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1. Introduction

A skydiver should have a basic understanding of the effects of altitude and low gas pressures on the human body. This is not only for one’s own benefit but also in order to recognise related problems in other skydivers and pilots.

Although three areas of concern can be identified, the two primary areas of concern for skydivers relate to:

- The effects of hypoxia on the body
- The effects of other factors such as health, temperature and jump type

The third area relates to the effects of decompression sickness, but as this only really occurs at altitudes above 25000ft AMSL it is not relevant to recreational skydiving.

Most people would think our breathing is regulated mainly by oxygen, but under normal conditions oxygen has very little effect. Understanding why this is so helps to explain why hypoxia is potentially dangerous, as is skydiving when suffering from a respiratory or circulatory disorder.

Before discussing this further a basic understanding of how we breathe is required.

This, and related topics, are very briefly covered in points 1 to 4. Point 5 deals with some frequently asked questions, and point 6 with symptom recognition and simple tests to assist in detecting hypoxia.

This document is not intended to serve as a comprehensive discourse but rather as brief introduction. For further reading simply search the internet for aviation medicine journals or topics related to respiratory physiology.
2. **We thrive on pressure!**

We breathe a mixture of different gases and hence refer to the partial pressure of the respective gas, for example, PO$_2$ and PCO$_2$ refer to the partial pressures of oxygen and carbon dioxide respectively. These may be measured in mm Hg (millimetres mercury). These values assist in understanding the rate at which these gases can diffuse and therefore enable us to gain oxygen (needed for cellular metabolism) and loose carbon dioxide (produced by cellular metabolism).

At sea level, air normally contains:

- 79% nitrogen
- 21% oxygen
- With a total pressure of the mixture being approximately 760 mm Hg, thus
- At 21% of the 760 mm Hg, oxygen exerts a partial pressure of 160 mm Hg.

Obviously, the greater the partial pressure of oxygen breathed in, the more oxygen there is to be gained.

As our cells produce CO$_2$, it is usually present in our blood at a higher concentration than the outside air. Luckily for us CO$_2$ diffuses roughly 20 times quicker through our tissues than O$_2$, so it is lost with the air we expire quite easily. This is a critical step to explaining the insidious (subtle and sneaky) development of hypoxia.

Normal values at sea level in the alveoli of the lung (area in the lungs where inspired air is exchanged with pulmonary blood) are in the region of:

- PO$_2$ = 104 mm Hg
- PCO$_2$ = 40 mm Hg

You will notice that this value for oxygen is substantially less than the 160 mm Hg present in the air inspired. This dilution is mainly due to the addition of some water (moisture from the airways) and the mixing of air already present in the lungs that has a lower oxygen value.
Many other factors influence the ability to get oxygen into the blood.

- **DISTANCE:** Increasing the distance the oxygen has to travel from the alveoli to the blood vessel is an important factor in lowering the diffusion rate – hence **inflammation and infection** can significantly reduce the ability of the inspired air to oxygenate blood.

- **RESISTANCE:** Narrowing of the airways also reduces the speed at which the air breathed in can reach the alveoli. As **asthma** sufferers will know, this can be debilitating.

- **FLOW:** Anything reducing blood flow to or through the lungs can also present a problem, as both adequate blood and ventilation are required in order to successfully acquire oxygen and release carbon dioxide.
3. **We need chemicals...**

It may surprise many to learn that our breathing is normally regulated more by blood carbon dioxide and pH than by blood oxygen levels.

The basic goal of respiration is to maintain the necessary concentrations of various chemicals in body fluids, notably O$_2$, CO$_2$ and H$^+$ (hydrogen ion).

- Elevated CO$_2$ and H$^+$ affect the brain causing a greatly increased strength of both inspiration and expiration.
- Oxygen only has a significant effect on breathing when alveolar PO$_2$ is 50% less than normal.
- Only when PO$_2$ has dropped to less than 40 mm Hg (from normal 104!) does ventilation show a **1.5-fold increase**.
- On the other hand, PCO$_2$ has only to increase to 50 mm Hg (from normal 40) to cause a **4-fold increase**!

