Hypoxia-Like Events in UK Typhoon Aircraft from 2008 to 2017

Desmond M. Connolly; Vivienne M. Lee; Amanda S. McGown; Nicholas D. C. Green

INTRODUCTION: Recent reports of in-flight, hypoxia-like events have prompted concern that aircraft life support systems (LSS) may not always provide effective altitude protection. An analysis was undertaken of hypoxia-like incidents reported in a UK front-line combat aircraft.

METHODS: A search of the UK Aviation Safety Information Management System database identified all Typhoon Defense Air Safety Occurrence Reports (DASORs) notifying in-flight symptoms over the decade 2008–2017. Qualitative analysis focused on the event narrative, altitude profile, timeline, symptom description, sortie characteristics, LSS function, postflight engineering investigation, and training implications. The plausibility and likelihood of hypobaric hypoxia were assessed, and the probable cause of symptoms ascribed.

RESULTS: There were 18 DASORs with notified symptoms of suspected in-flight hypoxia, 13 in solo pilots and 5 reports of symptoms affecting 7 of 10 aircrew in 2-seat aircraft. Two cases of probable hypoxia comprised one oxygen bottle failure and one mask-off cabin depressurization. In one report, hypoxia was assessed as plausible but unlikely, following birdstrike with failure of cabin pressurization during climb. Symptoms were explained by hyperventilation in 13 cases (65%) and twice by minor constitutional upset. Suspected hypoxia was managed by immediate selection of emergency oxygen and expedited descent in 10 of 18 occurrences (56%).

CONCLUSIONS: Only 2 cases of probable hypoxia have been reported in over 150,000 Typhoon flying hours. The Typhoon LSS has provided effective altitude protection including during cases of cabin depressurization. Symptom occurrences in Typhoon are idiosyncratic and unrelated; hyperventilation probably accounts for two-thirds of reports.

KEYWORDS: physiological events, hypobaric hypoxia, hyperventilation, altitude, Typhoon.

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In recent years, reports of in-flight, hypoxia-like symptoms have prompted concern that aircraft life support systems (LSS), and often those using On-Board Oxygen Generation Systems (OBOGS), may not be providing the required altitude protection.[7,17] In this context, an analysis was undertaken of hypoxia-like incidents reported in a UK front-line combat aircraft.

Military aircrew undertake practical training for familiarization with the subtle, nonspecific, often idiosyncratic and highly variable symptoms and signs of hypobaric hypoxia, a perennial physiological hazard at altitude.[4,12] Aircrew noticing similar symptoms during flight should suspect hypoxia as the cause and take urgent remedial action (emergency oxygen and descent). However, symptoms may develop during flight from numerous other causes, such as hyperventilation without underlying hypoxia,[9,10] effects of pressure change (e.g., ear/sinus discomfort, alternobaric vertigo),[5,21] decompression sickness (DCS),[5] acceleration atelectasis,[18] spatial disorientation and motion sickness. Symptoms could result from equipment failure (e.g., hypercapnia due to rebreathing of expired gas; contamination of breathing gas),[15] imposed breathing resistance or increased work of breathing (e.g., restrictive garments; mask valve faults) or from unrelated insults (e.g., dehydration from fluid restriction; hypoglycemia from a missed meal). Pathological symptoms might develop de novo during flight (e.g., spontaneous pneumothorax

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or angina pectoris)[1] as may delayed manifestations of preflight activities (e.g., positional alcohol nystagmus).[8]

Symptoms arising during flight are often mild, nonspecific and transient, leaving no residual evidence and without a clear underlying cause. Such incidents have been characterized recently as "unexplained physiological events," based largely on the lack of objective evidence of cause. Diagnosis must rely heavily on the narrative history of the event, the results of engineering investigations of LSS and pilot flight equipment, and any deductions that may be inferred. Multiple symptoms might point toward particular causes, but each report can only be judged qualitatively, with possible explanations either more or less likely to account for the events described. It may be impossible to establish or exclude any specific cause with certainty. Often, considerable ambiguity must remain, but this is not new. Past reviews of in-flight hypoxia acknowledge that many are unexplained, suggesting that some may result from transient equipment faults while others may be due to hyperventilation without underlying hypoxia.[3,19]

The aims of this work were to review all reports of in-flight, hypoxia-like symptoms in the Eurofighter Typhoon, over 10 yr of aircraft multirole operation, to establish those likely to be due to hypoxia and to assess the probable cause of nonhypoxia events, thereby quantifying the incidence of relevant aeromedical hazards. The in-flight management of suspected hypoxia was reviewed and factors promoting in-flight symptoms in the Typhoon considered, encompassing the aviation environment, aircraft operation and serviceability of the Typhoon LSS.

