

The First Derivation of the Frank Lead ECG at Alterations of Pulmonary Ventilation in Young Healthy Women

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Summary

The Frank orthogonal corrected ECG and its first derivation were recorded in 27 healthy volunteers (women aged 19-22 years) during normal ventilation at rest (control group), after voluntary hyperventilation lasting 75 seconds, and during hypoxic-hypercapnic ventilation (through the enlarged dead space) lasting 5 min. The projections of the magnitude and direction of the positive and negative QRS derivation maxima into the horizontal, frontal, left sagittal planes and their spatial distribution were constructed. The magnitude of the positive and negative QRS derivation maxima was significantly decreased during hypoxic-hypercapnic ventilation. A significant alteration in the direction only arose at the positive maximum during hypoxic-hypercapnic ventilation in the frontal plane. The intrinsicoid deflection was not significantly altered. The normal values of the maxima of the first QRS derivation in young healthy women are given. It is supposed that the decrease in amplitude of the maxima of the first QRS derivation is caused by slowed propagation of the depolarization wave under hypoxic-hypercapnic conditions and alteration of the direction of the positive maximum is caused by a greater participation of the right ventricle at the origin of the resulting QRS vector.

Key words

Electrocardiography – Vectorcardiography – The first derivation of ECG – Hypoxia – Hypercapnia – Hyperventilation – PQ interval – Intrinsicoid deflection – Frank orthogonal ECG

Introduction

In our previous papers, we demonstrated that both hyperventilation and hypoxic-hypercapnic ventilation prolong the QT interval of the electrocardiogram (Kujaník *et al.* 1985) and change the dependence of the QT interval on heart rate (Kujaník *et al.* 1993) and duration of the QT/QS₂ quotient (Kujaník *et al.* 1989, 1994). Prolongation of the QT interval is due to prolongation of its repolarization phase. The depolarization phase of the QT interval (QRS complex of ECG) shortens with increasing tachycardia.

However, there is not only simple QRS shortening but respiratory alterations also result in additional alterations inside the depolarization phase of the QT interval studied by instantaneous QRS vectors (Kujaník *et al.* 1991). The instantaneous QRS vectors are diminished and their direction is also altered (in the 30th millisecond of hypoxic-hypercapnic ventilation and in the 60th millisecond of hyperventilation). This

means that the QRS loop of the vectorcardiogram diminishes. We do not suppose that significant changes of the heart position are taking place, since the Frank lead ECG was registered in the mid-breath (end-tidal) position and the directions of the instantaneous vectors were not altered, with the exception of the 30th or 60th millisecond.

To explain the decrease in the instantaneous vectors we suggested (Kujaník *et al.* 1988, 1990) that an increased participation of the right ventricle plays a role in the alterations of QRS vectors. Changes in the direction of instantaneous vectors are assumed to be caused by an altered velocity of the QRS loop inscription. Its first derivative represents the velocity of its inscription. We therefore decided to investigate the alterations of the derivative maxima of the scalar QRS at hyperventilation and during hypoxic-hypercapnic ventilation.

Material and Methods

A group of 27 young female healthy volunteers (aged 19–22 years) was investigated in the supine position. The study was restricted to only one sex in order to minimize the variability of the QRS loop. The Frank lead orthogonal ECG and its first analogue derivation were recorded by electrocardiograph Chiracard 601 T (Chirana) at paper speed 100 mm/s. The electrodes were placed in the fourth intercostal space. The derivation device (Nemec and Baldovský 1982) was constructed by improvement of the original scheme of Husson (1979).

The first derivation of the Frank orthogonal ECG gives rise to several waves with different amplitudes. We evaluated the magnitude and the direction (angle) of the highest positive and highest negative waves only, since only those were clearly visible and could be measured correctly. The heart rate, PQ interval and the intrinsicoid deflection (the interval from the origin of the Q wave to the peak of the R wave) were measured from the underivated Frank lead ECG.

The planar projections of maxima of the first derivation into the horizontal, frontal, left sagittal planes (Pipberger *et al.* 1975) and their spatial counterparts were constructed from the derived leads (dx/dt, dy/dt, dz/dt) at the time of their maxima in the lead X. The formulas for calculation of the magnitude, direction, elevation and azimuth proposed by Hellerstein and Hamlin (1960) were used.

