SUMMARY  In 12 anesthetized open-chest dogs, ventricular epicardial activation maps were constructed and electrograms were recorded from the bundle of His, left bundle branch, and subendocardial Purkinje fibers 24 hours following Harris 2-stage ligation of the left anterior descending coronary artery. All animals developed ectopic ventricular depolarizations and/or ventricular tachycardia. The earliest area of epicardial activation was located along the border of the infarct in the left ventricle in all animals. Bipolar recording from various levels of the conduction system and ventricular myocardium revealed that the earliest recorded electrical activity originated in subendocardial Purkinje fibers which had survived the acute myocardial infarction. The origin of these arrhythmias was further studied by pacing through the electrode which had recorded the early Purkinje activity and comparing the surface ECG and activation sequence with that of the spontaneous rhythm. These data tend to support the hypothesis that ventricular arrhythmias occurring 24-72 hours following acute myocardial infarction have their origin in the subendocardial Purkinje network which has survived the infarction.

ECTOPIC VENTRICULAR DEPOLARIZATIONS occur in greater than 80% of patients during acute myocardial infarction.1,2 The majority of these arrhythmias occur during the first few days of the acute episode.3-4 Harris has described a canine model of acute anterior myocardial infarction which is particularly suitable for studying these arrhythmias.5 Four to eight hours following 2-stage ligation of the left anterior descending coronary artery, ectopic ventricular depolarizations appear, peak in frequency between 15 and 30 hours, and persist with decreasing frequency for approximately 72 hours. Electrophysiological studies performed on isolated superfused infarcted myocardium 24 hours following coronary occlusion have revealed survival, albeit in an abnormal state, of subendocardial Purkinje cells, in contrast to almost total absence of viable ventricular muscle cells.6-9 These Purkinje cells are characterized by diminished maximum diastolic potential, decreased action potential amplitude, diminished rate of depolarization (dV/dt), prolonged action potential duration, and enhanced diastolic (phase 4) depolarization. Spontaneous arrhythmias commonly arise in this setting.5-9 In addition, in vivo recordings from Purkinje fibers within the infarct have revealed Purkinje fiber activity preceding the onset of the QRS complex of the surface electrocardiogram during many ectopic ventricular complexes.6-10 These observations have led to the suggestion that the ventricular arrhythmias arising four to eight hours following acute myocardial infarction originate in the subendocardial Purkinje network underlying the infarction and are due to enhanced automaticity in these cells. Scherlag et al.10 have in addition suggested that some of these ectopic depolarizations are initiated in infarcted epicardial muscle. The present study, employing epicardial mapping techniques and bipolar electrode recordings from Purkinje fibers and the left bundle branch, verifies the origin of the experimentally induced accelerated idioventricular rhythm in the Purkinje network surviving the acute myocardial infarction.

Methods

Twelve healthy mongrel dogs weighing 10-15 kg were employed in these experiments. They were anesthetized with intravenous sodium pentobarbital (30 mg/kg) and ventilated with room air through a pharyngotracheal tube by a Harvard pump. The chest was opened through a small left thoracotomy in the fourth interspace and the pericardium was opened. The left anterior descending coronary artery was occluded by the Harris 2-stage procedure 3-7 mm from the border of the left atrial appendage. The chest was closed and the animal was allowed to recover. Preoperative and 4-6 hour postoperative electrocardiograms were obtained.

Twenty-four hours following the initial procedure the animals were reanesthetized with morphine (2.5 mg/kg) and sodium pentobarbital (10 mg/kg) and ventilated with room air through a tracheostomy by a positive pressure ventilator at a minute volume determined from a body weight nomogram. Body temperature was maintained at 101-102° F by a thermal mattress. The chest was opened in the fifth interspace and the heart was suspended in a pericardial sling. Standard electrocardiographic leads were monitored. A bipolar catheter electrode was inserted via the right femoral or internal jugular vein and placed in the right atrium to record the His bundle electrogram (HBE).