Evaluation of the reports requires some understanding of the function of the Typhoon LSS, the basic configuration of which is shown in the schematic in **Fig. 1**. Engine bleed air to the Environmental Control System (ECS) enables cabin pressurization and supplies a Molecular Sieve Oxygen Concentrator (MSOC), which adsorbs and purges nitrogen to deliver oxygen-rich breathing gas. Product gas oxygen content is monitored by two independent sensor systems and varies with MSOC cycle time, adjusted automatically with reference to cabin pressure. As with other OBOGS systems, oxygen content may fall with reduced inlet pressure (e.g., "throttle idle") or increased ventilatory demand (e.g., in two-seat variant aircraft). A water separator prevents sieve contamination that could degrade performance by blocking nitrogen adsorption.

Air is also supplied to the anti-G valve (AGV) of the aircrew services package (ASP) to inflate the anti-G trousers (AGT), while MSOC product gas passes to a pressure-demand regulator in the ASP to provide breathing gas to the pilot. The ASP can deliver pressure breathing for both altitude and 'G' protection, so breathing gas is routed via a compensated dump valve (CDV) to inflate a chest counter-pressure garment to match breathing pressure ($\pm$ 10 mmHg).

The pressurization schedule (**Fig. 2A**) limits the cabin to 8000 ft pressure altitude (PA) until aircraft altitude exceeds 23,100 ft, thereafter maintaining a differential pressure of 34.5 kPa (5.0 psi). The system is designed to maintain oxygenation at least equivalent to breathing air at sea level but is enhanced at cabin altitudes above 15,000 ft to prevent severe hypoxia in the event of rapid cabin decompression. However, oxygen content is limited to maximum 60% at cabin altitudes up to 15,000 ft to avoid acceleration atelectasis. Thus, there is a specification 'bottleneck' at cabin altitudes from 15,000 to 20,000 ft (**Fig. 2B**).

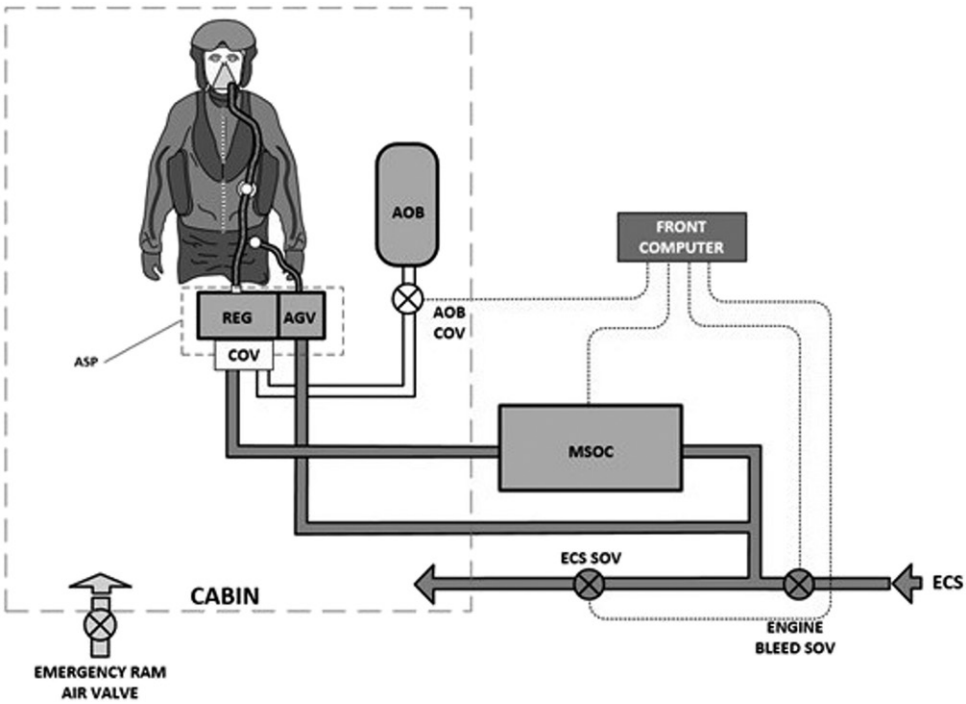

Fig. 1. Typhoon Life Support System overview. ASP: Aircrew Services Package, comprising REG: breathing gas oxygen regulator and AGV: anti-G valve; AOB: auxiliary oxygen bottle; COV: changeover valve; MSOC: molecular sieve oxygen concentrator; ECS: environmental conditioning system; SOV: shut off valve.

