A pacing model was designed that simulated "endless loop tachycardia," a complication found in the new generation of DDD (atrioventricular [AV] universal) pacemakers. The functional characteristics of the train of ventricular impulses simulating endless loop tachycardia were studied during both AV sequential pacing and basic ventricular drive. AV sequential pacing, by causing a decrease in ventriculoatrial (VA) conduction time of the first beat of the endless loop tachycardia, was associated with a decrease in the cycle length at which VA block occurred in 9 of 12 patients. The site of block was the His-Purkinje system in 4 of these 12 patients and the AV node in the remaining 8. At a cycle length with 1:1 VA conduction, a steady state VA conduction time was achieved in 2 to 4 beats (VA conduction time accommodation). The pattern of such accommodation depended on the site (His-Purkinje system versus AV node) of the maximal conduction delay. The steady state VA conduction time itself was altered with AV sequential pacing in patients showing His-Purkinje system delay, but not in patients with AV nodal delay.

The results suggest that in most patients, the cycle length of VA block and the longest steady state VA conduction time will depend on the retrograde conduction time of the first beat of the tachycardia. In addition, pharmacologic measures to prevent or terminate endless loop tachycardia will have to take into account the fact that both the His-Purkinje system and the AV node can be the site of initial block.

Although the clinically observed endless loop tachycardia somewhat resembles constant cycle length ventricular pacing, the electrophysiology of endless loop tachycardia remains essentially unknown. In this study, an attempt was made to create a model of endless loop tachycardia and characterize the underlying beat to beat electrophysiologic behavior during both the onset and the continuation of this arrhythmia as it occurs in the setting of AV sequential pacing. Although the latter pacing method has been shown to facilitate the retrograde conduction of V2 by way of both the AV node and the His-Purkinje system as compared with ventricular pacing alone (6), the relation between this phenomenon and occurrence of endless loop tachycardia is unknown. If such a facilitatory effect persists beyond V2, it is conceivable that the electrophysiology of endless loop tachycardia, occurring during AV sequential pacing, may be significantly affected. With this in mind, a unique pacing protocol was designed that permitted simulation of endless loop tachycardia in the settings of AV sequential pacing and ventricular pacing alone in order to elucidate: 1) the retrograde conduction over successive beats of the simulated tachycardia and to see if the facilitatory effect of AV sequential pacing persists beyond the V2 and 2) the functional behavior of the AV node and His-Purkinje system using...
beat by beat analysis as these relate to the initiation, sustenance or termination of the simulated endless loop tachycardia. In this report, we present the findings and the clinical implications of the phenomena observed during this study.

Methods

Study Group

We studied 12 consecutive patients (7 men and 5 women) with intact retrograde conduction, but without dual atrioventricular (AV) nodal pathways or AV bypass tracts of the Kent bundle type. The patients were selected from patients undergoing electrophysiologic studies for a variety of reasons. The nature of the procedure was explained to all and signed consent was obtained. All patients were in sinus rhythm and had narrow QRS complexes (less than 0.12 second). Cardioactive drugs were discontinued 48 hours before the study.

Electrophysiologic Studies

The studies were performed with patients in the nonseated postabsorptive state. After local anesthesia, quadrupolar electrode catheters were percutaneously introduced through the antecubital and femoral veins and positioned under fluoroscopic guidance. The catheter in the region of tricuspid valve permitted recording of His bundle potential and the catheters in the high right atrium and right ventricle recorded local electrograms and were also used for electrical stimulation.

The intracardiac electrograms (filtered at 30 to 500 Hz), surface electrocardiographic leads and time lines were simultaneously displayed on a multichannel oscilloscope (Electronics for Medicine VR-16) and recorded on a magnetic tape (Honeywell model 101). For analytic and illustrative purposes, recordings were subsequently reproduced on photographic paper at 100 or 150 mm/s speed.

Intracardiac electrical stimulation was performed, using a custom-designed DTU 101 digital stimulator (Bloom Associates, Ltd.) capable of delivering rectangular impulses of adjustable voltage and duration. Although complete electrophysiologic studies using the previously described techniques (7–10) were carried out in these patients, the pacing protocol relevant to this study is described in detail here.

