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The Vectorcardiographic QRS Loop in Pulmonary Ventilation Alterations in Young Healthy Women

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Summary

The influence of some pulmonary ventilation alterations (the normal ventilation at rest = control), the hyperventilation (HV) lasting 75 s, the hypoxic-hypercapnic ventilation (HXV) lasting 3 and 6 min) on the instantaneous QRS vectors was investigated in 42 young healthy women (19-24 years old). The magnitude and the direction of instantaneous QRS vectors in the 10th to the 70th ms and in QRS max were constructed from the Frank lead ECG. The significant alterations of the direction (angle) were found in the 30th ms and QRS max at HXV and in the 60th ms at HV. A significant decrease in the magnitude of instantaneous vectors was found in the 10th to 50th ms after 6 min of HXV, in the 30th to 50th ms at 3 min of HXV, in the 40th to 50th ms at HV. These alterations were the most marked in the horizontal plane. We suggest that the alterations of the instantaneous QRS vectors were caused by the influence of the autonomic nervous system or humoral agents, but not by heart position, Brody's effect or lung hyperinflation.

Key words
Electrocardiography – Vectorcardiography – Hypoxia – Hyperventilation – Frank lead ECG

Introduction

The alterations and deviations from normal pulmonary ventilation and tissue supply with oxygen may decrease the electrical stability of the heart in experiments (Szekeres and Papp 1967, Rogers *et al.* 1973, Kujaník *et al.* 1984) and pro- long the QT interval of ECG in healthy subjects (Kujaník *et al.* 1985) as well as in patients with pulmonary diseases (Tirlapur and Mir 1982). This QT interval prolongation is realized by a prolongation of its repolarisation phase since the depolarisation phase (QRS complex of ECG) is shortening in accordance with the tachycardia. However, it is still not known if it is only the simple QRS shortening or whether other alterations in the depolarisation phase of the QT interval occur. Therefore we decided to study this problem by means of the QRS loop of the vectorcardiogram (VCG).

The QRS loop of the VCG gives a continual picture of the end points of instantaneous summary vectors of ventricular myocardium depolarisation in time and space. It is a summary of the end points of the right and left ventricular vectors. Its magnitude and form depend on the magnitude and direction of both ventricular vectors. The voltage of the resulting QRS vector may also be low at high voltage ventricular vectors situated opposite to each other. If asynchronous depolarisation of the ventricular myocardium has occurred, the resulting QRS loop will be altered in form (the end points of instantaneous QRS vectors at the individual time intervals will be localized differently) and the magnitude and direction of instantaneous vectors will also be altered.

The influence of respiratory alterations on the vectorcardiogram or the Frank orthogonal electrocardiogram was studied by several authors. The alterations found were regarded as results of the influence of heart position during breathing (Simonson *et al.* 1957, Beswick and Jordan 1961, Riekkinen and Rautaharju 1976, Yamada 1985). The influence of other factors was considered also (Fiedler 1972, Mayer 1972, Ruttkay-Nedecký 1972, 1976 a,b).

The aim of our study was to compare the alterations of the magnitude and direction of the instantaneous QRS vectors, i.e. of the magnitude and form of the QRS loop in hyperventilation and hypoxic-

hypercapnic ventilation in the inter mediate position when the loop is not influenced by the altered heart position in the thorax, i.e. by altered electrical axis of the heart.

Methods

Forty-two young healthy women (age 19-24 years) were examined in the supine position. There are some differences in the QRS loop in men and women, therefore we chose members of only one sex. The Frank orthogonal corrected electrocardiogram X,Y,Z (Frank 1956) was recorded by the apparatus Chiracard 601 T (CHIRANA) at a speed 100 mm/s and sensitivity 2 cm/mV. The electrodes were situated in the fourth intercostal space. The magnitude and the direction (angle) of the instantaneous QRS vectors in the 10th, 20th, 30th, 40th, 50th, 60th, 70th ms and at the time of the maximal QRS vector (according to lead X) in the horizontal plane (XZ) and the left sagittal one (ZY) were calculated from the Frank ECG. Since the depolarisation in the 80th ms did not occur in all subjects, the instantaneous vectors at that time were not calculated. The heart rate and PQ interval (from the lead in which it was the longest), the QRS complex duration and the voltage (amplitude) of R wave in all three leads X, Y, Z were calculated from the ECG.

