Review Article

The Spatial Ventricular Gradient and its link with Dispersion of the Repolarization

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Abstract

The ventricular gradient, a notion conceived by Wilson and co-workers during the 1930s, has considerably contributed to a better understanding of the electrocardiographic 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 electrocardiographic 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 3D elaboration of the 2D concept of Wilson) body-surface mapping of local components of the ventricular gradient has emerged as a technique to assess local ventricular action potential duration heterogeneity. The latter is believed to contribute to the 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 overall heterogeneity of ventricular action potential morphology. This review addresses merely the nature and diagnostic potential of the spatial ventricular gradient. Main focus is on its role in electrocardiographically assessing dispersion of the 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.

Abbreviations:

Electrocardiogram	ECG
Ventricular gradient	VG
Action potential duration	APD
Wolff-Parkinson-White syndrome	WPW

Introduction

During the evolution of electrocardiography several new electrocardiographic parameters arose that initially enjoyed increasing popularity while they were later more or less abandoned because of severe criticisms or lack of understanding. One such a conceptually complex parameter is the ventricular gradient (VG), conceived by Wilson and coworkers in the nineteenthirties.[1,2] The VG never entered routine electrocardiography, although it is still figuring in leading textbooks.

Several review articles about the VG exist in the literature,[3-5] however, none of these focuses on the use of the spatial VG in regard to assessing ventricular dispersion of the repolarization, which is mechanistically linked to arrhythmogenesis. Dispersion of the 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 heart beat, 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 were excited at the same time instant, or when all ventricular action potentials would have 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 VG 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 VG may considerably contribute to experimental and clinical arrhythmology.

Origin of the concept of the ventricular gradient

In 1933, Wilson and colleagues published a paper in which, after identifying the transmembrane potential as the source of the electrocardiogram (ECG), forward calculations were given for the potential distribution around a muscle fiber.[9] Based on these principles, the same group reasoned that the QRST integral in an ECG lead (the total area under the curve over the QT interval; parts with positive deflections in the ECG are counted positive, parts with negative deflections negative) depends solely on the heterogeneity of the action potential durations (APD) 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 and colleagues regarding the properties of the QRST integral was many years later experimentally reaffirmed, first by Gardberg and colleagues in an isolated muscle strip,[10] and later in the complete heart by Abildskov and co-workers.[11] Wilson's "ventricular gradient", as the QRST integral was termed since its introduction[1] was, from the beginning on, recognized as a potential electrocardiographic tool to discriminate between primary and secondary T wave phenomena.[12]

Vectorial approach: the spatial VG

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 electrocardiographic 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 geometrical space, of which the magnitude, like in the scalar approach of the VG, has the unit ***voltage· time***.

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

Direction of the VG

In his mathematical analysis, Burger 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.[16] As the diastolic transmembrane potential is negative, this inverts the direction of the gradient which thereby, contrary to intuition, points to the briefest APDs in the heart instead of to the longest APDs.[1] This concept has been verified experimentally.[17]

The QRST integral in one ECG lead; QRST integral maps

In 1979, further theoretical elaboration of the VG concept by Plonsey showed that the (scalar) VG in a specific electrocardiographic or epicardial lead is obtained by weighting

(vectorially multiplying) the APD gradient by the vector lead field during volume integration.[18] The more a given lead is sensitive for the electrical activity in a particular area of the heart, the more the VG 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 VG ("the spatial VG") which yields one single vector of given size and direction for one heartbeat of a given person. The lead-dependent approach of the VG in body surface mapping may be useful to find localized inhomogeneities in the heart. By definition, the spatial VG, that can be derived from the 12-lead ECG, cannot discern between global and local phenomena as it lumps all APD inhomogeneity in the heart into one single integral.

The VG as an index for heterogeneity of action potential morphology

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

Cancellation and the VG

Cancellation of electromotive forces in the heart greatly reduces body surface potentials and ECG amplitudes. An estimated 75% of the electrical energy is cancelled during ventricular depolarization; during repolarization this estimated percentage is even 92-99%.[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 VG (decrease due to cancellation).

