The Disaster of 96: An educational way of explaining the physiological reactions produced as a consequence of exposure to low oxygen pressure at high altitude using the film *Everest* (2015)

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Received 20 November 2017; accepted 4 December 2017.

How to cite this paper: Domínguez Vías G. The Disaster of 96: An educational way of explaining the physiological reactions produced as a consequence of exposure to low oxygen pressure at high altitude using the film *Everest* (2015). J Med Mov [Internet] 2018;14(4): 227-236.

Summary

The 96 Mount Everest Disaster refers to the events that took place from May 10 to 11, 1996, when eight people caught in a storm were died, some during the ascent and, those who had already reached the Summit, while they descended. The film *Everest* (2015) faithfully reflects the previous symptoms that occurred during ascension, an important reason to understand the effects of altitude and low gas pressures on the human body. In this paper we address both problems, *Everest* can help students to understand and reflect on the challenges for body homeostasis that take place at great heights.

Keywords: Altitude, Barometric pressure, Alveolar oxygen pressure, Hypoxia, Pulmonary edema, Cerebral edema.

El Desastre del 96: Una forma educativa de explicar las reacciones fisiológicas producidas como consecuencia de la exposición a la baja presión de oxígeno a gran altitud usando la película *Everest* (2015)

Resumen

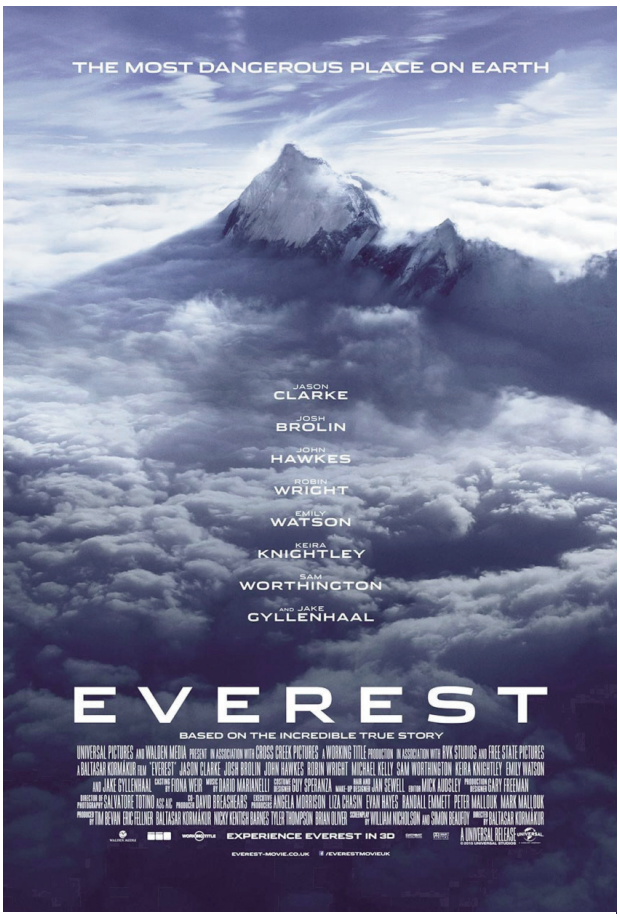
El Desastre del 96 del Monte Everest se refiere a los eventos ocurrido del 10 al 11 de mayo de 1996, cuando ocho personas atrapadas en un temporal perdieron la vida, algunos durante el ascenso y, aquellos que ya habían hecho cumbre, mientras descendían. La película *Everest* (2015) refleja fielmente los síntomas previos ocurridos durante la ascensión, razón importante para comprender los efectos de la altura y de las bajas presiones de gases sobre el cuerpo humano. En este trabajo se aborda ambos problemas, *Everest* puede ayudar al alumnado a entender y reflexionar sobre los desafíos para la homeostasis corporal que tienen lugar a grandes alturas.

Palabras clave: altitud, presión barométrica, presión de oxigeno alveolar, hipoxia, edema pulmonar, edema cerebral.

The author state that this article is original and has not been previously published.

