Cardiac Memory
Diagnostic Tool in the Making
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Classic definition of the term cardiac memory (CM) refers to the persistent T-wave changes on the ECG after a period of wide QRS rhythms that become evident once normal ventricular activation pattern is restored. It is related to the term ventricular electric remodeling sometimes used in basic science literature. Although CM itself is considered as an adaptive reaction to the change in the ventricular activation sequence, its manifestations (usually T-wave inversions, TWIs) are often confused with pathological conditions manifesting with TWI, such as myocardial ischemia or infarction. Although originally CM was described after restoration of the normal ventricular activation (narrow QRS), recently it was described and studied in wide QRS rhythms significantly expanding the clinical relevance of this phenomenon. Although complex molecular mechanisms of CM have been previously reviewed extensively, practical clinical application of this phenomenon received much less attention. In this article, we will focus on the clinically relevant aspects of CM including its diagnostic use in narrow and wide QRS rhythms. We will also examine how the fundamental CM property of being initiated by the changes in the geometry of the ventricular contraction might open new perspectives in wide QRST morphology analysis by establishing parallels between electric and mechanical functions of the heart.

History, Properties, and Adaptive Nature of Cardiac Memory
In 1915, White\(^1\) was the first to observe the phenomenon of transient TWI after single ventricular premature beats. Later in the 1940s, TWIs after conversion to sinus rhythm were described after paroxysmal tachycardias.\(^2\) Since then abnormal T waves of various duration have been documented after intermittent ventricular pre-excitation,\(^3\)-\(^5\) ventricular pacing,\(^6\)-\(^7\) intermittent left bundle branch block (LBBB),\(^8\)-\(^9\) ventricular tachycardia,\(^10\) and even QRS widening associated with sodium channel blocker toxicity.\(^11\)

In 1982, Rosenbaum et al\(^10\) introduced the term heart memory and presented the first unified hypothesis of how abnormal ventricular activation could lead to the development of T-wave abnormalities regardless of the wide QRS cause by a process he referred to as electrotonic modulation. In this seminal article, 3 principles of CM were formulated: (1) the direction of the T waves in sinus rhythm follows (remembers) the direction of the QRS complex during preceding episode of abnormal activation; (2) the amplitude of memory T waves increases the longer abnormal conduction continues, and (3) repeat episodes of abnormal activation after complete normalization of T waves result in more rapid and prominent accumulation of T-wave changes (hence the term memory). The authors hypothesized that CM represents adaptation of myocardial repolarization to the new activation sequence.

The adaptive nature of CM was subsequently confirmed in both animal and human studies. Costard-Jäckle et al\(^12\) and later Sosunov et al\(^13\) in an isolated rabbit heart demonstrated the presence of an inverse relationship between local activation and repolarization times resulting in homogeneity of the activation-recovery intervals throughout the ventricle during normal activation. Initiation of ventricular pacing disrupted this relationship, but within 60 to 120 minutes of pacing, this relationship was re-established suggesting adaptation of the repolarization to the new activation pattern. After the reversal to normal conduction, it took another 60 minutes for repolarization to readjust to the now new old activation sequence.

In humans, 1 week of ventricular pacing resulted in significant QT interval shortening during continuous pacing. However, when normal conduction was restored and QRS became narrow, QT interval became prolonged compared with the baseline, indicating that the heart has adapted to the new activation sequence.\(^14\)-\(^15\) No evidence of hypertrophy, changes in myocardial blood flow, and hemodynamics were observed in the setting of CM after 21 days of ventricular pacing in dogs further suggesting the adaptive nature of this process.\(^16\)

Electrophysiology, Molecular Mechanisms, and Triggers of CM
Repolarization Gradients
The ST segment and T waves on the 12-lead ECG are concurrent with the repolarization phase of the cardiac cycle. The concordance of QRS and T wave in a normal human ECG for many years was explained by the presence of a transmural repolarization gradient resulting from a shorter epicardial action potential duration (APD) so that epicardial cells being depolarized last repolarize first. Not surprisingly such 1-dimensional explanation applied to a complex...
3-dimensional structure as the heart seemed oversimplified and was called into question.\textsuperscript{17} Multiple regional repolarization gradients (apical-basal, right-to-left ventricle) have been discovered and their relative contributions along with the transmural gradient to the normal T-wave morphology remain the subject of an ongoing debate.\textsuperscript{17,18}