The alterations after a 75-second period of voluntary hyperventilation (submaximal ventilation with a three-fold higher ventilation rate than in the control group) and in the third and fifth minutes of hypoxic-hypercapnic ventilation (through the enlarged dead space formed by a tube of 1.3 l volume, 3 cm in diameter, 180 cm in length) were compared against the control values obtained during normal ventilation at rest.

The recording was performed immediately after the given type of ventilation in the end-tidal

position to eliminate the influence of alterations of the heart position. It was shown in our previous paper (Kujaník *et al.* 1992) that this manner of recording ensures an unaltered heart position (direction of the instantaneous QRS vectors) during quiet respiration at rest. Between hyperventilation and hypoxic-hypercapnic ventilation the heart rate was allowed to return to initial resting values.

The numerical data for each type of ventilation were calculated as the average of five consecutive heart cycles (QRS complexes) and were expressed as the arithmetical means \pm standard deviation. For statistical testing of differences, the nonparametric Z-test and Student's t-test were used by means of the program STATGRAPHICS (Graphic Software Systems, Inc.) in our Department of Medical Informatics. The $p < 0.05$ level of significance was adopted.

Results

Normal ventilation at rest (control)

The ECG values obtained during normal ventilation at rest were within normal limits and were used as the reference (control group – Tables 1–5). The heart rate and voltage of the R wave were within normal limits. Normal values of the derivation maxima are not known.

Voluntary hyperventilation

Voluntary hyperventilation lasting 75 s was accompanied by significant tachycardia, but the voltage of the R wave was not significantly altered. The values of positive and negative maxima of the first QRS derivative and of the other ECG parameters are summarized in Tables 1–5. The average magnitude of both derivation maxima was slightly decreased as compared to normal ventilation, but the differences were not significant. Duration of the intrinsicoid deflection was not significantly altered.

Table 1
Some ECG parameters

Parameter	Control	Hyperventilation	Hypoxic-hypercapnic ventilation	
			3 min	5 min
Heart rate (beats/min)	61.2 \pm 7.3	72.3 \pm 8.9***	76.1 \pm 10.3***	77.6 \pm 11.8***
R voltage lead X (mV)	0.714 \pm 0.337	0.692 \pm 0.347	0.663 \pm 0.345	0.649 \pm 0.334
R voltage lead Y (mV)	1.37 \pm 0.49	1.39 \pm 0.54	1.28 \pm 0.50	1.28 \pm 0.48
R voltage lead Z (mV)	1.08 \pm 0.30	1.04 \pm 0.29	0.98 \pm 0.28	0.99 \pm 0.23

Data are means \pm S.D. *** significantly different from controls ($p < 0.001$)

Table 2
Derivation maxima of the QRS complex of the Frank lead ECG (mV/s) and their direction (angle in grades) in the horizontal plane

Ventilation	Positive maximum		Negative maximum	
	Magnitude	Angle	Magnitude	Angle
Normal	1.14±0.36	52.4±19.1	1.11±0.29	-151.9±14.9
Hyperventilation	1.08±0.33	52.6±18.3	1.04±0.30	-155.0±16.7
H - H 3 min	1.02±0.34**	50.4±16.6	1.01±0.28	-153.2±13.0
H - H 5 min	1.03±0.30**	52.1±15.2	1.03±0.29	-152.9±14.5
H - H max	0.98±0.30***	51.0±14.8	0.97±0.24***	-153.6±13.8

Data are means ± S.D. Significantly different from normal ventilation: * $P < 0.05$, ** $P < 0.01$, *** $P < 0.001$. H-H = hypoxic - hypercapnic, Positive maximum = above the isoelectric line. Negative maximum = under the isoelectric line

Table 3
Derivation maxima of the QRS complex of the Frank lead ECG (mV/sec) and their direction (angle in grades) in the frontal plane

Ventilation	Positive maximum		Negative maximum	
	Magnitude	Angle	Magnitude	Angle
Normal	1.36±0.42	60.7±11.2	1.69±0.43	-125.8±10.7
Hyperventilation	1.35±0.42	62.5±10.3	1.64±0.42	-126.6±12.1
H-H 3 min	1.26±0.39*	58.9±13.1	1.53±0.34***	-127.1±13.3
H-H 5 min	1.24±0.38**	58.7±13.1	1.54±0.37**	-126.8±11.9
H-H max	1.18±0.39***	57.5±13.9*	1.45±0.33***	-127.6±12.3

For the legend see Table 2

Table 4
Derivation maxima of the QRS complex of the Frank lead ECG (mV/s) and their direction (angle in grades) in the left sagittal plane