Technical Details

Title: *Everest*.
Original title: *Everest*.
Country: USA.
Year: 2015.
Director: Baltasar Kormákur.
Music: Dario Marianelli.
Cinematography: Salvatore Totino.
Film Editing: Mick Audsley.
Screenwriters: William Nicholson, Simon Beaufoy.
Cast: Jason Clarke (Rob Hall), Ang Phula Sherpa (Ang Dorjee), Thomas M. Wright (Michael Groom), Martin Henderson (Andy 'Harold' Harris), Tom Goodman-Hill (Neal Beidleman), Charlotte Bøving (Lene Gammelgaard), Pemba Sherpa (Lopsang), Amy Shindler (Charlotte Fox), Simon Harrison (Tim Madsen), Chris Reilly (Klev Schoening), John Hawkes (Doug Hansen), Naoko Mori (Yasuko Namba), Michael Kelly (Jon Krakauer), Tim Dantay (John Taske), Todd Boyce (Frank Fischbeck), Mark Derwin (Lou Kasischke), Emily Watson (Helen Wilton), Sam Worthington (Guy Cotter), Keira Knightley (Jan Hall), Elizabeth Debicki (Caroline MacKenzie), Josh Brolin (Beck Weathers), Justin Salinger (Ian Woodall), Jake Gyllenhaal (Scott Fischer), Vanessa Kirby (Sandy Hill Pittman), Robin Wright (Peach Weathers), Mia Goth (Meg Weathers), Stormur Jón Kormákur Baltasarsson (Bub Weathers), Ingvar Eggert Sigurðsson (Anatoli Boukreev), Demetri Goritsas (Stuart Hutchinson), Chike Chan (Makalu Gau), Micah Hauptman (David Breashears), Clive Standen (Ed Viesturs), Nancy Baldwin (Janie), Lucy Newman-Williams (Linda), Vijay Lama (Colonel Madan), Avin Shah (Co-Pilot), …
Color: Color.
Runtime: 121 minutes.
Genres: Action. Adventures. Thriller. Drama. Based on real facts. Mountaineering. Survival. 90's.
Language: English.
Production Companies: Universal Pictures, Working Title Films, Walden Media, Cross Creek Pictures.
Synopsis: The story of the guide Robert "Rob" Edwin Hall, who on May 10, 1996, along with the guide Scott Fischer, joined in a joint expedition to ascend Mount Everest. Inspired by the events of the "disaster of 96", which took place during an attempt to reach the highest peak in the world, it narrates the adventures of two expeditions that face the worst known snow storm. In a desperate effort to adapt to the environment and to survive, the temper of the mountaineers is put to the test by having to face the inclemency of time, the difficulties of relief and a successive series of misfortunes caused by bad luck and planning.
Awards: Venice Festival: Official Feature Film Section (2015, out of competition). Satellite Awards: Nominated to Best Visual Effects (2015). Screen Actors Guild Awards (SAG): Nominated to Outstanding Action Performance by a Stunt Ensemble in a Motion Picture (2016). Camerimage (Jury Award): Nominated to Best 3D Film (2015). Visual Effects Society Awards (VES Awards): Nominated to Outstanding Supporting Visual Effects in a Photoreal Feature (2016) and



American poster of *Everest*.

Outstanding Models in a Photoreal or Animated Project (2016). Jupiter Award: Nominated to Best International Film (2016). Academy of Science Fiction, Fantasy & Horror Films, USA (Saturn Award): Nominated to Best Action/Adventure Film (2016).

Link:

http://www.imdb.com/title/tt2719848/?ref_=nv_sr_1

Trailer

Introduction

Despite the large number of works that document the adaptive physiology of the human body in a hypobaric environment, there are few films that collect a large catalog of symptoms in the face of a bad acclimatization during the ascent in an increasingly hostile environment. The film *Everest* (2015) is a great candidate that would complement in a very visual way the subject of sports and adaptive physiology in environments with low pressures of atmospheric gases. Likewise, some bases are established to determine the adaptation of the organism to different altitudes, its physiological consequences, and the concepts of an adequate interpretation and its clinical meaning. This decrease in barometric pressure is the main cause of all hypoxia problems in the physiology of high altitudes because, as the barometric pressure decreases, the atmospheric oxygen partial pressure decreases proportionally, remaining at all times 20.93% of the total barometric pressure.

