Cabin Pressure Altitude Effect on Acceleration Atelectasis After Agile Flight Breathing 60% Oxygen

Henry Tank; Gareth Kennedy; Ross Pollock; Peter Hodkinson; Rebecca-Anne Sheppard-Hickey; Jeffrey Woolford; Nicholas D. C. Green; Alec Stevenson

INTRODUCTION: A flight trial was conducted to determine whether breathing 60% oxygen during high performance flight maneuvers using contemporary pilot flight equipment induces atelectasis and to explore whether cabin altitude had any influence on the extent of atelectasis identified.

METHODS: On 2 separate days, 14 male aircrew flew as passengers at High [14,500–18,000 ft (4420–5486 m)] and Low [4000–6000 ft (1219–1829 m)] cabin pressure altitude in a Hawk T Mk1 aircraft breathing 60% oxygen. Sorties comprised 16 maneuvers at +5 G_z, each sustained for 30 s. Lung volumes (spirometry), basal lung volume (electrical impedance tomography, EIT), and peripheral oxygen saturation during transition from hyperoxia to hypoxia (pulmonary shunt fraction) were measured in the cockpit immediately before (Pre) and after (Post) flight.

RESULTS: Forced inspiratory vital capacity (FIVC) was significantly lower Postflight after High (−0.24 L) and Low (−0.38 L) sorties, but recovered to Preflight values by the fourth repeat (FIVC4). EIT-derived measures of FIVC decreased after High (−3.3%) and Low (−4.4%) sorties but did not recover to baseline by FIVC4. FIVC reductions were attributable to decreased inspiratory capacity. S_pO_2 was lower Postflight than Preflight in High and Low sorties.

DISCUSSION: Breathing 60% oxygen during flight results in a 3.8–4.9% reduction in lung volume associated with a small decrease in blood oxygenation and an estimated pulmonary shunt of up to 5.7%. EIT measures suggest persisting airway closure despite repeated FIVC maneuvers. There was no meaningful influence of cabin pressure altitude. The operational consequence of the observed changes is likely to be small.

KEYWORDS: atelectasis, forced inspiratory vital capacity, acceleration, altitude.

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Pulmonary acceleration atelectasis may account for some currently unexplained symptomatic in-flight physiological events.[7] In the United Kingdom, aircrew questionnaires have suggested that acceleration atelectasis has occurred during sorties in the Hawk T2 fast jet trainer.[20] The main factors associated with the development of acceleration atelectasis are sustained head-to-foot (+G_z) acceleration, anti-G trouser inflation, and high inspired oxygen concentrations (hyperoxia).[8,17] Common manifestations include postflight urge to cough, paroxysmal coughing, shortness of breath, chest tightness, and substernal discomfort on inspiration.[4,8,25] This is associated with marked attenuation of postflight vital capacity (VC)[16,17,25] by up to 60%.[24] The attenuation in VC is often reversed by deep breaths and, to provide a surrogate for the degree of atelectasis present, an inspiratory measure, rather than the more usual expiratory measure of VC, is typically used. This minimizes any atelectasis clearance prior to the VC measure. Forced inspiratory vital capacity (FIVC) is usually seen to revert to normal with repetition of the maneuver or following coughing or deep breathing. Research on the centrifuge has also demonstrated that a

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significant pulmonary shunt can develop,[10] a phenomenon known to be associated with atelectasis.[14]

The mechanism underlying development of acceleration atelectasis is understood to be absorption of gas trapped in the alveolar space when distal airways become occluded.[13,18,24] Exposure to $+G_z$ exaggerates intrapleural pressure gradients,[8] while anti-G trouser inflation splints the diaphragm and limits caudal expansion of the lung bases.[13] If alveolar gas contains a high concentration of oxygen that is rapidly absorbed once the airways close, the residual gas pressure can become inadequate to prevent alveolar collapse. It is generally accepted that retaining at least 40% inert gas (i.e., nitrogen) in the breathing supply, equivalent in practice to limiting the fractional inspired oxygen concentration (F_IO_2) to a maximum of 60%, prevents the development of meaningful atelectasis.[6] Previous research[15] exposing centrifuge subjects to a 4.5-min simulated air combat maneuver (SACM) consisting of +4.5 G_z peaks interspersed with +3 G_z nadirs monitored FIVC pre- and post-centrifuge undertaken with F_IO_2 varying from 21–100%. Postexposure FIVC, reported as a percentage of pre-exposure values, was decreased by 11%, 18%, 24%, and 26% with an F_IO_2 of 70%, 82.5%, 95%, and 100%, respectively, while no reduction was seen with an F_IO_2 of 50%. Based on a linear regression between F_IO_2 and the reductions in FIVC reported, a 5% fall in FIVC might be expected with an F_IO_2 of 60%.

