Linking: a dynamic electrophysiologic phenomenon in macroreentry circuits

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ABSTRACT The term “linking” has been used specifically to describe the mechanism for perpetuation of functional anterograde bundle branch block: namely, repetitive transseptal retrograde concealed penetration by impulses propagating along the contralateral bundle. We present selected examples that demonstrate linking-type phenomena actually have a wide spectrum of expression in human macroreentry circuits, particularly those incorporating either the bundle branches and His bundle or the normal pathway and Kent bundle. The examples presented are as follows: (1) persistent retrograde functional conduction delays in the His-Purkinje system during right ventricular pacing, (2) anterograde Kent bundle conduction at rapid rates, dependent on prior block in the normal pathway, (3) persistent anterograde functional infra-His block of atrial impulses during rapid ventricular pacing in the presence of a retrogradely conducting accessory pathway, and (4) transient advancement of His activation with ventricular fusion complexes during overdrive ventricular pacing of bundle branch reentrant tachycardia. Based on these examples, we characterize linking as a generalized electrophysiologic phenomenon in which each successive impulse entering a macroreentry circuit propagates preferentially along one limb because of functional block in the contralateral limb resulting from the effects of the prior impulse. It is proposed that such functional block may be dynamically maintained either by repetitive impulse interference, which perpetuates local refractoriness (examples No. 1 to 3), or by repetitive impulse collision (example No. 4). The general conceptual scheme outlined can be applied to specific electrophysiologic phenomena associated with a wide variety of reentry circuits in man.


THE TENDENCY of functional bundle branch block to perpetuate itself, once established during a sudden rate increase, is a familiar electrocardiographic phenomenon.1 Experimental findings reported by Moe et al.2 and by Glassman and Zipes3 support the previously postulated mechanism1 that such functional bundle branch block is sustained during successive beats because of repeated transseptal retrograde concealed penetration by impulses traversing the contralateral bundle branch. Rosenbaum et al.4 have termed this phenomenon “linking.”

In this article we report four interesting examples that, taken together, demonstrate that the concept of linking may be generalized so as to describe a broad class of electrophysiologic phenomena in which there is preferential conduction of successive impulses along one of two limbs of a macroreentry circuit due to persistence of functional block in the contralateral limb. Extrapolating from the examples presented, we offer two distinct dynamic mechanisms responsible for maintenance of the functional block. Under the proposed conceptual formulation for linking, it becomes readily apparent that many seemingly diverse phenomena associated with macroreentry circuits (e.g., accessory pathway conduction during atrial fibrillation and tachycardia “entainment”8) are, in fact, variations on a common electrophysiologic theme.

Methods Electrophysiologic studies were performed in patients in the unsedated state after informed consent was obtained. Cardioactive medications were discontinued five half-lives before the study. As described previously,9 quadripolar or hexapolar electrode catheters were inserted percutaneously with the use of local anesthesia and, with fluoroscopic guidance, the catheters were advanced to the right atrium, right ventricular apex, the region of His bundle, and, in patients with accessory pathways, the coronary sinus. All analog signals were displayed on an Electronics for Medicine VR-16 oscilloscope, recorded on a Honeywell multichannel tape recorder for subsequent retrieval, and printed on-line with the use of a Siemens-Elema Mingograf recorder at a paper speed of 100 mm/sec.

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Programmed electrical stimulation was accomplished with a Bloom digital stimulator that delivered fixed-current pulses via an isolation unit. Anterograde and retrograde studies were performed as previously described. In patients with an accessory pathway, its location and possible participation in supraventricular tachycardia were documented according to published criteria. Additional ventricular stimulation techniques, which have been used in prior investigations in our laboratory, will be described in the presentation of the examples.

Results

The human macroreentry circuits in which linking phenomena were demonstrated incorporate either the bundle branches and His bundle, or the normal pathway and Kent bundle. Whereas the first example to be presented represents the retrograde counterpart of anterograde functional bundle branch block, the second involves a common manifestation of linking in patients with Wolff-Parkinson-White syndrome. Two examples that are more complex follow. Complete electrophysiologic evaluation was performed in each patient; however, only findings pertinent to the theme of this communication will be outlined.

