Aircrew Conditioning Programme Impact on +Gz Tolerance

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INTRODUCTION: Physical conditioning may improve aircrew performance during exposure to high +G_z acceleration, although few studies have directly assessed this. The present study investigated the effects of a 12-wk Aircrew Conditioning Programme (ACP) on markers of G tolerance. The ACP comprises aerobic and muscle strengthening exercise performed twice weekly and targets improved fitness and reduced injury risk.

METHODS: There were 36 UK Royal Air Force and Royal Navy aircrew who volunteered; 17 performed the ACP (Ex) and 19 acted as a control group (Con). Centrifuge testing was performed before and after the intervention. Relaxed G tolerance (RGT) and straining G tolerance (SGT), which had the addition of muscle tensing, were assessed. G endurance was also determined via repeated simulated air combat maneuvers (SACMs). During these centrifuge runs a number of physiological variables were recorded.

- **RESULTS:** During the G profile to determine RGT, neither RGT, HR, nor blood pressure responses were affected by the ACP. During SGT profiles, a lower HR at a given $+G_z$ (+5.5 G_z) level following the ACP was observed (Ex: pre 146.0 \pm 4.4, post 136.9 \pm 5.6 bpm; Con: pre 148.0 \pm 3.2, post 153.1 \pm 3.3 bpm). BP was maintained and there was a tendency toward an improved SGT. The ACP increased the proportion of individuals completing the number of SACM profiles, although no meaningful differences were found between groups in other variables.
- **CONCLUSION:** Overall the ACP has no negative effect on RGT, reduced the physiological strain associated with a given level of $+G_z$ (during SGT), and tended to improve the ability to tolerate repeated G_z exposure.
- **KEYWORDS:** exercise, acceleration, aircrew, G tolerance.

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outine exposure to high, sustained acceleration in the head-to-foot direction (+ G_z acceleration) can have serious consequences for aircrew of agile fighter aircraft in terms of maintenance of cerebral perfusion and neck injury from muscle strain.¹⁹ The effects of sustained $+G_z$ exposure can include a loss of peripheral vision, gray-out, blackout, and loss of consciousness (G-LOC) with a 15% prevalence of G-LOC reported in UK Royal Air Force (RAF) aircrew in a survey in 2012.³⁵ The effects of $+G_z$ exposure are a result of reduced cerebral blood flow consequent to an increased head-to-heart hydrostatic pressure gradient and pooling of blood in the lower limbs.¹⁹ These effects may be compounded by the development of fatigue and result in an impaired ability to perform the demanding anti-G straining maneuver (AGSM) which is undertaken to ameliorate the effects of +G_z exposure. The AGSM uses continuous contractions of muscles in the lower limbs and abdomen combined

with a cyclical Valsalva maneuver to prevent lower limb pooling and maintain cerebral perfusion.¹⁹

Due to the physical effort required by the AGSM, muscle strength and aerobic fitness are likely to have a key role in performance during $+G_z$ exposure.⁴ In the longer term, exercise training may also be of benefit to counteract the risk of strain injuries, particularly to the neck, that are associated with exposure to high $+G_z$. The prevalence of neck injury in fighter pilots ranges from 63 to 83%.^{23,28,40} However, equivocal findings exist

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regarding the effects of physical conditioning programs on G tolerance,⁴ which can be assessed in a number of ways. Relaxed G tolerance (RGT) refers to the G level an individual can reach in a relaxed state that is associated with a specific symptom, typically a predefined level of peripheral light loss or G-LOC. RGT reflects an individual's underlying physiological response to +G_z. Straining G tolerance (SGT) reflects the G level an individual can attain, to a predefined symptom endpoint, while performing components of the AGSM. In contrast, G endurance refers to the duration of a specific G profile that can be sustained, typically when performing the AGSM and when using anti-G trousers (AGT).

Relaxed G tolerance has typically been shown to be unaffected by resistance training,⁶ and does not differ between individuals with a long history of strength or endurance training and untrained individuals.²² Some studies have reported improved G endurance in response to resistance training.^{2,18,38} but this is not always the case,⁶ with experimental design or data analysis in these studies confounding interpretation of their results.⁴

A new Aircrew Conditioning Programme (ACP) comprising specific muscle strengthening, aerobic, and anaerobic exercises aimed at improving function and G tolerance, reducing the risk of injury, and promoting general health and wellbeing in aircrew has been developed.³⁴ The present study was designed to test the efficacy of the ACP with regards to its effect on G tolerance. The specific aims of the present study were to evaluate the effects of the ACP on RGT, the physiological strain for a given +G_z under conditions where partial anti-G maneuvers (muscle tensing) were employed (SGT) and the ability to tolerate multiple exposures to high +G_z profiles which mimic combat flying (G endurance).