Because of the constraints of the mapping procedure, it was necessary to establish a unifocal ventricular tachycardia (UVT) or stable pattern of unifocal ectopic ventricular depolarizations in the animals in this study. If stable UVT was not initially present a stable pattern of unifocal ectopic depolarizations was established by pacing the heart through an atrial electrode at the sinus rate and crushing the sinus node. A sequence of 3-10 supraventricular beats followed by a pause was established with a digitally pro-
grammed stimulator.* The escape ectopic ventricular beat was used for mapping. Fusion beats were commonly observed. Measurements for mapping were made only from beats which were clearly ventricular in origin.

Mapping Technique

A bipolar plunge electrode (teflon-coated stainless steel: 0.005 inch diameter) was inserted into the noninfarcted left ventricular myocardium well within the distribution of the left circumflex coronary artery via a 23 gauge needle as the reference electrode. The epicardial surface of the ventricles was mapped with a hand-held bipolar roving electrode with terminals located 2 mm apart. The electrograms were displayed on a Tektronics 565 storage oscilloscope and the interval was measured by a custom designed interval counter.† The interval between the reference and roving electrograms was measured at the intrinsincoid deflections (midpoint of the most rapid transition of the electrogram) of each electrogram. The reproducibility of the measurements at each location was within ±2 msec. Forty-five to 60 predetermined locations on the left ventricle and 10 to 20 sites on the right ventricle were recorded to construct the ventricular epicardial activation maps. Isochronic maps were constructed from these data.

Localization of the Origin of Ectopic Ventricular Activity

Left bundle branch (LBB) depolarization was recorded through a Scher-type 10-lead electrode†† plunged into the base of the left ventricle, 1–2 mm caudal of the left circumflex coronary artery and into the ventricular septum across the plane of the mitral valve. In most experiments a close bipolar plunge electrode was placed in the area of the A-V junction to record the His bundle electrogram;‡‡ in the remainder a His bundle recording was obtained via a catheter electrode. Left bundle branch activity occurred following the His deflection of the His bundle electrogram. Purkinje fiber (PF) depolarization within, on the border of, and outside the area of myocardial infarction was recorded using either the Scher-type or routine plunge electrode. Ten to 15 plunge electrode recordings were obtained in the early activated area to locate and map the earliest Purkinje activation. The interval between the earliest recorded Purkinje fiber and the earliest site of epicardial activation was determined by subtracting the interval between these locations and the stable reference electrogram. The site of earliest ectopic activity was verified by electrically pacing the subendocardium through the electrode used to record the earliest Purkinje fiber‡‡ depolarization. It was then determined whether the resulting paced ventricular rhythm was comparable to the endogenous rhythm in morphology and activation sequence. All data were stored on magnetic tape with a Honeywell 5600 C tape recorder at 7/2 inches per second.

At the termination of physiologic studies, the heart with electrodes in place was removed and the left ventricle opened to confirm the location of the electrodes. The Purkinje system and bundle branches were stained with 2% tincture of iodine to confirm the location of the electrode in the left bundle branch. Routine microscopic studies were performed to confirm myocardial necrosis in the area electrically defined as myocardial infarction.

Results

All animals developed anteroseptal myocardial infarction by electrocardiographic and histologic criteria. The area of infarction was pale tan and noncontracting or bulging paradoxically. Only broad amplitude potentials (usually associated with distant depolarization or cavity potential) could be recorded from this epicardial, ventricular and intramyocardial muscle as compared to rapid, large amplitude electrograms recorded from normal areas. This area of electrically defined dead tissue corresponded with the area of histologically identified necrosis.

