F/A-18 Aviator Successfully Returned to Flight After an In-Flight Spontaneous Pneumothorax

Henry DeYoung; Yousef Ahmed; Jami Buckley

BACKGROUND: Spontaneous pneumothorax (PTX) is a diagnostic challenge in aviators given the common occurrence of musculoskeletal pain after flight and notorious underreporting of symptoms of other diseases in this group.

CASE REPORT: A 24-yr-old active duty F/A-18 Weapon Systems Officer performed an anti-G straining maneuver (AGSM) in response to a 6.5-g warm-up turn during a training flight at 16,000 ft (4876.8 m) above sea level. He immediately developed right-sided thoracic back pain. The flight was terminated, he landed, and the pain improved. Over the next 5 d, he noticed the insidious development of pleuritic chest pain and dyspnea. His symptoms prompted presentation to an aviation medicine clinic where a large right sided PTX was identified. After transfer to a local emergency department, a large bore chest tube was placed. A CT scan showed bilateral apical blebs requiring right and subsequently left video assisted thoracoscopy (VATS) with chemical/mechanical pleurodesis and apical wedge resection. Pulmonary function testing (PFT) showed a mild restriction defect 2-1/2 mo after surgery. The patient also completed cardiopulmonary exercise testing (CPET), performing better than his predicted reference range. After a high resolution CT showed no remaining signs of bleb or cyst disease and another month of healing he was returned to flight.

DISCUSSION: PTX should be considered in aviators with perithoracic pain after flight as several aspects of flight in high performance aircraft may increase the risk for PTX. These include positive pressure breathing through a facemask, repeated use of the AGSM, and the possibility of bleb expansion at altitude.

KEYWORDS: in-flight pneumothorax, anti-gravitational straining maneuver, cardiopulmonary exercise testing.

DeYoung H, Ahmed Y, Buckley J. *F/A-18 aviator successfully returned to flight after an in-flight spontaneous pneumothorax*. Aerosp Med Hum Perform. 2018; 89(11):1008–1012.

Although the risks of flying with a pneumothorax (PTX) are well documented, cases documenting the development of PTX in flight are very rare. Patients, pilots, and commercial passengers in these cases have ranged from having no underlying disease, to conditions such as Birt-Hogg-Dube syndrome, lymphangioleiomyomatosis, alpha-1-antitrypsin deficiency (AAT deficiency), and cystic pulmonary airway adenomatoid malformation.[11] Of these cases, only a few report military aircrew and pilots flying modern high performance aircraft who developed PTX in flight while performing an anti-G straining manuever (AGSM) (**Table I**).[9,13,17] The rarity of PTX sustained in flight, subtle symptoms, and the possibility for delayed presentation can make the diagnosis challenging.[2,15,16] Further complicating the clinical picture for aviators of high performance aircraft is the common development of postflight aches and pains due to acceleration $+G_z$ forces.[7] Still, there are several physiological factors specific to flight in high performance aircraft that may increase the risk for PTX in aviators with underlying pulmonary disease. In flight, jet aviators experience breathing through a positive pressure facemask, experience lower atmospheric pressure at high altitudes, and use the AGSM to maintain cerebral perfusion during high $+G_z$. Though these physiological extremes are necessary for flight in high performance aircraft, they likely contributed to the development of spontaneous PTX in the case presented here. Although rare, spontaneous PTX is an important consideration in aviators presenting with peri-thoracic pain after flight.

From Strike Fighter Squadron 106, Oceana Medical Health Clinic, Virginia Beach, VA, and Naval Special Warfare Logistics Support Unit One, Coronado, CA.

This manuscript was received for review in April 2018. It was accepted for publication in July 2018.

Address correspondence to: Henry DeYoung, M.D., Strike Fighter Squadron 106, Oceana Branch Medical Clinic, 1750 Tomcat Blvd., Virginia Beach, VA 23460; henrydeyoung73@gmail.com.

