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Non-invasive diagnosis of coronary artery disease using cardiogoniometry performed at rest

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Summary

Principles: Cardiogoniometry is a non-invasive technique for quantitative three-dimensional vectorial analysis of myocardial depolarization and repolarization. We describe a method of surface electrophysiological cardiac assessment using cardiogoniometry performed at rest to detect variables helpful in identifying coronary artery disease.

Methods: Cardiogoniometry was performed in 793 patients prior to diagnostic coronary angiography. Using 13 variables in men and 10 in women, values from 461 patients were retrospectively analyzed to obtain a diagnostic score that would identify patients having coronary artery disease. This score was then prospectively validated on 332 patients.

Results: Cardiogoniometry showed a prospective diagnostic sensitivity of 64%, and a specificity of 82%. ECG diagnostic sensitivity was significantly lower, with 53% and a similar specificity of 75%.

Conclusions: Cardiogoniometry is a new, non-invasive, quantitative electrodiagnostic technique which is helpful in identifying patients with coronary artery disease. It can easily be performed at rest and delivers an accurate, automated diagnostic score.

Key words: coronary artery disease; myocardial ischemia; cardiogoniometry; electrodiagnosis

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Introduction

Open my eyes that I may see wonderful things in your law (Ps. 119:18)

Since the late 1930s, assessments of three-dimensional data have been undertaken in order to glean information about the heart dipole [1, 2]; many different techniques have been suggested [3]. The best known and most widely – even though rarely – used vectorcardiography is the method developed by Frank [4]. However, traditional vectorcardiography is complicated to record, uses projections oriented according to body planes rather than the heart and electrode placement does not ensure an orthogonal system. We have developed a simple-to-use vectorcardiographic method, cardiogoniometry (CGM), which can surmount these traditional drawbacks and may provide easily accessible and useful quantitative information concerning the condition of the heart. CGM is a non-invasive, vecto-

Abbreviations

CAD	coronary artery disease
CGM	cardiogoniometry
ECG	electrocardiogram
IHD	ischaemic heart disease
LAD	left anterior descending coronary artery
LCX	left circumflex coronary artery
OSP	oblique sagittal plane
RCA	right coronary artery
SD	standard deviation
CMP	cardiomyopathy

rial, digital analysis of myocardial depolarization and repolarization. Its principles have been described earlier [5] and, in studies with smaller patient numbers [6–8], it has been shown to be useful in diagnosing ischaemic heart disease (IHD). However, only the recent advances in electronic data analysis have enabled the development of CGM into the new, non-invasive and automated cardiological diagnostic method presented here. CGM uses four thoracic leads to construct the vectorcardiographic loops in a three-dimensional system, defined by the three orthogonal axes, x, y, and z, approximatively related to the anatomical orientation of the heart.

Traditional 12-lead surface electrocardiography, performed at rest, is a non-invasive and widely available, albeit insensitive method for diagnosing ischaemic heart disease [9]. Furthermore, automated interpretation of the ECG is not always reliable [10] and the diagnostic yield depends highly on the ECG expertise of the reader [11]. We hypothesized that CGM can detect changes in the electric cardiac phenomena, even at rest, in patients at risk for myocardial ischaemia. The rationale for this is threefold: 1) CGM provides three-dimensional information on voltage and spatial orientation of the summation vector of the surface potential, which is not available in the ECG; 2) CGM provides measured quantitative computer analysis of this three-dimensional information; the rating does not require a qualitative evaluation by an expert; and 3) the CGM allows spatial analysis of beat-to-beat variability.

Patients and methods

Due to suspected ischaemic heart disease, 793 (270 women) patients were referred to a tertiary care cardiology clinic for a first elective coronary angiography. The CGM was performed at rest a few hours prior to the coronary angiogram. Written informed consent was obtained from all patients, and the local Ethics Committee had approved the study. During the recording, the patient lay in a supine position and, after normal expiration, held his or her breath for 12 to 15 seconds during the measurement. If this was not possible, shallow breathing with minimal thoracic excursion was allowed. Patients with atrial fibrillation, with frequent premature beats in the recording, left bundle branch block, and severe (grade III) valvular heart disease were excluded, as were patients with previous cardiac surgery. A CGM score to identify patients with coronary artery disease (CAD) was retrospectively derived on 461 patients (154 women) and prospectively validated on a further 332 patients (116 women). In 145 consecutive patients (51 women), the pre-angiography ECG was evaluated by an independent cardiologist (P.L.) for the presence or absence of IHD in the retrospective cohort. The same cardiologist, who had been blinded to the results of the CGM and coronary angiography, rated the pre-angiography ECG in all patients from the prospective cohort. Sensitivity and specificity of CGM and ECG in the prospective cohort were statistically compared using McNemar's test. Tests of differences between accuracies were constructed under Gaussian as-

