Indirect Measurements of Acceleration Atelectasis and the Role of Inspired Oxygen Concentrations

Ross D. Pollock; Sonny D. Gates; Jeremy J. Radcliffe; Alec T. Stevenson

BACKGROUND: A growing number of symptom reports suggestive of acceleration atelectasis in fast jet aircrew have raised the question as to whether traditional guidelines on inspired gas composition remain valid. The aim of this study was to assess the effects of inspired O_2 concentration on the development of acceleration atelectasis when wearing modern anti-G garments.

METHODS: There were 14 nonaircrew subjects who completed 5 centrifuge exposures to +5 G_z lasting 90 s. During exposures subjects breathed a gas mixture containing 21, 35, 45, 60, or 75% O_2. To assess the extent of atelectasis post-G_z, forced inspiratory vital capacity (FIVC), regional FIVC (EIT_{FIVC}), shunt, respiratory resistance, reactance, and compliance and peripheral O_2 saturation during a hypoxic exposure were measured.

RESULTS: Compared with baseline, FIVC was not statistically significantly altered. EIT_{FIVC} was 14.4% lower after the 75% O_2 exposure only with a greater symptom reporting with higher F_IO_2 in some individuals. A significantly greater shunt (~3–>6%) followed the 60 and 75% O_2 exposures. O_2 concentration during G_z had no effect on respiratory resistance, reactance, compliance, or hypoxemia.

DISCUSSION: There is evidence of mild acceleration atelectasis present when breathing 60% O_2, particularly in susceptible individuals, with 75% O_2 causing more obvious physiological compromise. An inspired oxygen concentration of <60% will prevent the majority of individuals from developing acceleration atelectasis.

KEYWORDS: aero-atelectasis, acceleration, fast jet, oxygen concentration.

Pollock RD, Gates SD, Radcliffe JJ, Stevenson AT. *Indirect measurements of acceleration atelectasis and the role of inspired oxygen concentrations*. Aerosp Med Hum Perform. 2021; 92(10):780–785.

Pilots flying highly agile aircraft are at risk of developing acceleration (aero-) atelectasis, a partial collapse of basal lung tissue during exposure to head to foot acceleration.[11,16] Acceleration promotes atelectasis when anti-G trousers (AGT) are inflated and, importantly, high inspired oxygen concentrations are breathed. Distortion of the lung under its own weight with exposure to +G_z (head to foot) acceleration and compression of basal lung regions from AGT inflation lead to airway closure. Subsequent absorption of oxygen from unventilated alveoli causes them to collapse and renders them atelectatic.[4] The importance of inspired oxygen concentration arises from its relationship to rate of gas absorption from the alveoli. Higher inspired oxygen concentration and, consequently, lower inspired nitrogen concentrations are associated with faster absorption.[4,8] Based on animal studies and human centrifuge experimentation, keeping the inspired oxygen concentration below 60% is believed to avoid meaningful acceleration atelectasis.[8,9,13] These findings have informed the design of some aircraft life support systems to limit the supplied inspired oxygen concentration at low equivalent pressure altitudes, thereby minimizing the development of acceleration atelectasis.

Acceleration atelectasis can result in a significant pulmonary shunt and the development of symptoms such as

From Human Performance, QinetiQ Ltd., Farnborough, United Kingdom; the National Hospital for Neurology & Neurosurgery, Queen Square, United Kingdom; and the Centre for Human and Applied Physiological Sciences, King's College London, United Kingdom.

This manuscript was received for review in January 2021. It was accepted for publication in July 2021.

Address correspondence to: Alec T. Stevenson, QinetiQ Ltd., Farnborough, United Kingdom; atstevenson@qinetiq.com.

DOI: https://doi.org/10.3357/AMHP.5859.2021

shortness of breath, chest tightness, and cough.[10] Anecdotal reports have suggested these symptoms are developing during some fast jet flight.[19] While excessive inspired oxygen concentrations delivered by onboard oxygen generation systems cannot be excluded as the cause,[3] the role of advancements in aircraft capabilities and anti-G systems should also be considered. In particular, the use of full coverage (FCAGT), as opposed to partial coverage (PCAGT) AGT, has become more common. FCAGT transmit pressure directly to the lower thorax[7] and may result in greater abdominal and basal lung compression than PCAGT. In addition, an extra 1–1.5 G_z protection is provided by FCAGT,[12] allowing higher G_z levels to be sustained without the need for pilots to perform the anti-G straining maneuver, which is known to mitigate against acceleration atelectasis.[23]

The influence of inspired oxygen concentration on the development of acceleration atelectasis has previously been investigated with subjects wearing PCAGT.[9,13,23] The aim of the present study was to investigate the influence of inspired oxygen concentration on the development of acceleration atelectasis when wearing FCAGT to determine whether limiting inspired oxygen concentrations to 60% still avoids the development of meaningful atelectasis. It was hypothesized that breathing gas mixtures containing over 60% oxygen during G_z exposures would be associated with evidence of atelectasis development.

