Epicardial Activation of the Intact Human Heart
Without Conduction Defect

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SUMMARY To describe the epicardial ventricular activation sequence in the intact human heart, we obtained epicardial maps from 11 patients with normal QRS undergoing open heart surgery. Epicardial breakthrough (EBT), defined as the emergence of a radially propagating epicardial wavefront, occurred in three to five sites in each patient, and was earliest in the anterior right ventricle, 7–25 msec (mean 17 msec) after the onset of the QRS in all patients. Subsequent EBT occurred in the inferior right ventricle (10 sites in 10 patients), in the anterolateral left ventricle (13 sites in 10 patients), and in the inferior left ventricle (eight sites in seven patients). Latest epicardial activation (LEA), defined as the latest site of recordable epicardial activity, occurred in the basal segments in all patients, anteriorly in the right ventricle in five patients, and inferiorly in six patients, four on the right and two on the left. LEA occurred 63–96 msec (mean 77 msec) after the onset of the QRS, and was recorded within 20 msec of the end of the QRS in all patients. Sequence of epicardial activation reflected a fusion process among the wavefronts. This descriptive and quantitative data should provide a suitable basis for comparison of abnormal ventricular activation sequences in patients undergoing surgery for preexcitation or ventricular tachycardia.

MOST DESCRIPTIONS of ventricular activation in the mammalian heart are derived from canine experimentation using epicardial and multiple intramyocardial electrodes.1,2,8,10 Sporadic observations have been made in the human heart under a variety of surgical conditions in the last 50 years, relating mostly to unipolar QRS morphology.8–13 In 1970, Durrer et al. described the epicardial and intramural activation sequence in seven extirpated, reperfused human hearts.14 The activation of the human heart was found to be different in several ways from the dog heart.

Extensive epicardial mapping of the intact human heart has become feasible, as a result of the experience gained with mapping of patients with cardiac arrhythmias. In this study, we systematically report observations of epicardial activation in the intact human heart, describing and quantitating the range of normality in epicardial activation sequence in 11 patients undergoing open heart surgery.

Material and Methods

Patient Selection

We reviewed ECGs of patients scheduled for open heart surgery at the University of Illinois Hospital and West Side Veterans’ Administration Hospital, Chicago. Patients with normal QRS morphology were asked to participate and we requested informed consent for epicardial mapping. Protocol was approved by Human Investigation Committee of University of Illinois. A medical history was obtained, and patients underwent physical examination, had ECGs, vectorcardiograms, chest films, M-mode echocardiograms and diagnostic cardiac catheterizations. A normal QRS morphology was defined by the presence of QRS duration not greater than 0.10 sec, and mean frontal plane axis of between 0° and +90°. We excluded patients who had previous myocardial infarction as determined by history, ECG, and absence of ventriculogram showing localized dyskinesia. Patients were also excluded if ECGs revealed either left or right ventricular hypertrophy.

Patients were operated via a median sternotomy, allowing good access to all aspects of the ventricles for epicardial mapping. Mapping was performed during sinus rhythm, before cardiopulmonary bypass, by previously described techniques.15 Simultaneous recordings were made of multiple surface ECG leads, chosen from the six available frontal plane leads and a precordial lead (usually I, II, III, V₅), a bipolar ventricular reference electrogram and bipolar electrograms from the exploring probe. The bipolar reference electrode consisted of two stainless steel suture electrodes (Flexon, Davis & Geck, Pearl River, NY) attached to the anterior right ventricular wall. The exploring electrodes were contained in a hand-held probe with three silver terminals 2 mm apart, embedded in a teflon tip (Elecath, Rahway, N.J.). Signals were isolated, amplified and recorded at 100 and 200 mm/sec paper speeds on a multichannel oscilloscopic recorder with filter settings of 40–500 Hz for the exploring electrograms and 0.1–40 Hz for the reference electrograms and surface cardiogram (Electronics for Medicine, Minneapolis, Minn). We explored 54–70 epicardial ventricular sites in each heart. Local epicardial activation times reflect the mean of five to 10 beats in stable sinus rhythm, and were measured in milliseconds from the earliest ventricular
deflection in surface or reference leads, to the point at which the first rapid deflection crossed the baseline in the local bipolar electrogram. The diaphragmatic and left lateral surfaces of the heart were explored partly by palpation, with a hand inserted in the pericardial sac, to avoid dislocation of the heart. Landmarks for localization included the atrioventricular and intraventricular sulci, major coronary arteries and the acute and obtuse margins of the heart. Hematocrit, arteriole blood gases, serum electrolytes and glucose were continuously monitored and maintained within normal limits throughout the mapping studies, which were completed within 15 minutes in all cases. The reproducibility of the timing measurements during analysis of the mapping data has previously been examined by us (unreported data) by: 1) duplicate recording from the same data points in several patients, and 2) blind measurement by a second observer involved in the study, in all patients. The range of accuracy in our measurements is ± 5 msec.