METHODS

Subjects

All subjects were either UK RAF or Royal Navy (RN) aircrew enrolled in Elementary Flying Training (EFT) or Basic Fast Jet Training (BFJ) courses. For reasons related to the participants military training, the BFJ volunteers were preselected to the exercise group (Ex, N = 17), while the EFT volunteers were preselected to the control group (Con, N = 19). All subjects were men and the characteristics of each group are displayed in **Table I**. Inclusion criteria included holding a current RAF or RN Medical Employment Standard without any temporary or permanent limitations. In addition, all subjects had passed an aircrew medical in the last 12 mo, including 12-lead ECG, and completed and had passed the RAF or RN Fitness test. Prior to testing all subjects were familiarized with the $+G_z$ levels and profiles used in the present study. Subjects attended a preintervention test session which was then repeated after a 12-wk intervention period.

Prior to participation in the study all subjects provided written informed consent. All procedures were approved by the UK Ministry of Defense Research Ethics Committee (MoDREC/14/532) and conformed to the Declaration of Helsinki.

Equipment

All centrifuge exposures were conducted on a 9.14-m human centrifuge (Farnborough, UK). During testing, subjects were harnessed into a deactivated ejection seat (Mk 16, Martin Baker Aircraft Company Ltd, Denham, Middlesex, UK) located in the gondola and they wore standard flying coveralls, aircrew boots, Mk10b helmet, and P/Q oronasal mask. In addition, subjects wore 5-bladder AGTs (Mk4, Survitec, London, UK) which were pressurized by an anti-G valve (VAS 110-022, Hymatic Ltd., Redditch, UK). During RGT and SGT profiles the anti-G valve was deactivated, i.e., AGT were worn but not inflated. The AGTs were activated only for the final series of +G_z exposures (simulated air combat maneuvers—SACMs).

Subjects assessed the extent of their peripheral light loss during acceleration exposures by referencing a bank of three lights positioned at eye level. This consisted of a central continuous white light and two red flashing lights positioned at a 60° angle relative to eye position. While on the centrifuge a 3-lead ECG was continually recorded to allow determination of heart rate (HR). Blood pressure (BP) and consequently systolic (SBP), diastolic (DBP), and mean arterial (MAP) BP were continually monitored by the volume-clamp method²⁹ with a cuff fitted to the middle finger of the left hand (Ohmeda 2300 Finapress, BOC Health Care, Madison, WI, USA). The left arm was supported on a padded armrest at heart level, allowing an estimate of heart level BP. Eye level systolic (SBP_{eve}), diastolic (DBP_{eve}), and mean arterial (MAPeve) pressure were determined by measuring the distance between heart and eye level of each subject and subsequently subtracting the hydrostatic equivalent blood pressure³ from the heart level blood pressure values obtained.

Procedure

A two group controlled trial was conducted in which the effects of a 12 wk ACP³⁴ on $+G_z$ tolerance and physiological outcomes was assessed. On each day of testing subjects began by performing two RGT gradual onset acceleration profiles followed by two SGT step acceleration profiles and finally four simulated air combat maneuvers (SACMs) (**Fig. 1**). G endurance has typically been assessed by performing a SACM until exhaustion; however, to better understand the effects of the ACP on performance during G, four discrete SACMs were performed. This ensured the same total workload, in terms of $+G_z$ exposure, was performed by each subject, rather than perform a single G profile until exhaustion.

RGT

Subjects performed two RGT centrifuge exposures, separated by 2 mins, each with an onset rate of $0.1 \text{ G} \cdot \text{s}^{-1}$. They remained relaxed throughout the centrifuge exposure with a visual fixation on the central light directly ahead of them. At the point where they were no longer able to perceive the two flashing red lights (i.e., 60° peripheral light loss), subjects depressed a button which placed a marker on the acceleration trace and stopped the centrifuge; this point was taken as the subject's RGT. The gradient of the HR, SBP_{eye}, DBP_{eye}, and MAP_{eye} recorded during the run was calculated in LabChart and divided by the acceleration onset rate ($0.1 \text{ G} \cdot \text{s}^{-1}$) to give the slope of each

