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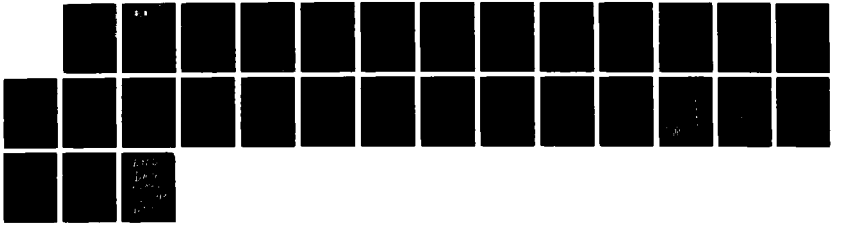

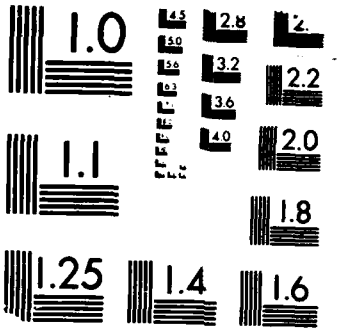

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A number of physiological adjustments occur when man is exposed to the hypoxia of high altitude. One of the most significant is a reduction in his maximal aerobic exercise performance. The component of systemic oxygen transport responsible for the early reduction in maximal aerobic power is the low arterial oxygen content, whereas decreased cardiac output is the reason for the persistence of the impairment after several days at high altitude.

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Human Acclimatization and Physical Performance at High Altitude

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Man is the only animal who purposely subjects himself to the rigors of the mountainous regions of the world for reasons other than pure survival. Man travels to high altitude for recreational pursuits such as skiing, trekking, mountaineering, camping, and sightseeing. In the past few decades, the number of people participating in high altitude activities has increased manyfold (17). The grandeur of mountainous regions and the ability to participate in mountain activities were once limited to mostly those who lived in the lower regions immediately surrounding the higher elevations, and to those who were fit and strong enough to hike up difficult trails. However, improved air and ground transportation has "opened up" the mountains to the masses. People of all ages and physical abilities can fly to mountain regions, and in some places, get to a summit with little difficulty. For example, it is estimated that in the 1987 season, 500,000 tourists made the one-hour ascent of Pikes Peak, Colorado (1830 m to 4300 m) either by car, bus, or railway; remained for a couple of hours on the summit, and then descended (Personal Communication, National Park Service).

Because there have been such large numbers of people participating either passively or actively at higher elevations, it is important to understand exactly how the hypoxia (reduced amount of oxygen) of high altitude can affect the human body in general, and physical performance in particular. The physiological changes and problems encountered during treks into high mountainous regions have been appreciated since their first recorded description in 326 B.C. (17). However, it has only been in the last 125 years that an oxygen deficiency in the inspired air was experimentally shown to be the primary source of many of the altitude-related changes and problems (17).

Basic Concepts

An uninterrupted supply of oxygen must be made available to man in order for him to function for more than a few minutes. Each cell of the body requires oxygen. However, because of the distance between most cells and the ambient air, the cells cannot receive the oxygen directly. The ability to transport oxygen to the cells depends on a delivery and exchange system that includes: movement of oxygen from the ambient air to the lungs; movement of oxygen from the lungs and into the blood; transportation of the oxygenated blood to the tissues; and finally, movement of oxygen out of the blood into the tissues.

The percentage of oxygen in the ambient air is constant (20.93%) up to at least 100,000 meters (328,080 feet) (6). However, as one ascends from sea level to altitude, one is exposed to a progressive reduction in barometric pressure (P_B). Since the the partial pressure of oxygen is determined by multiplying the percentage of oxygen by the P_B, the partial pressure of oxygen is also reduced at altitude. For example, at sea level, where the P_B is 760 Torr (see appendix, part A), the partial pressure of oxygen in the ambient air is 159 Torr (760 Torr * 20.93%). When one goes to 5500 meters (18,000 feet), which is about the highest altitude man can be acclimatized to and live and work for years (28), the partial pressure of oxygen is reduced to approximately 80 torr (380 Torr * 20.93%), or about half the sea-level value.

Oxygen (or any gas) moves from regions of higher concentrations to regions of lower concentrations. Therefore, if the partial pressure of oxygen in the inspired air is reduced, as it is at altitude, then the ability to move oxygen along each stage of oxygen exchange to the various cells of the body must also be reduced. The result is an impaired oxygen transport. However, there are numerous adjustments that occur within the body to minimize the effects of hypoxia and make it possible for an individual to

function despite a large reduction in the partial pressure of oxygen in the inspired air.

The first part of this paper will briefly review some of the physiological responses which occur when an individual is acutely and chronically exposed to the hypoxia of high altitude. The second part will describe how some of these responses adequately compensate for the hypoxia during bouts of submaximal, but not maximal, exercise. It should be noted that there are a number of factors associated with the high-altitude environment that can have a direct bearing on the well-being and physical performance of an individual which are not related to the hypoxia per se. These factors can include cold temperatures, high winds, low humidity, and solar radiation injuries (20). It is the intent of this paper to describe the physiological adjustments primarily due to hypoxia alone.

I. Physiological Adjustments

Ventilation

Man's initial response to high altitude is an increase in ventilation. The number of breaths taken each minute as well as the depth of each breath is increased. This increase in ventilation causes the air deep within the lungs to contain a higher pressure of oxygen than it would otherwise have. By increasing the pressure of oxygen within the lungs, more oxygen can diffuse (filter) into the blood for transport to the tissues (25).

The increase in ventilation also causes carbon dioxide (one of the waste products of cellular function) to be "blown off" making the blood less acidic which, in turn, initiates a series of steps culminating in an enhanced ability of oxygen to be released from the blood to the tissues (34). Obviously, the increase in ventilation is an extremely important and useful response to high altitude.