DOI: https://doi.org/10.3357/AMHP.5145.2018

Table I. Cases of In-Flight Pneumothorax Thought to be Due to the AGSM in Chronological Order from Top to Bottom.

YEAR/STUDY	UNDERLYING DISEASE	NO. OF SUBJECTS	OUTCOME	AIRCRAFT
1975, Ho[9]	None	1	Returned to flight	T-33
1994, Robb[17]	Prior PTX	1	Returned to flight	F-16
2008, Lin et al.[13]	Bullae with AAT deficiency	1	Grounded	F-16

PTX, pneumothorax; AAT, alpha-1-antitrypsin deficiency.
In column order from left to right: year of study and reference number, underlying disease if present, number of subjects detailed in the case/case series, if the individual was returned to flying status as applicable, and the type of aircraft flown.

CASE REPORT

During a standard training flight, a male 24-yr-old F/A-18 Weapons Systems Officer performed an AGSM during a 6.5-g warm-up turn. Immediately after, he developed sharp right-sided thoracic back pain just lateral to T5–T7. At this point the training flight was terminated, he returned to base, where he landed, and felt that his pain had significantly improved. Over the next 3 d, his pain continued to worsen, which he attributed to thoracic muscle spasm. Approximately 2 mo prior he had presented for his yearly physical complaining of intermittent thoracic back pain related to flight in the same region. Given his previous condition, he felt he did not require evaluation upon landing and attempted treatment with anti-inflammatories at home.

Three days later, he presented to "sick call" at a military aviation medicine clinic with the chief complaint of a "tweaked back muscle." His pain remained isolated to his lateral right thoracic spine, but he had now developed a pleuritic character to his back pain, as he was unable to take a deep breath without coughing. His blood pressure was 116/80, heart rate in the 50s, respiratory rate was 12, and S_pO_2 of 92–94%. On the right side, he had diminished breath sounds to auscultation, there was hyperresonance to percussion, and he appeared uncomfortable. A chest X-ray showed a large right sided PTX (**Fig. 1**). At this point, he was started on 15 L oxygen on a nonrebreather face mask while awaiting transfer to the nearest emergency department.

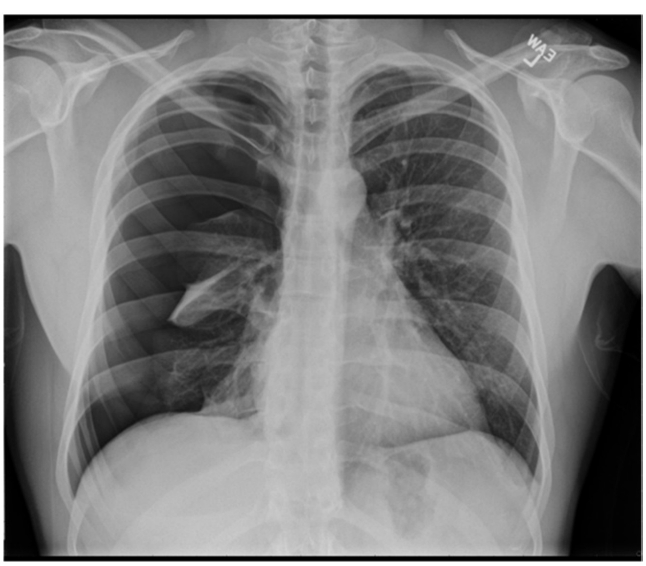

Fig. 1. PA chest X- ray from at the patient's initial presentation. The right hemithorax is hyperlucent with collapse of the right lung consistent with a large right pneumothorax without radiographic signs of mediastinal shift. At the time the chest X-ray was taken, there were no clinical signs of tension PTX.