There is a limited range of possible reasons why acceleration atelectasis reported in flight might result in a greater decrease in FIVC. The F_IO_2 could be higher than expected or, alternatively, the G profiles being flown may be more conducive to atelectasis formation in some aircraft types. Alternatively, evolving aircraft capabilities and newer designs of anti-G protection systems might predispose to the development of acceleration atelectasis, such as the use of full coverage anti-G trousers (FCAGT) with more efficient abdominal compression and splinting of the diaphragm. The improved G protection afforded by these garments[22] also means that pilots can sustain high levels of $+G_z$ acceleration for longer, potentially without recourse to the anti-G straining maneuver (AGSM), which is known to be effective at inhibiting atelectasis formation.[24]

It is possible that other features of the flight environment may affect atelectasis, such as the reduced ambient pressure.[3] Ernsting[5] reported the kinetics of trapped gas absorption in the dog lung, which followed a two-phased profile. Initially, absorption of oxygen from alveoli distal to closed airways was rapid, determined principally by gas solubility (independent of the presence of nitrogen); thereafter, the rate of absorption was proportional to regional blood flow and became slower with higher nitrogen concentrations. At altitude, the rate of the first phase was more rapid, while the second phase was slower. Ernsting concluded that the overall effect of altitude was to slow the rate of alveolar collapse and thereby impede atelectasis formation.

The aims of the current study were to determine whether breathing 60% oxygen (balance 40% nitrogen) during high performance flight maneuvers induces atelectasis and to explore whether cabin altitude had any influence on the extent of atelectasis identified. Measures were taken before and after (but not during) flight. The presence (and extent) of atelectasis was inferred from changes in FIVC and indices derived from electrical impedance tomography (EIT), which provides a surrogate measure of regional (basal) lung volume. Blood oxygen saturation and a derived estimate of pulmonary shunt were also recorded. The null hypotheses were that breathing 60% oxygen would prevent atelectasis development and there would be no influence of cabin altitude.

METHODS

Subjects

Recruited were 14 healthy male aircrew. All aircrew held a current flight medical and had previous fighter aircraft experience on platforms such as Alpha Jet, Hawk, Talon, Tornado, Tutor, and Typhoon. The study protocol was approved by the UK Ministry of Defense Research Ethics Committee and adhered to the principles outlined in the Declaration of Helsinki. Informed written consent was provided by all participants.

Equipment

Flights were conducted in a modified Hawk T Mk1 aircraft flying from Boscombe Down, Wiltshire, United Kingdom. For this trial the participant sat in the rear seat while the front seat was occupied by a safety pilot who handled the aircraft during maneuvers. Typhoon aircrew equipment assemblies (AEA) were worn by both the participant and safety pilot, and included a flying coverall, FCAGT, flight jacket (incorporating chest counterpressure; CCP), aircrew boots, an Mk 10 helmet, and P/Q oxygen mask. FCAGT pressurization, pressure breathing for G protection (PBG), and CCP inflation were provided from a Typhoon breathing and anti-G regulator (aircrew services package; ASP). FCAGT pressurization commenced at +2 G_z (±0.3 G), increasing by 10 kPa · G^{-1}. PBG began at +4 G_z, increasing at 1.6 kPa · G^{-1} with the CCP inflated to an equivalent pressure (±1.3 kPa). All AEA was fitted by a qualified Survival Equipment Technician on the first day of testing and proper fit was verified prior to each trial flight.

Protocol

Each participant underwent two flights on separate days. The flight sorties were, in so far as practicable, identical, only differing in the cabin altitude (ALT) to which the participant and safety pilot were exposed. The low altitude sortie (Low) was flown at flight altitudes where the target cabin altitude remained between 4000–6000 ft (1219–1829 m) pressure altitude (PA) (609–656 mmHg). The high-altitude sortie (High) was flown at flight altitudes where the target cabin altitude remained between 15,000–18,000 ft PA (4420–5486 m; 380–429 mmHg). The upper limit of 18,000 ft PA was selected to minimize risk of decompression sickness and the range represents the highest likely cabin altitudes during dynamic maneuvering in a high-performance fighter. Due to aircraft performance limitations with the Hawk T Mk1, the high-altitude sortie (High) was

undertaken with the cockpit depressurized so that a higher cabin altitude could be achieved. This allowed the G profiles to be flown at a lower flight altitude than would otherwise be necessary to achieve the required 18,000-ft PA cabin altitude [the cabin altitude remained 1000–2000 ft (305–610 m) lower than the aircraft altitude due to the effects of aircraft speed on cabin ram air flow]. To achieve and sustain the acceleration profiles for the High sorties, the safety pilot had to perform the turn in a descending spiral, starting at a flight altitude of around 20,000 ft PA (6096 m; 349 mmHg). Aircraft altitude for maneuvers in the Low sorties where the cabin was pressurized were around 6000 ft (609 mmHg). Cabin pressure data were not recorded in flight due to limitations with instrumentation, but cabin altitude was confirmed using aircraft instrumentation and recorded manually on the crew's kneeboard.

Performance limitations of the Hawk T1 placed greater constraints on the High sortie and risked limiting the number of G exposures that could be completed. Accordingly, the High sorties were flown first by all participants so that their total $+G_z$ exposure could always be replicated accurately during their subsequent Low sortie. Matching $+G_z$ exposures between sorties was prioritized over the possibility of introducing an order effect.

