CARDIAC ARRHYTHMIA AT HIGH ALTITUDE: EFFECT OF AGING

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The impression generally reflected in mountain medicine texts that cardiac arrhythmia is relatively rare at altitude is largely based on electrocardiographic studies at rest. (1) However, a survey of studies under conditions of exertion documents the presence of some cardiac arrhythmia at simulated high altitude or in the field in young normal subjects. The incidence correlated positively with the degree of elevation, and negatively with the degree of acclimatization.(1) As there was no published information on cardiac rhythm in older normal subjects climbing at altitudes, I recorded Holter monitor electrocardiographic data on myself on ascent and descent of Mt. Kilimanjaro (5895 m) over 5 days in 1986 at age 65.(1). A battery of cardiac studies at sea level, including coronary arteriography and thallium exercise stress showed no abnormality or arrhythmia at that time. During the ascent from 4710 to 5895 m there were frequent ventricular complexes predominantly of left ventricular origin, and multiple runs of ventricular bigeminy increasing in frequency and duration until the peak was reached. Ventricular ectopy was markedly reduced on initiation of descent, and virtually disappeared within 3 hours thereafter.

To determine the effect of age progression on cardiac rhythm under the same conditions I made an ascent of Mt. Kilimanjaro over the same route in 1996 at age 75. Sea level cardiac studies showed little change as compared to those in 1986 except that exercise capacity was less, and there were some degenerative changes in the mitral and aortic values on echocardiographic study. On ascent electrocardiogram (Holter Monitor), oxygen saturation (finger oximeter), and blood pressure (digital automatic) were recorded. A two channel Holter Monitor was employed to facilitate diagnosis of left vs right ventricular ectopy and measurement of P wave amplitude. On this occasion I turned back on reaching an altitude of 5100 m, because of increasing dyspnea and a mistaken impression that my heart rate was abnormally slow (oximeter dysfunction).

RESULTS

Holter data are displayed in Table 1. Maximal heart rate during climb was 85% of maximal rate during treadmill exercise test at sea level, and heart rate during sleep was comparable to that at sea level. Frequency of premature complexes of left ventricular origin (LV, VPC's) increased progressively at higher altitude during climb at rest, and during sleep. During ascent, at the highest altitude, a maximum of 56 complexes per hour developed, falling to 10 per hour shortly after initiation of descent. Ventricular complexes of right ventricular origin (RV, VPC's) were relatively infrequent, with no clear relations to altitude. Short runs of left ventricular tachycardia occurred during climb, rest and sleep, with increasing frequency during ascent at higher altitude, with a 14 complex run at 250 bpm during climb near 5100 m (Fig. 1). No right ventricular tachycardia occurred. Atrial tachycardia occurred predominately during sleep. No ST depression consonant with ventricular ischemia (duration > 20 sec) occurred. Amplitude of the P wave in lead V1 rose progressively during climb, rest, and sleep with increasing altitude, falling on descent.

Blood oxygen saturation fell progressively from 92% at 1800 m to 71% at 5100 m during climb. Blood pressure levels during ascent were not significantly different from those observed at comparable heart rate during treadmill exercise at sea level min.120/60, max. 160/80 mmHg).

DISCUSSION

Duration of the ascent from 4700 to 5100 m was approximately the same, 2_ - 3 hours, as that 10 years before. Average levels of heart rate and incidence of ventricular ectopy during climb were higher in this study than previously (123 vs. 116 bpm, and 56 vs 50 VPC's/hour). During the first two hours of descent from the highest altitude reached heart rate fell little in this study vs the earlier (120 vs 102 bpm), but ventricular ectopic frequency fell strikingly in both to 10 vs 9 VPC's/hour). In contrast to the prior study when ventricular tachycardia was not observed, 9 episodes were documented in this study. The low incidence of right ventricular ectopy was comparable in the two studies, but the degree of P wave amplitude increase was somewhat greater in this study (0.5 – 1.5 vs 0.1 – 0.2 mm).

Although cardiac arrhythmia has not been observed at rest in younger men at altitudes as high as 8848 m, premature ventricular complexes, ventricular bigeminy, and premature atrial complexes have been recorded during or after exercise in healthy men over an age range of 20 – 53 years at altitudes from 4600 to 7620 m.(1) There is experimental support for the postulate that this ectopy relates to increased sympathetic neural activity. Increased sympathetic nerve traffic is demonstrable on direct intraneural recording in young subjects during acute exposure to ambient hypoxia, with a synergistic effect of exercise and rapid rise in noreprinephrine levels.(2) Sympathetic stimulation and catecholamine release may bring about delayed after-depolarizations and triggering of arrhythmia in atria or ventricles.(3) Plasma catecholamine levels for given exercise levels (% Vo2 max) are higher in older than in young subjects, and exercise induced ventricular arrhythmia is frequent in the former group.(4)

Of note in both studies reported here is the sharp decrement in ventricular ectopy with lesser exertion during descent vs ascent at the same altitude. Greater incidence of ventricular ectopy and development of ventricular tachycardia in the second study at age 75 suggests increased sympathetic stimulation, not only during climb but during sleep as well. As far as the author is aware, there is the first report of ventricular tachycardia during sleep (6 complexes, 170 bpm, at 4700 m) at high altitude. This suggests that the sympathetic response to hypoxia as well as that to exercise may be enhanced in the older subject.