Following transport, he received a right-sided 12 French chest tube thoracostomy and was admitted. A high-resolution CT scan was performed, revealing subtle bilateral apical blebs. He was seen by Cardiothoracic Surgery and, given his occupation as an F/A-18 Aviator, the decision was made to proceed with video assisted thoracoscopy (VATS) with chemical and mechanical pleurodesis with doxycycline with apical wedge resection. After a successful, uncomplicated surgery, and a short admission period, the patient was discharged home. He returned 6 wk later to undergo VATS and chemical/mechanical pleurodesis with apical wedge resection on the left side. Again surgery went well without complications and he was discharged after a short admission.

Pulmonary function testing (PFT) was performed 2.5 mo after surgery in accordance with the Naval Aeromedical Waiver Guide (ARWG) (**Fig. 2**). The patient's PFT results revealed a mild restrictive defect consistent with right and left apical wedge resection. To assess if he had adequate cardiopulmonary reserve for the physiological extremes of flight in the F/A-18, he also underwent cardiopulmonary exercise testing (CPET) (**Fig. 3**). He performed better than predicted for his reference range comparison group. After 3 mo of recovery, he was returned to flight status and has re-engaged with the training syllabus. At this time he has flown many times without any further issues.

DISCUSSION

Naval aviators are a unique and relatively small population who undergo comprehensive screening prior to commissioning and clearance for flight.[19] Although this process includes a chest X-ray, subpleural blebs are difficult to see on chest X-ray and are likely to be missed.[3,19] In aviators of high performance aircraft who have subpleural blebs that go undetected, the risk of PTX may be increased. As previously mentioned, these physiological extremes of flight that likely contributed to the development of PTX in this case are breathing through a positive pressure face mask, relatively lower atmospheric pressure at high altitudes, and use of the AGSM to maintain cerebral perfusion during high G forces.

F/A-18 aviators breathe through a pressurized face mask, the CRU-103, which allows them to fly at higher altitudes. The oxygen mask in an F/A-18 typically delivers oxygen at 10 cm H_2O of positive airway pressure throughout a given flight, but is capable of reaching 72 cm H_2O when pressure breathing for g's.[4] In comparison, alveolar rupture and iatrogenic pneumothorax in mechanical ventilation has been associated with peak airway pressures of 50 cm H_2O and plateau pressures that exceed 35 mmHg, respectively.[10] Patients in these studies

		Ref	Pre	% Ref	Post	% Ref	%Chg
Spirometry							
FVC	Liters	6.03	** 4.25	** 70	** 4.20	** 70	-1
FEV1	Liters	4.95	** 3.81	** 77	** 4.07	** 82	7
FEV1/FVC	%	83	90	108	** 97	** 117	8
FEF25-75%	L/sec	4.99	4.60	92	6.03	121	31
FEF25%	L/sec		8.15		9.32		14
FEF50%	L/sec		5.60		7.47		34
FEF75%	L/sec		2.56		4.15		62
PEF	L/sec	10.85	8.42	78	9.34	86	11
FET100%	Sec		5.98		6.79		14
FIVC	Liters		4.26		4.19		-2
Lung Volumes							
TLC	Liters	7.90	** 5.55	** 70			
VC	Liters	6.03	** 4.27	** 71			
IC	Liters	3.90	3.32	85			
FRC PL	Liters	4.20	** 2.23	** 53			
ERV	Liters	1.95	** 0.83	** 43			
RV	Liters	1.98	1.28	64			
RV/TLC	%	25	23	91			
Vtg	Liters	4.20	4.07	97			
Diffusing Capacity							
DLCO	mL/mmHg/min	33.1	30.9	93			
DL Adj	mL/mmHg/min	33.1	30.9	93			
DLCO/VA	mL/mHg/min/L	4.79	5.97	125			
DL/VA Adj	mL/mHg/min/L		5.97				
VA	Liters	7.90	** 5.17	** 65			
IVC	Liters	4.25	** 3.81	** 90			
Resistance							
Raw	cmH2O/L/sec	1.03	1.46	142			
Gaw	L/sec/cmH2O	1.007	0.685	68			

