**Time: 1730-1736**

**Poster #: 11 .INTRAOPERATIVE OXYGEN CONCENTRATIONS INCREASE PERIOPERATIVE OXIDATIVE STRESS IN A DOSE-DEPENDENT MANNER – A RANDOMISED CONTROLLED TRIAL (PULSE Ox) .** Andrew Cumpstey1, Anna Clark1, Magdalena Minnion1, Renato Nogueira2, Helen Moyses1, Daniel Martin3, Jose Tanus-Santos2, Mark Edwards1, Michael Grocott1, Martin Feelisch1. 1University of Southampton, 2University of São Paulo, 3University of Plymouth

Background: The World Health Organization (WHO) recommends all anaesthetised patients receive 80% oxygen during surgery to reduce the risk of surgical site infection (SSI). Results from the PROXI trial (no difference in SSI rates between 30% and 80% oxygen but possibly worse postoperative outcomes with 80% oxygen) would caution against this but were considered 'mechanistically implausible' (WHO).Objective: To investigate whether administering higher inspired oxygen concentrations during anaesthesia might increase systemic oxidative stress (and therefore predispose to adverse outcomes).Methods: Twenty-eight adult patients undergoing major (central venous catheter required) abdominal surgery were randomly allocated to receive 30%, 55% or 80% oxygen throughout anaesthesia. Paired arterial and central venous blood gases (to measure oxygen extraction) and samples were collected 2-hourly. Total nitroso species (RxNO) and Nitric Oxide (NO) scavenging were quantified using gas phase chemiluminescence. Total free thiols (TFTs) and ferric reducing ability of plasma (FRAP) were measured colorimetrically.Results: Higher oxygen was associated with higher RxNO concentrations (Mean[SD] 52.6[19.1]/100.2[36.1]/91.2[34.4] nM for 30/55/80% respectively, $p = 0.05$) & reduced NO scavenging (4.4[0.7]/3.5[0.4]/3.6[0.6]uM, $p = 0.02$) at the end of surgery. Normalized TFTs increased throughout surgery but with no difference between groups (4.5[0.3]/4.1[0.3]/4.2[0.4], $p = 0.26$). FRAP concentrations did not change overall (866.5[52.0]/908.1[37.1]/810.6[57.4], $p = 0.42$) but decreased markedly in some patients. Why inter-individual perioperative oxygen sensitivity differs merits further investigation. Tissue oxygen extraction reduced significantly with 80% oxygen (0.28[0.10]/0.23[0.08]/0.20[0.06], $p < 0.001$), supporting previous data in critically ill patients.Conclusion: Higher intraoperative oxygen concentrations significantly increase markers of oxidative stress, lower systemic antioxidant capacity and decrease oxygen extraction during surgery in a dose-dependent fashion.Funding: Doctoral Fellowship (Southampton NIHR Biomedical Research Centre)

**Time: 1736-1742**

**Poster #: 15 .EFFECTS OF NALTREXONE ON SLEEP QUALITY AND PERIODIC BREATHING AT HIGH ALTITUDE.** Katharine Foster1, James Anholm2, Gary Foster3, Prajan Subedi2. 1Emergency Medicine, Loma Linda University School of Medicine, Loma Linda, CA 92354, 2Pulmonary & Critical Care, VA Loma Linda Healthcare System & Department of Medicine, Loma Linda University School of Medicine Loma Linda, CA 92357, 3Cardiology, St. Charles Health System, Bend, OR 97701