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**This weak effect of lowered PO$_2$ on breathing is due to strong opposition by CO$_2$ (and blood pH) on brain functions that govern how we stimulate muscles for inspiration and expiration.**
Consider the following sequence of events following exposure to low PO₂ pressures:

- As the PO₂ drops (to approximately 50% normal) increased breathing is stimulated (hyperventilation in response to hypoxia). This can result in about 2/3 increase in breathing.
- Remember that CO₂ is able to move through tissues at a rate roughly 20 times faster than O₂.
- The increased rate of breathing results in an increased rate of CO₂ loss. (This is a combination of ventilation and changes in heart rate)
- This has an immediate effect on blood pH (causing it to increase) as well as blood CO₂ (causing it to decrease).
- These changes in blood pH and CO₂ have strong inhibitory effects on neural mechanisms driving the muscles of inspiration and expiration, causing the respiration rate to drop.
- This overrides the drive from the low O₂ and thus reduces the ability to gain more O₂ from the air that is inspired.
- The drop in respiration rate also corrects blood pH and blood CO₂.
- Once corrected, the inhibition of the respiration rate is reduced.
- This means that the total response to low O₂ is perhaps as much as 2/3 above normal, which is actually a comparatively slight increase.
- This sequence continues and even if one consciously tries to achieve a higher rate of ventilation it becomes increasingly difficult to do so.
- Hypoxia may thus ensue, prior to which you may not consciously be aware of this tug-of-war between the need to increase O₂ and keep blood CO₂ constant.
What may surprise most people is that this basic pattern of breathing is present in all of us, and some of us do this on the ground normally.

That is, we have a period of deeper more rapid breathing, followed by respiration inhibition due to CO₂ loss, followed by a slow increase in blood CO₂ and stimulation of breathing again. This occurs over intervals of less than one minute to 3 minutes, and is referred to as periodic breathing.

An accentuated pattern of this type of breathing is what basically occurs in people with hypoxia.

This pattern can also be noted in people trying to recover from low O₂ exposure when sleeping (the diminished respiration of sleep worsens the pattern). Many skydivers can relate to interrupted sleeping patterns after a day of multiple skydives despite being tired and needing to sleep.
4. **Adapt or descend.**

The fact that we can adapt to increased blood concentrations of CO₂ is due to the fact that it has a *potent acute effect*, but *weak chronic effect*, on controlling respiration.

Therefore, after 2 days exposure to low PO₂ the increased rate of respiration and resultant low blood CO₂ has about one fifth of the initial effect. This explains how one can climb high altitude mountains with the correct acclimatization procedures. This adaptation may allow for 6-fold increase in ventilation.

The catch for skydivers is that we don’t hang around at an elevated base camp for hours, let alone days. The potent acute effect peaks within a few minutes – well within the time frame of normal plane climbing to exit altitude.

These effects are largely due to blood chemistry changes. But one doesn’t have to climb Mount Everest, or hop in an un-pressurized plane and keep climbing with no oxygen supply to wait for hypoxia to occur.

The required low O₂ (< 40 mm Hg) can be achieved in the lung (alveoli) even if the outside air has an adequate PO₂.

**Alveolar PO₂ values in the region of 40 mm Hg can be achieved by skydiving below hazardous altitudes with respiratory and/or cardiac abnormalities, such as emphysema or any condition resulting in:**

- Low rate of ventilation of alveoli
- Low rate of perfusion (blood flow) to functional areas of the lung
A similar sequence as detailed in point 3 earlier can also be induced at sea level by certain psychiatric conditions, such as various stress related responses. Someone suffering from hyperventilation will achieve a similar pattern as to the middle stage of the sequence detailed in point 3, except that the blood pH will elevate to a point where the person may pass out. As they are unable to control the accelerated breathing rate the familiar “breathe in and out of a brown-paper bag” routine may follow.

The relevance of this to skydivers in general, and AFF instructors in particular, is that should one observe a pattern of hyperventilation on the ground it should be monitored closely. If it continues in the plane it is probable that the student may pass out as the climb to altitude continues, or may have a reduced capacity for clear thought and/or for physical activity. It is critical to note that this may also occur after free fall when under canopy, so the bottom line is don’t take the risk. Call off the jump and descend to the ground with the student.

(See point 6)

Some abnormal conditions where O2 does strongly influence respiration can arise when increases in CO2 and H+ are prevented from changing, such as:

Pneumonia, emphysema (conditions where gas exchange is limited)

It is recommended that someone with a similar respiratory condition consult a medical doctor who specializes in lung physiology or aviation physiology prior to either flying a plane or performing a skydive.
5. **Some FAQs**

**What if I hyperventilate on purpose before the jump?**

You don’t have to purposefully try to increase your ventilation rate as this may occur even as an unconscious anticipatory signal from the brain. The problem is that within 1 minute the chemistry changes mentioned earlier intervene to adjust the rate again. In fact, this is why slow, calm breathing to relax the heart rate and ventilation rate are preferred techniques, as any sudden change in blood chemistry affects the ability of the nervous system to respond appropriately.

Also remember, excessive ventilation can cause a significant drop in blood CO₂ and therefore limit the ability to respond to an increased need for O₂. This can lead to a poorer response to acceptable altitudes as even a normal ventilation response may be suppressed.

**Getting hot or cold in the plane good or bad?**

Increased temperature has both a direct and indirect effect of increasing ventilation. A similar pattern can be noted in people with fever. Again, excessive heat can suppress your ability to respond to an increased need for O₂ on the trip up to altitude.