Preflight measurements were undertaken immediately prior to donning a flight helmet and again 15 min before takeoff. Soon after takeoff, a series of G exposures were performed as a subject 'warm-up' and to confirm operability of the anti-G system. This comprised a rapid onset rate turn to +4 G_z for 15 s followed by +6 G_z for the same duration. The trial maneuvers began 10–15 min after takeoff and within 30 min of preflight measurements. Trial maneuvers consisted of 16 repeats at +5 G_z maintained at this acceleration level for approximately 30 s, each attained using rapid acceleration onset rates (>6 $G \cdot s^{-1}$). The G profiles were separated by approximately 90 s of level flight. In addition, participants were instructed to avoid using the AGSM during the test G profiles if possible and apply lower body muscle tensing only to augment their G protection as required. Throughout all flights, participants breathed a 60% oxygen (balance nitrogen) gas mix, supplied from a series of compressed gas cylinders via the Typhoon ASP. After landing, the aircraft taxied to the apron, powered down, and was towed into the hangar where the measurements made pre-exposure were repeated. The time between landing and the performance of the postflight measurements was ~15 min and from the last $+G_z$ maneuver around 30 min.

Pre- and Postflight Measurements

Before (Pre) and after each flight (Post) a series of measurements were made in the aircraft hangar. For these measures the aircrew were fully clothed in their AEA and remained seated and harnessed in the ejection seat of the aircraft. Tests were undertaken using a standard respiratory mask (Hans Rudolph Inc., Shawnee, KS, USA) to which a pneumotachograph (Fleisch type, No. 2) and differential pressure transducer (Celesco low cost variable reluctance, 0–2 cm H_2O; Celesco Transducer Products, Inc., Chatsworth, CA, USA) had been fitted. Inspiratory and expiratory valves were housed in a machined plastic t-piece fitted upstream of the pneumotachograph. The mask inspiratory hose was connected to a set of manual valves which selected the breathing gas: either air, 100% oxygen, or a 14% oxygen (balance nitrogen) gas mix. The latter two were bottled gases supplied to the participant via an independent pressure demand regulator. The flowmeter was calibrated across a range of flows before testing began and immediately after the measurements had been performed using a calibration syringe. Following Pre measurements, participants immediately donned their oronasal P/Q oxygen mask and flight helmet and start-up procedures were commenced. Upon return to the hangar postexposure, participants again donned the test mask as soon as their oronasal P/Q oxygen mask and flight helmet had been removed.

While breathing air through the test mask, the participant was instructed to expire to their normal end-expiratory position and to indicate when this was reached. An experimenter then selected 100% oxygen, which the participant breathed until the expired nitrogen concentration fell below 1%. The participant was then supplied with the hypoxic gas mix (14% oxygen, balance nitrogen); when a stable end-tidal oxygen concentration was achieved, they completed four FIVC maneuvers (i.e., FIVC1, FIVC2, etc.) separated by periods of tidal breathing (around 45 s). Each FIVC comprised forceful emptying of the lungs followed by a maximal inspiration in accordance with current guidelines.[9]

Inspired and expired partial pressures of nitrogen, oxygen, carbon dioxide, and argon were initially measured using a respiratory mass spectrometer (MSX-671, Ferraris-Respiratory Europe Ltd., Hertfordshire, UK). However, a technical fault meant that in eight subjects, measurements of partial pressures of oxygen and carbon dioxide were made using a laser gas analyzer (O_2Cap, Oxigraf Inc., Sunnyvale, CA, USA). Peripheral arterial oxygen saturation (S_pO_2) was determined using a pulse oximeter (Model 3900, Datex-Ohmeda, Madison, WI, USA) fitted to the ear lobe. Ambient temperature, pressure, and humidity were recorded daily (WMR86A Backyard Pro Wireless Weather Station, Oregon Scientific, High Wycombe, UK). A further temperature measurement was made within the mask housing using a thermocouple (T-type, AD Instruments, Dunedin, New Zealand). All data were recorded using an analog-to-digital converter and PC-based acquisition system (PowerLab 16/30, AD Instruments, Dunedin, New Zealand).

In addition, changes in a surrogate measure of regional lung volume were investigated using EIT. This technique exploits the principle that with increasing air volume the lung parenchyma present greater resistance to the flow of an electrical current. By applying an imperceptible alternating current between successive pairs of electrodes and measuring the resultant voltage distribution circumferentially around the chest, cross-sectional imagery of thoracic impedance can be generated. At high temporal resolution changes in impedance with the breathing cycle can be visualized and quantified, for the whole section or a region of interest, and may be particularly sensitive to changes affecting basal lung regions. For this study

a Pulmovista®500 device (Dräger, Lübeck, Germany) was used with a 16-electrode belt fitted at the fifth intercostal space. Adhesive electrode gel (Tensive, Parker Labs, Fairfield, NJ, USA) was applied to the dry electrodes to improve skin contact and reduce movement of the belt; a form fit foam pad was used to ensure the electrodes across the back retained good contact with the skin, especially along the anatomical indentation formed by the thoracic spinous processes.