METHODS

Subjects

Volunteering for the study were 15 subjects, 13 men and 2 women (mean ± SD: age, 27.6 ± 6.6 yr; body mass, 82.3 ± 13.9 kg; height, 1.77 ± 0.08 m). All were centrifuge trained up to +9 G_z. All subjects underwent medical screening, including 12-lead electrocardiography and echocardiography. Any individual with a history of cardiovascular disease, respiratory disease, current smoker, or over the age of 40 yr was excluded. Ethical approval for the study was obtained from the UK Ministry of Defense Research Ethics Committee (696/MoDREC/15). The study adhered to the principles of the Declaration of Helsinki. All subjects provided written informed consent.

Equipment

Several measurement techniques were used to assess the development of acceleration atelectasis. The primary measure was the participants' forced inspiratory vital capacity (FIVC), with a reduction of 0.5 L[14] typically regarded as indicating the development of acceleration atelectasis. Other measurements included lung impedance, respiratory resistance, reactance, and compliance in addition to pulmonary shunt estimation. The measurement techniques have previously been described in full.[21] A brief description of each is given below.

Lung volume. Expired volume was determined by integrating the flow signal recorded from a pneumotachograph (heated Fleisch pneumotachograph No. 2 connected to a differential pressure transducer; PSE550 series; SMC, Tokyo, Japan). Flow values were corrected to account for any changes in the viscosity of the breathing gases, which changed throughout the experiment by design.[2,24] FIVC was determined by calculating the change in volume during a maximum inspiration from residual volume with an FIVC reduction of 0.5 L assumed to be representative of the development of atelectasis.[14] Inspiratory capacity was calculated as the volume difference between the average end-expiratory level (over three normal tidal breaths) to that at full inspiration.

Electrical impedance tomography (EIT) was used to quantify basal lung volume (Sheffield Mk 3.5 EIT system, Maltron, Rainham, Essex, UK). Eight electrodes were placed equidistantly around the circumference of the thorax at the level of the xiphoid process while a reference electrode was placed over the right anterior superior iliac spine. Current (212 μA root mean square) was applied at 30 different frequencies (2 kHz to 1.6 MHz) to a pair of electrodes and measured at the remaining electrode locations before the 'drive' electrodes were switched to another pair, with the process continuing until all drive electrode combinations had been satisfied, yielding one data set (frame). Purpose built software (Matlab v6.1, The Mathworks Inc., Natick, MA, USA) acquired and processed the resulting data to image the lung at 25 Hz. Pixel intensities were subsequently represented as a percentage change in impedance from the value recorded at the subject's residual volume. The 20 pixels which showed the greatest variation in intensity at maximal inspiration were used to identify a region of interest, the average intensity of which was plotted, with respect to time, over the recording period. From this, each FIVC could be identified and regional FIVC (EIT_{FIVC}) estimated as a relative (%) change in impedance from residual volume to maximal inspiration recorded during the FIVC.

Respiratory compliance. Respiratory system compliance, resistance, and reactance were measured using the forced oscillatory technique (FOT).[17] A single frequency oscillation of 5 Hz eliciting a peak-to-peak pressure change of 2–2.5 mmHg at the mouth was superimposed onto the normal breathing cycle using an audio amplifier (Crown Amcron Macro Tech 1200 watts, Crown Audio, Los Angeles, CA, USA) and 12″ loudspeaker (3000 W, Space 12, Vibe Space, Sutton, UK) connected to the face mask via a 9-mm internal diameter rigid plastic tube. Respiratory resistance (Rrs), reactance (Xrs), and compliance (Crs) were determined using previously described methods.[1,21]