APD heterogeneity under physiological and pathophysiological conditions

The VG integrates gradients in the action potential morphology throughout the heart; one major aspect of action potential morphology is action potential duration (APD). Multiple studies have been conducted to search for the existence of transmural, apicobasal and left-ventricular–right-ventricular APD gradients. An extensive review by Burton and Cobbe regarding this topic has been published recently.[23] The results of these studies are sometimes seemingly conflicting; this is 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 VG in animals or in man. Moreover, attempts to establish normal values of the VG in man have revealed a considerable variability between subjects.[25] Hence, it may be better to longitudinally measure intra-individual trends in the VG than to transversally measure inter-individual differences in the VG.

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 remodelled hypertrophied hearts after myocardial infarction, APDs in affected areas are prolonged.[29]

In a biological 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]

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

The VG and the T wave

The T wave in the ECG reflects the heterogeneity of the repolarization throughout the ventricles.[4] In 1964 Van Dam and Durrer attempted to simultaneously activate canine ventricles by high-intensity electrical stimuli.[17] The thus obtained T wave 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 and colleagues.[34] In both publications it was mentioned that the area under the primary T wave equals the VG. Abildskov and colleagues defined also 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).[34] 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 VG

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 VG should be insensitive to such changes. However, since the introduction of the VG it has been observed that extrasystoles appeared to change VG.[4] Wilson and colleagues[2] reported this phenomenon already and ascribed it to measurement errors, physiological variability due to respiration, and an altered mechanical contraction pattern due to alterations in the activation pathway. Since then, several mechanisms were identified that contribute to altered action potential morphology distribution throughout the heart when it is activated in an abnormal order, amongst others altered electrotonic influences[35], altered cellular electrophysiological properties due to premature excitation[36] and cardiac memory.

T-wave memory and the VG

In a classical publication, Rosenbaum and co-authors[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 V2 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, like altered gap juntion distribution, altered I_{to} and I_{Kr} distribution; alterations in $I_{Ca,L}$, altered electrophysiological responses to antiarrhythmic agents.[38] The VG would be an appropriate ECG parameter to keep track of such dynamic changes in action potential morphology distribution throughout the heart.

Repolarization heterogeneity, reentrant arrhythmias and the VG

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

It were Spach and Barr who measured, by multiple epicardial and transmural electrodes in recovered closed-chest dogs, selected on having normal upright T waves in lead II, the full activation and repolarization sequence.[41] They found that the epicardium was to repolarize first, although it depolarized last. The results of the classical mapping study in human hearts by Franz and coworkers[42] are in agreement with the findings of Spach and Barr[41] because they showed that, when pooling the results for all endocardial and epicardial mapping sites, there was a negative linear relationship between activation time and APD.[42] This inverse relation between the activation time and APD would contribute to a relative stabilization of the repolarization.[42] Hence, in the normal heart (normal electrophysiological matrix, normal activation sequence), the VG reflects a normal degree of APD heterogeneity that more or less compensates for a normal degree of activation time heterogeneity.

It were Abildskov and co-workers to suggest that QRST area was among the promising ECG parameters for reentry risk assessment and several articles followed showing that the

QRST area distribution over the body surface indeed correlated with arrhythmia vulnerability.[3] In this approach, arrhythmia risk was conceptually coupled to altered primary factors, *i.e.*, altered local APD heterogeneity reflected in an altered local lead dependent VG. Obviously, increased dispersion of the repolarization, caused by altered APD dispersion and likely to be reflected in an altered VG, is not sufficient for an arrhythmia to occur. A second condition, the occurrence of an appropriate triggering stimulus, like an ectopic beat, has also to be fulfilled.

Relation between the spatial VG and the QRS and T axes

It follows from the mathematical definition and calculus[2,16] that the spatial VG (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 the QRS and T axes do. 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 VG points usually anterior, inferior and to the left, roughly along the long axis of the heart, in the direction of the apex (see Figure 1).[25] As the VG points in the direction of the briefer action potentials, it can be deduced that corresponding apicobasal and/or endo-to-epicardial action potential morphology trends (briefest action potentials apically/epicardially) must necessarily exist.[1]

Relation between spatial VG, QRS and T integrals, and the QRS-T angle

Recently, Van Oosterom provided important insight into the ECG deflections caused by the ventricular depolarization process, action potential morphology heterogeneity, repolarization process, and their relationship.[43] 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, while the integral of the heart vector during repolarization (spatial T-wave integral) is proportional to the dispersion of the repolarization (provided that QRS and T don't overlap).

