Mechanism of Double Potentials Recorded During Sustained Atrial Flutter in the Canine Right Atrial Crush-Injury Model

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Background. During atrial flutter, double potentials may be recorded at specific sites in the atria. It has been suggested that double potentials represent sequential activations at the center of the reentrant circuit. An alternative hypothesis is that double potentials represent electrical activity in an area of slow conduction. Understanding their mechanism is important because double potentials have been considered a possible indicator of target sites for catheter ablation.

Methods and Results. We systematically studied double potentials in our canine model of atrial flutter produced by right atrial crush injury using a 64-channel computerized mapping system with 56 electrodes on the right atrium in seven mongrel dogs under general anesthesia. Activation maps were recorded during sinus rhythm before and after crush injury, during rapid pacing above and below the crush injury, and during sustained atrial flutter, entrainment of atrial flutter, and termination of atrial flutter induced with D-sotalol (2 mg/kg). During sinus rhythm before crush injury, activation was uniform, and double potentials were not recorded in any dog. After crush injury, activation proceeded up to and around the crush injury, and narrowly split double potentials were recorded in two of seven dogs. During rapid pacing above and below the crush injury, double potentials were recorded in five of seven dogs. During 14 episodes of atrial flutter (mean cycle length, 140±16 msec), double potentials were recorded at electrodes along the crush injury. The activation time of the early x component of the double potentials (25±13 msec) was similar to that of adjacent electrodes above the crush injury (24±11 msec), and the activation time of the late y component (89±13 msec) was similar to that of adjacent electrodes below the crush injury (91±14 msec). The timing of the x and y components was dependent on the location of the recording electrode, with x and y widely spaced at the end of the crush injury near the area of earliest atrial activation during atrial flutter, more equally timed at the center of the crush injury, and more closely timed at the end of the crush injury opposite the area of earliest activation. During transient entrainment, double potentials were accelerated to the pacing rate, but their activation time relative to adjacent electrodes was maintained. During abrupt termination of atrial flutter, the early x component of the double potential was always recorded, but the late y component was not, because of conduction block below the posterior end of the crush injury.

Conclusions. This study has shown in our canine model of atrial flutter that double potentials are recorded from the center of the reentrant circuit and that they represent sequential activations as the reentrant wave front passes on either side of the crush injury. (Circulation 1992;86:628–641)

Key Words • atrial flutter • potential, double

Recent studies in patients with type 1 atrial flutter have demonstrated that double potentials may be recorded by endocardial electrode catheters placed at some specific locations in the atria and not others. One hypothesis proposed for the mechanism of double potentials is that they represent sequential activations at the center of a reentrant circuit as the reentrant wave front passes on either side of the recording electrode. Several studies in experimental models of atrial flutter involving reentry around an arc of functional block support this hypothesis. An alternative hypothesis that has been proposed is that double potentials, like fragmented electrograms, may represent electrical activity in an area of slow conduction within the reentrant circuit. Delineation of their mechanism is of clinical importance because double potentials, which may identify the center of the atrial flutter reentrant circuit rather than a critical area of slow conduction, have been considered a possible target for catheter ablation techniques to cure atrial flutter. Furthermore, catheter ablation for atrial flutter using currently available methods for localizing the reentrant circuit has been only moderately successful.

Thus, to enhance our understanding of their mechanism, we systematically studied double potentials re-
corded in our canine crush-injury model of atrial flutter. In this model, a crush injury in the right atrium in the region of the pectinate muscles acts as a stable anatomic obstacle around which reentry occurs without a discrete area of slow conduction.

Methods

Seven mongrel dogs (weight, 20–30 kg) were studied under general anesthesia induced with 30 mg/kg intravenous pentobarbital sulfate and maintained throughout the study with additional doses of 1–2 mg/kg. The dogs were endotracheally intubated and ventilated with room air supplemented with oxygen to maintain arterial pH at ≈7.40, Po₂ at ≈100 mm Hg, and PCo₂ at ≈40 mm Hg. Arterial and venous cannulas were placed in the right or left femoral artery and vein by direct cutdown. The arterial blood pressure and surface ECG lead II were continuously recorded on a strip chart recorder (Clevite Brush Instruments, Inc., Cleveland, Ohio). A median sternotomy and right lateral thoracotomy were performed, and the heart was suspended in a pericardial sling. Two platinum-tip hook electrodes (Grass Instruments Co., Quincy, Mass.) were attached to the right and left atrial appendages for bipolar pacing. Pacing was performed with a programmable stimulator (Medtronic, Inc., Minneapolis, Minn.).