Prior to the anesthesia for the in situ electrophysiologic studies, the electrocardiogram of each animal revealed spontaneous ventricular tachycardia competing with sinus beats. Seven animals developed stable unifocal tachycardia and five manifested multifocal tachycardia. In the latter animals, programmed pacing established a stable pattern of escape depolarizations. Our studies revealed no differences between these two groups of animals. The spontaneous ventricular rate in infarcted dogs without autonomic stimulation was 196 ± 19.5 beats/min. In a previous study, they a control group of dogs following bundle of His ablation by cautery developed a spontaneous ventricular rate of 49.2 ± 8.4 beats/min also without autonomic stimulation. In each case, the ventricular tachycardia could be overdriven by supraventricular or ventricular pacing. In figure 1, the sequence of epicardial activation and the electrocardiogram of one dog which developed ectopic ventricular beats are demonstrated. The electrocardiogram shows ectopic ventricular depolarizations manifesting predominantly a QS pattern. Epicardial activation began inferiorly (solid black area) near the apex along the left border of the infarct (diagonal broken lines) and spread concentrically to excite the entire left ventricle in 51 msec. The earliest site of activation on the right ventricle occurred in the posterior right ventricle 32 msec after the onset of left ventricular activation. Figure 2 demonstrates the epicardial activation sequence in another dog 24 hours after acute myocardial infarction. The electrocardiogram reveals a unifocal ventricular tachycardia at a rate of 182 beats/min. The QRS morphology is a monophasic R wave and the earliest site of left ventricular epicardial activation (solid black area) is located anterior and superiorly along the border of the infarct; this contrasts with figure 1, in which the electrocardiographic QS pattern was associated with a more inferior and posterior site of origin. Also in contrast to the late right ventricular activation in figure 1, the earliest site of right ventricular activation in figure 2 was only 11 msec following the onset of left ventricular epicardial activation. The duration of left ventricular epicardial activation in figure 2 was 72 msec (termination of LV activation not shown).

In eight of the 12 animals, ventricular activation maps were similar to that shown in figure 1, that is, a posterior and inferior early site associated with the QS pattern on the ECG. Two dogs developed a monophasic R wave ventricular tachycardia similar to that shown in figure 2, and the remaining two dogs each demonstrated two different elec-

---

† Bloom Associates.
trocardiographic patterns of ventricular tachycardia. In each animal, the two different patterns were associated with different sites of earliest ventricular activation. Figure 3 demonstrates the activation sequences of one dog during two different episodes of spontaneous ventricular tachycardia. The electrocardiogram in figure 3A reveals a tachycardia with a QS morphology at a rate of 204 beats/min and the corresponding map shows the inferoposterior epicardial breakthrough. In figure 3B, a tachycardia at a rate of 195 beats/min with the monophasic R wave morphology is associated with the anterior, proximal epicardial site. The electrocardiographic patterns alternated between these two patterns over a two-hour period; epicardial maps were obtained twice for each pattern and the same early site was identified both times for each pattern.

The site of earliest epicardial activation in 14 of 14 ectopic ventricular rhythms investigated in 12 dogs was always located along the border of the infarct and initiation of activation of the left ventricle always occurred earlier than activation of the right ventricle. In those dogs in which the origin was located inferoposteriorly, right ventricular activation was delayed 20–30 msec, whereas in those with the anterior site, the activation of the right ventricle began 10 to 20 msec following the onset of left ventricular depolarization. The mechanism by which right ventricular activation occurred was investigated by comparing the relationship of the earliest recorded right ventricular potentials to the His depolarization. In those animals in which the earliest activation was recorded in the anterosuperior LV, the RV activation preceded the His potential by 2–12 (avg 10) msec. Conversely, in the other animals in which earliest activation was recorded posteroinferiorly, the RV activation followed the His depolarization by 28–40 (avg 32) msec. Right ventricular activation never preceded left bundle branch depolarization.

Following determination of the site of earliest epicardial activation, bipolar recordings from the left bundle branch at its bifurcation and the distal subendocardium of the left ventricle were made. Subendocardial recordings were obtained within the infarct and along the border zone, especially in the area of earliest epicardial activation. A representative recording from these sites during a sinus beat displayed at a rapid speed is shown in figure 4. The sequence of activation during sinus rhythm is shown in recordings from the bundle of His (HBE), left bundle branch (LBE), anterior wall subendocardial Purkinje fibers (PF, and PFa) and left ventricular free wall intramural electrode (REF). During sinus rhythm or supraventricular override pacing, the LBB potential followed the His bundle potential and preceded the onset of the surface electrocardiogram as well as left ventricular Purkinje fiber activity. The Purkinje fiber labelled PF, was located 1 cm within the infarcted zone whereas the one labelled PFb was in the border zone.

