Regional Asynchrony During Acute Myocardial Ischemia Quantified by Ultrasound Strain Rate Imaging

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OBJECTIVES

We propose a new method to easily quantify asynchronous wall motion due to postsystolic shortening (PSS). We also studied the relationship of the spatial and temporal extent of PSS to the extent of myocardium at ischemic risk after variable duration of ischemia.

BACKGROUND

Postsystolic shortening is a sensitive marker of asynchrony during ischemia. Current techniques for detection of asynchrony are either subjective, or invasive and time-consuming. Strain rate imaging (SRI) can noninvasively depict PSS as prolonged compression/expansion crossover.

METHODS

Nineteen open-chest pigs were scanned from apical views, before and after left anterior descending coronary artery occlusion. Strain rates were derived offline from tissue Doppler velocity cineloops. The time from electrocardiographic R-wave to the occurrence of compression/expansion crossover (T_{CEC}) was calculated. Prolonged T_{CEC} during ischemia was identified using a standardized analysis and both spatial (% of left ventricle) and temporal extent were quantified. The extent of myocardium at risk was measured in seven animals from dye-stained specimens.

RESULTS

Prolonged T_{CEC} was found in all ischemic segments. There was a good correlation (r = 0.91; p < 0.001) and good agreement between the spatial distributions of prolonged T_{CEC} and myocardium at risk. The extent of myocardium at risk was better approximated by T_{CEC} measurement (36 ± 7% vs. 39 ± 8%, respectively; p = NS) than by wall motion analysis (47 ± 17%, p < 0.05). The duration of occlusion did not prolong T_{CEC}.

CONCLUSIONS

Prolonged T_{CEC} consistently occurs in ischemic myocardium and is apparently not affected by the duration of ischemia. Standardized analysis of T_{CEC} in SRI closely quantifies the extent of ischemic myocardium. This new method may be a useful tool in other cardiac conditions associated with regional diastolic asynchrony. (J Am Coll Cardiol 2001;37:1141–8) © 2001 by the American College of Cardiology

Echocardiographic detection of wall motion abnormalities relies on visual interpretation of asynchrony in the contraction/relaxation sequence and amplitude of wall motion, which may introduce an error of up to 90 ms (1). Postsystolic shortening (PSS) or thickening (depending on direction of measurement) represents persistent contraction after aortic valve closure and is a marker of asynchrony (2,3). Postsystolic shortening has been reported in several clinical conditions, such as myocardial ischemia (3–6), stunning (7), hypertrophic cardiomyopathy (8), aortic stenosis (9), syndrome X (10) or left bundle branch block (11). However, PSS has so far been evidenced using either invasive or complex and time-consuming technology.

Tissue Doppler imaging enables accurate detection, quantitation and display of local myocardial velocities (12). Strain rate imaging (SRI) can be derived from tissue velocity data and can quantify intramyocardial contraction and expansion independent of translation (13). Recent studies on myocardial ischemia have shown that PSS (also referred to as postsystolic compression) can be easily detected using SRI (14–16). In-depth quantitation of the phenomenon was not performed, or only one region at a time was investigated. It remains unknown to what extent such changes in SRI are related to the extent of myocardium at risk and duration of ischemia.

Nonuniformity in the heart has been described in terms of myoarchitecture, mechanics, electrical properties, vascular supply and excitation-contraction coupling (17). In-depth studies of wall motion must take into account the variability in regional contraction and relaxation, both within the heart and between individuals. This study: 1) proposes a new method for spatial quantification of asynchronous compression/expansion sequence in an individual during ischemia, using a statistical approach, and 2) reports the topographical relation of regions with prolonged compression to the extent of myocardium at ischemic risk and duration of ischemia.

METHODS

Animal preparation. Domestic pigs weighing 30 to 40 kg were anesthetized with an infusion of Ketamine, Fentanyl and Amideate, intubated and mechanically ventilated. A median sternotomy was performed and the heart exposed in a pericardial cradle. After baseline echo measurements, the mid- or distal portion of the left anterior descending (LAD) coronary artery was ligated. Body temperature was kept constant with a heating pad.
In seven animals, triphenyltetrazolium chloride solution was injected intravenously at the conclusion of the experiment, with the LAD ligature in place, in order to differentiate normally perfused myocardium (stained tissue) from the myocardium at ischemic risk (unstained tissue). Five minutes after the dye injection, the animals were euthanized (Sleepaway; Fort Dodge Laboratories, Fort Dodge, Iowa); the hearts were excised, cannulated and pressure-perfused overnight with formaldehyde.