Usually, however, we experience being cold more than hot (sitting on the runway versus climb to altitude). Being cold is more dangerous as the exposure typically occurs at the altitude increases and the normal response to this is to increase the rate of oxygen consumption of the body. This further increases the need for adequate oxygen.
What effects occur at various Altitudes (all AMSL)

8000 ft: The ventilation rate begins to be affected. Blood saturation is around 93 % which is enough to result in a significant chemically-induced response. This increased ventilation rate is technically already a response to hypoxia.

9000 ft: The possibility exists for hypoxia to result in decreased mental judgement, memory, and motor nerve movements, specifically in those exposed repetitively and/or for long periods, and in those with respiration and/or cardiac deficiencies.

10 000 ft: The PO₂ in air has fallen from 160 mmHg to 110 mm Hg, and the PO₂ in the alveoli to 67 from 104! Yet despite this up to 10 000ft AMSL the saturation of blood with O₂ remains around 90%. This is good news for skydivers!

Beyond 10 000 ft: The O₂ saturation of blood falls progressively, and from 20 000 ft the PO₂ in air has fallen from 160 mmHg to 73 mm Hg, and the PO₂ in the alveoli to 40, yielding an O₂ saturation in blood of around 70%.

11 000 ft: Hypoxia may result in up to 80 % loss in mental proficiency in those exposed for long periods of time.

12 000 ft: Hypoxia effects that may be noticed from this altitude include drowsiness, lassitude, fatigue and/or euphoria. Headaches tend to be more prevalent and intense with increasing altitude, finally resulting in twitching, convulsions and coma from around 23 000 ft.

16 000 ft: From here on up to 20 000 ft the maximum ventilation response is reached – which is only about 65 % above normal. Contrast this to being at the same altitude on a mountain for several days where the maximum response can increase ventilation to 400 % above normal!

TIME!!!: These statements are largely flexible due to persons being acclimatized to the changes, having different health states, and different durations of exposure. Staying at 15 000 ft for 1 hour can result in a loss in mental proficiency of 50 %.

The “ceiling”: A person can normally remain conscious till the saturation in arterial blood falls to 50 %. This occurs after 20 000 ft AMSL, with the ceiling in an unpressurized plane anywhere above 23 000 ft AMSL. Exposure at 28 000 ft under this scenario can result in diminished consciousness within 1 minute and coma within 3 minutes (for an unacclimatized person).
15 000 ft barrier: Many skydivers may have heard pilots referring to a 15 000ft regulatory barrier. One of the reasons for this is that complicated compounds are formed in blood to assist in lowering the affinity of red blood cells for oxygen (so that other tissue cells can get some!), but from 15 000 ft this mechanism actually works as a negative as it also tends to reduce the ability of red blood cells to “pick up” oxygen in the lungs.

What about the cardiac effects?

At high altitudes the heart may have to work harder to get blood through the lungs as (some blood vessels constrict in response to the low oxygen). This can be dangerous and life threatening in those individuals already compromised. It can also result in a condition called acute pulmonary edema. It also affects other muscles resulting in a significantly decreased ability to do work at high altitudes.

Do we all respond the same?

No. There is significant variation between individuals exposed to the same conditions. Some of these variations may be related to levels of fitness or fatigue, others appear to be inherent.

How does strenuous activity during a jump affect us?

When having to perform an activity that requires strength our cells tend to use other forms of energy that do not require oxygen (anaerobic) as opposed to those that do require oxygen (aerobic or oxidative metabolism) – mainly as these anaerobic pathways can release energy much quicker than the aerobic ones.

This gives a source of energy that lasts for a few seconds to between 1 and 2 minutes. This also explains why some jumpers can do strenuous activity during free fall even though they may not be breathing, or have left the plane after emptying their lungs when screaming “Ready, set, go!”: The consequences are a build-up of lactic acid in blood and the tendency to continue to breathe at an increased rate once under canopy and/or when on the ground. This can go on for a few minutes to an hour and is due to an oxygen debt that has consequently arisen.

Repetitive jumps involving strenuous activity can result in a build up of lactic acid that can cause extreme fatigue – requiring some hours for recovery. The oxygen debt can recover between one to two hours, but can take days to recover if exhaustive depletion has occurred.

Note that different jumps involve different levels of energy expenditure – wingsuiting versus normal freefall.
6. Understanding the Signs that may occur

6.1 Symptoms of Hypoxia are due to a mixture of:

- Direct effects of a poor supply of oxygen to the tissues
- Attempts of the body to compensate for this
- Primary and secondary effects of other processes not directly related to the hypoxia.

