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Characterization of Double Potentials in a Functionally Determined Reentrant Circuit

Multiplexing Studies During Interruption of Atrial Flutter in the Canine Pericarditis Model

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Objectives. **We tested the hypothesis that double potentials recorded during atrial flutter in a functionally determined reentrant circuit reflect activation of the reentrant wave front around an area of functional conduction block.**

Background. **The center of the atrial flutter reentrant circuit in the sterile pericarditis canine model is characterized by double potentials.**

Methods. **We studied 11 episodes of atrial flutter in eight dogs during interruption of atrial flutter while pacing the atria. A multielectrode mapping system was used to record simultaneously from 190 electrodes on the right atrium (location of reentry).**

Results. **Interruption of atrial flutter occurred when the orthodromic wave front from the pacing impulse blocked in an area of slow conduction in the reentrant circuit. The response of the double potential with interruption of atrial flutter depended on the location of the recording site relative to this area of block. Two types of response were seen. When the double potential was recorded orthodromically distal to this area of block, interruption of atrial flutter was associated with disappearance of the second deflection, and continued pacing after interruption of atrial flutter was not associated with reappearance of the second potential. When the double potential was recorded at a site orthodromically proximal to the area of block, interruption of atrial flutter was not associated with disappearance of the second potential, and when rapid atrial pacing was continued, the double potential remained despite disappearance of the atrial flutter reentrant circuit.**

Conclusions. **Double potentials represent functional conduction block in the center of the reentrant circuit, with each deflection of the double potential reflecting activation on either side of the area of functional block. The data also demonstrate that double potentials are not limited to a reentrant circuit, as they were recorded on either side of an area of block in the absence of such a circuit.**

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We have previously shown that the functionally determined atrial flutter reentrant circuit in our canine pericarditis model is located on the right atrial free wall (1–3) and that the center of the atrial flutter reentrant circuit is characterized by double potentials, defined as two discrete deflections per beat separated by an isoelectric interval (1,4). The observation that double potentials are not recorded during sinus rhythm in this model (1,4) is consistent with the hypothesis that double potentials reflect an area of functional block, much like that reported by Allessie et al. (5,6) in the isolated rabbit left atrium and in an acetylcholine model of atrial flutter (7). Double potentials have also been recorded during studies in other experimental models of atrial flutter (6–9), and in patients with atrial flutter (10–14) and ventricular tachycardia (15–17). Although double potentials have been recorded during these several rhythms, it is still uncertain why they occur and what they mean, particularly when present in reentrant rhythms due to a functionally determined reentrant circuit.

In the presence of anatomic block, it seems clearly established that double potentials reflect sequential activation on either side of the block. For example, double potentials are recorded routinely from catheter electrodes placed in the coronary sinus, and clearly reflect activation (atrial and ventricular) on either side of an area of block (the atrioventricular groove). And, as had been predicted by Allessie et al. (7), in an anatomically determined reentrant circuit, double potentials have been demonstrated to reflect activation on either side of a central area of anatomic block during atrial flutter in a crush lesion model (9) and in a cryolesion model (18). However, in the canine pericarditis

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atrial flutter model, in which the reentrant circuit is functionally determined (1–4), there are two possible explanations for the presence of double potentials in the center of the reentrant circuit: 1) Double potentials may reflect sequential activation on either side of a central area of block, but in this case functional block; or 2) double potentials may reflect slow conduction through the center of the reentrant circuit (19), as very slow conduction through the center of the reentrant circuit could theoretically produce a double potential (20,21). In this study, we used the techniques of rapid atrial pacing and simultaneous multisite mapping during induced atrial flutter in the canine pericarditis model to test the hypothesis that the double potentials recorded during atrial flutter in a functionally determined reentrant circuit reflect activation of the circulating reentrant wave front around an area of functional conduction block rather than slow conduction through the center of the reentrant circuit.

Methods

Eleven episodes of induced atrial flutter were studied in eight adult mongrel dogs weighing 18 to 25 kg, following creation of sterile pericarditis (22). All the studies were performed in accordance with the guidelines of the Institutional Animal Care and Use Committee.

Creation of the model. Sterile pericarditis was surgically created in each dog with the techniques described by Pagé et al. (22) from our laboratory. At operation, three pairs of stainless steel wire electrodes (0 Flexon, Davis and Geck) coated with FEP polymer except for the tip were sutured on three epicardial atrial sites: the sulcus terminalis of the right atrium; the interatrial band, known as Bachmann's bundle, and the posteroinferior left atrium close to the proximal portion of the coronary sinus. The interelectrode distance of each electrode pair was about 5 mm. These wire electrodes were brought out through the chest wall and exteriorized posteriorly in the neck near the midline. In five dogs, electrodes were not placed in the sulcus terminalis site for ease in subsequent placement of the electrode array for later simultaneous multielectrode mapping studies in the open chest state.

Studies to determine inducibility of atrial flutter. Before performing studies in the open chest state, it was necessary to make certain that the atrial flutter could be induced. Therefore, beginning on the 2nd or 3rd postoperative day, induction of atrial flutter was attempted in the conscious, nonsedated state using the epicardial wire electrodes placed at the initial operation in all study dogs. Induction of atrial flutter was attempted by pacing performed from one of the electrode sites (sulcus terminalis, Bachmann's bundle region or posteroinferior left atrium) by using techniques described previously (1,2,22). During these studies, electrocardiographic (ECG) leads I, II and III and bipolar electrograms from the three electrode sites were monitored on an oscilloscope and recorded on photographic paper at a speed of 100 mm/s using an Electronics for Medicine VR-16 switched beam oscilloscopic recorder. The data were also recorded simultaneously on FM tape using a Honeywell 101 FM tape recorder.

Multisite mapping studies. When atrial flutter was shown to be inducible, the dogs were anesthetized with pentobarbital (30 mg/kg body weight, intravenously) and mechanically ventilated with a Harvard respirator. The body temperature of the dogs was kept within the normal physiologic range throughout the study by using a heating pad. The chest was opened through a median sternotomy. The heart was exposed, the epicardium was gently separated from the adherent pericardium, and then the heart was suspended in a pericardial cradle. After removal of the gauze strips from both atria, the talcum powder that had become encrusted was carefully peeled off. The stainless steel wire electrodes previously sutured on the sulcus terminalis were removed to permit placement of the epicardial electrode array in the three dogs with electrodes at this site. A new reference bipolar electrode was sutured on the tip of the right atrial appendage. Then, a previously described electrode array (2,3) containing 190 electrodes was secured to the right atrial free wall with a Velcro belt.

