Environmental Physiology at the Johnson Space Center: Past, Present, and Future

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Universities Space Research Association

UTMB, Introduction to Aerospace Medicine
July 19, 2007
three guiding principles at JSC

- Keep healthy astronauts healthy.
  - a different approach than treating the sick
  - an occupational health model
- Risk reduction -- ALARA
  - we don’t study decompression sickness (DCS), we limit the risk.
  - we don’t study acute mountain sickness (AMS), we limit the risk.
  - we must stay non-invasive in what we do.
- Operational reality
  - Use what you know, very often forced to extrapolate.
  - JSC is not a medical or academic research center.
consequences of these principles

- We do more prevention than treatment.
- We often lack specific data for specific questions because we respond to immediate needs.
- A non-invasive approach maximizes subject safety but limits research opportunity.
- We constantly assess risk as:
  - the probability of the event and consequence of the event.
environmental physiology

- **Pressure**
  - hypobaric and hyperbaric

- **Gases**
  - hypoxia and hyperoxia
  - hypercapnia – closed space issues
  - inert gas physiology / respiration

- **Temperature**
  - hypothermia and hyperthermia
    - thermal comfort
  - Protective clothing
    - diving, aviation, mountaineering, space

- **Acceleration**

- **Noise and Vibration**

- **Exercise / Performance**

- **Acclimatization / Adaptation**
  - engineering solutions when necessary
we don’t like rapid pressure change

“We’ve made it, Warren! ... The moon!”
environmental chambers at JSC

- Environmental Test Article
- 11-foot chamber
- 8-foot chamber
- Skylab simulation chamber
- Two hypo and two hyperbaric chambers
- Chamber B
- “giant” thermovaccum chamber
- Neutral Buoyancy Laboratory
- Space suit / personal rescue sphere
- Thermal chamber
In-suit Doppler Bubble Detector
reducing the risk of decompression sickness

<table>
<thead>
<tr>
<th>Program</th>
<th>Cabin Pressure, (psia)</th>
<th>Cabin Oxygen Concentration, volume %</th>
<th>EVA Suit Pressure, (psia)</th>
<th>EVA O₂ Prebreathe Time, minutes</th>
<th>EVA Prebreathe Conditions</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mercury</td>
<td>5</td>
<td>100</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Gemini and Apollo</td>
<td>5</td>
<td>100</td>
<td>3.75</td>
<td>0</td>
<td>-</td>
</tr>
<tr>
<td>Skylab</td>
<td>5</td>
<td>70</td>
<td>3.75</td>
<td>0</td>
<td>-</td>
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<tr>
<td>Shuttle</td>
<td>10.2</td>
<td>26.5</td>
<td>4.3</td>
<td>40</td>
<td>In-suit (36 hrs at 10.2 psia)</td>
</tr>
<tr>
<td></td>
<td>14.7</td>
<td>21</td>
<td>4.3</td>
<td>240</td>
<td>In-suit</td>
</tr>
<tr>
<td>ISS</td>
<td>14.7</td>
<td>21</td>
<td>4.3</td>
<td>120-140</td>
<td>Mask and in-suit; staged w/exercise</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>240</td>
<td>In-suit</td>
</tr>
</tbody>
</table>
classification of DCS

- **Type I – pain-only**
  - “pain” as just an awareness (Grade 1)
  - “pain” at a threshold (Grade 2)
  - “pain” enough to impair performance, and therefore stop a test (Grade 3)

- **Type II – serious DCS**
  - should stop an EVA
  - could result in long-term injury, or even death

- Ultrasound monitoring for venous gas bubbles is a non-invasive way to understand decompression stress and monitoring for arterial gas is a safety plan.
Doppler Ultrasound Technology

- Non-invasive measure of decompression stress
- Spencer 0 – IV Venous Gas Emboli Scale
- Monitor
  - Pulmonary artery – all of cardiac output
  - Subclavian vein
  - Mid-cerebral artery – where it really matters
  - Four chamber view of the heart
Tissues, especially adipose, dump bubbles in venous system
pulmonary artery VGE video
four-chamber ultrasound video
Argo II, 1994
Exercise during prebreath is now hot.
1983 – 2007: 914 exposures, 121 cases of DCS with 7 classified as Type II