This analysis clearly establishes the electrocardiographic relationship between primary factors (APD dispersion, measured in the ECG as the VG), secondary factors (dispersion in activation instants, measured in the ECG as the QRS integral) and the resulting dispersion of the repolarization (measured in the ECG as the T-wave integral). Of note, in electrocardiography, dispersion in ventricular activation is currently 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 in use as indexes of dispersion of the repolarization.[44]

A situation with increased QRS and T integrals is not necessarily associated with a large VG. 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 VG, which is the vectorial sum of the QRS and T integrals remains unchanged (no primary changes).

The spatial QRS-T angle, that increases by secondary changes,[45] has proven to be an important prognostic ECG index.[46] Possibly, combining the spatial QRS-T angle with the VG could still futher increase this prognostic value, as this would add information about absence or presence of primary changes. With pure secondary changes, only the spatial QRS-T angle

would be enlarged, while the VG remains unchanged. With additional primary changes (a less favorable condition) the VG would be enlarged as well.

Illustrative simulated ECGs

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

The ECG in panel A of Figure 2 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]

The ECG in panel B differs from that in panel A 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 VG have by definition the same magnitude and direction.

The ECG in panel C 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 secondary nature. The VG has magnitude zero, as there is no APD heterogeneity. Logically, the QRS and T integrals have equal magnitudes and opposite directions.

The ECG in panel D 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 VG is larger, and also the T integral is larger than normal, due to increased APD heterogeneity and increased dispersion of the repolarization, respectively.

The ECG in panel E, finally, 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 before, the spatial QRS-T angle is wide. Both the QRS and T integrals are larger, because there is more disparity of activation and of repolarization. As there are no APD changes, the VG remains the same as normal.

While dispersion of the repolarization is increased in the ECGs depicted in panel D and in panel E, the causes of this increased dispersion differ completely (see also Figure 1 in the review article by Burton and Cobbe).[23] In panel E, the increase in dispersion in the repolarization is caused by a gross transventricular repolarization front, that grossly follows the depolarization sequence; such a repolarization pattern is, possibly, not arrhythmogenic per se.[45] Contrastingly, in panel D, 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 VG

As elaborated above, changes in the VG are caused by primary changes in the electrophysiological matrix, while secondary changes have no direct effect on the VG. This characteristic of the VG is potentially useful in ECG diagnostics in the presence of abnormal

intraventricular conduction, e.g., in the diagnosis of ischemic heart disease in the presence of preexistent right or left bundle branch block. The diagnosis of ischemia from the 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 associates the VG did not perform well compared to other ECG parameters in the diagnosis of myocardial infarction in the presence of ventricular conduction defects.[50] This could be due to the large range in the normal values of the VG.[25] Also, Goldman and colleagues, in their 1969 article, stressed the potential additional value of serial ECG analysis (comparison of the suspect ECG with a previous non-suspect ECG of the same patient) for this diagnostic purpose.

Also, the VG could be useful in detecting primary electrical disease. E.g., the sharp APD gradients in the right ventricle that occur in the Brugada syndrome[31] are likely to increase the VG magnitude. The Brugada syndrome has multiple electrocardiographic 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. Also, intermittencies in its electrocardiographic expression are seen. Whether or not the VG magnitude is elevated in the typical Brugada ECG, and remains elevated in suspicious and intermittent episodes requires further study. More in general, with increasing knowledge about primary electrical diseases and their electrocardiographic manifestations, it is reasonable to surmise that the spatial VG will have diagnostic and prognostic value here.

More general use of the VG

Whether or not the VG is useful in the setting of ECG diagnosis has still to be validated. Additionally, and possibly more importantly, the spatial VG 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 VG offers unique information about the ECG in healthy and diseased substrates under various conditions.

Recently a novel general ECG descriptor, called 'total cosine between R and T' (TCRT), 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 that compares the TCRT-based results with VG-based results, and in which the explicitely formulated mathematical relation of TCRT with the VG is given.

Conclusions

The spatial VG represents the lumped heterogeneity of action potential morphology throughout the ventricles. It is, like multiple electrocardiographic parameters, sensitive to cancellation. In the normal heart, the VG expresses a certain amount of physiological 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 VG represents a less effective compensation, thus increasing the amount of dispersion of the repolarization and, in presence of triggering events, increasing arrhythmogeneity.