Persistent retrograde functional conduction delays in the His-Purkinje system during rapid ventricular pacing, with subsequent resolution (example No. 1). A 63-year-old man with atherosclerotic heart disease underwent electrophysiologic testing because of unexplained syncope. The surface electrocardiogram and His-Purkinje system responses during anterograde and retrograde assessment were normal. During incremental right ventricular pacing, behavior of the His-Purkinje system at rapid rates was studied in a systematic fashion, as shown in figure 1. In both panels, a basic atrial drive (S,) at 500 msec cycle length (CL) is followed by an 8 beat ventricular train (S,) having a CL of 300 msec. The first ventricular paced S, begins at a programmable pause after the last atrial paced S,. In figure 1, A, this pause is 700 msec in duration. Note that from the second through sixth paced ventricular beats a retrograde His deflection (H,) is observed after each ventricular electrogram. The S,H, interval, initially 170 msec, decreases to a steady-state value of 150 msec. Concurrently there is a retrograde Wenckebach phenomenon occurring in the atioventricular node. During the last two ventricular paced beats, however, the His bundle deflection has suddenly merged into the ventricular electrogram, indicating resolution of the marked His-Purkinje system delays seen in the previous beats.

When the pause preceding the S, train is shortened to 400 msec (figure 1, B), His-Purkinje system refractoriness is abbreviated and the emergent retrograde His deflections present in figure 1, A, are no longer seen. Concomitantly, the corresponding ventriculoatrial conduction times or S,A, intervals during the second and third ventricular paced beats in B are also shortened. Thus, the persistently emergent His deflections in A must represent functional conduction delays. The most likely explanation for this phenomenon is sustained retrograde functional right bundle branch block, the maintenance of which is dynamically "linked" in a repetitive fashion to anterograde concealment from each successive impulse after it activates the His bundle via the left bundle branch. This process is depicted schematically in figure 2. As we have noted previously, and demonstrated in the present case, the S,H, interval during such sustained retrograde functional right bundle branch block either attains its steady-state value immediately or decreases over 1 or 2 beats before equilibrating, owing presumably to accommodation of retrograde conduction along the left bundle–His axis.

It is of particular interest that the linking in panel A "breaks" during the seventh paced ventricular beat, despite a constant frequency of input to the His-Purkinje system. This phenomenon can best be explained by postulating migration of sight of block, as has been demonstrated during resolution of functional anterograde conduction delay and block in the His-Purkinje system.

Anterograde Kent bundle conduction at rapid rates, dependent on prior block in the normal pathway (example No. 2). An 8-year-old girl with Wolff-Parkinson-White syndrome underwent electrophysiologic evaluation because of repeated attacks of narrow QRS tachycardia. She was found to have a right-sided Kent bundle and easily inducible orthodromic reciprocating tachycardia. Figure 3 was obtained during rapid atrial pacing. As illustrated in figure 3, A, sudden initiation of atrial pacing at a constant cycle length of 270 msec results in anterograde propagation exclusively via the normal pathway with repetitive block in the bypass tract evident from the second paced beat on. The absence of an atrial echo after the last paced beat is due to retrograde block in the accessory pathway resulting from concealed anterograde penetration by the last atrial impulse. When atrial pacing is repeated at the same cycle length (figure 3, B) 3:2 followed by 2:1 block in the atrioventricular node is apparent during the first 5 atrial paced beats. Ventricular preexcitation is not observed to accompany the second through fifth atrial impulses. Anterograde conduction in 1:1 fashion over the accessory pathway is restored, however, in the wake of the fifth atrial impulse (indicated by an
asterisk) which blocks in both the atroventricular node and the Kent bundle.

This suggests that persistence of functional anterograde block in the bypass tract in figure 3, A, requires repetitive retrograde penetration of the accessory pathway by orthodromic impulses activating the ventricle. Concomitantly, sustained functional retrograde block in the bypass tract is maintained by repetitive antero-

![Figure 1](http://circ.ahajournals.org/)

**FIGURE 1.** Example No. 1. Persistent retrograde functional conduction delays in the His-Purkinje system during rapid ventricular pacing, with subsequent resolution. Tracings from top to bottom are electrocardiographic lead V1 and right atrial (RA) and His bundle (HB) electrograms, followed by the time line (T). In both A and B an 8 beat paced ventricular train (S2, heavy black arrows), at a constant cycle length (CL) of 300 msec, begins at a programmed S1S2 pause after the last beat of an atrial drive (S1, white arrows) having a basic CL of 500 msec. See text and figure 2 for additional explanation. A = atrial deflection; H = His deflection; S = stimulus artifact; V = ventricular electrogram.