Mapping Technique

For atrial epicardial mapping, we used a computerized system (Bard Electrophysiology, Billerica, Mass.) with 56 channels for atrial epicardial recordings and eight channels for surface ECG leads I, II, III, aVR, aVL, aVF, V₁, and V₅. Surface ECG signals were filtered at 0.05–100 Hz. Atrial epicardial signals were filtered at 30–300 Hz and digitally sampled at a rate of 1,000 samples per second with a 12-bit accuracy. The signals were automatically gained by the computer system, depending on the maximal amplitude of the signals recorded, with typical signal amplitude ranging from 5 to 10 mV. As preliminary studies had shown that the reentrant circuit was confined to the right atrium around the crush injury in this model, a single right atrial electrode plaque containing 56 electrodes was constructed by sewing 56 bipolar wires through two 0.5-mm sheets of silastic rubber shaped to fit the right atrial posterior epicardial surface. The wires were knotted between each layer of rubber, and the two layers were glued together with silicone rubber cement. The mapping plaque was sutured to the posterior right atrial epicardial surface (Figure 1A). Intraelectrode bipolar spacing was 2 mm, and interelectrode spacing was approximately 5 mm vertically and 8 mm horizontally.

In each dog, atrial activation maps were obtained during sinus rhythm before and after crush injury, during right atrial pacing (cycle length, 200 msec) above and below the crush injury, during sustained atrial flutter, during pacing entrainment of atrial flutter, and during spontaneous termination of sustained atrial flutter induced by intravenous infusion of d-sotalol. The computer mapping system recorded multiple 8-second windows of rhythm that were briefly analyzed on-line to ensure adequate data quality, then stored to hard disk and subsequently to individual floppy disks for detailed poststudy analysis. Local activation times of epicardial electrograms were de-
fined as the point of maximum slope of the electrogram as it crossed the isoelectric line and were referenced in the surface ECG leads to the earliest onset of the P wave during sinus rhythm, to the earliest deflection from baseline of the flutter wave during atrial flutter, and to the pacing stimulus artifact during rapid atrial pacing and pacing entrainment. Activation times were determined automatically by the mapping system computer but were manually edited in the event of computer error. Surface ECG and atrial epicardial recordings were printed directly from the computer mapping system, but because of the limited graphics capability of the computer program, activation maps were redrawn for correct anatomic representation.

**Creation of Atrial Flutter Model and Pacing Induction and Entrainment Methods**

The atrial crush injury was made with a surgical clamp by lifting the anterior portion of the right atrial plaque after baseline sinus rhythm and atrial pacing maps were recorded. The crush injury was placed on the right atrial free wall, parallel to and approximately 1.5 cm above the atrioventricular groove, extending from the base of the right atrial appendage 1.5–2.5 cm posteriorly toward the intercaval zone (Figure 1C). The crush injury was typically 3–4 mm wide. The crush-injury location relative to electrode positions was noted, and the anterior portion of the electrode plaque was resutured in place. After crush injury, activation maps were recorded during sinus rhythm and atrial pacing.