	CONT	ROL	EXERCISE			
	PRE	POST	PRE	POST		
Age (yr)	23.4 (0	0.4)	25.8 (0.4)†		
Height (cm)	179 (1.4)	180 (0.9)		
Mass (kg)	82.8 (2.0)	83.6 (2.2)	80.9 (2.1)	80.7 (2.3)		
BMI (kg · m ^{−2})	25.6 (0.5)	25.8 (0.5)	24.8 (0.5)	24.7 (0.5)		
HR (bpm)	65.2 (1.8)	62.2 (2.0)*	56.3 (2.2) [†]	59.1 (2.9)		
SBP (mmHg)	122. (2.6)	121. (2.9)	118. (2.2)	118. (1.8)		
DBP (mmHg)	67.2 (1.3)	66.6 (1.5)	64.3 (1.6)	64.6 (1.5)		
MAP (mmHg)	85.5 (1.6)	84.9 (1.8)	82.3 (1.4)	82.7 (1.6)		
MFPF (N) [‡]	3730 (333)	4232 (273)	4206 (248)	4800 (325)		
KE Torque (Nm)	205.7 (13.3)	210.9 (11.3)	210.7 (11.3)	216.5 (10.7)		
KF Torque (Nm)	83.2 (7.5)	83.8 (6.8)	92.9 (7.7)	84.7 (5.7)		
PF Strength (N) [‡]	1468 (85)	1391 (61)	1374 (78)	1286 (94)		
Lactate (mmol/L)	5.8 (0.9)	6.1 (0.8)	5.4 (0.3)	5.5 (0.6)		

All values are mean \pm SE. BMI – body mass index, SBP – systolic blood pressure, DBP – diastolic blood pressure, MAP – mean arterial pressure, MFPF – maximum foot pedal force, KE – knee extensors, KF – knee flexors, PF – plantar flexors. *Significantly different to pre-intervention (P = 0.034), [†]Significantly different from Control group (P < 0.01), [‡]main effect of Time (P < 0.05).

variable (i.e., change in variable per G level), and the means of both runs were determined. The AGSM was not performed and AGT were not used to remove any confounding effects they have on the underlying physiological responses to $+G_z$.

SGT

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The SGT was assessed via two centrifuge exposures consisting of +3 G_z for 15 s which subsequently increased every 5 s by +0.5 G_z until the point where the subject terminated the run or +7 G_z was reached. All increases in acceleration were at an onset rate of 1.0 G · s⁻¹ and AGT were not activated. A rest of 2 min was given between exposures. During the run subjects were requested to remain relaxed until 60° peripheral light loss occurred, at which point they performed sufficient lower body and abdominal muscle tensing to maintain clear vision. When they were no longer able to prevent peripheral light loss (as above), the subject terminated the run and that point was recorded as the SGT. Electromyography (EMG) was used to assess the levels of muscle activation of the knee extensors, flexors, and plantar flexors (see below) with muscle tensing



pre- to post-intervention. The SGT profiles were conducted to assess whether changes in lower body muscle strength induced by the ACP influenced G tolerance and if the physiological strain associated with a given workload varied post-ACP.

G Endurance

The final centrifuge exposures during each test session involved performing four SACMs separated by 2 min. Each SACM comprised four exposures to +7 G_z , each lasting 5 s, with a 15-s period at +5 G_z between peaks. The onset and offset rate used was $1.0 \text{ G} \cdot \text{s}^{-1}$. AGTs were activated and subjects performed the AGSM as required during the exposures. A rest of 2 min was given between exposures, with the centrifuge in idle $(1.5 \text{ G} \cdot \text{s}^{-1})$ to reduce nausea effects. Subjects were requested to use the stop button at any point if they were unable to prevent peripheral light loss. Immediately following each SACM they stated their rate of perceived exertion (RPE) using the modified Borg category-ratio scale.²⁶

A blood lactate reading was obtained via a finger prick blood sample (Accu-Check Softclix, Accu-Chek, Roche Diabe-





tes Care, West Sussex, UK) 3 min after the final SACM. The mean HR and muscle activation recorded during individual SACMs were also determined. Blood pressure during the SACM was not analyzed due to artifact in the signals during the AGSM, preventing reliable data. For each SACM, only data from those subjects who completed that entire SACM both before and after the intervention were included in the analysis.

Prior to the centrifuge exposures, several physiological measurements were also taken. Each subject had their resting HR, SBP, DBP, and MAP recorded after 5 min of quiet supine resting. HR was determined by 3-lead ECG (FE-262-B1/A-1 Isolation Amplifier, Fylde Electronics Laboratories, Preston, UK) while blood pressures were determined using an automated sphygmomanometer (IntelliSense 705IT, Omron Healthcare, Milton Keynes, UK). Following this the isometric maximum voluntary strength (MVC) of the left knee extensors (KE), knee flexors (KF), and plantar flexors (PF) were assessed. KE and KF MVCs were performed with the subject seated in a custom-built dynamometer with their knee in 90° of flexion. Their lower leg (approximately 3 cm distal to the ankle) was secured to a padded steel brace which was attached to a strain gauge via a rigid bar (for KE measurement the bar was placed posteriorly and for KF the bar was positioned anterior to the leg). Waist and shoulder straps were used to minimize subject movement. Each subject performed three MVCs (for both KF and KE) where they were asked to generate maximal force as quickly as possible and hold for 5 s. Standardized verbal encouragement was given throughout by the same tester for each subject.

