Case Report

A Case of Ventricular Return Extrasystoles with Two Consecutive Re-entry Sweeps to the Atrium

Yasuki Sakamoto, M.D., Kazumasa Hiejima, M.D., Shigeru Tsuchiya, M.D., and Toyomi Sano, M.D.

Summary

A case is presented of ventricular return extrasystoles showing 2 consecutive re-entry sweeps to the atrium. These rare arrhythmias were observed in a patient with atrial septal defect, who had received digitalis for a long period.

Additional Indexing Words:
Retrograde P wave  Re-entrant P wave  Pseudoreciprocal beat  Wandering pacemaker

Although there have been many case reports on the return extrasystoles, the number of those on which diagnosis was established is relatively small.1) Above all, the arrhythmia due to 2 or more successive re-entries is even rarer than single return extrasystoles.2)-5)

The following case of ventricular return extrasystoles documents 2 consecutive re-entries to the atrium in a patient maintained on digitalis for a long time.

Case Report

A 51-year-old woman was admitted twice to the Tokyo Medical and Dental University Hospital, for recurrent congestive heart failure due to atrial septal defect, on November 7, 1967, and September 16, 1968, respectively.

On the second admission, there were edema of the extremities, cyanosis of lips and distension of the neck veins. Her pulse was 40 per min. and was bigeminal. The blood pressure was 130/70 mm. Hg. The peripheral venous pressure was 220 mm. H2O.

Auscultation of the chest revealed a loud, grade 4/6, systolic ejection murmur at the pulmonic area and a harsh, grade 5/6, systolic regurgitant murmur in the...
5th intercostal space along the left sternal border. Fixed splitting of the second heart sound was present. These findings were consistent with the presence of atrial septal defect associated with relative tricuspid insufficiency previously diagnosed by

![Fig. 1](image1.png)

**Fig. 1.** An ECG showing complete right bundle branch block and right ventricular hypertrophy. The patient was digitalized by 0.1 mg. of digitoxin per day.

![Fig. 2](image2.png)

**Fig. 2.** A shows atrial premature beats in bigeminy. B shows frequent A-V junctional or ventricular premature beats with retrograde P waves in bigeminy.
catheterization. Rales were audible in the lungs.

Liver was palpable 6 cm. below the right costal margin in the midclavicular line.

Abnormal laboratory data were as follows: total bilirubin, 2.5 mg./100 ml.; alkaline phosphatase, 27.2 KAU; GOT, 50 units.

A chest X-ray film showed enlargement of the right cardiac contour and of the main pulmonary arteries.

The patient had received digitalis for about 5 years. On readmission, there were no signs due to digitalis intoxication, except for cardiac arrhythmias. The ECGs shown in Fig. 3 to Fig. 6 were recorded at that time.

The ECGs shown in Fig. 1 and Fig. 2 were taken before the present readmis-

Fig. 3. These tracings show A-V junctional or ventricular premature beats with fixed coupling and ventricular return extrasystoles. All numbers in each diagram correspond to hundredths of a second. See text for detail.

Fig. 4. The upper strip is an esophageal ECG, taken at 35 cm. from the lower lip. These strips show pacemaker wandering to the A-V junction and retrograde P waves.
sion when she was also receiving a maintenance dose of digitalis: an ECG taken on May 13, 1967 (Fig. 1) shows complete right bundle branch block and right ventricular hypertrophy, and Fig. 2, recorded on December 21, 1967, shows atrial premature beats in bigeminy in A and frequent ventricular or A–V junctional premature beats with retrograde P waves also in bigeminy in B. These retrograde P waves are diagnosed as such because of the negativity in leads II, III and aVF, and are confirmed by the esophageal ECG shown in Fig. 4.

In Fig. 3A taken on readmission, ventricular or A–V junctional premature beats with fixed coupling are repeated until R₄. It is impossible to tell whether this bigeminy is produced by the A–V junctional or ventricular focus, without more information. Therefore, ECGs are diagrammatized tentatively as shown in Fig. 3 and 6.

All these premature beats are followed by retrograde P waves. Each R–P interval of the premature beats is almost the same, being 0.16 sec. The configuration of the P wave and the P–P interval and R–R interval of the dominant rhythm are variable, suggesting that there is a wandering pacemaker. This is also ascertained by the esophageal ECG in Fig. 4, where the pacemaker resulting in R₃, R₄ and R₆ seems to be wandering from the S–A node to the region near the coronary sinus ostium. On the other hand, at R₇ in Fig. 3A, the R–R' interval is shortened.

Fig. 5. These ECGs are presented to show retrograde P waves and return extrasystole.

Fig. 6. A reveals change of configuration of antegrade and retrograde P waves. B shows wandering pacemaker and bigeminy. C. This tracing is analysed as in the diagram below, i.e., pseudo and true return extrasystoles. See text for detail.
suddenly from 0.48 sec. to 0.42 sec., whereas the R–P’ interval (the interval between
the beginning of the QRS complex and the retrograde P’ wave) of R₈ is prolonged
from 16 to 20, and the retrograde P’ wave is followed by a QRS complex (R’’=R₉)
showing the pattern of left bundle branch block. This second QRS complex (R₉)
is found to occur only with such prolongation of the R–P’ interval. Since this pro-
longation probably allows retrograde atrial impulse to return to the ventricle, this
beat can be interpreted as a return extrasystole. Fig. 5 shows a retrograde P’ wave
and a return extrasystole in lead aVF, as well. Furthermore, the return extrasys-
toles of R₃ and R₈ in Fig. 3B are followed by P waves showing the same pattern as
P’ wave. Hence, these P waves are explained as the second re-entry to the atrium
(P’’ wave). A similar P’’ wave is also seen in Fig. 6C. At all events, it is note-
worthy that the R’–P’’ interval is considerably longer than the R–P’ interval and
that no distinctive P waves are ever observed following the second re-entrant P
waves, differing from the other single re-entry to the atrium (Fig. 3A).

