Comparison of Resetting and Entrainment of Uniform Sustained Ventricular Tachycardia
Further Insights Into the Characteristics of the Excitable Gap

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Background. Resetting and entrainment have both been used to characterize the electrophysiological properties of the reentrant circuit in ventricular tachycardia. Several entrainment studies have suggested that the circuit has decremental properties, because the return cycle increases at faster pacing rates. Resetting, however, demonstrates a fully excitable gap in the majority of tachycardias.

Methods and Results. The response to resetting and overdrive pacing was analyzed in 18 ventricular tachycardias. Resetting demonstrated some duration of a fully excitable gap in 14 of 18 tachycardias. Overdrive pacing was performed at several cycle lengths with an incremental number of stimuli (1–15 beats) such that the first beat that interacted with the tachycardia (the nth beat) could be identified. The return cycles measured during resetting and the nth beat of pacing were identical (r=0.99). At relatively long paced cycle lengths, paced beats after the nth beat resulted in a constant return cycle in most tachycardias with a fully excitable gap. At rapid paced cycle lengths, an increase in the return cycle from the nth to the nth+1 beat was associated with progressive prolongation in the return cycle with each incremental paced beat until a longer equilibrium return cycle was reached or the tachycardia terminated in response to pacing.

Conclusions. We propose that the responses to resetting and overdrive pacing with or without entrainment appear to provide conflicting information about the characteristics of the circuit because they in fact measure entirely different electrophysiological parameters. The nth beat of pacing foreshortens the excitable gap to the extent that it arrives prematurely. Subsequent paced beats interact with an altered tachycardia circuit that has had less time to recover excitability. Resetting is the interaction of a single paced beat with the tachycardia and, as such, provides a more accurate assessment of the characteristics of the unaltered tachycardia circuit. (Circulation 1993;87:1229–1238)

KEY WORDS • resetting • entrainment • tachycardia, ventricular • excitable gap

Resetting1–4 and entrainment5–13 are distinct programed stimulation techniques that have been used to characterize the electrophysiological properties of uniform sustained ventricular tachycardia in humans. Both techniques have provided important evidence supporting the reentrant mechanism of this arrhythmia in the setting of chronic coronary artery disease.4,6,13–16 However, the use of stimulation protocols without precise attention to methodology has led to conflicting interpretations of the properties of the proposed reentrant circuit. Several studies using entrainment have concluded that some portion of the tissue within the circuit has decremental properties. This conclusion is based on the observed increase in the interval from the pacing stimulus to the last orthodromically entrained electrogram at progressively faster pacing rates.11,17 Analyses using resetting with single or double extrastimuli, however, demonstrate some duration of a flat response curve in approximately 70% of ventricular tachycardias.1 Furthermore, the flat resetting response typically represents 10–25% of the tachycardia cycle length1 and suggests the presence of a fully excitable gap in all segments of the tachycardia circuit.1,18

The purpose of this study was to analyze the response of uniform ventricular tachycardia to resetting and overdrive pacing to explain this apparent discrepancy and to determine which technique best describes the characteristics of the tachycardia circuit.

Methods

Study Population

The study group included 17 men and one woman ranging from 44 to 78 years of age. All patients had chronic coronary artery disease with healed myocardial infarction. The mean left ventricular ejection fraction was 31±14%. A single morphology of sustained, well-tolerated ventricular tachycardia from each of the 18 patients was included in the study. The average VT cycle length was 399±47 msec (range, 320–490 msec). Two patients were receiving no antiarrhythmic medica-
tions at the time of the study; six patients were receiving propafenone, seven patients were receiving type I agents with (two patients) or without (five patients) a type IB agent, and three patients were receiving amiodarone. All patients were undergoing electrophysiologic study for evaluation of spontaneous episodes of uniform sustained ventricular tachycardia or aborted sudden cardiac death, and all had reproducibly inducible ventricular tachycardia during programmed stimulation. Written, informed consent was obtained from all patients. The study protocol was approved by the Institutional Review Board of the University of Pennsylvania.