Body Fluid Alterations

There are 3 major fluid compartments within the body. At sea level, under resting conditions, the volume of fluid inside the cells, between the cells, and in the blood vessels (plasma volume) remain relatively constant. However, exposure to altitude quickly alters the normal distribution of body fluids. There is a marked movement of fluid out of the blood vessels and from the spaces between the cells, into the cells. The reduction in plasma volume, occurs almost immediately upon exposure to altitudes greater than 3000 m (10,000 feet). At altitudes greater than 4300 m (14,110 feet), plasma volume may be reduced by 20 to 30% (14). The total volume of body fluid may or may not be altered (14). With longer altitude exposures, there is some reversion of fluid compartments to sea-level values, but apparently never full recovery (28).

Altitude Sickness

The change in fluid status, especially the rapid flux that occurs within the first couple days of exposure to high altitude, is thought to play a major role in the cause of some altitude-induced medical problems (20). Among the numerous medical problems associated with high altitude, many of which impact on performance, are acute mountain sickness (AMS), high-altitude cerebral edema (HACE), and high-altitude pulmonary edema (HAPE).

AMS is a very common illness above 3000 m (10,000 feet) and is characterized, but not limited to, lassitude, headache, nausea, vomiting, insomnia, irritability and depression. The incidence of AMS varies from 8-100% of exposed individuals, and is directly related to the rate of ascent and to the final elevation obtained (13,20). Symptoms usually become noticable after only several hours of exposure; reach their peak severity within the first 24-48 hours; and then gradually recede over the ensuing 2-7 days as acclimatization (adaptation to the hypoxia) takes place (20).

Although AMS is self-limiting, it can be debilitating (20). AMS symptoms can be minimised or eliminated by gradual ascent and/or by ingesting certain medications prior to the ascent. The most commonly used medication is acetazolamide (17,20).

HACE, or edema formation in the brain, is potentially fatal if left untreated (21,37). Edema can be thought of as an accumulation of fluid in the spaces between or in the cells which will interfere with the ability of the affected tissue to function normally. Although the early signs of HACE resemble those of AMS, "staggering gait" and change of mental status (e.g., increased forgetfulness) help differentiate early HACE from AMS (20). HACE occurs in about 1% of individuals who ascend rapidly and who are not acclimatized to altitude (13). If HACE is suspected, immediate evacuation to a lower elevation (at least by 300 m or 1,000 feet) and medical aid are warranted (17,20).

HAPE, or edema formation in the lungs, also occurs in unacclimatized individuals who rapidly ascend to high altitude (13,17,20). The incidence of HAPE is higher than that of HACE but much less than AMS (20). Edema formation, by filling the lungs cavities with fluid, interferes with the ability of oxygen to diffuse into the blood. Thus, it should be no surprise that the early symptoms of HAPE include fatigue, breathlessness on exertion, and cough. Gurgling sounds from within the chest during breathing can also be heard. HAPE usually manifests itself 12-96 hours after ascent (20). Exercise at altitude, especially in younger males, may increase the susceptibility to HAPE (20). Fortunately, HAPE usually responds to evacution to a lower elevation readily, although medical aid should be sought (20). If left untreated, HAPE can lead to coma and death in less than 12 hours (17,20).

Fluid Status and Hormonal Changes

Changes in fluid status at altitude can be caused by, or can cause, an alteration in the concentration of those hormones that are involved with maintaining a normal fluid and electrolyte (e.g., sodium, potassium) balance in the body (24). Two of the more important hormones involved with fluid regulation are aldosterone and arginine vasopressin. Both of these hormones have an effect on the kidneys to reabsorb water to help limit a reduction the total blood volume. There are a number of scientists who believe that an overactivation of these hormones, as well as 2 other related substances (renin and angiotensin), are implicated as being at least partially responsible for AMS and edema formation at high altitude (24).

Cardiac Output

Changes in fluid status also play a role in the alteration of cardiac output at altitude. Cardiac output is the product of heart rate (beats per minute) and stroke volume. Stroke volume is the volume of blood pumped by the heart with each beat. Cardiac output, then, is the amount of blood that is pumped by the heart and is the volume of blood circulated within the body each minute. During the first couple of days at high altitude, cardiac output is increased at rest and at any level of oxygen uptake, except during maximal exercise. After the first couple of days, there is a progressive reduction in cardiac output (8,12). By the 10th day of continued exposure, cardiac output at rest and at any level of oxygen uptake is actually less than that found at sea level (12,15,28). The reduction in cardiac output seems to be primarily due to a reduction in stroke volume, which, in turn, is due to the reduction in plasma volume (8,12). For the rest of the altitude sojourn, the reduced level of cardiac output is stabilized (12).

Enhanced Oxygen Carrying Capacity

Blood can be thought of as being composed of 2 main compartments: plasma volume and red blood cells. Out of any given quantity of blood, approximately 55% will be plasma volume and 45% will be red blood cells, at sea level. Hematocrit is the percent of red blood cells within any quantity of blood. Carried within the red blood cells is hemoglobin, which is the compound responsible for picking up, transporting, and releasing oxygen at the lungs, in the blood, and at the tissues, respectively. With initial exposure to high altitude, the hematocrit and hemoglobin concentration are increased. However, there is no change in the number of red blood cells. The increases in hematocrit and hemoglobin are due to the reduction in plasma volume. As the exposure continues, there is an increase in red blood cell production. After 2 to 3 weeks, red cell mass is significantly increased (28). The result of increases in the hematocrit and hemoglobin concentrations at altitude is a greater oxygen capacity of the blood. In other words, for any volume of blood, more oxygen can be transported from the lungs to the tissues and into the cells.

II. Effects of Altitude Exposure on Exercise

Maximal Oxygen Uptake

The lowered oxygen pressure at high altitude impairs those types of physical activities which are dependent upon sustained oxygen uptake. Man's ability to perform muscular exercise is usually evaluated by measuring his maximal oxygen uptake (VO_2max), during severe exercise leading to exhaustion in a brief period of time. Numerous investigators have measured the effect of altitude on VO_2max (1,9,23). Drawing from the cumulative data, Buskirk (4) established the general relationship between altitude and aerobic work capacity, expressed as a percent of VO_2max at sea level (see Figure 1). Note

that VO_2max is not measurably altered by altitudes between sea level and about 1500 m (5,000 feet). However, above 1500 m there is a linear decrease in VO_2max, at the rate of 10% per 1000 m (3,280 feet). For example, in travelling from sea level to 1500 m, VO_2max remains unaltered, but if an additional 1500 m are ascended (i.e., at 3000 m), VO_2max will be 85% of that at sea level. The reduction in VO_2max occurs immediately upon exposure, persists for the entire sojourn (12,15), and occurs in both males and females (10). It should be emphasized that the relationship between altitude elevations and VO_2max, as presented in Figure 1, represents the average VO_2max decrement at a particular altitude. There is a wide range in individual values at any given level of hypoxia. For example, Young et al.(26) found that at 4300 m (14,110 feet) the average VO_2max decrement of 51 males was 27%, a value similar to what would be predicted from Figure 1. However, the distribution of values ranged from 9% to 54%.