Induction of atrial flutter. The same pacing protocol used during the closed chest state was used to induce atrial flutter in the open chest state. The only difference was that since the sulcus terminalis electrode pair was removed, it was not used as one of the pacing sites. In its place, the pair of electrodes placed at the right atrial appendage was used.

After sustained atrial flutter was induced, rapid atrial pacing was initiated at a rate 5 to 10 beats/min faster than the spontaneous rate, continued for up to 10 s, and terminated abruptly. If atrial flutter was not interrupted, rapid atrial pacing was again initiated, but with an increment in rate of 5 to 10 beats/min. This procedure was repeated until the atrial flutter was interrupted (3).

Data acquisition. Before, during and after the interruption of 11 episodes of atrial flutter in eight dogs, unipolar electrograms were recorded simultaneously from 190 electrodes distributed over the right atrial free wall along with ECG lead II, and processed using a previously described cardiac mapping system designed and built at Case Western Reserve University (2,3,23). The data were displayed on a graphic monitor while being stored in a circular buffer. This procedure permitted us to monitor and edit all events and stop filling the circular buffer whenever we wished to retain recorded data. Data were archived on either a floppy disk or a hard disk, both being resident to the host system.

Data analysis. Analysis of data consisted of selecting activation times and drawing isochronous maps with a minimal resolution of 1 ms (2,3,24). Bipolar signals were obtained in software by taking the potential difference between pairs of unipolar signals. To aid in the selection of an activation time at an electrode pair site, the bipolar signal and both unipolar signals were displayed on a graphic screen and a cursor was used manually to select an activation time. A time reference signal was selected either from the stimulus

JACC Vol. 22, No. 7
December 1993:2022–32

artifact in each beat during atrial pacing or from the electrode pair site activated earliest in each spontaneous beat and was depicted as zero activation time. For double potentials, the activation time at these sites was assigned to the deflection with the largest amplitude (25). The center of the reentrant circuit was defined by analyzing the isochronous lines. In this study, double potentials were defined as atrial electrograms with two discrete deflections per atrial beat separated by an isoelectric interval or a low amplitude baseline (12), and slow conduction was defined as conduction velocity less than 0.2 m/s (19).

Results

Simultaneous Multisite Mapping Studies of the Interruption of Atrial Flutter

Eleven episodes of the pacing interruption of induced atrial flutter in eight dogs were analyzed. The mean cycle length of stable atrial flutter was 158 ± 26 ms (range 135 to 190 s). The reentrant wave front in the right atrial free wall circulated in a clockwise direction in seven episodes and in a counterclockwise direction in four. Rapid atrial pacing to interrupt atrial flutter was performed from the right atrial appendage in seven episodes and the posteroinferior left atrium in four. The mean atrial pacing cycle length that interrupted atrial flutter was 140 ± 22 ms.

Disappearance of double potentials with the pacing interruption of atrial flutter. A representative example of the disappearance of double potentials associated with the interruption of atrial flutter is shown in Figures 1A through 1G. The top portion of Figure 1A shows an atrial activation map during sustained atrial flutter at a cycle length of 142 ms. Isochrones are at 10-ms intervals. Arrows show the main direction of the reentrant activation wave front. The activation wave front circulates in a clockwise direction around an area of apparent functional block, represented by dashed lines. The circled numbers 1 through 8 represent electrode recording sites in the reentrant circuit adjacent to lettered sites A through D at the center of the reentrant circuit. Double potentials were recorded at sites A through D. The lower panel of Figure 1A shows bipolar electrograms recorded from these lettered sites and selected adjacent numbered sites. Note the normal electrograms recorded from sites 1, 2, 4, 7 and 8 along with the double potentials from sites A, B, C and D. As can be appreciated from these records, each potential of the double potentials reflects activation time on opposite sides of the region of functional block. At electrode pair site D, the "a" potential activation time corresponds to the activation time at electrode site 4, and the "b" potential activation time corresponds to the activation time at electrode site 7. Thus, the activation times of the double potentials correspond closely to those of the circulating reentrant wave front on each side of the area of functional block of the reentrant circuit. Note that the interval from the "a" potential to the "b" potential recorded at site D is

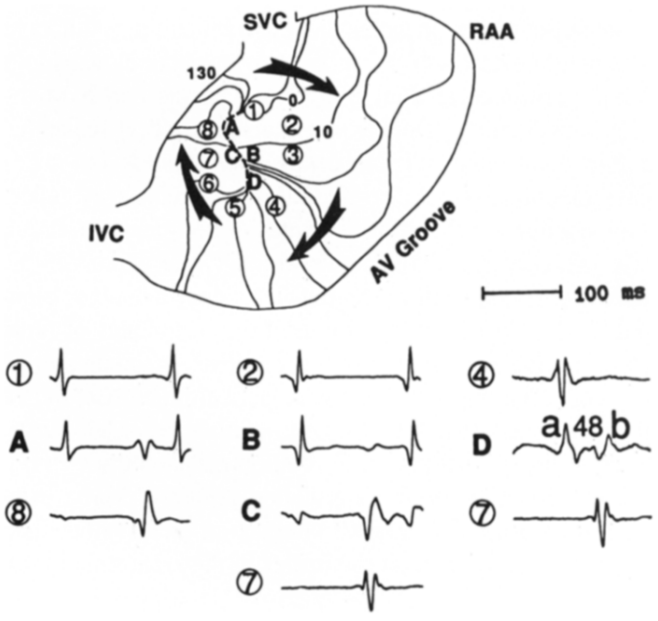


Figure 1A. Ten-millisecond isochronous maps during atrial flutter and selected bipolar electrograms. **Top**, Isochronous maps show sustained atrial flutter at a cycle length of 142 ms. **Uncircled numbers** show the activation time at each isochronous line. **Arrows** show the main direction of the activation wave front. The activation wave front circulates in a clockwise direction around an area of apparent functional block **(dashed line)**. In the **lettered sites adjacent to this dashed line**, double potentials were recorded. The **circled numbers** represent each electrode pair site selected arbitrarily from pair sites in the reentrant circuit. AV = atrioventricular; IVC = inferior vena cava; RAA = right atrial appendage; SVC = superior vena cava. **Bottom**, Bipolar electrograms recorded from these sites. The single electrograms were recorded from sites 1 through 8 and the double potentials from sites A to D. Each potential of the double potentials reflects activation time on opposite sides of the region of functional block. At electrode site D, the double potentials recorded were separated by 48 ms. The "a" potential corresponds to the activation time at site 4 and the "b" potential corresponds to that at site 7. The **circled numbers** 1 through 8 and A to D indicate the site of the electrode pairs in Figures 1B to 1G.