The magnitude and the direction of the instantaneous QRS vectors during normal ventilation at rest were considered as the control group. The alterations at the end of hyperventilation (submaximally deep 75 s tolerated ventilation with three times higher breating rate than in the control group) and in the 3rd and 6th minute after the onset of hypoxic-hypercapnic ventilation (through the enlarged dead space = a tube 1.8 liters in volume, 30 mm in diameter and length 2550 mm) were compared with the control group. This method of producing hypercapnic hypoxaemia is predominantly used in sports training. The record was always registered in the intermediate breathing position when the expressive inspiratory and expiratory movements of the thorax were eliminated voluntarily not to give rise to bigger alterations of the heart position. Between the hyperventilation and hypoxic-hypercapnic ventilation there was a pause till the recovery of the heart rate to values compared to normal ventilation.

The numerical data for every type of ventilation were calculated from five consecutive QRS complexes as a mean ± the standard deviation (x ± S.D.). The statistical evaluation of the differences compared with control values were calculated by means of Student's two sample analysis using the programme STATGRAPHICS (Graphic Software Systems, Inc.). The differences at the level at least 0.05 were regarded as significant.

Results

A. Normal ventilation at rest (control group):

The mean heart rate from the ECG was 75.5 ± 12.4 per min. The duration of the PQ interval, QRS complex (the longest in the lead Z) and the voltage of R waves are shown in Table 1. The instantaneous QRS vectors at the individual time intervals and corresponding angles (the direction) were within the limits of normal values (Chou *et al.* 1974, Ruttkay-Nedecký 1982, Bachárová and Melotová 1986) and were used as the control group. The values in the horizontal plane in which the differences from normal ventilation were the most marked are shown in Tables 2 and 3. The depolarization in the 70th ms (i.e. QRS longer than 0.07 s) occur in all 42 subjects,in the 80th ms in the horizontal plane only in 37 subjects (88.1 per cent).

B. Hyperventilation:

The mean heart rate was increased, the QRS complex in leads X and Z was shortened. The PQ interval and R voltage were not significantly altered (see Tables 1 to 3). The magnitude of instantaneous vectors is decreased from the 40th ms. Significant alterations occurred in the 40th and 50th ms and at the time of QRS max, i.e. in the middle part of the loop. The direction of instantaneous vectors were not significantly altered till the 60th ms. Only in the 60th ms the instantaneous vector was localized more terminally than during normal ventilation (a greater part of the loop was registered during 60 ms than we can see in normal ventilation). The depolarization in the 70th ms occured in all 42 subjects, in the 80th ms in the horizontal plane only in 35 subjects (83.4 per cent).

C. Hypoxic-hypercapnic ventilation:

After 3 min of this ventilation, the heart rate was increased, the PQ interval was not significantly prolonged, the R voltage (in the lead X only) and QRS duration were significantly decreased (Table 1). After 6 min of this ventilation the heart rate was increased, the PQ interval was significantly prolonged, the QRS complex (in leads X and Z) was significantly shortened, the R voltage was significantly decreased.

It may be seen from Tab. 2 and 3 that the decrease in the instantaneous QRS vector amplitude was even more marked from the onset of depolarization till the 50th ms than in hyperventilation. It was more distinct after the sixth minute of hypoxic-hypercapnic ventilation. It was significant from the 10th to the 50th ms. In the 60th ms significant alterations did not occur and in the 70th ms after 6 min of this type of ventilation, an increase in vector magnitude occurred.

Table 1
Some ECG parameters

Parameter	Control	Hyperventilation	Hypoxic-hypercapnic ventilation	
			3 min	6min
Heart rate [min^{-1}]	75.5±12.4	92.3±17.6 P<0.00001	96.5±16.9 P<0.00001	97.7±18.8 P<0.00001
PQ interval [ms]	124.3±26.8	124.4±26.5	128.6±31.0	130.5±29.4 P<0.03
QRS lead X [ms]	84.2±9.1	80.7±8.7 P<0.0025	80.6±7.6 P<0.005	79.0±8.6 P<0.0005
QRS lead Y [ms]	85.9±10.02	86.0±9.4	84.8±10.7	84.3±10.8
QRS lead Z [ms]	98.2±9.1	95.3±8.2 P<0.007	93.8±0.2 P<0.0003 *P<0.04	94.1±7.8 P<0.002
R voltage lead X [mV]	0.7±0.2	0.6±0.2	0.5±0.2 P<0.000001 P<0.000025	0.5±0.2 P<0.00000 *P<0.0000
R voltage lead Y [mV]	1.0±0.3	1.0±0.2	1.0±0.2	0.9±0.2 P<0.009 *P<0.025
R voltage lead Z [mV]	0.8±0.2	0.7±0.2	0.7±0.2	0.7±0.2

The results are the means ± S.D.; n=42. The statistical differences against the control group, * against hyperventilation values.