All experiments conformed to the Position of the American Heart Association on Research Animal Use. The study protocol was approved by the Institutional Animal Care and Use Committee of the Mayo Clinic.

Echocardiography. A commercially available ultrasound scanner (GE Vingmed System FIVe, GE Medical Systems, Horten, Norway, 3.5 MHz transducer) was used to collect digital loops in tissue Doppler imaging mode from epicardial approach. Three standard apical views were acquired: two-chamber, four-chamber and apical long-axis. Digital loops of single cardiac cycles were collected in expiratory apnea at baseline and during ischemia, with 60–65 frames/s. The Nyquist limit for velocity was set between 8.9 and 14 mm/s. All data were analyzed offline.

Data analysis. Each loop comprised two simultaneously acquired data sets: velocity information and conventional gray-scale data. A modified 18-segment perfusion model was used to divide the left ventricle (LV) into individual coronary perfusion territories (18). In this model, the LV in each apical view was divided into two cardiac walls, each of which was subdivided into three segments: basal, mid and apical.

STRAIN RATE IMAGE ANALYSIS. Strain rates were computed from velocity data using custom software (Bjorn Ostadt, Horten, Norway, 3.5 MHz transducer) was used to collect digital loops in tissue Doppler imaging mode from epicardial approach. Three standard apical views were acquired: two-chamber, four-chamber and apical long-axis. Digital loops of single cardiac cycles were collected in expiratory apnea at baseline and during ischemia, with 60–65 frames/s. The Nyquist limit for velocity was set between 8.9 and 14 mm/s. All data were analyzed offline.

Strain rates were calculated as the difference between two velocity points along the ultrasound beam divided by the distance between the points (5 mm in our analysis). Negative strain rate values (representing compression) were displayed in yellow-orange and positive strain rate values (representing expansion) in cyan-blue (Fig. 1A). Strain rate values corresponding to an outline manually set along the LV midwall (19) were reconstructed for the complete cardiac cycle in a longitudinal M-mode strain rate map (X-axis representing time and Y-axis representing the unfolded LV circumference). The

software facilitates midwall tracking throughout the cardiac cycle. This way, one strain rate map was generated from each loop, depicting the longitudinal shortening (compression) and lengthening (expansion) of the LV walls along the ultrasound beam. All strain rate maps were assigned a random number and the compression/expansion crossover (i.e., the transition from orange to blue color) was manually delineated by two investigators who were blinded to the animal data.

Analysis of the delays in the compression/expansion crossover was performed in two ways: 1) continuously along the entire outline (for better description of the regional dysfunction in both spatial and temporal extent), and 2) as a mean value per segment (for comparison with conventional wall motion analysis).

STANDARDIZED ANALYSIS OF THE REGIONAL COMPRESSION/EXPANSION Crossover. To quantify the spatial and temporal extent of postsystolic compression, we propose a technique similar to the centerline method used in interpreting ventriculograms (20). Briefly, the LV circumference (i.e., the mid-myocardial outline length) was divided into 120 equidistant parts (chords; i = 1, 2, . . . , 120). The time from electrocardiographic peak R-wave to the compression/expansion crossover (T_cec) was calculated for each chord with customized software, and heart rate corrected (corrected T_cec = T_cec / RR).

A database of mean T_cec of each chord (Mi) and standard deviation (SD) were calculated at baseline for each apical view from the corresponding T_cec values of all animals. To identify and quantify ischemia in a particular animal, we defined the standardized T_cec for each chord according to the formula: standardized T_cec = (T_cec(ischemia) − Mi) / SD, and expressed it in SD units. Chords with standardized T_cec < 1 SD unit were defined as normal T_cec, whereas chords with standardized T_cec > 1 SD unit were defined as prolonged T_cec or abnormal (Fig. 2).

