Accelerated Junctional Escape
A Clinical and Electrocardiographic Study

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SUMMARY
During the routine interpretation of electrocardiograms, 49 cases of spontaneously occurring accelerated, or borderline accelerated, junctional escape complexes (escape intervals of 1200 ± 20 msec) were encountered. Forty-seven of the 49 patients whose cardiograms demonstrated the accelerated junctional escapes had the clinical setting recognized to be associated with the more protracted accelerated junctional rhythm, nonparoxysmal junctional tachycardia. In addition, five patients demonstrated nonparoxysmal junctional tachycardia immediately preceding or subsequent to the cardiograms demonstrating accelerated junctional escape. It is suggested, therefore, that the two arrhythmias, i.e., accelerated junctional escape and nonparoxysmal junctional tachycardia, may be of similar clinical significance.

The determinants of the length of the manifest escape interval could be assessed in those cases where two or more junctional escape complexes occurred in sequence, allowing the true interectopic interval to be ascertained. The dominant role of the rate of atrioventricular conduction and the sequence of ventricular and junctional activation in determining the length of the manifest escape interval was apparent.

Additional Indexing Words:
Accelerated automaticity Escape rhythms Nonparoxysmal junctional tachycardia

PATHOLOGICAL ACCELERATION of ectopic pacemaker activity within the specialized conduction system may not become manifest if the dominant rhythm, usually supraventricular, discharges at a faster rate. Slowing of the dominant rhythm by interventions such as carotid sinus pressure in man or direct electrical stimulation of the heart or vagus nerve in the experimental animal has been used to unmask suppressed ventricular rhythms arising in the Purkinje system, distal to the atrioventricular junction.1-4 Similarly, pathologically accelerated junctional rhythms, nonparoxysmal junctional tachycardia (NPJT), may remain suppressed by a more rapidly discharging higher focus only to become manifest either with slowing of the dominant pacemaker or acceleration of junctional ectopic pacemaker, or both.5, 6

Isolated accelerated junctional escape impulses appearing spontaneously in man have received little or no attention. It is the purpose of this report, based on analysis of 49 routine cardiograms 1) to illustrate unmasking of accelerated junctional escape impulses by a variety of spontaneous mechanisms, 2) to show that the escape complexes occur in the same clinical setting as NPJT and thus to suggest that they should perhaps be viewed clinically in the same light as NPJT,6, 7 and 3) to present some of the more interesting electrocardiographic mechanisms accompanying accelerated junctional escape.

Material and Methods
Forty-nine cardiograms with sinoatrial rhythm (SA) interrupted by accelerated junctional escape complexes, the escape interval being 1200 ± 20 msec or less, were collected in the course of routine interpretation of tracings and correlated with clinical findings. The reasoning for including escape intervals up to 1200 msec is discussed below.

The escape cycle was measured in milliseconds as the interval from the beginning of the R wave preceding the escape complex to the beginning of the escape R wave and is referred to as the true junctional interectopic interval.

In instances where two or more junctional complexes followed the manifest escape pause, the interectopic junctional cycle length was determined. This interval is referred to as true junctional interectopic interval.

For each tracing the following were determined: 1) the rate of the basic, dominant pacemaker, 2) the manifest escape interval, 3) mechanism permitting an escape, 4) the true cycle length of the ectopic junctional pacemaker, 5) number of consecutive ectopic junctional complexes, and 6) clinical setting accompanying the accelerated escape rhythm.

Results
The clinical data and electrocardiographic (ECG) findings for each case are presented in table 1.
Table 1

Electrocardiographic and Clinical Data for 49 Cases of Accelerated Junctional Escape Impulses

<table>
<thead>
<tr>
<th>No.</th>
<th>Dominant R-R*</th>
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<th>True junctional rate‡</th>
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*All intervals are in msec.
†See Methods for derivation.

Abbreviations: APS = atrial premature systole; VPS = ventricular premature systole; MI = myocardial infarction; Post op = post open heart surgery; JPS = junctional premature systole; SA = sinoatrial; VT = ventricular tachycardia.
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The various ECG mechanisms permitting accelerated junctional escape are illustrated in figure 1.