Pacing protocol. The simulation of endless loop tachycardia in the settings of ventricular and AV sequential pacing was accomplished as follows.

Standard method (Fig. 1A). The right ventricle was paced at a basic cycle length ($V_1V_2$) of 500 to 800 ms for six to eight beats and then a premature ventricular beat, ($V_2$) was introduced. The $V_2V_1$ interval chosen was the shortest coupling interval at which $V_2$ consistently conducted to the atria. At these $V_2V_1$ intervals, a retrograde His bundle electrogram ($H_2$) could be visualized preceding the atrial deflection ($A_2$) in many cases (7 of 12 patients). After $V_2$, a train of ventricular impulses (eight beats) was programmed at a constant cycle length, such that each ventricular impulse in the train produced a 1:1 VA response. The $V_2V_3$ interval was the same as the cycle length of the train (that is, $V_2V_3 = V_3V_4 = V_4V_5$, and so forth). Thus, $V_2$ became the first beat of the simulated endless loop tachycardia. The stimulation protocol was repeated; each time the cycle length of the simulated endless loop tachycardia was decreased by 20 ms until retrograde block (that is, ventricular but not atrial deflection) occurred with any beat of the train.

Atrioventricular (AV) sequential method (Fig. 1B). The standard pacing protocol just outlined was repeated with the basic ventricular drive ($V_1V_2$), being replaced by an AV

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**Figure 1.** Pacing protocol. The ladder diagrams depict the standard and atrioventricular (AV) sequential methods. A. Standard method. The ventricle is paced at a basic drive ($V_1V_2$). The endless loop tachycardia is simulated by $V_2V_3V_4$, and so forth such that $V_2V_3 = V_3V_4 = V_4V_5$, and so forth. The coupling interval between the simulated tachycardia and the basic drive ($V_2V_3$) is fixed and the same as in the AV sequential method (B). In the latter method, the basic drive $V_1V_2$ is replaced by an AV sequential drive ($A_1V_1A_2V_2$) at the same cycle length. $A =$ atrium; AVN = AV node; HB = His bundle; PS = Purkinje system; V = ventricle.
sequential drive of the same cycle length. The $V_1V_2$ interval and cycle length of the train remained the same as in the standard method. The range of AV intervals used was 100 to 150 ms; these intervals were chosen so that facilitation of conduction might occur through both the His-Purkinje system and the AV node (6).

**Definition of terms.** A complete set of definition of terms used for retrograde conduction times and retrograde refractory periods has been published (7,8).

The **VA interval** was measured from the corresponding stimulus artifact to the beginning of the low atrial electrogram. Whenever the retrograde His bundle deflection was recognized during ventricular pacing, the **VH interval** was measured from the corresponding stimulus artifact to the beginning of the retrograde His bundle deflection, and the **HA interval** was measured from the beginning of the His bundle deflection to the onset of the low atrial electrogram.

**Figure 2.** Case I. The effect of atrioventricular (AV) sequential pacing on the cycle length of block. Each panel shows electrocardiographic lead $V_1$ and high right atrial (HRA) and His bundle (HB) recordings with time-line (T). The appropriate deflections are labeled in this and subsequent figures. **A,** Standard method. The ventricle is paced at a basic cycle length of 600 ms. A premature ventricular beat ($V_2$) "initiates" the train of ventricular beats, each conducting to the atrium, simulating endless loop tachycardia. The train cycle length (TCL) is 380 ms; because $V_2$ is the first beat of the train, the $V_2V_3$ interval is also 380 ms. The basic cycle length and the $V_1V_2$ interval are the same in all three panels. **B,** As the train cycle length is decreased to 360 ms, $V_1$ blocks in the retrograde direction. After AV sequential drive (C), the retrograde conduction time of $V_2$ ($V_2A_2$) is decreased to 120 ms (from 205 ms in A and B) and no retrograde conduction block occurs in the train of ventricular impulses, even though the train cycle length is 260 ms. (Although not labeled, the pattern of initial increase in ventriculoatrial [VA] conduction time followed by a subsequent decrease to steady state values of VA conduction time can be easily appreciated in C.) See text for details.
Steady state level of the VA conduction time was achieved when values for VA conduction time did not change by more than 5 ms during the simulated tachycardia.