The activation sequence during a spontaneous ectopic
ventricular depolarization is shown in figure 5. The analog data are displayed in the same order as in the previous figure. The Purkinje fiber in the subendocardial layer underlying the infarct (PF) was the earliest recording site to depolarize; subendocardial Purkinje activity preceded activation of the proximal conduction system, left bundle branch (LBE) and bundle of His (HBE) and ventricular muscle depolarization (ECG and REF). Note that the infarct zone Purkinje fiber activation preceded depolarization of the Purkinje fiber in the peri-infarction zone at the site of earliest epicardial activity identified previously by epicardial mapping. The His bundle was activated in a retrograde fashion. Depolarization of the Purkinje cell in the interior of the infarct preceded earliest viable ventricular mus-

**Figure 4** Depolarization of ventricular specialized conduction system (VSCS) and electrocardiogram during normal sinus rhythm. The first record is the His bundle electrogram (HBE) recorded by a catheter electrode. In the HBE, a indicates atrial septal activation, h indicates the His bundle depolarization and s indicates ventricular septal activation. The second record is the left bundle electrogram (LBE) recorded with a multiple-lead electrode in the left bundle branch. The ventricular and atrial electrograms in the HBE and LBE were electronically clipped. Note that left bundle activation (b) follows h. The third and fourth electrograms were recorded from the left ventricular subendocardium and show Purkinje fiber depolarization within the area of the infarct (PF) and in the border (peri-infarction) zone (PFb). These follow b. The fifth record recorded in noninfarcted myocardium is the reference electrogram (REF) used in the mapping protocol of each experiment. The last record is the lead II electrocardiogram (ECG). The timing signal indicates 100 msec intervals.

**Figure 5** Depolarization of the ventricular specialized conduction system (VSCS) and electrocardiogram during a ventricular ectopic depolarization. The records from top to bottom are His bundle electrogram recorded through a catheter electrode (HBE), left bundle electrogram (LBE), infarct zone Purkinje fiber (PF), border zone Purkinje fiber (PFb), reference electrogram (REF), and lead II electrocardiogram (ECG). The timing signal (T) indicates 100 msec intervals. Labeling is identical to figure 4. Note that activation of PF, precedes both b and h which are activated in the retrograde direction and PFb, REF and ECG representing the distal VSCS and ventricular myocardium which are activated in the antegrade direction.
cle activation by 10 msec. The data displayed in figure 6A were recorded during unifocal ventricular tachycardia and the electrograms represent a single complex at rapid sweep. Again note that the earliest left ventricular activity is the Purkinje fiber in the subendocardium of the infarct (PF,) shown in the third record. The His bundle and left bundle branch shown in the first and second records are activated in a retrograde sequence. Depolarization of the Purkinje fiber in the peri-infarction zone at the site of earliest epicardial activation (fourth record) and the reference electrogram (fifth record) are activated later than the Purkinje fiber in the infarct zone (PF,).

In each animal studied, the earliest recorded ventricular activity in either an ectopic ventricular depolarization or ventricular tachycardia originated in a left ventricular subendocardial Purkinje fiber in the area of the acute myocardial infarction. This depolarization always preceded the activity in the left bundle branch and the earliest activity in the viable myocardium bordering the infarct.