Similar to the theories of normal T-wave morphology initially inverted T waves of CM were explained by the change in the transmural repolarization gradient because of preferential epicardial APD prolongation.\textsuperscript{16,19} Later, it was discovered that not only the transmural repolarization gradient in CM is site-specific,\textsuperscript{20} but also other gradients exist including apical-basal and right-to-left ventricle.\textsuperscript{21,22} There is a general agreement across the studies that CM is associated with prolongation of repolarization in the early activated areas. However, repolarization changes at the late activated sites were variable ranging from shortening,\textsuperscript{23} minimal, or no change\textsuperscript{20,22} to significant APD prolongation.\textsuperscript{24,25} Discrepancy in these findings is a subject of an ongoing debate and is likely related to the differences in experimental models, pacing site, species, and methods of measurement. In addition, novel factors influencing local APD, such as Purkinje fiber—myocyte junctions have been described and merit further investigation.\textsuperscript{26} From the clinical perspective, the human data on QT interval shortening during continuous ventricular pacing\textsuperscript{14,15} are most consistent with reciprocal changes in APD between early (prolongation) and late-activated (shortening) areas.

**Molecular Mechanisms**

Molecular mechanisms of CM have been extensively reviewed recently.\textsuperscript{27,28}

In brief, CM comprises a spectrum of 2 distinct but overlapping phenomena which differ in the mechanisms by which repolarization changes are achieved: short-term and long-term CM. Short-term CM observed within minutes of ventricular pacing is thought to occur from modulation and modification of existing proteins and channel trafficking. It is relatively short-lived and dissipates within minutes. Long-term CM is seen after longer periods of abnormal activation and is longer lasting (days to weeks). It includes changes in gene transcription and protein synthesis.

Molecular changes observed in the setting of CM include alterations of multiple ion channels, receptors, and cell coupling, including the transient outward current, Ito, IKr, ICa,\textsuperscript{27,28} Na/Ca exchanger,\textsuperscript{23} AT1 receptors, stretch-activated receptors,\textsuperscript{29} and gap junction redistribution.\textsuperscript{23,30}

**Triggers of CM**

Perhaps the most important discovery in the physiology of CM was establishing its relationship to the mechanical function of the heart. The notion that it is the change in ventricular contraction pattern and local ventricular wall stress (as opposed to electrotonic modulation originally proposed by M. Rosenbaum) that triggers CM was first made on the basis of CM inhibition by the renin-angiotensin system blockade.\textsuperscript{31} It was later confirmed in elegant experiments with excitation–contraction

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**Figure 1.** A, Development and partial resolution of cardiac memory (CM) in a chronic canine model of epicardial left ventricular pacing. Leads I, aVF, and frontal plane vectorcardiogram shown at baseline, during ventricular pacing, 1 hour after interruption of ventricular pacing for 7, 21, and 3 days after cessation of ventricular pacing. Leads I and aVF are shown (top). At baseline (left column), the T waves during sinus rhythm are positive in both leads. Left ventricular pacing (column 2) results in wide QRS with secondary T-wave abnormalities and discordant T waves. Pacing interruption after 7 and 21 days results in the development of progressive (accumulation) T-wave inversions during sinus rhythm that persist during day 3 of recovery. The polarity of the T waves corresponds to the polarity of the paced QRS complex. **Bottom**, The corresponding frontal plane vectorcardiograms are displayed demonstrating gradual alignment of the T-vector loop during sinus rhythm with the QRS loop during ventricular pacing. Note the increase in the T-vector amplitude and rotation toward the direction of the paced QRS vector (arrows). Cross-hair represents calibration at 1 mV. **B**, Quantitative assessment of CM. The baseline and memory T-vector loops superimposed. CM can be measured by the change in the T-vector direction, amplitude, and 3-dimensional distance (T peak displacement [TPD] measured in mV) between the T-vector peaks (T control represents the baseline T-vector loop and T memory represents the T-vector loop after CM induction). TPD is the preferred method of CM measurement. Adapted from Yu et al\textsuperscript{19} with permission of the publisher. Copyright © 1999, Wolters Kluwer Health. Adapted from Plotnikov et al\textsuperscript{32} with permission of the publisher. Copyright © 2001, European Society of Cardiology. Authorization for this adaptation has been obtained both from the owner of the copyright in the original work and from the owner of copyright in the translation or adaptation.
uncoupling demonstrating that pacing-induced CM only develops if the change in the electric activation sequence is accompanied by the active mechanical contraction, whereas CM-like changes can develop as the result of local mechanical myocardial stretching alone without electric activation change.13

We think this fundamental property of CM provides an opportunity to consider CM as a potential indicator of myocardial contractility disturbance in clinical practice, as will be shown later.