Submaximal Exercise

Although VO_2max is measured during maximal effort of relatively brief duration, the decrease in VO_2max at high altitude is also reflected in more prolonged exercise of less severe intensity (10,28). Oxygen uptake at rest, or any fixed (i.e., absolute) exercise intensity up to very heavy levels does not differ from sea level (1,10). However, the oxygen uptake elicited by the fixed exercise intensity actually represents a greater fraction of the reduced VO_2max at altitude than it did at sea level (23). Therefore, the relative physical stress on the body will be increased in proportion to the altitude-induced reduction in VO_2max. For example, pedaling an ergometer at an exercise intensity of 200 watts requires an oxygen consumption of about 2.8 L/min at sea level and at any altitude (1). If an individual who has a VO_2max of 4.0 L/min at sea level and a VO_2max of 3.2 L/min at 3500 m (11,500 feet) (see Figure 1), pedals an ergometer at an exercise intensity of 200

watts in both environments, he will be exercising at 70% of his VO_2max at sea level and at 88% of his VO_2max at 3500 meters. Furthermore, at any altitude greater than 4500 m (14,750 feet), where this individual's VO_2max would be reduced by more than 30% (or less than the required 2.8 L/min), the 200 watts would represent a supramaximal exercise intensity and, thus, could not be sustained. Obviously, because of the greater relative stress, endurance time (performance of submaximal exercise at a fixed exercise intensity) will be reduced at altitude (1,19).

Athletic events

The physical limitations imposed by the hypoxia of high altitude can be further illustrated by contrasting the performances of athletic events at altitude with the performances of the same events at sea level. Malhotra and Sen Gupta (21) recorded the running speeds of healthy, nonathletic soldiers in a 1.6 km race held at sea level and at four higher altitudes. They found that the running speeds were reduced (i.e., the time to finish the run was increased) in direct proportion to the increase in elevation, to allow completion of the event. At sea level, the maximum speed was 16.5 km/h; at 2270 m (7500 feet), 14.2 km/h; at 3100 m (10,000 feet), 12.2 km/h; at 3500 m (11,500 feet), 11.7 km/h; and at 4000 m (13,000 feet), 10.6 km/h. Likewise, Grover and Reeves (11) reported that the average time to run a mile was increased from 4:49 min at 300 m to 5:11 min at 3100 m in high school athletes. As can be seen, the decrement in performance is directly related to the altitude where the event is taking place.

The magnitude of the performance decrements at altitude is also directly related to the duration of the event, i.e., the longer the event at any altitude, the greater the decrement. At the 1955 Pan American Games held in Mexico City (2380 m or 7800 feet), the increase in winning times were 3% longer in the 1500 m run, 6% to 7% greater in the 5000 m and 10000 m runs,

and 17% to 22% greater in the marathon (42,000 m) than the sea level times (16). Similar results were reported during the 1968 Olympics (also held in Mexico City). Craig (5) showed that there were steady increases in times for both track and swimming events lasting longer than 1 minute, ranging from 3% at 4 minutes to 8% at one hour.

Not all physical and athletic performances suffer because of the hypoxia of high altitude. "Explosive" (high intensity, short duration) activities, such as the short-put, broad jump, high jump, and other events primarily involving anaerobic metabolic processes, do not suffer because they do not depend on oxygen transport. In both the 1955 Pan American Games and in the 1968 Olympics, world records were broken in the short running events (e.g., 100 m to 400 m), which depend largely on anaerobic energy sources, presumably due to a reduction in air density and resistance (16).

Muscle Strength

Muscle strength also does not seem to be adversely affected during acute exposures to high altitudes, and there is some evidence indicating that it may be enhanced (3,27). Young et al. (27) measured muscle strength in 10 young men, before, during and after a 48-hour exposure to 4572 m (15,000 feet). A series of 7 strength assessments which included measures of both static and dynamic strength of a number of muscle groups were performed. Measurements were made of isokinetic (180°/sec, 36°/sec, and 0°/sec) knee extensor strength; isometric strength of the upper torso, knee, and trunk extensor muscles; and muscular endurance of knee extensor and elbow flexor muscles during repeated isokinetic contractions. Strength in each of the tests was increased above the sea-level values (though reaching statistical significance in only the 3 isometric measurements). The increase in strength scores, overall, averaged 8.3%. Likewise, Burse et al (3) found isometric handgrip strength to be increased during a 6-day exposure to 4300 m (14,110 feet). However, handgrip endurance time to fatigue tended to decrease.

The effects of a hypoxic exposure lasting more than a week on skeletal muscle strength have not been addressed. However, chronic exposures at 4300 m (7) and higher (22) altitudes have been shown to cause reductions in lean body mass which, presumably, could result in a reduction in muscle strength.

What Limits Aerobic Exercise Capacity at High Altitude?

The performance of aerobic exercise is obviously dependent upon oxygen transport. Figure 2 illustrates the close relationship between VO_2max and maximal systemic oxygen transport. The relationship exists over a wide range of values at sea level and during acute and chronic exposure to altitude (15). Since man's capacity for exercise is diminished at high altitude, it is reasonable to postulate that this is a consequence of some compromise in the oxygen transport system.

In transporting oxygen from the atmosphere to exercising muscle, a chain of processes is involved. With each breath, air is moved into the lungs followed by a transfer of oxygen across the lung tissue into the blood stream to combine with hemoglobin. The oxygenated hemoglobin is then transported through the blood stream, to the muscles where it diffuses out of tiny blood vessels (capillaries), into the muscle cells, and ultimately, to the mitochondria (see appendix, part B). If the mitochondria, or the "powerhouses" of the cell, are not supplied with an adequate amount of oxygen, all cellular functions will cease because sufficient energy will not be produced.