Data Analysis

Inspired and expired volumes were derived by integration of the inspired and expired flow, respectively. Tidal volume (V_T) and FIVC were identified using a cyclic peak/nadir detection algorithm available in the data analysis software. Respiratory rate (RR) and V_T were averaged over a 2-min period 30 s prior to commencement of the first FIVC while breathing the hypoxic (14% O_2) gas mix. Inspiratory capacity (IC) and expiratory reserve volume (ERV) were computed as the difference in the mean end-tidal volume from 3–5 tidal breaths prior to each FIVC and the minimum and maximum volumes during the expiratory and inspiratory phases of the FIVC, respectively. Reported volumes are all corrected to Body Temperature and Pressure, Saturated conditions (BTPS). For measurements made by EIT, equivalent impedance measures of FIVC ($FIVC_{EIT}$) were derived. All impedance changes were referenced to the minimum impedance recorded during the FIVC maneuvers and, therefore, represent increases from residual volume. The reported end-tidal partial pressures of oxygen ($P_{ET}O_2$) and carbon dioxide ($P_{ET}CO_2$) are those recorded during the expiratory phase of the FIVC maneuver, presented as the average across all four repeats. Mean S_pO_2 were extracted over a 2-min period while breathing air immediately after donning the test mask ($S_pO_{2\ normoxia}$), during the final period breathing 100% oxygen ($S_pO_{2\ hyperoxia}$), and 30 s before commencing the first FIVC maneuver while breathing the hypoxic gas mix ($S_pO_{2\ hypoxia}$). Pulmonary shunt (to the nearest percent) was estimated using techniques described elsewhere;[19] briefly, the relationship between the expired fractional end-tidal oxygen concentration ($F_{ET}O_2$) and S_pO_2 during the transition from hyperoxia to hypoxia was compared with standard curves generated from an established model of gas exchange with varying shunt fractions.[21]

Statistical Analysis

The principal comparative measure used to determine sample size was the measurement of FIVC. This is the only metric where previous data of the effect of atelectasis are available. We considered an effect size of greater than a Cohen's *d* of 1 as an important effect (i.e., the difference in FIVC with atelectasis should be larger than the between subject variation in FIVC normally observed). FIVC is approximately 5 L in adults with a standard deviation of 0.6 L. Given that field measurements of FIVC are likely to demonstrate a greater variability than those performed in the laboratory, based on a power of 80%, alpha of 0.05, a correlation between repeated measurements of 0.70 (determined from previous measurements of FIVC using the nitrogen washout technique by one of the experimenters), and an SD of 0.75 L to detect a meaningful pre-post flight difference (i.e., Cohen's $d = 1$), 10 participants were required. In order to account for participant dropout, 14 participants were recruited. Datasets were assessed for a normal distribution using the Shapiro-Wilks test. First, to guide subsequent analysis, the baseline (Pre) FIVC data (FIVC#) before the two sorties (Day) were compared using repeated measures analysis of variance (rmANOVA), interrogating main effects of Day vs. FIVC# vs. Subject. Subsequently, the differences between corresponding FIVC measurements (delta Pre-Post) across the two altitude (ALT) conditions were evaluated using rmANOVA for main effects of ALT vs. Subject vs. FIVC#. Specific post hoc pairwise comparisons to explore changes in FIVC# were conducted using paired *t*-tests. The outcomes of FIVC analyses guided parallel analyses of IC and ERV. EIT measures were analyzed within each ALT condition, as it was not possible to precisely replicate electrode placement between the two flights. EIT data were analyzed for main effects of Subject vs. FIVC#. Data for V_T, RR, and S_pO_2 were subject to rmANOVA to assess main effects of $+G_z$ (Pre/Post) vs. ALT (High/Low) vs. Subject. Specific post hoc comparisons employed paired *t*-tests and Wilcoxon signed rank tests for nonparametric data. For data presentation mean and 95% confidence interval after correction for between subject variability[1] are shown. IBM SPSS Statistics v.22 (Chicago, IL, USA) was used for the statistical analyses with significance for main effects of rmANOVA set at $P < 0.05$.

RESULTS

Demographic information on the 14 male study participants is shown in **Table I**. Cabin altitude at the start of maneuvering in the High condition was 18,000 ± 50 ft (5486 ± 1.5 m) PA and mean finishing altitude was 14,430 ± 1265 ft (4398 ± 386 m) PA. In the Low condition, the cabin altitude achieved was 4300 ± 800 ft (1311 ± 244 m) PA. The mean G_z level reached across the acceleration exposures successfully registered by the flight

Table I. Demographic Data of 14 Male Participants.

SUBJECT	AGE (yr)	HEIGHT (cm)	WEIGHT (kg)	TOTAL FLYING HOURS (h)
1	48	175	75	1471
2	34	178	89	2000
3	50	173	85	6500
4	55	176	88	3850
5	44	171	66	3200
6	37	182	65	2050
7	46	173	68	4365
8	23	174	80	180
9	23	182	77	450
10	41	193	80	3000
11	28	173	65	210
12	40	190	90	3000
13	35	180	81	2800
14	38	173	80	3500
Mean	39	178	78	2613
SD	10	6	9	1891

Table II. Absolute and Differential (Δ) Data for Repeated Measures Taken Pre- and Postflight at High and Low Altitude.

