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Changes in Electrocardiogram Among Temporary Residents at High Altitude

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Abstract. A prospective study was conducted on young healthy men who were residents of plains and stayed at high altitude for 24 months—the first 12 months at an altitude of 4,200 metres and subsequent 12 months at 3,600 metres. Their ECGs were recorded in the plains before departure for high altitude and after 3, 6, 11, 18 and 23 months of stay at high altitude. After their return to the plains further ECG examinations were carried out after 1, 3 and 6 months of return. ECG changes suggestive of right ventricular hypertrophy were present in a substantial number of individuals after three months of arrival and they persisted during their stay at high altitude. The ECG changes reversed completely in majority of individuals within one month of return to plains. These changes are considered to be due to hypoxia and are reversible.

1. Introduction

Permanent residents of high altitude have a high pulmonary artery pressure as compared to residents of the plains[1,2]. The temporary residents from the plains are also liable to develop pulmonary hypertension if they stay at high altitude for a sufficiently long period. Pulmonary hypertension causes right ventricular hypertrophy/strain which is reflected in ECG[3]. Most of the ECG studies conducted so far were on a small number of permanent residents or mountaineers during the course of their expeditions which lasted for a few weeks[3,4]. Only one study was conducted on temporary residents at high altitude for one year[3] but no long term study on a large number of subjects has been done. A prospective study was conducted on 202 young healthy male individuals who stayed at high altitude for twenty-four months. The ECGs were recorded at various intervals of their stay at high altitude and on return to plains.

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2. Material and Methods

A group of 202 young healthy male volunteers between the ages 18 and 40 years were taken for study. They travelled by road to a height of 4,200 metres where they stayed for 12 months. They later came down to 3,600 metres and stayed there for another 12 months before returning to the plains. ECG examination was carried out before going to high altitude and repeated after 3, 7, 11, 18 and 23 months of stay there as well as after 1, 3 and 6 months of return to the plains. The ECG record consisted of standard limb leads I, II, III, aVR, aVL and aVF and chest leads V-1, V-2, V-4 and V-6. The stay at the high altitude was interrupted for short periods up to 60 days in a year when these volunteers came down to the plains. Twenty three individuals left high altitude permanently during the course of study. All the individuals were engaged in moderately severe to severe physical exertion to which they were accustomed to before leaving for high altitude.

3. Results

The age-wise distribution of volunteers is shown in Table 1. The majority of them were between 21 and 30 years of age. The number of persons examined and the

Table 1. Age distribution of volunteers

Ages (years)	Number of persons examined
18-20	50
21-30	126
31-40	25
41	1

Table 2. Electrocardiographic changes suggestive of right ventricular hypertrophy during the period of stay at high altitude

Period after arrival at high altitude (months)	Number examined	Right axis deviation of more than 10° in frontal plane	Clockwise rotation in horizontal plane	Inversion of T-wave in chest lead	
				V-2	V-4
3	190	84 (44.2%)	88 (46.3%)	35 (18.5%)	11 (5.8%
7	165	90 (54.5%)	90 (54.5%)	38 (23.0%)	18 (10.5%)
11	175	100 (57.1%)	92 (52.5%)	32 (18.2%)	17 (9.1%)
18	148	87 (58.7%)	49 (33.1%)	18 (12.1%)	6 (4.0%)
23	176	95 (53.9%)	69 (32.9%)	24 (13.5%)	12 (6.8%)

Table 3. Persistence of ECG changes at various intervals on return to plane.

Period after return to plains (months)	Number examined	Right axis deviation in frontal plane by more than 10°	Clockwise rotation in horizontal plane	Inversion of T-wave in chest leads V2	V4
1	154	32 (20.7%)	6 (3.8%)	6 (3.8%)	—
3	120	10 (8.3%)	4 (3.3%)	3 (2.5%)	—
6	180	Nil	Nil	Nil	—

number whose ECG showed changes after 3, 7, 11, 18 and 23 months of stay at high altitude are shown in Table 2. The ECG changes (Fig 1, 2 & 3) were suggestive of right ventricular hypertrophy because there was right axis deviation of more than 10° and clockwise rotation in horizontal plane with shift of transition zone of QRS to the left. These changes occurred in slightly less than half of the individuals within three months of arrival and were found in more than half of them during subsequent examination at 7 and 11 months. There was suggestion of right ventricular strain in the form of