When maneuvering in this "bottleneck" range MSOC cycle responsiveness may lag slightly behind changes in cabin pressurization, resulting in transient dips below the lower oxygen limit. Dips into the "buffer zone" are allowed for up to 10 s but at no time is oxygen content allowed to fall below the absolute lower limit equivalent to breathing air at 5000 ft (Fig. 2B). Prolonged or unacceptable dips trigger automatic selection of 100% oxygen from the Auxiliary Oxygen Bottle (AOB), mounted on the ejection seat. The front computer operates change over valves (COV) to select and deselect the AOB to augment MSOC function when required (Fig. 1). The AOB includes a "reserve" of 70 L designated Emergency Oxygen (EO). An amber [MSOC] caption illuminates on the display warning

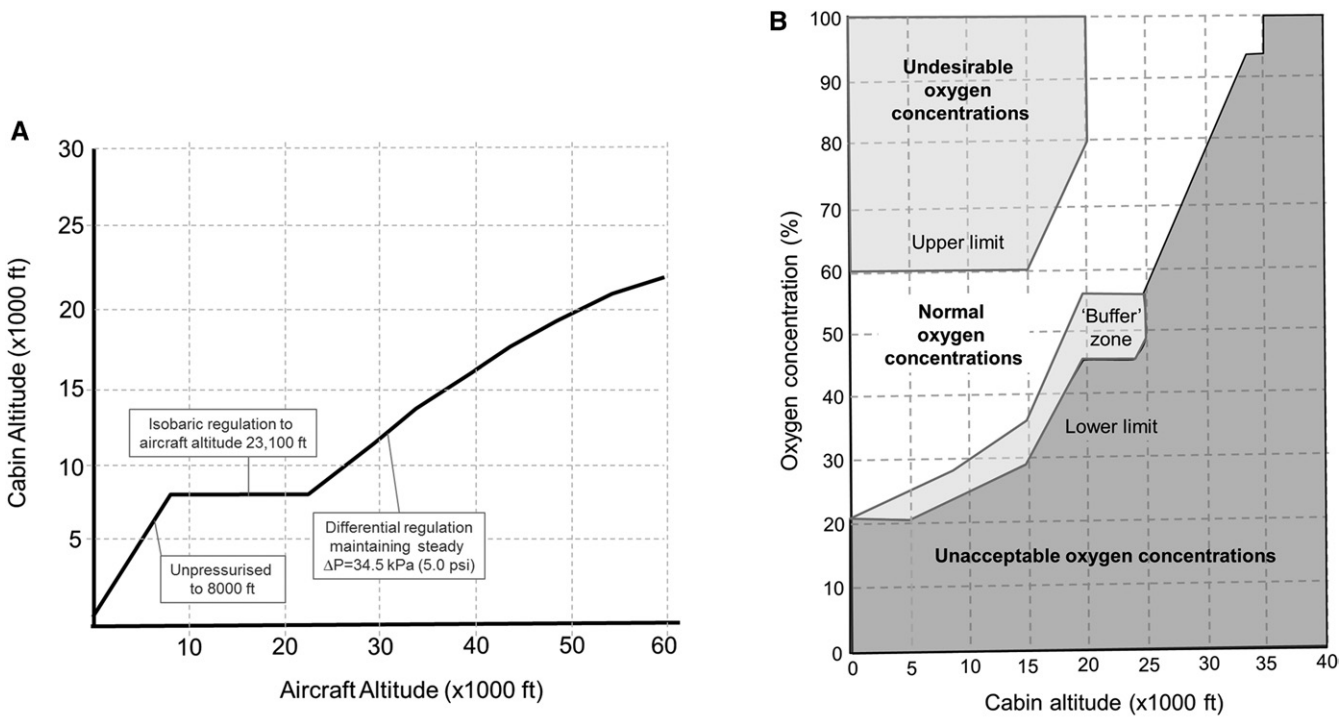

Fig. 2. A) Typhoon cabin pressurization schedule; B) intended Typhoon breathing gas oxygen concentration by cabin altitude.

panel if the AOB is selected; if this does not de-select automatically within 60 s then an oxygen system failure is diagnosed. If AOB contents deplete to 70 L then a red [OXY] caption indicates utilization of EO. The AOB may be operated manually but cannot be de-selected after automatic selection if a fault condition remains. The system is designed always to "fail safe" with default AOB selection. System function is recorded and can be reviewed after the flight.

METHOD

Subjects

In the UK, incidents generating concern over military aviation safety, including suspected hypoxia, are notified by raising Defense Air Safety Occurrence Reports (DASORs). The findings of resulting investigations are managed using the Military Aviation Authority Aviation Safety Information Management System (ASIMS), which includes a searchable database. Reports may be raised anonymously. In the current study, all reports were raised by an aviator directly involved in the in-flight occurrence, usually the individual experiencing symptoms. The database is used routinely for retrospective research and analysis, without a priori consent. The current study did not obtain personal, biometric or physiological data and ethical approval was not required. Approval for publication was provided by the relevant Defense authority.

Procedure

A search of the ASIMS database was undertaken using standard Boolean terms to identify all Typhoon reports, raised over the decade from January 2008 to December 2017 inclusive, containing any of the following words or word-stems in the free text 'Brief Title' or 'Narrative': hypo; hyper; nausea; nauseous; sick; ill; dizzy; dizziness; light-headedness; pain; ache; breath; neck; back; chest; tingling; numbness; pins and needles; disorientation; vision; visual. Variations were allowed for, including hyphens and spaces, e.g., 'lighthead', 'light head' or 'light-head'. Stems were used where two or more words could contain the same root, e.g., "hypoxia" or "hypoxic", so that no relevant DASORs would be omitted. Given the potential diversity of symptoms, several searches were conducted to ensure that no symptomatic events were overlooked. Irrelevant records were excluded, e.g., those where a search term appeared in an unrelated context (e.g., "ache" in "detached"), or where a search term appeared in a negative reference (e.g., "at no time were symptoms of hypoxia apparent").