Objective: This study examined the role of the Mu-opioid receptor (MOR) on breathing and sleep at high altitude (HA). We hypothesized that MOR blockade with naltrexone would result in higher nocturnal oxygen saturations, fewer apneas and improved sleep at high altitude.Methods: This double blind, placebo-controlled, crossover study included 9 healthy subjects aged, 27.9 ± 4.6 years. Two overnight trips spaced at least two weeks apart occurred from Loma Linda, CA (355m) to Barcroft Laboratory, CA (3810m) for each arm. Subjects took either 50 mg naltrexone or matching placebo at bedtime. Sleep metrics were recorded using WatchPATTM device (Itamar Medical Ltd.). Subjective data was measured with the Groningen Sleep Quality Scale, Stanford Sleepiness Scale and Lake Louise Score (LLS) for acute mountain sickness (AMS).Results: Mean overnight SpO2 was lower after taking naltrexone, 81 ± 6% vs. 83 ± 4% (mean difference 1.9 ± 2.1%), 95%CI=0.1–3.6, p=0.04). Minimum overnight SpO2 was lower on naltrexone 70 ± 6% vs. 74 ± 4% (dif. 4.6% ± 4.3%) CI=1.0–8.2, p=0.02). Total sleep time and total apnea-hypopnea index(AHI) were not different. Subjective sleep quality was significantly worse on naltrexone measured via Groningen ($p<0.03$) and Stanford Sleepiness Scale ($p<0.03$). AMS measure via the LLS was significantly worse while taking naltrexone ($p<0.03$).Conclusion: In contrast to our hypothesis, this study demonstrated a significant decrease in oxygen saturation and sleep quality with no change in sleep time or AHI. AMS scores were significantly worse after taking naltrexone. To our knowledge this is the first study to test physiologic effects of MOR blockade in humans at altitude. Further characterization of the MOR's vasoactive and sympathetic modulation and its effects on regulation of cerebral blood flow are needed to further interpret these results.

**Time: 1742-1748**

**Poster #: 20 .SLEEP AND BLOOD PRESSURE DURING A 12-MONTH STAY AT CONCORDIA STATION (3233 M), ANTARCTICA.** Michael Furian1, Paul Robach2, Stijn Thoolen3, Sarah Rommel3, Sebastien Baillieul1, Stephane Doutreleau1, Pierrick J Arnal4, Samuel Verges1. 1HP2 laboratory, Université Grenoble Alpes, Inserm (U1300), CHU Grenoble Alpes, Grenoble, 38000, France, 2Ecole Nationale des Sports de Montagne, 74400 Chamonix, France, 3French Polar Institute Paul-Émile Victor, Brest, 4Dreem, Paris, France

Introduction. Sleep architecture remains impaired when staying at the Concordia Station (3233m), Antarctica. The purpose of this study was to investigate sleep and blood pressure, and the pathophysiological role of hypoxia.MethodProspective cohort study in 23 subjects staying for 12 months at 3233m (N=11, mean±SD age 36±10y, BMI 24.3±3.1kg/m2) or in Dumont d'Urville, 20m (N=12, age 31±12y, BMI 22.3±3.1kg/m2), Antarctica. Before departure (BL) and in the 1st and 12th month at the Stations, sleep assessment (DREEM) and 24h ambulatory blood pressure (BP) monitoring was performed.ResultAt 3233m, subjects had less stage 3 sleep (%total sleep time, TST) in the 1st (mean±SE 18±2%TST) and 12th (18±2%TST) month vs BL (24±3%TST, both P<0.05). In contrast, proportion of stage 2 sleep and micro-arousals were higher in the 1st (50±2%TST, 10.1±1.0/h) and 12th (51±2%TST, 11.5±1.0/h) month vs BL (43±3%TST, 7.1±1.2/h, both P<0.05). At 20m, no changes occurred. At 3233m, nocturnal mean BP was higher in the 1st (85±2mmHg) and 12th (80±2mmHg) month vs BL (76±2mmHg, both P<0.05). The higher nocturnal BP was caused by a higher proportion of non-dipping defined by <10% Δnight-day BP, which was 0% at BL, 45% in 1st and 27% in 12th month at 3233m (P<0.05, 1st month vs BL). At 20m, no changes occurred. Conclusion. A 12-month stay at the Concordia Station at 3233m was associated with worse sleep and nocturnal BP compared to pre-departure. Since these impairments were not observed at Dumont d'Urville, preventive measures against hypoxia might be considered to improve outcomes in these crewmembers.

**Time: 1748-1754**

**Poster #: 36 .DIFFERENCES IN DNA METHYLATION BETWEEN ALTITUDE EXPERIENCED AND ALTITUDE NAÏVE HEALTHY VOLUNTEERS ON EXPOSURE TO HYPOBARIC HYPOXIA.**

Kay Mitchell1, Emma Garratt1, Michael Natoli2, Elie Antoun1, Matthew Hewitt1, Negusse Kitaba1, Andrew Cumpstey1, Thomas Smedley1, Nelson Diamond2, Timothy Beck2, Denny Levett1, Michael Mythen3, Andrew Murray4, Hugh Montgomery3, Daniel Martin5, Keith M Godfrey1, Richard Moon2, Karen Lillycrop1, Michael Grocott1. 1University of Southampton, 2Duke University, 3University College London, 4University of Cambridge, 5University of Plymouth