Potential diagnostic value of the VG is to be found in the electrocardiographic diagnosis of ischemia in the presence of preexistent conduction defects, and in variable manifestations of primary electrical disease. Additionally, the VG can be regarded as a general parameter that characterizes the primary component of dispersion of the repolarization. As such, the VG could be useful in risk assessment. To our knowledge, follow-up studies towards the predictive value for ventricular arrhythmias in known arhythmogenic substrates have as yet not been done.

As the direction and magnitude of the spatial VG can be computed from any routine clinical 12-lead ECG, its measurement is much less cumbersome in comparison to the determination of e.g. 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 in serial ECG analysis.

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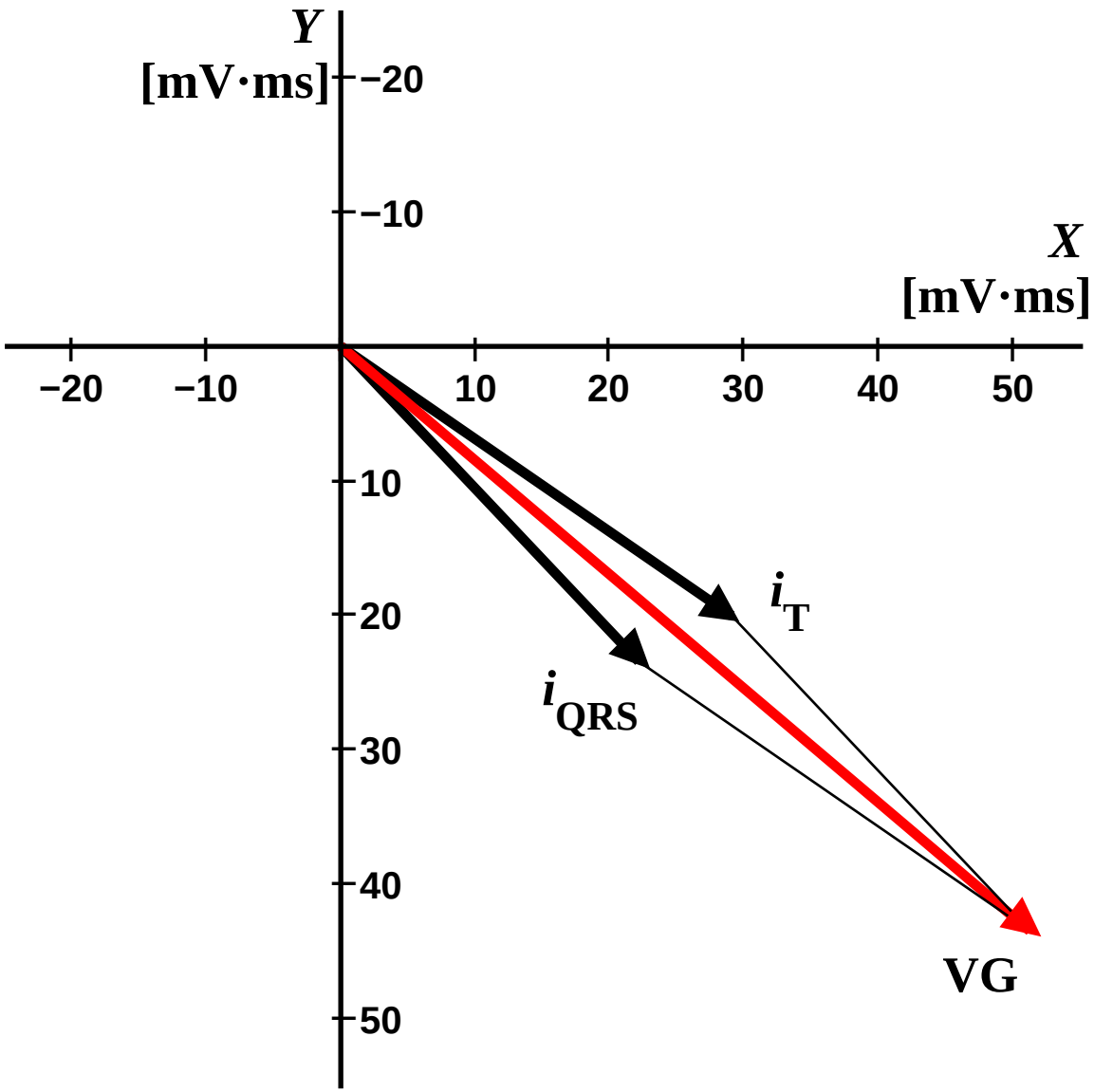
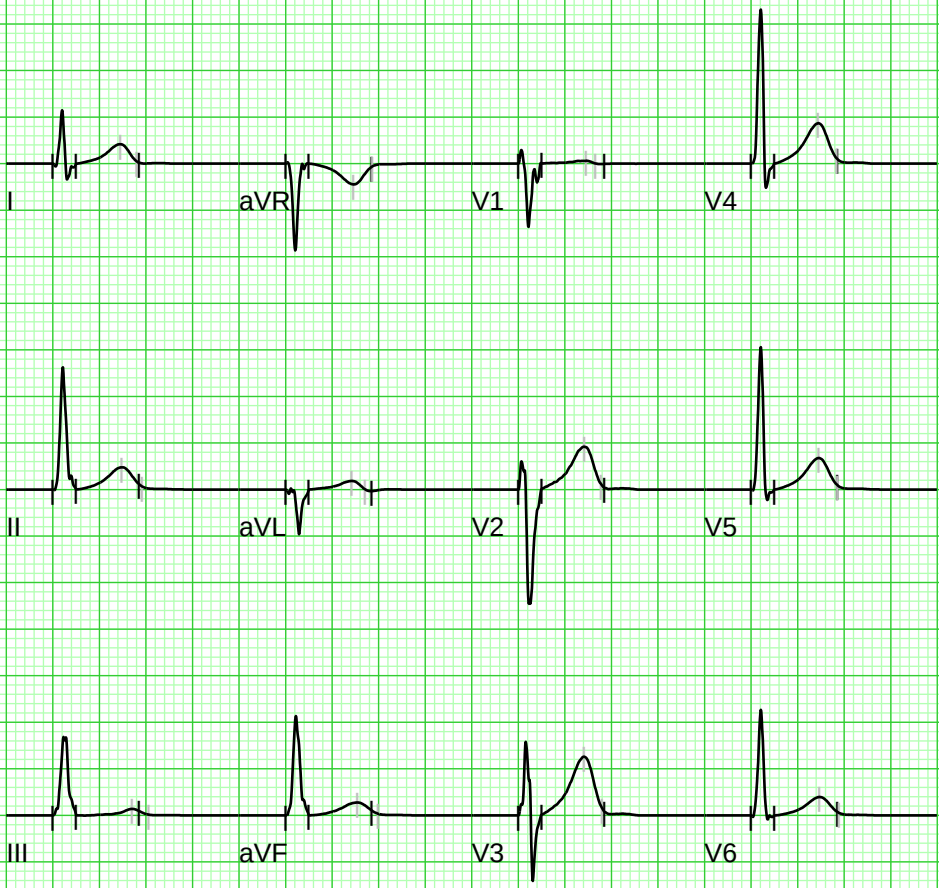
LEGENDS