After right atrial crush injury, attempts were made to induce sustained atrial flutter by programmed atrial stimulation introducing single (S,S1), double (S,S1S2), or triple (S,S1S2S3) premature beats to atrial refractoriness, after an eight-beat drive (S,S1) at 200-msec cycle length, or by rapid atrial pacing at a cycle length of 150, 120, or 100 msec for 10–20 beats. Sustained atrial flutter was defined as that lasting >10 minutes. Activation maps were obtained at the onset of atrial flutter and after 10 minutes of sustained atrial flutter. Attempts were then made to entrain atrial flutter at pacing rates 20–30 msec faster than the intrinsic atrial flutter cycle length from the right atrial appendage and at sites near or within the reentrant circuit around the crush injury. Pacing entrainment near or within the reentrant circuit was performed with a bipolar plunge electrode passed through the electrode plaque. During entrainment, activation maps were recorded so that the period of pacing and its cessation with resumption of atrial flutter was contained within the window. Criteria used to verify transient entrainment of atrial flutter included acceleration of flutter waves and local electrograms to the pacing length, constant fusion of flutter waves on the surface ECG during pacing, and at termination of pacing the resumption of tachycardia at the same cycle length and with the same flutter wave morphology as that noted before pacing.

**Termination of Atrial Flutter**

Activation maps were recorded in each dog at the moment of termination of atrial flutter induced by intravenous d-sotalol (2 mg/kg during 10 minutes) to determine the effect of arrhythmia termination on the double potential recordings. d-Sotalol was chosen for this study because of its high degree of efficacy in terminating atrial flutter in our model in previous studies.15

**Statistical Analysis**

The data are presented as mean±1 SD. Double potentials were defined as discrete activations separated by an isoelectric interval, with the earliest activation after the onset of the surface ECG P wave identified as x and the subsequent activation as y. The statistical significance of the differences in mean activation times for early x and late y components of the double potentials compared with activation times at adjacent electrodes was determined with Student's t test (two-tailed) for unpaired data.

**Results**

**Electrogram Characteristics Recorded During Sinus Rhythm Before and After Crush Injury**

Before crush injury, there was uniform spread of activation from the sinus node throughout the right atrium without evidence of slow conduction (Figure 2A, left panel). Electrograms recorded from the right atrium, particularly those overlying the area in which the crush injury would subsequently be placed, demonstrated no fragmentation and no double potentials (Figure 2A, right panel).

After crush injury, the activation wave front during sinus rhythm proceeded up to and then around the crush injury in opposite directions (Figure 2B, left panel). Electrogram recordings along the length of the crush injury demonstrated narrowly spaced double potentials in two dogs (Figure 2B, right panel) but no double potentials in the remaining five dogs.

**Electrogram Characteristics During Rapid Atrial Pacing Above and Below the Crush Injury**

During rapid atrial pacing (cycle length of 200 msec) via the bipolar plunge electrode placed above or below the crush injury, double potentials were recorded at locations along the length of the crush injury in five of seven dogs but not in the remaining two. During rapid pacing, the crush injury acted as an anatomic obstacle causing complete conduction block in six of seven dogs and marked conduction delay in one. The activation wave front from the plunge pacing electrode proceeded up to and then around the crush injury in opposite directions, producing a double potential recording at electrodes along the crush injury as a result of their sequential activation. Examples of the activation patterns and double potentials recorded during rapid pacing directly above and below the center of the crush injury are shown in Figures 3A and 3B from dog 7 using the high-density plaque. Note that the activation wave front spreads from the pacing electrode up to and then around both ends of the crush injury. As a result, double potentials are recorded along the length of the crush injury but not beyond the ends of the crush injury.

In contrast, pacing at the atrial appendage produced wave fronts nearly perpendicular to the crush injury, and in no case were double potentials recorded in the right atrium, particularly along the crush injury. An example of the activation pattern and electrograms
FIGURE 2. Effects of crush injury on atrial electrogram characteristics during sinus rhythm. Panel A, left: Activation map from dog 2 during sinus rhythm. Before crush injury, right atrial activation proceeds uniformly from the sinus node without conduction delay. Panel A, right: Electrogram recording from electrode F7, located at the center of the line where the crush injury will subsequently be placed, demonstrates no double potentials or fragmentation. Panel B, left: Crush injury produces line of complete conduction block around which activation wave fronts proceed in opposite directions, with collision below the crush injury. Panel B, right: After crush injury, electrode F7 records a double potential with narrow spacing. Activation time of x component is 20 msec, similar to the 19-msec activation time of adjacent electrode E6, just above the crush injury. Activation time of y component is 50 msec, similar to the 56-msec activation time of adjacent electrode G6, just below the crush injury. SVC, superior vena cava; IVC, inferior vena cava; RAA, right atrial appendage. Activation time lines represent 10-msec intervals. x and y, early and late components of the double potential. P, P wave; Q, QRS.