Acute exposure to altitudes greater than 3000 m leads to an increase in ventilation. Exercise is also a stimulus to ventilation, so that when exercise is performed at altitude, ventilation is consistently greater at any given submaximal exercise intensity than at sea level. Figure 3 shows ventilation in relation to oxygen uptake, at different exercise intensities,

when breathing oxygen or air at sea level and during acute exposure to various altitudes. In Figure 3, the ventilation at an oxygen uptake of 3 L/min was 55 L/min when pure oxygen was inhaled, 60 L/min at sea level when the subject was breathing air, 75 L/min at 1000 m (3300 feet), 85 L/min at 2,000 m (6600 feet), 100 L/min at 3,000 m (10,000 feet), and 130 L/min at 4,000 m (13000 feet).

The point to be made here is that it is not ventilation which limits exercise capacity at altitude. If one exercises at progressively increasing exercise intensities, VO_2 will plateau at its maximum while ventilation continues to increase (1).

The second step in oxygen transport is the diffusion of oxygen from the lungs to the blood. The pressure gradient between the partial pressure of oxygen in the lungs and the partial pressure of oxygen in the blood plays an important role in the rate of diffusion of oxygen. When one exercises at sea level, the blood returning to the lungs (venous blood) to be oxygenated has a low partial pressure of oxygen (much of the oxygen was used at the site of the exercising muscles). Since the partial pressure of oxygen in the lungs is high, the oxygen pressure gradient from the lungs to the venous blood is large, which favors rapid diffusion. At high altitude, however, the partial pressure of oxygen in the lungs is reduced, despite the compensatory increase in ventilation, so that the pressure difference from lungs to the blood is less, thereby slowing the rate of diffusion (18). Consequently, when exercise raises cardiac output and increases the velocity of the red blood cells through the lung capillaries, transit time becomes too short for equilibration between the lungs and the blood to occur and, as a result, blood oxygenation (saturation) falls (25).

Figure 4 shows the rise in oxygen saturation of blood with respect to time spent in the lung capillaries during heavy exercise at sea level and at an altitude of 3100 m (10100 feet). At sea level (in the left-hand panel),

blood transit time in the capillary could be shortened to less than 0.35 seconds before oxygen saturation of blood leaving the lung would fall to less than 95%. In the right hand panel, at 3100 m, it is apparent that the rise in oxygen saturation during blood transit time in the capillary is slower than at sea level, and 0.5 sec is insufficient time to achieve oxygen equilibrium between the lungs and the blood. The resultant widening of the lung-blood oxygen gradient at high altitude suggests that oxygen uptake is, at least in part, diffusion limited by the lowered partial pressure of oxygen at altitude.

The third component of oxygen transport is arterial oxygen content. Oxygen diffuses from the lung into the capillaries where it is transported in combination with hemoglobin in the blood to the tissues. The oxygen carrying capacity, i.e., the maximum amount of oxygen which can be transported if fully saturated with oxygen, is the product of hemoglobin concentration and 1.34 (a constant, which is the amount of oxygen that can combine with each gram of hemoglobin). At sea level, the oxygen carrying capacity is 20 vol% (15 grams of hemoglobin/100 ml of blood * 1.34 ml/oxygen) and is 97% saturated with oxygen. Thus, arterial oxygen content is 19.4 vol% (20 vol% * 97%) at sea level.

Within the first few hours of exposure to altitude, the oxygen carrying capacity is not altered from the sea level value of 20 vol% (10). However, the arterial oxygen content is decreased due to a reduction in the saturation of hemoglobin. At 4300 m, for example, partial pressure of oxygen in the lungs is reduced, thereby reducing the saturation of hemoglobin to about 87%. Therefore, the arterial oxygen content will be reduced to 17.4 vol% (20 vol% * 87%).

As already alluded to, after the first few hours, but within the first few weeks of altitude exposure, there is an reduction in plasma volume (14,28). Thus, the hematocrit ratio and the concentration of hemoglobin in

the blood is increased, effectively increasing the oxygen carrying capacity. At 4300 m., because of the reduction in plasma volume, the concentration of hemoglobin may be raised to 16.5 vol% of blood and the oxygen carrying capacity to 22.1 vol% (16.5 vol% * 1.34). Due to the aforementioned ventilatory adaptations, arterial oxygen saturation may be raised also, perhaps to 89%. Therefore, because of the increases in hemoglobin concentration and arterial oxygen saturation, the arterial oxygen content will be raised to 19.7 vol% (22.1 vol% * 89%), a value similar to the 19.4 vol% found at sea level.

With continued residence in the hypoxic environment, red cell production is stimulated and total red cell number increases, resulting in a "true" increase in hemoglobin concentration. This, together with partial restoration of plasma volume, leads to an expansion in total blood volume exceeding the sea-level value (12).

Thus, after the first few hours of altitude exposure, the arterial oxygen content is increased sufficiently to maintain systemic oxygen transport and is not the limiting factor in oxygen uptake.

The next major component of oxygen transport is the circulation of oxygenated blood to the tissues, including the exercising muscles. VO_2max is directly related to systemic oxygen transport (see Figure 2) and is considered to be a valid index of the exercise capacity of an individual because it reflects both the ability of the cardiovascular system to deliver oxygen to the exercising muscles and the ability of the tissues to utilise oxygen (1). Systemic oxygen transport has two major components, arterial oxygen content and cardiac output. As shown in Figure 1, VO_2max is diminished immediately upon exposure to altitudes greater than 1500 m (4,10). Since maximal cardiac output does not diminish for the first couple of days of exposure (12,15), the reduction in oxygen transport (and VO_2max) is closely related to the reduction in arterial oxygen content (23).

In Figure 5, oxygen saturation of hemoglobin in normal man has been plotted at sea level and at various altitudes at rest and during exercise. At 3100 m (10,150 feet), the normal resting arterial oxygen saturation is 93%. At 4540 m (14,900 feet), the resting saturation falls to 78% and at 5800 m (19,000 feet), to 69%. During exercise at sea level, arterial oxygen saturation does not usually fall. However, during exercise at high altitude, oxygen saturation does fall. At 4540 m, the saturation may fall to below 70% and at 5800 m, intense exercise may decrease arterial saturation to levels as low as 50% to 60%. This is obviously a factor that limits work performance at high altitudes since arterial oxygen content is directly related to arterial oxygen saturation.