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 VG 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 (panel A). To generate the primary T wave (panel B), all ventricular depolarization instants were set at zero time while maintaining the normal action potential duration heterogeneity. The secondary T wave (panel C) was obtained by using the normal ventricular depolarization sequence and by assigning all action potential durations the mean action potential duration. An ECG with increased dispersion of the repolarization (panel 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 (panel 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 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 of 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 further interpretation.


Y
[mV·ms]
−20
−10
X
[mV·ms]
−20
−10
10
20
30
40
50
10
20
30
40
50
i_T
i_{QRS}
VG

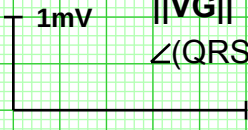


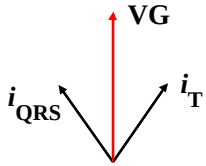
$||\boldsymbol{i}_{QRS}||$ = 48 mV·ms

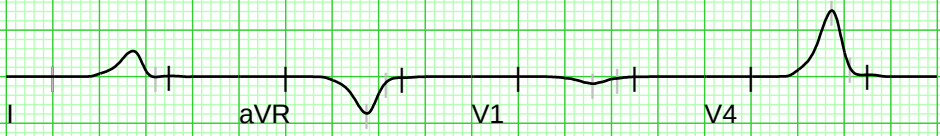
$||\boldsymbol{i}_{T}||$ = 49 mV·ms

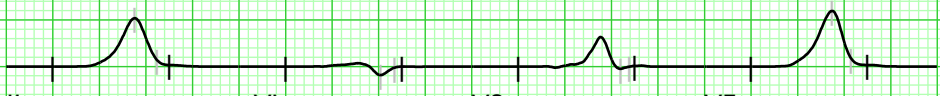
$||\mathbf{VG}||$ = 81 mV·ms

$\angle(QRS,T)$ = 70 °





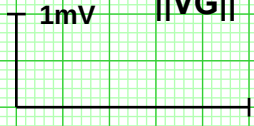


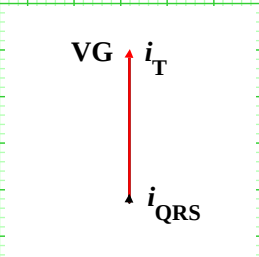


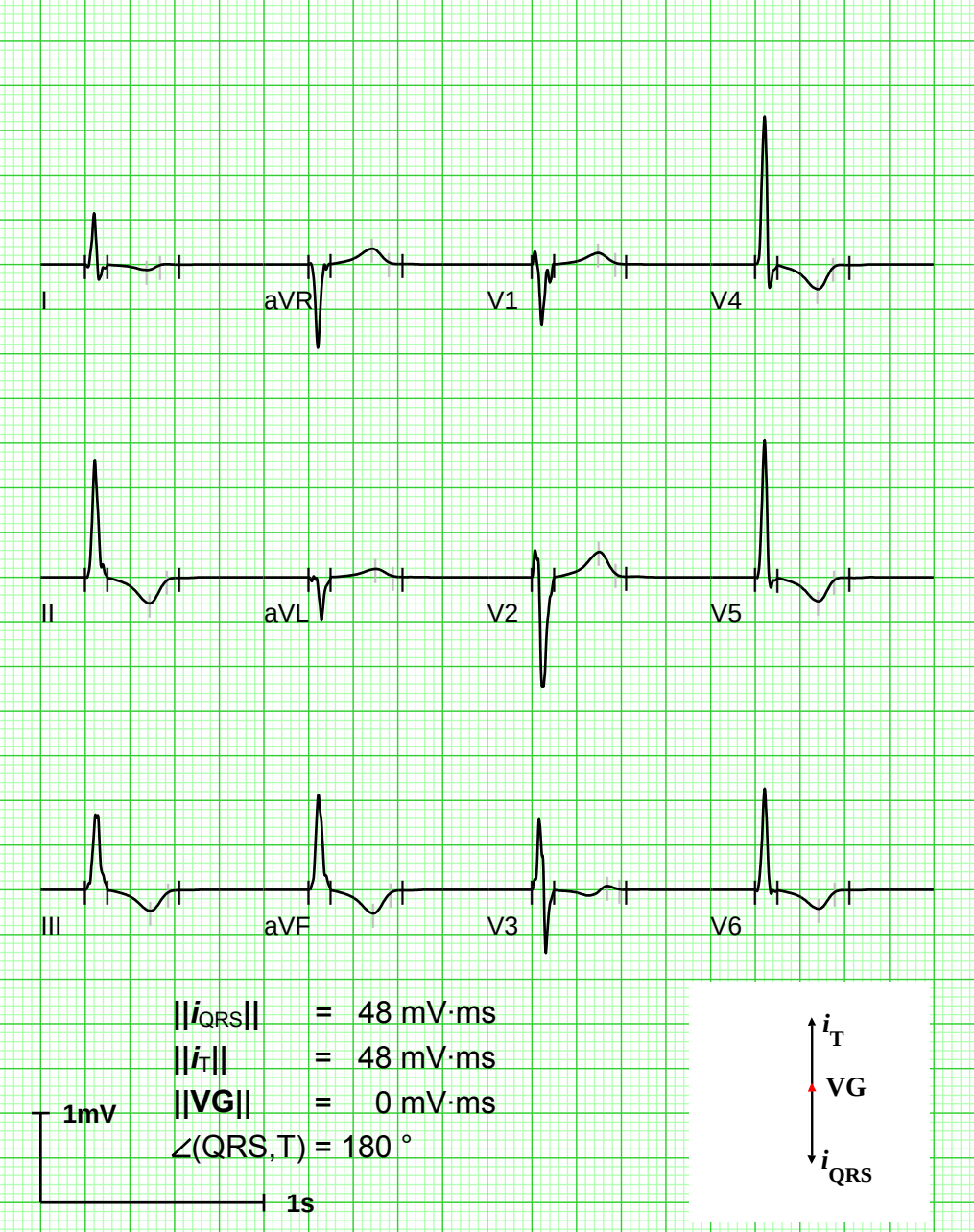
$||\boldsymbol{i}_{QRS}|| = 0$ mV·ms

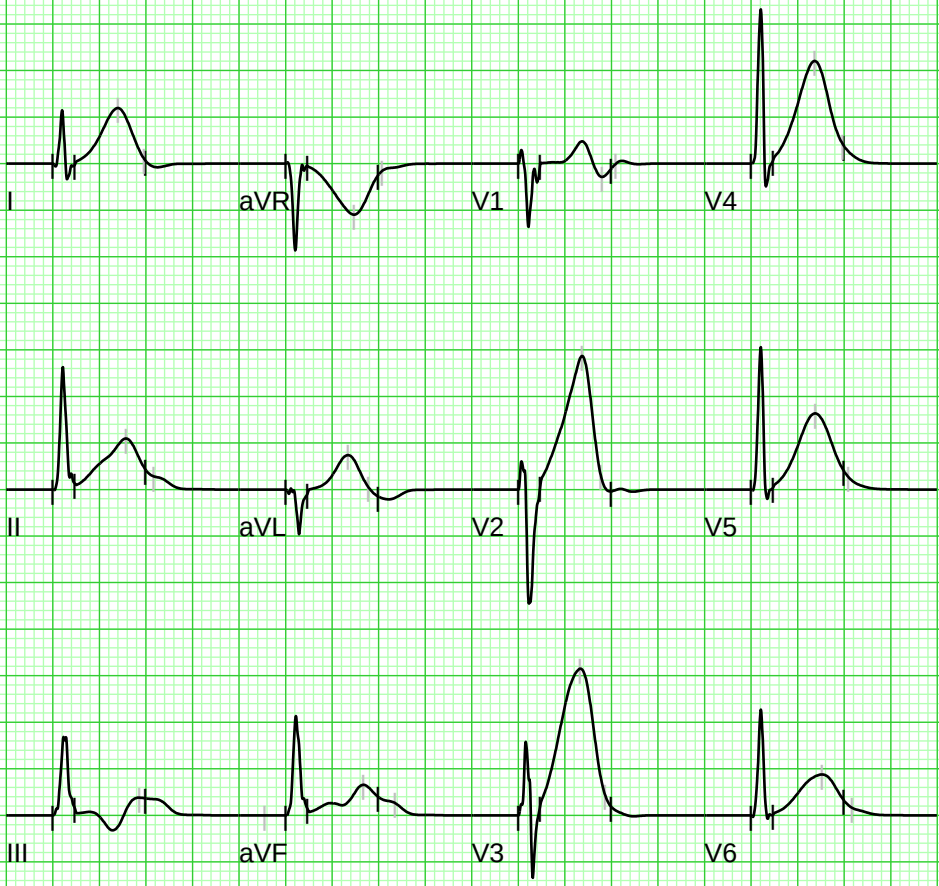
$||\boldsymbol{i}_{T}|| = 81$ mV·ms

$||\mathbf{VG}|| = 81$ mV·ms






I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
||i_{QRS}|| = 48 mV·ms
||i_T|| = 48 mV·ms
||VG|| = 0 mV·ms
∠(QRS,T) = 180 °
1mV
1s
i_T
VG
i_{QRS}

