Explosive Decompression with Resultant Air Gas Embolism in a Fourth Generation Fighter at Ground Level

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BACKGROUND: Arterial gas embolism (AGE) is a rare condition in the flying community most often only ever seen in flight while operating at high altitude or incidents involving hypobaric chambers. This article describes a severe case of AGE that occurred in a pilot of a fourth generation fighter aircraft at ground level. The environmental control system (ECS) malfunctioned, causing an overpressurized cockpit and a subsequent explosive decompression when the pilot opened the canopy to egress.

CASE REPORT: The ECS onboard fourth generation fighter aircraft is composed of many computer-controlled subsystems. When these components fail, the system can potentially overpressurize the cockpit. Combined with opening the canopy without prior venting, this overpressurization can lead to a situation akin to a diver surfacing too quickly. A pilot experienced this scenario and subsequently developed symptoms of arterial gas embolization—one form of decompression illness (DCI). We reviewed the design of the environmental control system and recommend that the cockpit must be slowly depressurized to decrease risk of injury from rapid decompression.

DISCUSSION: Literature review showed three similar cases of ground-based overpressurization causing AGE symptoms, although these cases were maintenance personnel intentionally testing aircraft cabin integrity and not associated with aircraft intending flight.[7] The lessons learned from this case can be used to identify and hopefully prevent severe DCI from ground level cockpit overpressurization and to further general understanding of aircraft ECS.

KEYWORDS: overpressurization, decompression illness, environmental control system, arterial gas embolism.

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While rare, sudden loss of cabin pressure at high altitude or exposure in altitude chambers are the most common causes of decompression illness (DCI) in the aviation community. These symptoms can range from commonly seen mild joint pains or cutaneous rash to rare complications of cardiopulmonary collapse, neurological emboli, or death.[12] When explosive or rapid decompression occurs, however, there is an increased risk of more serious injury. Data for exact comparisons is scant, but it is estimated in animal models that mortality may increase by 40% when decompression time is shortened from 0.041 s to 0.015 s. It is difficult to estimate an exact correlation for these cases due to differences reported in the literature for total pressure change tested, time of exposure, rate of pressure change, and lack of controlled human studies.[12]

In this article, we discuss the case of a pilot who experienced an environmental control system (ECS) malfunction that resulted in cockpit overpressurization while at ground level. The pilot opened the canopy to egress the aircraft without venting excess cabin pressure, causing explosive decompression. He subsequently developed a severe case of arterial gas embolism (AGE). In this regard, it is important for all personnel involved from air crew to maintenance and emergency medical personnel to understand the aircraft systems, methods to respond to these unique situations, and appropriate medical care. This would

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improve response times, overall situational awareness, and recognition of DCI symptoms even in a novel situation.

CASE REPORT

The patient was a healthy 27-yr-old male pilot with over 300 h flying jet aircraft. Of note, he had previously suffered decompression sickness symptoms 6 mo earlier when his aircraft failed to maintain cockpit pressure at high altitude. At that time, his only symptom was a scarlatiniform rash over the torso and upper extremities. He underwent a U.S. Navy Treatment Table 6 (TT6) dive with complete resolution of the rash and no other sequelae.

On the day of the incident, around 08:00, the pilot had planned a daytime training sortie in the local area, which has an elevation of approximately 50 ft (15 m). After a normal engine start, the pilot switched on the ECS. Instead of simply pressurizing the cockpit seal with only a slight increase in cockpit pressure, the ECS began an immediate, uncontrolled cockpit pressurization process. Within moments, the pilot felt pain in his head, face, and bilateral ears. He felt the need to Valsalva repeatedly and performed the maneuver several times to keep up with the increasing cockpit pressure. These sensations continued until the pilot shut off the ECS and the cockpit pressure stabilized. His ears no longer popped, but he still felt a lot of pressure on his head.

The pilot did not declare an emergency, which would normally lead to activation of emergency medical response, but called maintenance for aid. Upon checking his cabin pressure altimeter gauge, he observed the needle varying between the 4 and 5 o'clock position, representing an unlabeled portion of the gauge. The gauge was designed to annotate altitude from sea level to 50,000 ft (15,240 m) in 1000-ft (305-m) increments (**Fig. 1**).[8,9] After a few minutes, the ECS mechanic arrived and discussed the situation with the pilot over the intercom. They agreed to terminate the sortie, thinking that either the gauge and/or ECS must be broken.