48 ms. Figure 1B shows electrograms from all the numbered sites recorded simultaneously with ECG lead II. The relative sequence of activation is indicated by the arrows.

Figures 1C and 1D show the response of the double potentials at site D during interruption of atrial flutter by rapid atrial pacing at a cycle length of 125 ms performed from the right atrial appendage in the same episode shown in Figures 1A and 1B. In the ECG lead II tracing in Figure 1C, S_3 through S_9 correspond to each respective stimulus artifact. The interval from each stimulus artifact to the "a" component of the double potential was almost constant, ~65 ms from beats S_3 through S_8. Conversely, the interval from each stimulus artifact to each second potential, "b", prolonged from 125 ms during S_3 to 135 ms during S_5, until the stimulus from S_6 did not produce a "b" potential. Termination of pacing (Fig. 1D) demonstrates that the atrial flutter indeed has been interrupted. Also, neither double

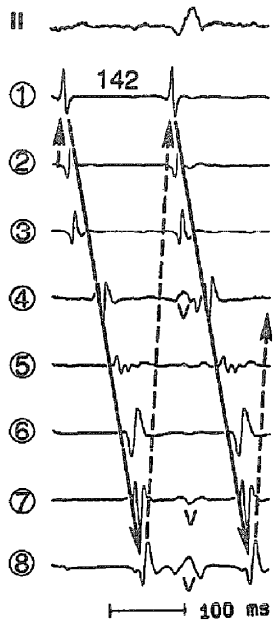


Figure 1B. Lead II of the surface electrocardiogram (II, top trace) and bipolar electrograms during the same episode of atrial flutter shown in Figure 1A. **Circled numbers** 1 to 8 represent each electrode pair site shown in Figure 1A. Arrows indicate the direction of the main activation wave front. v = ventricular electrogram; 142 = atrial flutter cycle length in ms.

potentials nor conduction block was recorded during sinus rhythm (Fig. 1D).

These observations can be explained by analysis of the activation maps shown in Figure 1E. They are drawn using the stimulus artifact for S_6 and S_7 as a time reference. The numbers in parentheses in the top map represent activation times measured from the previous stimulus artifact because that area was activated by an orthodromic activation wave front from the previous pacing stimulus, S_5 (open arrow). The circled numbers indicate the sites of the electrograms shown in Figure 1F, and are the same recording sites shown in Figure 1A. In the map of S_6 (top map of Fig. 1E), the antidromic activation wave front of S_6 collided with the orthodromic activation wave front (open arrow) of the previous beat, S_5. However, the orthodromic activation wave front of the S_6 pacing impulse blocked in an area close to the inferior vena cava. This also can be seen from the electrogram recordings shown in Figure 1F. Therefore, the area activated by the orthodromic wave front of the previous beat, S_5, was not activated by either the orthodromic or antidromic wave front of the S_6 pacing stimulus, so that there was localized conduction block for one beat. This is the so-called third criterion of transient entrainment and indicates that the atrial flutter reentrant circuit has been interrupted (26,27). Thus, indeed, with beat S_7, there was no longer any circus movement. Rather, there was radial activation of the right atrial free wall generated by the impulse from the pacing site (Fig. 1E). The disappearance of double potentials associated with interruption of the reentrant circuit by rapid atrial pacing was seen in 9 of the 11 episodes and was always associated with the disappearance of an area of functional block.

After interruption of the atrial flutter by the S_7 beat, pacing was not stopped until S_{14} (Fig. 1C and 1D). In Figure 1C a double potential reappears with beat S_8, the second component now labeled "c". This double potential remained until pacing was stopped (Fig. 1D). When pacing was stopped, sinus rhythm resumed (Fig. 1D). The observations concerning the presence of a double potential at site D during continued rapid pacing after interruption of atrial flutter can be explained by analysis of the atrial activation map in Figure 1G. As seen in the map, during pacing at a rate faster than the previous atrial flutter rate, localized functional block was present in the region of electrode site D in the presence of radial activation of the right atrial free wall generated by the impulse from the right atrial appendage pacing site. However, double potentials did not reappear at site A, B or C, as they were not associated with conduction block after interruption of atrial flutter by atrial pacing. In 9 of the 11 episodes studied, during continued pacing after the pacing interruption of the atrial flutter reentrant circuit, reappearance of a double potential was seen in one or more recording sites from which double potentials were recorded during induced atrial flutter. This was always because conduction block, usually localized, occurred during the continued rapid pacing.

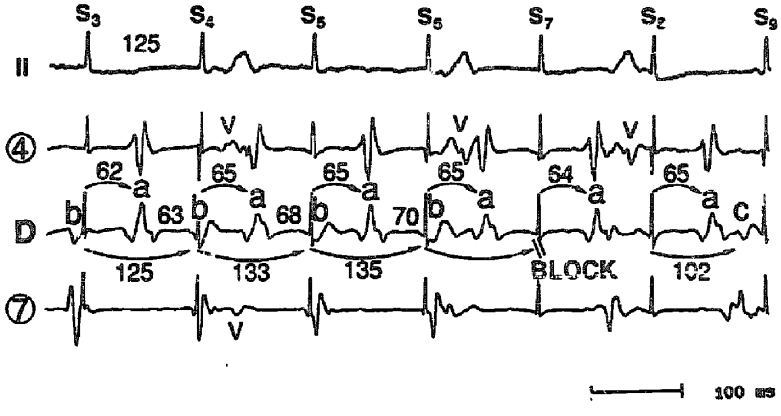


Figure 1C. Atrial pacing at a cycle length of 125 ms is performed during the same episode of atrial flutter shown in Figure 1A from the right atrial appendage. S_3 to S_9 correspond to the respective stimulus artifact. In electrode pair D, the numbers represent the interval (in ms) from each stimulus artifact to the respective potential of the double potential and the interval from an "a" potential to a "b" potential. Reentry is interrupted by rapid pacing in S_6. In S_8, a new potential, "c", appears. The **circled numbers** 4 and 7 and the letter D indicate the electrode pair sites shown in Figure 1A. Abbreviations as in Figure 1B. See text for details.