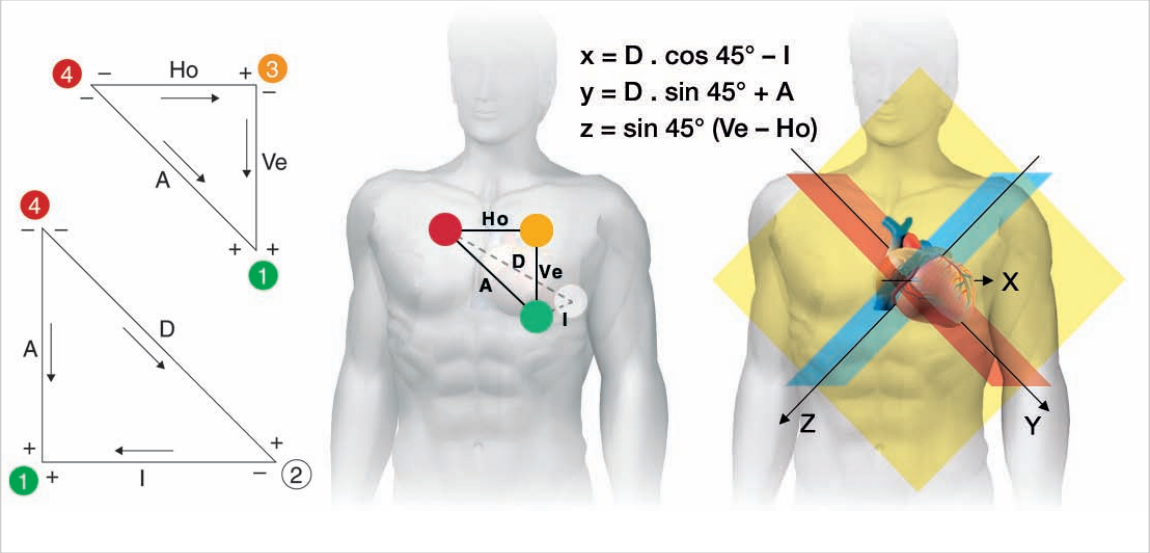


Figure 1

Principles of cardiogoniometry: Four electrodes are placed at four points on the patient's thorax as follows: Point 1 (green) at point V4 of Wilson, ie, in the 5th intercostal space in the mid-clavicular line; point 2 (white) sagittal to electrode 1 on the back (point V8 of Wilson); point 3 (yellow) is located perpendicularly above electrode 1 at 0.7 times the distance between points 1 and 2; point 4 (red) is placed to the right of point 3 at the same distance as between points 1 and 3. The leads are defined as follows: 4-2 D (dorsal), 4-1 A (anterior), 2-1 I (inferior), 4-3 Ho (horizontal), 3-1 Ve (vertical). Points 4-2-1 define the oblique sagittal plane OSP (red on the right drawing); points 4-3-1 define the frontal plane (yellow on the right drawing). The third plane (blue on the right drawing) is orthogonal to the two other planes and contains point 3; it is the sagittal plane perpendicular to the OSP. Projection x is oriented in an antero-dorsal direction and lies in the OSP and the sagittal plane perpendicular to the OSP. Projection y is oriented in a baso-apical direction and lies in the OSP (4-2-1) and the frontal plane (4-3-1). Projection z is oriented in a supero-inferior direction relative to the OSP and lies in the frontal plane (4-3-1) and the sagittal plane perpendicular to the OSP. Deduction of definitions of the projections x, y, and z is given elsewhere in detail [5].

ymptotic assumptions and relied upon the variances of the differences in sensitivities and specificities. A p-value <0.05 was considered significant.

Principles of cardiogoniometry

The trigonometric principles of CGM have been described in detail elsewhere [5], but can be summarized as follows: Four electrodes define two planes that are perpendicular to each other (figure 1): the frontal plane (electrodes 1,3,4) and the oblique sagittal plane (OSP) (electrodes 1,2,4). The three electrodes for each plane describe a Kirchhoff loop. Vectorial addition of the three potentials between pairs of these electrodes results in a vector that describes the electric field in each plane, according to Newton. The vectors of two orthogonal planes allow the construction of the heart vector, which gives, by its orientation, the direction of the field and, by its length, the strength of the electrical field generated by the heart. The electrical potential sampling rate was 1 kHz. CGM differs from conventional vectorcardiography by two relevant improvements. Firstly, the recordings are performed without intercalated resistances. This avoids the distortions present in historical vectorcardiographic methods using circuits with compensatory resistances. No distorting proximity effects were observed. Secondly, the projection planes of CGM are oriented in rough approximation to the anatomy of the heart, rather than the body planes (figure 2).

The coordinate system used for display is a Cartesian system (figures 2 and 3), defined by three orthogonal axes that cross in the origin, which has the value of zero for all axes (figure 2). The x-axis has an antero-posterior orientation with positive values posteriorly. The y-axis has a left oblique sagittal orientation (OSP) with positive values pointing to the apex, negative values pointing to the base of the heart. The x- and y-axes define the OSP, which is the main plane. The z-axis is perpendicular to the OSP with negative values pointing up. The frontal plane is defined by the y- and the z-axes. The plane that is defined by the x- and z-axes is also a sagittal plane that lies perpendicular to the OSP and separates the apical from the basal part of the heart.

The recording of the vector each millisecond results in the three traces x, y, and z (figure 4) from which the vector loops (figure 5) are constructed. The distance of each point on the different loops from the origin, ie, the length of each millisecond vector that corresponds to the strength of the electrical field, can be displayed over time.

