Severe Neurological Decompression Sickness in a U-2 Pilot

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Severe neurological decompression sickness (DCS) has been a rare entity in the U.S. Air Force, including the U-2 community. In over 50 yr of operation, few U-2 pilots reported severe neurological DCS in flight despite the extreme altitudes at which they operate. This article describes a near-fatal case of neurological DCS that occurred during a combat mission. The injury left the pilot with permanent cognitive deficits that correlated with focal lesions present on magnetic resonance imaging of his brain. To our knowledge, the images presented herein are the first to show radiological evidence of brain injury induced by altitude DCS. Though only a single case, the objective and clinical findings in the case pilot are similar to results documented in divers suffering DCS with central nervous system injury and victims of traumatic brain injury. DCS will remain a potentially serious threat to current and future air and space operations.

Keywords: U-2, case report, altitude, decompression sickness, neurological symptoms, traumatic brain injury, magnetic resonance imaging.

DECOMPRESSION sickness (DCS) remains a significant concern for U-2 pilots during high-altitude flight. DCS and its sequelae are recognized complications in persons exposed to changes in environmental pressure commonly seen in high altitude aviation or scuba diving. Symptoms vary widely, ranging from mild joint pain, to serious neurological manifestations, and even death. In contrast to diving-related DCS, death or incapacitation from DCS are virtually unheard of in modern aviation. This is due partly to redundant systems protecting aviators and the fact that descent from altitude serves as compressive therapy (1,7,13,14).

U-2 pilots are at significant risk of DCS due to frequent long sorties at extreme altitudes. The U-2S is a high-altitude surveillance aircraft flown by a single pilot. Typical missions involve flights over 70,000 ft (21,336 m) for greater than 9 h. The aircraft is pressurized with a differential ratio to maintain a cabin pressure below 35,000 ft (10,668 m), so pilots wear a full pressure suit in case of unexpected cabin decompression. Aviators also undergo denitrogenation ("pre-breathing") by breathing 100% oxygen for at least 1 h before flight. Pre-breathing establishes an oxygen gradient to offload nitrogen from tissues to the blood, thereby decreasing nitrogen stored in the body. These measures have been effective at preventing aviation-related DCS over time.

Despite increased risk, no deaths, incapacitation, or lasting injuries due to DCS have been reported among U-2 pilots in over five decades. In a 1996 survey of active/retired U-2 pilots, approximately 70% reported at least one episode of DCS during their career. Of those, 12.7% were severe enough to cause the pilot to alter the flight plan or abort the mission, but no lasting injuries were reported (2). In this article, we present a case of near-fatal DCS with neurological symptoms in a U-2S pilot during flight with permanent sequelae.

CASE REPORT

During a high-altitude reconnaissance mission in support of combat operations in 2006, a 47-yr-old male U-2S pilot experienced severe physical and cognitive manifestations of neurological DCS. The pilot performed standard preflight procedures, including donning a full pressure suit and pre-breathing for 1 h at rest. Approximately 2.5 h into flight [cabin altitude 28,000 ft (8534.4 m)], the pilot complained of bilateral knee pain which dissipated after he increased the internal pressure of his pressure suit. Moments later, he noticed ankle pain. Thinking it was caused by poor position, the pilot adjusted his rudder pedals with no relief. Simultaneously, he sensed the aircraft rolling, which he attributed to momentary spatial disorientation caused by head movement. Over the next 2 h, the pilot experienced gradually worsening concentration, confusion, fatigue, and headache. Recognizing his deteriorating condition, the pilot tried eating, drinking, increasing oxygen supply, and increasing suit pressure, with only minimal relief. The pilot rationalized his symptoms as "old man problems" and did not alert ground controllers.