Statistical Analysis

Some descriptive statistics are used to report essentially qualitative analysis. Reports were reviewed independently by three subject matter experts in aviation medicine or physiology, each with over 25 yr of experience including the conduct of Typhoon LSS evaluation for altitude and G protection. Analysis focused on the narrative, altitude profile, event timeline, symptom description, LSS function, sortie characteristics, postflight engineering investigation and implications for aircrew training. This information was collated from each report using a common template.

The likelihood of "significant" hypobaric hypoxia was assessed (with "significant" defined as "sufficiently severe or prolonged to explain symptoms arising under the circumstances described")

and the likeliest cause of symptoms proposed. Since this cannot be entirely unambiguous, the probability of hypoxia, relative to other possible causes, was defined as follows:

- REMOTE—Hypoxia is implausible.
- UNLIKELY—Hypoxia is unlikely; another cause is more likely.
- PROBABLE—Hypoxia is plausible and most likely.
- INDETERMINATE—Hypoxia is unlikely but no more plausible cause exists.

There was general agreement between all three subject matter experts on the majority of reports, both for the likelihood of hypoxia and most likely explanation of symptoms. For the minority for which there was some initial uncertainty, consensus was easily achieved during three-way discussion following explanation of the rationales underlying individual opinions. All authors agree on the outcomes being presented and have reviewed the manuscript.

RESULTS

In total, 18 reports of suspected in-flight hypoxia were submitted over the decade from 2008 to 2017, involving 20 symptomatic pilots, 13 flying solo and the others in 2-seat aircraft with dual occupancy. Thus, in two dual-occupancy aircraft, both pilots were symptomatic. The reporting period covered 152,112 flying hours (fh), giving an incidence of 11.8 reports per 100,000 fh (1 per 8475 fh). The number of reports is shown relative to number of flying hours each year in **Fig. 3**.

Despite increasing Typhoon flying hours, suspected hypoxia occurrences are infrequent, averaging less than 1 per year since a small reporting spike in 2012. The incidents in 2012 were unrelated, occurring at different times during the year and affecting different individuals in different aircraft, on different squadrons and at different operating locations, with none rated higher than UNLIKELY to be due to hypobaric hypoxia. There were no aspects in the report narratives to suggest a common cause and there was no apparent operational or engineering explanation for the reporting spike, or change in equipment or practice.

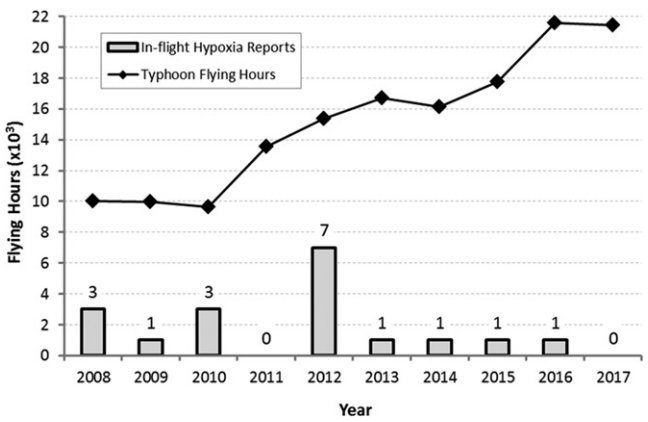

Fig. 3. UK in-flight hypoxia-like occurrences and Typhoon flying hours 2008-2017, encompassing the progressive introduction to service of aircraft with multirole capability.

Only 8 of 18 reports (44%) volunteered details of the symptoms interpreted as possible hypoxia (**Table I**). Six of these (75%) described "tingling" sensations with three (37.5%) reporting feeling dizzy, tipsy or light-headed. There were individual reports (12.5%) of feeling cold, uneasy, shallow breathing, flush or headache. The remaining 10 reports referred to unspecified "symptoms of hypoxia" or similar generic description.

The likeliest explanation for many of these symptoms is hyperventilation (hypocapnia). Characteristically, this causes paresthesia (tingling) in the extremities and in the lips, dizziness/light-headedness, a sensation of breathlessness and unease/anxiety, enough to account immediately for seven of the eight reported symptom complexes. Of course, this does not preclude hypoxia as an underlying cause. The description in Serial *h* of "rapid onset of a flush feeling and slight headache" excludes neither hypoxia nor hyperventilation but is also consistent with other causes such as hypercapnia (e.g., from rebreathing carbon dioxide) or carbon monoxide poisoning. Many narratives either stated or implied that symptoms resolved on oxygen and/or following descent, neither of which mandate hypoxia as the cause. In one instance a headache and "fuzzy feeling" persisted for a few hours after landing, while, in another, symptoms recurred briefly after landing but quickly resolved. Overall, in-flight "hypoxia-like" symptoms in Typhoon aircraft may be characterized as acute (abrupt onset), transient, responsive to immediate action drills and unlikely to persist.