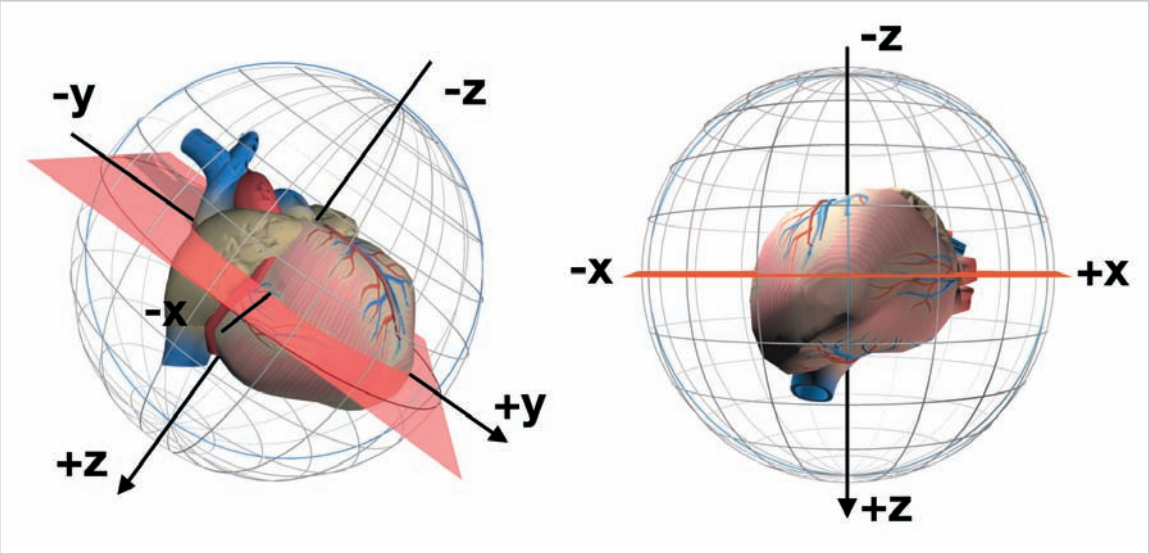
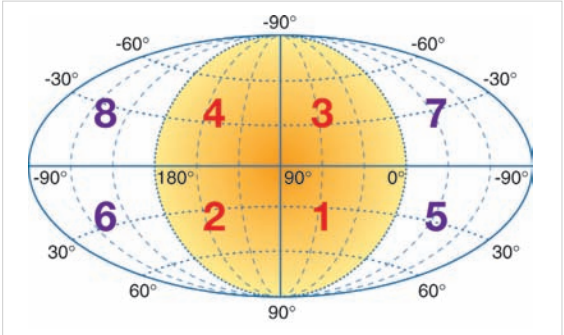


Figure 2

Projections x, y, and z. The projections x, y, and z are oriented relative to the heart. The OSP (red in the drawing) is the main plane, defined by the projections x and y. A globe is defined around the heart, with its centre at the origin of the orthogonal system, ie, at the point where the three rectangular axes x, y, and z cross. Degrees of longitude (angle alpha) and of latitude (angle beta) define precisely every point on the globe around the heart. The positive end of the y-axis projects to the apex of the heart and pierces the imaginary globe at $\beta = 0°$ latitude and $\alpha = 90°$ longitude. The negative end of the y-axis lies at $\beta = 0°$ latitude and $\alpha = -90°$ longitude. The spatial orientation of any vector measured can be defined with its projection onto the globe positioned around the heart.

Figure 3

The coordinate system of the heart vectors enables the imaginary globe around the heart to be divided into hemispheres and octants. The apical hemisphere (orange) is located between 0° and 180° longitude (angle alpha), the basal hemisphere correspondingly between 0° and –180°. The y-axis points to the apex of the heart and therefore to $\alpha = 90°$ longitude and $\beta = 0°$ latitude. The anterior (ventral) hemisphere extends from $\alpha = 90°$ over 180° to –90°, and the posterior (dorsal) hemisphere correspondingly from $\alpha = 90°$ over 0° to –90°. The latitude (angle beta) defines a superior ($\beta = 0° - -90°$) and an inferior ($\beta = 0° - +90°$) hemisphere. The octants are defined as follows: octant 1: apical inferior posterior; octant 2: apical inferior anterior; octant 3: apical superior posterior; octant 4: apical superior anterior; octant 5: basal inferior posterior; octant 6: basal inferior anterior; octant 7: basal superior posterior; octant 8: basal superior anterior. Numbers of apical octants are given in red; numbers of basal octants are given in purple.