**Stimulation Protocol**

The stimulation protocol used in our laboratory for the induction of ventricular tachycardia has been described previously. Briefly, single, double, and triple ventricular extrastimuli were delivered, scanning diastole, during paced drives at two or more cycle lengths from the right ventricular apex and outflow tract. Left ventricular stimulation was performed from at least one site if ventricular tachycardia was not induced with right ventricular stimulation. Synchronous square waves with a pulse width of 1 msec were delivered at twice diastolic threshold from a custom-designed programmable stimulator (Bloom Associates, Reading, Pa.). Five surface ECG leads (leads I, II, III, V1, and V6) and two to five bipolar intracardiac leads (filtered at 40–400-Hz bandpass) were monitored on an oscilloscope, recorded with an ink jet recorder at a paper speed of 100–200 mm/sec, and stored on magnetic tape. Recordings from the left ventricular site of tachycardia origin were available in five patients.

**Stimulation During Ventricular Tachycardia**

After the induction of well-tolerated, sustained ventricular tachycardia, premature extrastimuli were introduced from the right ventricular apex to study the

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**Figure 1.** Tracings showing corrected coupling intervals. Surface ECG leads I, II, and V1, as well as an intracardiac electrogram from the right ventricular apex (RVA) and time lines (T) are displayed during ventricular tachycardia. Top panel: A single extrastimulus (S) is delivered at a coupling interval of 340 msec. This stimulus arrives 90 msec before the next expected tachycardia beat (timing indicated by the arrows) and results in resetting, since the postspacing beat also arrives earlier than expected (less than compensatory pause). For single extrastimuli, the difference between the coupling interval and the tachycardia cycle length is an accurate representation of the degree of prematurity with which the impulse reaches the circuit. For double extrastimuli, the coupling interval needs to be corrected to account for the influence of the first extrastimulus. Middle and bottom panels: The first extrastimulus is delivered at a coupling interval of 360 msec, a coupling interval that does not result in resetting. The second extrastimulus, although it is only 100 msec and 120 msec shorter than the VT cycle length, arrives 160 msec and 180 msec earlier than the next expected tachycardia beat in the middle and bottom panels, respectively.
response of the tachycardia to resetting and entrainment. The return cycle, defined as the interval from the last pacing stimulus to the onset of the first postpacing beat, was measured from the right ventricular apex recording. Pacing was performed at the right ventricular apex from the distal pair of electrodes of a 6F Josephson quadrupolar catheter (USCI; intraelectrode distance, 0.5 cm), and the proximal poles were used for recording. The return cycle response pattern, i.e., the curve derived from plotting the coupling interval of the premature stimulus during resetting or the last stimulus of a pacing train during entrainment versus the return cycle, was determined in all patients. Previous studies have shown that the return cycle response pattern during resetting can be used to quantify the extent of the excitable gap. A flat response pattern indicates that the paced impulse propagated through the circuit with the same conduction time over a range of coupling intervals. This suggests that the premature stimulus encounters fully recovered tissue in all portions of the circuit over this range of coupling intervals. An increasing response pattern suggests that the premature impulse encounters incompletely recovered tissue in at least part of the circuit. This results in interval-dependent conduction slowing and a progressive increase in the measured return cycle as the coupling interval decreases.

In the event that pacing resulted in termination of ventricular tachycardia before completion of the protocol, programmed stimulation was repeated, and the pacing protocols during ventricular tachycardia were again performed after verification (by 12-lead ECG) that the same tachycardia was initiated and that the cycle length was sufficiently stable for analysis (<20-msec variation in cycle length over 20 consecutive beats).

Resetting represents the interaction of a single extrastimulus with the tachycardia, resulting in a less than compensatory pause. Single ventricular extrastimuli were introduced starting at a coupling interval 20 msec less than the VT cycle length. The coupling interval was decreased in 5–10-msec steps until the stimulus was refractory or resulted in termination of the tachycardia. Double ventricular extrastimuli were then delivered in the following manner: The coupling interval of the first extrastimulus was fixed at 20 msec above the first coupling interval that resulted in resetting or 20 msec above right ventricular refractoriness if resetting was not observed. The second extrastimulus was introduced at a coupling interval equal to the VT cycle length, and the coupling interval was decreased in 5–10-msec steps as described above. Thus, only the second extrastimulus interacted with the tachycardia circuit; the first extrastimulus served only to “peel back” refractoriness so that the second could reach the tachycardia circuit over a broader range of coupling intervals.