Shunt. Using a modified technique,[15,22] as described previously,[21] an estimate of pulmonary shunt was made by manipulating inspired oxygen concentration to achieve a wide range of alveolar O_2 concentrations. During this period peripheral arterial oxygen saturation (S_pO_2) was measured at the earlobe (Radical 7 pulse oximeter, Masimo Corporation, Irvine, CA, USA) while alveolar oxygen tensions were estimated from

$F_{et}O_2$ measured breath by breath by respiratory mass spectrometry (MSX671, Ferraris Respiratory, Middlesex, UK). The relationship between S_pO_2 and $F_{et}O_2$ data were then modeled using a biexponential of the form:

$$SpO_2 = a^* e^{b^* FetO_2} + c^* e^{d^* FetO_2}$$

Comparison of the modeled curves to those generated by an established mathematical model of gas exchange in which shunt can be manipulated[20] allowed the degree of pulmonary shunt (to the nearest percent) to be estimated. In addition, the minimum S_pO_2 ($minS_pO_2$) value recorded during the hypoxic period was determined.

On each day prior to testing, the time delay between the flow measurement and the respiratory gas measurement due to the ~15-m length of the mass spectrometer capillary tube was determined by delivering a bolus of known gas simultaneously to both measurement devices. In addition, the time delay associated with the measurement of S_pO_2 from the earlobe was corrected by identifying the delay from when the gas being breathed was switched from the hypoxic to hyperoxic gas.

Analog-to-digital conversion of all measures, with the exception of those derived by EIT, was performed using a PC-based data acquisition system (Powerlab 16SP, ADInstruments, Sydney, Australia) and recorded continuously on chart software (LabChart v7, ADInstruments).

Procedure

Each subject's testing was conducted on a single day on a human-rated long-arm (9.14-m radius) centrifuge (Farnborough, UK). During the experiment subjects were harnessed into an aircraft ejection seat reclined to 23° (10B-2 Mk1, Martin Baker Aircraft Company Ltd., Higher Denham, Middlesex, UK) and wore an aircrew coverall, inflatable sock bladders, flight jacket, FCAGT, and purpose built helmet and mask assembly. Under G_z acceleration FCAGT inflation began at +2 G_z and increased linearly at 10 kPa · G^{-1} (Aircrew Service Package, Honeywell Aerospace, Yeovil, UK). Supply of positive pressure breathing for G protection was disabled. Five discrete acceleration exposures were undertaken to a plateau at +5 G_z for 90 s each time using onset/offset rates of 1 G · s^{-1}. This acceleration exposure was chosen as previous research found this to be sufficient to elicit evidence of acceleration atelectasis in the majority of individuals when breathing 94% oxygen and wearing FCAGT.[21] During each exposure subjects breathed a gas mixture containing 21, 35, 45, 60, or 75% O_2 (balance nitrogen), with the order randomized for each individual. Subjects were instructed to maintain clear vision throughout using lower body muscle tensing only. As far as possible, unless required to do so by the study protocol, subjects were asked to avoid taking deep breaths or coughing during the test session.

An overview of the experimental procedures is given in **Fig. 1**. Identical measurements were made at baseline (prior to the first G_z exposure) and after each +G_z exposure. These began with subjects breathing a hyperoxic gas mix (94% O_2, balance N_2) for a period of 60 s (required for the shunt measurement) followed by a period breathing a hypoxic gas mix (14% O_2, balance N_2) until $F_{et}O_2$ stabilized (typically between 2 and 3 mins). The reduced inspired oxygen concentration breathing period was designed to produce a mild hypoxemia that would be more pronounced if gas exchange was impaired. Following $F_{et}O_2$ stabilization, sinusoidal pressure oscillations were applied to the oronasal mask for 30 s to allow respiratory compliance, reactance, and resistance to be measured using