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Recorded along the length of the crush injury during pacing at the right atrial appendage are shown in Figure 3C from dog 7 using the high-density plaque. Note in this example that the activation isochrons are nearly perpendicular to the crush injury, although conduction below the crush injury is somewhat slower than that above the crush injury, and no double potentials are recorded.
A

Anterior

Above *

CRUSH INJURY

Below *

Posterior

B

Anterior

Above *

CRUSH INJURY

Below *

Posterior

C

Anterior

Above *

CRUSH INJURY

Below *

Posterior

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Patterns of Double Potentials Recorded During Sustained Atrial Flutter

Fourteen episodes of sustained atrial flutter (mean cycle length, 140±16 msec) were mapped and analyzed in the seven dogs studied (see Table 1). In each dog, the atrial flutter reentrant wave front proceeded around the crush injury in either a clockwise or a counterclockwise rotation (as viewed from anterior to the chest), which was associated with either an upright or inverted P wave morphology, respectively, in ECG leads II, III, and aVF. Activation maps and electrogram recordings from around the reentrant circuit in two episodes of sustained atrial flutter induced in dog 1 are shown in Figure 4.

In all seven dogs, during both morphologies of atrial flutter induced, double potentials were recorded only at electrodes along the line of the crush injury. In the 14 episodes of atrial flutter in which double potentials were recorded (Table 1), the activation time of the early x component correlated with the activation time at electrodes directly above the crush injury, whereas the activation time of the late y component correlated with the activation time at the electrodes directly below the crush injury. For example, the mean activation time of the x component was 25±13 msec, not statistically different from the 24±11-msec mean activation time at adjacent electrodes above the crush injury. Similarly, the mean activation time of the y component was 89±13 msec, not statistically different from the 91±14-msec mean activation time at adjacent electrodes below the crush injury.

Examples of double potentials recorded during two episodes of atrial flutter induced in dog 1 are shown in Figure 5. During an episode of inverted P wave flutter (Figure 5A, left and right panels), the activation time of the x component of the double potential at electrode F7 is 45 msec, which is similar to the 38-msec timing at adjacent electrode E8 above the crush injury. The activation time of the y component of the double potential at electrode F7 is 85 msec, which is similar to the 78-msec timing at adjacent electrode G6 below the crush injury. During an episode of upright P wave flutter (Figure 5B, left and right panels), the activation time of the x component of the double potential is 20 msec at electrode F7, correlating with the 22-msec

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<td>D5</td>
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Mean±SD
25±13/24±11 89±13/91±14

DPe, early component of double potential; DPI, late component of double potential; ADJ, electrode adjacent to the electrode recording the double potential above or below the crush injury. No statistically significant differences were noted between the activation times of the double potential and adjacent electrodes.

Electrodes selected were generally those closest to the center of the crush injury.