Role of Vectorcardiography in Evaluation of CM
As originally noted by Rosenbaum et al.,10 not only the T waves follow the direction of the wide QRS complex in each ECG lead, but also the whole T-wave 3-dimensional vector aligns with the wide QRS vector when assessed using vectorcardiography.14

This 3-dimensional nature of repolarization changes in CM made vectorcardiography a preferred tool of its assessment so that instead of tracking disparate T-wave polarities and amplitudes in individual leads the change in the whole T-wave vector can be observed and measured. The vectorcardiography-based measurement of CM, T-vector peak displacement was developed.15 It allowed to express CM magnitude as a single numeric value and track its changes under different experimental conditions (Figure 1). T-vector peak displacement has been used in several experimental and clinical studies further advancing CM principles on a new quantitative level.14,20,32

Clinical Applications of Cardiac Memory in Narrow QRS Rhythms

Why Was CM Initially Described Only During Normal Ventricular Activation
Traditionally, CM has been associated with TWI during sinus rhythm with narrow QRS for several reasons:

First, normal T waves during sinus rhythm are positive in most leads, whereas TWI is usually associated with pathological conditions (eg, ischemia, strain, cerebral hemorrhage). Therefore, T-wave changes are more likely to be noticed when TWI is present. Second, in the most common situations leading to CM development (LBBB, right ventricular apical pacing), wide QRS deflections initiating CM are negative in the majority of the leads and therefore produce TWI.

Third, large secondary repolarization changes during wide QRS rhythms mask CM until the QRS becomes narrow (Figure 2).

However, as both depolarization and repolarization of the heart occur in a 3-dimensional space, CM always produces a mixture of positive and negative T-wave changes based on the wide QRS vector polarity. Although positive T-wave changes are often overlooked, they are as important as TWI in diagnostic applications of CM as will be seen below.

Detecting the Presence and Localizing Intermittent Wide QRS Rhythms
CM signature consisting of the specific combinations of negative and positive T waves can give a clue to the presence and localization of intermittent wide QRS rhythms including pre-excitation, ventricular tachycardia, and conduction abnormalities.

Figure 3 demonstrates CM signature after ablation of a left posteroseptal bypass tract. Figure 3A shows baseline ECG with manifest pre-excitation. ECG in Figure 3B obtained 30 minutes after the bypass tract ablation demonstrates tall peaked T waves across the precordium as well as inferior TWI corresponding to the vector of the delta wave. Tall positive memory T waves in the right precordial leads can be easily confused with hyperkalemia or ischemia. ECG in Figure 3C obtained 1 week after ablation shows a significant decrease in the amplitude of both positive and negative T-wave deflections. ECG completely normalized in 6 months (not shown).

A year later the patient was again noted to have T-wave abnormalities on his routine ECG. Follow-up Holter monitoring showed intermittent pre-excitation without any arrhythmias. In this case, CM was the only sign of the partial recurrence of antegrade conduction over the bypass tract.

Another patient with structurally normal heart and frequent episodes of palpitations presented with T-wave abnormalities during sinus rhythm on his ECG (Figure 4, left). Based on the presence of the inferior TWI and tall peaked T waves in the right precordial leads in the absence of ischemia, one can suspect the presence of a wide QRS rhythm originating in the inferior septal aspect of the left ventricle. Indeed, the patient had verapamil-sensitive ventricular tachycardia (Figure 4, right).
CM signature of intermittent LBBB will be discussed in the following section along with right ventricular pacing.

CM-related T-wave changes after intermittent right bundle branch block have not been described. The likely reason for that is that the delayed activation of the right ventricle produces mostly positive deflections in the right precordial leads resulting in an increase in the positive T-wave amplitude that goes unnoticed.

