Simultaneous Block of the Normal AV Pathway and Abnormal AV Bypass Tract by Digitalis Intoxication in a Patient with Type A WPW Syndrome*

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A comprehensive study on the nodal rhythm was made by ROTHBERGER and WINTERBERG and by SCHERF and COHEN. Numerous papers appeared also in regard to the anatomy and pathophysiology of the WPW syndrome, AV pathway and its bypass. These experimental and clinical studies brought forth some new concepts such as an aberrant ventricular conduction, a dual AV nodal conduction system, ventricular echoes, a supernormal conduction through the anomalous bypass, a concealed AV conduction, a functional bypass of the AV node in the WPW phenomenon and so on. Histopathological demonstration of a case of ventricular pre-excitation terminated in complete AV block by LEV et al. sufficiently illustrated the complexity of the anatomy and pathophysiology of the AV pathway and its bypass. In their case, there was no bundle of KENT, but unusually copious MAHEIM fibers were responsible for a delta wave. Evidence for an acquired type of WPW syndrome was presented by PRINZMETAL et al. and by SPECICHER and KLEPZIG.

The patient presented in this paper is a case in which digitalis intoxication induced a simultaneous block of the normal AV pathway and abnormal AV bypass tract. An effort was made to explain the site of block and the origin and pathway of the QRS complexes observed.

CASE REPORT

Early in September, 1966, T.Y., a 65-year-old male first noted an irregular pulse. On April 28, he consulted a physician because of vertigo, and a diagnosis of hypotension was made. On May 2, he consulted another physician who indicated a presence of arrhythmia, edema and pulmonary congestion. In spite of medical treatment and bed rest, orthopnea and cyanosis appeared on May 15, and he was sent to the Yamaguchi Red Cross Hospital on June 3, 1966.

On admission, physical examination disclosed a tall obese man weighing 66 kg, a manager of a motor car repair-shop. Physical examination revealed dry and moist rales all over the chest, and cyanosis was marked on the lips, fingers and toes. The heart rate was more than 150/min, and the pulse rate at the radial artery ca. 60/min. The blood pressure was 130/80 mmHg. The abdomen was distended, but neither the liver nor the spleen was distinctly palpated. A presence of ascites was obscure because of the adipose abdominal wall. Pitting edema was present on the lower extremities.

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He showed ortho- and tachypnea. The body temperature was 38.4°C. Immediately after admission, he was placed on a reclining bed, oxygen inhalation instituted, and 0.5 cc of 10 per cent phenobarbital injected. Then, 200 mg of procainamide and 0.5 mg of Alotec (isoproterenol derivative) were given every 8 hours, and 2 cc of neophyllin-M (dihydroxypropyl-theophyllin) every 12 hours. Streptomycin and penicillin were also given for the first 10 days. On the following day, June 4, ECG clearly demonstrated a presence of “f” waves, and thus the basic rhythm was revealed to be atrial fibrillation with rapid ventricular response. Immediately, digitalization was completed within 24 hours with lanatoside C intravenously, and 0.4 mg was given daily as maintenance dosis for the following 3 days. A chest X-ray taken on admission showed pulmonary congestion, an elevation of the diaphragm and a marked cardiac enlargement to both sides with a cardiothoracic ratio of ca. 70 per cent. Dyspnea decreased day by day and disappeared on June 6, when the heart rate was ca. 78/min, the pulse rate ca. 56/min and the blood pressure 100/70 mmHg. From June 4, oral digitalization was instituted with digitoxin tablets. The patient vomited, and the heart rate was 52/min on June 9. Therefore, digitoxin was stopped for 2 days, when ECG showed complete AV block, atrial fibrillation and normally configured nodal QRS complexes. Medical treatment consisting of digitoxin, Fluitran (trichlormethiazide), Amisaline (procaine amide), Segontin, Aspara-M (K-asparat and Mg-asparate) and Alotec was instituted again from June 11 and continued unchanged until discharge. Afterwards, in spite of consistent medical treatment, ECG showed various changes as described below.