After being in the cockpit for approximately 15 min, the pilot shut down the engine according to his normal shutdown procedures and immediately opened the canopy, resulting in an extremely loud "bang." He complained of ringing in the ears combined with a tumbling vertigo sensation. All of the moisture inside the cockpit condensed into fog and the pilot lost visibility until the canopy raised high enough for the moisture to escape. The pressure gauge reset to 0 ft cabin altitude after the fog cleared. The pilot managed to exit the aircraft on his own, but felt lightheaded. He walked back to the squadron building and took off his gear. At that point, he noticed a constant substernal pain that worsened with deep inspiration and did not change significantly with exhalation. He also noticed that his distant vision was blurry and that he could not see the end of a 10-ft (3-m) hallway clearly.

The pilot's squadron members then took him to the local hospital, where the medical team started him on 100% oxygen via nonrebreather facemask. The physical exam found normal breath sounds bilaterally without wheezing or crackles, but the member complained of a nonproductive cough every few minutes. There was absent right facial sensation in the V3 distribution with loss of vibration, temperature, and cutaneous touch to the area. No other neurological symptoms were found, with normal sensation to the rest of the face. The pilot also had blurry vision to the point where he could not see clearly past the foot of the bed, scoring approximately 20/100 bilaterally on a bedside Snellen's test. The chest pain was described as a sharp stabbing pain diffusely around the mid chest area and did not localize. The pain was not affected by palpation. The remainder of the exam, a portable chest X-ray, and ECG were negative.

After roughly 30 min on 100% oxygen, the patient showed mild improvement. His vision had returned to normal, though his cough, inspiratory chest pain, and facial numbness remained. Consultation was made with the USAF hyperbaric specialists and the pilot was presumptively diagnosed with AGE. The closest hyperbaric chamber was located 2 h away by ground so the treatment team called for helicopter transport. Air transport took roughly 20 min to arrive. The flight arrived at the dive chamber, which is located at approximately 128 ft (39 m) above sea level, in 30 min. The member remained on oxygen during the entire flight under the care of in-flight medical personnel. The helicopter flew at an altitude restriction of 1000 ft (304.8 m) above ground level and did not violate this during the flight over and around local mountains of around 800 to 1300 ft (244 to 396 m).

At the hyperbaric facility, the pilot underwent a TT6 dive to 60 fsw. Total time from the start of the incident inside the cockpit to the start of the dive was approximately 2 h, including transportation. Within 30 min of initiation of the dive, the pilot's pulmonary symptoms resolved and, within 60 min, the sensation to his face returned. After completion of the TT6 without any extensions, the patient was re-examined and found to be asymptomatic. The team decided to not admit the patient for observation as the member appeared to be fully treated and believed that there was low risk of new symptoms arising. The medical team then transported the pilot home by ground ambulance as the helicopter had returned to base earlier and counseled him on hydration, refraining from exercise, and avoiding alcohol. The medical team instructed the pilot not to fly for at least 72 h and to follow up the next morning in the flight medicine clinic.

During the appointment, the pilot stated that he had an uneventful evening and had gone to sleep by 22:00 local for approximately 7 h. After waking up, he complained of developing new sharp 4/10 lower back pains around the L3-L4 region and sharp 3/10 right upper neck pains around the C5-7 level. He also reported urinary incontinence during sleep when he woke up with wet trousers and a return of the lower sternal discomfort. He did not report the need to urinate overnight. On physical exam, he again had complete absent cutaneous sensation in the V3 distribution on the right side of the face, but remained ambulatory and had no nuchal rigidity. The chest discomfort was described as being nearly identical to the previous