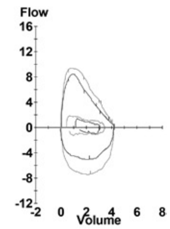

Fig. 2. Pulmonary function test results. The pulmonary function test results above were observed in the patient after bilateral apical wedge resection. There is a restrictive pattern as seen by the patient's diminished FEV_1 and FVC, increased FEV_1/FVC ratio, and diminished total lung capacity.

typically had acute respiratory distress syndrome, unlike the patient here, who was a healthy aviator. Given the patient's health, the pressurized face-mask was not likely a primary cause, but was likely a contributing factor in the development of his PTX.

Boyle's Law describes the inversely proportional relationship between pressure and volume in a closed system with a fixed amount of gas kept at a constant temperature. From 8000 ft to 23,000 ft (2438.4 to 7010.4 m) above sea level, the F/A-18 cockpit is pressurized to 8000 ft pressure altitude. Beyond 23,000 ft, the cabin pressure is scheduled to rise at a steady rate to as high as 15,000 ft (4572 m) pressure altitude at 35,000 ft (10,668 m) above sea level.[14] At 8000 ft pressure altitude the ambient pressure within the cockpit is equivalent to approximately 564 mmHg. In response to lower atmospheric pressure, gases within an enclosed space such as the human body may expand by as much 25–30% in volume. Subpleural blebs might demonstrate trapping, preventing communication with the tracheobronchial tree and pressure equalization, and could therefore lead to volume expansion and possible rupture.[1] This relationship appears to be meaningful as cases of spontaneous PTX appears to cluster in areas of low atmospheric pressure.[2,11,18]

Repeated use of the AGSM may lead to increased risk of the development of PTX in aviators of high performance aircraft with unknown pulmonary disease. The AGSM encompasses a combination of sustained muscle tensing and cyclical Valsalva maneuvers every 3 s throughout a G pull.[16] This maneuver allows aviators to increase their systemic arterial blood pressure and maintain cerebral blood flow while experiencing increased positive G force. A consequence of the AGSM is the elevation in the transpulmonary pressure and intra-alveolar pressure.[5,6,12] The "Macklin Effect" was first described by the Macklin brothers in 1944 as a mechanism for the development of spontaneous pneumomediastinum and subcutaneous emphysema due to alveolar rupture because of a large pressure gradient generated against a closed glottis. By this same mechanism, an individual performing an AGSM could also develop a spontaneous PTX through air leak into the pleural cavity. This scenario has been described as the underlying cause of spontaneous PTX in professional musicians, an adolescent running sprints, and an individual blowing up a large number of balloons.[5] There are approximately three other cases which have attributed the development of in-flight PTX to this maneuver.[9,11,13,17] Given that the patient in this case developed pain almost immediately after performing the AGSM, we believe the AGSM was the key cause of the patient's in-flight PTX.

The aviator in this case was a member of the U.S. Navy. According to the U.S. Navy ARWG, a single incidence of spontaneous pneumothorax is considered disqualifying for flight but is considered for a waiver after recovery and mitigation of recurrence.[19] In fact, a profession of risk, which includes aviators, is an indication for definitive management of spontaneous PTX. With evidence of bilateral pulmonary blebs on CT, the aviator described here required bilateral pleurodesis. Pleurodesis is a process of mechanically or chemically causing sclerosis of lung tissue and is often performed through VATS. VATS allows surgeons to avoid thoracotomy and makes use of a few small incisions and cameras to visualize the thoracic cavity. Both types of pleurodesis decrease or completely remove the pleural space, leading to pleural symphysis, preventing pneumothorax recurrence. Ultimately, these procedures reduce the risk of pneumothorax recurrence to approximately 1–5%.[18]

