HISTORICAL REVIEW

Elucidation of the spatial ventricular gradient and its link with dispersion of repolarization

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The ventricular gradient, a notion conceived by Wilson et al during the 1930s, has contributed considerably to a better understanding of the ECG manifestations of the cardiac repolarization process. The power of the ventricular gradient is its ability to assess the primary factors that contribute to the T wave (i.e., heterogeneity of action potential morphology throughout the ventricles) in the presence of secondary factors contributing to the T wave (i.e., heterogeneity in ventricular depolarization instants). Where T-wave morphology is an ECG expression of heterogeneity of the repolarization, the ventricular gradient discriminates between primary or secondary causes of such heterogeneity. Besides the spatial ventricular gradient (Burger's three-dimensional elaboration of Wilson's two-dimensional concept), body surface mapping of local components of the ventricular gradient has emerged as a technique for assessing local ventricular action potential duration heterogeneity. The latter is believed to contribute to localization of arrhythmogenic areas in the heart. The spatial ventricular gradient, which can be computed on the basis of a regular routine ECG and does not require body surface mapping, aims to assess the overall heterogeneity of ventricular action potential morphology. This review addresses the nature and diagnostic potential of the spatial ventricular gradient. The main focus is the role of the spatial ventricular gradient in ECG assessment of dispersion of repolarization, a key factor in arrhythmogeneity.

KEYWORDS Action potentials; Repolarization; Dispersion; Primary T wave; Secondary T wave; QRST integral; QRS axis; T axis; QRS-T spatial angle

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Introduction

During the evolution of electrocardiography, several ECG parameters arose that initially enjoyed increasing popularity but were later were abandoned because of severe criticisms or lack of understanding. One such conceptually complex parameter is the ventricular gradient, conceived by Wilson et al[1,2] in the 1930s. The ventricular gradient never entered routine electrocardiography, although it still figures in leading textbooks.

Several review articles about the ventricular gradient exist in the literature[3–5]; however, none focuses on use of the spatial ventricular gradient for assessing ventricular dispersion of repolarization, which is mechanistically linked to arrhythmogenesis. Dispersion of repolarization of a given heartbeat, electrocardiographically reflected in its T wave, arises from superimposition of (1) the heterogeneity, throughout the ventricles, of the action potential morphologies for that given heartbeat; and (2) the heterogeneity of the ventricular depolarization instants as they result from the conducted impulse that gave rise to the beat under consideration. Theoretically, a pure primary or secondary T wave would result when all ventricular myocardium was excited at the same time instant or when all ventricular action potentials had identical shapes, respectively. In practice, both primary and secondary factors contribute to the T wave, and these contributions cannot be unraveled by T-wave analysis alone. The power of the ventricular gradient is its ability to assess heterogeneity of the ventricular action potential morphology independent of secondary factors.

Although reentrant spiral-based tachyarrhythmias can be initiated in conditions of homogeneous action potential morphology,[6] multiple tachyarrhythmias potentially deteriorating into ventricular fibrillation appear in a situation of increased heterogeneity of action potential morphology.[7] Moreover, reducing action potential morphology heterogeneity has an antiarrhythmic effect.[8] Therefore, measurement of the ventricular gradient may considerably contribute to experimental and clinical arrhythmology.

Origin of the concept of the ventricular gradient

In 1933, Wilson et al[9] published a paper in which, after identifying the transmembrane potential as the source of the ECG, forward calculations were given for the potential distribution around a muscle fiber. Based on these principles, the same group reasoned that the QRST integral in an ECG lead (total area under the curve over the QT interval; parts with positive deflections in the ECG are counted as positive, parts with negative deflections are counted as negative) depends solely on the heterogeneity of the action potential durations (APDs) in the muscle fiber ("local variations in the excitatory process") and not on the order in which the muscle cells are activated.[1,2]

The postulate of Wilson et al regarding the properties of the QRST integral was experimentally reaffirmed many

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years later, first by Gardberg et al[10] in an isolated muscle strip and later in the complete heart by Abildskov et al.[11] Wilson's "ventricular gradient", as the QRST integral was termed since its introduction,[1] was, from the beginning, recognized as a potential ECG tool for discriminating between primary and secondary T-wave phenomena.[12]

Vectorial approach: The spatial ventricular gradient

The representation of the momentaneous electrical forces generated by the heart as a single dipole of which strength and direction are expressed in a vector of given magnitude and direction dates back to Waller.[13] Vectorcardiography emerged from these principles and aims to construct the heart vector from an assembly of ECG limb and chest leads.[14]

In the vectorial approach, the scalar definition of the QRST integral according to Wilson (i.e., the area under the QRST curve in a given ECG lead) is generalized as the spatial integral of the area formed by the moving spatial heart vector during the QRST interval. Consequently, the latter integral is a vector in three-dimensional geometric space, of which the magnitude, as in the scalar approach of the ventricular gradient, has the unit voltage · time.