Laboratory examinations on admission disclosed the following: red cell count $372 \times 10^4$, Hb 86 per cent, white cell count 9600 (neutrophils 78 per cent), serologic reactions for syphilis all negative, CRP +, RA-Test negative, ASLO 50 Todd units, icterus index 8, zinc turbidity test 12.0, serum total protein 7.0 g/dl, A/G ratio 0.85, alkaline phosphatase 2.3 Bodansky units, GPT 12 Sigma-Frankel units, GOT 21 Sigma-Frankel units, serum total cholesterol 172 mg/dl. Glucose tolerance curve was diabetic with fasting blood sugar of 129 mg/dl and the highest blood sugar of 195 mg/dl 1 hour after ingestion.

**Electrocardiographic Follow-up and Discussion**

In this paper, “anomalous QRS complexes” will be used to those widened, bizarre QRS complexes which differ from normally configured QRS complexes, including the QRS complexes of aberrant ventricular contraction and ectopic ventricular beats, “aberrant conduction” not only to abnormal intranodal and intraventricular conduction but also to accelerated conduction by way of the accessory pathway (pre-excitation or WPW phenomenon) and “ectopic beats” to any cardiac contraction of an ectopic impulse.

As shown in Fig. 1, ECG on June 3 simulated ventricular tachycardia. One QRS complex in V6 (7th beat) which terminated a relatively longer cycle showed a normal configuration, which would indicate that the pathway of the anomalous QRS complex was refractory at this moment, and this normally configured QRS complex might be a nodal escape. On the morning of June 4, a continuous ECG registration disclosed a presence of “f” waves, indicating that the basic rhythm was atrial fibrillation with rapid ventricular response. Therefore, intravenous digitalization was instituted immediately with lanatoside C. On June 5, the “f” waves became more distinct, and the QRS complexes showed a widened, bizarre, anomalous configuration. The duration of the QRS complexes was more than 0.12 sec, and the QRS complexes in all unipolar precordial leads were upright, indicating that these anomalous QRS complexes were due neither to complete bundle branch block nor to ventricular extrasystoles but to the pre-excitation syndrome type A. Therefore, an excitation which has occurred in the atrium reaches the ventricle through an AV bypass tract. On June 6 (Fig. 2), the heart rate was markedly decreased, and some of the QRS complexes showed a normal configuration as observed in V2, V3, V5 and V6. Most of these normally configured QRS complexes terminated a longer cycle as observed in V2 and V3.
which would indicate that these normally configured QRS complexes might be AV nodal escaped beats. Or it may be that an excitation in the atrium reaches the ventricle through the normal AV pathway because of a prolonged refractory period in the AV bypass tract by digitalization. Lanatoside C was discontinued on June 7. Oral digitalization with digitoxin which had been instituted on June 4 was completed on June 8 and maintained with 0.1mg of digitoxin daily except on June 9 when nausea occurred. On June 11 (Fig. 3), ECG showed atrial fibrillation and intermittent complete AV block with normal and anomalous QRS complexes. Namely, when complete AV block was present, ECG showed a nodal rhythm in atrial fibrillation with normally configured QRS complexes. This would indicate that, as observed in lead I, both normal and abnormal AV pathways were simultaneously blocked at

Fig. 1. ECG on June 3, 1966
Atrial fibrillation with rapid ventricular response and anomalous QRS complexes. The heart rate is ca. 200/min. The 7th beat in V4 terminating a longer cycle may be a nodal escape. The QRS complexes in all precordial leads were upright, indicating that these wide, bizarre QRS complexes are due to type A WPW syndrome.

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this moment because of digitalis intoxication, and the pacemaker was shifted to the AV node. The site of the pacemaker must have been above the bifurcation of the common bundle, and the AV block must have occurred proximal to this pacemaker. The basic rhythm in lead II was also atrial fibrillation with complete AV block, and the 3rd beat might be an ectopic beat originating in the AV bypass tract, involving antegrade (downwards) the AV nodal pacemaker. Complete AV block was intermittent because the RR interval varied markedly in leads III and aVR, and a normally configurated AV nodal QRS complex alternated irregularly with an anomalous QRS complex. This seemed to indicate that the basic rhythm was atrial fibrillation, and an excitation in the atrium reached the ventricle through either the normal AV pathway or the abnormal AV bypass tract according to their refractoriness. On

Fig. 2. ECG on June 6, 1966
Atrial fibrillation with normal and anomalous QRS complexes. The heart rate is ca. 85/min. Normally configurated QRS complexes in V2 and V3 terminate a longer cycle, indicating that the refractory period of the anomalous AV bypass tract is prolonged by digitalis intoxication, and the AV nodal pacemaker is made to escape.