		High			Low		
		Pre	Post		Pre	Post	
VARIABLE		CTRL	$FIVC_1$	$FIVC_4$	CTRL	$FIVC_1$	$FIVC_4$
FIVC	Absolute	4.87 ± 0.18	4.64 ± 0.33*	4.80 ± 0.27	5.08 ± 0.21	4.70 ± 0.25*	4.97 ± 0.31
(L BTPS)	Δ CTRL	–	−0.24 ± 0.18*	−0.07 ± 0.36	–	−0.38 ± 0.37*	−0.10 ± 0.35
IC	Absolute	3.57 ± 0.25	3.35 ± 0.20*	3.62 ± 0.28	3.81 ± 0.20	3.37 ± 0.34*	3.67 ± 1.16
(L BTPS)	Δ CTRL	–	−0.22 ± 0.31*	0.05 ± 0.31	–	−0.44 ± 0.4*	−0.14 ± 0.34
ERV	Absolute	1.34 ± 0.14	1.37 ± 0.14	1.33 ±0.31	1.32 ± 0.17	1.38 ± 0.25	1.35 ± 0.30
(L BTPS)	Δ CTRL	–	0.03 ± 0.19	−0.01 ± 0.33	–	0.06 ± 0.32	0.02 ± 0.29
$FIVC_{EIT}$	Absolute	21.1 ± 7.5	17.8 ± 7.9*	19.2 ± 7.7*	27.7 ± 11.4	23.3 ± 13.1*	25.2 ± 12.8*
(Impedance %)	Δ CTRL	–	−3.3 ± 2.6*	−1.9 ± 2.6*	–	−4.4 ± 4.1*	−2.4 ± 3.3*

CTRL data are the means of four baseline Preflight FIVCs; Δ CTRL represent changes from this mean during Postflight FIVC 1 and FIVC 4. Data (N = 14) are mean ± the 95% confidence interval after correction for between-subject variability.
*Denotes statistical significance on post hoc paired t-tests ($P \leq 0.05$).

recorder (N = 175) was +4.8 ± 0.1 G_z. The mean duration of the +G_z exposures was 33 ± 6 s. Mean G levels (Z = 1.96, P = 0.14) were not significantly different between the High and Low conditions; G durations were longer in the High than Low conditions (Z = 1.96, P < 0.001) by a short interval (35 vs. 32 s).

Lung volume and EIT data are summarized in **Table II**. For ease of comparison, group FIVC data are also shown in **Fig. 1**, including all four repeats performed before (Pre) and after (Post) each flight for both the High and Low cabin altitude conditions. The graph indicates that FIVC was decreased postflight on both days, but that measures tended generally to be higher, including preflight baseline data, on the day of the lower cabin altitude sortie. The rmANOVA examining baseline (Pre) FIVC data for the 2 d confirmed a significant difference between baseline FIVC values for the High and Low cabin altitude conditions (5.1 ± 0.65 vs. 4.9 ± 0.64, P = 0.036). Thus, rmANOVA to assess the influence of cabin altitude on FIVC was conducted using the differential values for corresponding pre- and postflight FIVCs, indicating a main effect of FIVC# (P = 0.048), but no effect of cabin altitude (P = 0.25). Post hoc paired comparisons highlighted that the first FIVC performed following the sortie was lower than mean baseline FIVC for both the High (P = 0.039) and Low cabin altitude conditions (P = 0.002), confirming an effect of flight to decrease FIVC. This recovered fully by FIVC4 such that, for both High and Low cabin altitudes, lung volume at FIVC4 was not significantly different from baseline (Table II). The changes in FIVC were due to a fall in IC, which was decreased from baseline on FIVC1 in both the High (P = 0.02) and Low (P = 0.002) cabin altitude conditions, but had recovered by FIVC4. In contrast, ERV was unaffected (see Table II). Changes in $FIVC_{EIT}$ were similar, with a decrease from baseline seen postflight for both the High (P = 0.003) and

