Vectorcardiographic Changes in Hypoxic Environment

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Abstract

VECTORCARDIOGRAPHIC (VCG) studies were made on 15 normal healthy adults under short duration moderate hypoxia in an altitude chamber at a simulated altitude of 15,000 feet. QRS maximum vector magnitude showed a significant diminution ($P<0.001$) on immediate ascent and partial recovery after a stay of 30 minutes. The magnitude increased beyond the ground level values on switching on to 100 percent oxygen breathing. Orientation of the maximum vector did not show any significant changes. The changes observed could be related to known circulatory adjustments under hypoxia. Thus VCG may be used in determining hypoxic response in a normal individual and be helpful in assessment of cases with cardiovascular abnormalities.

Introduction

Several studies have been reported on changes in electrocardiogram associated with acute exposure to moderate hypoxia. In some studies, healthy individuals were acutely exposed to altitude and changes in ECG and other parameters were correlated with possible cardiac dysfunction[7]. In others, hypoxia has been used as a stress in combination with exercise to unmask ECG abnormalities for evaluation of cases of ischaemic heart disease [1,10,18]. In such studies, many borderline ECG changes often present difficulties in interpretation as to the underlying pathophysiologic cause. ECG provides the insight into electro-physiological status of the heart which acts like a current generator inside the chest. It is well known that the magnitude as well as the waveform pattern, as recorded from the body surface electrodes, depend not only on the current generator but also on the nature of the conducting medium which is likely to change under dynamic conditions. Electrode positions and their proper weightage assume importance when volume conduction is involved in a heterogenous medium.

Short duration moderate hypoxia leads to cardiac, haemodynamic and respiratory changes which involve both the current generator as well as the conducting medium. Thus the information obtained from standard 12-lead ECG might be less revealing of these changes which could be improved by vectorcardiographic (VCG) studies. VCG provides a comprehensive three dimensional approach to the analysis with the vector concept as its multiple lead system with proper weightage is based on sound biophysical principle. With this in view, VCG studies have been conducted under short duration moderate hypoxia in a Decompression Chamber in healthy subjects and possible reasons for changes in VCG findings have been discussed.

Material and Methods

Instrumentation: Eight electrodes were applied to subject in a standard Frank lead configuration. Floating electrodes were used to minimise disturbance due to muscle tremors and care was taken to prepare the skin adequately by cleaning. The resistor network proposed by Frank was utilised to correct the distortion of the cardiac dipole field that is due to the shape of torso and the eccentric location of heart in the chest. The network output is theoretically proportional to the orthogonal components of the cardiac dipole.

Experiment: Fifteen adult healthy male subjects

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were taken up for this study. Each subject was recorded at ground level while sitting at rest inside a Decompression Chamber. The chamber was then raised quickly to a simulated altitude of 15,000 feet. Records were taken immediately after reaching the altitude and after a stay of 30 minutes while the subject was breathing rarefied air all the time. He was then given 100 percent oxygen to breathe and another record was obtained, before bringing him back to the ground level. All recordings were made at end-expiration to eliminate respiratory artefact.

Results

Maximum QRS vector (QRS_{max}) magnitude and orientation in frontal, left sagittal and horizontal planes were studied and analysed (Fig. 1). On exposure to altitude, QRS_{max} magnitude appreciably diminished in size compared to that of ground level values. It recovered to a large extent after a stay of 30 minutes at the altitude. However, its orientation did not show any appreciable change. The statistical data is given in Tables I and II. The decrease in magnitude that occurred on immediate ascent to altitude and the subsequent recovery after 30 minutes was found to be highly significant in all the three planes. The data is given in Tables III and IV. On breathing 100 percent oxygen the magnitude of QRS_{max} increased further, often becoming bigger than ground level value which is given in Table I.

TABLE I

Mean and s.d. of changes in QRS_{max} magnitude at Ground Level (GL), on ascent to altitude (0 mt), after 30 mt. of stay at altitude (30 mts) and after administering 100% oxygen (O_2).