To assess PF strength subjects sat upright in a rigid chair which was attached to a dynamometer. They sat with their hip, knee, and ankle at 90° and a specially designed clamp was placed over the distal portion of the knee to prevent heel lift.¹⁵ The same procedure as described for KF and KE MVCs was performed. A rest period of 1 min was given between contractions. Force signals were recorded in LabChart 7 (AD Instruments, Oxford, UK) via an analog-to-digital convertor (PowerLab 16/s; AD Instruments) and sampled at a frequency of 200 Hz. The maximum MVC recorded for each muscle group was used for analysis.

EMG (m320, Myon AG, Schwarzenberg, Switzerland) was recorded during all MVCs. After lightly abrading and cleaning the skin, and where necessary removing hair, two electrodes (1 cm diameter, Ambu BluSensor N, Ambu, St. Ives, Cambridgeshire, UK) separated by 1 cm were placed over each muscle and secured using medical tape. The location of EMG recordings were: vastus lateralis (VL)-two-thirds of the way along a line from the anterior superior iliac spine and the lateral side of the patella; biceps femoris (BF)-half way along a line from the ischial tuberosity and the lateral epicondyle of the tibia; and the gastrocnemius lateralis-one-third of the way along a line from the head of the fibula to the heel.²¹ EMG was recorded with a sampling frequency of 2000 Hz, gain of 1000 and band-pass filtered between 5-500 Hz. All signals were rectified and smoothed (4-Hz low pass filter). The peak rms EMG over a 50-ms period during the MVC was identified and used for subsequent normalization of EMG signals. Electrodes remained secured to the subject throughout the MVC testing and centrifuge exposures.

Following initial testing the Ex group completed the 12-wk ACP.³⁴ This is a structured, progressive exercise program which is delivered for 1 h, twice weekly, supervised by military physical training instructors and physiotherapists. The four main components of the ACP target: 1) whole body flexibility; 2) cardiovascular fitness; 3) stability and control of the neck, shoulder girdle, and trunk; and 4) strengthening of the neck, back,

abdominal, and leg muscles. Subjects in the control group were requested to continue to perform their normal exercise routine in the 12 wk between test sessions, which did not include any neck-specific or whole-body strength training.

Statistical Analysis

The mean values obtained from the two SGT and RGT centrifuge exposures were used for analysis. The normality of the data was assessed using the Kolmogorov-Smirnov test with Mauchly's test of sphericity used to assess variance homogeneity. As SGT was not normally distributed a log transform was applied. A 2-way mixed ANOVA (Group*Time) with Bonferroni post hoc analysis was conducted. Unless otherwise stated the results of the Group*Time interaction are reported. RPE was assessed using the Kruskall-Wallis test. An alpha level of 0.05 was used. All data are reported as mean \pm SE.

RESULTS

The ACP and experimental sessions were well tolerated, with only a single subject in the Ex group unable to complete the post-ACP experimental session due to a musculoskeletal injury unrelated to the intervention. Their data was excluded from analysis. Both groups were similar at baseline (P > 0.05 in all cases—Table I) except for the Con group being significantly (P < 0.05) younger and having a higher resting HR (P = 0.004—Table I).

Self-reported physical activity levels ascertained by the weekly activity log were similar in both groups apart from participation in the ACP by the Ex group. The Ex group completed more flight hours (33.0 \pm 3.6 h) than the Con group (8.8 \pm 1.2 h), but neither group participated in any air combat or high +G_z (>+5 G_z) sorties that may have influenced results, with the majority of time for all subjects having been spent in ground school.

Overall the ACP had no effect on body mass [F(1,34) = 3.307, P = 0.090] or BMI [F(1,34) = 3.305, P = 0.078], although in both instances there was a tendency toward an increase in the Con group in both parameters (P = 0.056 and 0.053, respectively). Resting SBP [F(1,34) = 0.236, P = 0.630], DBP [F(1,34) = 0.200, P = 0.657], and MAP [F(1,34) = 0.338,P = 0.565] were unaffected. A significant effect was found on resting HR [F(1,34) = 8.407, P = 0.007], with a lower HR postintervention in the Con group (P = 0.034) and a tendency toward this in the Ex group (P = 0.066). Overall there was no between group differences post-intervention in PF strength [F(1,33) = 0.018, P = 0.895], KE torque [F(1,33) = 0.003,P = 0.953], or KF torque [F(1,33) = 2.263, P = 0.143], although PF strength [F(1,34) = 4.487, P = 0.042] was lower in both groups following the intervention (Table I).