FIGURE 3. Facing page: Effects of crush injury on atrial electrogram characteristics during rapid pacing in dog 7. Panel A, left: High-density plaque activation map during rapid atrial pacing above the crush injury. Crush injury produces a line of complete conduction block around which paced wave front conducts in opposite directions. Panel A, right: Electrograms recorded from electrodes C4, D4, E4, and F4 record double potentials, representing sequential activations as the paced wave front proceeds up to and then around the crush injury. Electrodes A4, B4, and G4, where paced wave front was unidirectional, record only single potentials. Panel B, left: High-density plaque activation map during rapid atrial pacing below the crush injury. Again, paced wave front proceeds up to and then around the crush injury in opposite directions. Panel B, right: Electrodes B4, C4, D4, E4, and F4 record double potentials representing sequential activations, whereas electrodes A4 and G4 record only single potentials where the wave front was unidirectional. Panel C, left: High-density plaque activation map during rapid atrial pacing at the right atrial appendage. Pacing at atrial appendage produces an activation wave front essentially parallel to crush injury, although somewhat slower below crush injury. Panel C, right: Electrodes A4, B4, C4, D4, E4, F4, and G4 record no double potentials because wave front was unidirectional. S, pacing stimulus. Activation time lines represent 10-msec intervals.
FIGURE 4. Electrogram characteristics and activation patterns during sustained atrial flutter in dog 1. Panel A, left: Sequential electrogram recordings from the reentrant circuit and surface ECG lead aVF during sustained inverted P wave flutter; cycle length, 130 msec with a 2:1 ventricular response. Panel A, right: Atrial activation map demonstrating clockwise activation pattern (as viewed from anterior to the heart) during same episode of atrial flutter. Panel B, left: Sequential electrogram recordings from reentrant circuit and surface ECG lead aVF during sustained upright P wave flutter; cycle length, 130 msec with 2:1 ventricular response. Panel B, right: Atrial activation map demonstrating clockwise activation pattern (as viewed from anterior to the heart) during same episode of atrial flutter. SVC, superior vena cava; IVC, inferior vena cava; RAA, right atrial appendage. Dotted activation line separates earliest and latest activation times during atrial flutter. Solid vertical line represents reference mark at onset of P wave in surface ECG. Cross hairs along reference line represent computer-assigned local activation times of atrial electrograms. Activation time lines represent 10-msec intervals.

timing at adjacent electrode E₆ above the crush injury. The activation time of the y component at electrode F₇ is 105 msec, correlating with the 115-msec timing at adjacent electrode G₆ below the crush injury.

Relation of Double Potential Component Timing to Location Along the Line of the Crush Injury

In each dog, the activation timing of the x and y components of double potentials was correlated with
the location of recording electrodes along the line of the crush injury (Table 2). The xy interval was longest at electrodes located near the end of the crush injury adjacent to the area of earliest atrial activation during atrial flutter. In contrast, the xy intervals were more equally spaced at electrodes near the center of the crush injury and shortest at those near the end of the crush injury opposite the area of earliest atrial activation.

An example of double potentials recorded from electrodes along the length of the crush injury from dog 4 is shown in Figure 6. Note that the xy interval of the double potential is longest at electrode C\textsubscript{6}, located at the end of the crush injury near the earliest activation time during atrial flutter. In contrast, the xy interval becomes more equally spaced in time at electrode E\textsubscript{6} near the center of the line of the crush injury and shortest at electrode G\textsubscript{6} opposite the area of earliest atrial activation. As in previous examples, the activation times of each component of the double potentials correlated with activation times of adjacent electrodes above and below the crush injury.

**FIGURE 5.** Characteristics of double potentials recorded during atrial flutter in dog 1. Panel A, left: Electrogram from electrode F\textsubscript{7} along crush injury, during episode of inverted P wave atrial flutter, demonstrating typical double potentials, and adjacent electrodes E\textsubscript{6} and G\textsubscript{6} above and below the crush injury demonstrating single potentials. Panel A, right: Activation map of atrial flutter, demonstrating locations of electrodes F\textsubscript{7}, E\textsubscript{6}, and G\textsubscript{6}. Note that activation time of x component of double potential at electrode F\textsubscript{7} is 45 msec, correlating with 38-msec timing at adjacent electrode E\textsubscript{6} above the crush injury. Activation time of y component of double potential at electrode F\textsubscript{7} is 85 msec, correlating with 78-msec timing at adjacent electrode G\textsubscript{6} below the crush injury. Panel B, left: Electrogram from electrode F\textsubscript{7} along the crush injury, during episode of upright P wave atrial flutter, also demonstrating typical double potentials. Panel B, right: Activation map of atrial flutter, demonstrating locations of electrodes F\textsubscript{7}, E\textsubscript{6}, and G\textsubscript{6}. Note that activation time of the x component of the double potential at electrode F\textsubscript{7} is 20 msec, correlating with 22-msec timing at adjacent electrode E\textsubscript{6} above the crush injury. Activation time of y component at electrode F\textsubscript{7} is 105 msec, correlating with 115-msec timing at adjacent electrode G\textsubscript{6} below the crush injury. SVC, superior vena cava; IVC, inferior vena cava; RAA, right atrial appendage. Activation time lines represent 10-msec intervals.
TABLE 2. XY Intervals of All Double Potentials Recorded During Atrial Flutter Relative to Their Location Along the Crush Injury