Responses to hypoxia may be divided into the following categories:

<table>
<thead>
<tr>
<th>Category</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ventilation:</td>
<td>Hypoxia = Response is to increase ventilation (hyperventilation). This causes an excess loss of CO₂ = pH rises. This in turn inhibits ventilation. (termed hyperventilation induced hypocapnia)</td>
</tr>
<tr>
<td>Cardiovascular function:</td>
<td>Abnormally rapid heart rate (more than 100 beats/minute in an adult)</td>
</tr>
<tr>
<td>Cerebral function:</td>
<td>Highly sensitive = Effects range from decreased judgement and psychomotor disturbance to rapid loss of consciousness</td>
</tr>
<tr>
<td>Blood:</td>
<td>Skin and mucosal colour changes (darkening that may appear bluish = cyanosis)</td>
</tr>
<tr>
<td>Visual:</td>
<td>Partial or complete blindness</td>
</tr>
</tbody>
</table>
6.2 Recognising the symptoms:

The most common early warning signs are:

<table>
<thead>
<tr>
<th>Category</th>
<th>Symptoms</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cardiorespiratory</td>
<td>Tachycardia (increased heart beat rate)</td>
</tr>
<tr>
<td></td>
<td>Hyperventilation</td>
</tr>
<tr>
<td></td>
<td>Cyanosis</td>
</tr>
<tr>
<td></td>
<td>Dyspnoea (difficulty in breathing)</td>
</tr>
<tr>
<td>Neuromuscular</td>
<td>Incoordination, tremor, twitching, illegible writing and poor reproduction of geometric figures.</td>
</tr>
<tr>
<td></td>
<td>Failure to perform simple arithmetic calculations and immediate and delayed recall.</td>
</tr>
<tr>
<td></td>
<td>Perseveration (uncontrollable repetition of a specific response such as a word, phrase or gesture).</td>
</tr>
<tr>
<td>Visual symptoms</td>
<td>Headache and dysoesthesia (sensory impairment)</td>
</tr>
</tbody>
</table>

The symptoms can be divided into two types of categories, namely those that are perceived by the person experiencing hypoxia, and those that can be noted by someone observing such a person.
<table>
<thead>
<tr>
<th>Subjective effects (perceived by person experiencing hypoxia):</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Visual</strong></td>
</tr>
<tr>
<td><strong>Central Nervous System:</strong></td>
</tr>
<tr>
<td><strong>Neuromuscular symptoms:</strong></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Observed effects (person observing someone experiencing hypoxia)</th>
</tr>
</thead>
</table>
| **Impaired memory functions:** | Impaired immediate recall  
| | Impaired delayed recall  
| | Semantic memory errors |
| **Impaired computational functions:** | Simple arithmetic errors |
| **Impaired visual-motor functions:** | Motor incoordination  
| | Thought or motor block (simple calculations) |
| **Neuromuscular symptoms:** | Tremors, twitching |
| **Cardiorespiratory:** | Hyperventilation;  
| | Difficulty breathing;  
| | Increased pulse rate;  
| | Cyanosis |
6.3 Simple tests for hypoxia

In addition to having an awareness of the symptoms that may occur, we actually do many simple tests during AFF procedures in the plane that can assist in detecting hypoxia (see below).

Understanding these responses that a student can give are an important contribution towards increased safety as they may help to detect similar responses in experienced skydivers.

Some simple tests to perform include:

<table>
<thead>
<tr>
<th>Test type</th>
<th>Examples</th>
</tr>
</thead>
<tbody>
<tr>
<td>Simple recall and computational problems</td>
<td>“What is your opening altitude?” = 55000ft</td>
</tr>
<tr>
<td></td>
<td>“How many thousand feet left from 7000 ft to opening altitude?” = 1500 ft</td>
</tr>
<tr>
<td></td>
<td>“What are the priorities of freefall?”</td>
</tr>
<tr>
<td>Eye-hand coordination</td>
<td>“Show me 5500ft on your altimeter”</td>
</tr>
<tr>
<td>Semantic memory and visual-motor coordination</td>
<td>“Show me your malfunction drill” = (say correct words and perform correct actions)</td>
</tr>
</tbody>
</table>

We could also include some of these signs in our briefings:

“If you experience a loss of vision, or tingling sensation, please let us know.”

One could extend similar tests to experienced jumpers.

How often haven’t you heard one team member ask another in the plane:

“Am I facing the prop on this exit?” (AE)
“What formation is after the stairstep?” (FS)
“What is the climb-out sequence again?” (AE + FS)

Remember that the recognition of the dangers of hypoxia do not only relate to freefall and safely opening a parachute, but also to safe canopy flying and depth and computational requirements for safe landing.

Proactive safety entails recognising the insidious nature of hypoxia. An increased awareness of the symptoms and interpretation of the signs could help to significantly reduce injuries on landing that are often simply put down to “a judgement error”.

James Meyer (PhD Physiology) July 2007