Detection of Cardiac Arrhythmia at Very High Altitude Using a Wearable ECG Sensor

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INTRODUCTION

The fact that sudden cardiac death (SCD) is the most common cause of non-traumatic death in males at high altitude has generated much interest in the effect of hypobaric hypoxia on heart rhythms[1,2,3,4]. It is thought to be pro-arrhythmic and palpitations are commonly reported, but until recently, ECG equipment was ill-suited for large volume data collection[4,5]. Consequently, there is little hard data on the subject. Recent small studies indicate that altitude is associated with brady and tachyarrhythmia, though not ventricular arrhythmia associated with SCD[3,4,5]. In order to add to the body of knowledge, this pilot study used small, lightweight sensors to gather continuous ECG data on subjects at low, high, very high and extreme altitude (*Table 1*).

METHODS

Wearable ECG sensors (Cardea Solo, Cardiac Insight, USA) were used to gather 28 days of continuous cardiac data on 5 subjects before, during and after climbing Denali (20,310 ft./6190 m), the highest point in North America (*Ill. 1*) Subjects were four males and one female ages 34.8±5.9 years (range: 28 – 41). All had a normal cardiovascular exam and gave written informed consent prior to study participation.

Subjects travelled to Anchorage, Alaska on May 14th, 2018 (Day 0) then drove to Talkeetna (348 ft./108 m). The following day (Day 1) the first sensors were applied. A single button on the sensor initiates recording, then adds markers to the data. Each subject was given a small notebook with instructions on recording symptoms, sleep/wake times and intake of caffeine or medications.

Illustration 1. The route up the West Buttress of Denali (Mt. McKinley).

Each sensor was worn for seven days before being replaced.

On Day 4 the team flew to the Talkeetna Glacier and hiked to Camp 1 at 7800 feet (2377 m). Subsequent camps were at 11,000 ft. (3353 m), 14,200 ft. (4328 m) and 17,200 ft. (5243 m). 4 of 5 subjects reached the summit (20,310 ft./6190 m) while one remained at the highest camp. Monitoring continued after subjects returned to their homes at altitudes less than 482 ft. (147 m) except for one, who resides at 4400 ft. (1341 m).

Data from each of the 20 sensors was analyzed with proprietary software (Cardea Solo, v. 2.8.0.11). ECG's were manually reviewed to confirm computer identified abnormalities and to find additional arrhythmic events.

Meters	Altitude	Feet
> 5500	Extreme	> 18,045
3500 - 5500	Very High	11,483 - 18,045
1500 - 3500	High	4,921 - 11,483
0 - 1500	Low	0 - 4921

Table 1. Altitude definitions[7].

RESULTS

Five subjects had continuous ECG monitoring for 26.8±2.8 days (range: 22.8–29.4). Total duration was 3,213 hours (134 days). Software was able to analyze 86% of this (2,761 hrs.) while the remaining 14% had poor signal quality.

One of five subjects had 39 non-conducted P waves during the study period. *Figure 1* shows the altitude profile of the team and the number of these events. 34 occurred sleeping at the highest camp (17,200 ft./5243 m). This subject did not go to the summit before the last night sleeping there. The following night, the team slept at 11,000 ft. (3353 m) and there were an additional 4 dropped beats. For the next 10 days there were none, until the last night of monitoring when there was one additional dropped beat while sleeping at 482 ft. (147 m). These 39 non-conducted P waves resulted in pauses that averaged 2717± 416 ms (range: 2138 – 4043). The longest pause of 4043 ms was the sum of two consecutive non-conducted P waves and occurred on the fourth night sleeping at 17,200 ft. (5243 m) (*Figure 2*). PR prolongation preceded most pauses.

No subject reported palpitations or symptoms that could be attributed to arrhythmia. The climbers did suffer from rhinorrhea, cough, reactive airway (wheezing), heartburn, acute mountain sickness (headache and nausea), hypothermia, and profound exhaustion.

DISCUSSION

The findings of this study are consistent with other recent work which has found pauses, supraventricular tachycardia, atrial fibrillation, atrial flutter, and ST depression at >4100 m (13,451 ft.).[3,4,5] Pauses are the most common finding, seen in 8 of 15 subjects at >4800 m in one study.[5]

Bradycardia and pauses are frequent in patients with sleep apnea.[8] Since periodic breathing is common at high altitude, one wonders if a similar relationship may be present.[6] Monitoring nocturnal oxymetry and respiratory patterns along with ECG would help clarify relationships between respiration and arrhythmia at altitude.

Future work will involve larger numbers of subjects and the collection of data on these additional variables. More data would enable estimates of the prevalence and variety of altitude induced arrhythmia. Of particular need is data from older individuals and those with cardiovascular risk factors. This could help guide recommendations for individuals traveling to high altitude for recreation.

