CASE REPORTS

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Incapacitating ventilatory drive at high altitude

In October, 1997, I participated in a trek to the northeast face of Everest, the Kangshung face, with 6 others, Sherpas and yaks with herders. In preparation I had walked almost daily for about 3 months, climbed local hills of 300m or so many times at a rate of about 300m/hr, with only the expected (at my age of 75) moderate shortness of breath. After a week in Tibet, largely at 3650m, we climbed the first day from 3800m to 4240m over about 6 km. I liberally used an over-shoulder water supply and probably drank 1.5 liters during the climb. Even slow and moderate climbing caused me unexpectedly severe hyperventilation, and I was not able to keep up with the others, eventually needing to be relieved of my 7 kg day pack.

We each recorded oxygen saturation (SpO_2) with an Onyx pulse oximeter (NONIN) which is a self contained finger tip device with batteries and display on the finger, weight about 30g, and remarkably accurate. On arriving at 4240m, after a brief rest, my SpO_2 was 88%. The others ranged from 85-89%. Immediately after walking slowly 50M up a 10% grade my SpO_2 fell to 74% (heart rate 120), then returned to 84% in about 4 min of rest. Two younger fit members of the group walked more rapidly up the same grade, and their SpO_2 fell briefly to 77% and 78%, (HR 150 in both) while a 65 yr old male showed no fall, from 84% at rest to 84% immediately after the same ascent. Later my SpO_2 settled at about 84%. I was able to increase it to 88%-90% at rest by apneustic breathing without hyperventilation, by which I mean repeatedly taking a deep inspiration, breathholding at near TLC for 5-10 s, then expiration to FRC and immediate re-inspiration to near TLC. I assume this rise of saturation was due to opening of critically closed alveoli in my aging lungs.

I had no AMS symptoms, except a slightly reduced appetite, and no HAPE symptoms. Even slight exertion resulted in marked ventilatory drive. When I lay down even before getting into my sleeping bag, the posture change alone stimulated vigorous hyperventilation for 2-4 min, after which I was comfortable, felt normal and was soon asleep. I developed a diuresis, with which a full bladder awoke me about once an hour for the first 5 hrs of sleep, and then slept well. I was not aware of any peripheral edema, but did experience an unusual large wet stool in the early evening, without abdominal symptoms or other sequellae or further diarrhea, possibly a sign of visceral edema. The plan for the next 10 days was to climb over 5300m, descend into a valley at less than 4000m, perhaps do some minor climbing, then climb back at the end over another 5300m pass to return to the same roadhead. There is no other way out of that valley, and no facility for rescue should one be necessary, except the help of others and the animals. In the morning, the strong ventilatory drive with even the exertion of dressing persisted. So despite feeling perfectly well at rest, I decided not to continue, probably over-reaction from anxiety. I was easily able to walk back down to the road head with my day pack, and was then driven to Lhasa (a 2 day trip). I noted some continuation of the unusually strong ventilatory response to mild exercise for several days until I returned to low altitude

(Kathmandu). I was given a diagnosis of early HAPE in Kathmandu although I never had orthopnea, cough, substernal burning, pressure or pain, or other HAPE symptoms, and no undue desaturation at rest at altitude.

I suspect my experience represented a combination of three other factors:

1) A strong peripheral chemoreceptor drive, intensified by a week at Lhasa's altitude. At sea level, my HVR exceeds 1 L/min/%, and doubled with 12 days at 3810m altitude. One of our group, a 42 year old female pediatrician, experienced no increased ventilatory drive on 2 different occasions at over 5300M (by car) when her SpO_2 fell to 60% and 68% with short walks, suggesting a low HVR. She completed the trek without difficulty.

2) Greater than normal fall of O_2 saturation due to pulmonary shunting through critically closed alveoli as PA pressure rises with exercise. The rise of critical closing capacity above FRC is a well documented part of aging. These unventilated regions, at rest, may have little blood flow due to local hypoxic vasoconstriction, but with an increase in cardiac output and pulmonary arterial pressure augmented by altitude hypoxia, their precapillary sphincters may be forced open. Such exercise-induced shunt has been widely recognised, perhaps more often called a rise in low V/Q regions. I suspect that shunting also occurs upon lying down as pooled more desaturated venous blood floods into the lesser circulation, lowering arterial SaO_2 and perhaps further raising pulmonary arterial and/or wedge pressure;

3) Systemic fluid accumulation associated with a rise in central venous and right atrial pressure due to the right ventricular response to its rising afterload from hypoxic pulmonary hypertension and the associated exercise of climbing. Peripheral edema is also rather commonly observed at high altitude, although it is not usually assumed to represent right heart "failure".

I suspect this experience may be common. I am inclined to call it HAD for high altitude Dropsy, where D may also connote Drive of ventilation from Desaturation due to shunts.

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Acute high altitude hypoventilation that resulted in hyperventilation following a hyperoxic test.