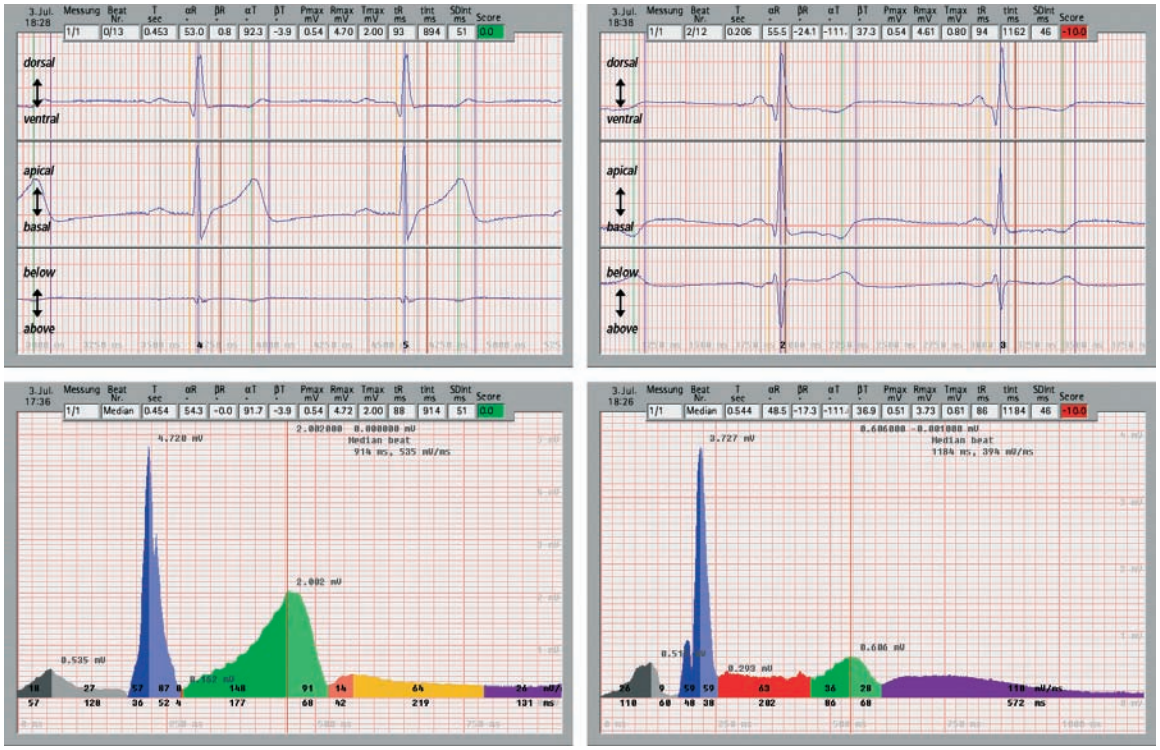


Figure 4

Traces from a healthy person (left) and a patient with myocardial ischaemia (right). Images above show the recordings of the projections of x (dorsal – ventral), y (apical – basal), and z (below – above). Images below show the time course of the median potential corresponding to the median distance of the tip of the momentary vector from the origin (abscissa in mV). Dark colours indicate potentials from the beginning of a loop to the maximum distance from the origin; light colours correspond to potentials from the maximum of the loop till its end. Potentials corresponding to the P-loop (atrial depolarisation) are shown in grey (P+ dark grey, P- light grey). Because potentials are non-vectorial and cannot be negative, the loop corresponding to the QRS-complex is designated the R-loop. The potentials corresponding to the R-loop (ventricular depolarisation) are shown in blue (R+ dark blue, R- light blue). The potentials measured after the end of the R-loop and before the onset of the T-loop are shown in red. The potentials corresponding to the T-loop (ventricular repolarisation) are shown in green (T+ dark green, T- light green). TP-potentials are shown in purple. The recording on the left side differentiates U-potentials (U+ orange, U- yellow).