The initial cycle length of block is defined as the longest cycle length at which VA block occurs.

The initial site of block is defined as the site of VA block (His-Purkinje system or AV node) at the initial cycle length of block.

Statistical analysis. Data were analyzed using the paired t test. Values are expressed as mean ± 1 standard deviation.

Results

As indicated in the Methods section, the coupling interval of V2 (the first beat of the simulated endless loop tachycardia) with either the basic ventricular or atrioventricular (AV) sequential drive was the same, that is, the V1V2 interval was fixed. The V2A2 interval, however, was shorter with the AV sequential method in all patients (Fig. 2). This facilitation of the retrograde conduction of V2 has been previously described by us (6), and values for the decrease in the V2A2 interval in each patient are given in Table I.

For clarity, the results may be organized in two sections: 1) the effect of a decrease in the V2A2 interval (in the setting of AV sequential pacing) on the functional characteristics of endless loop tachycardia, such as the cycle length of block and the longest steady state VA conduction time; and 2) the retrograde conduction characteristics of the simulated endless loop tachycardia, such as the beat to beat changes in retrograde conduction and the site of block.

Effect of AV sequential method on cycle length of initial block (Table 1). In 9 of the 12 patients, the cycle length at which retrograde VA block occurred decreased significantly with the AV sequential method (range of decrease 20 to 100 ms, mean 47 ± 24). A striking example was Patient 1, in whom no VA block was seen at a cycle length as short as 260 ms (Fig. 2). In two of the remaining three patients (Cases 5 and 8), the cycle length of block was unchanged, and in one patient (Case 7) it was increased with the AV sequential method.

Effect of AV sequential method on the longest steady state VA conduction time (Table 2). The shortest cycle length with 1:1 VA conduction is given in Table 2 for both methods. It can be seen that in 9 of 12 patients, a faster pacing rate with 1:1 VA conduction was achieved with the AV sequential method. The steady state VA conduction time at the shortest cycle length with 1:1 VA conduction represents the longest VA conduction time that the retrograde VA conduction system is capable of sustaining under a given set of circumstances.

The effect of the faster pacing rate attained during AV sequential pacing on the steady state VA conduction time varied. In five of nine patients, the same value of steady state VA conduction time was achieved at the faster pacing rate (Patients 6 and 9 to 12, Table 2). In contrast, the faster pacing rate during AV sequential pacing produced changes in values of steady state VA conduction time in Patients 1 to 4. This difference in the behavior of the VA conduction time in response to the shorter cycle length produced by AV sequential pacing appeared to be related to differences in the site of maximal delay (AV node compared with His-Purkinje system) and is discussed later.

Two of the remaining three patients (Cases 5 and 8) showed no change in shortest cycle length (with 1:1 VA conduction), but one patient (Case 5) showed a slight de-

Table 1. Electrophysiologic Data (in ms)

<table>
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<tr>
<th>Case</th>
<th>BCL</th>
<th>V1V2</th>
<th>V2A2</th>
<th>V2H2</th>
<th>H2A2</th>
<th>Block</th>
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<th>Site</th>
<th>AV Int</th>
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<td>400</td>
<td>AVN (V3)</td>
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*Mean decrease ± standard deviation = 59 ± 36 ms (p < 0.001 compared with standard method); †mean decrease ± standard deviation = 33 ± 37 ms (p < 0.02 compared with standard method). AV Int = atrioventricular interval; AVN = atrioventricular node; BCL = basic cycle length; CL = cycle length; HPS = His-Purkinje system; Site = site of block; — = no ventriculoatrial block until cycle length of 260 ms; + = see text for details.
Table 2. Steady State Ventriculoatrial (VA) Conduction Time at the Shortest Cycle Length With 1:1 VA Conduction (in ms)

<table>
<thead>
<tr>
<th>Case</th>
<th>Shortest CL With 1:1 VAC</th>
<th>Steady State VACT</th>
<th>Shortest CL With 1:1 VAC</th>
<th>Steady State VACT</th>
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*Patients showing decrease in shortest cycle length with 1:1 VA conduction with atrioventricular sequential method. †A shorter pacing cycle length was not attempted. CL = cycle length; VAC = ventriculoatrial conduction; VACT = ventriculoatrial conduction time.