Plane		GL	0 mt	30 mt	O_2
Frontal	Mean	1.55	1.34	1.47	1.65
	s.d.	0.57	0.55	0.56	0.67
Sagittal	Mean	1.56	1.34	1.44	1.62
	s.d.	0.64	0.65	0.60	0.44
Transverse	Mean	1.49	1.25	1.39	1.58
	s.d.	0.52	0.53	0.52	0.37

TABLE II

Mean and s.d. of changes in QRS_{max} orientation at GL and at altitude (0 mt, 30 mt) and after administering 100% oxygen (O_2).

Plane		GL	0 mt	30 mt	O_2
Frontal	Mean	46°	48°	45°	47°
	s.d.	14°	15°	12°	14°
Sagittal	Mean	136°	123°	134°	136°
	s.d.	23°	27°	28°	43°
Transverse	Mean	313°	316°	313°	310°
	s.d.	14°	16°	12°	17°

TABLE III

Mean difference, 't' values and significance of the changes of maximum QRS vector magnitude.

Plane	Mean diff.	't' values	Significance
Frontal			
GL.—0 mt	−0.21	4.20	$p<0.001$
0 mt—30 mt	+0.13	4.73	$p<0.001$
Sagittal			
GL—0 mt	−0.22	5.93	$p<0.001$
0 mt—30 mt	+0.10	4.50	$p<0.001$
Transverse			
GL—0 mt	−0.23	9.59	$p<0.001$
0 mt—30 mt	+0.15	6.40	$p<0.001$

TABLE IV

Mean difference, 't' values and significance of the changes in orientation of the maximum QRS vector.

Plane	Mean diff.	't' values	Significance
Frontal			
GL—0 mt	2.75	1.21	NS
0 mt—30 mt	−1.19	2.12	NS
Sagittal			
GL—0 mt	6.50	2.02	NS
0 mt—30 mt	−0.81	0.71	NS
Transverse			
GL—0 mt	2.87	0.74	NS
0 mt—30 mt	3.44	1.13	NS