Prior to the tragic outcome of the Disaster of 96, the film begins with one of the main problems that raised a multitude of issues: the need for the commercialization of Everest, whose aim is to offer a range of professionalized supports to help reach the summit to not professional or inexperienced mountaineers to eight thousand, which is what is called the elevations of land above 8,000 meters above sea level (m a.s.l.). The New Zealander Rob Hall created the concept of commercial expeditions guided to Everest for non-professional mountaineers. Other commercial operators followed the example of Rob Hall, among them *Mountain Madness* of the guide Scott Fisher. Rob Hall, guide and main leader of the commercial expedition *Adventure Consultants*, along with other secondary guides (Mike Groom and Andy Harris), Sherpas, and eight of their clients of different nationalities (Frank Fishbeck, Doug Hansen, Stuart Hutchinson, Lou Kasischkle, Jon Krakauer, Yasuko Namba, John Taske and Beck Weathers), began the journey to the summit of Everest.

In the minute 5 (5':15''), all participants of the expedition met on March 30, 1996 at Kathmandu airport (Nepal, 1,338 m a.s.l.). There Rob Hall makes reference of the achievements of reaching the top with life: 1) amputations of fingers by freezing (hypothermia), all this while the secondary guide Mike Groom shows the absence of fingers in the foot; 2) - the human being is not prepared to support the flight altitude of a Boeing 747 (Jumbo), which offers an intercontinental flight range of 15,000 m a.s.l. When the height of the South Col (7,906 m a.s.l.) is exceeded, the body begins to die, which is why it is also called "Death Zone" because it is not possible to acclimate the body to higher altitudes of 8,000 m a.s.l. - . At minute 8 (8':48''), it is decided to acclimate the organisms for 40 days with gentle ascents, to be able to attack the summit without difficulties on May 10 due to the stability that the climate presents on that spring date.

Progress of the ascension in stages

Effects of low oxygen pressure on the body

At minute 10 (10':22''), all the components of the equipment are transported by helicopter to the heliport of Lukla (2,860 m a.s.l.), a city in the Khumbu region of eastern Nepal where start the travel the great most visitors from the Himalayas. Table 1 shows the barometric and oxygen (O_2) pressures calculated (approximations) at the different heights shown in the film, in order to demonstrate to the students how the pressures of the gases decrease as height is gained. It is taken as an initial reference the barometric pressure at sea level (760 mmHg).

The temperature has been calculated in relation to the altitude (Table 1). The maximum temperature that marked the summit of Everest (-50 ºC) between May 10 and 11, 1996 was taken as reference. In the atmosphere, the temperature decreases with height. When the air gains altitude, it tends to expand by decreasing the pressure that the air generation loses its temperature as a result of its expansion. We must consider that the Everest air is dry, experiencing a temperature variation of 1 ºC per 100 m a.s.l., a difference of air saturated with moisture, where the temperature decreased with the height 0.5 ºC per 100 m a.s.l.

Then, approximately, the barometric pressures were calculated with the help of the Barometric Formula from the HyperPhysics website (http://bit.ly/2xLU2ZE). The temperature tends to decrease with height, so the calculation model will overestimate the pressure at a certain height (Table 1).

The concentration of O_2 in air at sea level is 20.93%, so the partial pressure of O_2 (PO_2) is 159 mmHg. This pressure in healthy individuals allows saturation of hemoglobin by 97%. Up to a height of approximately 6,048 m a.s.l., even when breathing air, the arterial saturation of O_2 remains at least as high as 90%[1]. Above that height, the saturation of arterial O_2 decreases rapidly. The effect of respiration with pure O_2 masks on the alveolar PO_2 (PO_2 alv) at different heights is different. When a climber breathes pure O_2 instead of air, most of the alveolar space previously occupied by nitrogen (N_2) becomes occupied by O_2, resulting in 100% arterial O_2 saturation at the top of Everest, or 99% at higher altitudes (up to 9,144 m a.s.l.).

Both atmospheric pressure and PO_2 decrease exponentially with altitude while the fraction of O_2 remains constant for about 100 km. When PO_2 falls, the body responds with altitude acclimation[1,2]. When the body reaches about 2,100 m a.s.l., saturation of hemoglobin begins to decrease drastically[3]. However, the human body has short and long-term adaptations that allow it to partially compensate for the lack of O_2. Subjects who live above 3,000 m a.s.l. they are in a relative state of hypoxia. This concept must be meditated, since, as it has been commented, the composition of the air is the same percentage, both at sea level and at the top of Everest. What varies is the barometric pressure and, in turn, the partial pressure of the gases. There is a limit to the adaptation, mountaineers refer to altitudes

Table 1. In this table, from the values offered in the film, it has been calculated all the parameters that directly affect the physiology of the human body without acclimatizing or acclimated (in parentheses).