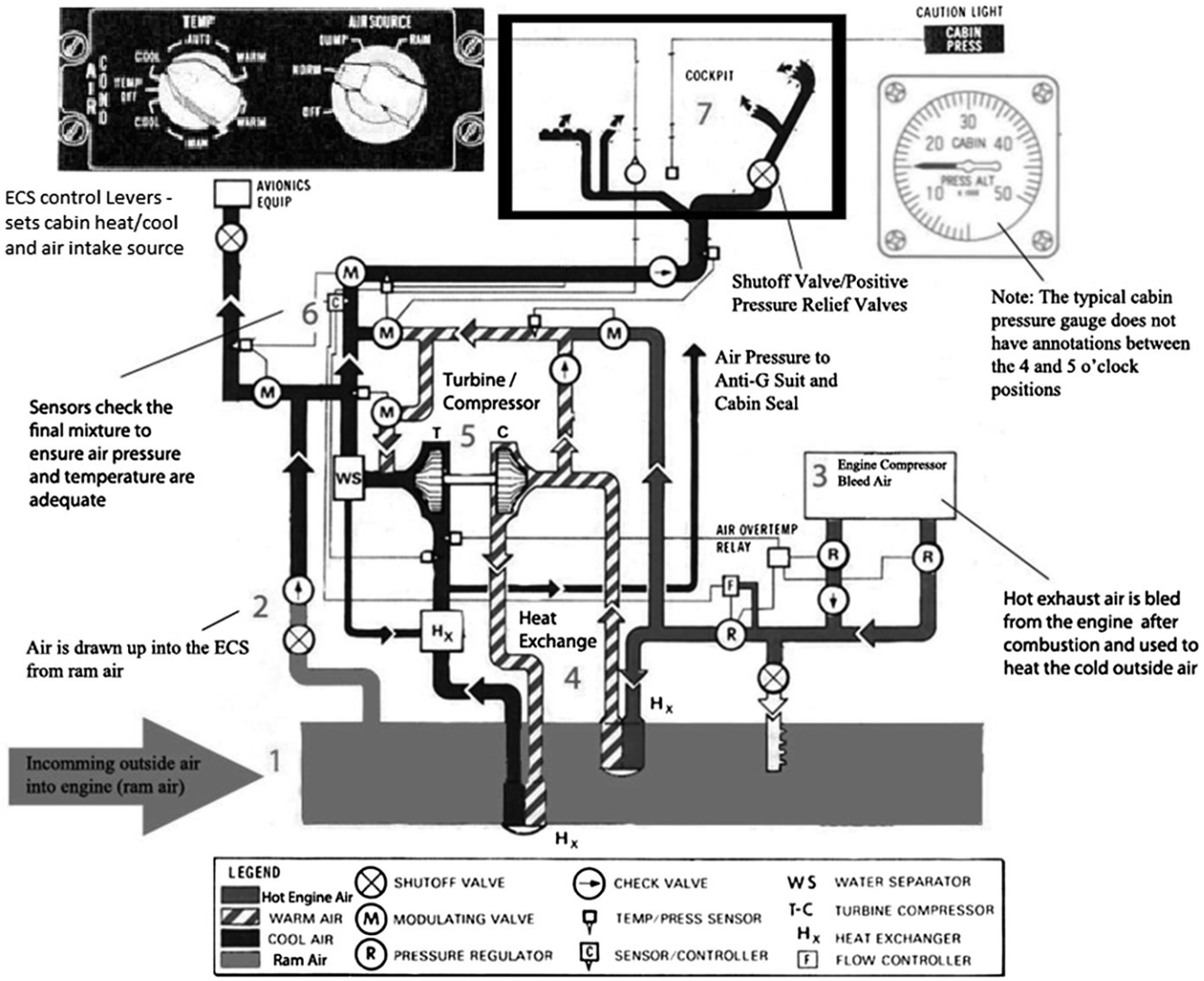

Fig. 1. Typical environmental control system flow diagram. 1) Air enters the aircraft through a ram air intake nozzle at the exterior of the aircraft. 2) The air flows up and continues into the cabin if it is of sufficient temperature and pressure. 3) Hot engine exhaust warms cold intake air at high altitude via heat exchange tubes at location 4. 5) Air compressors and turbines regulate the amount of airflow and pressure. 6) Sensors ensure temperature and pressure. 7) Cabin pressure is further adjusted by pressure relief valves.

day's presentation. There was no cough and the lung exam was normal. The remainder of the exam showed no other abnormalities. A chest X-ray and EKG done before transport were also normal.

A helicopter was called after roughly 15 min of exam which again took 20 min to arrive and then took the patient back to the dive chamber using the same route as before. The pilot was started on oxygen during the exam on the ground and remained on oxygen through the flight. Supplemental oxygen did not affect the patient's symptoms. While in flight, the patient noted increased pain in the lower back despite altitude restriction. Upon landing, he complained of worsening back pains and had sharp pains shooting down both legs to the knees with weight bearing and also with leg raises. He was able to move his legs and feet, but was unable to stand due to pain and was transitioned to a spine board to exit the aircraft despite being able to walk onto the aircraft initially.