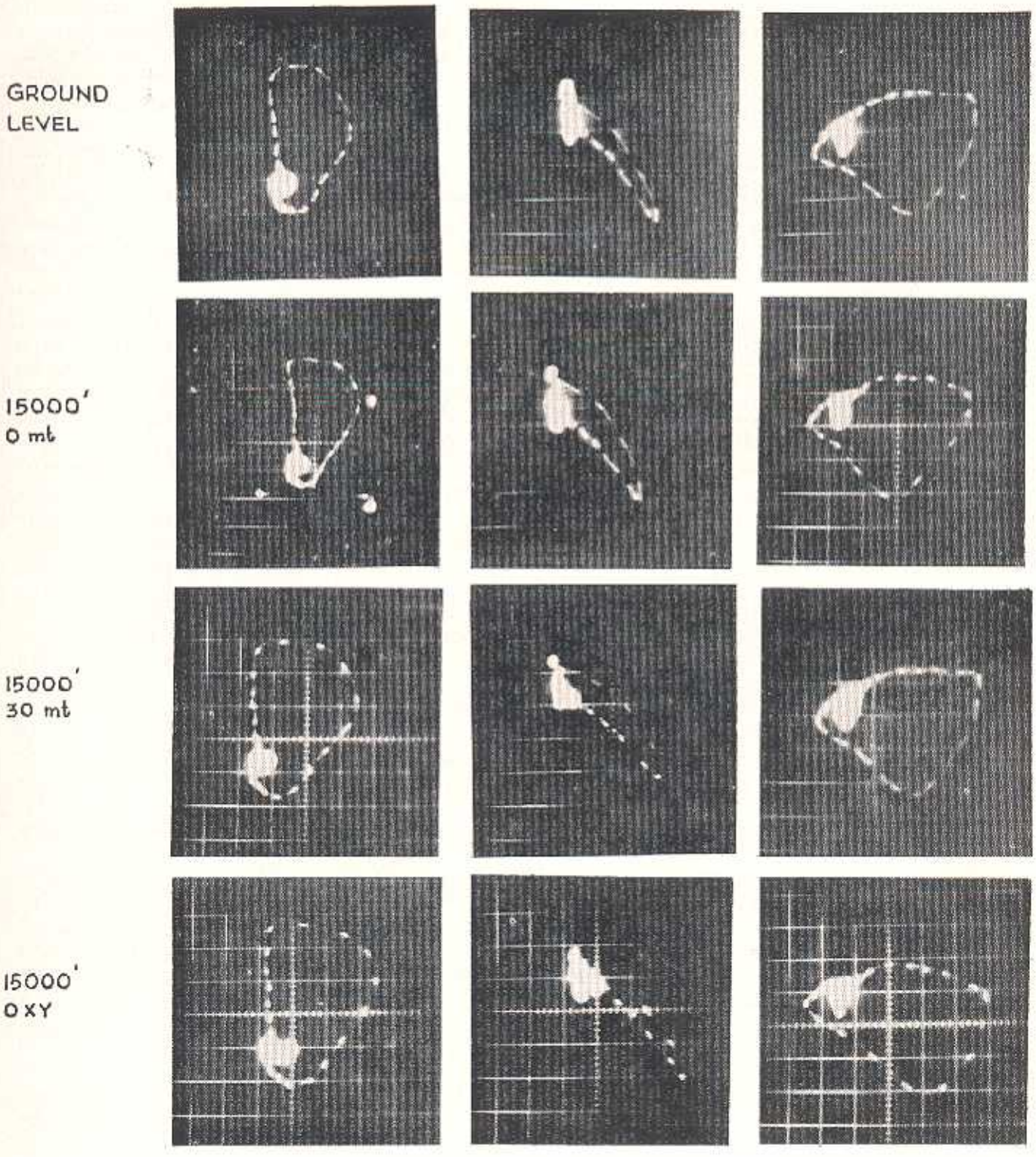

Fig. 1

Vectorcardiograms in 3 planes of a subject recorded at ground level and at simulated altitude of 15,000 ft in a Decompression Chamber immediately after ascent (0 mt), after 30 mts and after giving 100 percent oxygen (oxy). The effects of hypoxia and oxygen can be seen in the size of the vector loops and the maximum QRS vector magnitude.

Discussion

Changes in ECG have been commonly observed by many workers on exposure to altitude. These are described as clockwise rotation of the heart and right axis deviation [9-12] and have been related to hyperventilation with associated lower position of the diaphragm and right ventricular overload due to pulmonary vasoconstriction induced by hypoxia at altitude. The degree of right axis deviation has been found to be proportional to the altitude reached[8] and the duration of stay. However, on immediate exposure to hypoxia any change in electrical axis and rotation of the heart should lead to simultaneous changes in cardiac vector in mutually perpendicular planes i.e. increase in one associated with a decrease in another. This was not found to be the case with VCG findings. On immediate ascent to altitude the magnitude of QRS_{max} was noted to be reduced in all the three planes along with minor changes in orientation. The change in orientation was found to be statistically 'not significant.'