The pilot first notified ground controllers he was "feeling ill" 4 h later, but elected to continue. Minutes later, he reported severe weakness and difficulty breathing. As the pilot's cognition worsened, controllers provided step-by-step instructions for even the most basic cockpit

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operations. Suspecting DCS, controllers directed the pilot to return to the deployed base from which his flight originated. Hostile airspace prevented immediate descent, which lengthened the pilot's hypobaric exposure. As the pilot's mental status deteriorated, ground controllers experienced increasingly longer episodes of radio silence before all voice communications eventually ceased. Meanwhile, the pilot had to open his helmet facemask to clear debris after an episode of spontaneous emesis. This exposed him to an extremely hypoxic and hypobaric environment for the remainder of the flight. The pilot's symptoms progressed to include color vision loss, hemianopsia, visual disturbances, hearing loss, and repeated emesis. The pilot was unaware of his surroundings throughout descent to the airfield. Other pilots observed the U-2S descending in a stall before recovering spontaneously. The pilot made multiple attempts to land on the wrong runway, coming within 5 ft (1.5 m) of a likely fatal ground impact at one point. Fortunately, during one approach, the pilot regained enough situational awareness just prior to ground impact to land the plane safely.

Upon landing, rescue personnel found the pilot slumped over the instrument panel with emesis contaminating the cockpit. The flight surgeon controlling the scene was a hyperbaric medicine specialist. His initial clinical assessment was "severe DCS with neurologic symptoms and incipient cardiovascular collapse." Rescue personnel extracted the pilot and initiated intravenous hydration and 100% oxygen by aviator's mask. Treatment continued en route to a nearby host-nation hyperbaric facility via helicopter. The helicopter restricted flight to 300–500 ft (91.4–152.4 m) above ground level to minimize nitrogen bubble re-expansion.

Brief physical examination was accomplished on the runway. Notable findings included clinical signs and symptoms of shock with pale clammy skin, thready pulse, and lethargy. A bluish-purple, mottled rash consistent with cutis marmarota was seen over his torso. Peripheral oxygen saturation was 89% (off oxygen). Respiratory rate was slow and shallow while auscultation of the lungs revealed diffuse rales and crackles. His carotid pulse was thready and no radial pulses were palpable. Neurologically, the pilot had a Glasgow Coma Scale of 14 (opened eyes to speech) and was difficult to arouse. He had difficulty with serial 7s and judgement. Deep tendon reflexes were hyperactive with normal Babinski response. He was unable to stand or walk and finger-to-nose pointing was abnormal.

The pilot's past medical history was significant for two reported prior episodes of DCS with joint and skin manifestations in 1999 that resolved with surface level oxygen. During this deployment, the pilot denied any daily medications. His body mass index was 31. He denied prior tobacco use and alcohol ingestion during the day before flight. There were no recent hypobaric or hyperbaric exposures. The pilot had suffered from viral gastroenteritis 5 d prior to this incident. However, he was symptom free 2 d before the incident. He was a highly experienced pilot, also serving temporarily as squadron commander.

The pilot underwent hyperbaric oxygen treatment (HBOT) after arriving at the host nation medical facility. He initially underwent U.S. Navy Treatment Table 6 (USN TT6) with two extensions at 60 fsw. During the first oxygenation break, he vomited and remained difficult to arouse. By the third break, he reported improvement in symptoms and tolerated oral fluids. HBOT was halted and the pilot remained hospitalized. His medical work-up included transthoracic echocardiogram, various laboratory studies, electroencephalogram, and electrocardiogram. These tests were unremarkable. However, computed tomography of the brain demonstrated areas of low attenuation in the bifrontal and temporal regions. Therefore, MRI was performed to determine the extent of injury. The radiologist described multiple punctuate cortical and sub-cortical lesions involving the bifrontal and parietal lobes, likely due to microvascular ischemia (**Fig. 1**). The consulting neurologist and radiologist concluded the findings were consistent with high altitude DCS. Given the pilot's marked improvement after HBOT and these MRI findings, the flight surgeon deferred further tests.

