Atrial Flutter with Irregular Ventricular Response as a Contraindication to Digitalis

By Seymour B. London, M.D., and Rose E. London, M.D.

Although atrial flutter occurs at atrial rates of 250 to 350 per minute, or faster, the ventricular response is usually at one half the atrial rate because of partial block of atrioventricular conduction. This block is of physiologic origin, arising from the refractory period of the atrioventricular node, and it may occur without obvious cardiac disease.1-3 On the other hand, variations in the ventricular response during atrial flutter4 indicate increased block, which may be associated with disorders of the conduction system.

Two cases are presented that illustrate the potential danger of producing complete atrioventricular block by the use of digitalis in atrial flutter with greater than 2:1 block and emphasize the value of an external electric cardiac pacemaker in ventricular asystole that may follow such complete block.

Case Reports

Case 1

A 58-year-old woman with no history of cardiovascular disease or hypertension was found on a routine examination to be in good health except for a grossly irregular rapid heart action. The pulse rate varied between 120 and 130, and the blood pressure was 120/80. The heart tones were of good quality, there were no murmers, and there were no signs of enlargement or failure. An electrocardiogram (fig. 1) demonstrated atrial flutter with 2:1 and 3:1 ventricular response with Wenckebach phenomenon. The atrial flutter rate was approximately 300 and the ventricular rate 130.

Although the patient felt well, it was thought wise to correct the arrhythmia. She was hospitalized and was given 1 mg. of digoxin in divided doses. On the following day, an additional 0.5 mg. of digoxin was given. The atrial flutter persisted but now with fixed 4:1 block (fig. 2A). Because of the continued flutter three doses of 0.2 gm. of quinidine were given three hours apart. By the evening of the second day variable block had occurred with resultant irregularity of the pulse; an additional 0.25 mg. of digoxin was given. The next morning the electrocardiogram revealed a flutter wave rate of 300 and an irregular ventricular rate of approximately 60 (fig. 2B). Digoxin was discontinued because of the increased atrioventricular block, but quinidine was continued and the patient received five 0.2-Gm. doses at 3-hour intervals. At 8:00 A.M. on the fourth day, with an electrocardiogram (fig. 2C) showing no change from the previous day, the patient was given 0.25 mg. of digoxin, making a total dose of 2 mg. over 4 days. One hour later the patient suddenly vomited and fell out of bed. She recovered quickly but had four similar episodes during the next 4 hours. Each seizure, as described by the nurses, consisted of nausea, flushing of the skin, and transient unconsciousness, followed by pallor and perspiration. During the third seizure, an electrocardiogram (fig. 3) revealed persistent atrial flutter and long periods of ventricular asystole. Immediately an external electric pacemaker5 was applied and further seizures were terminated promptly by electric stimulation.

A trial of isoproterenol (Isuprel) sublingually, 7.5 mg. at 10-minute intervals was followed by temporary 2:1 atrioventricular response and frequent ventricular extrasystoles. Ten minutes after the third dose of isoproterenol, however, ventricular standstill of 12 seconds occurred and was associated with a major convulsive seizure. Therefore the heart was driven intermittently by the pacemaker for the next 12 hours until 4:1 to 6:1 atrioventricular conduction returned with a ventricular rate of 60 to 70. Thereafter the patient regained her usual state of well-being, and was discharged without further medication. Electrocardiograms showed persistent atrial flutter with variable block until approximately 1 year later, when she was found to have a normal sinus mechanism with entirely normal tracing. (fig. 4). She has remained well since.

Case 2

A 74-year-old retired iron worker had suffered a myocardial infarction 6 years previously followed by congestive failure, which was controlled by digitalis and salt restriction. His electrocardiograms (fig. 5) over the previous 2 years showed complete right bundle-branch block and a pro-

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Case 1. Lead II demonstrating atrial flutter with variable F-R relationship and Wenckebach phenomenon. AV represents the time between the atrial flutter impulse (A) and the ventricular activation (V). Alternate flutter impulses showing physiologic block are not indicated as penetrating the AV node. The interrupted oblique lines following flutter wave numbers 10 and 20 indicate a block of the impulse in the AV node producing Wenckebach phenomenon.

Figure 1

Atrial flutter with variable F-R relationship and Wenckebach phenomenon. AV represents the time between the atrial flutter impulse (A) and the ventricular activation (V). Alternate flutter impulses showing physiologic block are not indicated as penetrating the AV node. The interrupted oblique lines following flutter wave numbers 10 and 20 indicate a block of the impulse in the AV node producing Wenckebach phenomenon.