			Air			Breathing air			Breathing pure O_2			
Location	Altitude	Temperatura e(Tª)	PB (mmHg)	P O_2 air (mmHg)	P CO_2 alv (mmHg)	PI O_2 (mmHg)	P O_2 alv (mmHg)	Sat. Art. O_2 (%)	P CO_2 alv (mmHg)	PI O_2 (mmHg)	P O_2 alv (mmHg)	Sat. Art. O_2 (%)
Sea level	0	Variable	760	159	40 (40)	~150	~100 (~100)	97 (97)	40	713	663	>100
Lukla	2860	+9	537	112	~37 (~23)	~103	~57 (74)	93 (95)	40	490	440	>100
Namche Bazar	3750	-1	474	99	~33 (~21)	~90	~49 (~64)	85 (90)	40	427	377	>100
Tengboche Monastery	3867	-2	~467	~98	32 (20)	88	48 (63)	84 (89)	40	420	370	>100
Monument to mountaineers	4877	-12	401	~84	28 (~18)	74	39 (51)	80 (87)	40	354	304	>100
Everest Base Camp	5364	-17	371	~78	26 (~17)	68	35 (~47)	75 (86)	40	324	274	>100
Camp 1	5944	-23	337	~71	24 (15)	~61	31 (42)	73 (85)	40	290	240	>100
Camp 2	6492	-28	307	64	22 (14)	~55	27 (37)	71 (83)	40	260	210	>100
Lothse face	7132	-34	274	57	20 (13)	~48	23 (32)	68 (78)	40	227	177	>100
Camp 3	7315	-36	265	55	20 (~13)	~46	21 (~30)	67 (77)	40	218	168	>100
Camp 4	7951	-41	~236	49	18 (~12)	~40	17 (25)	64 (74)	40	189	139	>100
The balcony	8412	-46	214	~45	17 (11)	35	14 (21)	58 (66)	40	167	117	>100
The Hillary Step	8760	-49	~200	~42	16 (10)	32	12 (19)	50 (60)	40	153	103	>100
Everest Summit	8848	-50	~196	41	16 (10)	31	11 (18)	<50 (<60)	40	149	99	99

Altitude: Meters above sea level. BP = Barometric pressure of atmospheric air. P O_2 air= Oxygen pressure of atmospheric air. P CO_2 alv = Partial pressure of carbon dioxide in the alveolus. PI O_2 = Partial pressure of inspired oxygen. P O_2 alv = Partial oxygen pressure in the alveolus. Sat. Art. O_2 = Saturation of oxygen bound to hemoglobin in arterial blood (approximate and approximate data).

above 8,000 m a.s.l. as the *Zone of death*, where no organism can acclimate.

Paul Bert (1878) already described with precision that the disorders produced by the height on the organism[4] have their starting point in a lower barometric pressure (BP), which generates a lower inspiratory pressure of O_2 (PI O_2), with a decrease mild O_2 saturation in hemoglobin and a lower O_2 tension in arterial blood gases.

$$PI\ O_2 = FIO_2 \times (BP - 47)$$

As the altitude increases both the FIO2 (whose value is the percentage of O_2 in the atmosphere, 20.93%) and the vapor pressure of water (47 mmHg) are constant parameters, while the total BP and partial pressures of the O_2 go decreasing progressively with altitude. The water vapor pressure of the alveoli remains at 47 mmHg as long as the body temperature is normal, regardless of height.