Hypoxia was assessed as PROBABLE in just 2 of the 18 reports, affecting 2 (9%) of the 23 aircrew involved in these incidents. Hypoxia was assessed UNLIKELY in 12 (52%), REMOTE (implausible) in 7 (30%), and INDETERMINATE in 2 (9%) symptomatic aircrew in the same aircraft. Symptoms were not described by the aircrew involved in any of the PROBABLE or INDETERMINATE occurrences.

Only one pilot flying solo experienced PROBABLE hypoxia (risk likelihood 7.5×10^{-6} per solo flying hour). He removed his oxygen mask to investigate a microphone fault just as the cabin depressurized coincidentally due to mechanical (valve) failure. He experienced hypoxia breathing ambient cabin air at an equivalent 24,700 ft PA. The other pilot who experienced PROBABLE hypoxia was the rear seat occupant in a two-seat aircraft returning to base at an aircraft altitude of 42,000 ft

Table I. Specific Symptoms Volunteered in Eight Hypoxia-Like Typhoon Incident Reports.

SERIAL	SYMPTOMS DESCRIBED
a	*"cold and tipsy with tingling finger tips"*
b	*"tingling sensation over my whole body, predominantly in my arms and legs"*
c	*"slightly dizzy and uneasy"*
d	*"very rapid….tingling sensation in his hands and lower extremities"*
e	*"mild tingling was felt in the fingertips"*
f	*"tingling fingers, light headedness and shallow breathing"*
g	*"tingling lips"*
h	*"rapid onset of a flush feeling and slight headache"*

(cabin altitude 17,500 ft) when both AOBs were selected automatically for reasons which were undetermined. Both aircrew developed symptoms, but while the rear seat pilot was breathing from a faulty and empty AOB, the front seat pilot's AOB was feeding normally and the likelihood that he experienced hypoxia is REMOTE. Both are likely to have experienced symptoms of hyperventilation. In the rear seat pilot this is likely to have been secondary to hypoxia and possibly also increased breathing resistance.

There was one INDETERMINATE occurrence in a two-seat aircraft. Both pilots noticed transient symptoms at top of climb to 30,000 ft in an unpressurized cabin, with automatic AOB selection when cabin altitude exceeded 25,000 ft. Postflight investigation revealed a birdstrike had occurred; in this incident, hypoxia was unlikely but cannot be discounted entirely. For hypoxia to have occurred, the birdstrike would have had to cause fast decompression with suboptimal oxygen delivery (ECS malfunction, failure of pressurization and erratic MSOC function in the "buffer zone" (see Fig. 2B) with product gas oxygen not falling sufficiently low to trigger automatic AOB selection).

One report assessed as UNLIKELY is worth mention. An experienced nonhandling pilot, during an undemanding phase of flight, experienced minor symptoms consistent with hypoxia training (tingling lips) at a cabin altitude of 17,000 ft. Cabin pressurization and LSS functions were normal and the AOB was not selected. Upon planned descent the symptoms resolved spontaneously at a cabin altitude of around 8000 ft. During postflight investigation ASP and MSOC functional tests were passed but a persistent (covert) control/monitoring fault only cleared after replacement of both oxygen monitors and a computer. Significant hypoxia is UNLIKELY as LSS function was satisfactory throughout. Hyperventilation is a possible cause but there is no compelling explanation for this event.

Eight reports (44%) were associated with gradual loss of cabin pressurization, with three due to ECS failures (one secondary to birdstrike) and five due to pressurization valve failures. Maximum cabin altitudes ranged from 24,700 ft to 38,000 ft PA. The LSS performed reliably during all these events, delivering 100% oxygen as indicated and always failing safe when faced with sporadic, idiosyncratic system faults.

Five of the 18 reports (28%) concerned 2-seat aircraft with dual occupancy, disproportionately high given that 2-seat aircraft account for only 12% of the fleet's flying hours. Two of these resulted in PROBABLE or INDETERMINATE hypoxia affecting one or both occupants, but this apparently increased incidence is misleading given the low numbers and idiosyncratic nature of the events. These occurrences were not related to ventilatory demand on the MSOC.

Nine reports (50%) were associated with flight at cabin altitudes within the "bottleneck" region of the cabin pressurization schedule (Fig. 2B). Excluding the birdstrike incident, there were no occurrences to suggest that dips into the "buffer zone" were associated with insufficient oxygen delivery.

Two solo reports are attributed to nonspecific constitutional upsets that appeared minor and self-limiting, with symptoms possibly precipitated by the flight environment (but not suggestive of DCS). Finding just two such incidents, over a decade of Typhoon flying, is a remarkably low incidence rate and it is unrealistic to expect that no such incidents should ever occur.