JACC Vol. 22, No. 7
December 1993:2022–32

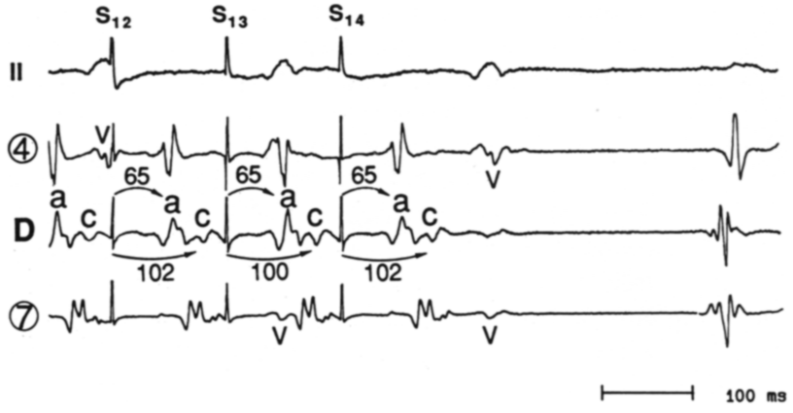


Figure 1D. Cessation of the period of atrial pacing shown in Figure 1D. S_{12} to S_{14} correspond to the respective stimulus artifact. In electrode pair D, the numbers represent the interval (in ms) from each stimulus artifact to the respective potential of the double potentials ("a" and "c"). The deflections at the right are the atrial deflections in the first sinus beat after the cessation of rapid atrial pacing. Symbols and abbreviations as in Figures 1B and 1C.

Nondisappearance of double potentials associated with interruption of atrial flutter. Figures 2A to 2E show atrial activation maps and selected bipolar atrial electrograms from a different study during an episode of atrial flutter at a cycle length of 150 ms. In the map shown in Figure 2A, the reentrant activation wave front circulates in a counterclockwise direction around an area of apparent functional block. The circled numbers represent electrode recording sites in the reentrant circuit adjacent to the lettered sites from which double potentials were recorded. The lower panel shows bipolar electrograms recorded from sites A through F, and 1 through 7. Once again, each potential of the double potentials reflects activation time on opposite sides of the region of apparent functional block. Thus, at electrode pair site A, the "a" potential activation time corresponds to the activation time at site 1 and the "b" potential activation time corresponds to the activation time at site 2. The interval from potential "a" to potential "b" was 44 ms. Note also that double potentials were recorded from sites in which relatively rapid conduction (absence of crowding of isochrones) was occurring on each side of the area of functional block (sites A, B, C and D).

Figures 2B and 2C show the response of the double

Figure 1E. Ten-millisecond isochronous maps during interruption of the same episode of atrial flutter shown in Figure 1A. **The upper and lower panels** show isochronous maps for the 6th (S_6) and 7th (S_7) paced beats, respectively, during rapid pacing at a cycle length of 125 ms delivered from the right atrial appendage (RAA). **The circled square wave** indicates the right atrial appendage pacing site. **The numbers in parentheses** are activation times in ms from the previous stimulus artifact. **The open arrow** during S_6 represents the orthodromic activation wave front of the previous pacing impulse. In the upper panel, **the solid black line (thin arrows)** represents block of the orthodromic activation wave front of the S_6 pacing impulse. This is associated with the interruption of the atrial flutter. S_7 is the first paced beat immediately after the interruption of atrial flutter. Abbreviations and symbols as in Figure 1A.

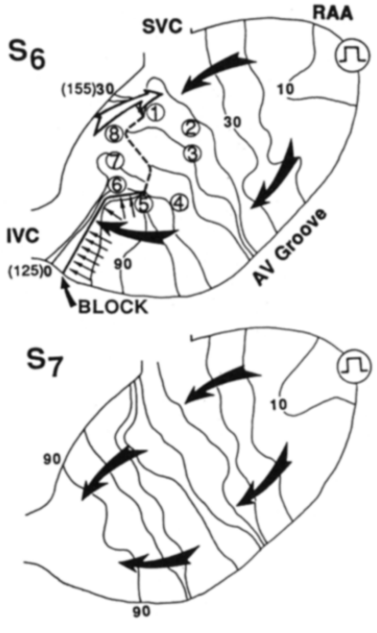
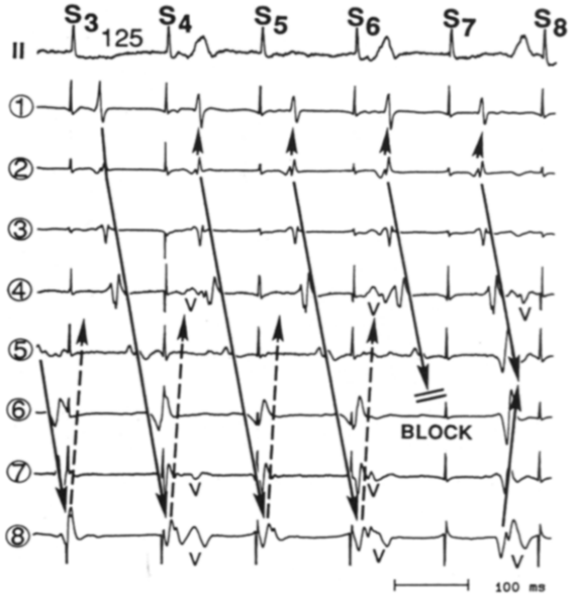


Figure 1F. Lead II of the electrocardiogram recorded simultaneously with bipolar electrograms obtained from the same episode shown in Figure 1B. The **arrows** show the relative activation sequence. S_3 to S_8 correspond to respective stimulus artifacts. The main activation wave front was blocked between electrode pair sites 5 and 6 in S_6. Abbreviations as in Figure 1B.