To further document that these early Purkinje fibers were the site of origin of the ventricular arrhythmias, the ventricles were paced through the electrode which had recorded the earliest Purkinje fiber within the infarct. Figure 6 demonstrates the data recorded during one such experiment. The electrocardiogram in 6A was recorded during spontaneous ventricular tachycardia. In figure 6B the ventricles were paced from the early Purkinje fiber site in the subendocardial layer of the infarct zone. The first and second records show the His bundle electrogram (HBE) and left bundle electrogram (LBE), respectively. The third record is deleted because the electrode was being used for pacing. The reference intramyocardial electrogram and electrocardiogram are in the fifth and sixth records. Note that the configuration of the QRS complex and the sequence of activation are almost identical to that occurring spontaneously. In order to exclude the possibility that pacing from any subendocardial site with personal infarct might result in a similar electrocardiographic pattern, the ventricles were paced from several sites within the infarct. Figure 7 demonstrates the alteration in QRS configuration that resulted when stimulation was applied at various subendocardial sites within the infarct. The electrocardiogram displayed below the heart was recorded during spontaneous ventricular tachycardia. The earliest recorded Purkinje fiber during this rhythm was located at site 3. The electrocardiograms at the right were obtained during pacing from each of the three sites to which the tracing are connected by the solid lines. Note that pacing through the electrode at site 3 resulted in a ventricular complex which appears almost identical to the spontaneous one; however, when stimulation was applied to the cephalad portion of the infarcted zone (clear area) the QRS morphology was markedly different. A stimulation site between these sites produced an intermediate biphasic complex. Movement of 10 to 15 mm of the pacing electrode in these infarcted hearts produced readily perceptible alterations in the surface QRS morphology.

Discussion

Ectopic ventricular arrhythmias are common 12 to 72 hours after Harris 2-stage ligation of the left anterior descending artery in the dog (phase 3 of Harris). On the

![Figure 6](http://circ.ahajournals.org/content/53/1/60.full)

**Figure 6** Depolarization of the ventricular specialized conduction system (VSCS) and electrocardiogram during (A) spontaneous ventricular tachycardia and (B) electrically stimulated ventricular tachycardia. The records are bipolar recordings from the His bundle (HBE), left bundle branch (LBE), border zone Purkinje fiber (PFb), noninfarcted myocardium (REF) and lead II electrocardiogram (ECG). The third record in A is the infarct zone Purkinje fiber (PF,); this record is deleted in B because the pacing stimulus was passed through the electrode used to record it. The time signal (T) indicates 100 msec intervals. A) Note that during the spontaneous VT, PF, precedes activation of the proximal VSCS as well as the distal VSCS and ventricular muscle. B) The left ventricle was paced through the electrode used to record PF, in figure 6A. Note that the sequence and timing of activation remains unchanged from the spontaneous arrhythmia.
basis of epicardial and intramural recordings and histologic studies Harris suggested that this delayed ectopic activity arose in the peri-infarction or boundary zone in which he included: 1) the circumferential tissue surrounding the infarct extending from endocardium to epicardium, 2) a layer between the necrotic muscle and the epicardial surface, and 3) a thin layer of subendocardial tissue. Recent evidence has suggested that these arrhythmias arise more specifically from the Purkinje fibers underlying the myocardial infarction. Friedman et al. and Scherlag and co-workers using microelectrode techniques have demonstrated the markedly abnormal electrophysiological and potentially arrhythmogenic characteristics of cells from the infarct zone studied immediately following rapid excision from the in situ heart. Furthermore, in in vivo studies, these investigators have recorded depolarization of Purkinje fibers in the infarcted area prior to the onset of the QRS complex during ectopic beats. However, in these studies earlier activation of Purkinje fibers in the peri-infarction zone or more proximal bundle branch system were not excluded. In addition, use of the surface electrocardiogram may not accurately assess the onset of ventricular activation.

Our experiments confirm that ectopic ventricular arrhythmias arising 24 to 48 hours after experimental myocardial infarction originate in subendocardial Purkinje fibers surviving in the area of ischemia. The Purkinje fiber depolarization recorded from the subendocardium of the infarct zone precedes the activation of the left bundle branch and the His bundle thus excluding supraventricular or proximal bundle branch sites as the origin of these arrhythmias. Similarly the infarct zone Purkinje fibers activation precedes depolarization of Purkinje fibers located in the border zone as well as the viable ventricular muscle. This finding excludes the latter as the site of origin of the arrhythmias. Thus the earliest activation occurs within the Purkinje network underlying the acute infarct. The earliest electrogram recorded from the left ventricular Purkinje network in these experiments was not necessarily recorded from the Purkinje fiber in which the arrhythmia originated; actually it may merely have been close to it. Nevertheless, since the Purkinje fiber within the infarct which was recorded was depolarized prior to either the more proximal or distal ventricular specialized conduction system (VSCS) or the ventricular muscle, the tachyarrhythmia must have originated from some fiber within this portion of the Purkinje system underlying the infarct.