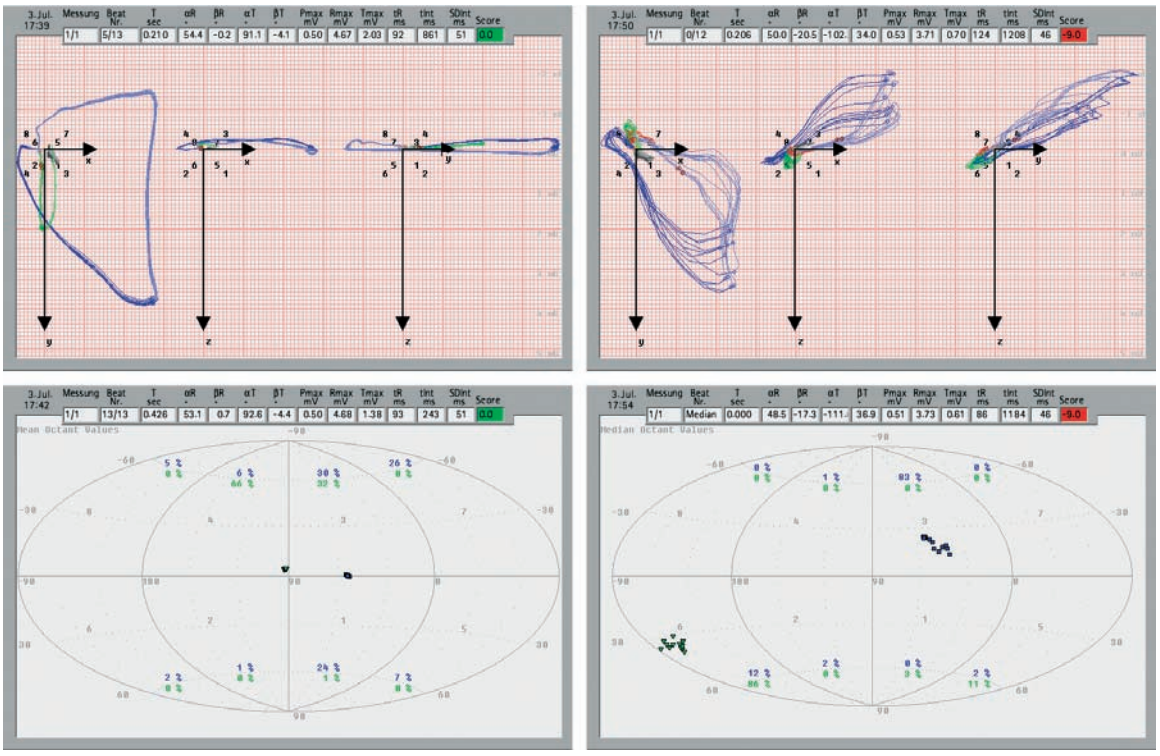


Figure 5

Vector loops and projections from a healthy person (left) and a patient with myocardial ischaemia (right). The images above show three orthogonal projections of the vector loops of several heart beats onto one plane: The loops on the left are the projection onto the OSP. The loops in the middle are the projection onto the sagittal plane perpendicular to the OSP, corresponding to the view onto the apex (four chamber projection in echocardiography). The loops on the right are the projection onto the frontal plane. Axes and projections of octant numbers are shown in black; P-loop in grey, R-loop in blue, RT-potentials in red, T-loop in green, TP-potentials are not shown. Note the pathological variability of the loops from heartbeat to heartbeat ("floating") in the patient on the right side. The images below show the projection of the maximal vector of each R-loop (blue) and T-loop (green) onto the globe around the heart. Note the pathological orientation of the maximal T-vectors (basal, inferior, anterior) in the patient on the right side. Numbers of octants are indicated in grey. Percentages of R-loop (blue) and T-loop (green) potentials are shown for each octant.

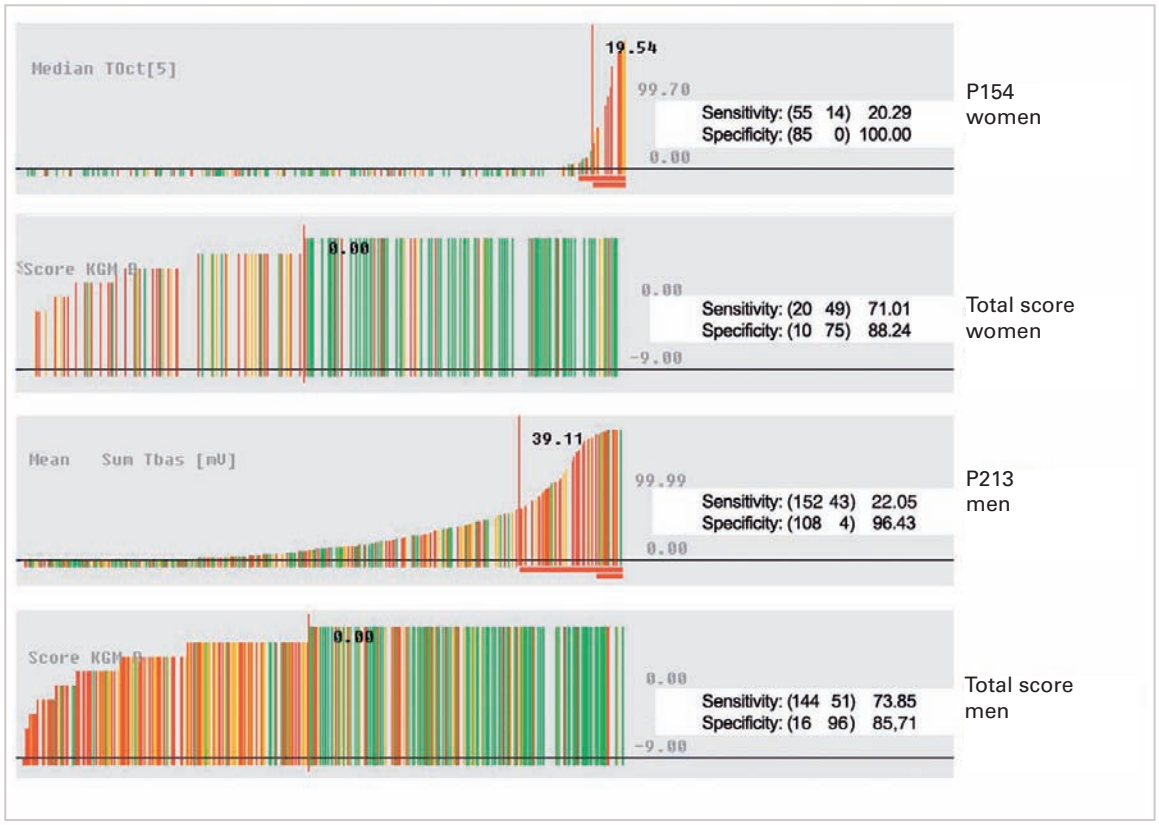


Figure 6

The distribution of measured values is given for one exemplary variable measured in women (above) and men (below) and for the global score (retrospective cohort). Values are sorted from the lowest to the highest. The ordinate corresponds to the measured values. Lowest and highest measured values are indicated on the right in grey. One vertical line is drawn for each patient. Green lines indicate patients without significant coronary artery stenoses. Yellow lines indicate patients with one vessel disease. Red lines indicate patients with two or three vessel disease. The sensitivity and specificity for each individual variable are given on the right side. The numbers in parentheses indicate false negatives (above left), true positives (above right), true negatives (below left), and false positives (below right). Red horizontal bars indicate pathological ranges for men (upper bar) and women (lower bar).

Table 1

Diagnostic cariables in cardiogoniometry.