The spatial extent of the region with prolonged T_cec was computed as the percent of contiguous chords with standardized T_cec > 1 SD unit in each view. Scattered regions of fewer than four chords with standardized T_cec > 1 SD unit were considered noise and not counted. The magnitude of T_cec prolongation was computed as the mean value of standardized T_cec of all abnormal chords and considered to reflect the degree of regional asynchrony.

WALL MOTION ANALYSIS. Wall motion score (WMS) was evaluated from the gray-scale loops by an independent observer experienced in the assessment of wall motion analysis and blinded to SRI data. The scoring system was based on the standard American Society of Echocardiography recommendations, with 1 = normal motion, 2 = hypokinesia, 3 = akinesia and 4 = dyskinesia (21). Segments with WMS > 1 were counted (% of LV).

To compare T_cec with the WMS, segmental T_cec was calculated by averaging the T_cec values from all 20 chords.
in that segment. An average value for segmental $T_{CEC}$ per WMS was calculated in each animal, then mean segmental $T_{CEC}$ per WMS was calculated by averaging all animals.

THE THREE-DIMENSIONAL RECONSTRUCTION OF STAINED CARDIAC SPECIMENS. The seven stained hearts were embedded in a polyurethane Styrofoam block, then cut orthogonal to the LV long axis into 3-mm-thick slices (20 to 25 slices/LV). Myocardium at ischemic risk (unstained zone, Fig. 1C) was sharply demarcated from normally perfused myocardium (red-stained zone). Calibrated digital pictures of LV slices were analyzed with Analyze software (22) and a three-dimensional computer replica of the cardiac specimen was obtained with good spatial resolution. The three apical views were reconstructed from longitudinal transsections through the LV according to the standard orientation of the ultrasound scan-planes with respect to the LV (Fig. 1C). Left ventricular midwall outlines were drawn similar to SRI data, and the extent of the myocardium at risk was measured as the percentage of the unstained myocardium in the outline. All measurements were performed blinded to the SRI data.

STATISTICAL ANALYSIS. Statistical analysis was performed using the Statistical Analysis Software (23). The influence of blood pressure, duration of occlusion and extent of

![Figure 1](https://example.com/figure1.png)

**Figure 1.** Strain rate maps at baseline and during ischemia, and corresponding perfusion-stained specimen, in apical long-axis view. (A) Baseline strain rate imaging frame (left image) and the corresponding longitudinal M-mode strain rate map over one cardiac cycle (right image). Orange represents compression, blue expansion and green low motion. The two vertical straight lines approximate the time of the aortic valve closure and of the mitral valve opening (identified from gray-scale two-dimensional loops). The compression/expansion crossover is indicated by the solid black line as the color transition from orange to blue. The time from the electrocardiographic peak R-wave to the compression/expansion crossover ($T_{CEC}$) is measured for each pixel line in the image. (B) Ischemic strain rate maps from the same view. A prolonged compression pattern (black arrows) can be observed in the apical and mid anteroseptal segments (supplied by the occluded left anterior descending coronary artery), while the rest of segments have normal compression/relaxation pattern. Note also the delayed onset of systolic compression (white arrows) in the same ischemic segments. (C) Computer reconstruction of the stained cardiac specimen at the same level in the heart. Myocardium at risk is represented by the white region (arrows), while the normally perfused myocardium is stained in red. The location of the apical postsystolic compression pattern in the strain-rate maps matches the location of the ischemic myocardium (Segments are abbreviated: bIL = basal inferolateral; mIL = mid inferolateral; aIL = apical inferolateral; aAS = apical anteroseptal; mAS = mid anteroseptal; bAS = basal anteroseptal).
myocardium at risk on the extent and magnitude of prolonged T_{CEC} were tested using multiple stepwise regression analysis. The limits of agreements between two measurements were tested according to Bland and Altman (24). Baseline and ischemic segmental T_{CEC} were compared using paired t tests. Multiple pairwise comparisons were performed using ANOVA and the Ryan–Einot–Gabriel–Welch test as appropriate. The correlation between the segmental T_{CEC} and WMS was tested with Spearman’s rank correlation coefficient (25). The normal distribution was tested with the Shapiro–Wilk statistic and transformations were performed when appropriate. A p-value < 0.05 was considered significant. All calculations were performed with the SAS procedures PROC TTEST, PROC REG, PROC CORR and PROC UNIVARIATE (23). Results are presented as mean ± SD.