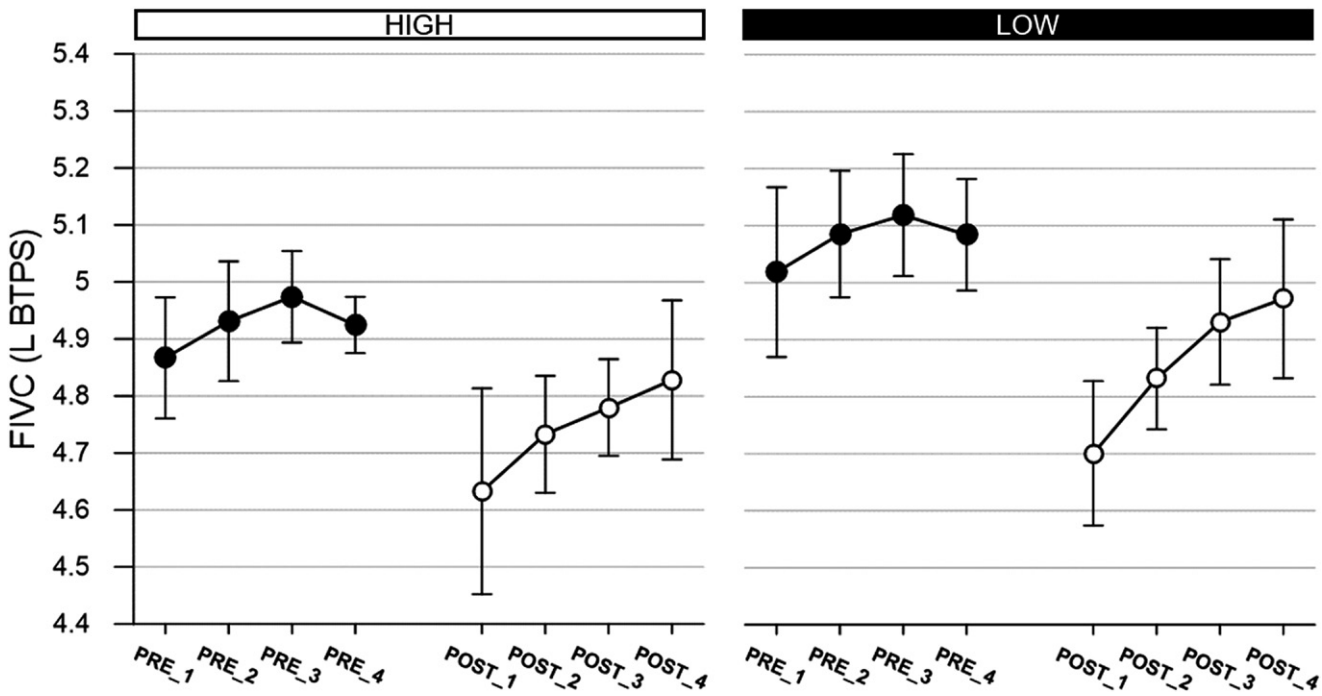

Fig. 1. Forced inspiratory vital capacity measured four times before (Pre; black circles) and after (Post; white circles) the High (left) and Low (right) cabin altitude sortie. Data (N = 14) are mean ± the 95% confidence interval after correction for between-subject variability.

Table III. Data for: S_pO_2 Measurements Whilst Breathing Air, Hyperoxic Gas (100% O_2), and Hypoxic Gas (14% O_2); Tidal Volume ($L \cdot s^{-1}$) and Respiratory Rate (Breaths per Minute) During Pre-FIVC Hypoxia; and End-Tidal Partial Pressures of O_2 ($P_{ET}O_2$) and CO_2 ($P_{ET}CO_2$) during FIVC.

	Pre	Post
$S_pO_{2\ normoxia}$ (%)		
High	97.7 ± 0.6	96.2 ± 0.8*
Low	97.6 ± 0.7	96.5 ± 0.7*
$S_pO_{2\ hyperoxia}$ (%)		
High	99.1 ± 0.6	98.7 ± 0.6*
Low	99.3 ± 0.8	98.6 ± 0.5*
$S_pO_{2\ hypoxia}$ (%)		
High	94.1 ± 0.9	92.3 ± 0.8*
Low	94.5 ± 1.0	92.7 ± 0.9*
Tidal Volume ($L \cdot s^{-1}$)		
High	1.21 ± 0.30	1.11 ± 0.15
Low	1.02 ± 0.16	1.03 ± 0.13
RR (bpm)		
High	11.9 ± 1.2	12.1 ± 1.1
Low	13.7 ± 1.4	13.6 ± 1.3
$P_{ET}O_{2\ hypoxia}$ (mmHg)		
High	68.4 ± 3.8	68.0 ± 3.1
Low	70.4 ± 2.8	69.2 ± 3.5
$P_{ET}CO_{2\ hypoxia}$ (mmHg)		
High	40.2 ± 1.7	38.2 ± 1.7*
Low	38.6 ± 1.6	36.4 ± 1.7*

Data (N = 14) are mean ± the 95% confidence interval after correction for between-subject variability.
*Denotes significance ($P \leq 0.05$) on post hoc paired data pre- and postflight.

Low (P = 0.002) altitude condition (see Table II). However, unlike measures of FIVC, regional (basal) EIT lung volume had not recovered to baseline values at FIVC4. EIT data were only available from 10 and 13 subjects in the High and Low condition, respectively. Loss of data was typically due to improper electrode contact, which was measured as part of the systems signal quality assessment prior to recordings with registration of data only possible if all 16 electrodes presented electrode-to-skin contact impedance below a predetermined threshold.

S_pO_2 was lower postflight than preflight for all of the three oxygen gas mixes inspired ($P < 0.05$), with no differences found between the High and Low cabin altitude conditions (see **Table III**. The lower oxygen saturations resulted in an increase in the estimated pulmonary shunt from 1.9 ± 2.5% to 4.9 ± 3.3% (P = 0.011) and from 1.9 ± 2.3% to 5.7 ± 3.3% (P = 0.003) in the High and Low cabin altitude conditions, respectively. Changes in the estimated shunt fraction were comparable between the two cabin altitude conditions. V_T and RR were unaffected by exposure to high +G_z flight breathing 60% oxygen following either High or Low cabin altitude sorties. There was also no effect on postflight $P_{ET}O_2$, but $P_{ET}CO_2$ was significantly decreased following both High (P = 0.021) and Low (P = 0.008) cabin altitude sorties (Table III).