RGT

There was a tendency toward an intervention effect on RGT [F(1,33) = 3.769, P = 0.061] (**Fig. 2**) with an increase in RGT of +0.12 G_z in the Ex group (P = 0.186) and a decrease in the



Fig. 2. Relaxed G tolerance (RGT), heart rate (HR), eye-level systolic blood pressure (SBP_{eye}), eye-level diastolic BP (DBP_{eye}), and eye-level mean arterial pressure (MAP_{eye}) recorded during gradual onset (0.1 G · s⁻¹) centrifuge exposures terminated at peripheral light loss. Slopes refer to the change in each variable with increasing +G_z. All values are mean \pm SE. Gray bars are pre-intervention while white bars are post-intervention measurements.

Con group of +0.11 G_z (P = 0.171). There was no effect of the ACP on the response of SBP_{eye} [F(1,33) = 0.881, P = 0.355] or MAP_{eye} [F(1,33) = 0.349, P = 0.559], while there was a tendency toward a difference in DBP_{eye} [F(1,33) = 4.024, P = 0.054], with post hoc analysis again revealing a tendency for an increase in the Con group (P = 0.067) only. The HR response during the RGT exposures was unchanged post intervention for both groups [F(1,33) = 1.071, P = 0.308].

SGT

There was a tendency toward an intervention effect on SGT [F(1,33) = 1.759, P = 0.144], with a nonsignificant increase in the Ex group (P = 0.17), while the Con group remained unchanged (P = 0.653—**Fig. 3**, top left panel). Overall no effect on the +G_z level at which muscle contractions began [F(1,32) = 1.662, P = 0.207] was found, although both groups began muscle contraction at a lower +G_z level post-intervention [F(1,32) = 4.375, P = 0.044] (Fig. 3, top center panel).

SBP [F(1,31) = 0.648, P = 0.427], DBP [F(1,31) = 0.839, P = 0.367], and MAP [F(1,31) = 0.829, P = 0.370] recorded at +5.5 G_z (Fig. 3, bottom row) were unchanged. However, a significant effect of the ACP was observed in HR [F(1,32) = 13.35, P = 0.001], with a lower HR post-intervention at +5.5 G_z in the Ex group (P = 0.004) with a tendency toward an increased HR in the Con group (P = 0.055; Fig. 3, top right panel). The levels of VL and BF activation at +5.5 G_z were unchanged [F(1,31) =

0.118, P = 0.734 and F(1,26) = 0.441, P = 0.513, respectively] post-intervention. In contrast, an effect on gastrocnemius lateralis activation was found [F(1,29) = 4.701, P = 0.038], with a small but significant (3.9%MVC P = 0.043) reduction in its activation in the Ex group while it was unchanged in the Con group (P = 0.226—**Table II**).

G Endurance

In the ACP group the proportion of subjects completing each SACM increased following the intervention (SACM 1: 94 vs. 100%; SACM 2: 88 vs. 100%; SACM 3: 88 vs. 94%; SACM 4: 81 vs. 88%; pre vs. post, respectively). In contrast the proportion of subjects in the Con group completing each SACM remained unchanged or decreased post-intervention (SACM 1: 95 vs. 95%; SACM 2: 95 vs. 89%; SACM 3: 89 vs. 84%; SACM 4: 74 vs. 68%; pre vs. post, respectively).

For SACMs 1 to 4, only those subjects who completed each individual SACM both pre- and

post-intervention were analyzed; the numbers of subjects in each group achieving this are shown in Fig. 4. There was no effect of the intervention on the mean HR during the SACM (P > 0.05 in all cases; see Table II). There was, however, a lower mean HR recorded in the Ex group during SACM 1 [F(1,29) = 5.106, P =(0.032], 2 [F(1,29) = 5.348, P = 0.029], and 3 [F(1,25) = 4.762, P = 0.039] than in the Con group, but not for SACM 4 [F(1,20) =2.108, P = 0.162]. With the exception of the BF muscle during SACM 3 [F(1,19) = 5.605, P = 0.029], muscle activation was unchanged (see Table II for statistics). During SACM 3 BF activation was reduced in the Con group (P = 0.032), while there was a lower activation in the Ex group compared with the Con group pre-intervention. RPE associated with each SACM was unaffected by the intervention in either group (SACM 1: $H_3 = 3.782$, P = 0.286; SACM 2: H₃ = 2.785, P = 0.426; SACM 3: H₃ = 5.744, P = 0.125; SACM 4: $H_3 = 3.374$, P = 0.337; Fig. 4). In those subjects who completed all four SACMs pre- and postintervention there was no change in blood lactate recorded after the final SACM [F(1,32) = 0.116, P = 0.736; Table I].