The pilot showed persistent cognitive and fine motor deficits (e.g., he could not remember how to shave) 12 h after initial treatment. Thus the flight surgeon initiated a second USN TT6 HBOT without extensions. After treatment, the pilot could recall details about the mishap and his global condition improved (i.e., he performed daily activities without assistance). Over the next 2 d, the pilot

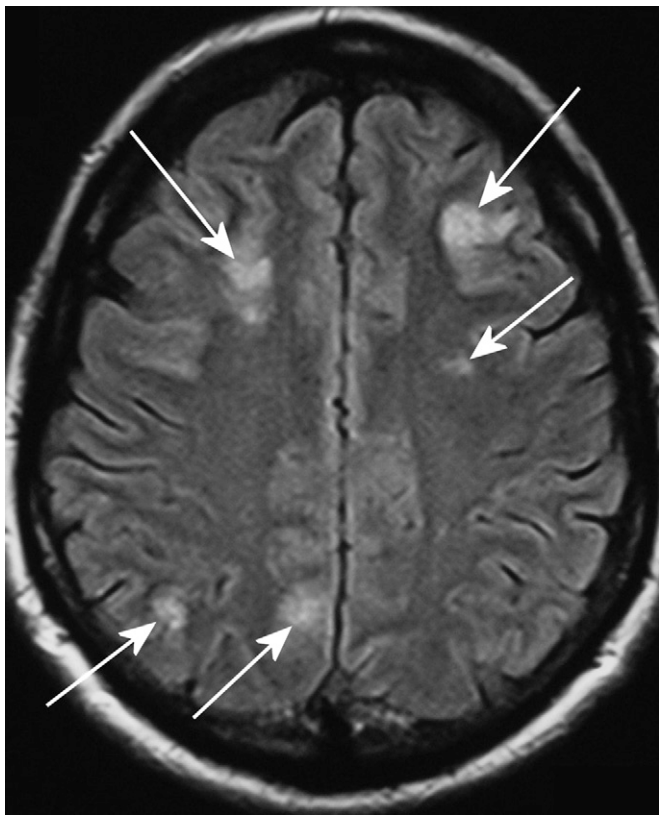

Fig. 1. Initial FLAIR MRI images through frontal and parietal lobes showing multiple cortical lesions involving the bifrontal areas, likely due to microvascular ischemia (solid arrows).

underwent two Treatment Table 9 sessions until his symptoms no longer improved with HBOT. A second MRI showed marked improvement; all cerebral lesions had almost disappeared except a right frontal lesion, which was smaller. He was discharged from the hospital 7 d after admission. The pilot remained with his squadron for another 8 d before returning home on an uneventful commercial flight. Investigation of the aircraft showed all systems functioned properly, including life support systems.

The pilot reported subjective resolution of his symptoms following HBOT. Accordingly, he was evaluated by a neurologist on return to home station who documented a normal neurological examination. Another MRI of the head was consistent with previous exams. This MRI demonstrated a focal lesion in the right frontal lobe (**Fig. 2**). It also showed multiple areas of gliosis involving cortical gray matter and subcortical white matter within the frontal lobes on T2 and FLAIR images. The lesions were in proximity to watershed zones, consistent with ischemic injury (**Fig. 3**). Similar lesions occurred in the right cerebellum (**Fig. 4**). Given the patient's clinical history and earlier MRI reports, the neurologist concluded these lesions represented sequelae of anoxic injury during his DCS incident.

The flight surgeon returned the pilot to flying status 3 mo later, after completing the required aeromedical consultations. The pilot continued to deny recurrent symptoms and stated his desire to return to flying. An altitude chamber ride was not deemed necessary. To mitigate risk, the pilot resumed flight in stages. Initially, he flew four sorties with an instructor pilot in the unit's companion jet trainer, the T-38. Next, he flew a two-seat training U-2 with an instructor pilot on two low-altitude and two high-altitude sorties. Only then did he return to flying solo in the U-2S. The pilot's initial flights took place without incident. However, he experienced dizziness and disorientation on his second solo. Symptoms resolved after inflating his pressure suit and the pilot landed uneventfully. After discussion with hyperbaric medi-