RESULTS

A total of 23 experiments were performed. Four animals were excluded (three died during ischemia; one had abnormal baseline LV function). From the remaining 19 animals, 98 circumferential SRI outlines (612 segments) entered the final analysis. From these, 594 segments were interpretable for wall motion analysis. The duration of LAD occlusion varied from 5 to 90 min. Ten animals had LAD occlusions <20 min and nine animals had >20 min of occlusion. The mean heart rate and mean blood pressure did not change significantly from baseline to ischemia (heart rate: 86 ± 17 beats/min and 89 ± 21 beats/min; blood pressure: 80 ± 14 mm Hg and 76 ± 18 mm Hg, respectively).

Spatial extent and magnitude of T_{CEC} prolongation. Figure 1 is an example of a longitudinal M-mode strain rate map in one animal before and during LAD ischemia. At baseline, the compression/expansion crossover occurred near the aortic valve closure. After LAD ligation, the apical and mid-anteroseptal regions showed a prolonged compression pattern (i.e., postsystolic compression) that persisted beyond the mitral valve opening, in asynchrony with the mid inferoposterior and basal segments (normally perfused segments) that had a normal compression pattern. Interestingly, in the ischemic segments brighter hues of orange color were observed in the postsystolic period than during systole. Note that, in this animal, the onset of systolic compression was also delayed in the ischemic segments. The association of delayed onset, prolonged compression pattern and brighter hues during postsystolic compression were highly indicative of regional ischemia.

A representative example of standardized analysis is given in Figure 2. Using this method, regions with prolonged T_{CEC} were detected in all ischemic segments. Prolonged T_{CEC} was found in 35 ± 9% of the LV (35 ± 7%, 37 ± 9%, and 34 ± 10% in two-chamber, four-chamber and apical long axis, respectively). The magnitude of standardized T_{CEC} was 2.3 ± 0.6 SD units (2.6 ± 0.6, 2.0 ± 0.6, and 2.2 ± 0.2 SD units in two-chamber, four-chamber and apical long axis, respectively). Standardized T_{CEC} in centrally ischemic regions (as guided by stained specimen) exceeded 3 SD units.

Figure 3 summarizes the mean ± SD of T_{CEC} at baseline and during ischemia for each apical view. T_{CEC} in the apical segments (i.e., territory of the occluded LAD) was prolonged more than 1 SD from the normal mean, but there...
was no change in $T_{CEC}$ in the rest of the segments supplied by nonoccluded arteries.

Interobserver and intraobserver variabilities for measurements of the $T_{CEC}$ were both <5% (range -10 to 15 ms).

**Extent of myocardium at risk versus prolonged $T_{CEC}$**

The extent of myocardium at risk calculated from seven cardiac specimens was $36 \pm 7\%$ of the LV ($29 \pm 3\%$, $41 \pm 8\%$, and $36 \pm 3\%$ in two-chamber, four-chamber, and apical long axis, respectively), whereas $39 \pm 8\%$ of the LV showed prolonged $T_{CEC}$ ($35 \pm 5\%$, $41 \pm 13\%$, and $40 \pm 4\%$, in two-chamber, four-chamber, and apical long axis, respectively). Figure 4 shows a good correlation between the extent of myocardium at risk and prolonged $T_{CEC}$. The span of prolonged $T_{CEC}$ matched or was slightly larger than the span of ischemic myocardium (mean difference 2.9%; $p = \text{NS}$). Multiple regression analysis showed that the only predictor of the extent of regions with prolonged $T_{CEC}$ was the extent of myocardium at risk ($r = 0.91$, $p < 0.001$).

**Segmental $T_{CEC}$ versus WMS.** Acute ischemia resulted in significant prolongation of $T_{CEC}$ in eight segments (Table 1). At multiple pairwise comparisons, four apical segments (the core of the ischemic region) were significantly different from the rest of the segments.