A TT6 dive started immediately. His chest discomfort, back, and neck pains improved once he reached 60 ft (18.29 m—approximately 26 psi) for 10 min. Pinprick and cold temperature sensation returned to normal in the right V3 distribution within 20 min. Vibration sensation returned to normal over the next 30 min. The remainder of the dive was unremarkable. The pilot again had complete resolution of symptoms at the conclusion of the TT6 dive.

The patient returned to the hospital by ground transport and received similar discharge instructions as the day prior following an uneventful 8-h monitoring period. Due to the repeat symptoms, he was also encouraged to take ibuprofen 800 mg three times daily for 3 d. In the subsequent days that followed, a magnetic resonance imaging of the brain showed no acute intracranial pathology and echocardiography was negative for a patent foramen ovale. A neurological evaluation 3 wk later confirmed that he had no lasting sequelae. Given full resolution of

symptoms, the pilot returned to flying status a month after the incident.

DISCUSSION

Based on the review of the literature, this case may be the first time that explosive decompression injury occurred to a pilot while inside an aircraft at ground level during routine flight operations. The medical definition of explosive decompression is a change in air pressure that occurs at a rate swifter than the rate at which air can escape from the lungs. This typically occurs with a significant change in pressure within 0.5 to 1 s.[3] When slower rates of change in pressure occur, the lungs can compensate by alveoli recruitment and diffusion of the pressure across a greater surface area. With a rapid decompression, however, respiratory gases in the pulmonary tree can expand faster than this compensation mechanism, resulting in pulmonary parenchymal tears. This kind of barotrauma allows gas to enter the pulmonary venous outflow and migrate through the left heart into the arterial circulation, causing AGE. Air trapping from a closed glottis in breath holding during this decompression can further increase intrathoracic pressures and worsen lung damage.[6] These air emboli often lodge in the brain, but can also travel to distant sites, producing a variety of symptoms such as neuropathies, pain, weakness, visual deficits, and even stroke-like manifestations.

Based on the cabin altimeter pressure gauge reading at the time of the event of around the 4–5 o'clock positon, and assuming linear movement of the needle in a counterclockwise direction to approximately negative 5000 to 10,000 ft (1524 to 3048 m), we estimated the pilot likely experienced a pressure differential of somewhere around 8.9–17.8 fsw (approximately 17.55 to 20.84 psi) as compared to surface pressure.[11] The subsequent rapid decompression induced by opening the aircraft canopy was sufficient to provoke AGE symptoms, which could be in as little as 4 fsw (approximately 1.8 psi differential) as described in diving literature.[2,5,7]

The patient's substernal chest pain was one of three classic manifestations of pulmonary barotrauma, with the other two being cough and hemoptysis.[4] Onset of his other symptoms (visual changes, vertigo, etc.) within seconds of decompression further support the diagnosis of AGE. Localized pulmonary injury, pneumomediastinum, and/or pneumothorax are also likely possibilities in these cases. Initial treatment of a suspected pulmonary barotrauma patient should consist of 100% oxygen delivery with clinical and radiographic assessment to rule out pneumothorax. As with all DCI events, a thorough neurological exam is required to catalog all potential deficits.

The definitive treatment for these DCI symptoms remains recompression therapy in a hyperbaric chamber.[10] There are multiple benefits to hyperbaric oxygen in these events. First, increased atmospheric pressure reduces bubble size, which may decrease symptom severity and local hypoxia. Second, a significant oxygen diffusion gradient develops, which not only assists with mobilizing and elimination of inert gases, but also increases tissue oxygenation, which may preserve hypoxic tissue and nearby watershed areas. Finally, treatment inhibits the inflammatory responses from the initial hypoxia and related reperfusion injury.[3]

The patient's presentation on the first follow-up visit with increasing back pain, difficulty walking, and urinary incontinence suggest he likely had an associated spinal cord hit either because of an inflammatory cascade that did not become clinically significant until later or represented a new onset AGE from residual pulmonary damage. Treatment in these recurrent cases remains the same, with a TT6 dive and possibly use of extensions as the recommended course of action. Adjunctive treatment research suggests the use of nonsteroidal anti-inflammatory drugs to decrease the risk of re-injures. Experimental models also suggest the use of mixed gas treatment tables such as heliox[1] to further decrease the number of recurrent symptoms. Mixed gas treatments, however, require advanced technical expertise and are likely not accessible in most instances.