Objectives: We investigated skeletal myocyte DNA methylation patterns in altitude experienced (AE) and altitude naïve (AN) lowlander volunteers on exposure to hypobaric hypoxia and subsequent return to normoxia. Methods: Twenty-one healthy male volunteers were exposed to environmental hypoxia over 3 days in a hypobaric chamber (maximum altitude equivalent to 3500m, PaO2 8 kPa). Vastas lateralis skeletal muscle biopsies were taken at baseline, at the end of hypoxia, and 3 hours after return to normoxia. Following quality control and normalisation procedures methylation levels of cytosine-guanine sequences (CpGs), generated using an Illumina HumanMethyaltion EPIC bead array, were compared between baseline and hypoxia, and between hypoxia and subsequent normoxia using paired t-tests. Associations between sites of methylation change and all known biological pathways were sought. Results: Methylation patterns altered in response to hypoxia in all participants, with further changes following return to normoxia. The number of differentially methylated CpGs (dmCpGs) was greater in AE than AN participants following exposure to hypoxia (ratio 2.06:1, $p<0.001$), and subsequent return to normoxia (ratio 4.14:1, $p<0.001$), and differed between them. DmCpGs were enriched in MAPK and PI3K-Akt signalling pathways following exposure to hypoxia, and subsequent return to normoxia. Conclusions: Altered DNA methylation patterns were associated with hypoxic exposure and subsequent return to normoxia in healthy humans with differences in methylation patterns between AE and AN individuals. Methylation changes were associated with signalling pathways that may underpin the skeletal muscle response to hypoxia.

**Time: 1754-1800**
**Poster #: 28 .ACUTE MOUNTAIN SICKNESS DOES NOT IMPACT VENTILATORY ACCLIMATIZATION FOLLOWING ACTIVE AND PASSIVE ASCENT TO 3600M.** Steven Landspurg1, Peter Figueiredo1, Emma Atkinson1, Janet Staab1, Mark Buller1, Reed Hoyt1, Philip Karl1, Tim Mesite1, Beth Beidleman1, Jon Femling2, Jason Williams2, Aaron Reilly2, Trevor Mayschak2. 1US Army Research Institute of Environmental Medicine, 2University of New Mexico Health Sciences Center

Introduction: Whether acute mountain sickness (AMS) differentially impacts the magnitude or time course of ventilatory acclimatization at high altitude (HA) remains controversial. Methods: To determine whether AMS impacts ventilatory acclimatization following both passive and active ascent to HA, 78 healthy Soldiers (mean±SD; age=26±5yr) were tested at baseline residence (BLR), transported to Taos, NM (2845m), then hiked (n=39) or were driven (n=39) to 3600m, and stayed for four days. AMS-C was assessed using the Environmental Symptoms Questionnaire at HA twice on day 1 (HA1), five times on days 2 (HA2) and 3 (HA3) and once on day 4 (HA4). If AMS-C was ≥0.7 at any timepoint, individuals were categorized as AMS-susceptible (AMS+, n=33); others were categorized as non-susceptible (AMS-, n=45). Portable real-time capnography was used to measure resting partial pressure of end-tidal carbon dioxide (PETCO2 mmHg) at ~09:00 at BLR, and after 19h (HA2), 43h (HA3), and 67h (HA4) at HA. Resting pulse arterial oxygen saturation (SpO2, %) was measured immediately after PETCO2. Results: Ascent conditions did not differentially impact ventilatory responses. PETCO2 and SpO2 did not differ between AMS+ and AMS- groups at BLR or any time point at HA. The PETCO2(mmHg) in AMS+ vs. AMS- groups, decreased($p<0.05$) from BLR (37.2±3.6 vs. 36.9±3.1) on HA2 (33.5±4.1 vs. 33.7±4.0), remained stable from HA2 to HA3 (33.4±4.1 vs. 33.8±3.9) and decreased($p<0.05$) from HA3 to HA4 (31.8±4.4 vs. 31.4±4.1). SpO2(%) decreased($p<0.05$) in both AMS+ and AMS-, from BLR (97.3±1.5 vs. 96.9±1.5) on HA2 (87.9±3.1 vs. 88.8±2.9) and remained stable from HA2 to HA3 (88.8±3.1 vs. 89.1±2.9) and HA4 (88.9±3.1 vs. 89.6±2.1). Conclusions: Ventilatory acclimatization occurred at HA, but AMS-susceptibility did not impact the magnitude or time course of acclimatization following active or passive ascent to 3600m. Authors' views not official U.S. Army or DoD policy. Funding: USAMRDC