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Inverted P wave atrial flutter

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<td>4</td>
<td>90 (E1)</td>
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<td>5</td>
<td>132 (D1)</td>
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<td>6</td>
<td>120 (D1)</td>
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<td>7</td>
<td>113 (B2)</td>
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<td>Mean±SD</td>
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All intervals are in milliseconds. Electrode location is approximate location of the recording electrode from the end of the crush injury closest to the area of earliest atrial activation during atrial flutter. ... Double potentials not recorded. Numbers in parentheses are electrode number recording double potential. Mean values at 0–5, 5–10, and 10–15 mm were statistically different from each other (p<0.05).

Effect of Pacing Entrainment of Atrial Flutter on Double Potential Recordings

The effect of pacing entrainment on double potential recordings was evaluated in five of seven dogs. During transient entrainment, double potentials recorded during atrial flutter continued to be recorded, but they were accelerated to the pacing cycle length. The relation of the activation time of the x and y components with respect to the timing of adjacent electrodes above and below the crush injury was also maintained. In three...
dogs, capture of both components of the double potential occurred orthodromically during entrainment without a change in the $xy$ interval, but in three dogs, capture of one component occurred antidromically with a resultant alteration in the $xy$ or $yx$ interval.

An example of orthodromic capture of double potentials during pacing entrainment of atrial flutter is shown in Figure 7 from dog 1. Note that the double potentials are accelerated to the pacing cycle length of 115 msec during transient entrainment of atrial flutter. The activation time of the $x$ component of the double potential from the pacing stimulus was 55 msec at electrode $F_7$, which is similar to the 48-msec activation time of adjacent electrode $E_6$ above the crush injury. The activation time of the $y$ component was 90 msec, which is similar to the 87-msec activation time of adjacent electrode $G_6$ below the crush injury. After cessation of pacing, the atrial flutter cycle length is 130 msec, and double potentials continue to be recorded but with different activation times relative to the onset of the $P$ wave. However, the relation between the activation times of the $x$ component (51 msec) and $y$ component (85 msec) and adjacent electrodes above (43 msec) and below (85 msec) the crush injury, respectively, is maintained.

An example of antidromic capture of double potentials during pacing entrainment of atrial flutter is shown in Figure 8 from dog 1. Note that the double potentials are accelerated to the pacing length of 115 msec during transient entrainment of atrial flutter. The activation time of the $x$ component of the double potential from the pacing stimulus was 37 msec at electrode $F_7$, which is similar to the 37-msec activation time of adjacent electrode $E_6$ above the crush injury. The activation time of the $y$ component was 15 msec, which is similar to the 15-msec activation time of adjacent electrode $G_6$ below the crush injury. After cessation of pacing, the atrial flutter cycle length is 135 msec, and double potentials continue to be recorded but with different activation times relative to the onset of the $P$ wave. However, the relation between the activation times of the $x$ component (13 msec) and $y$ component (106 msec) and adjacent electrodes above (10 msec) and below (120 msec) the crush injury, respectively, is maintained.

Response of Double Potentials During Spontaneous Termination of Atrial Flutter Induced by Intravenous Infusion of D-Sotalol

Termination of atrial flutter was observed during infusion of D-sotalol in each of the seven dogs studied as a result of development of a line of conduction block contiguous with and between the crush injury and the tricuspid valve annulus. Conduction block developed abruptly without cycle length oscillation in one dog, after a single premature eccentric activation originating elsewhere in the reentrant circuit in three dogs, and after transient atrial fibrillation in three dogs. In the four dogs in which termination of atrial flutter occurred abruptly, the $x$ component of the double potential was recorded, but the $y$ component was not. An example of a double potential recording from dog 1 during the last three beats of atrial flutter before its termination is shown in Figure 9A. Note that during the last beat of atrial flutter, the $x$ component of the double potential is recorded but not the $y$ component because of abrupt conduction block between electrodes $G_1$ and $G_2$ below the posterior end of the crush injury (Figures 9B–9D).