**Distinguishing Between TWI From CM and Ischemia**

Since the time CM was first described, its similarity to ischemic TWI made it a significant confounder in the diagnosis of myocardial ischemia often resulting in excessive cardiac testing. In particular, precordial TWI because of intermittent right ventricular pacing or LBBB (by far the most common presentations of CM encountered in clinical practice) can be easily confused with the Wellens’ syndrome characteristic of transient proximal left anterior descending coronary artery occlusion, a condition requiring prompt coronary intervention.33

In general, the direction of the ischemic T-waves points away from the area of ischemia. Because the ischemic region attributable to the proximal left anterior descending coronary artery lesion involves the left ventricle, the ischemic precordial TWI has a rightward axis in the frontal plane and is characterized by TWI in leads I and aVL (Figure 5, left). The rare cases of right coronary artery ischemia producing precordial TWI with the leftward axis (Figure 5, middle) have deeper TWI in inferior leads than that in the precordial leads. However, right ventricular apical pacing and LBBB produce QRS vectors positive in leads I and aVL resulting in positive T waves in these leads on resumption of normal conduction consistent with CM principles. It also produces precordial TWI deeper than the inferior TWI (Figure 5, right).

The combination of

1. positive T in lead aVL and positive/isoelectric T in lead I; and
2. precordial TWI>inferior TWI produces a unique pacing-induced CM signature that was 92% sensitive and 100% specific in differentiating pacing-induced TWI from ischemia in a retrospective study.34

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**Figure 3.** Cardiac memory after ablation of a left posteroseptal accessory bypass tract. **A**, Before ablation. Left posteroseptal bypass tract is present. **B**, Thirty minutes after ablation. Note tall positive peaked T waves in the right precordial leads as well as inferior T-wave inversions. **C**, One week after ablation. Both positive and negative T waves decreased in amplitude but have not normalized completely.

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**Figure 4.** Cardiac memory (CM) in a setting of recurrent monomorphic ventricular tachycardia. **Left**, Inferolateral T-wave inversions (TWI lead III>TWI lead II) and unusually tall T waves in leads V2–V3 during sinus rhythm suggest CM due to intermittent wide QRS originating in the inferoseptal area of the left ventricle. **Right**, Spontaneous monomorphic ventricular tachycardia observed later in the same patient. **Bottom**, Rhythm strip (lead II) during tachycardia demonstrating capture beat.
Although not studied in larger prospective trials, these easily applicable criteria have been used successfully in clinical practice to distinguish between CM and ischemia.35 Because the axis during right ventricular pacing and LBBB is similar with positive QRS complex in leads I and aVL, the criteria can also be extended to the cases of TWI because of intermittent LBBB.36

One of the study limitations is its relatively small size. Although not observed in the original study, some distal left anterior descending coronary artery ischemia might not produce enough T-wave right axis deviation to cause negative T waves in leads I and aVL.

The other limitation in using this diagnostic tool is a concern that T-wave changes because of CM can counterbalance or obscure ischemic TWI when CM and ischemia occur simultaneously in the same patient. No systematic data are available in patients with T-wave abnormalities because of coronary artery disease but our preliminary results in this population indicate that CM does not reverse ischemic TWI minimizing this risk. Notably, when TWI in leads I and aVL are present at baseline as the result of the structural heart disease, T waves usually stay inverted, despite the development of CM (T vector never fully aligns with the paced QRS vector).

The proposed diagnostic criteria in the future can be further improved by the use of vectorcardiography techniques.

Cardiac Memory in Wide QRS Rhythms

Despite the fact that processes underlying CM do not develop suddenly on QRS normalization but rather progress gradually during abnormal ventricular activation, for a long time it was thought that CM cannot be detected until QRS becomes narrow. Secondary T-wave changes in wide QRS rhythms occur immediately and are dominant, whereas CM-induced T-wave changes are initially subtle and while gradually increasing over time are obscured by the secondary changes. The assessment of CM by surface ECG in the setting of wide QRS is limited because of the dominance of the secondary T-wave changes. Repolarization changes because of CM in this situation have been largely overlooked, being masked by the large discordant T waves. Nevertheless, vectorcardiogram can readily detect CM in this situation. Figure 6 presents the vectorcardiogram of the patient shown in Figure 2 demonstrating the 3-dimensional T-vector displacement after 7 days of right ventricular pacing during the narrow (AAI pacing) and wide (DDD pacing) QRS. The actual T-vector change (T peak displacement) in the narrow and wide QRS is nearly identical in both magnitude and direction.14 As seen on both Figures 2 and 6, CM in wide QRS rhythms presents as a decrease in T-vector magnitude without directional change (another reason why they are usually missed) in contrast to the narrow QRS rhythm, where T vector becomes larger and rotates toward the paced QRS. Using quantitative vectorcardiographic analysis, CM can be detected in paced rhythm within minutes after the onset of pacing.