As the altitude exposure continues and altitude acclimatisation occurs, there is an increase in arterial oxygen content back to sea level values as we have already determined (12). VO_2max, however, does not rise above the level measured during the initial exposure to altitude in proportion to the increase in arterial oxygen content (12,28). This finding suggests that the sustained reduction in VO_2max at altitude is related to a reduction in maximal cardiac output. This has been found to be the case (9).

It should be re-emphasised that it is only the oxygen uptake during maximal exercise that is reduced at altitude. Oxygen uptake at rest and at any submaximal exercise intensity is not different from sea level (10,28). During an initial exposure to altitude during submaximal exercise, the reduction in arterial oxygen content is compensated for by an increase in cardiac output (1,12,23). In other words, the reduction in the amount of oxygen transported per unit of blood is offset by an increased volume of blood delivered to the tissues. As the altitude exposure is extended, and the arterial oxygen content is restored, a lesser volume of blood needs to be delivered so that cardiac output, at rest and during any submaximal exercise intensity, decreases to or even below, sea level values (12).

The final step in oxygen transport is tissue oxygenation. Once oxygenated blood enters the capillaries within the skeletal muscles, it is the ability of hemoglobin to hold onto oxygen which determines the ease with which oxygen is unloaded to the tissues. It has long been known that at altitude, the the ability of the hemoglobin to hold onto the oxygen is reduced at the level of the tissues, thus, favoring release of oxygen to the tissues. The increase in temperature and the increase in acidity of the local exercising muscles also reduce the affinity of hemoglobin for oxygen further aiding in the delivery of oxygen to the tissues (1).

With long-term acclimatisation to altitude, there is an increase in amount of capillaries per muscle tissue which reduces the length of the diffusion path for oxygen from blood to the cells, an increase in myoglobin ("muscle hemoglobin") content in skeletal muscle, and chemical adaptations within the muscle cell (6). All of these changes have a favorable effect on oxygen transport.

In summary, when man ascends to high altitude, one of the most significant consequences is a reduction in his maximal aerobic exercise capacity. The component of systemic oxygen transport responsible for the early reduction in maximal aerobic power is the low arterial oxygen content, whereas decreased cardiac output is the reason for the persistance of the impairment after several days at high altitude.

Appendix

A. The barometric pressure at any given altitude depends on the weight of the air column above it. Therefore, the weight of the air at sea level must necessarily exert a greater pressure than the reduced weight of air at any altitude. In the 1800's, scientists started defining altitude by the barometric pressure usually measured in the height of a column of mercury in a barometer. At sea level, the column reaches 760 millimeters (mm) and is referred to as 760 mm of mercury (Hg) or simply 760 mmHg. It is common practice to also refer to mmHg as "Torr" (an abbreviation of the name of the person who developed the barometer, Evangelista Torricelli). One mmHg = one Torr (17).

B. Mitochondria are small, sausage-shaped structures found in every living cell. Their function is to generate most of the energy for cellular work. A constant supply of oxygen must be made available to these structures so that energy can be continually produced.

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Figure Legends

Figure 1. Percent reduction in VO_2max as a function of altitude exposure. Note that VO_2max is not measurably altered until the altitude exposure exceeds 1500 m or about 5000 feet. Above 1500 m, there is a linear decrease in VO_2max at the rate of 10% per 1000 m. Adapted from Buskirk (4).

Figure 2. The relationship of VO_2max to systemic oxygen transport. Adapted from Hartley (15).

Figure 3. Ventilation (BTPS) in relation to oxygen uptake at different exercise intensities while breathing 100% oxygen, at sea level breathing air, and during exposure to 1000 m, 2000 m, 3000 m, and 4000 m. Modified from Astrand (1).

Figure 4. The effect of altitude exposure on the ability of the oxygen in the blood to become equilibrated with the oxygen in the lungs. Modified from West (25).

Figure 5. Effect of increasing altitude on blood saturation at rest and during exercise at sea level and at different altitudes. Modified from Banchero (3).