Ventilation	Positive maximum		Negative maximum	
	Magnitude	Angle	Magnitude	Angle
Normal	1.50±0.46	126.8±14.0	1.47±0.44	-69.5±10.3
Hyperventilation	1.48±0.46	125.0±11.9	1.39±0.43	-72.0±11.4
H-H 3 min	1.33±0.46***	126.0±12.1	1.31±0.37*	-68.4±12.7
H-H 5 min	1.34±0.39**	128.0±13.7	1.30±0.36***	-69.9±10.6
H-H max	1.26±0.43***	127.9±13.6	1.22±0.35***	-68.9±11.8

For the legend see Table 2

Hypoxic-hypercapnic ventilation

Hypoxic-hypercapnic ventilation (through the enlarged dead space) also resulted in significant tachycardia. The voltage of R waves was slightly

decreased but the differences were not significant. The values of derivation maxima and other ECG parameters are shown in Tables 1-5. A significant decrease in the magnitude of the positive maxima was obtained in all projections, the magnitude of the

negative maxima was also significantly smaller with the exception the horizontal plane. Since the largest decrease in the magnitude of the derivation maxima sometimes occurred after 3 or 5 min of hypoxic-hypercapnic ventilation, we defined the group of "Hypoxic-hypercapnic max" as that in which the maximal alterations were included independently of the time of their onset.

The direction of the negative derivation maxima was not substantially altered. However, the direction of the positive derivation maxima was significantly changed only in the group "Hypoxic-hypercapnic max" in the frontal plane (the angle was diminished). The duration of the intrinsicoid deflection was not significantly altered.

Table 5

Derivation maxima of the QRS complex of the Frank lead ECG (mV/s) and their direction in space (azimuth and elevation in grades)

Ventilation		Magnitude	Azimuth	Elevation
Normal	PM	1.65±0.48	52.4±19.1	45.3±9.0
	NM	1.78±0.43	-151.9±14.9	-49.8±9.4
Hyperventilation	PM	1.62±0.47	52.6±18.3	47.2±8.3
	NM	1.72±0.42	-155.0±16.7	-49.6±10.7
H-H 3 min	PM	1.49±0.46**	50.4±16.6	45.5±9.9
	NM	1.61±0.33**	-153.2±13.0	-49.2±13.1
H-H 5 min	PM	1.49±0.41**	52.1±15.2	44.6±11.1
	NM	1.61±0.35	-152.9±14.5	-49.1±10.7
H-H max	PM	1.42±0.43***	51.8±15.9	44.0±11.4
	NM	1.52±0.32***	-153.1±14.2	-48.5±11.8

PM = the positive maximum, NM = the negative maximum, Azimuth = the angle in the horizontal plane between the vector and X axis, Elevation = the angle between the spatial vector and the horizontal plane. For other legend see Table 2.

Discussion

Many morphological, physical and physiological factors support the opinion that the origin of vectorcardiographic alterations are classified as respiratory variability (Ruttkay-Nedecký 1972). Furthermore, different body and heart positions can alter the cardiac electric field. The data regarding QRS alterations under different conditions were published in several previous papers (Simonson *et al.* 1957, Beswick and Jordan 1961, Ruttkay-Nedecký 1976). In our study we tried to exclude the influence of alterations of the heart position on the ECG by its recording in the middle breath (end-tidal) position.

The evidence about the unaltered heart position is based on the fact that, with one exception (the positive derivative maximum during hypoxic-hypercapnic ventilation in the frontal plane), alterations of the direction of derivation maxima rose.

Furthermore, our previous results regarding the instantaneous QRS vectors (Kujaník *et al.* 1992) support this conception. In this paper we propose the hypothesis that such alterations of the cardiac electric field are caused by altered activity of autonomic nerves. This hypothesis was supported by Ruttkay-Nedecký and Osvald (1993) in the repolarization phase of ECG (T wave) and by our results.

We do not suggest the influence of decreased electrical conductivity at pulmonary hyperinflation (the resistivity of the lungs is several times higher than that of the heart) and of the Brody's effect (blood is a better conductor than the myocardium) on the QRS complex. The reasons for this suggestion are as follows: (1) the derivated Frank lead ECG was recorded in middle breath (end-tidal) position, when no hyperinflation was present, and the amount of blood in the heart was unchanged, (2) the reciprocal influence of both these factors is contradictory, i.e. the larger blood volume in

the ventricles increases and the pulmonary hyperinflation decreases the QRS voltage, (3) the pulmonary hyperinflation and Brody's effect are present only in some heart cycles depending on the breathing phase, not in each cycle, (4) the maxima of the first derivation were constructed as an average value from at least 5 heart cycles, i.e. the differences between individual cardiac cycles were eliminated.