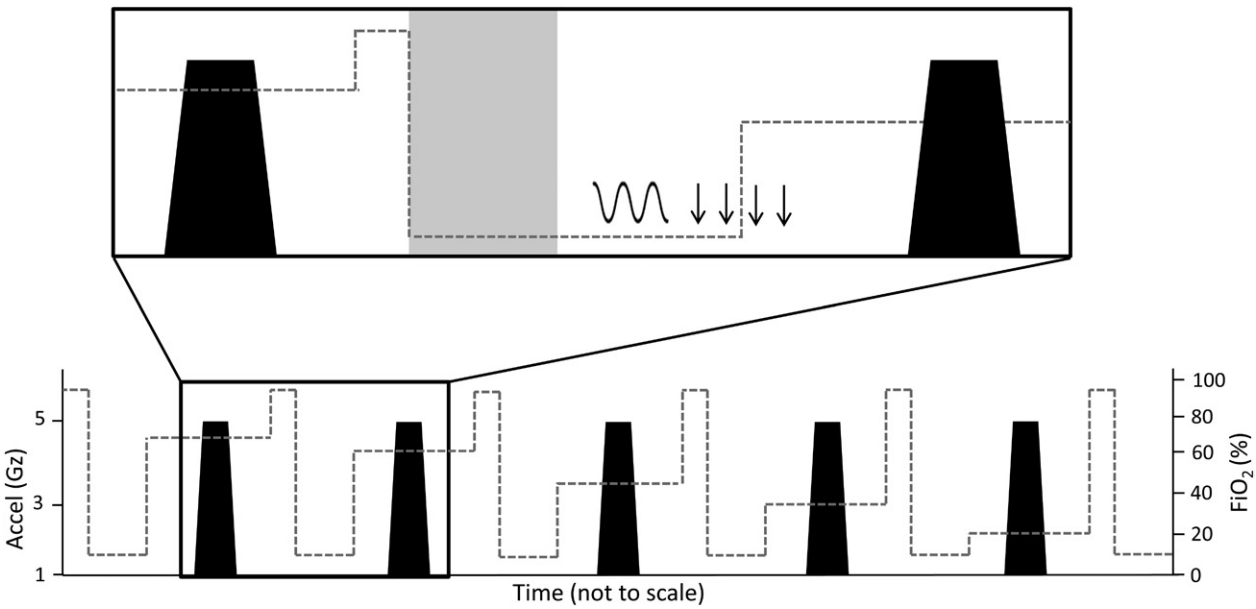

Fig. 1. Overview of the experimental protocol. Black bars indicate periods of G_z exposure while the gray dashed line indicates the inspired oxygen concentration (F_IO_2). The forced oscillatory technique (FOT) is performed during the period indicated by the sine wave while forced inspiratory vital capacity (FIVC) maneuvers are highlighted by arrows. The gray shaded area highlights the period during which pulmonary shunt is estimated. Identical measurements were made at baseline (immediately prior to the first G_z exposure) and after every G_z exposure.

the FOT. Following this, four FIVC maneuvers (separated by a minimum of 30 s) were performed, during which measures of regional lung volume were acquired by EIT. After completion of the second FIVC, the breathing gas was switched to that to be used during the following $+G_z$ exposure (21, 35, 45, 60, or 75% O_2, balance N_2). Any coughs that occurred after the FIVC maneuvers were noted. In addition, once all FIVCs were completed, subjects were asked whether they experienced an urge to cough or chest tightness/shortness of breath in the period between completion of the G_z exposure and the FIVC maneuvers. The $+G_z$ exposure commenced when FIVC measures were complete and a stable $F_{et}O_2$ concentration had been achieved. At the end of each $+G_z$ exposure, the subjects continued to breathe the gas mixture for 180 s to allow any G_z induced physiological disturbances not associated with atelectasis to resolve. The above processes were repeated for all inspired oxygen concentration conditions with approximately 10 mins between each G_z exposure.

Statistical Analysis

Statistical analysis was performed using IBM SPSS Statistics v22 (Chicago, IL, USA) with significance set at $P < 0.05$. The normality of the data was assessed with the Shapiro-Wilks test. A log transformation was performed on data that were not normally distributed (Rrs, Crs, and Xrs). The effect of inspired oxygen concentration on the dependent variable was assessed using one-way repeated measures ANOVA. If a significant main effect was established, planned contrasts against baseline values were performed with Bonferroni correction. Only the first FIVC performed post G_z was used for analysis. Unless otherwise stated data are mean (± SD).

RESULTS

Nausea after the second centrifuge run forced one subject to withdraw. Data obtained from this subject were excluded from analysis, therefore data are reported for 14 subjects. Experimenter error resulted in pulmonary shunt and S_pO_2 not being recorded from one subject following the 60% oxygen centrifuge exposure.

Respiratory symptoms associated with acceleration atelectasis were reported post G_z with a greater number typically reported with higher oxygen concentrations (**Table I**). The greatest number of reports of urge to cough or chest tightness/shortness of breath was given after the 60 and 75% oxygen centrifuge exposures (Table I). While 3 individuals reported an urge to cough after the 45% oxygen exposures, none described feeling chest tightness or shortness of breath. Overall the 21 and 35% oxygen exposures caused minimal respiratory symptoms. A spontaneous cough was noted in eight subjects upon completion of the FIVC maneuver after 75% oxygen exposure (Table I). This number reduced to three after the 60% exposure with one or zero subjects coughing in the 21–45% oxygen exposures.