Name	M / F	Description	Normal range
P20	F	Ratio of the maximal spatial velocity of the R-loop over the maximal spatial velocity of the T-loop. "Spatial velocity" is defined as the maximal distance measured in mV between two points separated from each other by 10 ms temporal distance on the R-loop or the T-loop, respectively.	≤22.5
P46	F	Median value of the angle alpha of the maximal vector of the R-loop	≤90°
P49	M	Median value of the angle beta of the maximal vector of the R-loop	≥–38°
P51	M	SD of the angle beta of the maximal vector of the R-loop for all measured heartbeats	≤2.15°
P64	M / F	Median angle alpha of the maximal vector of the T-loop	M: 10° – 130°; F: ≤113°
P67	M / F	Median angle beta of the maximal vector of the T loop	M: –56° – 1°; F: ≤1°
P72	M	SD for all measured heartbeats of the angle phi, defined as the angle between the maximal vectors of the R-loop and the T-loop	≤5.2°
P90	M	SD for all measured heartbeats of the ratio of the maximal potential (ie, the length of the maximal vector) of the P-loop over the maximal potential of the T-loop	≤0.09
P108	M	SD for all measured heartbeats of the "eccentricity" variable that describes the roundness of the R loop. If the R-loop is a perfect circle, P108 equals 0.	≤0.08
P127	M	% of the potential of the R-loop located in the apical superior anterior octant, ie, octant 4	≤51%
P136*	M	% of the potential of the R-loop located in the basal superior posterior octant, ie, octant 7	≤47%
P154	M / F	% of the potential of the T-loop located in the basal inferior posterior octant, ie, octant 5	M:≤1.25%; F: ≤19%
P157	M	% of the potential of the T-loop located in the basal inferior anterior octant, ie, octant 6	≤11%
P176	F	Temporal integral of the potential of the P-loop after the maximum of P	≤19.6 mVms
P213	M / F	% of the T-loop potential located in the basal hemisphere, ie, in the octants 5–8	M: ≤39%; F: ≤95%
P220	M / F	Median angle alpha of the vector pointing from the first point of the R-loop to the point 10 ms after ventricular depolarisation commences, ie, the initial orientation of the R-loop	M: < –130° or >45° F: < –126° or >56°
P273	F	Mean potential per ms of the T-loop that lies in octant 5	≤0.10 mV
P280*	F	Maximal potential of the T-loop that lies in octant 7	≤0.19 mV

M: men; F: women; SD: standard deviation; * variables removed after prospective evaluation of the diagnostic score

By using the median of heartbeats recorded over 15 seconds (excluding premature beats), the median potential course is obtained (figure 4).

The projections of each vector and, in particular, the maximal vectors of each loop on a globe surrounding the heart, with the origin of the coordinate system in its centre, are defined by two angles. The angle alpha defines the degree of longitude (the "meridian"), ie, how much anterior or posterior to the plane (defined by the y- and z-axes) the vector lies (figures 2 and 3). The angle alpha of a vector that lies in the y-axis and points to the apex is defined as 90°. Points on the apical hemisphere have positive values for the angle alpha (0° to 180°); points on the basal hemisphere have negative values for the angle alpha (0° to –180°). The angle beta defines the degree of latitude, ie, how much a vector lies above or below the OSP (defined by the axes x and y). Positive values for beta indicate a vector that points under the OSP (xy-plane); negative values are given for a beta that points above this plane (figures 2 and 3).

The coordinate system of the three orthogonal axes divides the space in eight octants (figure 3): Octants 1 to 4 are apical, 5 to 8 are basal. Octants 2, 4, 6, and 8 are anterior; octants 1, 3, 5, and 7 are posterior. Octants 1, 2, 5, and 6 are inferior, and octants 3, 4, 7, and 8 are superior.

Table 2

Patient characteristics and results of coronary angiography.

	Men		Women	
	Retrospective	**Prospective**	**Retrospective**	**Prospective**
Number of patients	307	216	154	116
Age (years)	61 (11)	61 (12)	65 (10)	66 (12)
Height (cm)	173 (7)	173 (10)	161 (6)	161 (6)
Weight (kg)	82 (12)	81 (12)	70 (15)	71 (16)
Body mass index (kg/m^2)	27 (4)	27 (8)	27 (6)	27 (6)
Pathological coronary angiography	195	158	69	49
Three vessel disease	63	45	24	7
Two vessel disease (RCA+LAD)	17	19	6	8
Two vessel disease (RCA+LCX)	11	13	3	4
Two vessel disease (LAD+LCX)	25	20	10	5
Single vessel disease (RCA)	24	20	7	4
Single vessel disease (LAD)	37	20	13	12
Single vessel disease (LCX)	12	8	3	3
Detailed information not available	6	13	3	6

Values are given as mean (standard deviation). For coronary angiography results, numbers of patients are given.
Left main artery stenosis was counted as two vessel disease. RCA: right coronary artery. LAD: left anterior descending coronary artery. LCX: left circumflex coronary artery.

Table 3

Sensitivity, specificity, positive and negative predictive values of cardiogoniometry and ECG.

		Men		Women		All	
		Retrospective	**Prospective**	**Retrospective**	**Prospective**	**Retrospective**	**Prospective**
CGM	Sensitivity	74%	65%	71%	61%	73%	64%
	Specificity	86%	84%	88%	79%	87%	82%
	Positive predictive value	90%	92%	83%	68%	88%	85%
	Negative predictive value	65%	47%	79%	74%	71%	58%
ECG	Sensitivity	45%	53%	57%	54%	49%	53%
	Specificity	83%	81%	77%	71%	81%	75%
	Positive predictive value	72%	88%	63%	57%	69%	78%
	Negative predictive value	60%	39%	72%	69%	64%	51%

The gold standard used to assess CAD was coronary angiography.