The first derivation of the QRS complex in Frank leads represents the velocity of propagation of the depolarization wave. Subsequently, the positive maximum of the first QRS derivation is the maximal velocity of the depolarization front at the time of the ascending R wave. The negative maximum of the first QRS derivation is the maximal velocity of the depolarization front at the time of the descending R wave. However, neither the spatial nor the planar velocity of QRS recording do not represent the conduction velocity in the myocardium. Takeuchi *et al.* (1977) reported that the correlation between the spatial magnitude and velocity is not very close.

Until now, the normal values of the derivative maxima in young healthy subjects were unknown. The maximal values of the first derivative of the Frank lead QRS complex in all three planes and in space and their direction (angle) in three planes, azimuth and elevation for young healthy women are therefore given in this study. The magnitudes are not the absolute values in mV/s but only relative ones compared to normal ventilation.

The significant decrease in the maxima of the first QRS derivation means that a significant decrease in the maximal velocities of the propagation of activation wave occurs during hypoxic-hypercapnic ventilation. When the QRS complex of the Frank lead ECG is shortened during the occurring tachycardia (Kujaník *et al.* 1992), the slope of R wave should be increased. If the derivative maxima are decreased, the velocity of depolarization and slope of the R waves are also diminished.

The duration of the intrinsicoid deflection is not significantly altered. Since the QRS complex of the ECG has a tendency to shorten (the depolarization in the 80th milliseconds occurs on hypoxic-hypercapnic ventilation only rarely), this shortening probably arises in the terminal part of the QRS. This subsequently occurs after the period of the intrinsicoid deflection and after the occurrence of the negative derivation maximum, the direction of which is not significantly altered.

We showed that the decrease in magnitude of instantaneous QRS vectors is caused by hyperventilation and hypoxic ventilation (Kujaník *et al.* 1988, 1992). To explain this effect we suggested that a higher participation of the right ventricle is involved in

the origin of the resulting QRS vector (Kujaník *et al.* 1988, 1990). During hypoxic-hypercapnic ventilation, the right ventricle works against a greater resistance because of vasoconstriction in the pulmonary vascular bed. Our current results support this hypothesis as the magnitude of derivation maxima was also decreased.

The derivation maxima can also be influenced by the voltage of the QRS complex. The decrease in QRS magnitude, with its unaltered duration, results in a diminution of the QRS slope and the first derivation of the QRS complex would be a reflection of that slope. However, this alteration was not found in our study.

It is possible to state that the positive maximum of the first QRS derivation arises in the period of depolarization of the right and left ventricles between the 30th and 40th milliseconds. The negative maximum arises after termination of depolarization of the right ventricle around the 50th milliseconds. Therefore, a decrease in the positive maximum means a decrease in the maximal velocity of the propagation of activation wave around the 35th millisecond i.e. in the period of prevailing tangential spread of the depolarization wave along the ventricular walls.

A decrease in the negative derivation maximum means a decrease in the maximal velocity of the depolarization wave around the 50th millisecond, in the period when depolarization of the left ventricle is terminating. During the hypoxic-hypercapnic ventilation a significant alteration of the derivation maxima was found, so that conditions for the slowing of impulse propagation were created only during hypoxic-hypercapnic ventilation. Hypercapnia stimulates catecholamine secretion from the suprarenal glands (Nahas *et al.* 1960). The catecholamine excretion is connected with hyperkalaemia (Williams *et al.* 1984). Hyperkalaemia and hypoxia can slow down the impulse conduction mainly between Purkinje fibres and the ventricular myocardium (Tan *et al.* 1989).

The duration of depolarization and repolarization together (the QT interval) is prolonged under hypoxic-hypercapnic conditions (Kujaník *et al.* 1985). This longer duration is caused by a prolongation of the ventricular action potentials, but action potentials of the subendocardial layer are shortened under hypoxia. Therefore, the prolongation of the QT interval occurs in the subepicardial layer and alterations of pulmonary ventilation may increase the electrophysiological heterogeneity of the ventricular myocardium.

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