The first report of the measurement of a spatial ventricular gradient appeared in 1954.[15] In 1957, Burger[16] published the analytical proof that this vectorial QRST integral, which he referred to as the "spatial ventricular gradient", is proportional to the volume integral of the APD gradient over the heart. This analysis established the spatial ventricular gradient as an index for APD heterogeneity throughout the heart.

Direction of the ventricular gradient

In his mathematical analysis, Burger[16] demonstrated that the maximum diastolic potential of cardiac myocytes figures as a proportionality constant in the equation for the integral of the heart vector over the QRST interval. As the diastolic transmembrane potential is negative, this inverts the direction of the gradient that, contrary to intuition, points to the briefest APDs in the heart instead of the longest APDs.[1] This concept has been verified experimentally.[17]

QRST integral in one ECG lead; QRST integral maps

In 1979, theoretical elaboration of the ventricular gradient concept by Plonsey[18] showed that the (scalar) ventricular gradient in a specific ECG or epicardial lead is obtained by weighting (vectorially multiplying) the APD gradient by the vector lead field during volume integration. The more a given lead is sensitive for the electrical activity in a particular area of the heart, the more the ventricular gradient in such a lead expresses the local properties of that part of the heart. In this review, we concentrate on the vectorial approach of the ventricular gradient ("spatial ventricular gradient"), which yields one single vector of given size and direction for one heartbeat of a given person. The lead-dependent approach of the ventricular gradient in body surface mapping may be useful for finding localized inhomogeneities in the heart. By definition, the spatial ventricular gradient, which can be derived from the 12-lead ECG, cannot discern between global and local phenomena because it lumps all APD inhomogeneity in the heart into one single integral.

Ventricular gradient as an index for heterogeneity of action potential morphology

In 1983, Geselowitz[19] theoretically proved that the QRST integral is determined by spatial heterogeneity in the area under the action potential rather than by heterogeneity in APD alone. Hence, heterogeneity in action potential resting amplitudes, peak amplitudes, upslopes, downslopes, and durations all contribute to the ventricular gradient. This somewhat more generalized concept casts the ventricular gradient into an index of heterogeneity of action potential morphology in the ventricles of the heart.

Cancellation and the ventricular gradient

Cancellation of electromotive forces in the heart greatly reduces body surface potentials and ECG amplitudes. An estimated 75% of the electrical energy is canceled during ventricular depolarization; during repolarization this estimated percentage is 92% to 99%.[20] In a similar way, action potential morphology gradients in different sites of the heart may have opposing directions and cancel out during integration.[21] This might explain why, in computer simulations using a model of the heart consisting of elementary cubic units, QRST integrals decreased when APDs were reassigned in a random selection of these units.[22] Notably, random APD assignment made the model more susceptible to the initiation of ventricular fibrillation. Obviously, this artificial mathematical manipulation dissociates the changes in the local APD gradients (increase due to random redistribution) from the changes in the ventricular gradient (decrease due to cancellation).

APD heterogeneity under physiologic and pathophysiologic conditions

The ventricular gradient integrates gradients in the action potential morphology throughout the heart; one major aspect of action potential morphology is APD. Multiple studies have searched for the existence of transmural, apicobasal, and left ventricular–right ventricular APD gradients. An extensive review by Burton and Cobbe[23] on this topic has been published. The results of these studies are sometimes conflicting, most likely due to differences in species, methodology, terminology, and interpretation.[24] With these partly conflicting results, it is not possible to make a fair educated guess of the normal size and direction of the ventricular gradient in animals or in man. Moreover, attempts to establish normal values of the ventricular gradient in man have revealed a considerable variability between subjects.[25] Hence, it may be better to longitudinally measure intra-individual trends in the ventricular gradient than to transversally measure interindividual differences in the ventricular gradient.