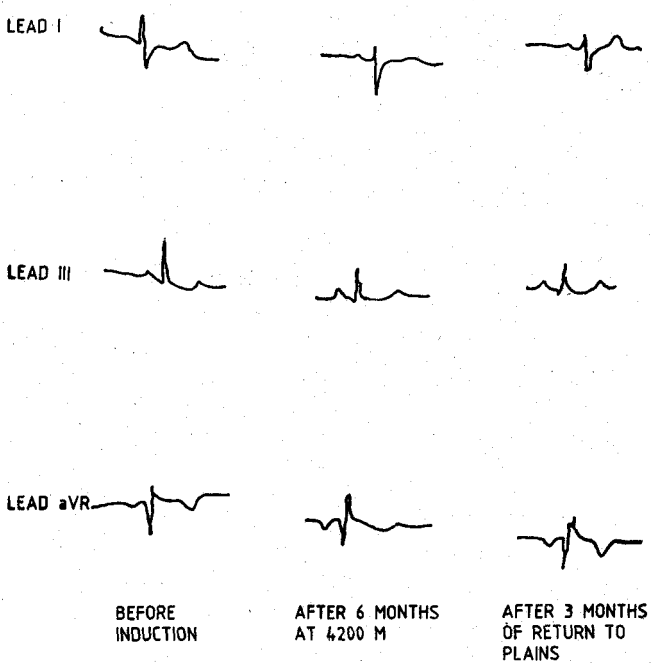

Figure 1. Clockwise rotation in frontal plane. Depth of S in lead III and height of R in aVR increased at high altitude and reversed on return to plains.

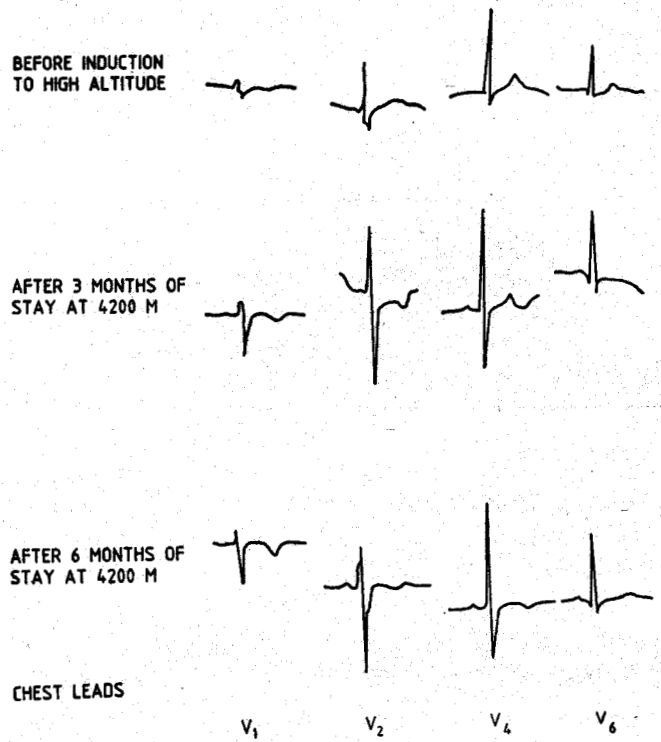

Figure 2. Clockwise rotation of electrical axis in transverse plane. Depth of S in chest lead V4 increased at high altitude. T becomes inverted in lead V2 after three months and in leads V4 also after six months of stay.

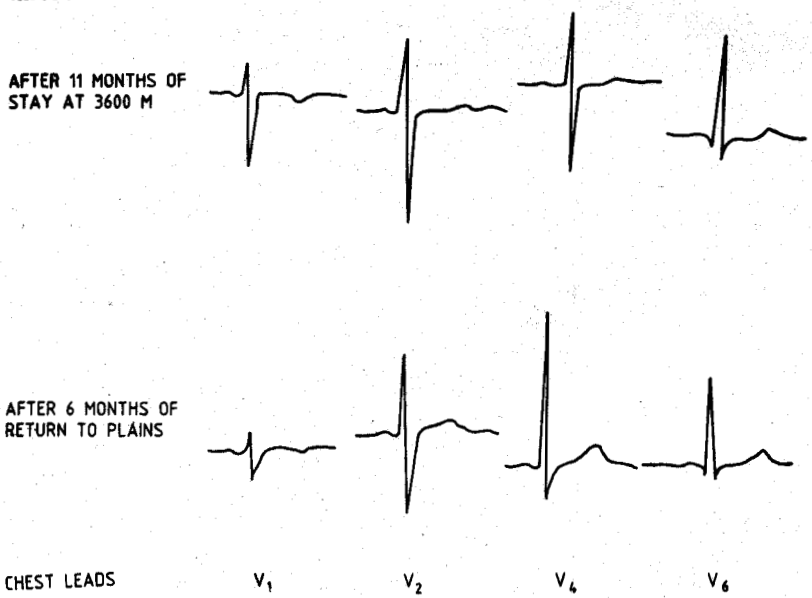

Figure 3. Reversal of clockwise rotation in transverse plane on return to plains. T wave became upright in lead V4 after descent to 3,600 metres and in lead V2 on return to plains.

