Case Reports

Digitalis-Induced Double Atrioventricular Nodal Rhythm Associated with Electrical Alternans

Edward K. Chung, M. D., F.A.C.C.*

Summary

An instance of digitalis-induced double atrioventricular nodal rhythm associated with ventricular electrical alternans has been described. To the knowledge of the author, it is the first such combination reported. One atrioventricular nodal pacemaker (group A) produces non-paroxysmal atrioventricular nodal tachycardia and the other pacemaker (group B) produces atrioventricular escape rhythm with electrical alternans. The influence of digitalis and hypokalemia in relation to such cardiac arrhythmia has been discussed. The necessity of early recognition of digitalis intoxication in elderly patients who develop non-paroxysmal atrioventricular nodal tachycardia and atrioventricular nodal escape rhythm during digitalization has been emphasized. The appearance of electrical alternans in this case is thought to be related to digitalis intoxication.

Additional Indexing Words:
Digitalis-induced arrhythmia A-V nodal rhythm Electrical alternans

It is well recognized that digitalis intoxication may produce atrioventricular nodal rhythm due either to atrioventricular block, causing atrioventricular nodal escape rhythm or acceleration of atrioventricular nodal pulse formation, resulting in atrioventricular nodal tachycardia1-3) or a combination of both factors.1) Double atrioventricular nodal rhythm (or tachycardia) resulting from digitalis excess also has been observed previously by different investigators.1,3) However, its association with electrical alternans has never been reported as far as the author can ascertain. Such an entity is described in the following presentation.

From the Cardiovascular Disease Clinical Research Program Project, Heart Station and Department of Medicine, Meharry Medical College, Nashville, Tennessee.
* Associate Professor of Internal Medicine, Assistant Director, Heart Station.
Present address: Associate Professor of Internal Medicine, West Virginia University School of Medicine, Morgantown, West Virginia.
Received for publication May 30, 1968.
CASE REPORT

A 72-year-old male was admitted to the hospital with marked congestive heart failure of one week duration due to arteriosclerotic heart disease. He had been taking digoxin 0.25 mg. and hydrodiuril 50 mg. daily for chronic congestive heart failure for at least 10 years prior to this admission. The significant findings on physical examination were: blood pressure 190/110 mm. Hg, slow heart rate at 43 beats per min., moderate left ventricular hypertrophy and bilateral moist basal pulmonary rales.

Roentgenogram of the chest showed moderate left ventricular hypertrophy with minimal bilateral pleural effusion. The electrocardiogram taken on admission revealed double atrioventricular nodal rhythm with electrical alternans (Fig. 1). The blood chemistry was normal except for a serum potassium of 2.8 mEq./L. All therapeutic measures, including discontinuation of digitalis and intravenous administration of potassium were unsuccessful and the patient expired 3 hours after admission. Autopsy revealed no evidence of myocardial infarction or pericardial effusion. Left ventricular hypertrophy and slight bilateral pleural effusion were present.

ANALYSIS OF ELECTROCARDIOGRAM

Fig. 1 exhibits double atroventricular nodal rhythms with electrical alternans. The P waves are inverted in lead aVF and upright in lead aVR and appear at a rate of 118 per min. These P waves are conducted in a retrograde fashion from the atroventricular node (group A). The ventricular rate is 43 per min. and the QRS deflections originate from the other pacemaker in the atroventricular
node (group B). Thus, the atria and ventricles are activated independently by two different pacemakers within the atrioventricular junctional tissue. The rate of group A pacemaker is faster than the inherent automaticity of impulse formation in the atrioventricular node, so that a non-paroxysmal atrioventricular nodal tachycardia exists. The QRS complexes originating from group B pacemaker represent 2:1 ventricular electrical alternans throughout the tracing. The impulse from group A pacemaker is unable to conduct to the ventricle in spite of the slow ventricular rate from group B atrioventricular nodal pacemaker, indicating that there is complete atrioventricular block. In addition, there is a prominent U wave due to hypokalemia.