Stability of the map was ensured by noting: 1) stable sinus rhythm throughout, with sinus rate varying by no more than 10 beats/min and 2) stable QRS morphology and axis monitored by the six or seven available ECG leads in all cases. The 12-lead postoperative ECG resembled the preoperative in QRS duration morphology and axis. No morbidity was encountered from the mapping procedure.

**Definitions**

Epicardial breakthroughs were defined as sites of emergence of a radially propagating wavefront at the epicardial surface, producing an island of early activation, completely surrounded by points of later activation. Latest epicardial activation was noted for the ventricles as a whole, and was considered the site of latest recordable ventricular activation. Sites of latest activation were also noted for each ventricle.

**Results**

Eleven patients fulfilled the criteria for selection. Clinical and electrocardiographic data are listed in Table 1. There were seven males and four females, age 35–64 years (mean 52 years). All patients had arteriosclerotic heart disease, except one with a secundum type atrial septal defect (normal QRS without right bundle branch block pattern). The QRS duration varied from 0.08–0.10 sec (mean 0.08 sec). Mean frontal plane QRS axis ranged from 0° to +60° (mean +35°).

**Epicardial Breakthrough (Table 1)**

All patients had three to five discrete sites of epicardial breakthrough — five patients had three breakthrough sites, three patients had four sites, and three

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**Table 1. Clinical and Epicardial Data**

<table>
<thead>
<tr>
<th>Pt No.</th>
<th>Age/Sex</th>
<th>Diagnosis</th>
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<th>QRS duration (msec)</th>
<th>Epicardial breakthroughs site and timing (msec)</th>
<th>Latest epicardial activation site and timing (msec)</th>
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<td>ARV ILV</td>
<td>65*</td>
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</table>

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*Overall latest epicardial activation.

Abbreviations: Axis = mean frontal QRS axis; CAD = coronary artery disease; ASD = atrial septal defect; BT1, BT2, . . . BT5 = 1st, 2nd, . . . 5th breakthroughs; ARV = anterior right ventricle; ALV = anterolateral left ventricle; IRV = inferior right ventricle; ILV = inferior left ventricle; F = female; M = male.
patients had five sites (table 1). Examples of typical patterns of epicardial activation are shown in figures 1 and 2. Figure 1 is the epicardial map from patient 7, who had coronary artery disease, and had three sites of epicardial breakthrough. The QRS duration was 90 msec in this patient. Earliest epicardial breakthrough was in the anterior paraseptal right ventricle (the area pretrabecularis), at 11 msec. We noted subsequent breakthrough sites in the anterolateral left ventricle at 28 msec (in the obtuse marginal region), and in the inferior right ventricle at 30 msec. Completion of epicardial activation occurred in the outflow tract region of the anterior right ventricular wall at 78 msec, 12 msec before the end of the QRS in this patient. The latest site of left ventricular activation was the inferior aspect of the left ventricle, 67 msec after onset of the QRS.

Figure 2 is the map from the patient (2) with second-degree atrial septal defect, who had five epicardial breakthrough sites, two in the right ventricle and three in the left ventricle. The QRS duration was 80 msec in this patient. Earliest epicardial breakthrough occurred 16 msec after QRS onset in the anterior paraseptal right ventricle. We observed subsequent breakthrough sites in the inferior left ventricle at 18 msec, anterolateral left ventricle at 20 msec, both in the basal and apical regions, and in the inferior right ventricle at 22 msec. The ventricles were depolarized by a process of fusion, with the latest area of epicardial activation in the right ventricular basal inferior wall at 70 msec, 10 msec before the end of the QRS in this patient. The latest left ventricular site activated was at the inferior basal region close to the obtuse margin at 60 msec.