The alteration in vector direction occurred in the 30th ms and at the time of QRS max, i.e. the record was accelerated compared with normal ventilation in the middle part of the loop. In the 50th ms a delay occurred compared with the 3 minutes' hypoxic-hypercapnic ventilation. The depolarization in the 70th ms occurred in all subjects, in the 80th ms in the horizontal plane after 3 min of this ventilation in 35 subjects (75 per cent) and after 6 min in 33 subjects (78.6 per cent).

Table 2

The magnitude of instantaneous QRS vectors in the horizontal plane [mV].

Time	Control	Hyperventilation	Hypoxic-hypercapnic ventilation 3 min	6 min
10th ms	0.14±0.16	0.12±0.13	0.12±0.1	0.09±0.06 P<0.03
20th ms	0.23±0.13	0.23±0.16	0.21±0.10	0.19±0.09 P<0.0006
30th ms	0.37±0.15	0.38±0.16	0.31±0.16 P<0.01 *P<0.002	0.29±0.14 P<0.0001 *P<0.002
40th ms	0.88±0.35	0.72±0.28 P<0.0006	0.65±0.23 P<0.00001 *P<0.005	0.62±0.24 P<0.0001 *P<0.002
QRS max	0.98±0.36	0.91±0.29 P<0.003	0.85±0.28 P<0.00001 *P<0.001	0.85±0.27 P<0.02
50th ms	0.75±0.36	0.64±0.32 P<0.0007	0.63±0.27 P<0.0005	0.62±0.28 P<0.0002
60th ms	0.37±0.21	0.34±0.21	0.35±0.19	0.37±0.20
70th ms	0.17±0.10	0.16±0.10	0.16±0.11	0.18±0.10 *P<0.04

The results are the means ± S.D.; n=42. The statistical differences against the control group, * against hyperventilation values.

Discussion

The ventilation manoeuvres used by us (hyperventilation and hypoxic-hypercapnic ventilation) produce a deepening of breathing which would cause extreme heart positions altering thus the electric field of the heart. We strove to eliminate the influence of extreme alterations of heart position upon the electric field of the heart by recording the Frank orthogonal electrocardiogram in the intermediate breath position and during shallow breathing in our conditions. In this way, no significant alterations of the instantaneous vector direction and therefore no significant alterations of the position and the electric axis of the heart occurred except for the 30th ms, 60th ms and at QRS max. Therefore it can not be assumed that alterations of the heart position during breathing appreciably influence the direction of instantaneous QRS vectors at the respiratory manoeuvres in our volunteers. We also do not regard the electric conduction alterations during pulmonary hyperinflation and Brody's effect (Brody 1956) as important, since this alterations can influence the instantaneous vectors in some cardiac cycles only, in dependence on the inspirium and expirium or on the heart filling with blood. They act during the whole QRS complex of ECG and not only in the 30th ms, 60th ms or in the time of QRS max. Moreover, their influences upon the voltage of the QRS complex of ECG and also upon the magnitude of the instantaneous QRS vectors are contradictory during the inspiration. They also eliminate each other at least partially.

We therefore consider the QRS loop alterations in our volunteers to be due to the influence of autonomic nerves on the electric field of the heart. It is predominantly the beta-adrenergic influence which is

Table 3

The direction of instantaneous QRS vectors in the horizontal plane [degrees].

Time	Control	Hyperventilation	Hypoxic-hypercapnic ventilation	
	3 min	6 min		
10th ms	-108.3±28.9	-106.4±24.8	-107.0±20.6	-113.9±30.0
20th ms	-69.6±21.3	-65.7±28.9	-70.4±21.3	-73.5±26.5
30th ms	-1.2±35.1	7.2±34.4	6.6±33.4	4.7±46.3 P<0.05
40th ms	51.9±29.3	44.5±28.0	48.5±27.5	46.4±27.7
QRS max	51.3±15.2	49.1±16.1	52.2±16.1	56.8±14.4 P<0.005 *P<0.00002
50th ms	70.5±22.3	70.7±25.7	72.4±22.3	63.8±25.0 **P<0.04
60th ms	102.8±26.4	113.1±26.2	111.3±25.3	104.5±33.6 P<0.02
70th ms	114.3±49.1	122.2±51.8	105.6±64.6	118.6±50.6

The statistical differences against the control, * against the hyperventilation, ** against 3 min hypoxic-hypercapnic ventilation

documented by tachycardia and shortening of the QRS. When hyperventilation lasts 75 s, we assume predominantly sympathetic nervous influences on the heart and/or local humoral influences (alkalosis, ionic alterations), since there is not enough time for the development of general humoral influences. However, hypoxic-hypercapnic ventilation lasting 3 or 6 min would provide enough time for the development of both nervous and humoral regulatory mechanisms. Circulating catecholamines, predominantly epinephrine, probably acquire the important influence on heart control under those conditions (Nahas *et al.* 1960).