Entainment is the delivery of a train of paced stimuli during ventricular tachycardia resulting in continuous resetting of the tachycardia circuit, demonstrated by the presence of a less than compensatory pause after the termination of pacing. Overdrive pacing resulted in entrainment if the following criteria were met: 1) fixed ECG or intracardiac fusion was seen at a constant paced cycle length; 2) there was progressive fusion at faster pacing rates; and 3) all of the tissue necessary for the continuation of the tachycardia was transiently accelerated to the paced rate, with the resumption of the tachycardia at the previous rate once pacing was stopped. The first two criteria are sufficient but not necessary to document continuous resetting of the circuit; the third criterion is both necessary and suffi-
cident. Overdrive pacing was performed at several paced cycle lengths (one to six cycle lengths) during each episode of ventricular tachycardia. The number of different paced cycle lengths used during attempted entrainment depended primarily on the patient's tolerance of the tachycardia. At each paced cycle length, an incremental number of paced beats (one to 15 beats) was added to each successive pacing trial, such that the first beat that influenced the tachycardia, the nth beat, could be identified. In addition, this method ensured that the tachycardia was not inadvertently terminated and reinitiated during long pacing drives.

Corrected Coupling Intervals

The prematurity with which a stimulated impulse interacts with the tachycardia is determined by how early the impulse enters the circuit relative to the ensuing tachycardia wave front. When a single extrastimulus is delivered, the degree of prematurity can be expressed by the coupling interval of the synchronously timed pacing stimulus. During resetting with double ventricular extrastimuli or the nth beat of pacing, the extrastimuli delivered before the stimulus that resets the tachycardia, even though they do not interact with the circuit, affect the relative prematurity of that impulse. Under these circumstances, the actual prematurity is not determined by the coupling interval of the extrastimulus but rather is best expressed by measuring how premature the extrastimulus is relative to the expected tachycardia beat (Figure 1). We refer to this parameter as the corrected coupling interval.

Statistical Analysis

Student's t test was used for the comparison of paired data and for the unpaired comparison of tachycardia episodes that terminated with pacing and those that did not. Least-squares linear regression was used to determine the relation of the return cycles observed during resetting and the nth beat of pacing.

Results

Resetting With Premature Stimuli

Single extrastimuli reset 15 of 18 and double extrastimuli reset 12 of 12 episodes of ventricular tachycardia. The resetting response patterns observed were flat plus increasing in 14 and purely increasing in four tachycardias; no purely flat responses were seen. The mean duration of the flat portion was 41.1±32.2 msec (10.7±8.4% of the VT cycle length), and the mean total
duration of the resetting zone was 125.0±44.8 msec (31.0±9.5% of the VT cycle length). Termination was observed during resetting trials in six tachycardias; in all cases, termination occurred after an increasing return cycle response was demonstrated.

**Comparison of Resetting With the nth Beat of Pacing**

The return cycles determined during resetting and after the nth beat during overdrive pacing were essentially identical when extrastimuli with equivalent corrected coupling intervals were compared. In the 26 trials (16 of 18 tachycardias) in which resetting and the nth beat of overdrive pacing provided equivalent corrected coupling intervals, the mean return cycles were 496.1±80.0 and 499.8±80.9 msec (p=NS), respectively. The correlation between the return cycles determined with the two methods was 0.99 (Figure 2). In 44 trials of pacing, there was no overlap in coupling intervals for the two techniques; in these cases, the corrected coupling intervals for the nth beat were shorter than could be achieved during resetting because of the effects of rapid pacing on local refractoriness.

**Response to Overdrive Pacing After the nth Beat:**

**Tachycardias With Increasing Resetting Curves**

The pattern of the return cycle response to paced beats after the nth beat was variable and depended on the pacing cycle length and the pattern observed during resetting (Figure 3). In four tachycardias with purely increasing resetting responses, the return cycle during overdrive pacing always increased for the nth+1 compared with the nth beat and continued to increase with the delivery of each successive beat at every paced cycle length. Three of the four tachycardias eventually terminated with the delivery of incremental paced beats.