- 86.8% no DCS
- 0.8% Type II
- 12.5% Type I

783 exposures, 326 with VGE detected

- 58.4% VGE 0
- 6.8% VGE II
- 10.7% VGE III
- 5.2% VGE I
- 18.9% VGE IV
the probability of DCS

- simple probability models are:

  \[ P(\text{DCS}) = \frac{\text{dose}^a}{\text{dose}^a + b^a} \] “Hill function”

  \[ P(\text{DCS}) = 1 - e^{-\text{dose}} \] “survival function”

  \[ P(\text{DCS}) = \frac{1}{1 + e^{-(B_0 - B_1 \times \text{dose})}} \] “logistic function”

- dose as simple one variable tissue ratio (TR)
- dose as more complex multivariable expression (TR, age, time, exercise, bubble volume, gender, etc.)
\[ P1N2 = P_0 + (P_a - P_0) \times [1 - \exp\left(-\ln(2) / t_{1/2}\right) \times \text{time}] \]

\[ TR = \frac{P1N2}{P_2}, \text{where } P2 \text{ is 4.3 psia suit pressure} \]

Diagram shows a graph with the following points:
- \( P_0 = 11.6, P_a = 0 \)
- \( TR = 2.40 \)
- \( TR = 2.14 \)
- \( TR = 1.70 \)

Y-axis labeled as \( P1N2 \) (psia)
X-axis labeled as resting prebreath time (min)
decompression sickness as dose-response

![Graph showing the relationship between dose and response](image)

- **Response (dcs, vge)** vs **Dose** (function of prebreath, altitude, time, age, etc.)
Fig. 1. The DCS and VGE failure distributions for data used in this analysis. Notice that each curve is "S" shaped, which helps to define an appropriate hazard function.

Fig. 3. The P(DCS) at either 3.5, 4.3, or 6.0 psia with (solid line) or without (dashed line) exercise at a particular time after decompression. The ratio of P1N2 to P2 (TR) in Eq. 5 was 1.65 for each curve, but notice the P(DCS) increases as P2 decreases at any particular time after decompression. The 95% confidence interval is provided for the curve specific to the 4.3 psia exposure that included exercise.

Fig. 5. Predicted vs. observed DCS incidence in 66 groups used to fit Eq. 5. The area of a circle is proportional to the number of people in a group. The three dark circles are results from NASA tests at 4.3 psia with TRs between 1.60 and 1.65 where exercise is I2 circles above identity line) and is not (circle below identity line) part of the test (4). The model neither over or under estimates the entire data set, but did over estimate the incidence of DCS in several small groups that reported no symptoms.
the data we have

EMU, $\mu$ - gravity

shirt-sleeve, unit gravity

DCS data from:
NASA and USAF databases
Literature database
mild Type I DCS video
6 / 42 (14%) pain got worse

19 / 42 (45%) pain got better

17 / 42 (41%) pain stayed constant
Type II, or “serious” DCS

- Seven Type II cases in 914 NASA exposures (1983 – 2007)
  - four of the seven had no $O_2$ prebreathe
examples of serious DCS

- substernal disturbance
- unproductive cough
- dyspnea
- disruptions of:
  - motor
  - sensory
  - cognitive pathways in brain and spinal cord
- paralysis
- ataxia
- dysmetria
- vertigo
- numbness
- aphasia
- amnesia
- altered mood
more examples

- tinitus
- diplopia
- nystagmus
- hemianopsia
- confusion
- belligerence
- scatomo
- nausea

- cold sweat
- dyskinesia
- syncope
- severe headache
- vomiting
- pallor
- hallucinations
- depression
the puzzle of exercise and DCS

- exer. at depth
- exer. at altitude
- exer. minutes before
- exer. during recomp.
- exer. hours before
- exer. during prebreath
- exer. days before
- exer. after recomp.
- micronuclei activation

aerobic fitness linked to all these
cutis marmorata – several hours later!
treatment of DCS is a real challenge in space
back to the Moon, but in a different way
we want to stay longer and do more EVA
spacecraft atmosphere trade study

- **Underlying Assumptions:**
  - Efficient and frequent EVAs drive the exploration program.
  - Low pressure suit is always preferred to high pressure suit.
  - There is an operational value to a short in-suit prebreathe.
  - Vehicle atmosphere may not prevent risk of DCS during EVA.
    - Shuttle and ISS atmospheres are examples.
  - Dedicated hyperbaric treatment capability may not be present.