Figure 5. T-wave inversions (TWI) because of ischemia and cardiac memory (CM). Left, Left anterior descending coronary artery (LAD) ischemia. Note: TWI in leads I and aVL. Middle, Dominant right coronary artery (RCA) ischemia. Although the TWI pattern is similar to CM because of the right ventricular pacing (positive T waves in leads I and aVL), the ratio of inferior/precordial TWI is different (maximal inferior TWI>maximal precordial TWI). Right, TWI because of CM. T waves in leads I and aVL are positive; precordial TWI>inferior TWI.

Figure 6. Vectorcardiogram of the patient in Figure 2 during AAI and DDD pacing in frontal (A) and transverse (B) projections before (baseline) and after the induction of cardiac memory (CM; day 7). On day 7 during AAI pacing, the T vector assumes the direction of the paced QRS complex while increasing in magnitude. At the same time in DDD mode, the T-vector magnitude decreases with no change in direction. Black arrows indicate the direction and magnitude of the projection of T-peak displacement as the result of CM and are similar in AAI and DDD modes. Sagittal plane (not shown) showed similar changes. Adapted from Shvilkin et al14 with permission of the publisher. Copyright © 2010, Elsevier. Authorization for this adaptation has been obtained both from the owner of the copyright in the original work and from the owner of copyright in the translation or adaptation.
Clinical Application of CM in Wide QRS: Age Determination of LBBB

The finding that CM decreases discordant T-wave amplitude in wide QRS rhythms has important clinical implications. For example, it can be used to determine whether LBBB is old or new which becomes valuable in the setting of chest pain.

For many years, American College of Cardiology/American Heart Association guidelines for the management of patients with ST-segment–elevation myocardial infarction considered new or presumed new LBBB, a class I indication for reperfusion therapy when associated with symptoms suggestive of ischemia creating a decision-making dilemma. Recently, this recommendation has been withdrawn largely because of the inability to determine the LBBB age which resulted in overly aggressive management of the chronic LBBB patients.

In clinical practice determining whether LBBB is actually new is virtually impossible as prior ECGs for comparison even if available are usually dated well beyond the relevant period of time (from the onset of symptoms which ranges from minutes to a few hours). LBBB in the vast majority of patients presenting with chest pain is old.

The criterion for LBBB age determination was developed on the premise that new LBBB would have larger T vector (and taller T waves on a 12-lead ECG) compared with the old one in accordance with CM development in wide QRS rhythm (Figure 7). As the duration of LBBB increases the discordant secondary T waves become smaller (Figure 7A). A retrospective analysis >1700 LBBB ECGs showed that indeed the new onset LBBB (defined as <24-hour duration) was rare (3%) and had significantly larger T waves (as well as smaller QRS vector magnitude) compared with the chronic LBBB (Figure 7B and 7C). Although the best results in LBBB age determination were achieved using quantitative vectorcardiography technique, a simplified 12-lead ECG criterion using the ratio of the maximal precordial S wave/maximal precordial T-wave amplitude (as approximations of the QRS and T vectors, respectively) was also developed. A conservative cutoff of S/T<2.5 allowed to detect 100% of the new LBBBs.

The T-vector magnitude in LBBB changes significantly during the first 24 hours. In cases of painful LBBB syndrome when ECG is recorded within seconds/minutes of symptoms onset (often during an exercise stress test), S/T ratio can be as low as 1.4. The majority of the T-wave changes occur within the first 24 hours of LBBB persistence when it assumes chronic QRST configuration. In the true chronic LBBB, the S/T ratio is close to ≥3.0 (Figure 7A and 7C). It is important to recognize that LBBB is often a dynamic phenomenon and can be intermittent or rate-dependent. Repeated episodes of intermittent LBBB can