The U.S. Naval ARWG also states that designated aviators with a single episode of spontaneous PTX must also have normal PFT to receive a waiver.[19] Because the patient's PFT showed mild pulmonary restriction, we hoped that, by using CPET, we could prove the patient had normal physiological reserve and was safe to return to flight status (Fig. 2). CPET provides a comprehensive functional assessment of the cardiovascular, respiratory, muscular, and metabolic systems during exertion. Some of the relevant parameters of CPET include the $\dot{V}O_{2max}$ and $\dot{V}_E/\dot{V}CO_2$ slope. $\dot{V}O_{2max}$ is the best indicator of a person's ability to inhale and use oxygen and is a universal marker of

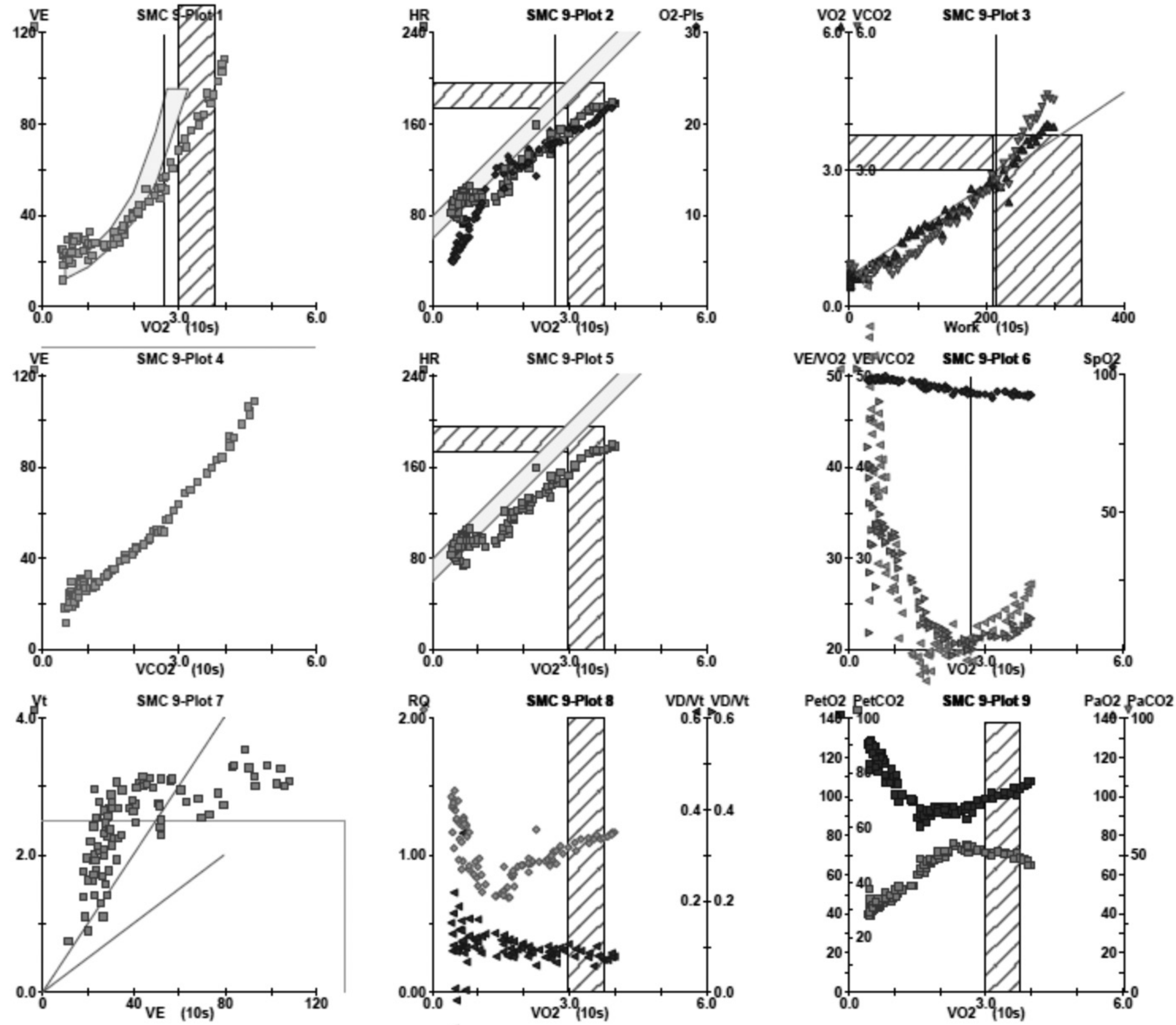

Fig. 3. Cardiopulmonary exercise testing or CPX. The CPX nine-panel plot graphic array shown above was produced from the patient's CPX test after postoperative recovery. The results shown above show that despite the patient's history of pulmonary restriction from apical wedge resection, he has no signs of decreased exercise capacity. The patient had a $\dot{V}O_{2max}$ of 4.034 $ml \cdot kg^{-1} \cdot min^{-1}$, 119% of predicted seen in plots 1 and 3. The $\dot{V}_E/\dot{V}CO_2$ slope was 17.4, better than his predicted $\dot{V}_E/\dot{V}CO_2$ slope of 21.9 and is seen in plot 4. HR, heart rate (bpm); $\dot{V}_E$, ventilation ($L \cdot min^{-1}$); $\dot{V}O_2$, oxygen consumption ($L \cdot min^{-1}$); $\dot{V}CO_2$, carbon dioxide consumption ($L \cdot min^{-1}$); V_T, tidal volume (L); $P_{ET}O_2$, partial pressure of end-tidal oxygen; $P_{ET}CO_2$, partial pressure of end-tidal carbon dioxide.

disease severity in many cardiopulmonary diseases, including pulmonary restriction. The $\dot{V}_E/\dot{V}CO_2$ slope is similarly useful in assessing overall disease severity in a number of conditions, including pulmonary restriction, reflecting the matching of ventilation and perfusion within the pulmonary system. The patient in this case had a $\dot{V}O_{2max}$ and $\dot{V}_E/\dot{V}CO_2$ slope that were both better than his predicted reference range (Fig. 3).