Table I. Respiratory Symptoms Reported Following Exposure to 5 G_z for 90 s While Breathing Gas Mixtures Containing Between 21 and 75% O_2.

	F_IO_2 DURING PRIOR G_z EXPOSURE				
	21%	**35%**	**45%**	**60%**	**75%**
Urge to cough	0	0	3	4	3
Chest tightness / SOB	1	0	0	2	4
Cough after FIVC	0	1	1	3	8

Data are number of subjects reporting or displaying the symptom. F_IO_2, inspired oxygen concentration; SOB, shortness of breath; FIVC, forced inspiratory vital capacity.

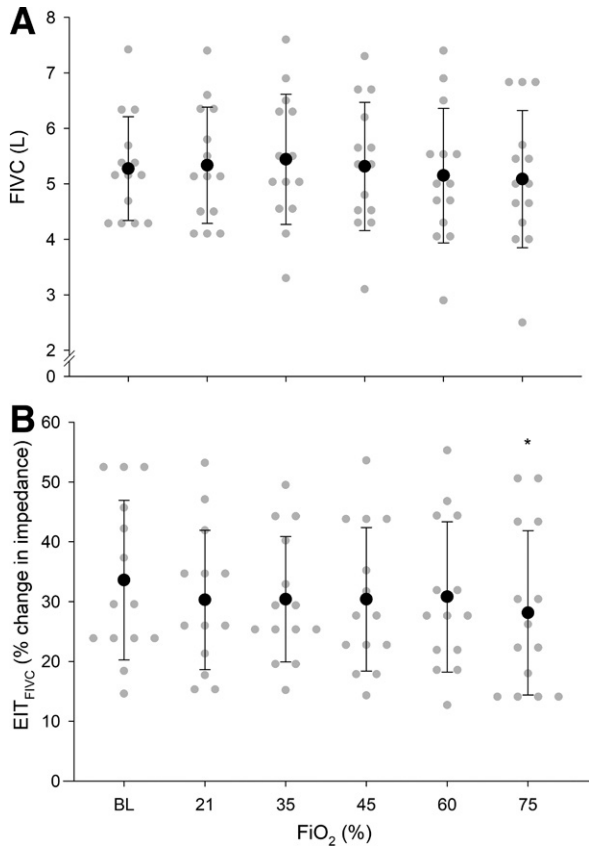

Fig. 2. Mean (± SD) forced inspiratory vital capacity (FIVC; panel A) and regional FIVC (panel B) at baseline (BL) and following centrifuge exposures while breathing a gas mixture with an inspired oxygen concentration (F_IO_2) of between 21 and 75%. Gray symbols represent individual subject data points; *significantly different from baseline ($P = 0.021$).

No main effect of inspired oxygen concentration on FIVC was present [$F(3.44, 44.26) = 2.14$, $P = 0.101$; **Fig. 2A**]. In contrast, a significant effect of inspired oxygen concentration on EIT_{FIVC} was found [$F(3.04, 39.56) = 3.05$, $P = 0.039$; **Fig. 2B**]. Post hoc analysis revealed that an oxygen concentration of 75% significantly reduced EIT_{FIVC} ($P = 0.021$). Inspired oxygen concentrations between 21 and 60% had no effect on EIT_{FIVC} ($P > 0.05$ in all cases).

There was a main effect of inspired oxygen concentrations on the degree of pulmonary shunt that developed [$F(3.47, 45.15) = 4.12$, $P = 0.009$]. Compared to baseline of just under 3%, pulmonary shunt was increased to more than 6% when breathing 60% and 75% oxygen ($P = 0.006$ in both cases; **Table II**). There was no difference in pulmonary shunt after 21, 35, and 45% oxygen exposures ($P > 0.05$ in all cases), but mean shunt values increased

Table II. Respiratory Function Related Parameters Recorded After Exposure to +5 G_z for 90 s Performed While Breathing Varying Oxygen Concentrations.