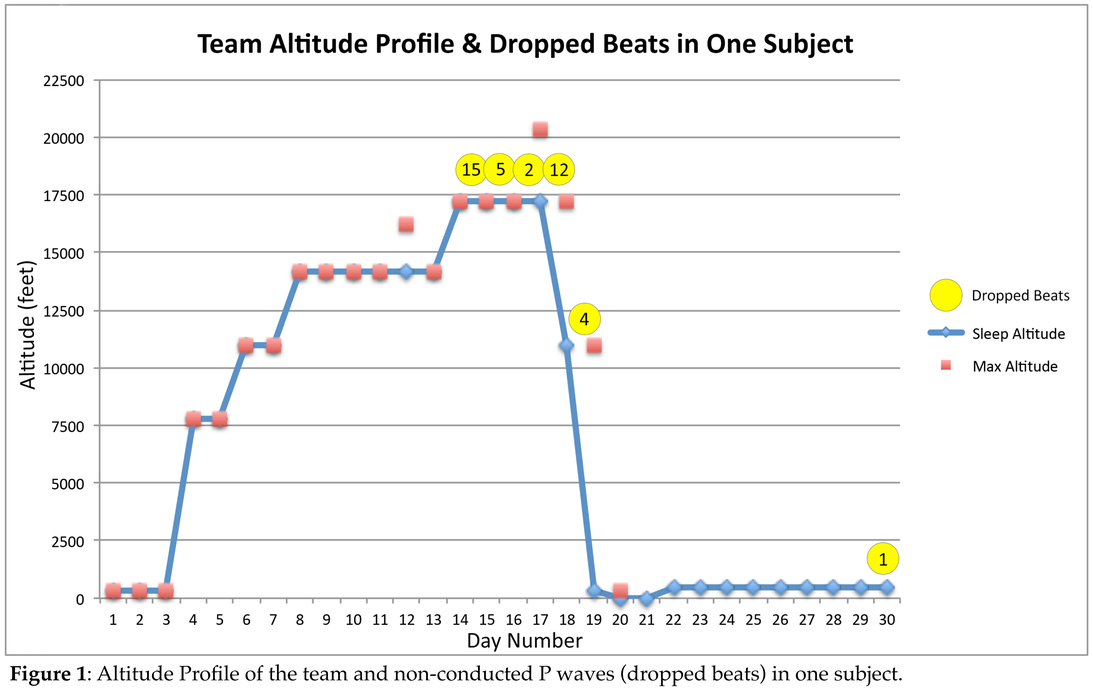

Figure 1: Altitude Profile of the team and non-conducted P waves (dropped beats) in one subject.

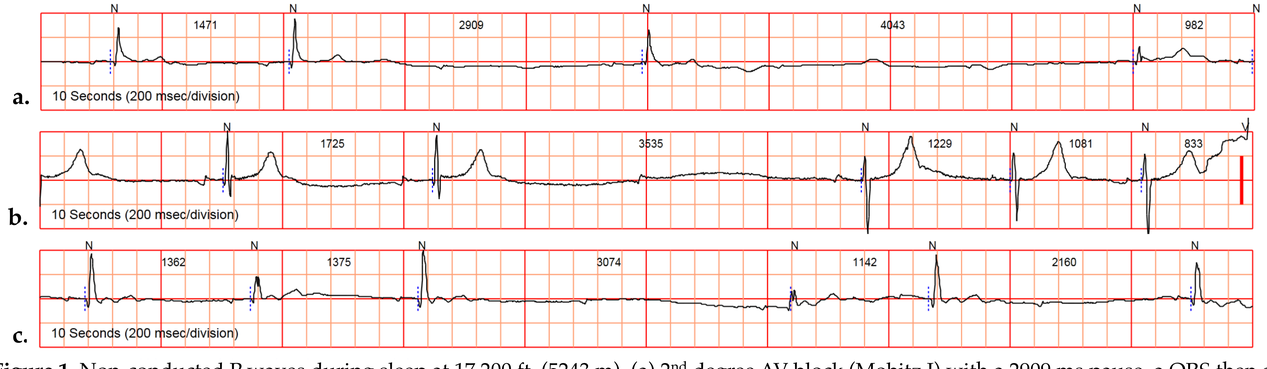

Figure 1. Non-conducted P waves during sleep at 17,200 ft. (5243 m) (a) 2nd degree AV block (Mobitz I) with a 2909 ms pause, a QRS then a 4043 ms pause containing two non-conducted P waves. Day 18. (b) Mobitz I with a 3535 ms pause. Day 16. (c) Mobitz II then Mobitz I patterns with 3074 and 2160 ms pauses. Day 18.

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