It appears that the depolarization wave usually exits into the left ventricle before activating the right ventricle despite the fact that the infarct borders the right ventricle along the length of the interventricular septum and free edge of the right ventricle. Delayed activation of the right ventricle probably occurs because no Purkinje fibers cross the septum connecting the left and right ventricular Purkinje networks. Right ventricular activation would thus have to occur 1) by retrograde conduction over the left bundle branch to the bifurcation of the His bundle followed by antegrade conduction down the right bundle branch or 2) via transseptal muscle conduction. Our data suggest that transseptal conduction activates the right ventricle when the earliest site of LV depolarization is located anteriorly near the summit of the interventricular septum and bifurcation of the A-V conduction system. On the other hand, when LV activation begins inferoposteriorly, right ventricular activation is delayed and follows the His, suggesting that the RV is activated via the ventricular specialized conduction system; however, RV activation by both routes is not excluded by our data. In a similar situation involving excitation initiated in the left ventricle, Van Dam and Jansel have shown that slow transseptal conduction causing delayed right ventricular activation occurs during complete right bundle branch block in dog and man. In addition, there is minimal participation of the His-Purkinje system in interventricular activation of the contralateral ventricle in this situation.

Scherlag and colleagues have reported epicardial depolarization in the infarcted tissue which preceded Purkinje activation and thus have suggested that some of the arrhythmias may originate in infarcted myocardium. We were unable to record epicardial, intramyocardial, or subendocardial ventricular muscle activation of the type associated with local electrical activity (i.e., rapid deflection, short duration complexes) within the borders of the infarcted tissue nor were we able to record myocardial or epicardial activity in the surrounding peri-infarction zone which preceded the Purkinje activity within the infarct. Our data in this regard are in substantial agreement with those of Friedman et al. who were unable to document intramyocardial electrical activity or early epicardial activation.

Several lines of evidence suggest that surviving Purkinje fibers in the infarct zone participate in the initiation of ventricular arrhythmias in man following acute myocardial infarction. Although Purkinje fiber activity in the area of infarction has not been recorded in human hearts, several authors have reported histologic and electronmicrographic evidence suggesting survival of Purkinje fibers after human myocardial infarction. Furthermore, evidence from epicardial activation studies in man and experimental animals after myocardial infarction suggest that these Purkinje fibers

---

**Figure 7** The effect of varying stimulation sites within the infarct zone on the QRS morphology of the ventricular rhythm. The posterior view of the left ventricle indicates the viable myocardium (stippled area), infarcted area (clear area) and the three sites at which stimulation was applied to the subendocardial surface of the left ventricle (numbered solid circles). The ECG shown below the heart was recorded during spontaneous VT. The tracings to the right were recorded during pacing from the sites each is attached to by a solid line. During the spontaneous VT, the earliest site of epicardial activation (solid black area) was located near the site of the earliest occurring PF, (site 3). Note that pacing from this site produces a ventricular rhythm almost identical to that occurring spontaneously.
survive functionally as well. An arrhythmia similar in many respects to that studied in this report occurs commonly in the first few days following acute myocardial infarction in man. Accelerated idioventricular rhythm associated with myocardial infarction (idioventricular tachycardia) is a nonparasystolic ectopic ventricular rhythm with a rate similar to the sinus rate (60-110/min) which emerges with sinus slowing and rarely is associated with ventricular fibrillation. Although the arrhythmia studied in this report had a much faster rate (160-240/min), the sinus rate of the dog is also considerably faster than in man. In fact the sinus and idioventricular rates in each are very similar. In the canine model as in man, the arrhythmia can be exposed by sinus slowing. Fusion beats are common in the human arrhythmia as well as in the canine model. Finally like the human arrhythmia the ectopic ventricular rhythm in the dog usually ceases 72 to 96 hours following infarction. Although the dog under certain circumstances the ventricular arrhythmias stabilize into a unifocal pattern, the pattern is usually multifocal unlike its human counterpart.