Such changes, on the other hand, could be related to the alteration of the resistivity of the thorax or the conductivity of the medium. In second manned Skylab Mission under subgravity condition, a statistically significant increase in QRS_{max} magnitude was noted in all the crewmen[19] and this was related to the centripetal shift of fluid during weightlessness. Hypobaric hypoxia leads to increased pulmonary vascular resistance thereby diverting some blood to the periphery at the initial stage and associated reduction in conductivity of the thorax. This can explain the reduction in the magnitude of max QRS vector on immediate ascent. Haemodynamic studies have shown that hypoxia leads to an increase in cardiac output. On immediate exposure this is most affected by an increase in heart rate[6] without any change in stroke volume. Unchanged stroke volume, despite the change in the left ventricular filling pressure, implies increased left ventricular contractility. Augmentation of myocardial contractility may be due to increased heart rate or sympathetic nervous system stimulation[16] both of which occur during systemic hypoxia. As the exposure to hypoxia continued, a partial recovery of the magnitude of QRS_{max} has been observed in all cases. This may be explained by two factors. Firstly, it could be due to a change in intracavity blood volume. It has been observed that cardiac output remains elevated[13] though the heart rate gradually falls towards resting level from initial rise found on immediate exposure to hypoxia. This must be associated with an increase in stroke volume and a larger intracavity blood mass. Brody[3] predicted that an increase in intracavity blood would augment potentials from radially oriented cardiac dipole and attenuate those from tangentially oriented dipoles. Since the radially oriented dipoles have the most marked influence on the QRS vector, the net effect of increased diastolic volume would then lead to increased magnitude of QRS_{max}. Millard et al[11] using a series of physiological interventions in experimental animals, have confirmed the validity of Brody effect. Secondly, the change in the magnitude of QRS_{max} could be affected by alteration in the resistivity of the thorax. Haemodynamic studies on animals[16] have shown that hypoxia leads to a significant increase in pulmonary blood volume when the exposure is continued. Thus, the volume of blood in the lungs increases at a time when the capacity of pulmonary vascular bed may be descreasing secondary to vasoconstriction. This seemingly paradoxical pair of responses, i.e. increased pulmonary blood volume as well as mean pulmonary arterial pressure may be one of the aetiological factors in the generation of high altitude pulmonary oedema [14-15-17]. Saltz et al[16] have demonstrated that the increase in pulmonary blood volume occurs within few minutes of exposure to altitude, with a secondary shift of blood from the peripheral to the central circulation. Recovery of QRS_{max} magnitude shortly after level-off at altitude as observed in the VCG could be related to this change. While studying the effect of hypoxia at 3658 meters in normal man[2] no significant change was observed in ventricular functions within 0 to 8 hours after arrival to altitude. ECG, phonocardiogram, carotid pulsation, impedance cardiogram, systolic time interval, ejection time and other cardiac indices were recorded in this study. Changes were observed only after 8 hours pointing to possible left ventricular dysfunction in addition to local changes in the pulmonary vessels.

Breathing 100 percent oxygen by a hypoxic individual leads to pulmonary vasodilatation[4] and further increase in the blood volume in the thorax. VCG showed an appropriate increase in the magnitude of QRS_{max} which could be related to increased blood volume and fall in the electrical resistivity. Paradoxical effect of oxygen on performing hypoxic

individual has been reported[3]. This phenomenon has no clear explanation, although several factors are probably involved[7]. Temporary arterial hypotension, possibly mainly from vasodilatation, can follow sudden restoration of pure oxygen to hypoxic individual. Oxygen induced constriction of cerebral arteries, possibly already affected by hypocapnoea associated with hypoxia, decreased air density and anxiety have been implicated. Coupled with hypoxia of more rapid onset in an individual performing work, hypotension and cerebral arterial constriction would then markedly aggravate the effect in the early 'recovery period.' Changes in maximum QRS vector magnitude could indicate the tendency of pulmonary vasodilatation associated with increasing central blood volume due to peripheral vasoconstriction effect with 100 percent oxygen breathing following exposure to hypoxia.

Conclusion

VCG studies have indicated certain changes in maximum QRS vector magnitude on short duration hypoxia in healthy adults which could be explained and correlated with known cardiovascular adjustment to hypoxic stress. These findings may provide additional information besides that obtained from standard ECG and be helpful not only to study the response to hypoxia in a healthy subject, but also in the assessment of cases with cardiovascular abnormalities. However, the number of subjects in the present group is small and as similar studies have not been reported in literature, further investigations are indicated and the present study is being extended further.

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