**Tachycardias With FlatResetting Curves**

Some duration of a flat response curve was demonstrated with resetting in the remaining 14 tachycardias. In eight tachycardias with flat resetting curves (group A), the response to overdrive pacing was dependent on the paced cycle length. At relatively long paced cycle lengths (21 trials), the return cycle did not increase significantly (<10 msec) from the nth to the nth+1 beat. In all 21 trials in this group, the corrected coupling interval of the nth beat corresponded to the flat portion of the resetting response curve for that tachycardia. When the return cycle was fixed after the nth and the nth+1 beats, the return cycle remained relatively constant and comparable to the flat portion of the resetting response curve when incremental paced beats were added at the same cycle length. When this occurred, the criteria for entrainment were satisfied. The mean paced
cycle length that resulted in a fixed return cycle response was 85.2±7.6% of the VT cycle length (range, 68.7–98.5%).

During overdrive pacing at faster rates (18 trials; mean paced cycle length, 79.6±8.3% of the VT cycle length), an increase in return cycle was observed with the delivery of longer pacing drives. In all cases, an increase in the return cycle of ≥10 msec from the nth to the nth+1 beat predicted progressive prolongation in the return cycle with the delivery of incremental paced beats until either a longer equilibrium return cycle was reached or the tachycardia terminated in response to pacing. Once a stable return cycle was observed, the tachycardia was entrained and did not terminate as additional paced beats were delivered.

In six tachycardias with some duration of fully excitible gap, overdrive pacing resulted in consistent increases in the measured return cycle with each incremental paced beat at all paced cycle lengths (group B). In group B tachycardias, even at the longest paced cycle lengths that interacted with the tachycardia (mean, 86.9±4.7% of the VT cycle length), the corrected coupling interval of the nth beat corresponded either to the increasing portion of the resetting curve (n=3) or to the flat portion of the curve within 20 msec of the curve's inflection point (i.e., where the curve changed from flat to increasing, n=3). There was no difference in the mean VT cycle length (409.4±49.2 versus 405.0±44.6 msec, p=NS) or the duration of the fully excitible gap (63.8±31.1 versus 43.3±12.1 msec, p=NS) in group A compared with group B tachycardias.

Specific Examples of the Response to Resetting and Overdrive Pacing

An example of the differential response to pacing at different cycle lengths is shown in Figure 4. This ventricular tachycardia with a cycle length of 420 msec had a flat resetting response over a range of coupling intervals from 350 to 240 msec. During pacing at 360 msec (86% of the VT cycle length), the nth beat results in a return cycle equivalent to that observed during the flat zone of resetting. The nth+1 beat does not cause an increment in the return cycle. With the delivery of subsequent paced beats at this cycle length, the return cycle remained constant no matter how many paced beats were delivered, and the tachycardia was entrained. During overdrive pacing at 300 msec (71% of the VT cycle length), a paced cycle length within the range of coupling intervals that resulted in a flat response curve during resetting, the return cycle increased from the nth to the nth+1 beat. The return cycle continued to increase after each incremental beat until the tachycardia terminated after a six-beat drive was delivered.

The return cycle response during overdrive pacing depended not only on the paced cycle length but also on the number of beats delivered in the pacing train. The response of a ventricular tachycardia with a cycle length of 440 msec is shown in Figure 5. At relatively long paced cycle lengths, the return cycles were constant as additional beats were delivered. At a faster paced cycle length, there was an initial increase in the return cycle until a new equilibrium return cycle was reached after seven beats, after which the tachycardia was entrained. During overdrive pacing at shorter cycle lengths, there was even greater prolongation of the return cycle with the delivery of each beat after the nth beat until the tachycardia eventually terminated in response to pacing.

Location of the Conduction Delay Resulting in Increasing Return Cycles

In five patients, endocardial recordings from the left ventricular site of origin demonstrated that the conduction delay responsible for increases in the return cycle measured during overdrive pacing occurred within the tachycardia circuit in the orthodromic direction. This is illustrated in Figure 6. Resetting demonstrated a flat response curve over a range of coupling intervals from 350 to 250 msec. Each beat after the nth beat of pacing at a cycle length of 400 msec resulted in a progressive increase in conduction time through the circuit, as indicated by the increased duration from the stimulus to the onset of the electrogram at the site of origin. After five beats, a new equilibrium return cycle was reached, the conduction time through the circuit remained constant, and the tachycardia was entrained.