**FIGURE 2.** Schematic representation of linking phenomenon in example No. 1 (and figure 1). In this and in subsequent figures, any depicted site of functional block (shaded region) is merely diagrammatic and not intended to imply precise location of block. A. During the first beat of the S2 train in figure 1, A, the paced impulse (asterisk) invades an unstressed His-Purkinje system and retrogradely activates the His bundle via the right bundle route. With the sudden shortening of input interval that attends the second beat of the S2 train in figure 1, A, retrograde block occurs in the right bundle and, consequently, the impulse can gain access to the His bundle only by transeptal propagation to the left bundle (B). His activation (which has been delayed because of the aforementioned events) then occurs retrogradely via the left bundle (C). At the same time, the impulse (dashed line) attempts antegrade to traverse the right bundle, but is prevented from doing so by distal tissue (shaded region) that is still refractory from retrograde penetration by the paced impulse (C). The stage is thus set for recurrence of retrograde block (in the right bundle) during the next paced beat in panel B (owing to prior antegrade concealment at the same site). In this way the events depicted in B and C temporally alternate with — and are thus linked to — one another, resulting in sustainment of retrograde functional right bundle branch block during subsequent paced beats. HB = His bundle; RB = right bundle; LB = left bundle; VM = ventricular myocardium.
grade concealment, as depicted schematically in figure 4, A to C. With the occurrence of block in the normal route (figure 3, B), such retrograde concealment is prevented, thereby permitting restoration of anterograde conduction via the Kent bundle. In fact, with the return of accessory pathway conduction during the sixth beat, as illustrated in figure 3, B, there is nearly maximal preexcitation due to more atrioventricular nodal delay compared with that seen during sinus rhythm (figure 4, D). During the next atrial paced beat, infra-His block develops in the normal pathway due to sudden shortening of HH cycle (note arrows indicating His deflections in figure 3, B), so that a completely preexcited complex results (figure 4, E). Functional anterograde block in the normal pathway (below the His) is then maintained by repetitive retrograde penetration from successive antidromic impulses propagating over the bypass tract (figure 4, F), thereby comprising a “reversal” of the linking pattern in figure 3, A (compare figure 4, B and C with E and F). It should be mentioned, however, that during 1:1 Kent bundle conduction in figure 3, B, we cannot definitively exclude two alternative scenarios to the one postulated above: (1) Linking is maintained by repetitive concealment at an atrioventricular nodal (rather than infra-Hisian) level, or (2) which is less likely, there is some degree of successful normal pathway conduction (rather than complete block with linking), so that ventricular fusion (as opposed to complete preexcitation) occurs.

One may question why, in figure 3, B, accessory pathway conduction is not restored by the fourth atrial paced beat following 3:2 block in the atrioventricular node. Inspection of figure 3, B, however, reveals a greater temporal proximity to preceding orthodromic ventricular activation during this beat compared with that which exists before restoration of 1:1 conduction over the Kent bundle during the sixth beat (unlabeled ventricle-to-next-atrial-stimulus intervals of 285 msec and 380 msec, respectively). Consequently, the accessory pathway is less recovered from retrograde penetration during the fourth atrial paced beat.

Persistent anterograde functional infra-His block of atrial impulses during rapid ventricular pacing in the presence of a retrogradely conducting accessory pathway (example No. 3). A 30-year-old woman with Wolff-Parkinson-White syndrome had recurrent episodes of symptomatic supraventricular tachycardia. Electrophysiologic investigation revealed that she had a right-sided bypass tract. Figure 5 is taken from the portion of the study in which systematic incremental ventricular pacing was performed. In all three panels, an atrial train (S,) at a basic cycle length of 650 msec is followed by an 8 beat train of ventricular stimuli (S,) that begins after a 600 msec intervening pause between the two trains. In figure 5, A, at a ventricular cycle length of 310 msec, there is 1:1 retrograde conduction over the accessory pathway producing the A, deflections. Following the second ventricular (V,) electrogram, an emergent retrograde His deflection (H,) coincides with the initial portion of the A, electrogram. With the third beat, the SH interval has decreased to 70 msec from its previous value of 100 msec. By the next beat and thereafter, retrograde His deflections are no longer emergent from the V, electrograms. This sequence of events reflects abortment of sustained retrograde His-Purkinje system linking (of the type seen in example No. 1) due to bilateral His-Purkinje system accommodation.\textsuperscript{10}

When the S, train CL is shortened to 300 msec (figure 5, B) there is a 20 msec increase in retrograde S,H interval during the second paced ventricular beat vs the corresponding value in figure 5, A. Such a magnitude of increase in retrograde His-Purkinje system conduction delay is within the expected range for a 10 msec decrease in ventricular coupling interval (from 310 msec in A to 300 msec in B).\textsuperscript{13}