- **Atmosphere Design Considerations:**
  - Don’t want a significant risk of fire – NASA has bad experience with 100% O₂.
  - Limit hypoxia – you need O₂ breath-by-breath.
  - Prevent DCS and VGE.
    - Better to prevent rather than treat DCS, or to constantly embolize the lung.
  - Optimize atmosphere to allow safe and efficient EVAs.
### future spacecraft atmospheres

<table>
<thead>
<tr>
<th>Environment</th>
<th>$P_B$ psia mmHg</th>
<th>$F_{O_2}$ (%)</th>
<th>$P_{iO_2}$ mmHg</th>
<th>$P_{AO_2}$ mmHg</th>
<th>Actual Altitude m</th>
<th>Actual Altitude feet</th>
<th>Equivalent Air Altitude m</th>
<th>Equivalent Air Altitude feet</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>CEV + LSAM</strong></td>
<td></td>
<td></td>
<td></td>
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<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>normal</td>
<td>8.0</td>
<td>414</td>
<td>32.0</td>
<td>117</td>
<td>77</td>
<td>4,877</td>
<td>16,000</td>
<td>1,829</td>
</tr>
<tr>
<td>best case</td>
<td>8.2</td>
<td>424</td>
<td>34.0</td>
<td>128</td>
<td>86</td>
<td>4,816</td>
<td>15,800</td>
<td>1,158</td>
</tr>
<tr>
<td>worse case</td>
<td>7.8</td>
<td>403</td>
<td>30.0</td>
<td>107</td>
<td>68</td>
<td>5,029</td>
<td>16,500</td>
<td>2,438</td>
</tr>
<tr>
<td><strong>HABITAT</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>normal</td>
<td>7.6</td>
<td>393</td>
<td>32.0</td>
<td>111</td>
<td>71</td>
<td>5,182</td>
<td>17,000</td>
<td>2,286</td>
</tr>
<tr>
<td>best case</td>
<td>7.8</td>
<td>403</td>
<td>34.0</td>
<td>121</td>
<td>80</td>
<td>5,029</td>
<td>16,500</td>
<td>1,524</td>
</tr>
<tr>
<td>worse case</td>
<td>7.4</td>
<td>383</td>
<td>30.0</td>
<td>101</td>
<td>63</td>
<td>5,364</td>
<td>17,600</td>
<td>2,895</td>
</tr>
</tbody>
</table>

$P_{iO_2}$ is inspired $O_2$ partial pressure, computed as $(P_B \text{ mmHg} - 47) \times F_{O_2}$ (as decimal fraction).

$P_{A O_2}$ is computed acute alveolar oxygen partial pressure from alveolar oxygen equation.
Acute Mountain Sickness

- Signs and symptoms including headache, nausea, dizziness, fatigue, vomiting and sleeplessness following a recent gain in altitude with at least several hours at the new altitude in a hypoxic environment; likened to a bad hangover.
The incidence of AMS is highly variable. Some may show mild AMS symptoms as low as 1,981-2,438m (6,500 - 8,000 ft). One report claims that 25% of people are affected with quick ascent to 1,891m, with 90% of symptoms resolving in 3 – 4 days. Houston (1982) claims that 25-30% of people at 3,048m (10,000 ft) will experience some type of AMS. This doubles at 4,200m (14,000 ft) and nearly all people will show some signs of AMS by 5,486m (18,000 ft). Roach (1998) says about 5% of people who develop AMS at 3,962m (13,000 ft) will go on to develop life threatening pulmonary and / or cerebral edema.
“typical” response to hypobaric hypoxic exposure

- Ascent causes a decrease in \( P_aO_2 \) sensed by the peripheral and central chemoreceptors, leading to increased rate of pulmonary ventilation \( (V_E) \) — **but some show little change in \( V_E \).**

- Hyperventilation in response to hypoxia increases \( P_AO_2 \) and subsequently decreases \( P_ACO_2 \) and leads to a transient alkalosis.