To understand how an aircraft can potentially cause a DCI event, it is useful to understand how a typical ECS similar to the aircraft in this scenario provides pressurization (Fig. 1).[8,9] Air enters the aircraft at position 1 through a ram air intake nozzle at the exterior of the aircraft. The air flows up to position 2 and continues into the cabin if it is of sufficient temperature and pressure as measured by probes (6). Hot engine exhaust warms the cold air at high altitude (3) via heat exchange tubes at position 4. Air compressors and turbines at position 5 regulate the amount of airflow and pressure. A final check valve will ensure proper temperature and pressure before air enters the cabin (7). All of this is controlled by the onboard computer and adjusted by the cockpit controls.[9]

At altitude, the ECS system in single seat aircraft often maintains the pressure in the cabin only 5 psi higher than outside air pressure in a system known as isobaric (constant) differential pressure control.[8] The turbine/compressor pump that pressurizes the air is opposed by the opening of positive pressure relief valves in the cockpit that dump any excess pressure to the outside. In practice, 5 psi is the equivalent of lowering the cabin altitude from 40,000 ft (12,190 m) to 16,000 ft (4880 m).[11] In some fourth generation fighter aircraft this system has a theoretical maximal burst potential of 7.35 $\pm$ 0.35 bars (7.253 atm or 106.6 psi),[8] but normally only operate at a 5-psi differential pressure range. This system is superior to completely sealed systems in that it generally weighs less and can minimize the threat of rapid decompression when ejecting.

A gel mesh seal held in place by straps around the canopy edge and inflated by the ECS system also serves to maintain cockpit pressure. Even if all the exhaust valves are closed, this seal will slowly leak air out of the cockpit at a typical nonlinear rate between 30–50 $ft^3 \cdot min^{-1}$ (0.849 to 1.416 $m^3 \cdot min^{-1}$) when the cockpit is pressurized at a 5-psi differential with the outside.[8] This rate will slow as the pressure differential decreases. The average fourth generation fighter cockpit volume is around 150 ft^3 (4.248 m^3); therefore, after roughly 3 to

5 min with the ECS system switched off, the cockpit pressure would approach ambient air pressure. The cabin seal in this case may have prevented leaking since the engine remained on and the ECS malfunctions kept a higher than normal flow to the seals.

The ECS in this case clearly malfunctioned. For the event to occur, the positive pressure relief valves at position 7 likely failed. In addition, either the computerized control sensors at position 6 must have also failed to detect the increased pressures and continued to operate the compressor pump at position 5 or the compressor may have failed to respond to a shutoff signal. This event would require at least two or more redundant safety systems to fail simultaneously, which accounts for its rarity. While the ECS was turned off earlier, the pilot kept the engine on, which could also further complicate the situation as a lot of the air pressure comes directly from the engine intakes. A complete teardown and inspection of the ECS system on this aircraft, unfortunately, identified no failed components. After reassembly and inspection, this aircraft returned to service and reported no further ECS malfunctions to date.

On review of this case, we realized a lack of understanding of this type of event from aircrew and emergency responders. Should future pilots find themselves in an overpressurized cockpit on the ground, we recommend they immediately shut down the ECS along with the aircraft engine, and wait inside a closed cabin until cockpit pressurization normalizes to ground level. This will allow the canopy seal air leak to slowly equilibrate to outside pressures and reduce the risk of rapid decompression. Breathing supplemental 100% oxygen would add an additional protective factor. If potential for rapid or explosive decompression is unavoidable, the pilot should avoid breath holding during canopy or hatch opening. Most, if not all aircraft, have a manual exhaust valve that maintenance personnel can access or a pilot operated dump valve on the ECS controls. Crews must take care when using these options to reduce excess cockpit pressure at a controlled rate over several minutes to reduce risk of rapid decompression. This information should be useful for development of future emergency response checklists.

Aircraft ECS are generally highly reliable life support systems. When they malfunction or are being ground tested, the potential for pulmonary barotrauma and AGE exist. Aerospace medical personnel should become familiar with the aircraft they support and be cognizant of how to respond appropriately in the event of an overpressurization event. They should also review response plans and exercise them to better stabilize patients, transport appropriately, and utilize emergency hyperbaric medicine services.

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