The wandering pacemaker is conspicuous in Fig. 6, in which the 5th beat in
B is so-called upper A–V junctional in origin. The configuration as well as the R–P’
interval of retrograde P’ waves are also variable as seen in Fig. 4 and 6A.

In the QRS–P–QRS complex at the initial part of Fig. 6C the R–P’ interval is
shorter than those mentioned above. It may have turned back to the ventricle at
the lower part of the A–V junctional tissue, or this may be a pseudoreciprocal beat.
The diagram is shown according to the latter interpretation.

The complicated arrhythmias disappeared following discontinuation of digitalis,
but such a bigeminy as shown in Fig. 2B has appeared occasionally until present.

**Discussion**

The diagnosis of a ventricular return extrasystole is frequently difficult and
misleading. According to Cohen,1) the diagnosis is made when an A–V beat
of the variety with a P wave following the QRS complex or a QRS complex
resulting from an ectopic ventricular beat is followed by a retrograde P wave
with an abnormally long R–P interval and, shortly thereafter, by a second pre-
mature complex. In this context, QRS–P–QRS complexes as shown in Fig. 3,
5 and 6C were diagnosed to be ventricular return extrasystoles, because of the
existence of retrograde P waves and appearance of those complexes only in the
presence of prolonged R–P interval.

In Fig. 3B and 6C there were the 2nd retrograde P’’ waves without the
succeeding ventricular activation, in spite of the fact that the R’–P’’ intervals
were far more prolonged than the R–P’ intervals. The reason why the 2nd
ventricular activation did not occur is not clear. However, it is reasonable to
assume as follows: the mechanism underlying the return extrasystoles has been
interpreted by assuming a longitudinal dissociation of the A–V junctional tissue
which is produced by the existence of dual or multiple pathways within it.6)-11)
The concept of the dual pathway of the A–V node may not be literally untena-
ble12),13) now, but, since the A–V junctional tissue consists of a net work of inter-
communicating smaller fibers\textsuperscript{14},\textsuperscript{15} which have a different refractoriness,\textsuperscript{16} repeated re-entry sweeps can occur, such as A–V junctional tachycardia.\textsuperscript{17},\textsuperscript{18} In this case, because of the relative refractoriness in the A–V junctional tissue after the passage of the preceding impulses, the 2nd retrograde ones took a longer time. Meanwhile, the other region of the A–V junction, showing a longitudinal dissociation in the early phase, was able to recover more completely. Then, this impulse excited most of the cells of the A–V junction, leaving few cells available for propagation of the impulse downward.

As for the reason why no P waves were present following the 2nd re-entrant P' waves, 3 mechanisms can be considered. The first is the most likely one, in which the 2nd retrograde P' wave discharged the sinus node. This was going to renew its rhythm, but the A–V junctional escaped beat (R\textsubscript{4} or R\textsubscript{9} in Fig. 3B or the last QRS but one in Fig. 6) appeared slightly ahead of it; the 2nd, a less likely one, is that, on the way where the 2nd retrograde impulses (arrows in Fig. 3B) descended from the atrium, a part of the excitation wave turned retrograde to the atrium again. Although the impulses were blocked before the arrival at the atrium, they returned to the ventricle with a considerable delay, which show R\textsubscript{4} and R\textsubscript{9}; the 3rd one, which is also less likely, is as follows: a small movement of the base line is noticed before R\textsubscript{4}. This is probably an artifact, but, since the interval between the beginning of the first P wave to this movement in Fig. 3B is almost equal to the R\textsubscript{3}–R\textsubscript{5} or R\textsubscript{5}–R\textsubscript{7} interval in Fig. 3A, it cannot be ruled out to be a rudimentary P wave. Its diminution may be due to the fact that the sinus node impulse encountered with the refractory period of the majority of the atria, which would be caused by the 2nd re-entry.

Return extrasystole is found most often in the presence of A–V junctional rhythm.\textsuperscript{4} It cannot be denied, however, that extrasystoles, in our case, showing the pattern of left bundle branch block are ventricular in origin since 85 per cent of the return extrasystoles originating from the A–V junctional tissue was reported to show the pattern of right bundle branch block.\textsuperscript{19},\textsuperscript{20}

Ventricular return extrasystoles with their site of origin in the A–V junction have been encountered in digitalis intoxication or sensibility, atherosclerotic heart disease, rheumatic carditis, diphtheria, congenital heart disease, during carotid sinus stimulation and with instability of the autonomic nervous system.\textsuperscript{1} In our case, the return extrasystoles disappeared following discontinuation of digitalis. However, since many factors as described above were present in this case, the pathogenesis of the return extrasystoles is not attributable to digitalis only.
References