Hyperventilation is considered the likeliest explanation for 13 of 20 reports of in-flight symptoms (65%), including 10 of 13 aircrew flying solo (77%), often associated with faster than expected AOB depletion. Some examples will help to illustrate why hypoxia was discounted and hyperventilation was attributed as the cause.

- *Example 1.* A transient MSOC warning caption (signifying AOB selection) on takeoff was noted to have recurred at top of climb, by which time AOB contents had depleted by 50%, suggesting a high rate of consumption. Cabin pressurization and the LSS were functioning normally so 100% oxygen was being supplied to the pilot. Symptoms suggestive of hypocapnia developed while instigating recovery actions, including feeling cold, "tipsy," and tingling fingers.
- *Example 2.* At cabin altitude 10,500 ft, a computer fault caused intermittent AOB selection and eventual depletion whereupon "initial symptoms of hypoxia" were experienced before descent was initiated. The pilot probably inspired ambient cabin air, against resistance, through the mask anti-suffocation valve. With the apparent urgency of the situation, this is likely to have promoted hyperventilation.
- *Example 3.* Pressurization valve failure during climb at 34,000 ft resulted in cabin decompression with increased noise, a sensation of "ears popping" and "beginning of hypoxia symptoms". However, the LSS functioned normally with immediate automatic AOB selection delivering 100% oxygen. This was maintained until well into recovery of the aircraft at low level and meaningful hypoxia is unlikely at any time, whereas hyperventilation was a likely response to this unpleasant in-flight incident.
- *Example 4.* After establishing level flight at 43,000 ft for 30 min, a student handling pilot in a 2-seat aircraft experienced rapid onset of bilateral, symmetrical paresthesiae in both upper and lower limbs, interpreted as "moderate symptoms of hypoxia". Cabin pressurization and the LSS functioned normally throughout so the pilot was not hypoxic at any time. Hyperventilation is the most likely explanation.
- *Example 5.* While maneuvering at 41,000 ft the MSOC caption illuminated indicating automatic AOB selection, by design. "Mild tingling was felt in the fingertips" and descent was initiated. The OXY caption illuminated after about 5 min indicating AOB depletion to 70 L and a high rate of consumption of 100% oxygen consistent with hyperventilation, resolving spontaneously with further descent.
- *Example 6.* During a dark night transit over water at 42,000 ft (cabin altitude 17,500 ft) a red OXY caption indicated AOB depletion to 70 L, with no prior MSOC captions to signify earlier AOB selections due to a wiring fault identified postflight. The report notes "…within seconds there were symptoms of hypoxia: tingling fingers, light-headedness and shallow breathing" consistent with hyperventilation; hypoxia

is not credible due to continuing supply of 100% oxygen. Other factors, including delayed air traffic clearance to descend, compounded another unpleasant in-flight incident.

DISCUSSION

The incidence of in-flight symptoms in Typhoon is low (11.8 per 100,000 fh) and the likelihood of symptoms being attributable to hypoxia is even lower, with just 2 of 20 symptomatic aircrew experiencing PROBABLE hypoxia. As in previous reviews, many narratives equated the symptoms experienced during flight with those of past hypoxia familiarization training, supporting the benefit of such training and implying hypoxia and/or hyperventilation as the likely underlying cause.[3,19] Pilots experiencing provocative in-flight symptoms will be unable to discriminate whether they are due primarily to hypoxia or hyperventilation. Instead, they should always interpret and manage suggestive symptoms as suspected hypoxia.[16]

The importance of responding immediately and instinctively to suspected hypoxia cannot be overstated; the immediate action drills are selection of emergency oxygen (in Typhoon, manual AOB selection) and descent to a safe altitude (10,000 ft PA ambient or below). Only 10 of 18 incidents (56%) were managed in this way. In the rest, either manual AOB selection did not occur or was not reported, or descent was delayed or was interrupted unnecessarily. No criticism is intended of how aircrew managed these incidents; their task was to manage in moments events that months of later review cannot explain without ambiguity. Nonetheless, there is scope to reinforce the correct immediate in-flight management of suspected hypoxia, i.e., always conduct emergency oxygen drills and expedite descent.

In healthy military aircrew, in-flight symptoms may be described as physiological events if they result from normal responses to an environmental stressor. This obliges recognition that removal of the stressor will result in spontaneous complete recovery, leaving no residual evidence of abnormality. Thus, subsequent postflight investigation will rarely find an underlying fault with either the aircraft or the aviator. Explanation must then depend on careful review of the narrative of the event and detailed inquiry into the underlying circumstances.