DISCUSSION

This study investigated whether a minimum 60% oxygen (balance 40% nitrogen) breathing induces acceleration atelectasis in pilots during high +G_z flight and explored whether cabin altitude had any influence on the extent of atelectasis identified. This gas mix was chosen because most combat aircraft using onboard oxygen generator technology supply around 60% oxygen to the crew at cabin altitudes where dynamic G_z maneuvering is conducted, so it is representative of real-world conditions.

Change in FIVC was used as the primary measure of atelectasis in this study. Multiple exposures to +5 G_z while breathing 60% oxygen were found to be associated with mean reductions in FIVC of 4.9% (−0.24 L) and 3.8% (−0.17 L) when FIVC1 was compared with the mean preflight FIVC in the High and Low cabin altitude sorties, respectively. These findings are consistent with previous research;[14] one unpublished study reported a 4.3% decrease in FIVC after exposing subjects to 75 s at +4 G_z while breathing 60% oxygen.[11] When using contemporary pilot flight equipment and life support systems, the current study therefore demonstrates that an F_IO_2 of 60% is still sufficient to moderate the development of acceleration atelectasis, although a reduction in lung volume still occurs. As expected, postflight FIVC recovered with successive breathing maneuvers. Symptoms were not formally assessed in the current study, but a cough, or an urge to cough, was seen during postflight FIVCs (illustrated in **Fig. 2**) with relatively few symptoms reported in flight. The reduction in FIVC is attributable to inspiratory limitation, with a loss of inspiratory capacity that broadly matches the reduction in FIVC (rather than loss of functional residual capacity).

Placement of EIT electrodes in this study was used to measure changes within the lower lobes of the lungs,[23] and so, the decrease in magnitude of the impedance change with lung inflation during the postflight FIVCs suggests that there was a reduction in basal lung volume. The fall in $FIVC_{EIT}$ as a percentage of pre-exposure values was much larger than accompanying changes in FIVC (5 vs. 15%) and did not recover to pre-exposure values at FIVC4. This suggests that basal lung regions remain resistant to re-expansion and the discrepancy with normalized estimates of FIVC demands further consideration.

It is possible that the 'recovery' in overall lung volume actually represents recruitment of lung regions known as a Pendelluft phenomenon.[12] Alternatively, artifacts caused by changes in impedance at the electrode-to-skin interface, for example by increased sweating, could be present. However, EIT measures were relative, representing a change from that recorded at residual volume (RV), so any offset in voltage at the electrodes would have largely been negated. EIT derived measures of V_T and FIVC are repeatable,[2,23] but the effects of cabin altitude could not be compared with this technique as identical electrode placements could not be guaranteed on consecutive days. Nevertheless, good correlation was observed between Pre measurements on each test day (r^2 = 0.87). EIT data in Table II and Fig. 2 show three FIVC maneuvers of a symptomatic subject pre- and postexposure. The first Post $FIVC_{EIT}$ was approximately 24% lower than Pre and toward the end of inspiration the subject coughed (depicted as a double peak in the impedance trace). FIVC end-expiratory impedance then increased, indicating recruitment of lung tissue clearly visible in the EIT images. Successive FIVC maneuvers were higher but did not recover to preflight values.