At global analysis, segmental $T_{CEC}$ during ischemia was correlated with wall motion score ($r = 0.65$, $p < 0.0001$). The mean segmental $T_{CEC}$ per WMS group during ischemia is shown in Figure 5A. Overall, segments with WMS $> 1$ were found in $43 \pm 16\%$ of the LV ($41 \pm 15\%$, $47 \pm 15\%$ and $43 \pm 18\%$ in two-chamber, four-chamber, and apical long axis, respectively). In those animals with measurement of the myocardium at risk, wall motion abnormalities were found in $47 \pm 17\%$ of the LV ($46 \pm 16\%$, $50 \pm 19\%$, and $45 \pm 21\%$ in two-chamber, four-chamber, and apical long axis, respectively). The span of segments with WMS $> 1$ significantly overestimated the extent of ischemia (mean difference 14.3%; $p < 0.05$).

**Influence of duration of occlusion.** A weak correlation was observed between the spatial extent of prolonged $T_{CEC}$ and the duration of occlusion ($r = 0.52$, $p < 0.05$). No significant correlation was found between the magnitude of standardized $T_{CEC}$ and duration of occlusion or blood pressure ($p = \text{NS}$). Even when only the four central ischemic segments were analyzed there was, again, no correlation between the segmental $T_{CEC}$ and the duration of ischemia ($p = \text{NS};$ Fig. 5B). Also, no correlation was found between mean WMS in the four ischemic segments and duration of occlusion ($p = \text{NS}$).

**DISCUSSION**

This study quantifies the spatial and temporal extent of asynchronous diastolic motion occurring after acute LAD ischemia, as assessed by SRI, in an open-chest animal

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**Table 1. Mean Difference in $T_{CEC}$ Values Between Baseline and Ischemia for Each Cardiac Segment (Values in ms, as Mean ± SD)**

<table>
<thead>
<tr>
<th>Segment</th>
<th>Two-Chamber</th>
<th>Four-Chamber</th>
<th>Apical Long Axis</th>
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<td></td>
<td>Inferior</td>
<td>Anterior</td>
<td>Septal Lateral</td>
</tr>
<tr>
<td>Basal</td>
<td>$-6 \pm 32$</td>
<td>$2 \pm 41$</td>
<td>$-3 \pm 52$</td>
</tr>
<tr>
<td>Mid</td>
<td>$11 \pm 46$</td>
<td>$19 \pm 47$</td>
<td>$-42 \pm 56^*$</td>
</tr>
<tr>
<td>Apical</td>
<td>$94 \pm 56^*$</td>
<td>$97 \pm 52^*$</td>
<td>$129 \pm 55^*$</td>
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*Segments with significantly prolonged $T_{CEC}$ during ischemia, as compared with baseline ($t$ test; $p < 0.001$); †Segments with $T_{CEC}$ not different from segments, but significantly prolonged from the rest of the segments within the same view (multiple comparisons between segments; both $t^2 p < 0.05$). Note that segments supplied by distal anterior descending coronary artery were all affected, but there is no significant change in $T_{CEC}$ of segments supplied by nonoccluded coronary arteries.

$T_{CEC}$ = time to compression/expansion crossover.
In this study, we consistently documented the presence of PSS during LAD ischemia indicated by a prolonged T_{CEC} in the ischemic segments. This finding supports the concept that altered compression/expansion crossover is a sensitive marker of ischemia. There was a statistically significant separation of ischemic standardized T_{CEC} from normal, whereas there was no change in T_{CEC} in regions outside of the occluded LAD territory (Fig. 3). The local compression patterns were still present in the ischemic region at the time of mitral valve opening (Fig. 1B), which concurs with sonomicrometer studies (3). The range of T_{CEC} prolongation during ischemia varied between 50 and 150 ms beyond normal. Such small delays in T_{CEC} could be easily missed at conventional analysis (1).

In this study, the regions with postsystolic compression were spatially consistent with the myocardium at risk as measured from stained cardiac specimens, even though a dynamic parameter, such as T_{CEC}, was compared with the anatomic myocardium at risk (which depends on the anatomy of coronary circulation and collateral blood flow supply).

**Influence of duration of ischemia.** The magnitude of T_{CEC} prolongation was not affected by the duration of ischemia within the tested period, which would have been desirable. As neither WMS nor T_{CEC} varied with duration of ischemia, further studies are required to elucidate this effect. Considering that animals with shorter occlusions had presumably more viable myocardium in the area at risk, our results suggest that during the tested period, the delay in T_{CEC} detects the presence of regional diastolic asynchrony rather than the amount of viable tissue present in the ischemic segments.