Discussion

The major finding of this study is that resetting with premature extrastimuli and continuous resetting using overdrive pacing (with or without entrainment) appear to provide conflicting information about the characteristics of the circuit because they assess entirely different electrophysiological parameters. If only the first beat of a pacing drive that interacts with the tachycardia is considered, the nth beat, the return response is identical to that observed during resetting over a wide range of corrected coupling intervals. After the nth beat, the subsequent return cycle response depends on several variables, many of which are independent of the electrophysiological parameters of the tachycardia. However, if the pacing continues in the presence of the tachycardia, the return cycle response may be determined not only by the coupling interval but also by the number of paced beats delivered. These findings have important implications for the treatment of tachycardias by overdrive pacing.
trophy physiological characteristics of the tachycardia circuit. The paced cycle length, number of beats delivered, presence of a fully excitable gap, the duration of the excitable gap relative to the pacing cycle length, and the coupling interval of the \( n \)th beat all have a dynamic effect on the return cycle. In most ventricular tachycardias with a fully excitable gap, a constant return cycle and entrainment can be demonstrated with the delivery of incremental paced beats at relatively long paced cycle lengths. During pacing at shorter cycle lengths, an increase in the return cycle from the \( n \)th to the \( n+1 \)th beat predicts subsequent increases in the return cycle until a new equilibrium is reached or the tachycardia terminates. Entrainment is present only when a new equilibrium is reached, leading to a fixed, albeit longer, return cycle. Progressive increases in return cycle, without the establishment of a new equilibrium, eventually result in tachycardia termination.

We hypothesize that the difference between the two techniques may be explained as follows: resetting is the interaction of a single extrastimulus with the tachycardia; overdrive pacing with or without entrainment, on the other hand, represents continuous resetting of the previously reset circuit.

This distinction is illustrated by the schematic examples presented in Figure 7. At any instant in time, the tachycardia circuit is composed of fully excitable, partially excitable, and inexcitable tissue. The first beat that interacts with the tachycardia during pacing (called the \( n \)th beat) advances it to the extent that it arrives prematurely. Thus, the \( n+1 \)th beat encounters a circuit that has already been altered by the effects of the \( n \)th beat—namely, the tissue has had less time to recover excitability. During overdrive pacing at long cycle lengths, the \( n+1 \)th beat may still encounter fully excitable tissue. It will propagate through the circuit without a change in conduction time so that subsequent beats will encounter the circuit at the same state of recovery, and the measured return cycle will stay stable. Under these conditions, the criteria for entrainment will be met and the return cycle will be the same as measured during resetting. At shorter paced cycle lengths, the \( n \)th beat advances the circuit and decreases the available excitable gap to a greater extent. The \( n+1 \)th beat may encounter partially refractory tissue, and its conduction time through the circuit will be prolonged. Because of this delay, subsequent beats will encounter tissue that has had even less time to recover, resulting in further conduction slowing and progressive increase in the measured return cycle. This process will eventually result in entrainment with a prolonged return cycle only if a new equilibrium is reached.

The observation that progressive increases in the return cycle can occur with entrainment at paced cycle
lengths within the range of corrected coupling intervals that result in a flat response curve during resetting emphasizes several important points. First, the cycle length of pacing is not the correct way to express the degree of prematurity with which an impulse interacts with the tachycardia circuit. Previously delivered beats, even if they do not interact with the circuit, affect the degree of prematurity with which the impulse is able to reach the circuit. This effect can be accounted for during resetting with double extrastimuli or the nth beat during pacing by use of corrected coupling intervals. Once the first beat of a pacing train has interacted with the circuit, however, the concept of corrected coupling interval does not apply because subsequent beats do not interact with the original tachycardia circuit. The idea that a train of paced beats alters the effects of subsequent extrastimuli was first considered by Gardner et al. in devising a new method for tachycardia termination. As shown in Figure 8, a single extrastimulus at a coupling interval that causes resetting with a flat return cycle response results in tachycardia termination when delivered after an eight-beat drive train at a relatively long paced cycle length. Second, the response to be measured is determined by a number of variables in addition to the paced cycle length. The number of beats in the pacing drive, the duration of the fully excitable gap, and the state of recovery from refractoriness at the time that the nth beat interacts with the circuit all affect the return cycle response.