The epicardial breakthrough sites in the 11 patients are summarized in figure 3. Forty-two sites were identified. Earliest epicardial breakthrough in the 11 patients was in the anterior right ventricular wall adjacent to the septum. We observed subsequent breakthrough sites in all patients. In 10 patients, we noted

![Epicardial map from patient 7.](image)

**Figure 1.** Epicardial map from patient 7. In this and subsequent maps, the anterior, inferior and left lateral views are shown. There is some overlap in the anterior and left lateral views in a zone adjacent to the anterior interventricular sulcus. The QRS in lead III is displayed above the time scale in msec, with 0 as the QRS onset. Each data point reflects the timing (in msec) of arrival of local activation at each point. Isochronous lines are drawn at 5-msec intervals up to 40 msec, and 10-msec intervals, thereafter. This example demonstrates the minimal criteria for definition of epicardial breakthrough, as seen in the inferior right ventricle, where the earliest activated site occurs only 2 msec earlier than adjacent sites. In 27 of the 42 breakthrough sites identified in the 11 patients, the interval between earliest and subsequently activated adjacent sites was 3 msec or greater. All sites fulfill criteria as defined in the text for identification of early breakthrough.
breakthrough in the inferior right ventricle; 13 sites, representing 10 patients, were noted in the anterolateral left ventricle, and eight sites, representing seven patients, were noted in the inferior left ventricle.

The timing of epicardial breakthroughs was examined. All breakthrough sites appeared 7-48 msec after the onset of the QRS. Earliest breakthrough in the anterior right ventricle occurred 7-25 msec after the onset of the QRS, with a mean of 17 msec in the 11 patients. Second breakthrough, seen in all patients, occurred at 18-45 msec (mean 28 msec). Third breakthrough, seen in all patients, occurred at 20-47 msec (mean 33 msec). Fourth breakthrough, seen in six patients, occurred at 20-48 msec (mean 35 msec). Fifth breakthrough, seen in three patients, occurred at 22-38 msec (mean 30 msec).

Latest Epicardial Activation (table 1)

The site and timing of areas of latest epicardial activation were analyzed. The sites found are summarized in figure 4. All sites of latest epicardial activation were found at the base of the heart, four anteriorly in the conus region, one anteriorly near the acute margin of the right ventricle, and six distributed along the atrioventricular groove, inferiorly, four on the right and two on the left. The anterolateral basal left ventricle was never the site of terminal ventricular activation in these patients. Recorded epicardial activation was complete by 63-96 msec, with a mean of 77 msec after the onset of QRS. All latest sites were recorded within 20 msec of the end of the QRS. In 10 cases, latest activation was identified before the end of the QRS by 2-20 msec (mean 14 msec). In one patient, latest epicardial activation was recorded 16 msec after the visible end of the QRS complex.

Analysis of the site and timing of latest activation of the two ventricles separately was also performed. As shown in table 1, we found a site of latest epicardial activation on each ventricle. For the right ventricle, in nine of 11 patients (all except patients 3 and 5), this was also the latest activated site on the epicardium as a whole. Latest right ventricular activation occurred 60-96 msec (mean 75 msec) after the onset of the QRS and was located anteriorly in six patients and inferiorly in five patients. In the case of the left ventricle, the latest site was also the latest site on the epicardium as a whole in two patients (3 and 5). Latest left ventricular activation occurred 60-87 msec (mean 70 msec) after onset of the QRS, and was located in the inferior left ventricle in all patients, at the base in eight patients, and as a sink removed somewhat from the basal rim in three patients (1, 8 and 9).