During ischemia/infarction, APD gradients in the heart change dynamically due to dynamic APD changes in the ischemic area. During a brief initial period, ischemia induces APD prolongation[26]; thereafter APD shortens. Shortening is more pronounced in ischemic epicardial areas than in ischemic endocardial areas.[27,28] In remodeled hypertrophied hearts after myocardial infarction, APDs in affected areas are prolonged.[29]

In a biologic model (canine arterially perfused left ventricular wedge preparation) of the congenital long QT syndrome types 1, 2 and 3, transmural APD gradients are accentuated by preferential prolongation of M-cell APDs.[30]

In the Brugada syndrome, action potential morphology heterogeneity is increased by heterogeneous early phase 1 action potential notch accentuation and heterogeneous loss of APD dome and APD prolongation in the right ventricular epicardium.[31]

Data from a wedge model of short QT syndrome suggest that in this disease, APD gradients are enhanced by preferential endocardial and M-cell APD abbreviation.[32]

Body surface maps of patients with Wolff-Parkinson-White (WPW) syndrome show longer activation-recovery intervals over the preexcited area, suggesting longer APDs in the preexcited area. This explains why the direction of the spatial ventricular gradient differs between patients with left or with right accessory pathways. Preexcitation-induced APD changes in patients with WPW syndrome may occur due to cardiac memory.[33]

Ventricular gradient and the T wave

The T wave in the ECG reflects the heterogeneity of repolarization throughout the ventricles.[4] In 1964, Van Dam and Durrer[17] attempted to simultaneously activate canine ventricles by high-intensity electrical stimuli. The T wave thus obtained approximates the hypothetical T wave that depends only on action potential morphology heterogeneity throughout the heart and that does not depend on the ventricular depolarization sequence. In 1971, this hypothetical T wave was called "the primary T wave" by Abildskov et al.[34] In both publications it was mentioned that the area under the primary T wave equals the ventricular gradient. Abildskov et al[34] also defined the hypothetical "secondary T wave" (the T wave that arises on the basis of depolarization heterogeneity only, in the absence of any action potential morphology heterogeneity). The T wave that is measured in a regular ECG originates from the resultant of these primary and secondary factors.

Secondary T-wave changes and the ventricular gradient

Altered ventricular activation sequences (e.g., occurring with extrasystoles, intermittent bundle branch block, and cardiac pacing) cause wide QRS complexes and secondary T-wave changes. With unaltered action potential morphology distribution, the ventricular gradient should be insensitive to such changes. However, since the introduction of the ventricular gradient, it has been observed that extrasystoles appear to change the ventricular gradient.[4] Wilson et al[2] had already reported this phenomenon and ascribed it to measurement errors, physiologic variability due to respiration, and an altered mechanical contraction pattern due to alterations in the activation pathway. Since then, several mechanisms have been identified that contribute to altered action potential morphology distribution throughout the heart when it is activated in an abnormal order, including altered electrotonic influences,[35] altered cellular electrophysiologic properties due to premature excitation,[36] and cardiac memory.

T-wave memory and the ventricular gradient

In a classic publication, Rosenbaum et al[37] described how, after several hours of right ventricular pacing, the T waves of normally conducted sinus beats remain altered with respect to the sinus beats prior to pacing. This T-wave memory lasted for several days. Also, intermittent bundle branch block was demonstrated to induce changes in the T waves of normally conducted beats. They reported that in lead V_2, the ventricular gradient of the paced beats changed as T-wave memory developed. Taken together, these observations proved that altered secondary conditions that are present for a relatively short time can induce primary changes that persist for a (much) longer time. Since this seminal study, multiple mechanisms for long-term and short-term cardiac memory have been revealed, such as altered gap junction distribution, altered I_{to} and I_{Kr} distribution, alterations in $I_{Ca,L}$, and altered electrophysiologic responses to antiarrhythmic agents.[38] The ventricular gradient would be an appropriate ECG parameter for keeping track of such dynamic changes in action potential morphology distribution throughout the heart.