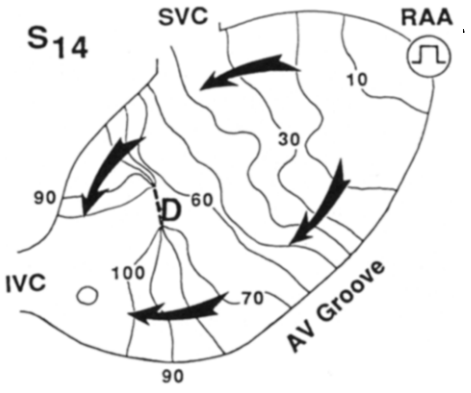


Figure 1G. Ten-millisecond isochronous map at the last paced beat S_{14} in the same episode shown in Figure 1D. The letter D represents the same electrode pair site D shown in Figure 3A. Abbreviations and symbols as in Figure 1A and 1E.

potentials at site A during the interruption of atrial flutter by rapid pacing performed from the right atrial appendage at a cycle length of 141 ms. In this example, the interval from the "a" potential to the "b" potential remained almost constant during rapid atrial pacing, and the double potentials remained despite the interruption of atrial flutter associated with the occurrence of block of the orthodromic activation wave front produced by beat S_8 (Fig. 2D). This is explained by analysis of the isochronous maps of beats S_8 and S_9 shown in Figure 2D.

In the isochronous map of beat S_8 (Fig. 2D), block of the orthodromic activation wave front occurred in the area of the pectinate muscles. In the next beat, S_9, a double potential was still recorded at site A (Fig. 2B) despite the disappearance of the atrial flutter reentrant circuit (Fig. 2D). The observations 1) that the site of the double potential recording was orthodromically proximal to the area of localized block that interrupted the atrial flutter, 2) that this site was activated at the same rate and from the same direction during each pacing impulse, and 3) that the area of block denoted by the dashed lines in Figure 2D remained during the period of rapid pacing even after the interruption of atrial flutter explains why the "a" and "b" potentials remained constant throughout pacing.

The continuation of the double potential during atrial pacing despite interruption of atrial flutter was seen in 3 of 11 studies. However, as now can be anticipated from our new understanding of the response of double potentials to rapid pacing, depending on the location of the recording site, one may expect to see either of the responses of double potentials during rapid pacing interruption of atrial flutter. Thus, a site proximal to the area of block could be expected to maintain the double potential during pacing despite the interruption of atrial flutter, but a site distal to the area of block should show disappearance of one of the double potentials associated with the localized conduction block of the wave front from the pacing impulse that interrupts the atrial flutter. This is illustrated by the response of the electrode pair at site F, shown in Figure 2A, which was located orthodromically distal to the localized conduction block. The interval from the "b" component to the "a" component (the shorter of the two intervals of the double potential) in electrode pair site F was 64 ms during atrial flutter before rapid atrial pacing (Fig. 2A). As shown in Figure 2E, during rapid atrial pacing, the interval from each stimulus artifact to the "a" component of the double potential was constant at 70 ms from S_5 through S_8. However, the interval to each "b" potential (i.e., the second potential of the double

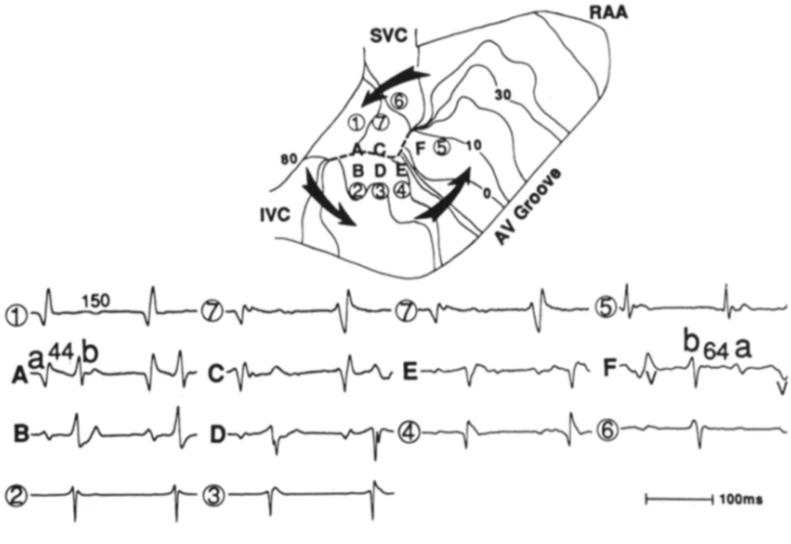


Figure 2A. Ten-millisecond isochronous map **(top)** and selected bipolar electrograms **(bottom)** during atrial flutter before pacing. **Top,** The numbers show the activation time for each isochrone, the **arrows** show the main direction of the activation wave front and the **dashed line** represents a region of functional block during atrial flutter at a cycle length of 150 ms due to counterclockwise rotation of the circulating reentrant wave front. Double potentials were recorded in **the lettered sites adjacent to this dashed line.** The **circled numbers** represent each electrode pair site selected arbitrarily from pair sites in the reentrant circuit. The **circled numbers** 1 through 7 and the letters A to F indicate the site of the electrode pairs in Figures 2B, 2C, 2D and 2E. Abbreviations as in Figure 1A. **Bottom,** Bipolar electrograms recorded from these sites showing the double potentials recorded from sites A to F. Each potential of the double potentials reflects activation time on opposite sides of the region of functional block. At electrode site A, double potentials separated by 44 ms were recorded; the "a" potential corresponds to the activation time at site 1 and the "b" potential corresponds to the activation time at site 2. At electrode site F, double potentials separated by 64 ms were recorded. v = ventricular deflection.

JACC Vol. 22, No. 7
December 1993:2022–32

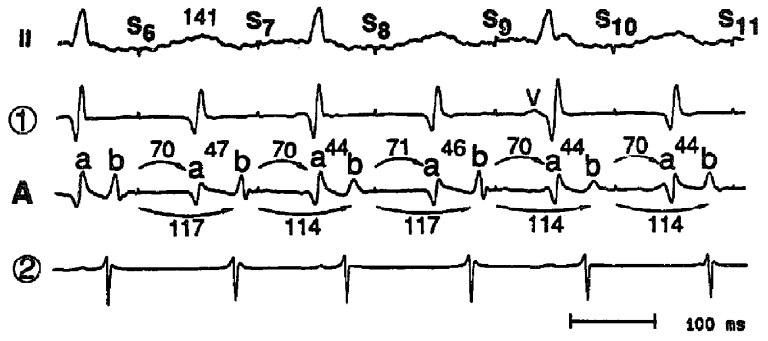


Figure 2B. Atrial pacing at a cycle length of 141 ms from the right atrial appendage during the same atrial flutter shown in Figure 2A from the right atrial appendage. S_6 to S_{11} correspond to the respective stimulus artifact. In electrode pair A, the numbers (in ms) represent the interval from each stimulus artifact to the respective potential of the double potential and the interval from an "a" potential to a "b" potential. Reentry is interrupted by rapid pacing in S_8. **Circled numbers** 1 and 2 and the **letter** A indicate the electrode pair sites shown in Figure 2A. Abbreviations as in Figure 1B. See text for details.

potential) prolonged gradually. Finally, the stimulus from S_8 did not produce a "b" potential. Thus, during the episode illustrated in Figures 2A through 2E, the double potential at site A persisted during the pacing interruption of atrial flutter, but the double potential at site F disappeared. The latter was the same type of response shown in Figure 1C from electrode recording site D.