Sequence of Epicardial Ventricular Activation

The pattern of epicardial activation from the moment of earliest epicardial breakthrough to the mo-
ment of latest epicardial activation was studied. The anterior right ventricle was depolarized entirely from the anterior right ventricular wavefront in all patients. The inferior right ventricle was depolarized predominantly from the emerging inferior right ventricular breakthrough, when present (10 patients). When additional inferior left ventricular breakthroughs were present (six patients), these contributed partially to inferior right ventricular depolarization, by crossing the septum and fusing with the inferior right ventricular wavefront 25–50 msec after the onset of QRS (fig. 2). This effect was most marked when the two contralateral breakthrough sites were distant from one another, e.g., a basal inferior right ventricular wavefront fusing with an apical inferior left ventricular wavefront (patient 10). In the absence of an inferior right ventricular breakthrough (patient 3), the inferior right ventricle was activated from the inferior left ventricle. The inferior left ventricle was depolarized

Figure 3. Summary of 42 sites of epicardial breakthrough in 11 patients with normal QRS. Views of the heart are as for figure 1. Each circle represents one area where breakthrough is found. Numbers of patients showing breakthrough in each area are shown. LV = left ventricle; RV = right ventricle.

Figure 4. Summary of 11 sites of latest epicardial activation. Semicircular areas abutting the AV groove represent areas where latest epicardial activation is found. Numbers of patients showing latest epicardial activation in each area are shown. LV = left ventricle; RV = right ventricle.
predominantly from inferior left ventricular emerging wavefronts, when present (seven patients). Although six of these patients had additional inferior right ventricular wavefronts, the right ventricular wavefronts did not appear to influence depolarization of the left ventricle; the wavefront on the left side of the septum was "dominant" in the inferior wall (fig. 2). In the absence of discrete left inferior breakthrough sites, there was always (four patients) a breakthrough on the inferior right ventricle and one or two breakthroughs on the anterolateral left ventricular wall. These two wavefronts fused with each other 50–60 msec after the onset of the QRS, to depolarize the inferior left ventricular epicardium (fig. 1).

In the anterolateral left ventricle (in all cases expect patient 11) one or two sites of epicardial breakthrough were responsible for most of anterolateral left ventricular activation. In all but one patient (patient 2, fig. 2), the anterior right ventricular breakthrough was sufficiently early and sufficiently close to the septal region, so that it influenced significantly the process of depolarization of the paraseptal region of the left ventricle, by fusing 20–60 msec after the onset of the QRS, with the anterolateral left ventricular wavefronts (fig. 1). In no patient was the inferior left ventricular breakthrough early enough or close enough to the obtuse margin to influence significantly the pattern of activation of the anterolateral left ventricular epicardium.

Discussion

In the dog heart, 3–7 ventricular activation begins on the left septal surface, near the junction of apical and middle thirds. This coincides with the onset of the QRS complex, and is followed within five msec by endocardial activation of the base of the left anterior and posterior papillary muscles. From here, left ventricular endocardial layers are depolarized rapidly as a truncated cone from apex to base, over 10 msec. The septum is activated in a centrifugal fashion, predominantly from left to right, spreading from the initial site. The left ventricular free wall depolarizes with an endo-epicardial sequence, the latest areas to be activated being the basal free wall and septum (40 msec after the onset of QRS). The depolarization of the right ventricle begins in the endocardium near the base of the anterior papillary muscle, at the insertion of the moderator band, five msec after initial left septal activation. Rapid right ventricular subendocardial activation results within 10 msec, and contributes to septal activation. Right ventricular free wall activation occurs tangentially, spreading to the epicardium as a single breakthrough opposite the base of the anterior papillary muscle, at 10–15 msec, from where more or less concentric isochronic spread occurs, terminating in the conus region by 30–35 msec. Thus, at the epicardial surface, three breakthrough sites are described, first in the right ventricular pretrabecular area as above, and later in two left ventricular sites, anteriorly adjacent to the midportion of the septum at 15–20 msec, and posterobasally at about 20–25 msec. These three epicardial sites correspond to the sites of earliest endocardial activation via the right bundle branch, and the anterior and posterior ramifications of the left bundle branch, respectively.

Over the last 50 years, sporadic observations have been made in the intact human heart under a variety of surgical conditions. 8–13 These studies were primarily concerned with the investigation of QRS morphology at various points over the epicardial surface. Only a limited number of recording sites were explored in these early studies, limiting the spatial resolution of the chronology of excitation. In most of these studies, the arrival of local subepicardial activity was inferred from the timing of the intrinsic deflection in unipolar leads. This was the case in the study by Roos et al., 14 in which portions of the epicardial surfaces of six patients with lung carcinoma were mapped, together with intramural recordings from a single platinum electrode introduced into the left ventricle, at the time of pneumonectomy. Only a single epicardial breakthrough site (anterior right ventricle) was identified with certainty in these patients, probably in part due to the limited number of recording sites used, 7–24 and the absence of any data from the inferior surfaces of the ventricles.