[8] From these results, we were able to show the patient had normal cardiopulmonary functional limitations and had more than adequate physiological reserve. We believe CPET would be a beneficial addition to the Naval ARWG. CPET results could be used in aviators with pulmonary disease and abnormal PFT to better assess their functional status and suitability for return to flight.

Through positive pressure breathing, bleb volume expansion at altitude, and repeated use of AGSM, the risk for development of an in-flight spontaneous PTX may be increased. In aviators presenting with perithoracic pain, PTX should be considered and treated promptly. Treatment with VATS and chemical and mechanical pleurodesis is effective in mitigating the development of subsequent PTX. CPET is also useful in the disposition of aviators following treatment. Last, PTX due to subpleural blebs is not a condition that precludes further service as an aviator in high performance aircraft.

ACKNOWLEDGMENTS

The views expressed in this article are those of the author(s) and do not necessarily reflect the official policy or position of the Department of the Navy, Department of Defense, or the U.S. Government.

All authors listed had full access to the data listed in this manuscript and had a significant role in writing this manuscript.

The authors whose names are listed below certify that they have no affiliations with or involvement in any organization or entity with any financial interest (such as honoraria, educational grants, participation in speakers' bureaus, membership, employment, consultancies, stock ownership, or other equity interest, or expert testimony or patent-licensing arrangements) or nonfinancial interest (such as personal or professional relationships, affiliations, knowledge, or beliefs) in the subject matter or materials discussed in this manuscript.