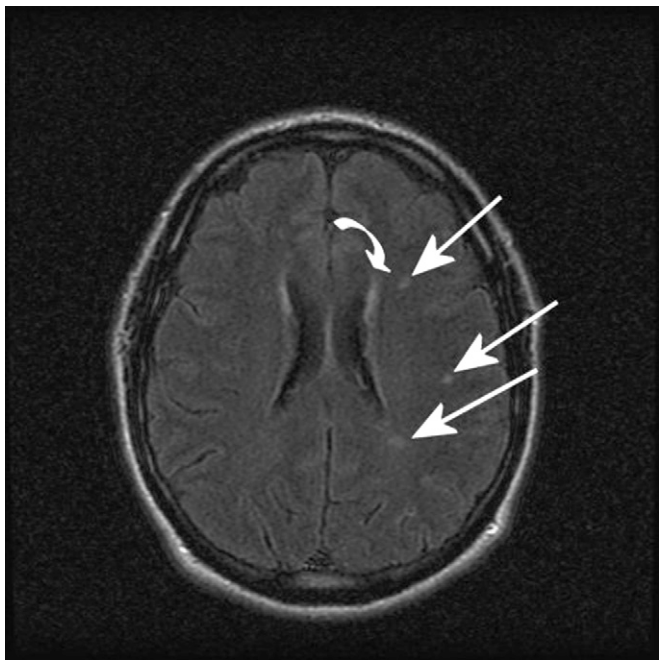

Fig. 3. FLAIR MRI image demonstrating punctate areas of hyperintensity within the brain (straight arrows). Lesions are located predominately along gray-white matter junctions of the inferior-lateral left frontal lobe. A similar lesion is noted in the periventricular white matter along the left atrium of the ventricle (curved arrow). Lesions do not enhance (images not shown) and are most consistent with gliotic foci secondary to microvascular ischemic changes.

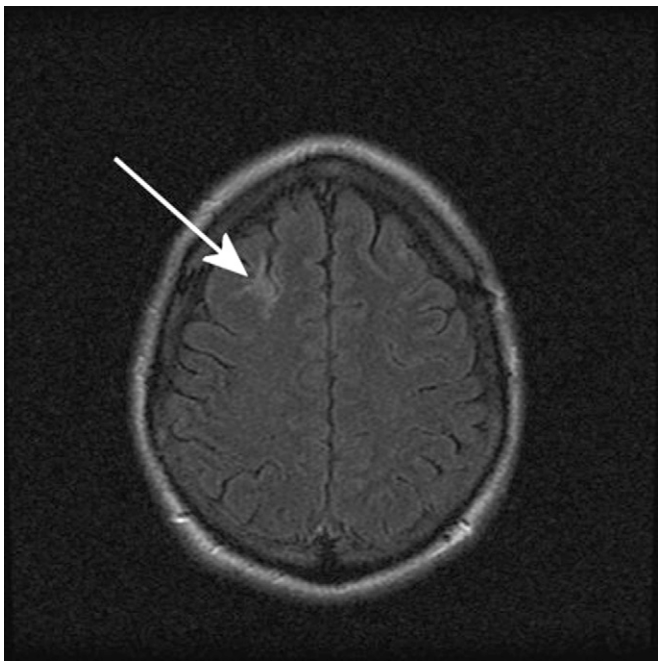

Fig. 2. Subsequent FLAIR MRI image completed at home station demonstrating a persistent lesion along the right frontal sulcus consistent with microvascular ischemia secondary to DCS (solid arrow).