The ventricular origin of accelerated idioventricular rhythm in man has been verified by Gallagher et al. by His bundle recordings in patients with digitalis toxicity. The arrhythmia caused by digitalis intoxication, identical to that associated with acute myocardial infarction, has been shown by Kastor and co-workers to originate in the ventricular specialized conduction system in the left ventricle. Thus it appears that accelerated idioventricular rhythm, associated with either digitalis toxicity or acute myocardial infarction, in the experimental canine model and probably in man, originate in the distal specialized conduction system of the left ventricle.

Although the present study did not address itself directly to the electrophysiologic mechanism responsible for these arrhythmias, our data suggest that they are due to increased automaticity; however, a re-entrant mechanism cannot be excluded. As emphasized by Scherlag et al. and observed in our studies these arrhythmias can be exposed by sinus slowing (mechanical disruption of sinus node, vagally induced sinus arrest, etc.) and abolished by overdrive pacing (supraventricular or ventricular). In addition, we observed a warm-up phase in the ventricular tachycardia following overdrive suppression. These arrhythmias are readily suppressed by propranolol as well as lidocaine as would be predicted for an automatic rhythm in contradistinction to a re-entrant arrhythmia.

Similarly, Wellens et al. employing electrical stimulation of the heart to assess the mechanism of sustained ventricular tachycardia in man, studied 3 to 20 hours following the onset of acute myocardial infarction, concluded that this arrhythmia was due to increased automaticity rather than re-entry. During the first 24 hours following the acute infarction (excluding the first 120 minutes), these episodes of ventricular tachycardia remained stable and did not degenerate into ventricular fibrillation; they appeared to be non-parasystolic and could not be terminated by successive premature stimuli. Although these data do not conclusively prove enhanced automaticity they nevertheless do support it as the mechanism of this arrhythmia. Certainly the electrophysiologic abnormalities previously described in Purkinje fibers following infarction satisfy the conditions required for re-entry and this re-entry may participate in other arrhythmias in the setting of acute myocardial infarction.

Two electrocardiographic QRS morphologies were observed in these canine experiments on ventricular arrhythmias found in acute myocardial infarction; each morphology was associated with a different region of early epicardial activation. One would expect under relatively similar conditions in man that specific electrocardiographic patterns would be associated with characteristic sites of earliest epicardial activation. The QRS morphologies in man would undoubtedly be different than those observed in these canine experiments due to the differing heart and body geometry found in the dog. Furthermore, these studies were performed in open chest dogs in the right lateral decubitus position.

Although this study investigated the site of origin of ectopic ventricular arrhythmias during acute myocardial infarction and the subsequent epicardial activation sequence, our results have implications in the investigation of ventricular arrhythmias associated with chronic ischemic states (i.e., ventricular aneurysm, chronic or healed myocardial infarction). The area of earliest epicardial breakthrough significantly influences the configuration of the surface electrocardiogram. However, an electrocardiogram still is capable of only indicating the vicinity from which an ectopic beat originates and cannot define accurately the actual site of origin of the arrhythmia. This study emphasizes the fact that electrophysiologic studies of arrhythmias must include endocardial mapping (or selected endocardial recordings) in addition to epicardial sequencing to fully evaluate these arrhythmias.

Acknowledgment

The authors wish to express their sincere appreciation to Mr. Ralph Iannuzzi for his expert technical assistance.

References

8. Friedman PF, Fenoglio JJ, Wit AL: Time course of reversal of electrophysiological and ultrastructural abnormalities in subendocardial Purkinje fibers surviving extensive myocardial infarction in dogs. Circ Res 36: 127, 1975
Echocardiographic Spectrum of Ebstein's Anomaly of the Tricuspid Valve

Zia Q. Farooki, M.B.B.S., James G. Henry, M.D., and Edward W. Green, M.D.