Discussion

The electrophysiological mechanism of double potentials recorded during atrial flutter has been evaluated in our canine right atrial crush-injury model. The study demonstrated that double potentials are recorded near the center of the reentrant circuit, along a line of conduction block created by the crush injury. Furthermore, double potentials appear to represent sequential activations from the reentrant wave front as it proceeds around the crush injury on either side of the recording electrodes.

Effect of Atrial Crush Injury on Right Atrial Electrogram Characteristics During Sinus Rhythm

Activation maps during sinus rhythm before crush injury revealed no evidence of conduction delay in the right atrium, and double potentials were not recorded as a result. The crush injury produced a line of marked conduction delay or complete conduction block in the right atrium around which the activation wave front proceeded in opposite directions. This resulted in the recording of narrowly spaced double potentials in two of seven dogs but not in the other five.

In previous studies in humans, it has also been noted that double potentials recorded during atrial flutter are usually not recorded at the same electrodes during sinus rhythm. A possible explanation for this observation is that the line of block responsible for reentry and double potentials during atrial flutter in humans is functional rather than anatomic and thus is not present during sinus rhythm. It is unknown, however, whether atrial flutter in humans is a result of reentry around a functional obstacle or a fixed anatomic obstacle, although its electrocardiographically consistent nature suggests that a fixed obstacle may play a role. This study has shown that even a line of fixed anatomic block may not result in the recording of double potentials during sinus rhythm. However, depending on the orientation of the crush injury to the sinus rhythm activation wave front, narrowly spaced double potentials or splitting of electrograms may be seen in some cases as the wave front proceeding in opposite directions around the crush injury sequentially activates electrodes along the crush injury.

Electrogram Characteristics During Rapid Atrial Pacing Above and Below the Crush Injury

Rapid pacing directly above and below the crush injury increased the likelihood of recording double potentials in five of seven dogs compared with sinus rhythm. This is probably a result of placement of the plunge pacing electrode near the center of the line of the crush injury, producing an initial wave front during pacing that was perpendicular to the crush injury, followed by a second wave front moving around the crush injury in opposite directions, resulting in the sequential activation of the electrode recording double potentials. The lack of double potentials during pacing above and below the crush injury in two of seven dogs may have resulted from placement of the plunge pacing electrode more toward the posterior end of the crush injury, resulting in initial and secondary wave fronts
FIGURE 7. Effects of transient entrainment on double potentials recorded during atrial flutter in dog 1 (orthodromic capture). Panel A, left: Double potentials from electrode F7, and panel A, right: an activation map during pacing entrainment of atrial flutter showing locations of electrodes F7, E6, and G6. During pacing, double potentials are captured orthodromically and accelerated to pacing cycle length of 115 msec. Activation time of x component of double potential at electrode F7 was 55 msec, correlating with 48-msec activation time at adjacent electrode E6 above the crush injury. Activation time of y component was 90 msec, correlating with 87-msec activation time at adjacent electrode G6 below the crush injury. Panel B, left: Atrial flutter cycle length is 130 msec after cessation of pacing, and double potentials continue to be recorded. Panel B, right: Relation between activation times of x component of 51 msec and y component of 85 msec at electrode F7 and adjacent electrodes E6 of 43 msec and G6 of 85 msec, respectively, is maintained. SVC, superior vena cava; IVC, inferior vena cava; RAA, right atrial appendage. Activation time lines represent 10-msec intervals.

being more parallel to the crush injury. This is clearly evident from data during rapid pacing at the right atrial appendage, where the paced wave fronts above and below the crush injury were essentially parallel and no double potentials were recorded in any dog.