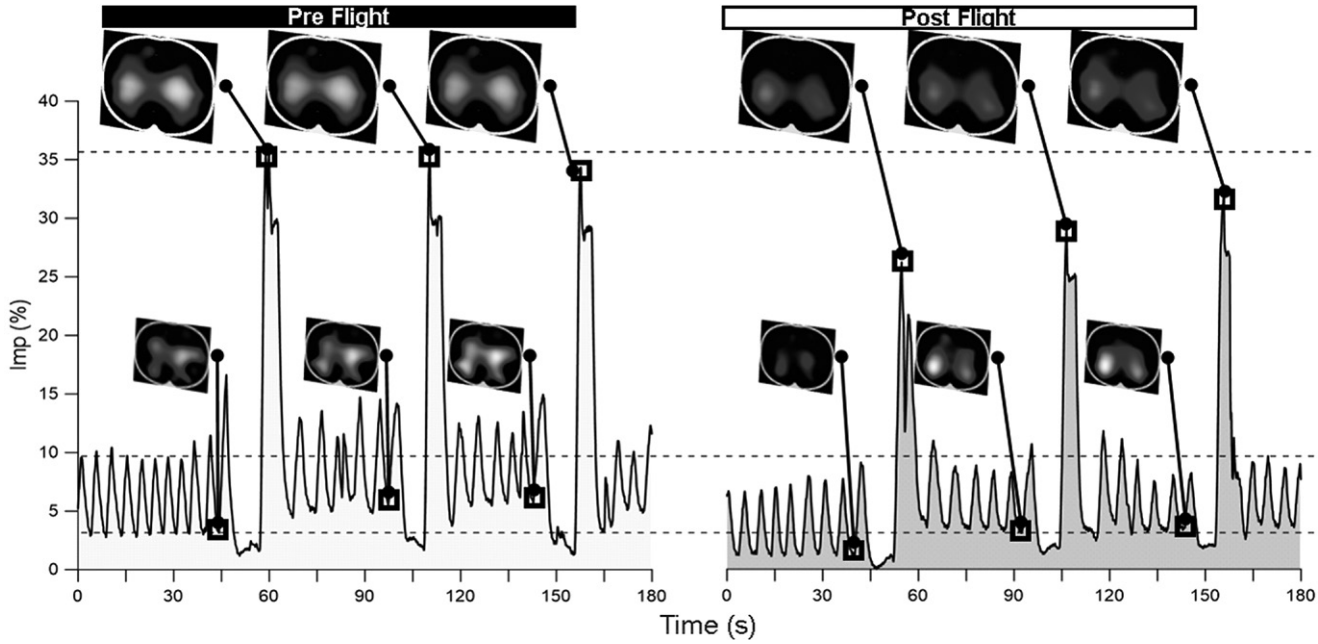

Fig. 2. Images of regional lung impedance acquired by electrical impedance tomography (EIT) over time for the whole image during the performance of three forced inspiratory vital capacity (FIVC) maneuvers (each separated by a period of tidal breathing) performed pre- (left) and postflight (right) in participant 5 (low altitude sortie). The top row of images is that recorded at maximal inspiration and the bottom at the end of a normal breath (i.e., functional residual capacity; FRC). To visualize changes a greater gain was used for images at FRC (×4.5) than at maximal lung volume, therefore comparison of images can only be made within each row. The dashed lines represent the impedance recorded at average end expiration, end expiration plus mean impedance change during tidal breathing, and peak impedance during a maximal breath preflight. Images were referenced as changes from the image recorded at residual volume and thus scales are configured to contrast relative changes in impedance. Note that this subject coughed during the performance of the first FIVC postflight (observed in the impedance trace as a double peak).

The postflight reduction in peripheral arterial oxygen saturation was consistent between subjects under the conditions investigated (Table III). The decrease observed in this study, although highly statistically significant, is modest and unlikely to have operational relevance. It does, however, indicate impairment of blood oxygenation. It is possible that mild atelectasis (as evidenced by the decrease in FIVC) was sufficient to result in a small pulmonary shunt. The reduction in arterial oxygen saturation lends support to the possibility suggested by the EIT data of a persistent functional impairment that is not reflected by overall measures of lung volume. Persistent reduction in arterial oxygen tension has been demonstrated following 75-s exposure to +4 G_z on the centrifuge, providing the subject avoided deep breathing.[10] A flight trial involving a series of maneuvers at +5.5 G_z sustained for 30–40 s over a total sortie duration of 30–40 min has also demonstrated postflight reductions in S_pO_2 (breathing air) that are of similar magnitude to those reported in the current study.[14] These findings suggest compromised lung function and blood oxygenation postflight, possibly due to atelectasis. However, it is widely accepted, and supported here, that the provision of 60% oxygen is sufficient to prevent gross atelectasis formation and will, in any case, provide adequate oxygenation during flight. Further studies using different F_IO_2 may still be required to delineate the mechanism and to determine whether more significant postflight reductions, potentially of operational relevance, occur with higher F_IO_2.

One of the aims of the study was to investigate whether cockpit pressure altitude had a protective effect on the development of acceleration atelectasis. In this study we found no difference in outcomes between flights carried out at cockpit pressure altitudes of 4000 and 18,000 ft (1219 and 5486 m), a maximum difference in barometric pressure of approximately 276 mmHg. It is possible that a different outcome might result with a higher F_IO_2, which could result in greater development of acceleration atelectasis.

In prioritizing consistent +G_z exposures across both conditions, due to performance limitations of the Hawk T Mk1 aircraft, possible order effects could not be controlled out. The statistically significant difference in baseline FIVC on the two flight days indicates that this introduced a systematic, methodological confound, which is nonetheless of interest. Preflight baseline FIVCs were consistently greater on the second day of testing (Low cabin altitude) ($P = 0.036$). It is possible that the initial fit of specific AEA garments may have relaxed following the first sortie and thus afforded greater expansion of the lungs during maximal inhalation. This perhaps warrants further study to examine how garment fit changes with repeated use and if there are implications to G protection. Notably, however, if any slackening of AEA fit did occur, the magnitude of changes in lung volumes during flight was unaffected.

Further limitations of the study, additional to the control of exposure orders, include delays between laboratory measurements and +G_z exposures. Despite endeavors to minimize this

interval, mandatory in-aircraft pre- and postflight procedures could not be shortened. Additionally, a nonflying control condition was not included and, therefore, could not be compared with postflight data. The acceleration exposures were not fully representative of air combat in magnitude or duration but were repeatable and within aircraft capability at both altitudes studied.

The major findings from this study are: a small reduction in postflight lung volume occurred, but recovered with repeated maximal inspirations, suggesting limited development of atelectasis; regional surrogate measures of basal (caudal) lung volume by EIT also decreased but did not recover to preflight values; peripheral arterial oxygen saturation was decreased postflight, suggesting formation of a small (5–6%) pulmonary shunt; and none of the measurements were influenced by cabin altitude. In summary, postflight measurements of FIVC and $FIVC_{EIT}$ indicate that mild degrees of atelectasis can occur following multiple in-flight exposures to +5 G_z while breathing 60% oxygen at low and high cabin altitudes. $FIVC_{EIT}$ data imply that mildly atelectatic regions may not fully resolve with simple postflight breathing maneuvers.

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