Following the third V, electrogram in figure 5, B, however, the SH interval suddenly increases markedly (by 50 msec) and, by the next beat, reaches a steady-state value of 180 msec, which is maintained for the duration of the S, train. If one assumes that the third through eighth emergent His deflections result from retrograde activation, then the crescendo-like sequence of SH values resembles neither the picture of retrograde His-Purkinje system accommodation,\textsuperscript{10} as in A, nor that of sustained retrograde right bundle branch block\textsuperscript{10} described above (example No. 1). Furthermore, in the normal His-Purkinje system a progressive increase in retrograde SH intervals during rapid ventricular pacing is observed only during functional 3:2 His-Purkinje system Wenckebach block,\textsuperscript{10} a process that is clearly not illustrated in figure 5, B.

A much more likely explanation for the findings in figure 5, B, is that the H deflections seen during the third through eighth beats of the S, train are anterograde in origin, with persistent block below the His maintained by a linking phenomenon in which the Kent bundle participates. This dynamic process is initiated by retrograde block in the His-Purkinje system during the third paced ventricular beat,\textsuperscript{14, 15} with subsequent anterograde activation of the atrioventricular node and His bundle from the impulse propagating retrogradely to the atria via the accessory pathway (as
depicted schematically in figure 6, A and B). The anterogradely traveling wavefront fails to produce a ventricular echo, however, due to block below the His caused by residual His-Purkinje system refractoriness engendered by the third paced ventricular beat (figure 6, C). This anterograde concealment in turn sets the stage for recurrence of retrograde His-Purkinje system block during the next paced impulse (figure 6, B), and so the process of alternating retrograde and anterograde block in the His-Purkinje system perpetuates itself for the duration of the S₂ train. Thus, the initial 50 msec jump in SH interval is due to a switch from retrograde to anterograde His activation, a finding analogous to that observed during initiation of orthodromic reciprocating tachycardia by single ventricular extrastimuli in patients with accessory pathways.¹⁴ The
subsequent increase in SH to a steady-state value is then readily explained by "crescendo" atrioventricular nodal accommodation to the sudden rapid input of successive anterograde atrial impulses.

When the S2 train CL is further shortened to 290 msec (figure 5, C) there is greater retrograde SH delay (140 msec) in the second beat compared with its counterpart in figure 5, B. This is followed by retrograde His-Purkinje system block in the next beat with anterograde (reentrant) activation of the His bundle and associated infra-His block, as in B. In contrast to the situation prevailing in the latter panel, however, bilateral infra-His linking is not subsequently maintained, despite an identical corresponding HH interval of 300 msec. Instead, an anterograde ventricular echo (Ve) with a left bundle branch block pattern (labeled by an asterisk) follows the fourth V2, preemptioning the fifth ventricular stimulus. The basis for resolution in figure 5, C, of a linking process that, by contrast, persists in B is not readily apparent. The difference in behavior may be related to a more distal level of His-Purkinje system block in C (due to a shorter retrograde input interval) that, in turn, might have facilitated migration of site of block.

Orthodromic reciprocating tachycardia at a CL of 280 msec becomes established after the first ventricular echo in figure 5, C. This occurrence attests to the functional nature of the His-Purkinje system linking phenomenon illustrated in B since, during tachycardia, the His-Purkinje system is able to transmit successive anterograde impulses at a CL 20 msec shorter than that generated by the S2 train in B. Inasmuch as the patient had other episodes (not shown) of orthodromic tachycardia with narrow QRS complexes (at a CL of 280 msec and HV interval of 55 msec), the sustained left bundle branch block pattern during tachycardia in C is also functional in nature. In fact, such sustained aberrant conduction represents a linking phenomenon that occurs in the wake of concealed retrograde penetration of the left bundle by the fourth V2 impulse.16

Transient advancement of His activation with ventricular fusion complexes during overdrive ventricular pacing of bundle branch reentrant tachycardia (example No. 4). A 30-year-old man with a history of aortic valve replacement had recurrent sustained wide QRS tachycardia. In the electrophysiology laboratory he was found to have incomplete right bundle branch block with a prolonged HV interval. Ventricular tachycardia due to a bundle branch reentry mechanism (with typical left and right bundle branch block morphologies) was reproducibly initiated by right ventricular extrastimulation. Examples of the laboratory-induced tachycardia have been published previously (see figures 7 and 8 in Akhtar19).