Repolarization heterogeneity, reentrant arrhythmias, and the ventricular gradient

Heterogeneity of the repolarization is associated with risk of life-threatening cardiac arrhythmias, as it is functionally linked to dispersion of refractoriness,[39] which facilitates reentrant ventricular tachycardias.[40]

Spach and Barr[41] measured the full activation and repolarization sequence by multiple epicardial and transmural electrodes in recovered closed chest dogs, selected based on having normal upright T waves in lead II. They found that the epicardium repolarized first although it depolarized last. Results of the classic mapping study in human hearts by Franz et al[42] are in agreement with the findings of Spach and Barr,[41] showing, when pooling the results for all endocardial and epicardial mapping sites, a negative linear relationship between activation time and APD.[42] This inverse relation between activation time and APD would contribute to a relative stabilization of repolarization.[42] Hence, in the normal heart (normal electrophysiologic matrix, normal activation sequence), the ventricular gradient reflects a normal degree of APD heterogeneity that compensates for a normal degree of activation time heterogeneity.

Abildskov et al[3] suggested that the QRST area was among the promising ECG parameters for reentry risk assessment, and several subsequent articles showed that the QRST area distribution over the body surface indeed correlated with arrhythmia vulnerability. In this approach, arrhythmia risk was conceptually coupled to altered primary factors, that is, altered local APD heterogeneity reflected in an altered local lead dependent ventricular gradient. Obviously, increased dispersion of repolarization, caused by altered APD dispersion and likely reflected in an altered ventricular gradient, is not sufficient for arrhythmia occurrence. A second condition, the occurrence of an appropriate triggering stimulus, such as an ectopic beat, also must be fulfilled.

Relation between the spatial ventricular gradient and the QRS and T axes

It follows from the mathematical definition and from calculus[2,16] that the spatial ventricular gradient (spatial QRST integral) is simply and straightforwardly the vectorial sum of the vectorial QRS integral and the vectorial T integral. By definition, the QRS and the T integrals assume the same direction (not the same magnitude) as do the QRS and T axes. In humans, the QRS axis usually points slightly posterior, inferior, and to the left, and the T axis usually points anterior and also inferior and to the left. Consequently, the ventricular gradient points usually anterior, inferior, and to the left, roughly along the long axis of the heart, in the direction of the apex (Figure 1).[25] As the ventricular gradient points in the direction of the briefer action potentials, it can be deduced that corresponding apicobasal and/or endocardial-to-epicardial action potential morphology trends (briefest action potentials apically/epicardially) must necessarily exist.[1]

Relation between spatial ventricular gradient, QRS and T integrals, and the QRS-T angle

Van Oosterom[43] provided important insight into the ECG deflections caused by the ventricular depolarization process, action potential morphology heterogeneity, repolarization process, and their relationship. Van Oosterom, after endorsing Burger's mathematical proof[16] that the integral of the heart vector over the QRST interval is proportional to the volume integral of the heterogeneity of APDs, demonstrates that the integral of the heart vector during depolarization (spatial QRS integral) depends on the heterogeneity of the activation instants, whereas the integral of the heart vector during repolarization (spatial T-wave integral) is proportional to the dispersion of repolarization (provided QRS and T do not overlap).

This analysis clearly establishes the electrocardiographic relationship between primary factors (APD dispersion, measured in the ECG as the ventricular gradient), secondary factors (dispersion in activation instants, measured in the ECG as the QRS integral), and the resulting dispersion of repolarization (measured in the ECG as the T-wave integral). Of note, in electrocardiography, dispersion in ventricular activation currently is uniquely assessed by QRS duration, thus neglecting QRS amplitude. However, to electrocardiographically assess the repolarization process, both T-wave amplitude and T-wave area (mostly scalar rather than vectorial) are being used as indices of dispersion of repolarization.[44]

A situation with increased QRS and T integrals is not necessarily associated with a large ventricular gradient. In a normal heart, pure secondary changes (e.g., altered intraventricular conduction sequence with ventricular ectopy) yield wide and bizarre QRS complexes and T waves with large QRS and T integrals. However, in this case the angle between the QRS and T axes will be large (the ECG will be discordant), and the ventricular gradient, which is the vectorial sum of the QRS and T integrals, remains unchanged (no primary changes).

The spatial QRS-T angle, which increases by secondary changes,[45] has proven to be an important prognostic ECG index.[46] Possibly, combining the spatial QRS-T angle with the ventricular gradient could still further increase this prognostic value, as this would add information about the absence or presence of primary changes. With pure secondary changes, only the spatial QRS-T angle would be enlarged, while the ventricular gradient remains unchanged. With additional primary changes (a less favorable condition), the ventricular gradient would be enlarged as well.