Table 4

Numbers of patients according to coronary angiography results and cardiogoniometry scores.

			CGM score					
			0 negative	**–1 positive**	**–2 positive**	**–3 positive**	**–4 positive**	**≤–5 positive**
Retrospective cohort	Men	Normal	96	8	2	2	3	1
		CAD+	51	60	34	22	8	20
	Women	Normal	75	5	3	2	0	0
		CAD+	20	20	13	7	5	4
Prospective cohort	Men	Normal	49	4	3	0	1	1
		CAD+	55	35	27	11	12	18
	Women	Normal	53	6	6	1	0	1
		CAD+	19	21	5	3	1	0

The gold standard used to assess CAD was coronary angiography. CAD: coronary artery disease

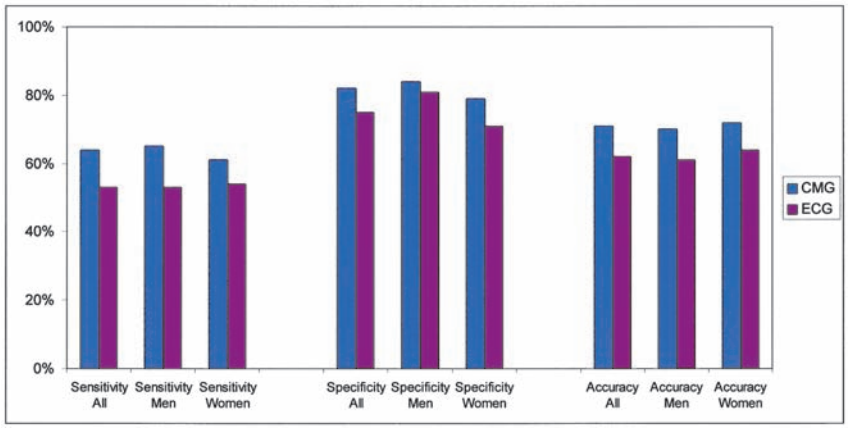


Figure 7

Diagnostic sensitivity, specificity, and accuracy of CGM and ECG.

Choice of variables for the diagnostic score

Using the CGM recordings from 461 patients, a diagnostic score was retrospectively derived to identify patients with CAD. The diagnostic score was developed in a two-step procedure. Firstly, among the 350 variables describing the cardiogoniometric loops that were measured in each patient, we used only those for which limits of normal ranges could be defined, ie, variables with dichotomising quality that showed ranges in which only – or mostly – pathological cases were found, and no measure from a patient with a normal coronary angiogram. No univariate statistical tests were used for choosing diagnostic variables. In a second step, variables were grouped according to their electrophysiological aspects (time course, potential, direction of vectors, R or T values, beat-to-beat variability), and redundant variables were eliminated. Variables that were physiologically not plausible were discarded. CAD may alter the surface potential of the global cardiac activity in different ways, depending on the affected area of the myocardium. Therefore, a combination score of penalizing variables has been used. The number of variables used for the score was deliberately restricted in order to avoid over-determination. Several variables, as described below, were retained (13 for men and 10 for women) to formulate a diagnostic score (table 1). For each value outside the normal range, a negative point was given (figure 6). Normal limits for the variables were defined so as to ensure the best discrimination between normal and pathologic cases. When the score did not equal 0, a CGM diagnosis of CAD was made. No positive points were given. Coronary arteriography was used as the gold standard for the presence or absence of CAD. For the purpose of this study, an empirical cut-off value of 50% was chosen for a stenosis in one of the three main coronary arteries to define CAD. However, 96% of all patients who were rated as having CAD had stenoses ≥75% or multiple 50% stenoses in series. Prospective CGM scoring was performed automatically and independent of the assessment of the coronary angiogram. All assessors were blinded as to the respective results from the other method.

ECGs used the traditional 12-lead standard recordings taken in the supine position at rest and in accordance with the recommendations of the American Heart Association [12]. The ECGs were rated on the basis of the Utrecht ECG Coding System [13, 14].

Results

Characteristics for the 461 patients (154 women) in the retrospective cohort and the 332 (116 women) in the prospective cohort are given in table 2.

Results from the retrospective cohort

Coronary angiography showed stenoses in 195 men (64%) and 69 (45%) women. The angiography results are given in table 2. Detailed information was not available for six men and three women with pathological coronary angiography. Four women and thirteen men had acute or subacute anterior wall myocardial infarction. Six women and twenty men had acute or subacute posterior wall myocardial infarction. Using one non-invasive recording at rest, CGM correctly diagnosed 240 men (78%) and 124 women (81%) for the presence or absence of CAD. This results in a higher CAD diagnostic sensitivity with the CGM than with the ECG; both methods show similar specificity (table 3).

For all assessed values, the ranges of patients with and without CAD overlapped. CGM was considered positive if at least one value lay beyond the defined limit of normality for the respective variable. The combination of thirteen (men), respectively ten (women), variables was necessary to derive the diagnostic score. The CGM scores for men and women with and without coronary artery stenoses are given in table 4.