Figure 7 demonstrates the patient's spontaneous wide QRS tachycardia. It too represents bundle branch reentry, but with an activation sequence opposite to that of the classic laboratory-induced type.17-19 In other

FIGURE 3. Example No. 2. Anterograde conduction, via a right-sided Kent bundle at rapid rates dependent on prior block in the normal pathway. Tracings from top to bottom are electrocardiogram leads I, II, and V1 and right atrial (HRA) and His bundle (HB) electrograms. In both A and B atrial pacing (S, white arrows) is at a basic CL of 270 msec. Black arrows in B indicate location of His bundle deflections during 1:1 Kent bundle conduction. See text and figure 4 for additional explanation. BCL = basic CL; other abbreviations as in figure 1.

FIGURE 4. Schematic representation of linking phenomena in example No. 2 (and figure 3). Both the accessory pathway (AP) and normal pathway are depicted. A, During sinus rhythm a wavefront emanating from the right atrium (RA) activates the ventricles over both pathways. B, With the first atrial paced beat in figure 3, A, functional block of the impulse (asterisk) occurs in the bypass tract (shaded region), but the wavefront continues to propagate over the normal pathway. C, When the impulse (dashed line) turns around, however, and invades the AP, block occurs again — this time due to incompletely recovered tissue resulting from the preceding anterograde penetration. Concealment by the retrogradely blocked impulse in turn engenders additional local refractoriness that causes block in the bypass tract of the next atrial (anterograde) input (B). Thus, during the paced atrial train in figure 3, A, dynamic maintenance of bidirectional functional block in the AP permits anterograde impulse propagation to proceed exclusively over the normal pathway. D, Owing to atrioventricular nodal (AVN) block of the fifth atrial beat as in figure 3, B, retrograde concealment into the Kent bundle is prevented, thereby permitting anterograde conduction of the next beat over the AP (as shown here). In contrast to the case of normal sinus rhythm (A), there is more AVN delay in this instance (due to faster atrial rate) and, consequently, nearly maximal preexcitation. E, During the next (i.e., seventh) atrial beat in figure 3, B, ventricular activation proceeds solely via the Kent bundle because of functional block in the normal pathway below the His. F, When the ventricular wavefront (dashed line) retrogradely invades the His-Purkinje system, it blocks bilaterally at sites of prior concealed anterograde penetration. Such retrograde concealment then engenders anterograde block below the His in response to the next atrial input (E). Thus, during subsequent paced atrial beats, there is exclusive 1:1 conduction over the Kent bundle associated with sustained functional infra-His block in the normal pathway. (An atrioventricular nodal site of dynamically maintained block is an alternative linking scenario during 1:1 pure AP conduction.) E and F may be viewed, therefore, as representing a linking process that is the "reverse" of that depicted in panels B and C. Abbreviations as in figure 2.
FIGURE 5. Example No. 3. Persistent anterograde functional infra-His block of atrial impulses during rapid ventricular pacing in the presence of a retrogradely conducting right-sided accessory pathway. Tracings from top to bottom are electrocardiogram leads I and V1 and high right atrial (HRA) and His bundle (HB) electrograms. Time line (T) is at the bottom. After a pause ($S_1S_2$) of 600 msec, which follows the last beat of an atrial drive ($S_1$, white arrows) of basic cycle length 650 msec, an 8 beat paced ventricular train ($S_2$, heavy black arrows) is introduced with three different constant CL (310, 300, and 290 msec in panels A, B, and C, respectively). See text and figure 6 for additional explanation. Ae = atrial echo; Ve = ventricular echo; other abbreviations as in figure 1.

FIGURE 6. Schematic representation of linking phenomenon in example No. 3 (and figure 5). A. During the first beat of the $S_2$ train in figure 5, B and C, the ventricular paced impulse (asterisk) retrogradely invades both the accessory and the normal pathway; these two wavefronts eventually collide in the atrioventricular node. B. By the third $S_2$ beat in figure 5, B and C, however, functional complete retrograde block in the His-Purkinje system has occurred, so that the impulse emerging from the bypass tract encounters no opposing wavefront as it anterogradely invades the AVN. C. Forward propagation of the returning anterograde impulse (dashed line) is eventually arrested when the wavefront invades the regions of the His-Purkinje system that have not recovered from retrograde penetration. Such anterograde block and concealment in turn causes retrograde block of the next paced $S_2$ (in figure 5, B), thereby recapitulating the events depicted in B. Thus, during subsequent paced beats (of the $S_2$ train in figure 5, B), there is alternation of functional anterograde and retrograde block in the His-Purkinje system, while retrograde accessory pathway transmission remains unhindered. Abbreviations as in figure 4.
words, His activation occurs retrogradely via the right bundle and ventricular depolarization proceeds exclusively from reentrant anterograde impulse propagation along the left bundle (as depicted schematically in figure 8, A). Note in figure 7 the longer conduction time along the retrograde vs the anterograde limb of the circuit (VH interval of 240 msec and HV of 70 msec, respectively, both unlabeled).