- There is also a hypoxia-induced diuresis as the kidney attempts to establish normal pH with the excretion of bicarbonate — **but some show little change in urine output.**
acute hypobaric hypoxia video
the spectrum of hypoxia

- A sudden ascent to high altitude could kill you due to acute hypoxia while a gradual ascent to the same altitude could result in AMS or no symptoms at all.

- Symptoms of AMS take longer to develop (hrs-days).

- Severe and prolonged forms of AMS may lead to High Altitude Pulmonary Edema (HAPE) and High Altitude Cerebral Edema (HACE) and death.
Based on this committee’s recommendations:

- A diagnosis of AMS is based on a recent gain in altitude, at least several hours (>2) at the new altitude, and the presence of headache and at least one of the following symptoms: gastrointestinal upset, fatigue or weakness, dizziness or lightheadedness and difficulty sleeping.

- A score of three points or greater on the AMS Self-Report Questionnaire alone or in combination with the clinical assessment score is diagnostic of AMS.
## Self Report Questionnaire

Each question asked and the sum is calculated as the AMS self report score.

<table>
<thead>
<tr>
<th>Question</th>
<th>Score</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Headache</td>
<td>0     No headache&lt;br&gt;1 Mild Headache&lt;br&gt;2 Moderate Headache&lt;br&gt;3 Severe Headache, incapacitating</td>
</tr>
<tr>
<td>2. Gastrointestinal Symptoms</td>
<td>0     No gastrointestinal symptoms&lt;br&gt;1 Poor appetite or nausea&lt;br&gt;2 Moderate nausea or vomiting&lt;br&gt;3 Severe nausea &amp; vomiting, incapacitating</td>
</tr>
<tr>
<td>3. Fatigue and/or Weakness</td>
<td>0     Not tired or weak&lt;br&gt;1 Mild fatigue/weakness&lt;br&gt;2 Moderate fatigue/weakness&lt;br&gt;3 Severe fatigue / weakness, incapacitating</td>
</tr>
<tr>
<td>4. Dizziness / lightheadedness</td>
<td>0     Not Dizzy&lt;br&gt;1 Mild dizziness&lt;br&gt;2 Moderate dizziness&lt;br&gt;3 Severe dizziness, incapacitating</td>
</tr>
<tr>
<td>5. Difficulty sleeping</td>
<td>0     Slept as well as usual&lt;br&gt;1 Did not sleep as well as usual&lt;br&gt;2 Woke many times, poor night's sleep&lt;br&gt;3 Could not sleep at all</td>
</tr>
</tbody>
</table>
Clinical Assessment

The interviewers' ratings of three signs is added to the self-report score (Roach 1993)

6. Change in Mental Status
   0  No Change in Mental Status
   1  Lethargy / lassitude
   2  Disoriented/confused
   3  Stupor / semiconsciousness
   4  Coma

7. Ataxia (heel to toe walking)
   0  No Ataxia
   1  Maneuvers to maintain balance
   2  Steps off line
   3  Falls down
   4  Can't stand

8. Peripheral Edema
   0  No peripheral edema
   1  Peripheral edema at one location
   2  Edema at two or more locations

This system helped to standardize the diagnosis of AMS.
a debate is underway

- Despite over a century of research there remains a vigorous debate on the etiology and pathophysiology of AMS.

- Certainly the brain is the target organ of and responder to O₂ deprivation.
Paul Bert (1833-1886)

- A French Physiologist considered the founder of Aerospace Medicine.

- Demonstrated, that the symptoms of AMS could be prevented or relieved by oxygen breathing and so "Proved" that it was the decrease in partial pressure of oxygen & subsequent hypoxia at high altitude, that caused AMS.

- This doctrine that low partial pressure of $O_2$ alone is the cause for AMS has held true for 150 years.
But over the last thirty years, researchers have begun to question the conventional wisdom that the symptoms of AMS are solely due to low $O_2$ partial pressure.
“the diminution of barometric pressure acts upon the living beings only by lowering the oxygen tension in the air, in the breath, and in the blood which supplies their tissues…. The increase in barometric pressure acts only by increasing oxygen tension in the air and blood…. ” Paul Bert, 1878.