Hypobaric hypoxia is a phenomenon that is defined as the decrease in the supply of oxygen to tissues due to a drop in the partial pressure of this gas by exposure to a low pressure atmosphere. The term hypoxia refers to a decrease in PO_2, and the term hypobaric to a BP lower than atmospheric pressure. To observe this phenomenon it is necessary to go to the Alveolar Gas Equation:

$$P\ O_2\ alv = PI\ O_2 - P\ CO_2\ alv/R$$

Where R is a parameter whose value is 0.8, reflecting the relationship between CO_2 production and O_2 consumption. It depends on the metabolism of tissues in a stable state. In the case of CO_2, during exposure to very high altitudes, the partial pressure of alveolar CO_2 (P CO_2 alv) decreases from the value at sea level of 40 mmHg to lower values. In the acclimated person, who increases their ventilation approximately five times, the P CO_2 alv decreases to approximately up to 7 mmHg due to the increase in respiration. CO_2 and water vapor reduce the alveolar O_2, reducing the concentration of O_2.

The partial pressure of alveolar O_2 (P O_2 alv) for the native who lives at these altitudes is a normal constant for their habitat, without judging a priori, whether the subject is in hypoxia or not, since the human organism in native subjects acclimated and / or adapted, it carries normal gas parameters, with a pH within normality and a decreased P CO_2 alv. This is due to a greater elimination of CO_2 by a mild hyperventilation, showing that the values of adapted and acclimated are no longer important, with respiratory rates as normal as in subjects living at sea level.

Acute effects of hipoxia

In the 11th minute (11':21''), all have ascended to the Namche Bazaar (3,750 m a.s.l.). Right next (12':07'') The first signs of discomfort begin in the climber Beck Weathers. Due to a rapid ascent and bad acclimatization, he begins to suffer from *altitude sickness* (Figure 1). The normal saturation values of O_2 in arterial blood ranges from 95% to 100%, but non-acclimatized climbers and patients with lung disease often have a lower percentage unless they use supplemental O_2. A non-acclimatized person can usually remain conscious until the arterial saturation of O_2 decrease to 50%[1,2]. Some of the most important acute effects of hypoxia on climbers not acclimatized who breathe air begins about that height. The symptoms are dizziness, laxity, mental and muscular fatigue, sometimes headache, occasionally nausea and sometimes euphoria. These effects progress to a phase of seizures or convulsions by enzymes of 5,500 m a.s.l. and they end above 7,000 m a.s.l. in the person not acclimated in the coma, followed shortly after death.

Figure 1. Hypoxic pre-symptom of *Altitude Sickness*. Beck Weathers (to the right of the photograph) begins to feel the signs of altitude illness at 3,750 m a.s.l.

In the 13th minute (13':51''), they arrive at the Tengboche Monastery, with an altitude of 3,867 m a.s.l. From there it gives way to the 14th minute (14':28''), with the visit to the monument to the mountaineers in Thokla, at 4,877 m a.s.l. Climbers can appreciate a rescue maneuver, where a group of people descend to a sherpa breathing pure O_2 (Figure 2). It is observed that it suffers from the acute effect of hypoxia (acute altitude sickness).

Right in the 18th minute (18':40''), is when the medical officer of the expedition, Caroline MacKenzie, explains how they should function in this new environment before reaching the top: "From a medical standpoint getting you to the top of Everest is really about O_2.

And the lack of it. To give you the best chance of summiting you need to prepare your bodies for the thin air up there. During the next month, before the final ascent, you will make three partial climbs of acclimatization and then you will return to the base camp".

Figure 2. A sherpa is transferred to the hospital after the first immediate medical attention due to an acute condition of altitude sickness.