Site of initial block during endless loop tachycardia (Table 1). Block in His-Purkinje system. In Patients 1 to 4, the site of block appeared to be the His-Purkinje system. In Figure 4B (Case 2), a retrograde 3:2 Wenckebach block in the His-Purkinje system is seen with the standard method. There is a 20 ms increase in the V₁H₃ interval compared with the V₂H₂ interval, followed by block in the His-Purkinje system with V₄ (no His bundle deflection is seen). The patient had relatively rapid AV nodal conduction (H₂A₂ and H₃A₃ = 50 ms) and it is unlikely that block was in the AV node. A similar phenomenon is noted in Fig. 4D with the AV sequential method. A 3:2 His-Purkinje system Wenckebach block was noted in Patient 3 with the AV sequential method, although the site of block could not be clearly documented to be the His-Purkinje system with the standard method. However, with substantial delay in the V₃H₃ interval (Table 1) and a rapid HA interval of 40 ms, the His-Purkinje system was the likely site of block.

An interesting example of His-Purkinje system conduction delay was seen in Patient 4 using the standard method. Unlike the other patients, accommodation either required several beats (Fig. 5A) or did not occur at all for the duration of the paced train (Fig. 5B). A closer analysis of the illustration clearly reveals that the inability of VA conduction time to quickly accommodate was primarily localized to the His-Purkinje system, rather than the AV node. The pattern and site of block (that is, the His-Purkinje system seen in Figure 5C is similar to that of Patients 2 and 3. With the AV sequential method, the facilitation of retrograde His-Purkinje system conduction (S₂H₂) (Fig. 5D) results in immediate accommodation in the His-Purkinje system and persistent 1:1 VA response for the duration of the train. In Patient 1, a retrograde His bundle deflection was not seen.
Figure 3. Patients 1 to 12. Patterns of ventriculoatrial (VA) accommodation. The ventriculoatrial conduction times (VACT in ms) of each beat of the “train” at the shortest cycle length with 1:1 VA conduction are plotted to show the patterns of VA accommodation during both the standard method (solid lines) and atrioventricular (AV) sequential method (interrupted lines). For clarity, patients are shown in groups of four. In addition, patients who showed VA block in the His-Purkinje system are grouped together (Patients 1 to 4). Patients 5 to 12 showed block in the AV node. Patients 1, 2, and 4, the pattern of increase in VA conduction time followed by a decrease to steady state value is seen. The “crescendo” and “decrrescendo” patterns of VA conduction time accommodation are seen in Patients 3 and 5 to 12. In Patient 4, because of the unusual pattern of His-Purkinje system delay seen at shorter cycle lengths during the standard method, a longer cycle length (420 ms) was used for the purpose of comparison. See text for details.

Discussion

Advantages and limitations of the pacing model. Many variables may determine the behavior and outcome of an endless loop tachycardia that occurs with a DDD pacemaker in situ; for example, the coupling interval of a premature beat (V2) may vary or changes may occur in autonomic tone affecting the ventriculoatrial (VA) conduction time of the cycle length of block (440 to 600 ms) in Patients 6, 8, 10 and 12 made it unlikely that the His-Purkinje system was the site of block; also V3 clearly fell outside the relative refractory period of the His-Purkinje system established during the routine study (Fig. 7). In Patient 9, the site of block could not be localized to the AV node with certainty.

Figure 4. (See page 1494.) Patient 2. His-Purkinje system Wenckebach block. A. Basic ventricular drive V1V2 at a cycle length of 600 ms. A premature ventricular beat (V2) conducts in retrograde manner to the atrium with S2H2 equal to 180 ms. V2 is followed by a train of eight ventricular paced beats at a train cycle length (TCL) of 420 ms. All the paced ventricular beats conduct to the atrium; however, no His deflection is seen after V1, V4, and so forth. B. The basic cycle length and V1V2 and V2H2 intervals are the same as in A. However, now the train cycle length is decreased to 400 ms. The faster rate of the train results in a progressive increase in SH intervals (S2H2 = 180 ms, S4H4 = 200 ms) and block in the His-Purkinje system, that is, V4 but no H4 is seen. C. After AV sequential drive, no H2 deflection is seen after V2. The train cycle length is 340 ms. At a shorter cycle length (D), retrograde His-Purkinje system delay is seen in V3 with block in V4.
A STANDARD METHOD