Attempted overdrive termination of the tachycardia by right ventricular pacing (at a CL of 270 msec) begins 290 msec after the fourth tachycardia beat in figure 7. This results not only in fusion of spontaneous and paced complexes, but also in advancement of retrograde His activation so that the encompassing HH interval is shortened from 310 to 290 msec. During the next 3 ventricular paced beats, QRS fusion is more pronounced and advancement of His activation persists, with slight increases in retrograde conduction time along the right bundle–His axis that result in a small degree of HH interval prolongation. On cessation of pacing the tachycardia resumes, beginning one full CL (310 msec) after the last paced beat.

The electrophysiologic events occurring during ventricular pacing in this example are a manifestation of a

![Diagrams](http://circ.ahajournals.org/)

**FIGURE 7.** Example No. 4. Transient advancement of His activation with ventricular fusion complexes during overdrive pacing of bundle branch reentrant tachycardia. Tracings from top to bottom as in figure 3, except for addition of right ventricular (RV) electrogram. Atrioventricular dissociation is noted during wide QRS tachycardia of right bundle branch block morphology. Four right ventricular paced beats (S, heavy black arrows) at a cycle length of 270 msec are introduced beginning 290 msec after the fourth spontaneous tachycardia complex. See text and figure 8 for additional explanation. Abbreviations as in figure 1.

**FIGURE 8.** Schematic representation of linking phenomenon in example No. 4 (and figure 7). A, Diagram depiction of spontaneous tachycardia in figure 7. As shown here, each circulating impulse traverses the right bundle retrogradely, gains access to the left bundle via the distal His bundle, then proceeds anterogradely to activate the left ventricle, and finally returns transseptally to the right ventricle (and bundle branch). B, During right ventricular pacing (as in figure 7) at a cycle length shorter than that of the tachycardia, each paced impulse (asterisk) retrogradely activates the His bundle via the same pathway utilized during tachycardia (i.e., right bundle route); when the paced impulse propagates transseptally, however, it collides in the left ventricle with the wavefront that was derived from the previous impulse but that has now emerged from the left bundle (dashed line). Since the ventricles are activated from two opposite directions, a fusion beat results (intermediate in morphology between a “pure” tachycardia and a “pure” right ventricular paced complex). The linking process depicted in B occurs during each of the 4 paced beats in figure 7. When pacing is terminated (C) the impulse emerging from the left bundle (and derived from the last paced beat) no longer encounters an opposing wavefront and is free, therefore, to retrogradely reenter the right bundle and reinitiate the tachycardia.
linking process that is different from that described thus far. In the first three cases, linking developed during the introduction of paced impulses into a “dormant” macroreentry circuit. By contrast, in the present example reentrant excitation is ongoing before pacing. Consequently, at the onset of ventricular pacing, retrograde His activation can occur only via the “committed” right bundle route, since the wavefront traveling transseptally to the left ventricle encounters and collides with the reentrant anterograde wavefront (figure 8, B). As long as block does not develop in the retrograde right bundle route, preferential retrograde conduction along that limb will continue during subsequent paced ventricular beats owing to repeated collisions in the left ventricle (and perhaps in some peripheral ramifications of the left bundle as well). The anterograde wavefront involved in each of these collisions is derived from and therefore linked to the wavefront from the previous beat that successfully propagates retrogradely via the right bundle. During pacing, the QRS complex narrows because of nearly simultaneous onset of right ventricular (paced) and left ventricular (anterograde) activation (figure 8, B). When ventricular pacing ceases, the last returning anterograde impulse emerging from the left bundle limb has no new leftward-bound transseptal counterpart with which to collide and thus is free to reestablish reentrant excitation of the circuit (figure 8, C).