- Consequently, maintaining sea level equivalent partial pressure of $O_2$ at any and all altitudes we “assume” no signs and symptoms of AMS should be seen.
Variable Pressure with Supposedly Equivalent Normoxia

<table>
<thead>
<tr>
<th></th>
<th>A</th>
<th>B</th>
<th>C</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>21% O₂ @ 760 mmHg</td>
<td>31% O₂ @ 523 mmHg</td>
<td>49% O₂ @ 349 mmHg</td>
</tr>
<tr>
<td></td>
<td>Sea Level</td>
<td>10,000 ft</td>
<td>20,000 ft</td>
</tr>
<tr>
<td></td>
<td>( P_AO_2 = 104 ) mmHg</td>
<td>( P_AO_2 = 103 ) mmHg</td>
<td>( P_AO_2 = 104 ) mmHg</td>
</tr>
</tbody>
</table>

Equivalent normoxic air altitudes: A = B = C
no AMS is expected?

Variable Pressure with Supposedly Equivalent Hypoxia

<table>
<thead>
<tr>
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<td>14% O₂ @ 760 mmHg</td>
<td>21% O₂ @ 523 mmHg</td>
<td>32.5% O₂ @ 349 mmHg</td>
</tr>
<tr>
<td></td>
<td>Sea Level</td>
<td>10,000 ft</td>
<td>20,000 ft</td>
</tr>
<tr>
<td></td>
<td>( P_AO_2 = 61 ) mmHg</td>
<td>( P_AO_2 = 61 ) mmHg</td>
<td>( P_AO_2 = 61 ) mmHg</td>
</tr>
</tbody>
</table>

Equivalent hypoxic altitudes: A = B = C
all equal time-course and incidence of AMS symptoms?
Accumulated anecdotal evidence shows descent is far more effective for relief of AMS than enriched $O_2$ breathing alone.

Essentially opening the doorway for further investigation of an independent pressure factor.
**Tucker, 1983**

- Starts his experiments with subjects living at 1,524 m (5,000 feet).
- Takes them to 15,000 feet on air and site pressure on 14% O\(_2\).

<table>
<thead>
<tr>
<th></th>
<th>Normoxic, P(_A)O(_2) = 103 mmHg</th>
<th>Hypoxic, P(_A)O(_2)&lt;103 mmHg</th>
</tr>
</thead>
</table>
| **Normobaric, P\(_B\) = 760 mmHg** | Altitude 1520  
P\(_A\)O\(_2\) = 77  
No AMS symptoms | Altitude 1520 m  
P\(_A\)O\(_2\) = 47.1  
Mean AMS Score: 3.2 |
| **Hypobaric P\(_B\)<760 mmHg** | Altitude 4570 m  
P\(_B\) = 430 mmHg  
P\(_A\)O\(_2\) = 45  
Mean AMS Score: 6.7 |
Confirm the effect of hypobaria on the pathophysiology of AMS – hypobaric hypoxia caused modest hypoventilation combined with mild edema relative to normobaric hypoxia.

<table>
<thead>
<tr>
<th>Normobaric, ( P_B = 760 ) mmHg</th>
<th>Hypoxic, ( P_AO_2 &lt; 103 ) mmHg</th>
</tr>
</thead>
<tbody>
<tr>
<td>Altitude 1520 m ( P_AO_2 = 76 ) No AMS symptoms</td>
<td>Altitude 1520 m ( P_AO_2 = 47.1 ) Mean AMS Score: 2.0</td>
</tr>
<tr>
<td>Hypobaric ( P_B &lt; 760 ) mmHg</td>
<td>Altitude 4570 m ( P_AO_2 = 74.5 ) Mean AMS score: 0.4</td>
</tr>
</tbody>
</table>

Lake Louise scoring system
The pressure effect seems real, so to understand the total hypoxic stress means you have to understand the interaction between $O_2$ partial pressure and ambient pressure.