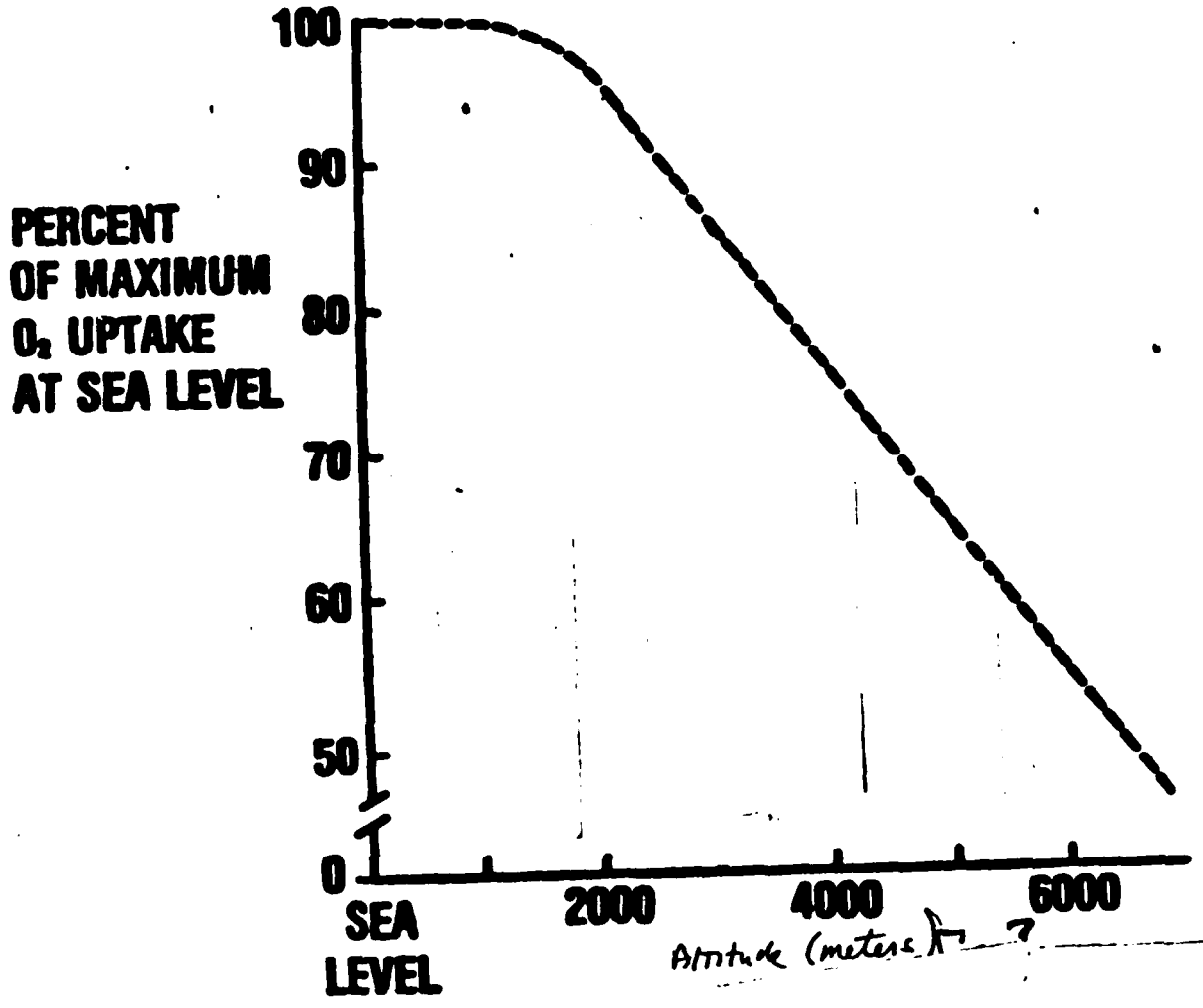

PERCENT OF MAXIMUM O_2 UPTAKE AT SEA LEVEL
100
90
80
70
60
50
0
SEA LEVEL
2000
4000
6000
Altitude (meters)

Figure #1

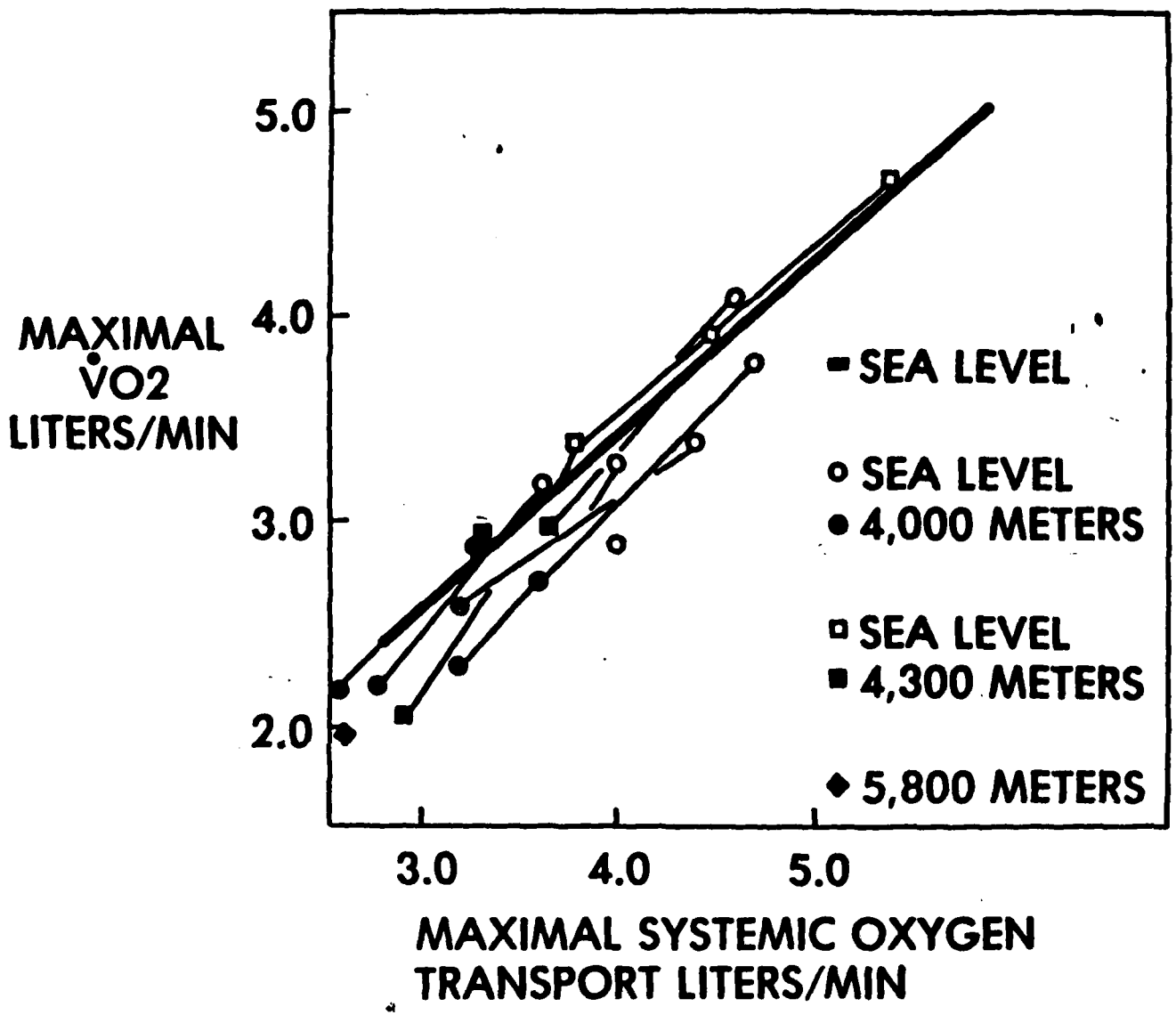

MAXIMAL
$\dot{V}O_2$
LITERS/MIN
5.0
4.0
3.0
2.0
SEA LEVEL
SEA LEVEL
4,000 METERS
SEA LEVEL
4,300 METERS
5,800 METERS
3.0
4.0
5.0
MAXIMAL SYSTEMIC OXYGEN
TRANSPORT LITERS/MIN