inverted T wave in chest leads V_2 and V_4 in minority of subjects during the course of their stay at 4,200 metres which reversed in some cases when they descended to 3,600 metres.

The number of individuals whose ECG examination was done after 1, 3 and 6 months of their return to plain and the number who still showed ECG changes are shown in Table 3. The changes persisted in a few subjects after one and three months of return and none of the subjects showed the change after six months of return. This suggests that right ventricular hypertrophy/strain is a reversible phenomenon.

4. Discussion

The electrocardiographic changes suggestive of right ventricular hypertrophy/strain at high altitude have been reported earlier[3–6]. Penaloza[3] was the first to publish his observations on a few subjects transported by road from sea level to a height of 4,330 metres where the subjects remained for one year. Jackson and Milledge carried out electrocardiographic studies on mountaineers during their ascent to heights of 5,800 and 7,300 metres respectively[4–6]. Penaloza had also carried out an electrocardiographic study on highlanders who had been born and brought up there[1,2]. All these workers noted electrocardiographic changes suggestive of right ventricular strain/hypertrophy which they attributed to pulmonary hypertension. Haemodynamic studies on natives of high altitude revealed raised pulmonary artery pressure. In a recent study in Peru where a few young soldiers were transported rapidly by road with cardiac catheters, it was seen that there was a rise in the pulmonary artery pressure[8]. In an experimental study carried out on rats exposed to simulated altitude of 5,500 metres, right ventricular hypertrophy developed over a period of five weeks exposure and regressed following five weeks of relief from hypoxia[9,10].

The present study on young male residents of the plains who stayed at a height of 4,200 metres for 12 months followed by stay at 3,600 metres for another 12 months, showed right axis deviation (clockwise rotation of heart by more than 10° in frontal plane) and shift of transition zone to left in transverse plane in more than 50 per cent of individuals. These changes were found after three months of arrival at high altitude and persisted during their stay. They reversed in majority of subjects within one month of return to plains and in all within six months. Some of the subjects showed inverted T wave in chest lead V_2 and a still lesser number showed inverted T in chest lead V_4. This suggests that they had probably developed right ventricular strain which was also reversible on return to plain.

The susceptibility to develop right ventricular hypertrophy/strain in some persons only is evident from the results of this study. Weir *et al* had reported that cattle born and brought up at high altitude showed individual susceptibility to develop pulmonary hypertension and it is determined genetically[11,12]. In a morphometric

study of pulmonary vasculature in a group of highlanders at different heights carried out by Wagenvoort and Wagenvoort, the individual susceptibility to develop pulmonary hypertension was also seen[14]. In a recent study at Peru, haemodynamic studies were carried out on highlanders after their stay of one and a half years at sea level and subsequent arrival at high altitude. It was found that all of them did not show rise in pulmonary artery pressure. Even among those who developed the rise, it was not of the same degree. It was suggested by the authors that there is some individual factor responsible for development of high altitude pulmonary hypertension[8]. In our study of plain dwellers ECG changes in nearly half of the subjects only were found. This suggests that there is an individual susceptibility to develop pulmonary hypertension, which was however temporary and reversible.

The mechanism responsible for development of hypoxic pulmonary hypertension, as suggested by ECG changes, has remained controversial. The condition is reversible in temporary residents as brought out in the present study. It is generally accepted that hypoxia causes vasoconstriction of pulmonary vessels. This may be due to excessive release of histamine from the mast cells of the lungs, which acts directly on the muscular coat of pulmonary blood vessels[11,15]. Alternatively hypoxia may exert its direct effect on the musculature of terminal portions of pulmonary arteroles[16]. Singh & Chohan, however, consider disorders of blood coagulation to be responsible for pulmonary hypertension. According to them hypoxia causes sludging of erythrocytes and formation of fibrin thrombin[17].

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