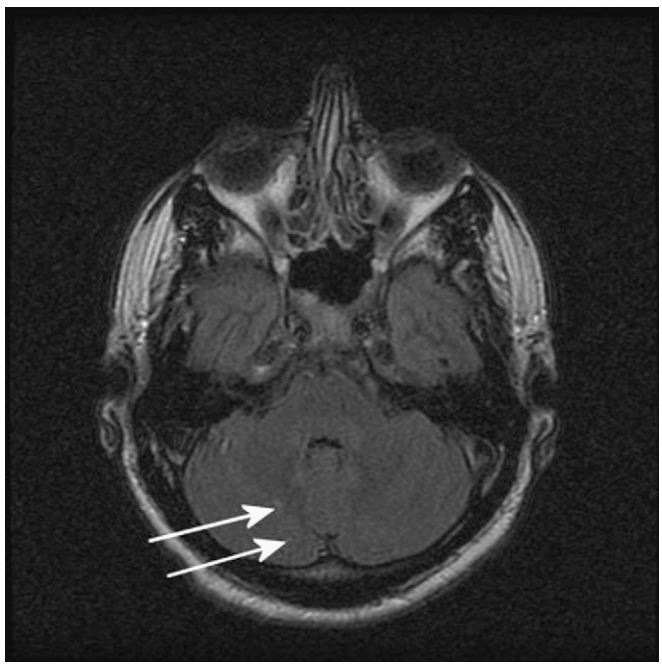

Fig. 4. FLAIR MRI image depicting similar punctate lesions in the right cerebellum (solid arrows).

cine consultants, the pilot's symptoms were felt to be manifestations of anxiety. He was placed on duties not including flying and released after 1.5 h of oxygen by aviator's mask. The pilot drove unassisted to his home, located at 1800 ft (548.6 m) elevation difference from the base. The pilot experienced a severe retro-orbital headache 36 h later. Over the next 3 wk, he experienced short-term memory loss, recurrent headaches, and "feeling in the fog." His symptoms partially improved with time, but he did not seek acute treatment.

Due to potential recurrence of DCS symptoms, the pilot underwent evaluation by the Aeromedical Consultation Service (ACS) to obtain a waiver for continued flying. During interviews, the pilot admitted to multiple unreported prior incidents of joint DCS. He also reported being hospitalized in 1991 "for dehydration" which, in retrospect, the pilot believed was actually neurological DCS. His ACS evaluation included normal neurology exam, transesophageal echocardiogram with bubble study, and electroencephalogram. Repeat MRI showed stable gliotic lesions. Significantly, a neuro-psychology exam showed cognitive deficits with variable performance decrements corresponding to the brain areas demonstrating lesions on MRI. These findings indicated the pilot would have difficulty managing complex problem-solving tasks in novel flying situations. Any re-injury could cause further deficits and he had a higher risk of seizure (though the absolute risk could not be quantified). Overall, the ACS concluded the pilot's risk of incapacitation was greater than 1% per year, which is the generally accepted cut-off standard for acceptance of incapacitating risk frequency. Consequently, the ACS recommended permanent disqualification.

The pilot retired from the military and never returned to flying. At the time of this publication, he reported persistent headaches, degraded visual acuity, joint pain, personality changes, short-term memory loss, and cognitive deficits. These deficits prevent him from obtaining a commercial pilot's license or other comparable employment. The pilot also reported ongoing difficulty obtaining clinical care and disability compensation from the Department of Veteran Affairs. His difficulty may be due in part to the unique mechanism of injury and subtle nature of his cognitive deficits.

DISCUSSION

This case is unique considering the pilot suffered a severe brain injury during flight that was nearly fatal and left him with permanent cognitive disabilities. In aviation, DCS with central nervous system involvement is rare. In a prospective series of 447 altitude chamber subjects at Armstrong Laboratory, only 0.5% exhibited frank central nervous system involvement (11). Similarly, residual deficits are rarely reported and are short-term in nature. Deaths are virtually unknown after 1959 and the institution of HBOT (1,11,13,14). In contrast, deaths and permanent injury are more prevalent among divers afflicted with central nervous system DCS. Also, the spinal cord is most commonly affected in divers, whereas the brain is usually affected in aviators (14).

This case was also unique to the U-2 community at the time. Historically, DCS incidence has been difficult to assess in the flying community due to reluctance among pilots to report incidents for fear of being disqualified from flying. Bendrick et al. found the prevalence of DCS among U-2 pilots is higher than previously reported, but no instances of permanent disability or death (2). Our search of historical records, published literature, and safety records uncovered few cases of neurological DCS comparable in severity to the subject case. One occurred in 2002 when neurological symptoms manifested after landing (9). At least three other in-flight cases occurred after 2006. Whether these cases reflect an increase above expected incidence of neurological DCS or a natural consequence of policy changes that promote reporting is a matter of conjecture.