Mechanism Permitting Escape

In 24 instances atrial premature systoles (APS) were responsible for the pause allowing the junctional pacemaker to escape (fig. 2, 3, 4). The average SA rate in this group was 98.4 beats per minute (an R-R interval of 610 msec, range 440-880 msec) with an average escape interval of 763 msec (range 540-1000 msec) (fig. 5, 6). In 13 instances, ventricular premature systoles (VPS) preceded the junctional escape. The average SA rate for this group was 82.1 beats per minute (an R-R interval of 731 with a range of 520-920 msec) and a junctional escape interval of 923 msec (range from 680 to 1220 msec). In six cardiograms SA slowing or SA depression and in five, Wenckebach Type I A-V block (fig. 7) were the mechanisms responsible for the interval allowing the junctional focus to escape. In cases in which SA slowing or depression was the cause, the average SA rate was 73.3 per minute (R-R 818 msec, range of 720-1000 msec). With Wenckebach block the SA rate was 92.3 beats per minute (R-R of 650 msec with a range of 520-780 msec). The average escape interval was 910 msec (range from 720-1000 msec) and 848 msec (range 840-1060 msec), for SA depression and Wenckebach A-V block respectively. In one instance, concealed junctional premature impulses resulted in a compensatory pause permitting junctional escape. The SA rate in this patient was 83 beats per minute, R-R of 720 msec and the manifest escape interval was 960 msec (fig. 8).

The Manifest Escape Interval and the True Interval

Twenty-three of the 49 cardiograms demonstrated a single, isolated escape while in 26, two or more consecutive junctional impulses were recorded (table 1, figures 4, 6, 8). In 14 of the 26 both the manifest escape interval and the true junctional rate were the

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Lewis diagram demonstrating the various mechanisms permitting accelerated junctional escape (SA = sinoatrial node; A = atrium; J = junction; V = ventricle; E = manifest escape; J = interjuncional cycle). 1) Conducted atrial premature systole (APS) with escape and true interjuncational interval being equal. 2) APS blocked above the junctional site with the manifest escape and true interjuncional intervals being equal. 3) Ventricular premature systole (VPS) activating the ventricles before reaching the junctional site resulting in a manifest escape interval longer than the true interjuncional cycle length. 4) APS penetrating to the level of the junctional focus, discharging it, but failing to reach the ventricle, resulting in an escape interval longer than the true interjuncional interval. 5) Conducted APS with delay of conduction below the accelerated junctional focus with a resultant manifest escape interval shorter than the true interjuncional interval. 6) Concealed junctional impulse with manifest escape interval longer than the true interjuncional interval.

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Accompanied by a run of NPJ. The manifest escape interval in F measures 760 msec. In B, C, D, and E the escape interval cannot be estimated.

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Figure 2

Accelerated junctional escape complexes (marked by the dots) following conducted (A, B) and blocked (C) APS and VPS (D, E, F). In A the APS is followed by a manifest escape R-R of 660 msec and a true escape interval of 700 msec. In F the VPS is followed by a run of NPJ. The manifest escape interval in F measures 760 msec. In B, C, D, and E the escape interval cannot be estimated.

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Figure 3

Accelerated junctional escape following atrial premature systole (APS) with concealed A-V conduction. The actual junctional escape is best estimated from the cycle which follows the conducted APS. The nonconducted APS, on the other hand, reaches and discharges the junctional focus but fails to excite the ventricles (concealed conduction), thus the fictitiously long manifest escape interval encompassed by the last conducted sinoatrial (SA) impulse and the junctional escape.

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same (±20 msec). The mechanism of the pause allowing junctional escape in these 14 cases was APS in nine, SA slowing or depression in four, and Wenckebach A-V block in one. In the remaining cardiograms there was a discrepancy between the manifest escape interval and the true interjunctional interval, with eight manifesting a longer escape than true interval. The mechanism permitting escape in seven of these were VPS. In the four cardiograms demonstrating a shorter manifest escape than true interval, APS were the initiating event.

In five patients both isolated accelerated junctional escapes and protracted NPJT were recorded (fig. 9, 10).