Authors and affiliations: Henry DeYoung, M.D., Strike Fighter Squadron 106, and Jami Buckley, M.D., Department of Aviation Medicine, Oceana Branch Medical Clinic, Virginia Beach, VA, and Yousef Ahmed, M.D., Naval Special Warfare Group One, Coronado, CA.

REFERENCES

1. Baumann MH. Pneumothorax and air travel: lessons learned from a bag of chips. Chest. 2009; 136(3):655–656.
2. Bintcliffe O, Maskell N. Spontaneous pneumothorax. BMJ. 2014; 348:g2928 Erratum in: BMJ. 2014; 348:g3302.
3. Cantin L, Bankier AA, Eisenberg RL. Multiple cystlike lung lesions in the adult. AJR Am J Roentgenol. 2010; 194(1):W1–W11.
4. CRU-103 Chest Mounted Oxygen Regulator. Cobham; 2009. [Accessed 2017 November]. Available from http://www.cobham.com/mission-systems/oxygen-systems/military-aerospace-oxygen-systems/regulators/cru-103-oxygen-regulator/docview/.
5. Dejene S, Ahmed F, Jack K, Anthony A. Pneumothorax, music and balloons: a case series. Ann Thorac Med. 2013; 8(3):176–178.
6. Eiken O, Kolegard R, Lindborg B, Mekjavic IB, Linder J. The effect of straining maneuvers on G-protection during assisted pressure breathing. Aviat Space Environ Med. 2003; 74(8):822–826.
7. Hämäläinen O. Thoracolumbar pain among fighter pilots. Mil Med. 1999; 164(8):595–596.
8. Herdy AH, Ritt LEF, Stein R, de Araújo CGS, Milani M, et al. Cardiopulmonary exercise test: background, applicability and interpretation. Arq Bras Cardiol. 2016; 107(5):467–481.
9. Ho BL. A case report of spontaneous pneumothorax during flight. Aviat Space Environ Med. 1975; 46(6):840–841.
10. Hsu CW, Sun SF. Iatrogenic pneumothorax related to mechanical ventilation. World J Crit Care Med. 2014; 3(1):8–14.
11. Hu X, Cowl CT, Baqir M, Ryu JH. Air travel and pneumothorax. Chest. 2014; 145(4):688–694.
12. Lauritzsen LP, Pfitzner J. Pressure breathing in fighter aircraft for G accelerations and loss of cabin pressurization at altitude–a brief review. Can J Anaesth. 2003; 50(4):415–419.
13. Lin YC, Chiu WK, Chang H, Cheng YL, Chen JC. Spontaneous pneumothorax in flight as first manifestation of alpha-1 antitrypsin deficiency. Aviat Space Environ Med. 2008; 79(7):704–706.
14. NAVAIR. NATOPS flight manual Navy model F/A-18E/F (OPNAVINST) 37107: NATOPS general flight and operating instructions. Patuxent River (MD): Naval Air Systems Command; 2008.
15. Peterson DC, Martin-Gill C, Guyette FX, Tobias AZ, McCarthy CE, et al. Outcomes of medical emergencies on commercial airline flights. N Engl J Med. 2013; 368(22):2075–2083.
16. Postmus PE, Johannesma PC, Menko FH, Paul MA. In-flight pneumothorax: diagnosis may be missed because of symptom delay. Am J Respir Crit Care Med. 2014; 190(6):704–705.
17. Robb DJ. Cases from the aerospace medicine residents' teaching file. Case H57. Complete spontaneous pneumothorax in-flight in an F-16 pilot during a high-G maneuver. Aviat Space Environ Med. 1994; 65(2):170–172.
18. Tschopp JM, Bintcliffe O, Astoul P, Canalis E, Driesen P, et al. ERS task force statement: diagnosis and treatment of primary spontaneous pneumothorax. Eur Respir J. 2015; 46(2):321–335.
19. U.S. Navy Aeromedical Reference and Waiver Guides. In: Center NMOT, editor. Washington (DC): United States Navy; 2016:1–21.