Table 1. Holter Data on Ascent 1800 m to 5100 m, and P wave Amplitude Descent 5100 m to 2700 m. Values for VPC's Represent Average Numbers Over the Period of Designated Activity.

	1800 m to 2700 m	2700 m to 3720 m	3720 m to 4000 m	3720 m to 4700 m	4700 m to 5100 m	5100 m to 4700 m	4700 m to 2700 m
MAX HR, BPM DURING CLIMB	132	138	121	121	123	120	130

Min HR, bpm during sleep⊗		45	38	48	56			
LV, VPC'S/HR								
	Climb	4	8	7	16	56	10	21
	Rest⊗	1	8	8	11			
	Sleep⊗	2	3	4	6			
RV, VPC'S/HR								
	Climb	4	2	2	1	6	2	5
	Rest⊗	0	4	1	4			
	Sleep⊗	1	1	3	2			
LV, Ventricular Tachycardia *		4 (105) Sleep		3 (135) Rest	3 (192) Climb	3 (121) Climb	4 (207) Climb	
					6 (70) Sleep	14 (251) Climb	3 (105) Climb	
							4 (108) Climb	
Atrial Tachycardia *			6 (145) Sleep	5 (165) Rest	7 (200) Climb			
				3 (133) Sleep	8 (168) Sleep			
				9 (163) Sleep	6 (158) Sleep			
P Wave Amplitude, mm⊥		0.7, 0.5, 0.5	1.0, 0.7, 1.0	1.2, 1.0, 1.0	1.5, 1.0, 1.1	1.5	1	1.0, 0.7, 0.5

* First figure refers 40 number of complexes, send in parentheses to bpm
⊥Measurements are from lead V1: during climb at rest, and during sleep respectively
⊗Data secured at the higher altitude. HR, heart rate. LV, left ventricle. RV, right ventricle. VPC's, ventricular premature complexes

As regards possible implications of these findings, it may be noted that a survey of deaths among mountain bikers in Austria during the period 1985 – 1992 indicated that 30% of 210 deaths were sudden, that over 50% occurred in men over 60 years, and there was an increased risk with physical exertion.(5) In a survey of deaths occurring in British expeditions at altitudes greater than 6500 m during the period 1968 – 1987, 3 of 15 deaths were of uncertain cause.(6) Thus cardiac arrhythmia may account for a significant percentage of fatalities at high altitude, as well as syncopal episodes, and perhaps some of the deaths attributed to "falls". Thus it would seem prudent to appraise the propensity to arrhythmia at sea level with appropriate recommendations of activity in certain potentially susceptible populations such as older persons, and those with known cardiovascular disease who may anticipate ascent to high altitude.

CONCLUSIONS

The incidence of ventricular ectopy, largely of left ventricular origin, increased in

association with development of ventricular tachycardia in this older normal subject at age 75 vs 65 years under comparable temporal and logistic conditions during climb at high altitude. The degree of exertion (ascent vs descent) had a modulating effect on ectopy on both occasions. These findings, together with the development of ventricular tachycardia during sleep on the second altitude exposure, suggest a progressively increasing sympathetic response to exercise under hypoxic conditions, and the possibility of increased sympathetic sensitivity to the hypoxic stimulus with advancing age. Also the focus is on sympathetic stimulation of left ventricular arrhythmia rather than pulmonary hypertension in arrhythmogenesis under unacclimatized conditions at high altitude.

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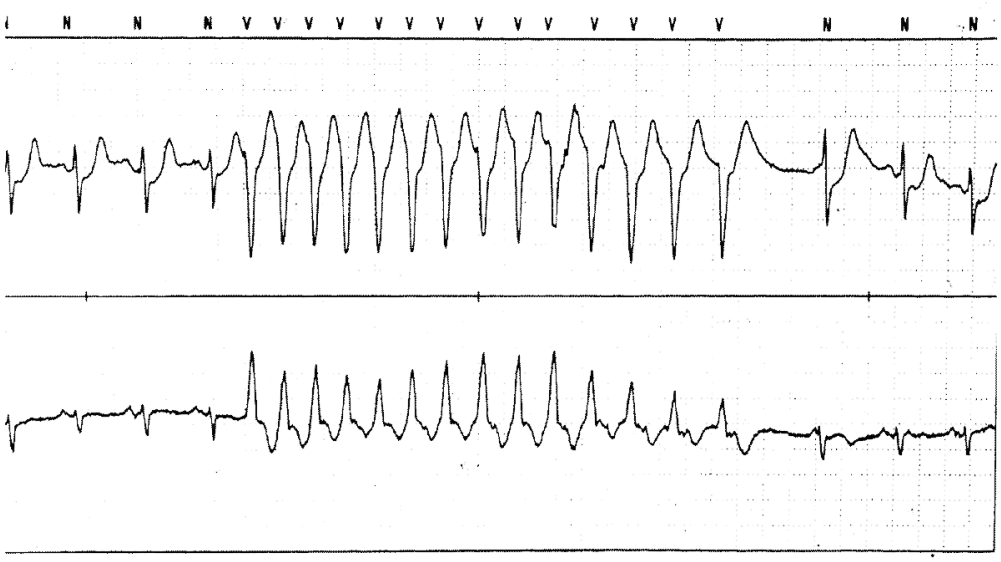
(7)

FIGURE 1 Fourteen complex run of left ventricular tachycardia at 250 bpm during climb near 5100 m altitude. ECG lead I above, lead V1 below.



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