DISCUSSION

The main findings of the present study were that 12 wk of a validated mixed exercise training program (ACP) did not negatively impact RGT and reduced the physiological strain

the ACP.

improved ability to tolerate the

SACM profiles was greater after

found no effect of 10-wk resis-

tance training on RGT in air-

crew,⁶ while untrained, resistance trained, and endurance trained

individuals have similar RGTs.²²

While not significant, there was a

tendency for RGT to improve by $+0.11 \text{ G}_z$ in the Ex group,

although this level of improve-

ment is unlikely to provide a

meaningful benefit to aircrew

performance. Interestingly, a

reduced vascular stiffness in precapillary vessels has been noted

in individuals with low RGT compared to those with a high

RGT.¹⁶ This difference is likely

due to individuals with a reduced

vascular stiffness having greater

+G, induced pressure distension

of dependent vessels¹⁶ and,

consequently, reduced systemic

vascular resistance. In young

individuals, resistance training

Previous investigations have



Fig. 3. Straining G tolerance (SGT), $+G_z$ level at which muscle contraction commenced, heart rate (HR) at $+5.5 G_{zr}$ mean arterial blood pressures (MAP) at $+5.5 G_{zr}$, systolic blood pressure (SBP) at $+5.5 G_{zr}$, and diastolic blood pressure (DBP) at $+5.5 G_z$. Gray bars represent pre-intervention and white bars post-intervention measurements. With the exception of SGT, all figures show mean \pm SE *Significantly different from preintervention (P = 0.004). Bars in contraction onset indicate a main effect of Time (P = 0.044).

for a given $+G_z$ level, which was associated with a tendency toward an improved SGT. Furthermore, for G endurance, while the physiological variables assessed during SACMs were not affected by the ACP, the proportion of individuals with an is associated with an approximately 11% increase in systemic vascular resistance.²⁷ It is therefore possible that the small increases noted in RGT could be due to an increased systemic vascular resistance consequent to the ACP. The lack of a

Table II. Mean (SE) Heart Rate (HR) and Muscle Activity (% Maximal Voluntary Contraction) of the Vastus Lateralis (VL), Biceps Femoris (BF), and Gastrocnemius Lateralis (GL) at $+5.5 \text{ G}_z$ During the Straining $+\text{G}_z$ Tolerance Profiles, and Mean Peak HR and Mean Muscle Activity Across Each of the Simulated Air Combat Maneuvers (SACM) with Group*Time Interaction Statistical Results During Each SACM.

	SGT AT +5.5 G _Z		SACM 1		SACM 2		SACM 3		SACM 4	
	PRE	POST	PRE	POST	PRE	POST	PRE	POST	PRE	POST
HR										
Control	148.0 (3.2)	153.1 (3.3)	152.5 (2.8)	151.9 (2.8)	154.1 (2.9)	150.8 (3.1)	155.8 (3.0)	150.2 (3.4)	156.9 (3.5)	156.1 (3.3)
Exercise	146.0 (4.4)	137.0 (5.6)	138.1 (3.0)	138.9 (3.1)	140.1 (3.1)	141.9 (3.3)	141.4 (3.3)	142.2 (3.8)	145.0 (3.8)	144.6 (3.6)
			F(1,29) = 0.685,		F(1,29) = 0.007,		F(1,25) = 0.293,		F(1,20) = 0.326,	
VI			r —	0.415	r =	0.932	r =	- 0.595	r = 0	0.575
Control	39.1 (5.1)	36.4 (5.5)	24.2 (2.3)	26.1 (3.8)	19.4 (2.1)	23.8 (4.0)	19.7 (2.4)	23.8 (4.0)	24.0 (3.6)	27.2 (3.6)
Exercise	40.4 (6.5)	37.1 (7.2)	24.5 (3.2)	25.8 (3.6)	22.8 (4.1)	26.1 (4.0)	18.7 (3.2)	24.1 (3.3)	19.7 (3.7)	23.8 (4.0)
			F(1,29) = 0.043, P = 0.838		F(1,25) = 0.026, P = 0.874		F(1,22) = 0.004, P = 0.953		F(1,22) = 0.057, P = 0.814	
BF										
Control	25.2 (3.6)	18.1 (2.7)	26.4 (3.1)	23.2 (3.6)	22.7 (3.0)	22.5 (3.9)	23.5 (3.5)	18.9 (3.8)*	26.3 (4.3)	21.0 (3.9)
Exercise	26.9 (5.9)	23.5 (6.2)	17.3 (3.5)	18.7 (3.3)	14.6 (2.8)	19.5 (3.2)	12.9 (2.8) [†]	18.0 (2.9)	12.0 (2.4)	18.8 (2.7)
			F(1,26) = 1.117, P = 0.300		F(1,22) = 3.160, P = 0.089		F(1,19) = 5.605, P = 0.029		F(1,17) = 3.411, P = 0.090	
GL										
Control	18.1 (2.8)	20.2 (3.4)	15.6 (2.2)	15.7 (2.6)	13.8 (1.8)	14.4 (2.3)	14.9 (2.2)	13.4 (2.1)	15.5 (2.3)	13.5 (2.1)
Exercise	19.1 (2.9)	15.2 (2.7)*	13.3 (1.7)	13.4 (2.2)	14.7 (2.2)	14.0 (2.3)	19.3 (5.0)	14.6 (2.4)	19.4 (5.1)	15.1 (2.4)
			F(1,27) = 0.212, P = 0.649		F(1,26) = 1.836, P = 0.187		F(1,22) = 1.669, P = 0.206		F(1,17) = 0.240, P = 0.631	

* Significantly different from preintervention (P < 0.05), [†]significantly different from Control group (P = 0.018).