		F_IO_2 DURING PRIOR G_z EXPOSURE				
	BL	21%	35%	45%	60%	75%
$minS_pO_2$ (%)	90.8 (2.7)	90.8 (3.1)	90.2 (3.5)	90.3 (3.9)	90.4 (2.7)	90.2 (3.1)
Shunt (%)	2.9 (4.2)	4.6 (4.5)	5.1 (5.3)	5.6 (5.3)	6.3 (5.2)*	6.1 (5.3)*
Crs ($L \cdot kPa^{-1}$)	0.19 (0.12)	0.20 (0.07)	0.21 (0.12)	0.21 (0.12)	0.24 (0.15)	0.21 (0.11)
Xrs ($kPa \cdot s^{-1} \cdot L^{-1}$)	–0.23 (0.15)	–0.20 (0.12)	–0.18 (0.08)	–0.19 (0.12)	–0.18 (0.08)	–0.19 (0.08)
Rrs ($kPa \cdot s^{-1} \cdot L^{-1}$)	0.41 (0.15)	0.43 (0.15)	0.40 (0.08)	0.41 (0.15)	0.38 (0.15)	0.40 (0.08)

Data are mean (± SD). $minS_pO_2$ is the minimum O_2 saturation recorded during a hypoxic exposure post G_z, pulmonary shunt was estimated during the same period. The forced oscillatory technique was used to determine respiratory compliance (Crs), reactance (Xrs), and resistance (Rrs). *Significantly different to baseline (BL) measurement ($P < 0.05$). $N = 14$ except 60% $minS_pO_2$ and shunt where $N = 13$.

with increasing inspired oxygen concentrations. There was no effect of inspired oxygen concentration on the minimum S_pO_2 recorded during a period of hypoxia post G_z exposure [$F(4.26, 51.10) = 0.604$, $P = 0.672$; Table II]. Respiratory resistance, reactance, and compliance were unaffected by inspired oxygen concentration ($P > 0.05$ in all cases; Table II).

DISCUSSION

To our knowledge this is the first study to investigate the effect of inspired oxygen concentrations on the development of acceleration atelectasis wearing FCAGT. The use of a breathing gas containing oxygen concentrations of 75% resulted in the development of atelectasis as indicated by decrements in EIT_{FIVC} and shunt along with increased symptom reporting. In partial agreement with our hypothesis, an oxygen concentration of 60% resulted in atelectasis, but only as determined by shunt measures while symptoms were also reported. Overall, there was no evidence of atelectasis development with oxygen concentrations <60%.

Previous research has reported a significant reduction in FIVC of between ~4 and 10% when breathing 60 and 70% oxygen during exposure to $+G_z$ during simulated combat maneuvers and single exposures to 4 G_z;[9,13] this was not the case in the present study. Acceleration atelectasis is typically assessed by FIVC measurement,[13,23] but this has been shown to result in atelectasis going undetected.[13] Furthermore, there is a large interindividual variability in the development of acceleration atelectasis.[21] EIT_{FIVC} has been suggested to be more sensitive to atelectasis due to it imaging the specific region of the lung most likely to be affected by atelectasis rather than the entire lung.[21] The decline in EIT_{FIVC} with oxygen concentrations of 75% in this study is similar to that following 90-s exposures to +5 G_z while breathing 94% oxygen;[21] in contrast, FIVC was unaffected by oxygen concentrations of 75% in the current study, but was decreased in previous studies when breathing 94% oxygen.[21] This suggests that alveolar collapse at the base of the lung is similar with inspired oxygen concentrations of 75 and 94%, while the reduction in FIVC seen when breathing oxygen concentrations of 94% may include lung regions outside the EIT imaging plane. This is further supported by half the subjects experiencing a spontaneous cough after the run breathing 75% oxygen, yet no statistically significant reduction in FIVC being found. Imaging studies including computed tomography suggest some degree of acceleration atelectasis formation is possible with inspired oxygen concentrations as low as 44.5%.[6] While there were four reports of symptoms associated with atelectasis while breathing 45% oxygen, there was no clear evidence from the remaining measures, with the mild symptoms reported unlikely to have any detrimental effect on aircrew performance or safety.