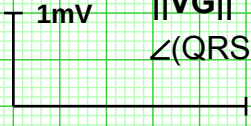


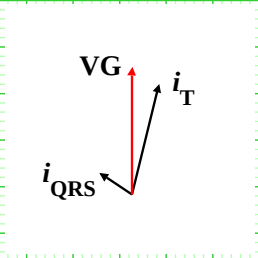
$||\boldsymbol{i}_{QRS}||$ = 48 mV·ms

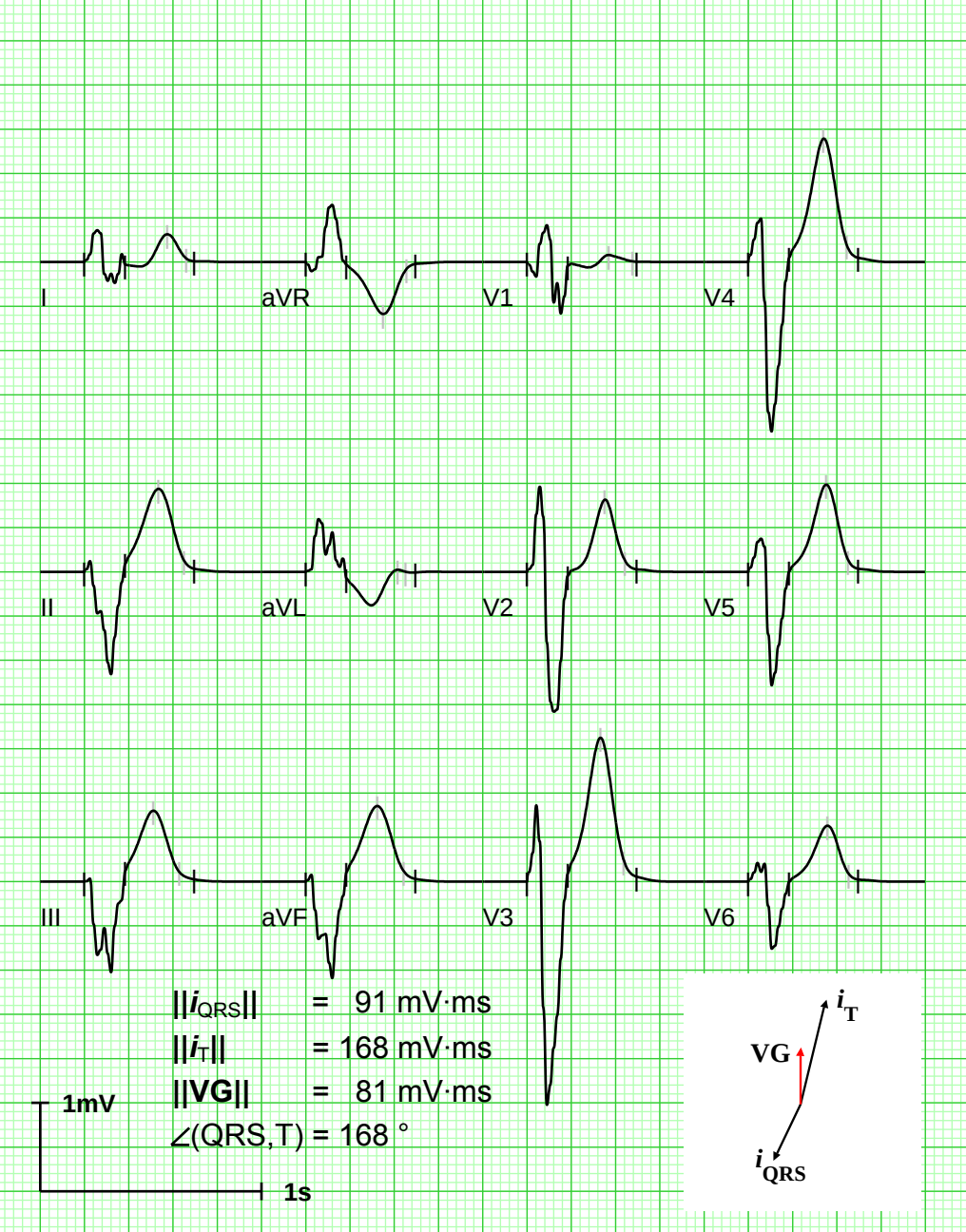
$||\boldsymbol{i}_{T}||$ = 169 mV·ms

$||\mathbf{VG}||$ = 191 mV·ms

$\angle$(QRS,T) = 70 °






I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
||i_{QRS}|| = 91 mV·ms
||i_T|| = 168 mV·ms
||VG|| = 81 mV·ms
∠(QRS,T) = 168 °
1mV
1s
i_T
VG
i_{QRS}