**Time: 1800-1806**

**Poster #: 4 .RELATIONSHIP OF NOCTURNAL OXYGEN SATURATION TO SLEEP QUALITY AT HIGH ALTITUDE.** Diana Biggs1, Andrew Burns1, Greta Carlson1, Ilaria Ferrari1, Lukas Sloan1, Linda E. Keyes1. 1University of Colorado

Objective: Few studies have investigated sleep quality vs. nocturnal SpO2, but rather have compared sleep fragmentation and nocturnal wakening to sleep quality. We evaluated the hypothesis that nocturnal hypoxia at high elevation would lead to poorer sleep quality.Methods: This prospective observational cohort study of adult lowlanders compared sleep quality measured by the Groningen Sleep Quality Scale (GSQ) and nocturnal SpO2 during participants' first 24h at high altitude (2470-2700m). SpO2 was measured continuously overnight using the Nonin WristOx.Results: We report preliminary data on 9 participants, (mean age = 46 yo, female = 6), none with a history of OSA. Mean basal nocSpO2 was 87%, 95% CI [85-89], mean minimum nocSpO2 75%, 95% CI [70-80] and mean percent time SpO2<88% 51%, 95% CI [27-75]. Mean GSQ was 7, 95% CI [4-10]. GSQ scores were not associated with minimum nocSpO2 (R2 = 0.0) or percent of time SpO2<88% (R2 =0.0). Lower mean nocSpO2 was associated with better perceived sleep quality (lower GSQ) (R2=0.6).Conclusion: Contrary to our hypothesis, despite participants reporting poor sleep quality the first night after arrival to high altitude, lower nocturnal SpO2 was not associated with worse sleep quality. We are unsure why higher mean nocturnal SpO2 was associated with worse sleep quality but suspect other unmeasured variables affect sleep quality.Funding: This work was supported by a Wilderness Medical Society Hultgren grant.

**Time: 1806-1812**

**Poster #: 44 .THE RELATIONSHIP BETWEEN SLEEP DURING THE FIRST NIGHT OF EXPOSURE TO 3600M ON ACUTE MOUNTAIN SICKNESS THE NEXT MORNING.** Bradley Ritland1, Peter Figueiredo1, Steven Landspurg1, Jon Femling2, Jason Williams2, Janet Staab1, Reed Hoyt1, Mark Buller1, J Philip Karl1, Aaron Reilly2, Trevor Mayschak2, Emma Atkinson1, Tim Mesite1, Beth Beidleman1. 1US Army Research Institute of Environmental Medicine, 2University of New Mexico

Objective: Sleep disturbances are common at high altitude (HA) (> 3500m), but the relationship between sleep and the incidence and severity of acute mountain sickness (AMS) is debated. The objective was to investigate whether sleep on the first night at HA was associated with AMS the next morning following active and passive ascent to 3600m. Methods: 78 healthy Soldiers (mean±SD; age=26±5yr) were transported from their baseline residence (BLR) to Taos, NM (2845m), where they hiked (n=39) or were driven (n=39) to HA (3600m) and assessed for two days (HA1 and HA2). Sleep was measured via actigraphy on the first night of sleep at HA (HA1) and used to calculate sleep awakenings (events/hr), duration (min), onset latency (min), wakefulness after sleep onset (WASO, min), and sleep efficiency (%). Mean pulse oxygen saturation (SpO2) was measured using pulse oximetry during sleep. AMS-Cerebral factor score (AMS-C) was assessed using the Environmental Symptoms Questionnaire on day 2 at HA (HA2). If AMS-C values were ≥0.7 on HA2, individuals were classified as AMS-susceptible (AMS+, n=23); others as non-susceptible (AMS-, n=55). Results: Ascent conditions did not differentially impact sleep measurements. There were no differences in sleep awakenings, onset latency, WASO, or sleep efficiency between the AMS+ and AMS- groups. In the AMS+ group compared to the AMS- group, sleep duration (min) was lower (392±57 vs. 433±63, p=0.009) and mean SpO2 (%) was lower (79.7±6.1 vs 82.0±3.9, p=0.05). Sleep duration (r=-0.32, p=0.004) and mean SpO2 (r=-0.32, p=0.005) values on the first night at HA were negatively correlated with AMS-C values the following morning on HA2. Conclusion: When Soldiers passively or actively ascend to high altitude (3600m), sleep duration and arterial oxygen saturation during the first night at HA is associated with AMS the next morning. Authors' views not official U.S. Army or DoD policy. Funding: USAMRDC