Fig. 4. Top row: proportion of subjects (%) completing each SACM. Middle row: mean \pm SE heart rate (HR) recorded during each simulated air combat maneuver (SACM) pre- (gray) and post- (white) intervention. Horizontal lines indicate a main effect of Group (P < 0.005). Numbers located within each bar indicate the number of subjects that were able to complete that SACM both pre- and post-intervention. Bottom row are boxplots, due to the nonparametric nature of the data, of the rate of perceived exertion (RPE) pre- (gray) and post- (white) intervention for each group during each SACM.

significant effect may be attributed to an insufficient training duration for circulatory adaptations to fully develop.³⁶

Studies which have investigated the effects of aerobic exercise, either through training programs¹⁷ or analysis of aerobically trained individuals,^{22,41} indicate it has no negative effect on RGT or G endurance. However, the prevailing opinion regarding aerobic training in the aviation community is that it will lower overall G tolerance⁴ due to its effects on sympathetic outflow and the renin-angiotensin system,¹³ which have the potential to lower blood pressure.¹⁴ Thus, it is important to emphasize that there were no negative effects noted on any aspect of G tolerance investigated, despite the inclusion of aerobic training components in the ACP. This further highlights that aerobic training is unlikely to lower G tolerance and that it should be included in aircrew training programs given its potential to reduce the effects of fatigue associated with performing the AGSM as well as the overall benefits to pilot health.

To our knowledge no study has assessed SGT using the G_z profiles used here with only one component of the AGSM performed: muscle tensing. We found a tendency toward an improved SGT associated with performance of the ACP, although the upper limit for the SGT profile of +7 G_z, set for ethical reasons, may have limited the efficacy of this measure as 16 subjects were able to tolerate this level of $+G_z$ prior to the intervention. Importantly, during the SGT profile, at $+5.5 \text{ G}_{z}$, a level all participants were able tolerate, a significantly lower HR was observed. As a direct relationship between HR and fatigue during repeated +G_z exposures exists,7 this suggests that by reducing the level of physiological strain, the ACP may be effective in delaying the onset of fatigue.

Importantly, despite a lower HR at this $+G_z$, head-level BP was maintained. While the precise mechanism cannot be determined, an altered baroreflex response or vascular resistance are likely candidates. Sympathetic neural drive, an indicator of altered baroreceptor responsive-

ness, is unaffected by exercise training in healthy populations,^{9,30} while baroreflex responsiveness to lower body negative pressure is similar in resistance trained and untrained individuals.³⁷ Furthermore, during the RGT exposure, BP and HR responses were not altered by the ACP, suggesting baroreceptor responsiveness was unaffected. In contrast, as indicated above, systemic vascular resistance is reduced by resistance training.²⁷ If this were to have occurred as a result of the ACP, it could account for the maintenance of blood pressure despite a reduced HR. Furthermore, as a close association between lower limb muscle mass and compliance of the capacitance vessels exists,¹² any muscle mass gains induced by the ACP might increase systemic vascular resistance. They may also act to reduce vascular pooling through activation of the venosomatic reflex, which induces skeletal muscle contraction in response to venous distension and stretching.³⁹ A reduction in blood pooling in the capacitance vessels of the lower limbs will improve venous return and stroke volume, supporting blood pressure during $+G_z$ exposures.

The reduced HR observed may be related to a lower muscular effort. This was assessed using EMG and, while muscle activation was unchanged in the VL and BF in both groups, there was a significant decline in PF activation in the Ex group, indicating a lower muscular effort required at a given workload. Whether this presents a meaningful improvement and is sufficient to reduce HR is debatable given that the reduction in activity was only 4% of maximal effort. The measurement of bilateral activation and of more muscles such as the gluteal and abdominal muscles would help provide further clarification of this. Resistance training has been reported to improve G endurance (assessed by SACM profiles) by some,^{2,17,38} but not all studies.⁶ The mechanisms responsible for the proposed improvement are poorly understood. Neuromuscular and biochemical adaptations to resistance training^{20,25,31} are expected to improve fatigability and the ability to repeatedly contract muscle during $+G_z$ exposure⁸ and would be beneficial when routinely performing the fatiguing AGSMs, and this could subsequently reduce its effectiveness.¹⁹ Tesch et al.³⁸ have suggested that improved motor unit synchronization and recruitment of high threshold motor units may play an important role in improved G endurance following training. In contrast, Epperson et al.¹⁸ speculated that resistance training could improve the efficiency of muscular contraction (i.e., lower the percentage of maximum contractile force required to maintain vision), thereby improving G endurance.