Illustrative simulated ECGs

To illustrate the concepts mentioned, we generated five distinct ECGs with ECGSIM,[47] an *in silico* model for ECG genesis (Figure 2). Subsequently, we analyzed these five ECGs using LEADS, our research-oriented ECG–VCG analysis program.[48] This program derives a vectorcardiogram (VCG) from a conventional 12-lead ECG by using a VCG reconstruction matrix.[49]

The ECG in Figure 2A was generated by using the ECGSIM default parameter setting. It is a normal ECG, with a normal ventricular depolarization sequence and normal APD heterogeneity. It is concordant in most leads and has a QRS-T angle of 70°. This is well below the upper normal value of 105°.[25]

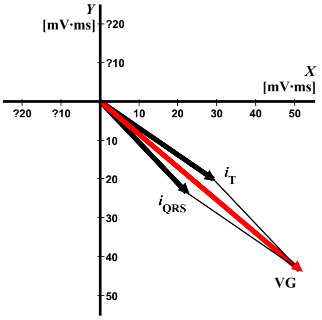

Figure 1 Spatial QRS integral (i_{QRS}), T integral (i_T), and ventricular gradient (VG, the QRST integral) for a normal ECG and projected in the frontal plane. The QRS and T integrals have the same directions as the QRS and T axes, respectively. The ventricular gradient is the vectorial sum of the QRS integral and the T integral.