The permanence of the group at high altitudes for days, weeks, even if they were months or years, allows a better acclimatization to the low PO_2, so that it produces less adverse effects on the body, and it is possible that mountaineers work more without the effects of hypoxia or rise to even greater heights. The main mechanisms that will work for the new adaptation are known[1,2]: 1) a large increase in pulmonary ventilation; 2) an increase in the number of erythrocytes; 3) an increase in lung diffusion capacity; 4) an increase in the vascularization of peripheral tissues; and 5) an increase in the ability of tissue cells to use O_2 despite a low PO_2 (cell acclimation). The organism can adapt to the altitude through an immediate or long-term acclimatization. At high altitude and for a short period, the lack of O_2 is detected by the arterial chemoreceptors of the carotid bodies, although the aortic and pulmonary chemoreceptors can also play a role, and cause an increase in the respiratory rate (hyperventilation). The immediate increase in pulmonary ventilation when ascending removes large amounts of CO_2, reducing PCO_2, and increasing the pH of body fluids. In this way, respiratory alkalosis is produced, which inhibits the respiratory center of the brainstem, opposing the effect of low PO_2 on the stimulation of respiration by means of the peripheral arterial chemoreceptors of the carotid and aortic bodies. After 2-5 days, this inhibition disappears, possibly due to the reduction of the concentration of bicarbonate ions in the cerebrospinal fluid and brain tissues, allowing the respiratory center to respond fully to the stimulation of peripheral chemoreceptors by hypoxia and ventilation increases up to approximately five times compared to normal. The inability to increase the respiratory rate causes an inadequate response of the carotid body, pulmonary conditions or kidney problems. At higher altitudes the heart beats faster, this is because it increases blood volume and cardiac output up to 30% immediately after a person makes an ascent, then decreasing cardiac output to normal values in a period of time (weeks) as blood hematocrit increases. The latter will allow the amount of O_2 that is transported to peripheral body tissues to remain approximately normal. Digestion becomes less efficient because the body suppresses the digestive system in favor of increasing the reserves of the cardiorespiratory system[5]. This is one of the possible explanations for which the guide Scott Fischer had serious digestive problems throughout the film, along with a picture of altitude sickness that changed from mild to severe, despite the continuous injections of dexamethasone.

In the 20th minute (20':11''), they arrive until the Camp One (5,944 m a.s.l.). There are references on taking care of the cold and the consequences of hypothermia: "Beware of hypothermia, it produces difficulty to speak or irrational behavior. I have seen mountaineers who took off all their clothes at 8,000 m height because they were hot".

Then (20':24''), she explains what is cerebral edema and pulmonary edema (Figure 3): "Do you know what a cerebral edema is? The brain swells causing the loss of motor capacity and finally death. In lung edema, the lungs become fluid with water until you drown. The only cure is to get off the mountain quickly".

Figure 3. Sequence of frames of the signs of pulmonary edema, where a mountaineer is seen shot down by violent cough (A, B) and bloody expectoration (C, D).

During the minute 24 (21':04''), in Camp Two (6,492 m a.s.l.), they are taught to operate the breathing equipment. They give orders of opening of the regulator, to verify that it sounds the exit of O_2, to connect tubes to the valve and mask, to breathe through it and to regulate the pressure. Doug Hansen begins to manifest acute pulmonary edema by the noise of his cough (21':36''). The guide Rob Hall expresses his concern, recommending that tonight he sleeps with O_2 and that he goes to the expedition doctor. After auscultation (Figure 4), the

prescribed treatment is adequate, the use of dexamethasone[6]. The leader of the Mountain Madness expedition, Scott Fischer, also injected dexamethasone (44':53'') after descending from Camp Two to Base Camp, and decide, the next day, to continue to Camp Three without hardly resting, nor to treat its slight edematous sign (Figure 5).

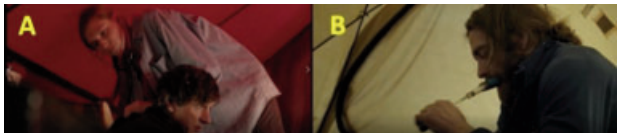


Figure 4 and 5. Auscultation and treatment with dexamethasone after manifesting a picture of altitude sickness.

There are two serious forms of altitude sickness, high-altitude pulmonary edema (HAPE, fluid in the lungs) and high-altitude cerebral edema (HACE, fluid in the brain). They may be preceded by mild symptoms (headache, insomnia, lack of appetite, mild daze), or appear suddenly in a previously healthy mountaineer, because of a very steep ascent. Both have a high mortality rate and can occur when a day or a day and a half has passed at too high (usually, above 3,500 m a.s.l.). The symptoms of HAPE (Figure 3) are severe, and include significant respiratory distress, dry cough, bloody sputum, chest pressure or pain, palpitations and fatigue. Because of the pulmonary edema a sound of bubbling can be heard with the rattle during breathing. The lips, outer edges of the ears and nails may appear cyanotic because of the lack of O_2. The appearance of HACE is the most serious and rapid form of presentation of altitude sickness. The symptoms of HACE are mainly nausea, vomiting, headaches, visual disturbances, irritability, incoordination, distraction, confusion, possible loss of consciousness, seizures and even coma. Beck Weathers experienced loss of vision during the ascent (Figure 6). It is not known if it is part of the symptomatology of altitude sickness or that it is transient altitude blindness that appears from 2,500 m a.s.l. Although it is postulated to a combination of factors (lack of O_2, hemorrhages in the retina, inflammation of the cornea as a result of the cold and an inflammation of the conjunctiva caused by solar radiation), it is probably attributed to a complication by an old operation In the view (52':44'').