Figure 2

A. Case 1. Fixed 4:1 block following digitalization. Second hospital day. (Lead aVp.) B. Case 1. Increase in block on third hospital day with further digitalization. (Lead aVp.) C. Case 1. Tracing taken 1 hour prior to onset of Stokes-Adams seizure on fourth hospital day. (Lead aVp.)

Discussion

The determining factor in each case appeared to be not the dosage of digitalis used but rather the pre-existing abnormality of conduction of the atrioventricular node. The longed P-R interval of 0.30 second. Over the period of 3 years, he developed repeated syncopal episodes. He was hospitalized because of syncopal attacks and varying cardiac rhythms (fig. 6A and B). Because of increasing congestive heart failure he was given 2.25 mg. of digoxin over a 4-day period.

Despite several Stokes-Adams seizures of short duration, he improved by the fifth hospital day, being alert, talkative, and without complaints. On the morning of the final hospital day the patient became extremely cyanotic and unresponsive, and sweated profusely. An electrocardiogram at this time showed atrial flutter with complete heart block. Over a period of an hour the patient's condition deteriorated and complete ventricular asystole occurred with persistent atrial flutter (fig. 7). Accordingly, the external electric pacemaker was applied, and the blood pressure and pulse rate were maintained artificially for 10 hours. No spontaneous ventricular activity appeared during this time, and gradually the clinical status deteriorated, despite the use of metaraminol and levaterenol in large quantities. The urinary output was good but the respiratory rate gradually slowed and eventually stopped, and the ventricles failed to respond to further excitation of the pacemaker.

Circulation, Volume XXIII, June 1961
lary escape. In the second case, prior to the onset of atrial flutter, there was first-degree heart block with a P-R interval of 0.30 second. In the first case, the depression of the atrioventricular node was manifest by the irregular ventricular response, which occurred in a pattern indicative of first- and second-degree atrioventricular block of the Wenckebach type (fig. 1). This important consideration is well pointed out by Besoain-Santander, Pick, and Langendorf, who considered the presence of an atrioventricular ratio greater than 2:1 to be evidence of a “disturbance of atrioventricular conduction corresponding to P-R prolongation during sinus rhythm.” The clinical importance of this electrocardiographic sign is attested by our two cases in which proper appreciation of atrioventricular depression might have prevented the serious consequences of drug therapy.
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Figure 5
Case 2. Electrocardiogram demonstrating prolonged AV conduction with normal sinus rhythm.

Figure 6
Top. Case 2. V1 on the day of admission demonstrating atrial flutter with irregular ventricular response. Bottom. V1 on following day showing normal sinus rhythm.

Figure 7
Case 2. Upper tracing shows atrial flutter with complete heart block and ventricular asystole with ventricular response to percussion (indicated by black dots). Middle tracing shows pacemaker driving the ventricle and atrial tachycardia. Lower tracing shows absence of spontaneous ventricular activity on discontinuation of pacemaker.
In view of the possibility of producing atrioventricular block during the conversion of atrial flutter to sinus rhythm, it is well advised that constant cardiac monitoring, with a pacemaker-monitor be undertaken in all cases showing impaired conduction. Certainly in the first case, the use of an electric pacemaker\(^5\)\(^,\)\(^6\) was lifesaving in maintaining ventricular activity until the drug effects had subsided.

**Summary**

Digitalis may have an additive effect on impaired atrioventricular conduction, so that atrial flutter with variable ventricular response may progress to complete heart block with ventricular asystole.

Two cases are presented of atrial flutter with variable ventricular response in whom ventricular standstill occurred during digitalis therapy.

External electric stimulation of the heart can maintain the circulation in digitalis-induced ventricular standstill.

**References**


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Do not rashly use every new product of which the peripatetic siren sings. Consider what surprising reactions may occur in the laboratory from the careless mixing of unknown substances. Be as considerate of your patient and yourself as you are of the test-tube.—SIR WILLIAM OSLER. *Aphorisms from His Bedside Teachings and Writings.* Edited by William Bennett Bean, M.D. New York, Henry Schuman, Inc., 1950, p. 103.
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SEYMOUR B. LONDON and ROSE E. LONDON

Circulation. 1961;23:920-924
doi: 10.1161/01.CIR.23.6.920

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