The effects of exercise training on G endurance have typically been assessed by performing SACM profiles until exhaustion. A modified version of this was completed in the present study, where four distinct SACMs were performed, allowing a more detailed analysis. Our findings revealed that the proportion of individuals completing all four SACMs was greater following the ACP. This, however, did not translate into a difference in any of the markers (HR and muscle activation) used to objectively assess performance during comparable points during each SACM. Furthermore, RPE was unchanged, which is unsurprising given the close relationship between RPE and HR.^{5,32} The reduced HR observed during the SGT profile in the Ex group would be anticipated to reduce fatigue development,⁷ although this did not translate to an improved SACM performance.

It is important to note that while frequent exposure to $+G_z$ acceleration can cause physiological adaptations and potentially improve G tolerance,^{11,33} the participants did not perform any high $+G_z$ flying during the study period. As such any differences noted between groups cannot be explained by the type of flying or training the participants received during the study period. Therefore, the additional improvements in the Ex group noted in the current study can be attributed to the ACP as opposed to other training related factors. G endurance is governed both by an individual's RGT and the effectiveness/efficiency of a person's AGSM,²² which can vary markedly between individuals.^{10,24} The relative inexperience of the current subject cohort to $+G_z$ and the AGSM may, therefore, have prevented any ACP-induced benefits to G tolerance from being observed. Greater AGSM training may be required to learn this unnatural and complex maneuver⁴ to ensure an effective performance. A reduced effectiveness compared with more experienced aircrew is indicated by the blood lactate measures measured post-SACM (Ex and Con 5.5 to 6.1 mmol \cdot L⁻¹, respectively). These values are higher than those measured in more experienced aircrew after performing similar SACM profiles to exhaustion (4.2 and 5.2 mmol \cdot L⁻¹),^{1,8,42} suggesting a greater physical demand on the current subjects despite not performing the task until exhaustion.

While the primary focus of the current study was to assess the effect of the ACP on G tolerance, it should be noted that the ACP was also designed to reduce strain injuries to the neck³⁴ and to contribute to overall health. Prior to the ACP RAF aircrew did not perform a structured exercise program. In this short-term study it is not possible to assess the ACP's effectiveness in regard to injury prevention, rather a detailed analysis of injury rates recorded after a sufficiently long period since the implementation of the ACP will be required.

The present study had some limitations. Firstly, the SGT profile was limited to a maximum of $+7 G_{z}$, due to ethical considerations relating to the $+G_z$ level aircrew would typically be exposed to when using the anti-G system in the present study, potentially reducing the efficacy of this measurement as 16 subjects were able to achieve this level before the intervention. Given the number of subjects capable of reaching this level, future studies should increase this limit as the profile used was well tolerated. Secondly, group allocation was not randomized but had to be based on where aircrew were in their training due to the difficulties in performing a supervised exercise program at a wide range of locations within the United Kingdom. This also explains the slightly younger age of the Con group as they were at a slightly earlier career point, and that the subjects were relatively inexperienced in terms of $+G_z$ exposures and AGSM. It is possible that having a more experienced subject pool with greater familiarity of the AGSM could influence the results. Thirdly, no women entered the BFJ or EFT training courses over the 2-yr period of the study and all subjects were men. Fourthly, it was not possible to record reliable BP values during the SACM due to significant artifact in the signal occurring from performance of the AGSM. Finally, the ACP was only performed 2 d/wk rather than 3 as would often be recommended for training programs. This is was due to the time constraints and logistical challenges associated with performing supervised exercise sessions in serving military personnel.

Exposure to high $+G_z$ is physically demanding and has the potential to result in G-LOC. The role of physical conditioning programs to aid performance under $+G_z$ and improve G tolerance is not completely understood. Here we have shown that the ACP does not negatively impact relaxed G tolerance, can reduce the physiological strain associated with a given $+G_z$, and

has the tendency to improve an individual's SGT, possibly as a result of an increased systemic vascular resistance. In the population studied these improvements did not translate to greater G endurance, as assessed by SACM profiles, although a greater proportion of individuals were able to complete the SACM profiles after performing the ACP. The results of the study indicate that the ACP is a suitable conditioning program for use by the fast jet aircrew community to improve aircrew performance in a controlled high $+G_z$ environment by reducing the physical workload associated with a given $+G_z$ level.

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