In 1970, Durrer et al. 14 reperfused and revived seven hearts removed from automobile accident victims, suspended the beating hearts in perfusate, and performed detailed epicardial and intramural mapping. In these hearts, minor variations in the activation process were observed, but the usual pattern of depolarization was to excite synchronously three left ventricular endocardial areas, 0–5 msec after the start of the left ventricular cavity potential: 1) an area high on the anterior paraseptal wall just below the attachment of the mitral valve; 2) a central area on the left surface of the interventricular septum; and 3) a posterior paraseptal area about one-third of the distance from apex to base. Rapidly expanding fronts from these areas became confluent by 15–20 msec, reaching the epicardial surface of those sites overlying the areas of earliest endocardial excitation by 30 msec. The latest site to be activated was either the posterobasal or posterolateral area of the left ventricle. Endocardial right ventricular activation started near the insertion of the anterior papillary muscle 5–10 msec after the onset of the left ventricular cavity potential. Then, rapid invasion of the septum and adjoining free wall occurred, resulting in right ventricular epicardial breakthrough in the area pretrabecularis after about 20 msec. The isochrones proceeded tangentially, reaching ultimately the pulmonary conus and the posterobasal area at 60–70 msec. In general, ventricular septal activation proceeded from left to right and in an apicobasal direction.

In Durrer’s study, 14 the epicardial excitation pattern reflected the movement of the endocardial and intramural fronts. Earliest epicardial breakthrough occurred in the anterior right ventricle near the septum in the 20–25-msec interval, spreading concentrically, activating the posterobasal region slightly later than the anterior atroventricular sulcus region or the pulmonary conus. Larger variations were seen in the
epicardial sequence over the left ventricle. Three early points of epicardial activation were seen: 1) an area on the anterior surface paraseptally, close to the atrio-
ventricular sulcus, 2) an anterior paraseptal area located halfway between apex and base, and 3) a posterior paraseptal area halfway from apex to base. In some hearts an additional area of left ventricular epicardial breakthrough was found near the apex posteriorly. The location of the latest activated area in the left ventricle was generally the posterobasal left paraseptal region, or in a more lateral location posteriorly.

Our study was performed to resolve the question of whether the data from the reperfused hearts of Durrer et al.14 accurately reflected epicardial activation in the intact human heart, and to quantitate further the range of normality. In our study, 11 patients with normal QRS were mapped before institution of cardiopulmonary bypass. All patients had three to five epicardial breakthrough sites, and all 11 patients had first breakthrough in the anterior right ventricle. Subsequent breakthrough sites appeared in the anterolateral left ventricle in 10 patients, in the inferior right ventricle in 10 patients, and in the inferior left ventricle in seven patients. All breakthroughs occurred 7–48 msec after the onset of QRS. The earliest right ventricular epicardial site ranged from 7–25 msec (mean 17 msec). Figure 3 shows that most of the epicardial breakthrough sites identified in this study were consistent with the data of Durrer et al.14 However, in virtually all hearts, we found a previously undescribed breakthrough site, on the inferior right ventricular wall. In four patients, this was located near the base adjacent to the septum. In another five, it was found toward the acute margin posterobasally, and in one, toward the right ventricular apex.

Latest epicardial activation corresponded to the terminal portion of the surface QRS, and was recorded within 20 msec of the end of the QRS in all of our cases. The site of latest activation on the epicardium (fig. 4) was always at the atroventricular sulcus, and was seen variously in the conus (four patients), the anterobasal right ventricle (one patient), and along the posterobasal region in six patients (four on the right and two on the left). The basal anterolateral left ventricle was never a site of latest epicardial activity.