**Time: 1812-1818**

**Poster #: 24 .CHARACTERIZATION OF THE INFLAMMATORY RESPONSE TRIGGERED BY NORMOBARIC HYPOXIA.** Sonja Hersel1, Frank Splettstoesser1, Katrin Reiners2, Sarah Zaffarana2, Laura de Boni3, Henning Weis3, 4, Fabian Hoffmann3, 5, Jan-Niklas Hoenemann3, 5, Jens Jordan3, Ulrich Limper3, Jens Tank3, Stilla Frede1. 1Department of Anesthesiology and Intensive Care Medicine, University Hospital Bonn, Germany, 2Institute of Clinical Chemistry and Clinical Pharmacology, University Hospital Bonn, Germany, 3Department of Cardiovascular Aerospace Medicine, Institute for Aerospace Medicine, German Aerospace Center, Cologne, Germany, 4Department of Nuclear Medicine, University of Cologne, Germany, 5Department of Internal Medicine III, Division Cardiology, Pneumology, Angiology and Intensive Care, University of Cologne, Germany

Objective: Hypoxia and inflammation share interlinked cellular pathways. We investigated the hypothesis that exposure to hypoxia is sufficient to trigger pro-inflammatory processes in human immune and endothelial cells, which could be involved in inflammatory signaling at high altitudes. Methods: White blood cells (WBCs) were collected from normoxic healthy donors. Primary human pulmonary microvascular endothelial cells (HPMECs) were purchased from PromoCell. Cells were incubated for up to 24h under normoxic or hypoxic conditions with 10% or 1% oxygen (O2). Regulation of pro-inflammatory and hypoxia-inducible genes and proteins was evaluated by RT-PCR, ELISA and immunoblot (HIF-2α stabilization). Plasma samples were taken from participants of the MyoCardioGen 3 (MCG3) study who were exposed to sustained severe hypoxia (35 days, lowest O2 concentration 9.5%). The study included three participants who had suffered myocardial infarction and one healthy subject. Plasma from the MCG3 participants was analyzed by Luminex assay. In addition, extracellular vesicles (EVs) were investigated using nanoparticle tracking analysis (NTA). Results: WBCs and HPMECs exposed to 1% O2 showed a slight elevation of tested pro-inflammatory cytokines, which was absent under normoxic or 10% hypoxic conditions. A cellular response to hypoxia could be proven in all in vitro experiments. Luminex results showed changes in pro-inflammatory cytokine concentrations in plasma of MCG3 study participants. NTA analysis confirmed a change in EV numbers in in vitro and in vivo hypoxia experiments. Conclusion: Our study indicates that 1% O2 slightly increases the pro-inflammatory state in WBCs and HPMECs. Since donors exposed to sustained hypoxia showed a more pronounced regulation of inflammatory cytokines, we conclude that inflammatory processes in vivo are influenced by communication between different cell types. Unidentified humoral factors or EVs secreted by cells under hypoxic stress may represent a possible connection.Funding: No external funding.

**Time: 1818-1824**

**Poster #: 40 .MANIPULATION OF IRON STATUS ON CEREBRAL BLOOD FLOW AT HIGH ALTITUDE IN LOWLANDERS AND ADAPTED HIGHLANDERS .** Alexander Patrician1, Christopher Willie1, Ryan Hoiland2, Christopher Gasho3, Prajan Subedi3, James Anholm3, Michael Tymko1, Philip Ainslie1. 1Centre for Heart, Lung & Vascular Health, University of British Columbia Okanagan, 2Department of Anesthesiology, University of British Columbia, 3Pulmonary/Critical Care, Loma Linda University