It is well known that on ascent to high altitude, the human body has several compensating mechanisms. Hyperventilation is the most significant. Some authors (1,2,3,8) suspect that a poor hypoxic ventilatory response (HVR) may be related to the presentation of Acute Mountain Sickness (AMS). We have observed, that some people with acute mountain sickness, hypoventilate, and hence have a low partial arterial oxygen tension (PaO_2), on arrival to 3510 m. Here we present one case that has relative hypoventilation and in which we performed basic pulmonary testing including hyperoxia.

CASE REPORT
A 47 years old, white male Chilean physician on a trip to Sucre (2500 m.) for a Medical conference, remained in La Paz (3510 m.) for a couple of days. A few hours after his arrival at the airport (4100 m), he began to have a headache, that gradually increased, and was unrelieved by acetaminophen or aspirin. He described that he had a slight flu before his trip. He had never smoked and had not ingested any alcohol prior to or after his arrival. He described an episode of cerebral stroke some years ago, that left no sequelae and had a normal life in his country.
His weight was 68 K. Height 1.80 m BP: 136/97 mmHg Temp: 36.7°C Pulse: 75/min Resp: 18/min. The electrocardiogram , chest examination and chest x-ray were normal. RBC count: 4.7 million/mm3, Hematocrit = 42%, Hemoglobin = 14 gm%, WBC count: 8200 with 80 % neutrophils, 18 % lymphocytes and 2 % monocytes. The day after arrival, blood gases breathing ambient air and 100 % oxygen by Hans Rudolph mouthvalves are shown in table 1. Ventilation was measured using a mouthpiece attached to a Hans-Rudolph valve and recollected in a Tissot, through an Analog-Digital converter hooked to an Apple II+ computer, during five minutes. Capillary blood, alveolar and expired air were measured in a PHM71 Mk2 Acid-Base Analyzer from Radiometer. For the hyperoxic tests a Douglas bag was filled with oxygen and connected to the input respiratory valve and ventilation measurements were repeated.

DISCUSSION:
Recent studies (9) show that at moderate altitudes (up to 3000 m) 25% of visitors suffer from acute mountain sickness. This was associated with poor physical conditioning or underlying pulmonary problems. Although some studies have shown that people with the highest ventilatory response to hypoxia have the least symptoms of AMS (10,2,8), others fail to show a correlation (11,12) which makes the subject a controversial matter. Relative hypoventilation in AMS has been previously described (10).

It is apparent that in this case there is relative hypoventilation. Relativity applied to hyperventilation at high altitude has been discussed before in our www page above (7). It is also evident that when given oxygen there is an increase in alveolar ventilation. This may be related to the paradoxical hyperventilation of some normal persons when given oxygen to breathe at altitude (4). Subjects with HAPE have also been found to hyperventilate on supplemental oxygen (15). As we previously showed (5), Chronic Mountain Sickness (CMS) patients demonstrate this type of response with a higher incidence than normal inhabitants of high altitude. Kryger et. al. (6) show hyperventilation on oxygen administration to be present only in CMS patients. It is significant that when given 100 % oxygen no intra-pulmonary shunt abnormalities were observed. Furthermore, the maximum PIO_2 achieved at 3600 m from a 98% oxygen cannula corresponds to approximately 430 mmHg, and with this mixture a normal person achieves a PaO_2 of at least 200 mmHg (7). This case confirms Bartsch's observations that there is a widened A-a O_2 difference. But we question ourselves, shouldn't the patient be hyperventilating with such low PaO_2? At 2500 m, he felt very well in Sucre and one week later returned to La Paz, for two days but had no symptoms whatsoever. From this, we make the supposition that the hypoventilatory response was transitory. Unfortunately we were unable to measure

his ventilation and blood gases a second time. Clinical normality with no headache suggested a normal physiological response. Later, blood gases measured at sea level were completely normal. This implies that either the respiratory center "learned" the appropriate reaction to hypoxia or that a reversible infection (perhaps viral) temporarily diminished his CNS ventilatory response. We don't know, however if the paradoxic hyperventilation on oxygen administration is a permanent event in most of these patients with AMS.

Although a person may have a normal response to hypoxia before the ascent, some mechanism such as viral or bacterial disease may temporarily alter their respiratory center, thereby producing a limited hypoxic ventilatory response. We have observed another case of a Japanese in whom clinically, hypoventilation was evident with a respiratory frequency of 8 per minute. Temporary hypoventilation on ascent to altitude may be an explanation for some cases of maladaptation to high altitude. These patients when given 100 % oxygen, as a test, may hyperventilate, which implies a temporary disorder of the respiratory center, not completely explained until now.

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	Breathing ambient air					**Breathing 100 % Oxygen**		
	PaO2 mmHg	PaCO2 mmHg	PAO2 mmHg	PACO2 mmHg	VA(BTPS) ml/min/m2	PaO2 mmHg	PaCO2 mmHg	VA(BTPS) ml/min/m2
NORMAL	60	30	60	30	4200	195	32	3910
CASE	38	33	52	37	1297	215	31	1467

Table 1. Acid-base status and ventilation of normal newcomers (13,7) and this case breathing ambient air and during hyperoxia. VA=Ventilation in ml/min/m2 calculated from VA=VCO2*.863/PACO2

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