Clinical Data

Twenty patients were receiving digitalis at the time the cardiograms were obtained, 16 patients were recovering from open heart surgery, and nine patients were being treated for acute myocardial infarction. Two patients demonstrated electrolyte disturbances while the two remaining patients had no demonstrable heart disease nor were they being treated with cardiac drugs at the time the records were obtained.

Discussion

Normally junctional pacemaking activity is passive and becomes manifest only with slowing, arrest, or failure of the primary, dominant pacemaker to penetrate the junction. The inherent physiological rate of a junctional pacemaker varies from 40 to 50 beats per minute, may be as slow as 30 beats per minute, but rarely exceeds 60 beats per minute. Consequently, a physiological junctional escape interval is rarely shorter than 1000 msec, escape intervals between 1000 and 1200 msec representing a gray zone for abnormal acceleration. In pathological situations the automaticity of a junctional pacemaker may be enhanced, resulting in NPJT. The rate of NPJT varies from 70 to 130 beats per minute. In seven of the 49 patients presented here, the manifest junctional escape interval was longer than 1000 msec, the duration varying from 1040 to 1220 msec. These borderline, or gray zone cases of accelerated junctional automaticity were included not only because of the difficulty in estimating accurately the true junctional rate when an isolated impulse occurs, but as will be discussed below, the true interjunctional interval may be less than 1000 msec in some of these patients. In addition, inclusion of these cases provided an opportunity to assess the clinical correlates of escape intervals in the borderline range. In 47 cases, accelerated junctional escape complexes occurred in the same clinical setting as does NPJT, namely, in digitalis intoxication, following open heart surgery, with acute myocardial infarction, and in electrolyte disturbance. In only two patients no heart disease was recognized nor were cardiac drugs being administered; these two patients

Figure 4

Accelerated junctional escape following APS. A. The manifest escape interval and true junctional cycle length are the same (650 msec), indicating that conduction below the junctional focus was the same for the atrial premature systole (APS) and junctional impulse. B. The escape interval (550 msec) is shorter than the true junctional cycle length (600 msec), suggesting that the APS was delayed in its conduction through the A-V junction. C. The APS is blocked above the junctional focus, does not interfere with its rhythmicity, and thus the escape and true interjunctional cycle length are approximately the same. The somewhat longer escape interval may be due to depression of the junctional focus by the sinus impulse.

Figure 5

Accelerated junctional escape following atrioventricular (A-V) junctional premature impulse. In V, the junctional focus is discharged by an atrial premature systole (APS) and terminated by an SA impulse 800 msec later. The manifest junctional escape cycle measures 920 msec in L2. In L3, the junctional impulse is delayed in its conduction to the atrium and the resultant distance between the retrograde F and subsequent SA impulse equals an SA cycle. The actual interval between premature ectopic junctional and the accelerated escape is longer than the manifest P-P, permitting a junctional escape.
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Figure 6

Accelerated escape after APS and VPS (atrial and ventricular premature systole). The escape follows a conducted APS in $L_2$. In each zone of $V_1$, the third P wave is an APS, conducted aberrantly, and is followed by a VPS. It may be, however, that both of the bizarre QRS represent VPS. The true interjunctional interval is 800 msec. The escape interval following the APS in $L_2$ equals the interjunctional interval (800 msec) while the escape interval following the premature ventricular contraction (PVC) is longer (920 msec).

were in the borderline group for accelerated junctional escape. Thus, while it cannot be assumed unequivocally that accelerated junctional escape complexes have the same significance as NPJT, the occurrence of the two in similar clinical situations tends to suggest such a relationship. Furthermore, the fact that in 25 patients, at one time or another, more than one escape complex was recorded and five patients exhibited protracted NPJT immediately prior to or subsequent to the tracing demonstrating accelerated junctional escape complexes, strongly supports an hypothesis of similar clinical significance of NPJT and accelerated junctional escape.