An increased pulmonary shunt due to acceleration atelectasis could increase the severity of hypoxemia during a subsequent hypoxic insult (e.g., with hypobaric hypoxia or further $+G_z$ exposure). However, although the estimated pulmonary shunt was significantly increased from a baseline of 3% to over 6% with inspired oxygen concentrations of 60 and 75%, this did not affect susceptibility to subsequent hypoxic hypoxia, with similar S_pO_2 recorded during the post-$+G_z$ hypoxic periods. An increase in pulmonary shunt of 3% is likely to result in a reduction in S_pO_2 of ~0.9%;[20] the lack of significant reduction in the present study is likely related to a high level of interindividual variability in the development of atelectasis and the study not being powered to detect differences in S_pO_2.[21]

Compared to baseline there was no significant change in pulmonary shunt of EIT_{FIVC} after the exposure breathing 21% oxygen; however, from Fig. 2B and Table II it can be seen that both of these variables showed signs of a reduction. Although changes in these measures only reached statistical significance at higher inspired oxygen concentrations, these observations would be consistent with some residual effect of $+G_z$, independent of F_IO_2. It is possible that some degree of compressive atelectasis and/or impairment of surfactant function occurred that was not immediately resolved upon return to +1 G_z.[18] The greater shunt with inspired oxygen concentrations of 60 and 75% would then reflect the additional contribution of absorptional atelectasis.

The FOT is a novel method for the noninvasive determination of lung resistance, reactance, and compliance. Animal models have established the suitability of this technique for detecting changes in lung compliance and reactance as a result of atelectasis,[5] although in the present study no changes in these variables were detected when using a oscillatory frequency of 5 Hz. While there was no effect on these variables, it should be noted that lung compliance derived from esophageal pressure measurements is reduced by acceleration atelectasis.[9] The relative insensitivity of the technique and small extent of atelectasis developed may explain

the lack of any recordable effect in the current study and suggest it may not be a technique suitable for future studies of acceleration atelectasis.

A limitation of the present study is that only a discrete duration and level of G_z was investigated. An exposure of +5 G_z was regarded as the worst-case scenario for the development of acceleration atelectasis as higher accelerations would require performance of the anti-G straining maneuver, which has a protective effect on the development of atelectasis.[23] Future studies should consider the effects of cumulative exposures to G_z acceleration and the effect of varying G levels with an aim to investigate more operationally relevant $+G_z$ profiles. Due to the need to allow any $+G_z$ induced physiological disturbances not associated with atelectasis to resolve, the first measurement of atelectasis (pulmonary shunt) was made approximately 3 min post-G_z exposure with FIVCs not measured until approximately 7 min post-exposure (the last measurements to be made as they also act to reverse atelectasis). This delay may be sufficient for some atelectasis to spontaneously resolve and as such the current findings may underestimate the true extent of its development.

Overall, the present study supports the use of breathing gas mixtures of 60% oxygen or less (and hence greater than 40% nitrogen) to prevent acceleration atelectasis in most individuals. With increasing oxygen concentration above 60%, a greater incidence of acceleration atelectasis will occur during acceleration exposure while wearing FCAGT. Further research is required to determine whether the current findings remain true for varying levels and cumulative exposures to $+G_z$.

ACKNOWLEDGMENTS

This work was performed as part of the Aircrew Systems Research program, which was funded by the Defence Science and Technology Laboratory (DSTL). The authors would like to thank all the subjects who volunteered for the study and the engineering staff at the Farnborough centrifuge facility for their technical assistance. We are grateful to the QinetiQ physicians for providing medical supervision.

Financial Disclosure Statement: The authors declare they have no conflicts of interest.

Authors and Affiliations: Ross D. Pollock, M.Sc, Ph.D., Sonny D. Gates, B.Sc., Jeremy J. Radcliffe, M.D., and Alec T. Stevenson, B.Sc., Ph.D., Human Performance, QinetiQ Ltd., Farnborough, United Kingdom; Jeremy Radcliffe, National Hospital for Neurology and Neurosurgery, Queen Square, United Kingdom; and Ross Pollock, Centre for Human and Applied Physiological Sciences, Kings College London, United Kingdom.