A variety of explanations have been proposed for AMS and the effect of barometric pressure.
hypobaric hypoxia vrs normobaric hypoxia

- Decreased gas density relative to 1 ATA
- Decreased quantity of gas in solution relative to 1 ATA
- Increased insensible water loss relative to 1 ATA
- Transient $N_2$ gradient out of tissues and CNS
- Potential for VGE

- Gas density at 1 ATA
- Gas in solution at 1 ATA
- Insensible water loss at 1 ATA
- Transient $N_2$ gradient into tissues and CNS
- No potential for VGE
in the past….

- NASA’s past habitats and vehicles did not expose the astronaut to a significant hypoxic condition.

- Our only experience is with the shuttle staged denitrogenation protocol where astronauts are at the physiological equivalent of 4,000 feet altitude.

- Likelihood of AMS almost nil.

- Specifies the development of human missions to the Moon, and then Mars.
- In order to accomplish this task NASA is required to build new interplanetary spacecraft, landers, space suits, rovers and surface habitats.
The atmospheres for these spacecraft, landers, surface habitats, and rovers will likely be hypobaric, and a little hypoxic.

Future Moon and Mars missions with CEV, LSAM and lunar habitat will require efficient EVA egress with minimal prebreathe time while still avoiding DCS and VGE.

The combination of hypobaria and hypoxia simulates the conditions encountered by mountain climbers.
SO.....

Are we putting future astronauts at an increased risk for AMS ?

Assume that we are, and develop a plan to mitigate the risk --- the JSC philosophy.
what happens if astronauts develop AMS?

- Based on extrapolation of current research, it seems unlikely that anyone will experience severe AMS.
- The bigger issue is likely “performance”, we want to maximize performance.
- The bigger issue is a mitigation plan.
We are dealing with performance issues and mission success, not life and death, with the AMS anticipated in the CEV.

We want to maximize performance and minimize any medical issues that impact mission success.

Montgomery (1989) stated that the incidence of AMS at 1,981m (6,500 ft) was approximately 12% and further stated that 50% of these subjects took medication for relief of symptoms.
prevention and treatment of AMS

- Preadaptation
- Preselection
  - The best predictor of AMS is history of prior episodes.
- Mild AMS is treated by:
  - Halting or slowing ascent
  - Acclimatization
  - Acetazolamide (125-250 mg BID)
  - $O_2$ therapy via mask or canula
other considerations

- Potential negative synergy between mild hypoxia and adaptation to μG.

- Does μG change the incidence of AMS?
  - redistribution of lung fluid
  - increased interstitial edema
  - altered incidence of HAPE?
optimum HCT for $O_2$ transport

**Fig. 5.5**—A, relative viscosity vs hematocrit for human blood and camel blood. B, the transport of oxygen through a glass tube vs hematocrit, with a constant driving pressure.
anticipated work in environmental physiology

- NASA / JSC has worked with:
  - USAF
    - Brooks AFB
    - Write-Patterson AFB in the distant future
  - Canadian Space Agency
    - DR&D – Toronto
  - Japanese Space Agency
  - Universities / Medical Centers
    - Duke University
    - University of Texas
    - University of Pennsylvania
    - Mayo – looking to the future
potential work to do in DCS

- Quantify PFO as a risk factor toward serious DCS.
- Understand the role of micronuclei in the genesis of bubbles.
- Consequence of air break in prebreathe – in progress.
- Exercise and accelerated N₂ washout.
- Exercise and change in micronuclei distribution.
- Validation of the current denitrogenation procedure for lunar EVAs.
- Data Mining -- Biophysical / statistical modeling of DCS.
- Effective DCS treatment at remote sites.
- Gender and risk of DCS and VGE.
- Application of ultrasound technology to monitor and understand decompression stress.
- Use of argon as an inert gas available on Mars.
potential work to do in AMS

- Quantify the risk and impact of AMS for modest hypoxic exposures.
- Specific experiments about AMS based on the atmospheres and conditions for the proposed CEV, LSAM, and surface habitats.
- Determine who may be at risk for developing AMS.
- Understanding the physics and physiology of the ambient pressure effect on AMS.
- Validate risk mitigation plans for AMS.
thank you from the folks at JSC
johnny.conkin-1@nasa.gov