Our data is consistent with that of Durrer et al.14 in respect to sites and timing of epicardial breakthrough, with the exception of the inferior right ventricle where a new finding of epicardial breakthrough is described. In respect to sites and timing of latest epicardial activation, we are also generally in agreement. In nine of our 11 patients, the basal right ventricular epicardium was activated last. We, as Durrer, found the single most common latest site of epicardial activation to be the outflow region of the right ventricle. However, an equal number of patients had latest activation of the posterobasal regions (paraseptal, marginal) of the right ventricle.

As described above, when the latest activated site of the individual ventricle was analyzed, the latest left ventricular activated site was always the postero-basal region in our study; this is consistent with Durrer's data. However, Barbato et al.13 stated that latest left ventricular activation occurred in the lateral half of the anterior surface and lateral surface, in normal human hearts. We believe that the paucity of data from the inferior aspect of the left ventricle in their cases probably obscured even later sites in this region.

The process of fusion of epicardial wavefronts yielding the pattern of activation of the epicardial surface, reflected the number, timing and location of the epicardial breakthrough sites. The onset of epicardial activation in the anterior right ventricle at 7–25 msec after QRS onset implies that the thinness of the right ventricular wall allows more rapid transmission to the epicardium of the later endocardial wavefront in the right ventricle. In the left ventricle, earlier endocardial activation and epicardial breakthroughs are, in part, a function of thickness and transmural activation velocity across the left ventricular free wall.

Our finding of an almost invariable breakthrough in the inferior right ventricular epicardium is difficult to interpret. There is no anatomic evidence to suggest that the right bundle branch splits into major divisions or fascicles, as may be the case on the left side.16–21 An alternative explanation seems more likely — that the relative thinness of the free wall of the inflow region of the right ventricle compared with the outflow and apical region, as shown by Eckner et al.,42 allows early emergence of activation at the basal inferior epicardial surface, of a wavefront which envelopes the right ventricular subendocardial layers almost instantaneously. Why these sites have not been documented previously may be related to the relative inaccessibility of the diaphragmatic wall of both ventricles and the paucity of data from these regions in many previous studies. We do not have an explanation for the absence of these breakthroughs in the cases of Durrer et al.14 and Fontaine et al.23 Since the patients studied by Fontaine et al.23 included many with right ventricular abnormalities, possibly some form of intramural right ventricular activation delay accounted for the absence of inferior right ventricular breakthrough in sinus rhythm in their patients.

Our findings in the left ventricle may indicate that the anterobasal paraseptal left ventricular breakthrough corresponds to high anterior activation of the endocardium via the anterior border fibers or fascicle of the left bundle branch, that the left ventricular inferior breakthroughs correspond to endocardial activation via the posterior border fibers or fascicle, and that the apical (anterior or inferior) left ventricular breakthroughs reflect emergence of the apical front generated by endocardial activation of the midseptal region.

Limitations of the Study

Our patients had cardiac disease (coronary disease in the majority), and subtle derangements in ventricular activation may have been present, resulting in departures from the truly “normal” pattern. Such departures would be expected to affect largely the mid
and late QRS sequence, as suggested by recent isopotential body surface mapping studies. Our study, therefore, is limited to a statement regarding epicardial activation sequence of patients with normal QRS, rather than that of normal patients. These limitations, however, would not be expected to affect the early QRS activation sequence in the absence of diagnosable infarction or intraventricular conduction defects. For these reasons we believe the observations regarding site and timing of epicardial breakthrough events are valid.

Clinical Implications

Our data provides a base which can be used to interpret mapping studies in patients with arrhythmia. Several sites of epicardial breakthrough can normally be seen in both left and right ventricles, both anteriorly and inferiorly, often in areas close, but not directly contiguous, to the atrioventricular sulcus. We anticipate that this data should be useful as a basis for comparison with the abnormal epicardial activation sequence in ventricular preexcitation and ventricular tachycardia. Knowledge of the range of normality in epicardial activation sequence should also provide a basis for interpretation of the disordered sequence seen in chronic or surgically induced intraventricular conduction defects. We hope that our data may also be helpful in the interpretation of computerized isopotential body surface mapping studies, in which the appearance of normal sinks or minima are usually interpreted as indicating epicardial breakthrough phenomena. The location of these phenomena on the basis of the body surface studies involves some extrapolation. Knowledge of the range of normal breakthrough phenomena should assist this extrapolation process.

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