REFERENCES

1. Bates JHT, Irvin CG, Farré R, Hantos Z. Oscillation mechanics of the respiratory system. Compr Physiol. 2011; 1(3):1233–1272.
2. Blumenfeld W, Turney S, Cowlet RA. Mathematical model for flow in the heated Fleisch pneumotachometer. Med Biol Eng. 1973; 11(5):546–551.
3. Borges JB, Hedenstierna G, Bergman JS, Amato MBP, Avenel J, Montmerle-Borgdorff S. First-time imaging of effects of inspired oxygen concentration on regional lung volumes and breathing pattern during hypergravity. Eur J Appl Physiol. 2015; 115(2):353–363.
4. Dale WA, Rahn H. Rate of gas absorption during atelectasis. Am J Physiol. 1952; 170(3):606–613.
5. Dellaca RL, Andersson Olerud M, Zannin E, Kostic P, Pompilio PP, et al. Lung recruitment assessed by total respiratory system input reactance. Intensive Care Med. 2009; 35(12):2164–2172.
6. Dussault C, Gontier E, Verret C, Soret M, Boussuges A, et al. Hyperoxia and hypergravity are independent risk factors of atelectasis in healthy sitting humans: a pulmonary ultrasound and SPECT/CT study. J Appl Physiol. 2016; 121(1):66–77.
7. Eiken O, Bergsten E, Grönkvist M. G-protection mechanisms afforded by the anti-G suit abdominal bladder with and without pressure breathing. Aviat Space Environ Med. 2011; 82(10):972–977.
8. Ernsting J. Influence of alveolar nitrogen concentration and environmental pressure upon the rate of gas absorption from non-ventilated lung. Aerosp Med. 1965; 36(10):948–955.
9. Green I. Synopsis of recent work done on the problem of pulmonary atelectasis associated with breathing 100 percent O2 and increased positive "g." Farnborough (UK): RAF Institute of Aviation Medicine; 1963. Report No.: 230.
10. Green ID. The degree of pulmonary arterial to venous shunt produced by breathing 100% oxygen during increased positive acceleration and whilst wearing an anti-g suit. Proceedings of the Physiological Society. J Physiol. 1963; 169(Suppl.):96–97.
11. Green ID, Burgess BF. An investigation into the major factors contributing to post flight chest pain in fighter pilots. Farnborough (UK): RAF Institute of Aviation Medicine; 1962. Report No: 1182.
12. Green NCh. Effects of and protection against long duration acceleration. In: Rainford DJ, Gradwell DP, editors. Ernsting's aviation medicine, 5th ed. Oxford (UK): Butherworld Heinman; 2016.
13. Haswell MS, Tacker WA Jr, Balldin UI, Burton RR. Influence of inspired oxygen concentration on acceleration atelectasis. Aviat Space Environ Med. 1986; 57(5):432–437.
14. Hyde AS, Pines J, Saito I. Atelectasis following acceleration: a study of causality. Aerosp Med. 1963; 34(2):150–157.
15. Kjaergaard S, Rees S, Malczynski J, Nielsen JA, Thorgaard P, et al. Non-invasive estimation of shunt and ventilation-perfusion mismatch. Intensive Care Med. 2003; 29(5):727–734.
16. Levy P, Jaeger E, Stone R, Doudna C. Clinical problems in aviation medicine: aeroatelectasis: a respiratory syndrome in aviators. Aerosp Med. 1962; 33(8):938–944.
17. MacLeod D, Birch M. Respiratory input impedance measurment: forced oscillation methods. Med Biol Eng Comput. 2001; 39(5):505–516.
18. Magnusson L, Spahn DR. New concepts of atelectasis during general anaesthesia. Br J Anaesth. 2003; 91(1):61–72.
19. Monberg R. Acceleration atelectasis - an old problem in a new setting [Abstract]. Aviat Space Environ Med. 2013; 84(4):427.
20. Olzowka AJ, Wagner PD. Numerical analysis of gas exchange. In: West JB, editor. Pulmonary gas exchange. New York: Academic Press; 1980:263–306.
21. Pollock RD, Gates SD, Storey JA, Radcliffe JJ, Stevenson AT. Indices of acceleration atelectasis and the effect of hypergravity duration on its development. Exp Physiol. 2020. [Accessed 24 September 2020]. Available from https://onlinelibrary.wiley.com/doi/abs/10.1113/EP088495.
22. Sapsford DJ, Jones JG. The PIO2 vs. SpO2 diagram: a non-invasive measure of pulmonary oxygen exchange. Eur J Anaesthesiol. 1995; 12(4):375–386.
23. Tacker WA Jr, Balladin UI, Burton RR, Glaister DH, Gillingham KK, Mercer JR. Induction and prevention of acceleration atelectasis. Aviat Space Environ Med. 1987; 58(1):69–75.
24. Turney SZ, Blumenfeld W. Heated Fleisch pneumotachometer: a calibration procedure. J Appl Physiol. 1973; 34(1):117–121.