Recent reports attest to the benefit of hypoxia training whereby prior symptom familiarization enables aircrew to recognize later occurrences.[20,22] This training typically imposes severe hypoxia equivalent to breathing air at pressure altitudes up to 25,000 ft, promoting secondary hyperventilation and symptoms of hypocapnia that will become an integral part of the hypoxia experience. Such symptoms will be subjectively indistinguishable from those due to hyperventilation without underlying hypoxia. For a concise review of the physiology of hypocapnia, see Laffey et al.,[14] and for consideration of the relative influence of hypoxia and hypocapnia on cerebral perfusion, oxygenation and cognition, see Friend et al.[6] It is perhaps inevitable then that many occurrences of suspected hypoxia will be considered, with the benefit of hindsight, to be due to hyperventilation.[3,19] In-flight hyperventilation remains poorly researched with little progress made since Gibson's 1979 review and later case reports.[9,10] Observed changes in patterns of aircrew pulmonary ventilation remain largely anecdotal and inadequately explained. However, the propensity to hyperventilate should be expected to be highly variable both between and within individuals.[13]

In particular, hyperventilation and its consequences during flight in agile aircraft remain poorly understood. Numerous factors associated with the flight environment, far more likely than hypoxia, will promote it, as indicated in **Table II**. Many of these are likely to be present during flight in highly agile aircraft and yet, fortunately, symptomatic occurrences remain relatively uncommon.

The normal partial pressure of carbon dioxide in mixed expired gas is ~27 mmHg (~3.6 kPa). In 1957, Balke et al. collected samples of mixed expired gas from aircrew during flight when training on various fast jet aircraft types, recording values ranging between about 10 and 45 mmHg (1.3 to 6.0 kPa).[2] Although these values will under-estimate alveolar partial pressures, hypocapnia should be expected to occur commonly during flight in agile aircraft and carbon dioxide levels should be expected occasionally to fall low enough to promote symptoms. On the other hand, some pilots may tend to retain carbon dioxide through hypoventilation when using some breathing systems, suggesting an influence of imposed external breathing resistance, itself known to promote hyperventilation in a minority of individuals. Lastly, if the data are considered in normalized form, presented as proportions of all measurements

Table II. Some Factors That May Promote Hyperventilation in Military Aviation.

FACTOR	EXAMPLES
Helmet/mask equipment	Unfamiliarity; poor fit; pressure points; discomfort
Inspiratory resistance	'Sticky' oxygen mask inspiratory valve; demand regulator 'lag'
Expiratory resistance	Stuck open inspiratory valve with compensated expiratory valve; regulator 'overboost'
Increased breathing pressure	Excessive 'safety' pressure; positive pressure breathing
Increased work of breathing	Anti-G trouser inflation; constrictive aircrew clothing; tight harness
Workload	Cognitive demand; pace of events; cockpit warnings/alarms
Hypoxemia	Sustained $+G_z$ acceleration; atelectasis
Sustained $+G_z$ acceleration	Oxygen debt; fatigue; 'heavy' chest wall; vestibular stimulation
Thermal stress	Heat; cold (immersion); pyrexia
Vibration	Turbulence; cabin pressure fluctuations
Motion effects	Spatial disorientation; motion sickness
Drugs / hormones	Medication; alcohol; progesterone (ovarian cycle)
Stress	Mission priority; operational factors; training stress; personal factors
Anxiety / fear	Combat; fear of flying
Hypobaric hypoxia	Being above 10,000 ft PA without a functional life support system

made in each aircraft type, the curves vary somewhat between types, suggesting that ventilatory responses vary systematically between aircraft and their breathing systems while recognizing that these data will also reflect differences in the corresponding pilot cohorts (e.g., mean age) and in the sortie profiles associated with different stages of training. It therefore appears perfectly feasible to characterize patterns of ventilatory response between aircraft types, and, indeed, Harding went some way to achieving this for the Hawker Hunter T Mk 7 aircraft.[11] It may therefore be possible to establish the likelihood of experiencing symptomatic hypocapnia (or, conversely, unacceptable carbon dioxide retention) with different flight profiles and to prepare and train aircrew accordingly. This might reasonably encompass control of rate and depth of breathing in response to symptom occurrence, and the correct conduct of immediate actions in response to hypoxia-like symptoms, i.e., platform-specific emergency drills.

The potential relevance of in-flight physiological monitoring warrants consideration. No aircraft or lives were lost due to hypoxia as a result of the events reported here, so monitoring would have offered no benefit with respect to enhancing outcomes in this context. On the other hand, it is (debatably) unlikely that monitoring would adversely affect outcomes while it might potentially have enabled more effective in-flight management of physiological occurrences. In the latter respect, monitoring of capnic state could be regarded as important as monitoring of oxygenation state. At the very least, in-flight monitoring would greatly facilitate accurate retrospective evaluation of in-flight physiological events, but would require redundant monitoring of nearly 8500 fh in the Typhoon to provide data on a single symptomatic occurrence, and 75,000 fh to record an episode of hypoxia.

There are obvious limitations to the current study. In the absence of hard data, retrospective review has relied heavily on the pilots' subjective recall and descriptions of events and the reviewers' later interpretation of the available, often incomplete, information. Inherently, therefore, such review will be susceptible to bias and prone to error. Even with retrospective benefits of time, reflection, discussion, and lengthy deliberation, it is challenging to differentiate the true nature of some events with confidence. It has been necessary to generalize when providing an overview, accepting that individual events may remain contentious and subject to debate.