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June 13, ECG still showed atrial fibrillation and intermittent complete AV block with normal and anomalous QRS complexes. On June 16 (Fig. 4), complete AV block disappeared, and the basic rhythm returned to atrial fibrillation with an increase in the heart rate. Most QRS complexes showed an anomalous configuration similar to those on admission. Some QRS complexes showed a normal AV nodal configuration, being followed by an anomalous QRS complex dissimilar to those on admission (I and aVR). The coupling interval of the normally configured AV nodal QRS complex with the anomalous QRS complex was the same in all leads (0.40 sec.), which might indicate that these anomalous QRS complexes were due to idioventricular beats (I and aVR) or idionodal beats by way of the AV bypass tract (aVF). Another explanation for the pairing of a normally configured nodal escaped beat with an anomalous premature beat in aVF is that an antegrade spread of an excitation in the AV node induces the normally configured nodal QRS complex and its retrograde spread returns to the ventricle by way of the AV bypass tract in a sense of ventricular echo. On June 18, the basic rhythm was atrial fibrillation, and most of the QRS complexes showed anomalous configurations similar to those on admission. On June 20 (Fig. 5), the basic rhythm was sinus rhythm mostly with anomalous QRS complexes. The configuration of several supraventricular premature beats was the same as that of the basic one. This would indicate that the pathways of the basic and premature contractions were the same. There was one apparently normal QRS complex in lead II (3rd strip) which had a normal PQ interval and no delta wave. On June 27, atrial fibrillation with rapid ventricular rate similar to that observed on ad-

![ECG on June 11, 1966](image)

Atrial fibrillation with intermittent complete AV block and mostly with normally configured QRS complexes. The heart rate is ca. 35/min. Complete AV block is observed in leads I and II. The 3rd beat in lead II may be an ectopic premature contraction originating in the anomalous bypass tract, involving antegrade (downwards) the AV nodal pacemaker. Complete AV block is disappeared in leads III and aVR. The RR interval in these leads varied markedly, and the normal QRS complex alternates irregularly with the anomalous one. All time intervals are expressed in hundredth of a second.

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mission occurred after he had a visitors and mentally exhausted. It continued for about 12
hours and subsided spontaneously. On July 1
(Fig. 6), the basic rhythm was sinus rhythm
with apparently normal (II and aVR) and anomalous (I, III, aVL and aVF) QRS com-
plexes. The ECG on July 1 (Fig. 6) was differ-
ent from that on June 20 (Fig. 5) in that the
PQ interval of the basic QRS complex was
longer and the duration of the QRS complex
shorter, taking a more normal configuration.
Only the supraventricular premature beats by
way of the AV bypass tract resembled those
on June 20 and those on admission (pre-exci-
tation contour). This difference could be attri-
buted to a difference in the amount of ventric-

![ECG on June 16, 1966](image)

Atrial fibrillation mostly with anomalous QRS complexes. The heart rate is
cia. 60/min. A few normal QRS complexes terminating a longer cycle are paired
with an anomalous QRS complex which is dissimilar to the anomalous QRS
complex of pre-excitation contour on admission (I and aVR). The coupling
interval of the normally configured AV nodal QRS complex with the ano-
malous QRS complex is the same in all leads (0.40 sec.), which may indicates
that these anomalous QRS complexes are due to idioventricular beats (I and
aVR) or idionodal beats by way of the AV bypass tract (aVF) or a ventricular
echo by way of the AV bypass tract to the nodal escaped beats (aVF).

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ular musculature undergoing premature excitation. The absence of a delta wave and the presence of apparently normal QRS in leads II and aVR could be explained by the position of electrodes in these leads, i.e., the direction of electrical propagation of an initial portion of an aberrant ventricular activation might be at right angles (tangent) to the right arm and vertical to the lead II axis, making the initial portion of the QRS to be isoelectric. That the configuration of the basic QRS complex differed only slightly from that of the supraventricular premature QRS complex in leads III and aVF is evidence that the pathway of the premature

Fig. 5. ECG on June 20, 1966
Sinus rhythm with anomalous QRS complexes associated with supraventricular premature contraction of anomalous configuration. The heart rate is ca. 60/min. The 4th beat in lead II (3rd strip) is apparently normal. In this record, the configuration and amplitude of the basic and premature QRS complexes are the same, indicating that the pathway of the basic and premature ventricular contraction are the same.