Unipolar electrograms recorded from the area of double potentials. Figure 3 shows the bipolar electrogram (upper trace) and one of the two unipolar electrograms recorded at electrode pair sites A, C, and D shown in Figure 2A. The arrow shows the point used to measure the respective activation times. Asterisks indicate the lower amplitude component of each unipolar double potential. The lower amplitude component (asterisk) of the unipolar electrogram in A and C shows no negative component. This finding indicates that the activation wave front that causes the lower amplitude component traveled toward each electrode (positive deflection) but did not subsequently travel away from that electrode. It is also consistent with the activation wave front blocking in the center of the reentrant circuit. Furthermore, the activation wave front traveled perpendicularly toward the functional center of block at sites B, D, and E so that the lower amplitude component of the double potential in the unipolar electrogram recorded at sites A and C would be expected to record only a positive potential (Fig. 3). However, this was not true for site D. Note that the activation wave front relative to sites A and C traveled almost parallel to the area of functional block. Therefore, the lower amplitude component of the double potential recorded at site D might be expected to demonstrate both a positive and a subsequent negative component (Fig. 3). In fact, the lower amplitude component of the unipolar double potential electrogram at site D does show both a positive and a negative component. This is explained by observing the direction of the activation wave front on the other side of the area of functional block opposite to each of the recording sites.

Discussion

Responses of double potentials during rapid pacing of atrial flutter due to a functionally determined reentrant circuit. As is clearly shown in this study, during atrial flutter, double potentials always were recorded in the center of the atrial flutter reentrant circuit. Each deflection of the double potentials reflected activation time on opposite sides of the region of apparent functional block. Thus, the activation times reflected in the double potentials corresponded closely to those of the circulating reentrant wave on each side of the area of functional block of the reentrant circuit, confirming the original observations of Okumura et al. (1) made during sequential site mapping studies in the same sterile pericarditis model.

There were two different responses of recorded double potentials during rapid pacing of atrial flutter in the present study. This is explained by using the schema shown in Figure 4. In the left panel, the atrial flutter reentrant wave front is shown circulating in a clockwise direction around an area of functional block, represented by dashed lines in the center of the reentrant circuit. The letters "b" and "a" each represent one deflection of the double potential recorded

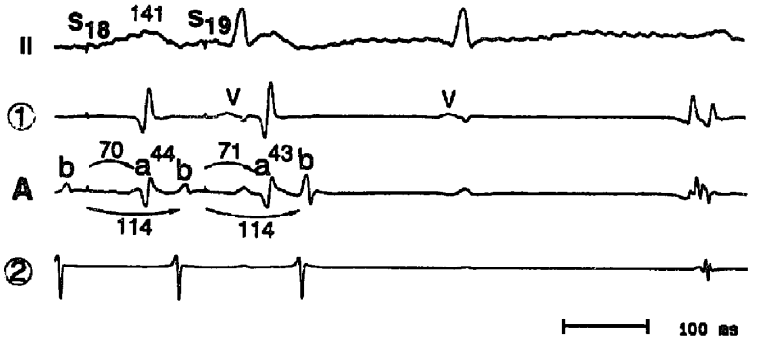


Figure 2C. Cessation of the same episode of atrial pacing shown in Figure 2B. S_{18} and S_{19} correspond to the respective stimulus artifact. In electrode pair A, the **numbers** represent the interval (in ms) from each stimulus artifact to the respective potential of the double potentials. The deflections at the right are the atrial deflections in the first sinus beats after the cessation of rapid atrial pacing. Abbreviations and symbols as in Figure 1B and 2B.

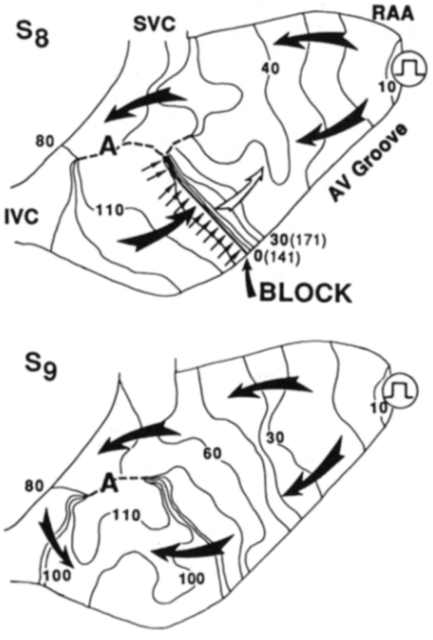


Figure 2D. Ten-millisecond isochronous maps during the interruption of the same atrial flutter shown in Figure 2B. The **upper** and **lower panels** show isochronous maps for the 8th (S_8) and 9th (S_9) paced beats, respectively, during rapid pacing at a cycle length of 150 ms delivered from the right atrial appendage. The **circled square waves** indicate the right atrial appendage pacing site. The **numbers in parentheses** are activation times from the previous stimulus artifact. The **open arrow** during S_8 represents the orthodromic activation wave front of the previous pacing impulse S_7. In the **upper panel**, the **solid black line (thin arrows)** represents block of the orthodromic activation wave front of the S_8 pacing impulse. This is associated with the interruption of the atrial flutter. The letter A represents the same electrode pair site shown in Figure 2A. Symbols and abbreviations as in Figure 1A.