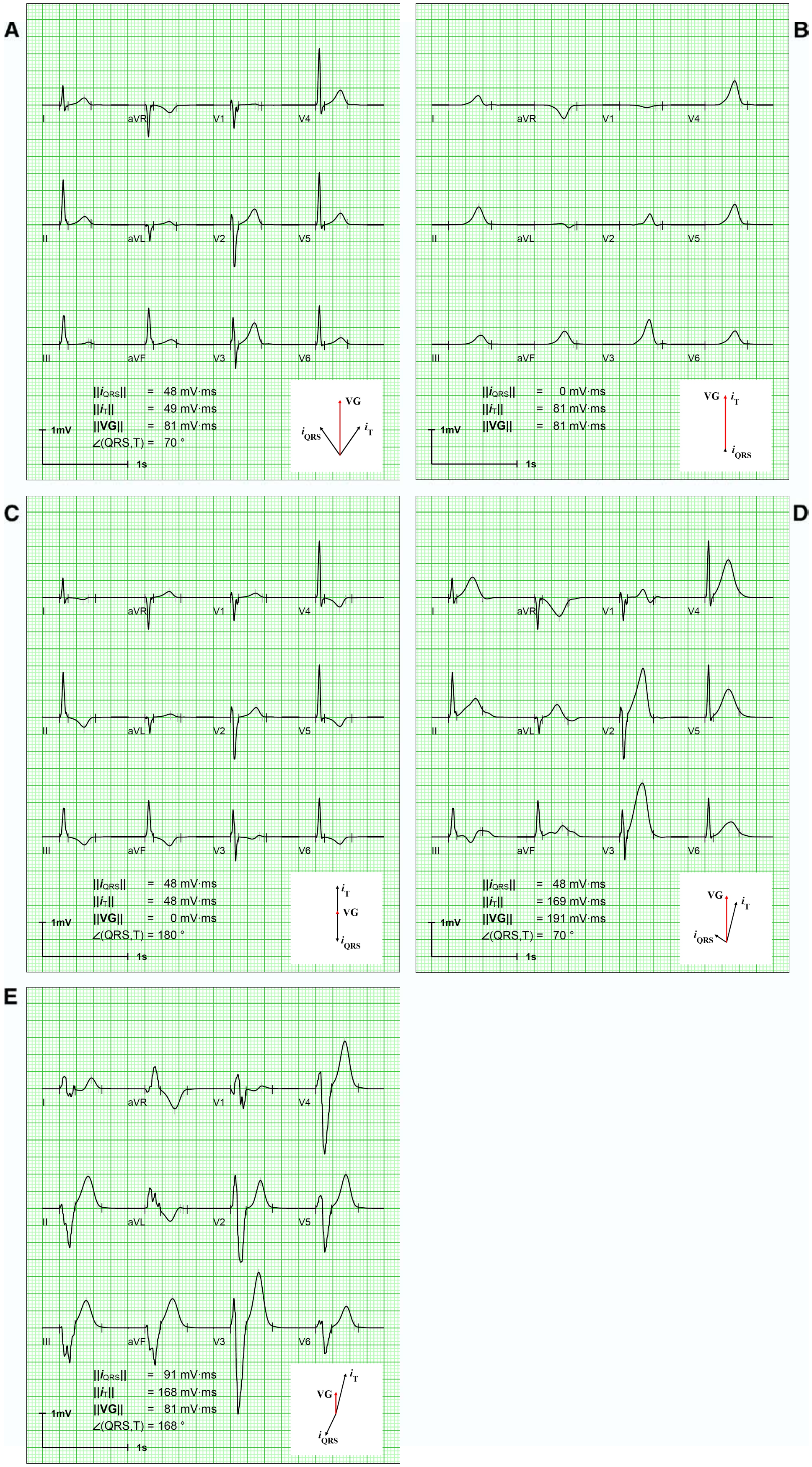

Figure 2 ECGs simulated with variations of heterogeneity in ventricular activation timing and action potential duration. During all simulations, maximal diastolic potentials and mean action potential duration were kept unaltered from the ECGSIM default situation, which yields a normal ECG **(A).** To generate the primary T wave **(B),** all ventricular depolarization instants were set at zero time while maintaining the normal action potential duration heterogeneity. The secondary T wave **(C)** was obtained by using the normal ventricular depolarization sequence and assigning all action potential durations the mean action potential duration. An ECG with increased dispersion of repolarization **(D)** was accomplished by using the normal ventricular depolarization sequence and by increasing action potential duration heterogeneity without changing the mean. Finally, a right ventricular-paced complex **(E)** was simulated by changing the ventricular depolarization order while maintaining normal action potential durations. Onset and end QRS and end T instants are indicated in the ECG leads by *vertical lines.* As ECGSIM models ventricular electrical activity, no P waves are present. *Insets* depict the corresponding QRS and T integrals and the ventricular gradient denoted as I_{QRS}, I_T, and ventricular gradient (VG). All pictures were made in the spatial plane set up by the QRS- and T-integral vectors. The ventricular gradient was always plotted vertically, the QRS integral pointing to the left, and the T integral pointing to the right. The QRS-T spatial angle and the angles of the QRS integral and the T integral with the ventricular gradient are faithfully depicted in the figures, as there is no projection distortion. The ratio of the vector magnitudes is also faithfully depicted; however, for visualization purposes, scaling was adjusted per inset. See text for details.

The ECG in Figure 2B differs from that in Figure 2A in that it was generated without dispersion in the ventricular depolarization instants: all myocardial cells depolarize at the same moment in time. APD heterogeneity is still normal (default) in this simulation. As a consequence, the ECG has no QRS complex and consists solely of the primary T wave. With absent QRS complex, the T integral and the ventricular gradient by definition have the same magnitude and direction.

The ECG in Figure 2C was simulated on the basis of the normal default ventricular depolarization sequence, but here all APDs have the same magnitude (average of the normal default APD distribution). As a consequence, the T wave is purely of a secondary nature. The ventricular gradient has magnitude zero, as there is no APD heterogeneity. Logically, the QRS and T integrals have equal magnitudes and opposite directions.

The ECG in Figure 2D was generated with a normal default ventricular depolarization sequence but with accentuated APD heterogeneity by proportionally increasing the longer APDs and decreasing the shorter APDs while maintaining the normal default average APD.[47] It has an almost normal QRS-T angle, as the gross APD distribution pattern throughout the ventricles was maintained. However, the ventricular gradient and the T integral are larger than normal due to increased APD heterogeneity and increased dispersion of repolarization, respectively.

The ECG in Figure 2E is a simulation of right ventricular pacing (by changing the ventricular depolarization sequence accordingly while maintaining the normal default APD distribution). Hence, it reflects the consequences of pure secondary changes. As discussed earlier, the spatial QRS-T angle is wide. Both the QRS and T integrals are larger because of a greater disparity of activation and repolarization. Because there are no APD changes, the ventricular gradient remains the same as normal.