Figure 58-2

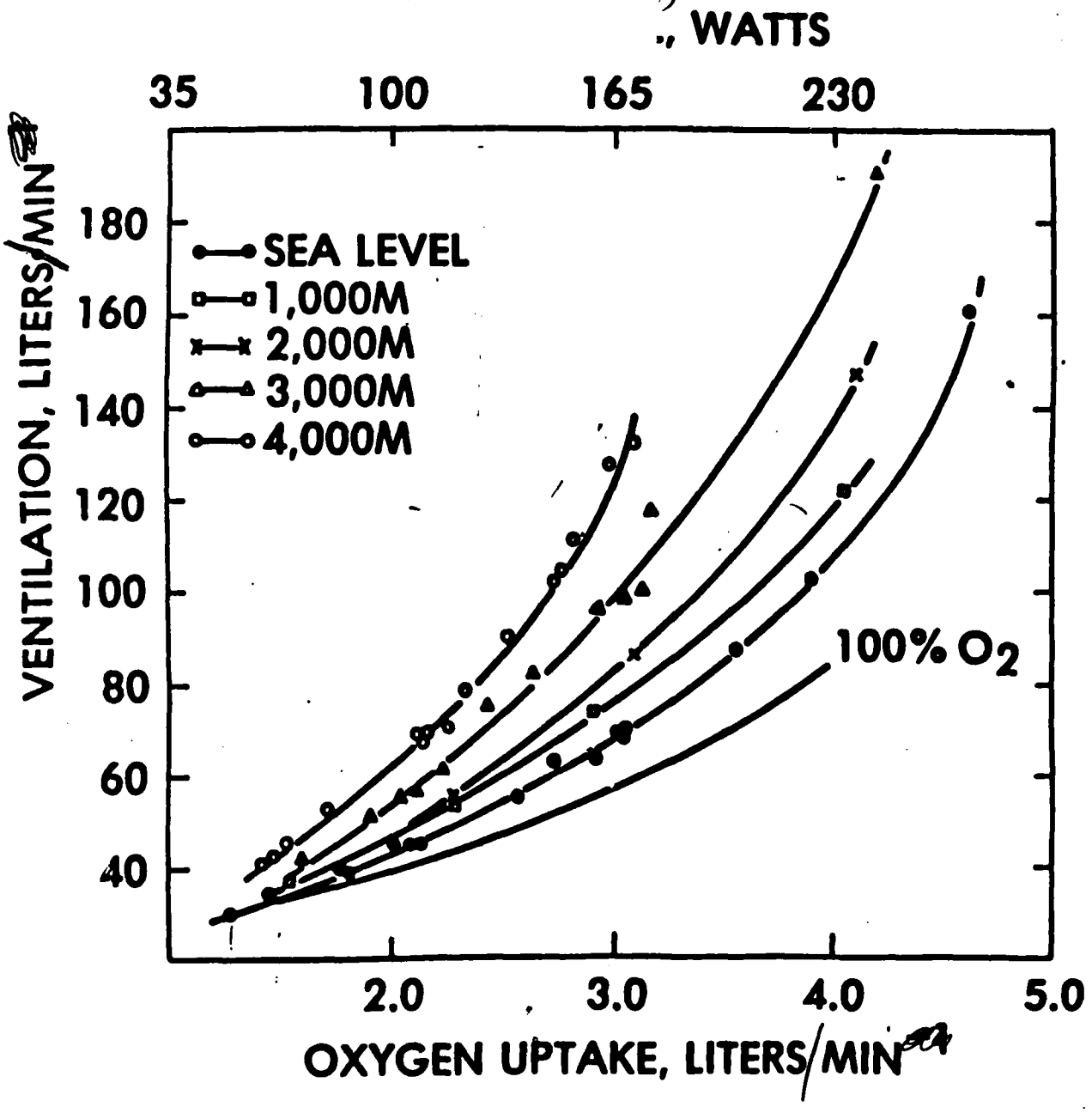
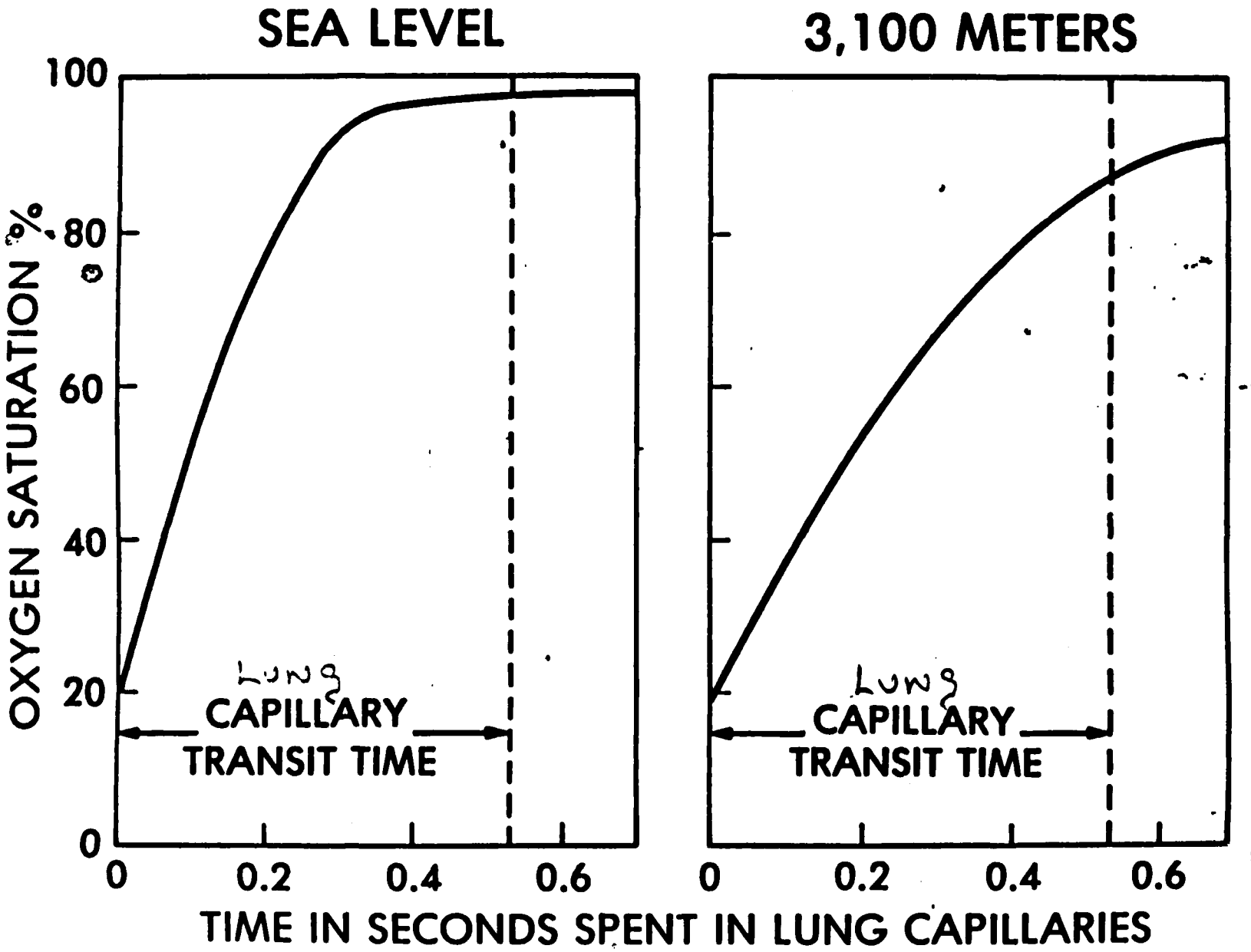
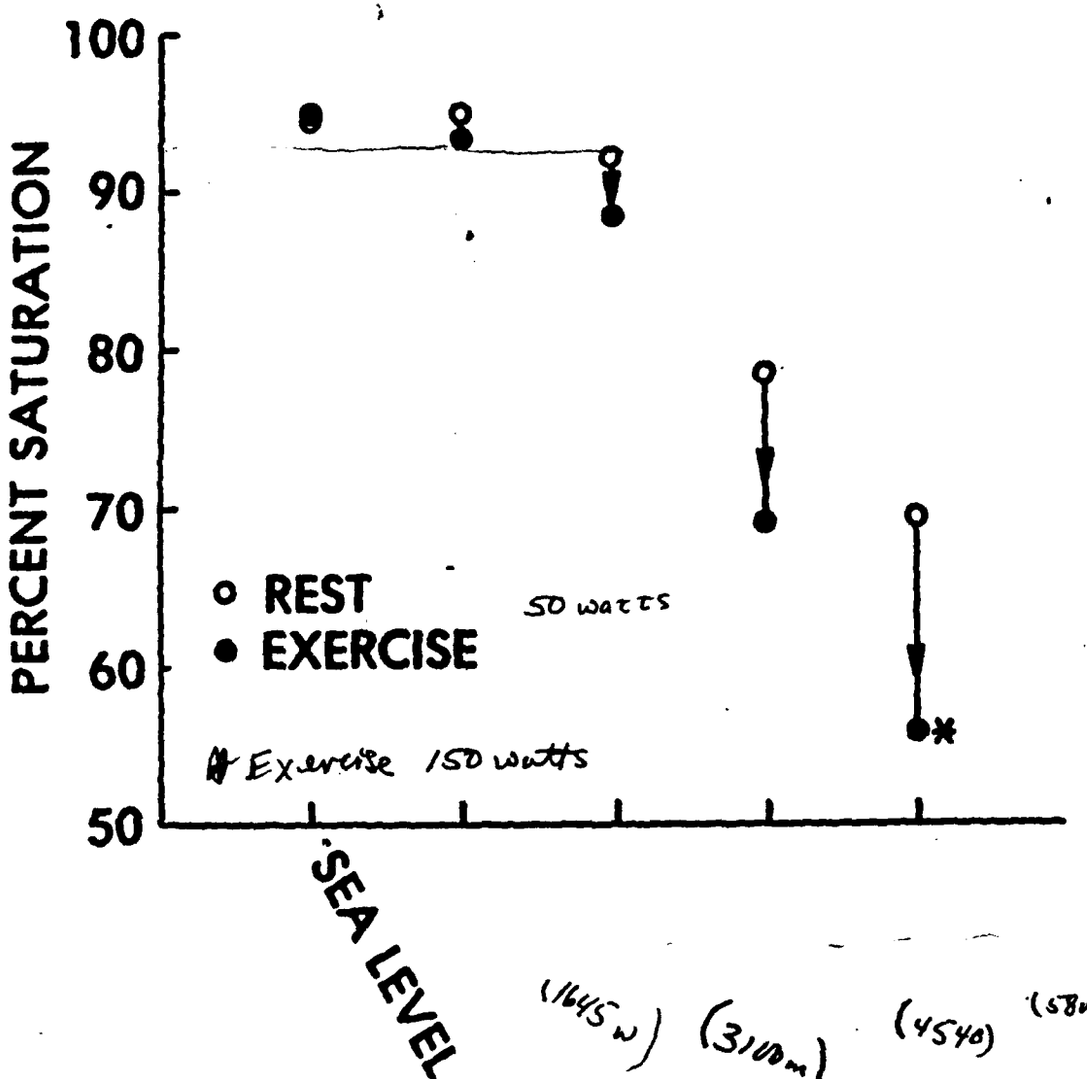

Exercise Intensity
WATTS
35
100
165
230
VENTILATION, LITERS/MIN
180
160
140
120
100
80
60
40
SEA LEVEL
1,000M
2,000M
3,000M
4,000M
100% O2
2.0
3.0
4.0
5.0
OXYGEN UPTAKE, LITERS/MIN

Figure 3


SEA LEVEL
3,100 METERS
100
80
60
40
20
0
OXYGEN SATURATION %
Lung
CAPILLARY
TRANSIT TIME
Lung
CAPILLARY
TRANSIT TIME
0
0.2
0.4
0.6
0
0.2
0.4
0.6
TIME IN SECONDS SPENT IN LUNG CAPILLARIES



Banchero et al., 1966

Figure 05

END

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