Objective: Cerebral blood flow (CBF) increases during hypoxia to counteract the reduction in arterial oxygen content. The onset of tissue hypoxemia coincides with the stabilization of hypoxia-inducible factor (HIF) and transcription of downstream HIF-mediated processes. It has yet to be determined, whether HIF down- or upregulation can modulate hypoxic vasodilation of the cerebral vasculature. Therefore, we examined whether: 1) CBF would increase with iron depletion (via chelation) and decrease with repletion (via iron infusion) at high-altitude, and 2) explore whether genotypic advantages of highlanders extend to HIF-mediated regulation of CBF. Methods: In a double-blinded and block-randomized design, CBF was assessed in 82 healthy participants (38 lowlanders, 20 Sherpas and 24 Andeans), before and after the infusion of either: iron(III)-hydroxide sucrose, desferrioxamine or saline. Results: Across both lowlanders and highlanders, baseline iron levels contributed to the variability in cerebral hypoxic reactivity at high altitude ($R2=0.174$, $P<0.001$). At 5,050 m, CBF in lowlanders and Sherpa were unaltered by desferrioxamine or iron. At 4,300m, iron infusion led to 4±10% reduction in CBF (main effect of time $p=0.043$) in lowlanders and Andeans. Conclusion: Iron status may provide a novel, albeit subtle, influence on CBF that is potentially dependent on the severity and length-of-stay at high altitude. Funding: The 2016 UBC Mt Everest Expedition and the 2018 Global REACH expedition to Peru was funded as a whole, by a Canada Research Chair (CRC) and the Natural Sciences and Engineering Research Council (NSERC) Discovery Grant and the Canadian Foundation for Innovation to P.N.A. A.P., M.M.T., R.L.H., were supported by an NSERC Doctoral Grants.

**Time: 1824-1830**

**Poster #: 46 .TRANSIENT HYPOXIA-INDUCED DEOXYHEMOGLOBIN FORMATION SERVES AS AN MRI CONTRAST FOR PERFUSION IMAGING IN PATIENTS WITH STENO-OCCLUSIVE DISEASE.**

Ece Su Sayin1, 2, Vittorio Stumpo3, 4, Jacopo Bellomo3, 4, Julien Poublanc2, Marco Piccirelli3, 4, James Duffin1, Vepeson Wijeya2, Athina Pangalu 3, 4, Andrea Bink3, 4, Bence Nemeth3, 4, Zsolt Kulcsar3, 4, David Mikulis1, 2, Olivia Sobczyk2, Jorn Fierstra3, 4, Joseph Fisher1, 2. 1University of Toronto, 2University Health Network, 3University Hospital Zurich, 4University of Zurich

Background: Susceptibility agents are required to generate contrast for calculating resting perfusion measures (such as mean transit time, cerebral blood volume, and cerebral blood flow) using dynamic susceptibility contrast (DSC) MR perfusion. Currently this requires the intravascular injection of gadolinium (Gd), engendering medical risks, cost, along with image, and environmental drawbacks. Hypoxia-induced deoxyhemoglobin (dOHb) is intrinsic, reversibly paramagnetic, and relatively low-cost.Objective: Here we use hypoxia-induced dOHb as a suitable agent for DSC perfusion and validate against a clinical standard, Gd, in patients with steno-occlusive disease (SOD).Methods: We studied 10 patients between the ages of 39 and 74 (8 M) with known steno occlusive disease in a 3-Tesla scanner running-BOLD acquisition sequences. Transient hypoxia was induced via an automated gas blender running feed-forward gas algorithm targeting 2 consecutive reductions of pulmonary PO2 from 95 mmHg to 40 ± 3 mmHg followed by full reoxygenation within a single inhalation. A second BOLD sequence was acquired following an intravenous injection of 5 ml of Gd. All images were analyzed, and resting perfusion measures were calculated using a standard tracer kinetic model.Results: The calculated perfusion measures and their distribution showed similar voxel-wise proportional changes in BOLD signal throughout the brain. Bland-Altman analysis indicated little bias or difference in hemodynamic measures between methods.Conclusions: The resting perfusion measures obtained from brief transient hypoxia are spatially and quantitatively comparable to those obtained using Gd in the same patients with varying patterns of SOD. The main advantages of transient hypoxia as a contrast agent include it being non invasive; reduced risk of allergy, renal or fetal toxicity; no accumulation in organs, and no environmental damage, making it a suitable contrast for DSC perfusion imaging.Funding: Dr Joseph Fisher Critical Care Research Fund.