V1

HRA

HB

T

A

V2 A2 120

A3 A4 A5 A6 A7 A8 A9 A10

S-H <140

B

TCL=400

A1 A2 A3 A4 A5 A6 A7 A8 A9 A10

S-H <140

C A-V SEQUENTIAL METHOD

TCL=340

A-V 150 A2 A3 A4 A5 A6 A7 A8 A9 A10

S-H <140

D

TCL=320

A1 A2 A3 A5 A7 A9

S-H <140
paced ventricular beats. It is apparent that the model of endless loop tachycardia in this study addresses only the retrograde conduction aspect of this arrhythmia. Furthermore, the atrioventricular (AV) interval is usually fixed and there is a programmable upper rate limit that is not exceeded during the pacemaker arrhythmia. Thus in an individual patient, the decrease in the cycle length of the train of ventricular impulses could be duplicated only by a simultaneous change in the AV interval or perhaps the upper rate limit, or both. Despite these shortcomings, this pacing model permits an insight into the potential clinical problem as it provides an opportunity to study the retrograde conduction characteristics of a train of ventricular impulses preceded by an AV sequential drive and allows a comparison with straight ventricular pacing.

Figure 5. Patient 4. Loss of ability of the His-Purkinje system to "accommodate" with progressive decrease in cycle length. The basic cycle length and V1V2 interval are the same in all panels. A, At a train cycle length (TCL) of 400 ms, a retrograde His bundle (HB) deflection is seen after each of the first five beats (V2 to V6). The SH interval decreases from V7 to V10, and there is no His deflection after the ventricular electrogram. The subsequent decrease in ventriculoatrial (VA) conduction time is clearly related to His-Purkinje system "accommodation." By decreasing the train cycle length to 380 ms, the retrograde His deflection is seen after each beat of the train (H2 to H10), demonstrating inability of the His-Purkinje system to respond to decreasing cycle length by decreasing its refractoriness. With further decrease in the cycle length, a block in the His-Purkinje system is seen (see text for details). D, With facilitation of retrograde conduction through the His-Purkinje system after AV sequential drive (that is, S1H2 < 145 ms), no VA block occurs at a train cycle length of 360 ms.
Mechanism of decrease in $V_2A_2$ interval with AV sequential pacing. Compared with the standard method, it is apparent that the AV sequential method produced a decrease in the $V_2A_2$ interval with subsequent changes in the functional characteristics of the train of ventricular impulses. One electrophysiologic mechanism of decrease in the $V_2A_2$ interval relates primarily to the earlier excitation of the AV node and the His-Purkinje system by the last paced atrial impulse of the AV sequential drive (6). As can be deduced from Figure 1, $V_2$ after an AV sequential drive encounters progressively more recovered tissue above the level of collision of the atrial and ventricular impulses, compared with $V_2$ during the standard method. However, this facilitatory effect of the AV sequential method appears to last beyond the first beat and is exhibited irrespective of the site of block, whether it be the AV node or the His-Purkinje system. Specifically, in 9 of 12 patients, the decrease in cycle length of block tended to approximate the
Figure 7. Patient 10. The decrease in cycle length of block in patients with atrioventricular (AV) nodal block. During the standard method (A and B), there is 1:1 ventriculoatrial conduction at a cycle length of 480 ms with VA block occurring at a relatively long cycle length of 460 ms. With AV sequential drive (C and D), the decrease in the V2A2 interval is accompanied by a decrease in the cycle length of VA block. VA Wenckebach block occurs most likely in the AV node.

decrease in V2A2; that is, the greater the decrease in the V2A2 interval, the shorter the cycle length of tachycardia without block (Table 1).

In Patients 5, 7 and 8, the reason for lack of decrease in the cycle length of block with the decrease in the V2A2 interval is not clear. It can be postulated, however, that in patients with relatively slow VA conduction the facilitatory effect of a decrease in the VA conduction time of the first beat is not enough to permit a change in the cycle length of block. Slow retrograde AV nodal conduction is perhaps more sensitive to small fluctuations in autonomic tone (12), accounting for the increase in cycle length of block seen in Patient 7 with a decrease in V2A2.