Discussion

The common unifying electrophysiologic theme in the examples that have been presented consists of a dynamic scenario in which the fate of each impulse entering a macroreentry circuit (potential or actual) is functionally linked to electrophysiologic sequelae of the previous beat, so that successive impulses are permitted to traverse completely only one of two alternate routes of impulse propagation. It is apparent, therefore, that the linking concept has a general application extending far beyond the familiar setting of functional anterograde bundle branch block to which the term was originally applied.4

Linking: two potential mechanisms. The examples presented suggest that there are two possible mechanisms that may account for linking behavior. This can be appreciated by considering a generalized linking phenomenon, as shown schematically in figure 9. Figure 9, A, depicts a situation in which successive impulses, symbolized by an asterisk, enter a macroreentry circuit and travel preferentially down one of its limbs due to sustained functional block in the other limb, as indicated by the shaded region. Panels B and C show the two alternative mechanisms whereby this functional block can be dynamically maintained. Both schemata depict the fate of successive anterograde and retrograde impulses, labeled “A” and “R,” respectively, as they invade the site of functional block over time. In the first mechanism (figure 9, B) each new anterograde impulse (A) blocks due to refractoriness engendered by retrograde concealed penetration from the previous beat ($R_{n-1}$). When the impulse that traverses the unblocked limb turns around to become $R_{n}$, it too blocks as a result of residual refractoriness left by the concealed anterograde impulse. This successive “interference” effect will persist indefinitely until external input into the circuit ceases, input frequency changes by a critical degree, site of block migrates distally,10,11 or conduction in the opposite limb is altered.

In the second mechanism shown in figure 9, C, block is dynamically maintained instead by successive actual collisions between each new anterograde impulse (A) and each returning retrograde impulse ($R_{n-1}$), derived from the previous input. On cessation of external input to the circuit, however, the last returning wavefront ($R_{n-1}$) encounters no new anterograde impulse, which would otherwise impede its progress. Since local tissue refractoriness is not a barrier to impulse propagation in this mechanism, $R_{n-1}$ is then free to reenter the opposite limb anterogradely. This form of linking can begin when external impulses enter the circuit either in the setting of preexisting sustained reentry or, less often, after initial development of anterograde unilateral functional block (with collision occurring before completion of the first reentrant cycle). The linking effect might be abolished, however, were the input frequency suddenly to increase so as to cause bilateral block of impulse propagation; in that case, if no additional input were presented to the circuit, there would be no subsequent returning retrograde impulse available to (re)initiate reentry.

In the examples that we have presented, the macroreentry circuits consisted either of the His bundle/bundle branch system with intervening myocardium (examples No. 1 and 4) or the normal pathway and Kent bundle with intervening atrial and ventricular tissue (examples No. 2 and 3). Presumably the first of the two proposed mechanisms (figure 9, B) was operative in examples No. 1 to 3, whereas the second (figure 9, C) accounted for the findings in example No. 4.

*The particular directional orientation of each impulse is purely an arbitrary designation in this generalized characterization of the linking process.
**Relation to concealed conduction.** The mechanisms for linking described above actually may be considered to represent two types of concealed conduction in the sense that in both cases a returning retrograde impulse (e.g., R in figure 9, B and C) is unable to propagate beyond the site of block; yet, by its mere invasion of that site at an appropriate time, forward progress of the next anterograde impulse (A in figure 9, B and C) along that limb of the circuit is prevented. It is only the temporal relationship between successive anterograde and retrograde impulses at the site of block that differs, i.e., being out of phase and perpetuating local refractoriness in the first mechanism (interference) vs being precisely in phase in the second, with resultant mutual extinction of impulses (collision). Grossly, however, the effect is the same: unilateral functional block is dynamically maintained during the linking process.

Despite such conceptual similarities between the two mechanisms, linking by interference, specifically, has been attributed to “repetitive concealed conduction.” An interesting property of this type of linking is that, once established, it can persist even as the input frequency is gradually decreased (over a certain range, of course) to a level below that prevailing before the onset of linking. Such behavior has been well documented during sustained functional or “rate-dependent” bundle branch block and during 1:1 conduction over an accessory pathway.

The particular process of linking by interference (and its resolution) observed in example No. 1 could explain changes in retrograde His-Purkinje conduction observed during some instances of antidromic reciprocating tachycardia in patients with Wolff-Parkinson-White syndrome. Two groups of investigators have reported the sudden disappearance of persistently emergent retrograde His potentials during antidromic tachycardia, with concomitant shortening of ventricular conduction time and tachycardia cycle length. Such a phenomenon probably reflects spontaneous resolution of sustained (right or left) retrograde functional bundle branch block similar to that seen in example No. 1 during ventricular pacing.

In patients with Wolff-Parkinson-White syndrome, the possible occurrence of intermittent propagation of consecutive anterograde impulses over the accessory vs the normal pathway during atrial fibrillation is well known. Example No. 2 provides a pacing model of this phenomenon and strongly suggests that its clinical occurrence may well be dependent on the establishment of an appropriate interference pattern of linking.