Investigators have used entrainment in an attempt to measure the excitable gap in order to investigate the physiology of the reentrant circuit,11,13,17,21 explain the events resulting in tachycardia termination,17,22 and quantify the effects of antiarrhythmic medications.23 Many of these studies have provided fundamental insights about the physiology of reentry; however, it is important to realize that they measured the electrophysiological properties of the previously reset tachycardia.
and not the tachycardia circuit. In addition, these studies typically used a fixed pacing duration (usually 10–15 beats or 10 seconds) at various paced cycle lengths without any attention to the number of beats that actually reached the circuit or even whether or not the tachycardia was actually entrained. In studies that required demonstration of fixed fusion,11,13,17,23 entrainment was clearly demonstrated during overdrive pacing. However, fixed fusion can be observed in two different conditions that would result in two different measured return cycles: 1) both the nth and the nth+1 beat have encountered fully excitable tissue (open squares in Figure 5, return cycle 520 msec, and Figure 7A); or 2) the nth+1 and subsequent beats have encountered partially refractory tissue, but a new equilibrium return cycle has been reached (filled circles in Figure 5, return cycle 560 msec, and Figure 7B). In other words, the return cycle measured after paced beats after the nth beat is markedly influenced by the effects of the nth beat on the tachycardia wavelength even if overdrive pacing has resulted in entrainment.

The interaction of a single extrastimulus with the reentrant circuit over a range of coupling intervals—resetting—is the only method to correctly determine the electrophysiological characteristics of the unaltered tachycardia circuit. In addition, because the corrected coupling intervals are changed in fixed 5–10-msec decrements, resetting is much more precise than entrainment. The relative prematurity of the nth beat during overdrive pacing, in contrast, is impossible to control in small, discrete steps. Finally, particularly during long pacing drives at relatively short cycle lengths, rapid pacing during attempted entrainment may actually change the electrophysiological characteristics of the tissue within the circuit. This will be the case if the circuit boundaries are, even in part, functionally derived,24 if conduction delays within the circuit are dependent on refactoriness, or if there are frequency-dependent effects on conduction in nonuniform anisotropic tissue within the circuit.25,26 Obviously, given the well-recognized use-dependent effects of many antiarrhythmic drugs,27,28 these effects may be even more important in patients who are receiving these agents. The present study was not designed to demonstrate these effects.

With the addition of incremental paced beats after the nth beat during attempts at entrainment, 15 of 18 tachycardias terminated at one or more paced cycle lengths. Entrainment, as it was originally defined, implies a stable condition and is distinct from and incompatible with tachycardia termination.6 Our results do not contradict the classic studies of entrainment but demonstrate that, when pacing is performed without regard to the number of beats that actually interact with the tachycardia circuit, the illusion of stability can be maintained.

Limitations

The extended duration of the pacing protocol required that only patients with well-tolerated ventricular tachycardia be included in the study. In many cases, this required studying patients receiving antiarrhythmic medications because the induced tachycardias were not sufficiently well tolerated in the baseline state. The
degree to which the use-dependent properties of many of these medications may have contributed to the observed differences between resetting and overdrive pacing is unknown. It is also unknown whether our observations apply to more rapid ventricular tachycardia. Many studies resulted in tachycardia termination during resetting or attempted entrainment and required reinduction of the same ventricular tachycardia for completion of the protocol. In some patients, minor (≤100-
ms) changes in tachycardia cycle length were observed that could have altered the duration of the excitable gap and affected the direct comparison of the response to resetting and entrainment. We believe that these changes are small relative to differential effects on the circuit of the two pacing techniques that we were trying to quantify.

Conclusions

The observation that the return cycle increases at progressively paced cycle lengths during overdrive pacing with or without entrainment does not contradict the finding that most tachycardias have a fully excitable gap demonstrated with resetting and does not support the presence of a zone of slow conduction with decremental conduction properties. The response to entrainment does not reflect the electrophysiological characteristics of the tachycardia circuit but rather those of the previously reset tachycardia circuit. In addition, the return cycle response to overdrive pacing is a dynamic variable that can change on a beat-to-beat basis; the measurement of a single return cycle after pacing without regard to the effect of each individual stimulus in the pacing train can result in inconsistent results. Because of all of these considerations, resetting with single or double premature stimuli is the best technique to determine the electrophysiological characteristics of the circuit in ventricular tachycardia.

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