In summary, over the decade from 2008 to 2017 there were just two occurrences of symptoms assessed as likely to be due to in-flight hypoxia in UK Typhoon aircraft (one event every 75,000 fh). Hyperventilation is the likely explanation for 13 of 20 (65%) symptom reports over the reporting period (one event per 11,700 fh). Of the 18 occurrences of suspected in-flight hypoxia, only 10 (56%) were managed by immediate manual selection of emergency oxygen and expedited aircraft descent. There is therefore scope to enhance in-flight management of suspected hypoxia and ground-based hypoxia training to reinforce platform-specific immediate action drills, and possibly also understanding and management of in-flight hyperventilation. Other than a single episode of hypoxia attributable to a faulty AOB in 2008, the Typhoon life support system has provided altitude protection effectively, in accordance with its design, including in response to loss of cabin pressurization.

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REFERENCES

1. Al-Janabi F, Mammen R, Karamasis G, Davis J, Keeble T. In-flight angina pectoris; an unusual presentation. BMC Cardiovasc Disord. 2018; 18(1):61.
2. Balke B, Wells JG, Clark RT. In-flight hyperventilation during jet pilot training. J Aviat Med. 1957; 28(3):241–248.
3. Cable GG. In-flight hypoxia incidents in military aircraft: causes and implications for training. Aviat Space Environ Med. 2003; 74(2):169–172.
4. Deussing EC, Artino AR Jr., Folga RV. In-flight hypoxia events in tactical jet aviation: characteristics compared to normobaric training. Aviat Space Environ Med. 2011; 82(8):775–781.
5. Files DS, Webb JT, Pilmanis AA. Depressurization in military aircraft: rates, rapidity, and health effects for 1055 incidents. Aviat Space Environ Med. 2005; 76:523–529.
6. Friend AT, Balanos G, Lucas S. Isolating the independent effects of hypoxia and hyperventilation induced hypocapnia on cerebral haemodynamics and cognitive function. Exp Physiol. 2019; 104(10):1482–1493.
7. Gertler J. Air Force F-22 Fighter Program. Congressional Research Service Report For Congress 7-5700, October 2012.
8. Gibbons HL. Alcohol, aviation, and safety revisited: a historical review and a suggestion. Aviat Space Environ Med. 1988; 59:657–660.
9. Gibson TM. Hyperventilation in aircrew: a review. Aviat Space Environ Med. 1979; 50(7):725–733.
10. Gibson TM. Hyperventilation in flight. Aviat Space Environ Med. 1984; 55(5):412–414.
11. Harding RM. Human respiratory responses during high performance flight. Advisory Group for Aerospace Research and Development AGARDograph No. 312. Neuilly sur Seine (France): NATO Science & Technology Organisation; November 1987.
12. Johnston BJ, Iremonger GS, Hunt S, Beattie E. Hypoxia training: symptom replication in experienced military aircrew. Aviat Space Environ Med. 2012; 83(10):962–967.
13. Karavidas MK, Lehrer PM. In-flight hyperventilation among airline pilots. Aviat Space Environ Med. 2009; 80(5):495–496.
14. Laffey JG, Kavanagh BP. Hypocapnia. N Engl J Med. 2002; 347(1):43–53.
15. Law J, Watkins S, Alexander D. In-flight carbon dioxide exposures and related symptoms: association, susceptibility, and operational implications. Houston (TX): NASA Johnson Space Center; 2010. NASA Technical Paper TP–2010–216126.
16. Petrassi FA, Hodkinson PD, Walters PL, Gaydos SJ. Hypoxic hypoxia at moderate altitudes; review of the state of the science. Aviat Space Environ Med. 2012; 83(10):975–984.
17. Phillips JB, Horning DS, Dory RE. A comparison of pulse-oximetry, near-infrared spectroscopy (NIRS), and gas sensors for in-cockpit hypoxia detection. Dayton (OH): Naval Medical Research Unit – Dayton; 2012.

Technical Memorandum 12-60. Available from: https://apps.dtic.mil/dtic/tr/fulltext/u2/a571028.pdf.
18. 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. 2021; 106(1):18–27.
19. Rayman RB, McNaughton GB. Hypoxia: USAF experience 1970–1980. Aviat Space Environ Med. 1983; 54(4):357–359.
20. Smith AM. Hypoxia symptoms in military aircrew: long-term recall vs. acute experience in training. Aviat Space Environ Med. 2008; 79(1):54–57.
21. Subtil J, Varandas J, Galrão F, dos Santos A. Alternobaric vertigo: prevalence in Portuguese Air Force pilots. Acta Otolaryngol. 2007; 127(8):843–846.
22. Woodrow AD, Webb JT, Wier GS. Recollection of hypoxia symptoms between training events. Aviat Space Environ Med. 2011; 82(12):1143–1147.