SUMMARY Sixteen patients aged between one day to 18 years with Ebstein's malformation of the tricuspid valve were studied with ultrasound. The findings were compared with a group of 74 patients without Ebstein's malformation. Two features were considered specific for Ebstein's malformation: 1) ability to record the anterior tricuspid leaflet (ATL) farther to the left of the left sternal border than in the control group; and 2) abnormally prolonged interval between the "C" points of the tricuspid and the mitral valve echoes (M,T,I). All other parameters measured were non-specific for Ebstein's anomaly of the tricuspid valve. The sail sound was recorded in ten patients with Ebstein's malformation and occurred at the time when the anterior tricuspid leaflet was in the most posterior position.

In a patient with congenital heart disease, an M,T,I interval greater than 0.03 sec and recording of an anterior tricuspid leaflet near the apex of the heart strongly suggest the diagnosis of Ebstein's malformation. These two criteria were not fulfilled in any patient who did not have Ebstein's malformation. Conversely, however, absence of these two features does not rule out Ebstein's anomaly of the tricuspid valve.

☐ EBSTEIN'S ANOMALY of the tricuspid valve was first described by W. Ebstein in 1866.1 It accounts for approximately 1% of all congenital heart defects.2 The clinical spectrum and the natural history of this disease have recently been reviewed.3 4 Although the pathology of the tricuspid valve in Ebstein's anomaly is extremely variable,3 three features are relatively constant: 1) redundancy of the valve tissue; 2) adherence of varying portions of the grossly abnormal septal and posterior cusps of the tricuspid valve to the right ventricular wall, resulting in the formation of an "atrialized" portion of the right ventricle; and 3) a normally attached, large anterior tricuspid leaflet.

The anterior tricuspid leaflet motion can easily be recorded by ultrasound technique. Echocardiographic studies have been reported previously in 33 patients with Ebstein's anomaly.5-11 We have studied 16 patients with Ebstein's anomaly and have compared the echocardiographic findings with a group of 74 patients without Ebstein's anomaly.

Materials and Methods

The echocardiographic examinations were performed with Ekoline 20, Cambridge strip chart recorder, and 2.5 or 5.0 mHz transducers. The tracings were recorded on a photographic paper at a speed of 50-100 mm per second. The time lines were 0.04 seconds apart. The following were recorded/measured (figs. 1 and 2):

1) “c” point was defined as the most posterior position of the anterior tricuspid leaflet (ATL) or anterior mitral leaflet (AML) at the onset of systole (fig. 1); 2) “d” point was defined as the point at which slow anterior motion of the closed atrioventricular valve changes to rapid anterior opening motion (fig. 1); 3) “T,” was defined as the most anterior position reached by the fully open ATL (fig. 1); 4) “M,” was defined as the most anterior position reached by the fully open AML; 5) M,T,I: interval between the “c” points of AML and ATL; 6) M,T,A: interval between the “d” points of AML and ATL; 7) M,T,o: interval between the “o”
Subendocardial origin of ventricular arrhythmias in 24-hour-old experimental myocardial infarction.
L N Horowitz, J F Spear and E N Moore

*Circulation*. 1976;53:56-63
doi: 10.1161/01.CIR.53.1.56

*Circulation* is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 1976 American Heart Association, Inc. All rights reserved.
Print ISSN: 0009-7322. Online ISSN: 1524-4539

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://circ.ahajournals.org/content/53/1/56

Permissions: Requests for permissions to reproduce figures, tables, or portions of articles originally published in *Circulation* can be obtained via RightsLink, a service of the Copyright Clearance Center, not the Editorial Office. Once the online version of the published article for which permission is being requested is located, click Request Permissions in the middle column of the Web page under Services. Further information about this process is available in the Permissions and Rights Question and Answer document.

Reprints: Information about reprints can be found online at:
http://www.lww.com/reprints

Subscriptions: Information about subscribing to *Circulation* is online at:
http://circ.ahajournals.org//subscriptions/