It is apparent that the manifest junctional escape interval, as determined from the surface cardiogram, may not reflect the true rate of the accelerated junctional pacemaker. This discrepancy occurs for a number of reasons. For example, it is impossible to determine with any degree of certainty when, or if, the junctional focus was influenced by an APS or VPS; or, in the case of a conducted APS, to be certain of the conduction time across the atrioventricular conduct-

Figure 7

The two $L_2$ strips were recorded a day apart. The upper row demonstrates atrioventricular (A-V) dissociation, nonparoxysmal junctional tachycardia (NPJT) with an interjunctional interval of 920 msec. The bottom tracing shows Wenckebach, Type 1 A-V block, each cycle terminated by an accelerated junctional escape.

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Isolated accelerated junctional escape (marked by the dot below) and nonparoxysmal junctional tachycardia (NPJT). Portions of a single tracing demonstrate isolated junctional escape in V_r. In aV_r and aV_r the QRS, identified by a question mark below, which follow the escape accelerated junctional escape may represent a second escape; this is suggested by the somewhat foreshortened P-R interval. V_r represents a run of NPJT interrupted by premature ventricular contraction (PVC). A-V dissociation is seen with the first three cycles following which P waves are no longer recorded. These may be buried in the junctional QRS, or the atrial activity may be suppressed. The manifest escape intervals are longer than the true interjunctional cycle because the ventricles are activated before the impulse reaches the junctional focus.

Figure 9

Accelerated junctional discharge on 3/13 and nonparoxysmal junctional tachycardia (NPJT) on 3/14. The junctional focus marked by the dot below the complexes during the NPJT probably activates the atrium as suggested by the terminal upright portion of the QRS, representing a retrograde P wave.

Figure 10

As a rule, when VPS initiate the compensatory pause permitting a junctional escape, the manifest escape interval is longer than the true interjunctional cycle (fig. 1-3). This is so because the junctional focus is discharged after the onset of QRS and thus the manifest escape interval appears longer than the true
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junctional rate. Less commonly, blocked APS, in which the block of the APS occurs distal to the site of the junctional focus, result in a manifest escape interval which is longer than true junctional interval (fig. 1-4, 3). Similarly, a concealed junctional discharge which fails to reach the ventricles may result in manifest escape cycle longer than the true junctional cycle (fig. 1-6).

All of the above mechanisms were seen in the present study. Ventricular premature systoles invariably discharged the ectopic junctional focus after the onset of QRS and in instances in which both the manifest escape and true junctional intervals could be determined, the manifest escape interval was longer than the true junctional interval. On the other hand, conducted APS sometimes were associated with manifest escape intervals shorter than true junctional intervals, but the disparity was much less pronounced than with VPS. The difference in the effect of APS and VPS on the length of the escape interval is not unexpected.

Atrial premature systoles affect the junctional focus before inscribing the QRS, whereas VPS inscribe the QRS before reaching the junctional ectopic focus. Blocked APS which do not disturb the junctional focus result in equal manifest escape and true ectopic intervals. On the other hand, if the impulse reaches and discharges the ectopic pacemaker without reaching the ventricles, the escape interval must be longer than the true interectopic junctional cycle length.

The phenomenon of suppression of a pacemaker (in this instance of the accelerated junctional pacemaker) by SA impulses, APS, or VPS was not a factor contributing to the duration of the manifest escape interval. If this had been the case, escape intervals longer than the true interjunctional cycle lengths should have been recorded with both APS and VPS. This, however, did not occur. With APS the manifest escape was shorter or equal to the true junctional cycle, except in one case (fig. 4). The dominant role of the rate of atrioventricular conduction and the sequence of ventricular and junctional activation in determining the length of the manifest escape interval is, by this reasoning, apparent.

It is of interest that the more rapid junctional escape impulses were recorded in association with the most rapid basic SA rates. While it is obvious that this would have to be so in order for the latent automaticity to become manifest, it also raises another question. Assuming that the rate of junctional and ventricular automaticity is influenced by the basic sinus rate and the mechanism for a pause allowing escape, as suggested by experimental data, are then the currently accepted rate changes for pathological acceleration of junctional automaticity too rigid? While this question cannot be answered with certain-
seven cases presented here with longer than 1000 msec manifest escape intervals were apparently without heart disease or other recognized concomitants of accelerated escape.

References
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