Figure 7. Use of the maximal precordial S/T ratio in left bundle branch block (LBBB) age determination. S/T represents the ratio of the maximal precordial S wave/maximal precordial T-wave amplitude. A, As the duration of LBBB increases, the maximal precordial S/T ratio increases from 1.64 at 6 hours (=new) to 3.22 at 15 days (=old). B, A typical example of resolution of a new LBBB (<8-hour duration). S/T ratio is 1.61. Resolution of LBBB results in no apparent T-wave abnormalities during narrow QRS. C, A typical example of resolution of an old LBBB (>3-day duration). S/T ratio is 2.93. Typical changes of cardiac memory with precordial TWI are evident during narrow QRS. D, A new onset LBBB (<75 minutes) in a setting of an acute left anterior descending coronary artery thrombosis. S/T ratio is 1.68 consistent with the new LBBB. Note ST segment changes in the baseline tracing consistent with ischemia and loss of R waves after resolution of LBBB. Adapted from Shvilkin et al with permission of the publisher. Copyright © 2010, Elsevier. Authorization for this adaptation has been obtained both from the owner of the copyright in the original work and from the owner of copyright in the translation or adaptation.

Figure 8. Correlation between cardiac memory (CM) magnitude (measured during DDD pacing) and the left ventricular dysynchrony (difference in time to the maximal longitudinal strain between septal and lateral left ventricular walls). With the increase in dysynchrony CM magnitude decreases. Adapted from Shvilkin with permission of the publisher. Copyright © 2013, Springer. Authorization for this adaptation has been obtained both from the owner of the copyright in the original work and from the owner of copyright in the translation or adaptation.
cause accumulation of CM-related T-wave changes, sometimes making the distinction between new and old LBBB difficult.

The new LBBB S/T criterion held true in a small subgroup of patients with acute coronary syndrome (Figure 7D) but needs to be confirmed in larger clinical trials.

Using this criterion in low-risk patients can help justify conservative management and avoid unnecessary coronary interventions.

Cardiac Memory in the Setting of Biventricular Pacing
Dynamic T-wave changes have been reported in the setting of biventricular pacing. This situation is of a particular interest as it demonstrates that the CM can develop on top of already existing CM-induced repolarization changes and the heart is capable of relearning. Whether the new T waves follow the biventricular or left ventricular paced QRS direction within the already existing CM-induced repolarization changes and the heart is capable of relearning. Whether the new T waves follow the biventricular or left ventricular paced QRS direction as well as clinical significance of these changes is the subject of further research.

Through a Looking Glass: Cardiac Memory as a Reflection of Ventricular Function
As discussed above, CM is initiated by the changes in local myocardial contractility. Understandably, a question arises whether CM magnitude can be used as a reflection of pacing-induced contractile pattern dysfunction, and whether wide variation of pacing-induced CM magnitude between individuals (approximately 8-fold) can be explained by the differences in the left ventricular contraction pattern during ventricular pacing. If true, this can open an opportunity to assess the risk of pacing-induced ventricular dysfunction based on ECG analysis. Indeed, the preliminary data suggest that pacing-induced CM magnitude is tightly correlated with the synchrony of left ventricular contraction during right ventricular pacing. Patients with more pronounced septal to lateral wall dysynchrony developed less CM (Figure 8). The dysynchrony was associated with accelerated septal contraction (septal flash) resulting in the smaller stroke volume.

To further extend this concept given the positive correlation between CM magnitude and QRS/T ratio at the onset of ventricular pacing, wide complex QRST morphology (and in particular QRS/T vector magnitude ratio) potentially can be viewed as the reflection of the ventricular contractile disturbance and used for risk assessment of dysynchronous heart failure.

Of further interest, recently vectorcardiography-derived T-wave area in LBBB was found to be predictive of response to cardiac resynchronization. Further studies are needed to confirm relationships between wide QRST morphology and contractile function of the heart.

Conclusions
Cardiac memory is a universal adaptive property of the heart reflecting the adjustment of repolarization to the new activation sequence and manifesting by T-wave changes. CM can be detected during both narrow and wide QRS rhythms, and the knowledge of its features improves ECG diagnosis. Further research in CM and wide QRST morphology can provide insight into mechanical function of the heart and potentially be used in risk assessment of dysynchronous heart failure and its correction.

Disclosures
None.

References


**Key Words:** bundle-branch block ■ electrocardiography ■ vectorcardiography ■ ventricular remodeling
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_Circ Arrhythm Electrophysiol._ 2015;8:475-482
doi: 10.1161/CIRCEP.115.002778

_Circulation: Arrhythmia and Electrophysiology_ is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
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Print ISSN: 1941-3149. Online ISSN: 1941-3084

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