These observations suggest that the orientation of the paced activation wave front to the anatomic obstacle associated with reentry is important to the recording of double potentials. This could also explain observations in previous studies in humans, in which double poten-
Double potentials recorded during atrial flutter were the result of sequential activations along a line of conduction block produced by the crush injury as the reentrant wave front passes on either side of electrodes at the center of the reentrant circuit. This conclusion is based on the facts that the electrodes recording double potentials were always located along the line of the crush injury, the activation times of the x and y components of the double potential were similar to the activation times of adjacent electrodes above and below the crush injury, and the positions of electrodes recording double potentials along the line of the crush injury influenced their timing.

The observations made during transient entrainment and termination of atrial flutter further support the conclusion that double potentials in this model are
FIGURE 9. Effects of abrupt termination of atrial flutter on double potentials in dog 1. Panel A: Double potential recording at electrode F7 during last three beats of an episode of upright P wave flutter and its termination. Note that during last beat of atrial flutter, x component is recorded but not y component. Panel B: Sequential electrograms from around the reentrant circuit demonstrate that termination of atrial flutter was caused by abrupt conduction block between electrodes G2 and G6 below posterior end of crush injury, resulting in recording of x component but not y component of double potential. Panel C: Atrial activation map of last beat of atrial flutter before termination. Note bunching of isochrons below posterior end of crush injury, suggesting some drug-induced slowing of conduction. Panel D: Atrial activation map of abrupt termination of atrial flutter reveals location of conduction block below posterior end of the crush injury. I, site of conduction block. SVC, superior vena cava; IVC, inferior vena cava; RAA, right atrial appendage. Activation time lines represent 10-msec intervals.

result of sequential activations from the reentrant circuit. During transient entrainment, double potentials were accelerated to the pacing cycle length. In addition, the relation between the activation times of the x and y components of the double potentials and the activation times of electrograms from adjacent electrodes above and below the crush injury was maintained during transient entrainment. This consistent relation probably would not be maintained if the double potentials were not directly linked to the reentrant circuit. Termination of atrial flutter by abrupt conduction block, usually below the posterior end of the crush injury, resulted in recording of the early x component of the double potential but not the y component, which also supports
the conclusion that each component represents sequential activations from the reentrant wave front on opposite sides of the crush injury. Overall, these observations are consistent with those made by other investigators both in humans and in other canine models of atrial flutter in which double potentials are recorded.\textsuperscript{1–6}

**Possible Clinical Implications of the Study**

Our data are compatible with those of Olshansky et al.,\textsuperscript{1} who proposed that double potentials recorded during atrial flutter in humans may represent sequential activation of electrodes near the center of the reentrant circuit. Our data do not exclude the possibility that double potentials may also be recorded at sites other than the center of the reentrant circuit—for example, at sites of collision of wave fronts spreading away from the reentrant circuit or in areas of slow conduction within the reentrant circuit. At such sites, however, intermittent block between double potentials may be seen, distinguishing them from those recorded at the center of the reentrant circuit.\textsuperscript{1} Therefore, recording double potentials with electrophysiological characteristics similar to those noted in our study may help identify the center of the reentrant circuit,\textsuperscript{1,6} which may in turn help localize critical sites to target for interventions such as catheter ablation. In our model, for example, the region between the line of block where double potentials are recorded and the tricuspid valve annulus may be critically important to maintain reentry and might therefore be an appropriate target area for ablation.\textsuperscript{17} This hypothesis will require further study.

**Limitations of the Study**

A limitation of this study is that electrograms and activation patterns in the left atrium, Bachmann’s bundle, and interatrial septum were not evaluated in this group of seven dogs. A single-electrode plaque was used in this study to maintain a relatively high electrode density over the right atrium, because in our previous studies,\textsuperscript{12–14} reentry in this model appeared to be confined to the right atrial free wall. However, these earlier studies using three electrode plaques over the right atrium, left atrium, and Bachmann’s bundle did not record double potentials outside the right atrium either.

**Summary**

This study has demonstrated in our canine right atrial crush-injury model of atrial flutter that double potentials 1) are recorded from the center of the reentrant circuit at electrodes located along a line of anatomic block produced by the crush injury and 2) represent sequential activations at the recording electrode as the reentrant wave front passes on either side of the crush injury.

**Acknowledgment**

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