Relation between the site of block and functional characteristics of endless loop tachycardia. Patients with relatively rapid conduction through the AV node (Cases 2 to 4 with short H2A2 intervals of 40 to 50 ms) were those in
whom His-Purkinje system was the initial site of block, whereas those with longer H2A2 intervals had block in the AV node (Table 1). The site of block indicates the site of maximal conduction delay and is likely to influence the behavior of the endless loop tachycardia. For example, the initial increase in VA conduction time with subsequent decrease was reflected in an accommodation pattern seen only in patients showing His-Purkinje system block (Fig. 3). This pattern probably reflects the response of the His-Purkinje system to cycle length abbreviation, manifesting initially as His-Purkinje system delay with subsequent accommodation.

In Patients 5 to 12 in whom the AV node was considered the site of maximal delay, smooth crescendo and decrescendo curves of VA conduction time accommodation, much like those seen in the AV node in the anterograde direction (11), were seen.

**Patients with change in steady state VA conduction.** In patients with His-Purkinje system block the steady state VA conduction time altered with the AV sequential method. The increase in steady state value of VA conduction time with this method in Patients 1, 2 and 3 (Table 2) may be explained by the facilitation of conduction with subsequent decrease in cycle length with 1:1 VA conduction time through the His-Purkinje system after AV sequential drive. The AV node is not the site of maximal conduction delay, and has enough “reserve” to respond to the shorter input with an increase in conduction time through the AV node and, thus, a longer steady state VA conduction time occurs. Although the VA conduction times are not labeled, this phenomenon is apparent in Fig. 2A and C. In Patient 4, the unusually prolonged delay in His-Purkinje system conduction using the standard method (Fig. 5A and B) was not seen with the AV sequential method, thereby resulting in a smaller steady state value (Fig. 5D).

**Patients with no change in steady state VA conduction.** More interestingly, five of the eight patients with AV nodal block (Cases 6 and 9 to 12) showed no change in the longest steady state VA conduction time even though shorter cycle lengths were achieved with the AV sequential method. Thus, in these patients, the longest steady state VA conduction time depended on the inherent conduction properties of the AV node and was unaltered by the AV sequential method. The delay, if any, in the His-Purkinje system at the shorter cycle length was probably not significant enough to increase the VA conduction time.

**Functional characteristics of retrograde conduction.** Lastly, certain functional characteristics of retrograde conduction observed during this pacing model, germane to both endless loop tachycardia and constant cycle length ventricular pacing, may be summarized: 1) the site of maximal delay of the retrograde conduction pathway can be either the AV node or the His-Purkinje system and will manifest itself as the initial site of block; 2) the pattern of VA accommodation can suggest the initial site of block; and 3) the coupling interval of the train of impulses to the basic drive is important because the VA conduction time of the first beat can affect the cycle length of the initial block as well as the longest steady state VA conduction time in patients with His-Purkinje system delay.

**Clinical implications.** It is beyond the scope of this study to draw conclusions regarding the programming of pacemakers to prevent tachyarrhythmias. A model of endless loop tachycardia, such as that in this study, however, is helpful in predicting the retrograde conduction behavior of the arrhythmia in situ.

The effect of the decrease in the V2A2 interval on the cycle length of block should be taken into account when assessing patients for DDD pacemakers. This also brings to light a potential pitfall associated with determining the cycle length of VA block by randomly switching on the stimulator, as is usually the case in routine electrophysiologic studies. The train of ventricular impulses that start earlier in diastole, would have a longer V2A2 interval and would tend to block at longer cycle lengths. However, if the stimulator was switched on later in diastole, the resulting abbreviated V2A2 interval might be associated with VA block occurring at shorter cycle lengths. Hence, studies comparing retrograde conduction in different patients, or after an intervention such as drug administration in the same patient, will need to address the effect of this variable.

Lastly, measures to terminate endless loop tachycardia have to take into consideration that 1) both His-Purkinje system and AV node can be the site of initial block, and 2) a drug such as procainamide, which acts on both sites (13), may be effective in preventing or terminating the tachycardia.

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**References**


