction is then maintained as previously described for atrial beats 4 to 6 in Figure 2; however, there is a progressive increase in atrioventricular conduction time, indicated by a decreasing interval from each R-wave peak to the following P wave (Fig 3, atrial beats 3 to 9). Finally, atrioventricular conduction does not occur following the tenth atrial beat. The progressive increase in atrioventricular conduction time via the slow pathway is presumed to have progressed to complete block, and atrioventricular conduction of the tenth atrial beat by the fast conduction system is blocked because of retrograde concealed conduction from the slow pathway.
of the atrioventricular node. Differential ischemia of the atrioventricular junction during the hospital course of this patient could have set the stage for longitudinal dissociation and the appearance of a dual atrioventricular conduction system.

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Electrode Causation of Pacemaker Inhibition*

Seymour Furman, M.D., and Vincent De Caprio, M.S.

Electromechanical nonphysiologic signals caused by the movement of intracardiac metal portions of an endocardial grasping electrode were shown to be "falsely" recycling an implanted pacemaker. The signals were similar in amplitude and slew rate to the ventricular electrogram and were terminated by ending the movement of the two metallic surfaces.

In pacemakers that sense cardiac activity, "false" recycling has been a recognized complication. One of its mechanisms has been that of fracture of the electrode, with the two fragments lying in an electrolytic solution,

*From the Department of Surgery, Division of Cardiothoracic Surgery, Montefiore Hospital and Medical Center, Bronx, NY. Supported in part by Public Health Service grant HE 04666-16.

Reprint requests: Dr. Furman, 111 East 210 Street, Bronx 10461

Figure 1. Normal sensing and pacing. Last three stimuli appear at pacemaker's escape interval. Second and fourth stimuli have been "falsely" recycled.