Although dispersion of repolarization is increased in the ECGs depicted in Figures 2D and 2E, the causes of this increased dispersion differ completely (see also Figure 1 in the review article by Burton and Cobbe[23]). In Figure 2E, the increase in dispersion of repolarization is caused by a gross transventricular repolarization front that grossly follows the depolarization sequence; such a repolarization pattern possibly is not arrhythmogenic per se.[45] In contrast, in Figure 2D there is fragmented repolarization throughout the ventricles, which may render a substrate more vulnerable to triggers that may induce reentrant arrhythmias.

Potential diagnostic value of the ventricular gradient

As elaborated earlier, changes in the ventricular gradient are caused by primary changes in the electrophysiologic matrix, whereas secondary changes have no direct effect on the ventricular gradient. This characteristic of the ventricular gradient is potentially useful in ECG diagnostics in the presence of abnormal intraventricular conduction, for example, in the diagnosis of ischemic heart disease in the presence of preexistent right or left bundle branch block. The diagnosis of ischemia based on 12-lead ECG is difficult in these cases because conduction disturbances and ischemia may cause similar ECG changes. However, in a study by Goldman and Pipberger,[50] the ventricular gradient did not perform well compared with other ECG parameters in the diagnosis of myocardial infarction in the presence of ventricular conduction defects. This could be due to the large range in normal values of the ventricular gradient.[25] Also in their 1969 article, Goldman and Pipberger stressed the potential additional value of serial ECG analysis (comparison of the suspect ECG with a previous nonsuspect ECG of the same patient) for this diagnostic purpose.

The ventricular gradient could be useful in detecting primary electrical disease, for example, the sharp APD gradients in the right ventricle that occur in the Brugada syndrome[31] likely would increase the ventricular gradient magnitude. The Brugada syndrome has multiple ECG manifestations, of which the "coved-type" T-wave morphology in the right precordial leads is considered specific and the "saddleback-type" T-wave morphology is suspicious. In addition, intermittencies in its ECG expression are seen. Whether or not the ventricular gradient magnitude is elevated in the typical Brugada ECG and remains elevated in suspicious and intermittent episodes requires further study. With increasing knowledge about primary electrical diseases and their ECG manifestations, it is reasonable to surmise that the spatial ventricular gradient will be of diagnostic and prognostic value.

More general use of the ventricular gradient

Whether the ventricular gradient is useful in the setting of ECG diagnosis has not been validated. Additionally, and possibly more importantly, the spatial ventricular gradient is, like heart rate, QRS duration, QT interval, and spatial QRS-T angle, a general descriptor of the ECG. As an overall measure of action potential morphology heterogeneity in the heart, the ventricular gradient offers unique information about the ECG in healthy and diseased substrates under various conditions.

A novel general ECG descriptor, called "total cosine between R and T" and claimed to be based on the concept of the ventricular gradient, was shown to have prognostic value in postmyocardial infarction patients.[51] This promising result awaits further confirmation by a study comparing the results based on total cosine between R and T with results based on ventricular gradient and in which the explicitly formulated mathematical relation of total cosine between R and T with the ventricular gradient is given.

Conclusion

The spatial ventricular gradient represents the lumped heterogeneity of action potential morphology throughout the ventricles. It is, like multiple ECG parameters, sensitive to cancellation. In the normal heart, the ventricular gradient expresses a certain amount of physiologic APD heterogeneity that tends to compensate for heterogeneity in the depolarization instants of the ventricular myocytes. From this point of view, a decrease as well as an increase in the ventricular gradient represents a less effective compensation, thus increasing the amount of dispersion of repolarization and, in the presence of triggering events, increasing arrhythmogeneity.

The potential diagnostic value of the ventricular gradient is to be found in the ECG diagnosis of ischemia in the presence of preexistent conduction defects and in variable manifestations of primary electrical disease. Additionally, the ventricular gradient can be regarded as a general parameter that characterizes the primary component of dispersion of repolarization. As such, the ventricular gradient could be useful in risk assessment. To our knowledge, follow-up studies on its predictive value for ventricular arrhythmias in known arrhythmogenic substrates have not been performed.

Because the direction and magnitude of the spatial ventricular gradient can be computed from any routine clinical 12-lead ECG, its measurement is much less cumbersome in comparison to the determination of, for example, QRST integrals in body surface potential maps. In the setting of routine clinical electrocardiography, this advantage of the spatial ventricular gradient probably outweighs the disadvantage that it does not directly localize the myocardial source of disparity of action potential morphology. Of course, changes in the ventricular gradient are detected most effectively by serial ECG analysis.

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