Prospective validation of the diagnostic score

Coronary angiography showed stenoses in 158 men (73%) and 49 (42%) women. The angiography results are given in Table 2. Detailed information was not available for nine men and four women with single vessel disease and four men and two women with two vessel disease. Two men had acute or subacute anterior wall myocardial infarction. Prospective application of the CGM score for identification of patients with coronary artery disease resulted in an overall diagnostic sensitivity of 64%, and specificity of 82% (table 3). The CGM scores are given in table 4. For comparison, the overall diagnostic sensitivity and specificity of the ECG were 53% and 75%,

respectively (table 3), in the prospective cohort. The diagnostic sensitivity, but not specificity, was significantly better for CGM than for ECG ($p < 0.007$). Overall prospective diagnostic accuracy was better with CGM than with ECG ($p < 0.003$): 71% (men 70%, women 72%) for CGM and 62% (men 61%, women 64%) for ECG (figure 7). Review of the usefulness of the chosen variables in men confirmed the high diagnostic value of all variables but P136.

Among the nine male patients who were falsely identified by CGM as having ischaemia, four had cardiomyopathy (CMP) (one alcoholic CMP, two dilatative CMP, one CMP of unknown origin), one had left ventricular hypertrophy, one had giant coronary arteries, one had a muscle bridge over the LAD with "milking," and one had experienced sinus tachycardia with elevation of troponin before the CGM recording.

The diagnostic value of variables used among women proved excellent for all variables but P280, which led to only false positive results and must therefore be invalidated.

Among the fourteen female false positive cases, two had left ventricular hypertrophy, one had a muscle bridge over the LAD with "milking," one had an acute myocardial infarction due to hypovolaemic shock, one had severe cor pulmonale with elevated troponin and postulated ischaemia due to congestion, one had troponin-positive acute myocarditis, and one had circumscript antero-lateral hypokinesia with open coronary arteries, suggesting myocardial infarction due to a thrombus that resolved prior to angiography. Thus, in seven patients, a possible cause for myocardial ischaemia in the absence of CAD could be identified.

Discussion

Non-invasive detection of myocardial ischaemia can be performed using surface ECG [15, 16], echocardiography and scintigraphy, which has largely been replaced by newer, expensive techniques employing radionuclide imaging [17] solely available at tertiary centres. The sensitivity of diagnostic methods that are performed at rest and yet available outside tertiary centres is low, and stress testing is therefore required to improve the diagnostic yield [18, 19]. Thus, surface ECG is currently the only inexpensive and widely-available method for non-invasive out-of-clinic assessment of myocardial ischaemia at rest. Although vectorcardiography has never gained broad acceptance in general clinical practice, the contribution of a third dimension in the analysis of cardiac electrical potentials is obvious [20, 3]. Nevertheless, this information cannot be obtained accurately from a conventional 12-lead-ECG because intercalated resistances distort the information, and because ECG leads do not form an orthogonal system. We present here a vectorcardiographic method that is easier to record than the conventional ECG (4 leads instead of 12), clearly displays accurate three-dimensional surface electrophysiological information and provides automated diagnostic evaluation of digitalised measurements. The variables used in the CGM score serve to identify the abnormal spatial localization of cardiac potentials and abnormal beat-to-beat variability. The use of these variables is plausible since alterations of repolarisation and depolarisation potentials are commonly used in the ECG diagnosis of IHD [21, 22], eg, a negative T-wave in the ECG corresponds to a high percentage of basal T-loop potentials (P213 in CGM). Two CGM variables, P136 and P280, initially thought helpful for diagnosis proved non-contributory to the prospective assessment. The reasons for this are unclear; further studies with greater patient numbers and stratification according to the distribution of the affected myocardial areas are thus warranted.

This study has several limitations. Firstly, there may be a referral bias, as all patients were sent to a tertiary medical centre for invasive cardiac assessment due to suspected myocardial ischaemia. Our results may therefore not be generalised to other populations. Secondly, the angiographic findings provide morphological information on coronary artery stenoses that only partly correlates with the presence and extent of ischaemia [23–25]. However, even though overt ischaemia may be absent at rest in asymptomatic patients with coronary artery stenoses, CGM showed a 64% sensitivity in identifying CAD patients. We hypothesize that, even in the absence of frank ischaemia, subtle electrophysiological alterations occur in CAD that may be detected by CGM, thereby identifying patients with silent myocardial ischaemia [26, 27]. Moreover, there were eleven false positive patients (seven women) with plausible reasons for myocardial ischaemia in the absence of significant coronary stenoses (tachycardia with troponin elevation, myocardial bridges [28], giant coronary arteries [29], left ventricular hypertrophy, myocardial infarction, troponin-positive acute cor pulmonale). If these cases are excluded from the analysis, specificity would rise to 91% in men and 88% in women.

In summary, a CGM score for non-invasive identification of patients with CAD has been retrospectively established and prospectively validated in a cohort of patients in a university hospital setting.

Conclusion

Based on these CGM results achieving a diagnostic sensitivity higher than the ECG with a comparable diagnostic specificity, we conclude that CGM may be a helpful tool in identifying CAD. As it is easy to apply, non-invasive, provides an automated score and can be performed at rest, we believe that it may become an inexpensive addition to the cardiological diagnostic armamentarium, possibly useful for early CAD diagnosis as well as in patients who do not tolerate exercise testing. Further studies on the diagnostic usefulness of CGM in a general population with a lower pretest probability for CAD are warranted.

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