from a site at the center of the reentrant circuit during atrial flutter, with each deflection reflecting activation on opposite sides of the area of functional block. The dotted region of the circulating reentrant wave front represents the excitable gap of the reentrant circuit, and the serpentine lines represent areas of slow conduction. In the right panel, the two different responses of the double potential at the interruption of atrial flutter by rapid atrial pacing are shown. Xn represents the paced beat which interrupts the atrial flutter reentrant circuit, and Xn-1 represents the orthodromic wave front of the previous beat. In each type of response, the antidromic activation wave front of Xn collides with the orthodromic activation wave front of the previous beat, Xn-1. And in each type of response, the orthodromic wave front of Xn also blocks, but at a different site in each type of block. This results in the disappearance of the "b" deflection in the type I response, but the continued presence of the "b" deflection in the type II response. Thus, in the type I response, because of the location of the block of the orthodromic wave front of Xn, activation of the area responsible for generating the "b" deflection did not occur. Therefore, the interruption of the atrial flutter was associated with the disappearance of the "b" deflection. As previously noted, this was seen in 9 of the 11 studies. In the type II response, because block of the orthodromic wave front of Xn occurred orthodromically more distal, activation of the area responsible for generating the "b" deflection did occur. Thus the double potential continued to be present. As previously noted, this was demonstrated in 3 of the 11 studies.

Double potentials recorded during atrial flutter remained (Fig. 2, B and C) or resumed (Fig. 1, C and D) in electrograms recorded from some electrode pairs after the interruption of reentrant excitation in all episodes. The explanation was always the presence of a functional area of block, with each deflection of the double potential reflecting sequential activation on each side of the area of block. We also found that the lower amplitude component of the double potential recorded from a unipolar electrogram in some, but not all, sites had no negative component (Fig. 3). The latter is also consistent with the interpretation that the lower amplitude component of the double potential reflects an activation wave front coming toward the recording site and then blocking. In sum, our data are consistent with the notion that double potentials reflect activation on either side of an area of functional block. However, although this was shown to occur primarily in the center of a reentrant circuit, double potentials reflecting sequential activation on either side of an area of block are not limited to a reentrant circuit.

Interpretation of double potentials in other studies. Allessie et al. (5), on the basis of studies in the rabbit left atrium, first speculated that the central area of apparent block around which the reentrant wave front circulates in a functional reentrant circuit was inexcitable due to electrotonic influence of the continuously circulating reentrant excitation wave front. Implicit in this is that double potentials recorded at the center of the reentrant circuit are due to electrotonus and reflect block. In subsequent studies in the rabbit atria, this group (6) showed that this latter interpretation was not correct, as membrane resting potentials recorded from the

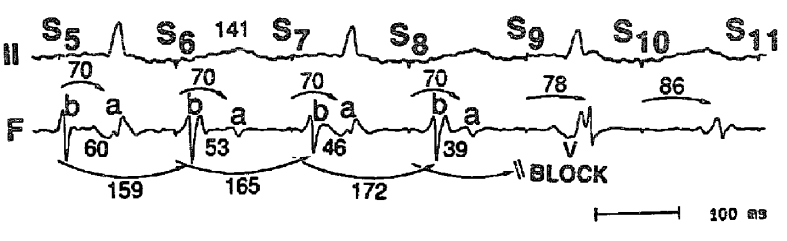


Figure 2E. Bipolar electrograms from site F during atrial pacing at a cycle length of 141 ms delivered from the right atrial appendage recorded simultaneously with lead II of the electrocardiogram. From the same episode shown in Figure 2A. Site F is indicated in Figure 2A. S_5 to S_{11} correspond to the respective stimulus artifact. The numbers (ms) at electrode pair F represent the interval from each stimulus artifact to the respective potentials.

JACC Vol. 22, No. 7
December 1993:2022–32

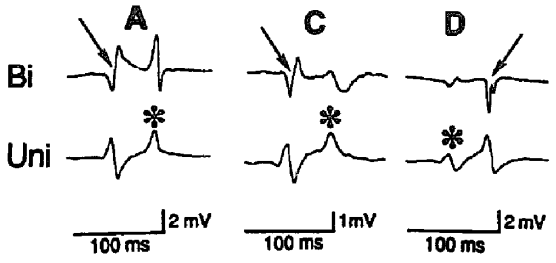


Figure 3. Bipolar (Bi) and unipolar (Uni) electrogram signals recorded simultaneously from electrode pairs A, C and D in the same episode of atrial flutter shown in Figure 2A. **Arrows** show the sites of the activation time of the main deflection of the double potential in the bipolar electrograms. **Asterisks** represent the lower signals in the double potentials. The lower potentials in electrode pairs A and C demonstrated no negative component.

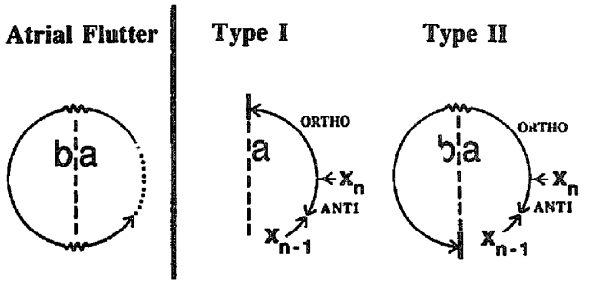


Figure 4. The schema on the responses of double potentials during interruption of the reentrant circuit by rapid pacing. X_{n-1} = the paced beat before X_n. X_n = the paced beat that interrupted the reentrant circuit; a and b = the respective potential in the double potentials; **dashed lines** = functional block; **solid line** = the localized block of the orthodromic Xn wave front. **Arrows** show the main activation wave front. See text for details. ANTI = antidromic; ORTHO = orthodromic.

functional center did not show ". . . such a degree of depolarization that they [could] be expected to be inexcitable." Rather, they proposed that the functional center of the reentrant circuit ". . . is continuously invaded by multiple centripetal wavelets which are blocked in the very center of the circuit. Here the impulse encounters fibers that are still in their refractory phase because they [have just been] activated by another centripetal wavelet just half a revolution time earlier." Thus, the double potentials they recorded (6) in the functional center of the reentrant circuit indicate block and serve as a marker for the location of the functional obstacle around which the reentrant wave front circulates.

Later studies by Allessie et al. (7) of atrial flutter in an acetylcholine infusion canine model also demonstrated double potentials recorded only during atrial flutter and only at the functional center of the reentrant circuit. They concluded that the double potentials reflected activation ". . . of [reentrant] waves passing along at either side [of the area of functional block]." They further stated that ". . . this sign [double potentials] can be regarded as indicative of functional conduction block, although it can also be expected in case of a thin scar caused by an intra-atrial incision." As we indicated in the introduction to this study, the latter "expectation" proved true for equivalent lesions, as double potentials have now been recorded from the anatomically created (crush lesion and cryolesion) center of the reentrant circuit in two recently described atrial flutter models (9,18).