**DISCUSSION**

The diagnosis of recurrent congestive heart failure was made in this patient, however, it was difficult to judge clinically whether or not it was a manifestation of digitalis intoxication. Toxicity due to digitalis was suspected because he was an elderly patient who had been taking diuretics prone to produce hypokalemia. Non-paroxysmal atrioventricular nodal tachycardia, various atrioventricular conduction disturbances and other various ectopic tachycardias are known to be associated with such a situation. The atrioventricular node has two distinctive actions; passive and active impulse formation. When the primary pacemaker fails to produce impulses (sinus arrest) or fails to conduct through sinoatrial or atrioventricular junctional tissues due to sinoatrial block or atrioventricular block, atrioventricular nodal rhythm occurs as an escape rhythm. If the atrioventricular node is acting as a pacemaker resulting from acceleration of impulse formation beyond its inherent automaticity, atrioventricular nodal tachycardia occurs as a paroxysmal or non-paroxysmal form. The rate varies between 150 and 200 per min. in paroxysmal tachycardia and 70 to 130 per min. in non-paroxysmal tachycardia. The presence of non-paroxysmal atrioventricular nodal tachycardia almost always indicates organic heart disease or digitalis intoxication, whereas paroxysmal nodal tachycardia may occur in healthy hearts.

Digitalis-induced atrioventricular nodal tachycardia has been reported by many different observers and was thought to be a more commonly manifestation of digitalis excess than the so-called paroxysmal atrial tachycardia with block. My experience agrees with the study of Pick et al. in that 4 out of 5 cases studied with double atrioventricular nodal rhythm or tachycardia were thought to be due to digitalis excess. Group A atrioventricular nodal pacemaker produces a rate of 118 per min. which controls only the atria. This manifests an example of non-paroxysmal atrioventricular
nodal tachycardia. Because of antegrade atrioventricular block, none of the impulses from group A pacemaker is conducted to the ventricle, since digitalis has depressive effects on atrioventricular conductivity and causes complete atrioventricular block. As a result of complete atrioventricular block, the other pacemaker (group B) in the atrioventricular junctional tissue controls the ventricles producing atrioventricular nodal escape rhythm.

In atrioventricular nodal rhythm or tachycardia, atrioventricular dissociation commonly occurs. Almost always the atria are controlled by the sinus node or by an ectopic atrial rhythm (as in atrial fibrillation, flutter or tachycardia) and the ventricles are controlled by the atrioventricular node and rarely by a ventricular ectopic focus (idioventricular rhythm). In a rare form of atrioventricular dissociation, the atria and ventricles are activated by different ectopic foci located in the atrioventricular node resulting in double atrioventricular nodal rhythm (or tachycardia). In the present case, a combination of 2 factors, active impulse formation of group A pacemaker and complete atrioventricular block producing passive impulse formation of group B pacemaker, result in double atrioventricular nodal rhythm with complete atrioventricular dissociation. In addition, it is interesting to note that group B atrioventricular nodal rhythm exhibits 2:1 ventricular electrical alternans throughout the tracing. A 2:1 ventricular alternans has been thought to be of no apparent clinical importance during tachycardia but otherwise can be encountered in coronary heart disease, myocarditis or congestive heart failure. In general, the most common cause of electrical alternans was thought to be pericardial disease, but in a previous study, this was not definitely substantiated.

The exact mechanism of electrical alternans is not yet clearly understood and various hypotheses have been described elsewhere. The hypothesis of alternating prolongation of the refractory phase of some part of the heart is the most attractive. In the presence of tachycardia, every alternate impulse may find some region of the myocardium still normally refractory from previous activation so that electrical alternans, in this instance, may be a normal and physiological phenomenon. However, if the electrical alternans occurs during a slow heart rate, this may indicate abnormal prolongation of the refractory phase of some part of the heart after previous excitation, and a subsequent impulse finds this region of the myocardium refractory. Consequently, electrical alternans in this instance would be considered pathologic. In the present case, electrical alternans is present throughout the tracing in spite of a very slow heart rate (group B), indicating definite pathology. Although electrical alternans occurring during atrioventricular nodal tachycardia was observed previously, a very slow atrioventricular
nodal escape rhythm associated with electrical alternans has never been reported as far as the authors can ascertain.

It is well documented that digitalis intoxication is frequently encountered in hypokalemia.\textsuperscript{4,12-14} Hypokalemia alone can cause cardiac arrhythmias as well as accelerate those induced by digitalis.\textsuperscript{13,14} It is also known that the myocardium may lose potassium due to heart failure, thus, predisposing to digitalis intoxication.\textsuperscript{15-17} In elderly patients, such as herein reported, who are being treated for congestive heart failure with digitalis and diuretics, digitalis intoxication may be easily produced since such patients have poor myocardial reserve and are especially sensitive to digitalis.\textsuperscript{18,19} The sudden appearance of rapid or slow heart action, particularly, atrioventricular nodal escape rhythm or non-paroxysmal atrioventricular nodal tachycardia during digitalization in elderly individuals, should make one suspicious of digitalis toxicity rather than the need for increased digitalis.

\textbf{REFERENCES}