Figure 6. A. Beck Weathers the moment which notice eye pain (A) and loss of vision (B).

Dale Kruse, mountaineer of the group of Scott Fischer, showed the two serious forms of edema (Figure 7) after starting ascent from Base Camp (5,364 m a.s.l.) to Camp Two (6,492 m a.s.l.) without taking rest in Camp One (5,944 m a.s.l.). They decide to go down to Camp One and there Scott Fischer decides his transfer to Base Camp (41':22'').

Figure 7. Dale Kruse in serious condition for manifesting pulmonary and cerebral edema.

The characters of the film, as they presented the mild symptoms of altitude sickness, were treated immediately with rest on the same ground for 24-48 hours, accompanied by a good hydration and a diet rich in sugars. With this treatment is enough. It was forbidden to climb when mountaineers presented symptoms of altitude sickness, even if it was mild, since they can evolve into more serious forms. The imprudence of the leader Rob Hall before the refusal to follow the ascent of Doug Hansen a few hundred meters from the top (1h:05':30''), already severely affected by altitude sickness and without O_2 reserves, caused the deadly delay of his returns just when the worst consequences of a storm began. It has beeb already commented that above an altitude of 8,000 m a.s.l., in which the partial pressure of O_2 is not sufficient to maintain human life, it is called *the zone of death*. When the symptoms are more severe or worsen, as was the case, the affected person should immediately descend to the lowest possible height, and always accompanied. Sometimes a descent of 400 meters is usually enough to notice an improvement. Another measure, if the O_2 regulating valve had not failed due to freezing, would have been to administer O_2 through the mask, an amount of 3 to 5 liters per minute at a concentration of not less than 40%. For the treatment of headache can be used minor pain relievers such as acetaminophen or aspirin, among others. As for

insomnia of height, especially if it is caused by periodic causes of breathing, it should be treated with acetazolamide, but never with hypnotics or sedatives, as they can make breathing even worse. The medication never replaces the decrease.

Hypothermia and freezing

Much of the most serious and fatal deaths in Everest occur in this area of death, either directly by loss of vital signs or indirectly by incorrect decisions made under stress or physical weakness. In this area no organism can acclimatize. The human body uses its O_2 supply faster than it can replace. A prolonged period in this area triggers a deterioration of body functions, loss of consciousness and, finally, death[7,8]. It is seen as the secondary guide of Rob Hall, Andy Harris, after unsuccessfully assisting Rob Hall and Doug Hansen, suffering a severe mental deterioration with paranoia and hallucinations (1h:25':40''). At temperatures lower than 0 °C, he manifests an illusory sensation of hyperthermia and is detached from all clothing (Figure 8). Another possible reason for the origin of heat or "burning" may be due to the adrenaline and vasoconstriction discharge for a long time. This helps preserve body temperature. In case of extreme cold or when the body is exposed to cold for prolonged periods, this protective strategy can reduce blood flow in some areas of the body to dangerously low levels. The areas where this occurs will freeze. The blood cools due to this vasodilation and loses heat. In addition, there is an edema due to vasodilation and the feet swell (the person hurts their shoes and takes them off). Goes into sleepiness, favored by hypothermia and, having a toxemia due to vasodilation, the so-called "substances of fatigue or hypnotoxins" act on the midbrain sleep center. The combination of cold temperatures and low blood flow can cause severe injuries in tissues that are freezing. This produces the formation of intra/extracellular ice crystals with hypertonicity, denaturation of proteins, destruction of cell membranes, plasma hyperviscosity and decreased nerve conduction. Having anoxia and consequently acidosis, histamine is released, which leads to vasodilation

Figure 8. Andy Harris takes off his clothes when he manifests a sensation of heat and burning by freezing.

(Ricketts phenomenon), due to the predominance of the sympathetic over the parasympathetic; the individual has great euphoria and security. It is thought that Andy Harris disappears after sliding and falling from a great height.