There was a trend toward a slightly larger extent of regions with postsystolic compression in animals with longer occlusion times. This trend might be due to larger areas at risk or tethering effects (30). Myocardial strain alterations were also found in normal myocardium adjacent to large infarcts with microvascular obstruction (31).

**Segmental T_{CEC} versus WMS.** Segmental T_{CEC} was longer in abnormally functioning segments than in normokinetic ones, but there was no significant difference in segmental T_{CEC} between akinetic and dyskinetic segments, which might be regarded as a limitation. These results suggest that during severe ischemia, T_{CEC} prolongation reflects the regional diastolic dysfunction, irrespective of the severity of systolic dysfunction. We believe that a combined assessment of both regional systolic (32,33) and diastolic function is required for a more complete analysis of regional function. On the other hand, wall motion analysis, but not measurement of T_{CEC}, significantly overestimated the extent of myocardium at risk. Thus, although T_{CEC} was not superior to WMS in estimating the severity of dysfunction, it was more accurate in quantification of the extent of ischemia. Whether measurement of T_{CEC} would be more sensitive for detection of ischemia than the conventional method cannot be answered from this study.
Implications. Quantitative assessment is an important prerequisite for complete description of the dynamic changes that occur during ischemia (3). Conventional assessment of wall motion based on visual assessment is highly subjective and at best semiquantitative. Therefore, reproducible, quantitative, and sensitive indicators of regional ischemia are required. The low temporal resolution used in stress echocardiographic studies, together with the limited ability of human vision to discern small differences in myocardial asynchrony (1), do not allow an objective quantitative assessment of complex wall motion. Tissue Doppler velocity and SRI can accurately quantify local mechanical function with higher temporal accuracy than any current clinical method. Strain rate imaging allows rapid, noninvasive and quantitative assessment of regional systolic function (13,32,33) and asynchrony in contraction/relaxation sequence, facilitating continuous gradation of the ischemic effect in both temporal and spatial domains.

The mechanisms involved in regional asynchrony and PSS are not completely understood. Postischemic shortening has been related to myocardial viability (4,6,34), and with a rapid, noninvasive tool, new studies might expand its clinical utility and bring new insights into its mechanism. It is not clear whether regional asynchrony is a cause or aggravating factor of the global diastolic dysfunction. Our method based on SRI data may be an easy way to detect asynchrony in other clinical conditions associated with regional diastolic dyssynergy (8–11) and monitor its ultimate functional impact on global LV function.

Finally, the recognition of abnormality relies on knowledge of normal temporal nonuniformity in the regional compression and expansion patterns. By using a database, the interpretation may become easier and more accurate without a priori knowledge of individual baseline data. Further studies are underway to test the value of this method for clinical use.

Study limitations. As a derivative of velocities, strain rate is more prone to noise. In our study, 14% of images were discarded because of artifacts; this could be partially avoided with new SRI software available today. Only one cardiac cycle was acquired, and averaging several cardiac cycles may decrease the noise. Lower systolic tissue velocities in apical ischemic segments in humans (35), together with a difference between the Doppler angle of incidence and true apical motion, may introduce errors in strain rate calculation. User interaction was required in this study for delineation of the crossover, although the inter- and intraobserver variability was low. Only one coronary artery occlusion was tested; however, similar findings are reported for other coronary arteries (15,16). Although we did not test the influence of load on the T_{CEC}, experimental work suggests a small effect (36). The influence of duration of occlusion on T_{CEC} was studied in different animals, and further studies are necessary. Although our study brings evidence that the change in one-dimensional deformation was a sensitive indicator of ischemia, interpretations from one axis cannot completely describe complex three-dimensional myocardial strains.

Conclusions. This study demonstrates that prolonged regional compression/expansion crossover as assessed by SRI consistently occurs in territories with severe ischemia. A novel method is proposed for identification and quantification of regional asynchrony using strain rate data, based on a statistical approach, thus making this method relevant to a clinical scenario where knowledge of data prior to the cardiac event is not known. Increased duration of ischemia did not seem to prolong more the temporal extent of postsystolic compression. More objective and quantitative information about the temporal nonuniformity and the regional extent of asynchronous motion can be obtained using SRI technique.

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