Cosío et al. (11) in studies of atrial flutter in patients speculated that the double potentials they recorded only during atrial flutter could be explained either by a line of complete block or by slow conduction, but that the inherent limitations of the catheter mapping technique prevented more than just speculation. In studies of patients with atrial flutter (12) and ventricular tachycardia (17), our laboratory used catheter electrode recording and pacing (entrainment) techniques to provide indirect but strong evidence that in both of these rhythms, double potentials are recorded from the functional center of the reentrant circuit and that the potentials probably reflect activation on either side of an area of block. However, in our study of atrial flutter (12), we did also describe persistent double potentials that seemed not to have any relation to the reentrant circuit and that were also recorded during sinus rhythm. Furthermore, Saoudi et al. (13) described double potentials recorded during atrial flutter that were demonstrated to be dissociated from the reentrant circuit. This is consistent with the demonstration in the present study that not all double potentials reflect block at the center of a reentrant circuit.

Kay et al. (16) have interpreted double potentials recorded in a patient with ventricular tachycardia as bracketing a discrete region of slow conduction. However, their data can readily be reinterpreted as reflecting activation on either side of an area of functional block, with the interval between each deflection of the double potential being explained in part by conduction of the circulating reentrant wave front through an area of slow conduction that is part of the reentrant circuit (17). In fact, a variant of this is most probably the explanation for the AH interval recorded from the AV junction. The AH interval is a double potential that at first blush seems to reflect activation bracketing an area of slow conduction, the AV node. However, the sites from which the atrial (A) and His (H) deflections are recorded are anterior to the location of the AV node (28,29). At such sites, there clearly is anatomic block between atrial tissue from which the A deflection is recorded and the His bundle from which the H deflection is recorded. But also clearly, for the impulse to get from the atrium to the His bundle, it passes through an area of slow conduction, the AV node. Thus, it can be said that the AH interval reflects sequential activation on either side of a region of anatomic block but that the activation interval between these two potentials includes conduction through an area of slow conduction, the AV node. Moreover, there are many other examples of double potentials recorded from the heart during sinus rhythm that clearly reflect block. Thus, electrograms recorded from the coronary sinus show double potentials that reflect local

activation of the left atrium and left ventricle. Electrograms recorded from the bundle branches show double potentials that reflect local activation of either the right or the left bundle branch and the adjacent ventricle. A double potential recorded in patients that might immediately bracket an area of slow conduction is the split His potential (HH^1). But even this example may have other explanations. Thus, published examples of split His potentials not uncommonly show fractionated signals rather than an isoelectric interval between the H and H^1 deflections (30), consistent with slow conduction (19,21). In that instance, the split His potential may not really be a double potential. Furthermore, in some instances, split His potentials appear to reflect longitudinal dissociation in the His bundle, in which case this double potential is a reflection of block with sequential activation on either side of the block (31). In short, double potentials recorded from patients during tachycardia or sinus rhythm seem, with rare exception, to represent sequential activation on either side of an area of conduction block.

Nevertheless, it has been experimentally demonstrated that electrotonic transmission across inexcitable gaps may result in marked conduction delay (19–21,32,33), so that very slow conduction could theoretically account for the double potentials. Thus, double potentials may represent discontinuous electrotonic transmissions or continuous conduction. Regarding the latter possibility, the studies of Dillon et al. (19) in a canine model of ventricular tachycardia clearly showed that slow continuous conduction across an area of apparent functional block in the center of the reentrant circuit was characterized by fractionated electrograms of long duration. We never recorded such electrograms at the center of the reentrant circuit, nor were they a part of the double potentials. Dillon et al. (19) also emphasized the importance of spatial resolution of the electrodes in the array to allow the possibility of recording such electrograms. Although the two electrode arrays were not quite the same (theirs had 192 electrodes with a 1-mm interelectrode distance between each electrode of a pair and a 3.5-mm distance between electrode pairs; ours had 190 electrodes with a 1.5-mm interelectrode distance and a distance of 4.2 to 6 mm between electrode pairs), we believe the spatial resolution of our electrode array was sufficiently comparable that we should have recorded such electrograms if they were present. In addition, the response to pacing in the present study, as diagrammed in Figure 4, seems to provide strong evidence that double potentials which are recorded during a tachycardia that is due to a functional reentrant circuit simply reflect block. The data during rapid pacing after interruption of the atrial flutter support this view as well.

Clinical implications. As indicated, double potentials have been recorded in patients from the atria during atrial flutter and from the ventricles during ventricular tachycardia. It is generally thought that both of these rhythms are amenable to cure with catheter ablation techniques if the appropriate portion of the respective reentrant circuit could be identified and ablated. Mapping of these arrhythmias is difficult with currently available catheter electrode techniques. Thus, one looks for electrographic markers of a critically important portion of the reentrant circuit, perhaps an area of slow conduction, that could be ablated. Identification of such markers should permit application of ablative energies at that site to provide effective therapy. We submit that double potentials rarely reflect such markers. Because double potentials recorded during a tachycardia seem to reflect an area of block, and usually in the center of the reentrant circuit, we suggest that ablation at such a site seems best avoided, as it might simply change a region of functional block in the center of the reentrant circuit into a region of anatomic block. However, the presence of a double potential in which one potential is fractionated may reflect block in which activation on one side of the block is slow conduction in an isthmus. The latter might occur, as suggested by Fitzgerald et al. (34) (Fig. 9), and, in fact, identifies a favorable area for ablation.

Conclusions. In summary, we have demonstrated that in a functionally determined reentrant circuit, double potentials recorded at the center of the reentrant circuit reflect sequential activation on each side of the center. Furthermore, we have presented mapping data supporting the hypothesis that the double potentials are a result of sequential activation on either side of an area of functional block, rather than a result of slow conduction across the center of the reentrant circuit. We have also demonstrated that double potentials reflecting activation on either side of a region of functional block may be present apart from a reentrant circuit. And finally, we have suggested that although double potentials theoretically may immediately bracket an area of slow conduction, in fact, classic double potentials (each potential of the double potential is a single, nonfractionated deflection) probably rarely do.

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