A total of eight people die from hypoxia and freezing. It also made the difficult decision to kill Beck Weathers because he was in a coma and frozen. However, later on that same day and inexplicably for the medical field, he reappeared walking (Figure 9) to the camp with difficulty after regaining consciousness (1h:44':11'').

Figure 9. Beck Weathers "resuscitates" and walks to the camp with severe hypothermia and frostbite on parts of the body that are not covered.

Quickly, before being evacuated by helicopter, he was treated with O_2 (1h:45':46''). The basis of treatment of freezing injuries is rapid rewarming of the affected limb by immersion in water at 40-42 °C for 15-30 min (Figure 10). Lower temperatures appear to be less effective in achieving tissue recovery, and higher temperatures can cause thermal burns. The application of dry heat and slow heating are contraindicated. During treatment, the appearance of an erythema of violaceous coloration and the softening of the area that allows the skin to be folded are favorable signs of recovery.

Figure 10. Beck Weathers is treated with O_2 and submerging the frozen limbs in warm water.

After the initial treatment there is a slowly progressive recovery of viable tissues and necrosis of the unrecoverable area. The definition of the extent of damage occurs after a variable time, from one to three months, after exposure to cold. Then surgical debridement of the

necrotic tissues and the necessary amputations will be carried out. In the final credits it is shown that Beck Weathers lost both hands and nose due to severe freezing (1h:53':00'').

Results and Discussion

It is suggested that the fatal results of both expeditions are due to the low qualification of the clients, and the confidence provided by the use of O_2 bottled only in cases of emergency, leading to dangerous situations, together with the added pressure of to be accompanied by a journalist, Jon Krakauer, from the important magazine *Outside* for mountaineers. The decision not to use supplementary O_2 while performing his guidance role to Anatoli Bukrèyev, secondary guide of Scott Fischer, is also criticized. Krakauer himself states that, within the threshold of *the area of death*, many of the misguided decisions that were made on May 10 were caused by two or more days without adequate oxygenation, a restricted diet and lack of rest[9]. In addition, researchers at the University of Toronto discovered that the adverse weather conditions of May 11 were the cause of a drastic fall in O_2 levels of around 6%, which resulted in a 14% reduction in the consumption of O_2[10].

Table 1 shows the variation of the arterial saturation of O_2 during the ascent to the summit of Everest, however, it is necessary to measure these saturation values after the acclimation period. Several articles show by pulse oximetry that the arterial saturation of O_2 increases after a habituation of 30 days in the Base Camp[11]. In the *American Medical Research Expedition to Everest*, they claim that increased saturation at high altitudes could be due to hyperventilation states[12]. On the other hand, they state that hyperventilation in ascents at high altitudes is facilitated by a lower viscosity of air, which increases the percentage of arterial O_2 saturation[13,14]. In 2009, other studies drew their conclusions by studying a group of mountaineers who climbed Everest[8,15-18]: (1) the decrease in P O_2 alv is proportional to the decrease in BP while the saturation of O_2 is maintained despite the large barometric changes with altitude; (2) increases in hemoglobin concentration compensate arterial O_2 content up to levels reaching 7,000 m a.s.l.; (3) they could not demonstrate neurocognitive alterations that could support serious disturbances of cerebral hypobaric hypoxia. This point does not agree with the manifestations suffered by the real characters and those shown in the fiction. This is strongly affirmed by other authors, where they do recognize the presence of these neurocognitive alterations by cerebral hypobaric hypoxia in extreme heights, regardless of whether or not they are acclimated. They claim that, in acute and chronic conditions of cerebral hypobaric hypoxia, there is a blockage of the mechanisms of cerebral self-regulation[19].

Conclusions

All the data collected in this work suggest that the film Everest is a worthy candidate for clinical study to understand basic concepts of adaptive physiology in hostile environments. The variables used in relation to changes in pulmonary gas pressure, after modifying the barometric conditions, help to reflect on the physiological changes suffered by a mountaineer, with or without acclimatization, to extreme heights.

Acknowledgements

Dr. Domínguez-Vías thanks the help of Professor Dr. José Juan Vallo de Castro of the University of Cádiz, for the cession of his literary material and invaluable help for the development of ideas in the field of adaptive physiology to the height.

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