The decrease in the magnitude of instantaneous QRS vectors is in agreement with the findings of Fiedler (1972) concerning hypoxic conditions. This decrease means that the QRS loop is diminishing (the end point of the instantaneous QRS vector describes the smaller trajectory in the different planes). However, the diminishing of the loop is not uniform. It diminishes most between the 40th and 50th ms (in all types of altered ventilation), less in the initial part (in the hypoxic-hypercapnic ventilation only). In the 60th ms, there are no substantial alterations in any type of ventilation. On the other hand the loop is enlarging in the 70th ms during the 6 minute hypoxic-hypercapnic ventilation. The record of the loop is accelerating, also the duration of the QRS complex of the Frank ECG is shortening and its amplitude is diminishing.

We assume that the decrease in the magnitude of instantaneous QRS vectors during changes in ventilation is predominantly caused by increased participation of the right ventricle in the shaping of the resultant QRS vector. The increase in the resulting vector of the right ventricle situated opposite the vector of the left ventricle results (at the unalterated magnitude of the summary vector of the left ventricle) in a decrease of the summary QRS vector of both ventricles.

In the 70th ms, the instantaneous vector, on the other hand, increases during hypoxic-hypercapnic

ventilation. A similar but even larger increase is typical for right ventricular hypertrophy of type C. Therefore, their common cause is probably the hypoxic vasoconstriction in the pulmonary circulation and the alterations during the hypoxic-hypercapnic ventilation can be regarded as the first phase of the QRS loop alterations during type C of right ventricular hypertrophy.

The diminution of the instantaneous vectors also occurs during deep inspiration, but it has different characteristics (Lamb 1957, Ruttkay-Nedecký 1976) since it has no effect till the 20th ms; the largest one is in the second half of the loop. The diminution under our conditions is already significant in the 10th ms and it lasts only till the 50th ms. Alteration in the direction of instantaneous QRS vectors means that the registration of QRS loop trajectory in time is altered. Although in the first 20 ms the tendency is the beginning to shorten the loop registration in the 30th ms and during the QRS max the loop registration is accelerated in the hypoxic-hypercapnic ventilation since the angle of this instantaneous vector is greater than that one during normal ventilation.

A similar situation occurs in the 60th ms at the hypoxic ventilation. Therefore the QRS loop is smaller and it is registered faster in both types of ventilation. This fact correlates with the shorter duration of the QRS complex of the ECG, eventually depolarisation in the 80th ms occurs in a lesser number of subjects than during normal ventilation at rest, respectively.

The hypoxic-hypercapnic ventilation decreases the voltage of the R wave, lengthens the PQ interval duration and alters the direction and the magnitude of the instantaneous QRS vectors more than the hyperventilation. The increase in heart rate and QRS duration is aproximately the same in these types of ventilation.

For this reason, similar alterations of the instantaneous vectors would also be expected. However, the alterations are bigger at hypoxic-hypercapnic ventilation, where the PQ interval is lengthening and the QRS complex is shortening. Perhaps, impulse conduction is also slowed down in the conductive tissue in spite of the adrenergic activation under hypoxic conditions. This opinion is supported by findings on *in vitro* myocardium of the dog (Veenstra *et al.* 1987) that acidosis and hyperkaliaemia produce the impulse conduction delay in Purkinje cells but their effect is contradictory in the ventricular myocardium – the impulse conduction delay is produced by hyperkaliaemia but the more rapid conduction by acidosis. Even more expressive Purkinje ventricular junctional conduction delay is produced by acidosis and hyperkaliaemia. The tendency to delayed loop registration in the 10th and 20th ms after 6 minutes of hypoxic-hypercapnic ventilation may also mean delay in the depolarization propagation in the initial part of the QRS loop.

Differences in the influence of hyperventilation and hypoxic-hypercapnic ventilation on the instantaneous QRS vectors may also be caused by the fact that, during inspiration sympathetic nervous influences on the heart are activated (Trzebski 1981), while the hypercapnia produced by the ventilation through the enlarged dead space stimulates the production and secretion of catecholamines from the suprarenal glands (Nahas *et al.* 1960). The distribution and density of sympathetic innervation which controls the heart predominantly by means of norepinephrine is not identical with the distribution and the density of blood capillaries from which adrenaline is predominantly transported into the myocardium. However, this problem recquires further research.

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