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**FIGURE 9.** Schematic representation of a generalized linking phenomenon. A, Depiction of a hypothetical macroreentry circuit into which successive impulses (asterisk) enter and preferentially traverse one limb as a result of persistent functional block (shaded region) in the contralateral limb. B and C, Two distinct mechanisms whereby the functional block can be dynamically maintained. Each of the two panels is a “blow-up” of the region of block as it is invaded by successive (i.e., n - 2, n - 1, n, ...) anterograde (A) and retrograde (R) impulses over time. Panel B shows impulse interference, whereas C depicts impulse collision. See text for additional explanation.
in the normal/accessory pathway macroreentry circuit. This thesis receives further support from Prystowsky et al., who recently reported a similar example, and from Klein et al., who systematically documented the occurrence of concealed conduction in accessory pathways.

Example No. 3 is particularly remarkable in that it demonstrates that linking by interference can result in sustained functional bilateral block in the His-Purkinje system. A similar phenomenon probably occurred in example No. 2 as well during 1:1 Kent bundle conduction. Conceivably, a single persistent intra-His site of functional block distal to the His recording site might also account for such sustained anterograde (and retrograde) block below the His. These two alternative sites of block cannot be distinguished from one another, however, without additional recordings from the bundle branches.

Relation to tachycardia “entrainment.” Impulse collision rather than interference is the fundamental mechanistic feature that distinguishes the type of linking observed in example No. 4 from that seen in each of the first three cases. During ventricular pacing in example No. 4 the occurrence of fusion beats gives the impression that the preexisting bundle branch reentrant tachycardia transiently “speeds up” to the pacing rate. This is merely an electrocardiographic illusion, however, since true reentrant tachycardia does not exist during this type of linking, owing to repeated collisions that prevent circulating impulses from completely traversing the circuit. Only after pacing is terminated do the collisions cease, thereby permitting unimpeded reentrant excitation to occur once again.

Waldo et al. have used the term “transient entrainment” to describe an analogous phenomenon observed in orthodromic reciprocating tachycardias involving an accessory pathway. Using incremental overdrive pacing, these investigators were able transiently to entrain such tachycardias to an average of 30 beats/min faster than the spontaneous rate of tachycardia. At more rapid pacing rates, bilateral block in the macroreentry circuit developed and termination of the tachycardia typically ensued. The ability to demonstrate such an entrainment zone presumably reflects the existence of an excitable gap in the reentry circuit. 5, 20

During attempted overdrive pacing, entrainment-type phenomena have also been observed in the settings of atrial flutter, ventricular tachycardia, and even atrioventricular nodal reentry. To our knowledge, however, example No. 4 represents the first documented case of linking by collision producing an entrainment effect in a well-defined ventricular macro-reentry circuit that utilizes both bundle branches and the bundle of His.

Possible occurrence of linking in small reentry circuits. In this report, models of macroreentry were chosen to facilitate demonstration and analysis of the linking process. Nevertheless, it should be appreciated that linking probably also occurs in circuits that are considerably smaller. For example, in patients with so-called dual atrioventricular nodal pathways, Wu et al. documented the existence of two alternative (i.e., shorter or longer) steady-state AH intervals during atrial pacing at a constant CL. The particular AH interval that was manifest depended on whether the first paced beat successfully traversed the fast pathway or instead blocked in that limb and engaged the slower pathway. A mechanism of linking by interference (perpetuating refractoriness in the fast pathway) would account for the maintenance of such unilateral functional block during subsequent paced atrial beats.

Sustained monomorphic ventricular tachycardia is another arrhythmia in man that can originate from a small region of the heart, most likely the subendocardium. A linking mechanism might explain the occurrence of an abrupt change to a different uniform QRS morphology during sustained ventricular tachycardia despite maintenance of the same CL. For example, linking by interference around local diseased tissue could cause impulses emerging from the tachycardia circuit (or automatic focus) to exit preferentially via one of two or more potential pathways. A sudden resolution of this linking phenomenon, as in example No. 1, might well result in a different ventricular activation sequence (and hence an altered QRS morphology) without a change in the rate of tachycardia. On the other hand, sudden development or resolution of linking by an interference process involving a portion of the actual ventricular tachycardia circuit could alter total conduction time of a single revolution, resulting in a change in CL as well. Finally, the demonstration of an entrainment effect during overdrive pacing of typical sustained monomorphic ventricular tachycardia, which is analogous to the phenomenon observed in example No. 4, probably indicates the occurrence of linking by collision in the tachycardia circuit.

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