excitation was not too far removed from that of the basic excitation. The anomalous QRS complexes in leads I, III, aVL and aVF were similar in configuration to those observed on admission, but their amplitude and duration on July 1 were less than those on admission. This difference could be ascribed to a difference in the amount of ventricular musculature undergoing pre-excitation which might be a result of the fact that the basic rhythm on admission was

Fig. 6. ECG on July 1, 1966

Sinus rhythm with more normally configurated basic QRS complexes and supraventricular premature beats of anomalous QRS complexes similar to those on admission and those on June 20 (pre-excitation contour). The heart rate is ca. 80/min. The difference in configuration between the basic QRS complexes on June 20 and those on July 1 and that between the basic and the premature QRS on July 1 may be attributed to a difference in the amount of ventricular musculature undergoing premature excitation. An absence of a delta wave and a presence of an apparently normal QRS complex in leads II and aVR can be explained by the position of electrodes in these leads. Namely, the direction of electrical propagation of an initial portion of an aberrant ventricular activation may be at right angles (tangent) to the right arm and vertical to the lead II axis.
atrial fibrillation and that on July 1 sinus rhythm. In configuration, the anomalous premature QRS complexes were similar to the anomalous basic QRS complexes. The amplitude of the anomalous premature QRS complexes was, however, greater than that of the anomalous basic QRS complexes and equal to that of the anomalous QRS complexes on admission. This might indicate that the pathway of ventricular excitation in atrial fibrillation was the same as that of premature ventricular contraction. Or it might be that the configuration of the QRS complex in sinus rhythm was due to fusion of initial ventricular activation by way of the anomalous pathway and activation by way of the normal AV pathway, whereas the

Fig. 7. ECG on July 16, 1966
Sinus rhythm with anomalous QRS complexes in the form of type A WPW syndrome. The heart rate is ca. 60/min. The basic QRS complexes are similar to those on admission in configuration but not completely the same. This difference in configuration and amplitude can be ascribed to a difference in the degree of ventricular aberration partly due to the difference in the basic rhythm which is atrial fibrillation on admission and sinus rhythm on July 16.

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configuration of the QRS complex of premature beats in sinus rhythm and beats in atrial fibrillation was due to ventricular activation entirely by way of the anomalous pathway. On July 16 (Fig 7), ECG showed sinus rhythm with WPW syndrome type A associated with supraventricular premature beats of anomalous configurations. The basic QRS complexes were similar to those on admission in configuration but not completely the same. This difference in configuration and amplitude could be ascribed to a difference in the degree of ventricular aberration possibly due to the fact that the basic rhythm on admission was atrial fibrillation and that on July 16 sinus rhythm.

Identification of a fusion beat is difficult in the presence of atrial fibrillation and in the absence of ectopic nodal or ventricular rhythm. In this patient, no distinct fusion beats were observed even when the basic rhythm showed sinus rhythm or complete AV block. Identification of parasystole is impossible unless there are at least more than 3 QRS complexes of another origin in one strip. In all strips registered, no parasystole was observed except for extrasystolic bigeminy. Although a large number of arrhythmic influence including mental excitement and overdoses of various drugs increase the degree of nonuniformity of the ventricle, nonuniform recovery of excitability may be a factor of major importance in enhancing the propensity of a heart to the formation of extrasystoles, fibrillation and ventricular aberration.

**Summary**

A 65-year-old male entered the Yamaguchi Red Cross Hospital with a tentative diagnosis of ventricular tachycardia. However, continuous ECG registration disclosed a presence of "f" waves, indicating that the basic rhythm was atrial fibrillation with a rapid ventricular response. Immediately, a rapid digitalization was instituted with lanatoside C. After digitalization, ECG showed a) atrial fibrillation with anomalous QRS complexes in the form of type A WPW syndrome, b) atrial fibrillation with intermittent complete AV block with normally configured AV nodal QRS complexes, c) atrial fibrillation with normal and anomalous QRS complexes and idioventricular beats following the normally configured nodal QRS complexes, d) sinus rhythm with apparently normal and anomalous QRS complexes and supraventricular premature beats of anomalous configuration and e) sinus rhythm with anomalous QRS complexes in the form of type A WPW syndrome. The duration of the anomalous QRS complex in sinus rhythm was shorter than that in atrial fibrillation.

**References**


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