This is the first case we are aware of documenting radiological evidence on MRI of neurological DCS in a pilot. Objective radiological evidence of neurological injury is unusual in DCS. Diagnostic modalities evaluated thus far (CT, MRI, SPECT, and PET imaging) have shown low sensitivity in detecting pathological changes in the brain with aviators or divers afflicted with neurological DCS. Consequently, they have little use in acute diagnosis (5,8). Similar to results with soldiers suffering traumatic brain injuries, the utility of imaging in documenting clinical progress of neurological DCS patients has not been proven. When present, MRI and CT lesions tend to correlate with a higher degree of structural damage and greater likelihood of residual deficits (3,5,6,8). In this case, there was direct correlation between the pilot's physical exam and MRI findings (Fig. 3 and 4). Confusion, temporary amnesia, and personality changes in this pilot are indicative of a temporal-frontal lobe injury—the clinical equivalent of stroke. He also displayed cerebellar findings, including ataxia and impaired equilibrium. One could argue his lesions resulted from previous DCS injuries or another undiagnosed condition. Gliotic lesions have been found in asymptomatic divers and chamber attendants (4,5). While no studies have been performed in aviators, routine altitude exposures of U-2 pilots could plausibly result in similar findings. Unfortunately, no earlier studies are available for comparison in this case as neuroradiological studies are not routine screening exams. However, standard three-sequence MRI studies performed at the home station (diffusion, apparent diffusion coefficient, and T-2 weighted sequences) indicated this pilot's lesions were acute. Clinically, the pilot had no antecedent signs or symptoms on multiple flying physical exams to suggest a pre-existing neurological condition. Additionally, improvements noted in sequential MRI studies by the host nation correlated clinically with the pilot's neurological status as HBOT progressed. Most importantly, deficits persisting in this pilot today are consistent with his MRI lesions.

The degree to which one person is more susceptible to DCS than another is a matter of debate. Generally accepted risk factors increasing susceptibility to aviation DCS include higher altitude, longer exposure, greater in-flight activity, and lack of pre-oxygenation. However,

multiple individual factors, including age, obesity, hydration status, physical condition, gender, and prior DCS (12,14), may increase susceptibility. Altogether, any of these factors, or the combination, could have influenced this pilot's condition. Unfortunately, studies to date have been unable to predict an individual's specific risk of DCS with certainty (10,12,14).

Ultimately, if prevention fails, pilots must recognize DCS and seek treatment. Current protocol is to descend immediately and initiate surface level oxygen via aviator's or tight-fitting non-rebreather mask. HBOT must be initiated immediately for severe symptoms. For mild cases (i.e., joint pain), HBOT is indicated if symptoms do not improve within 30-60 min of treatment, or if symptoms worsen. Hyperbaric medicine specialists should be consulted for guidance (8).

Diagnosing and treating DCS can be difficult under typical flying conditions. One is tempted to criticize the case pilot for not landing sooner. In that sense, he recognized a degree of complacency in his early decision making. On the other hand, the pilot sustained a frontal lobe lesion that likely impaired his judgment. More generally, aviators routinely make compromises for practical reasons that can delay seeking treatment for DCS. For one, pilots and astronauts place considerable pressure on themselves to complete their missions. Combat especially may force military pilots to subjugate personal safety to mission needs. Two, diagnosis of DCS is difficult, even for medical experts. For example, the pilot attempted a logical sequence of actions to correct likely physiological problems (i.e., cramped seating, hypoglycemia, hypoxia, etc.) before considering DCS. These were reasonable actions flight surgeons perform when troubleshooting in-flight problems. Finally, flights over oceans or wilderness place pilots far from available HBOT. In this case, controllers concluded the pilot would be safer landing at an airfield known to him rather than closer, but unfamiliar emergency fields. While this increased the pilot's exposure, on-scene medics endorsed the decision in order to provide lifesaving HBOT on landing.

We reported lessons learned from this near-fatal physiological incident to improve flying safety. Despite the stellar safety record in aviation-related DCS since 1960, this case demonstrates that DCS remains a serious potential threat to flight operations. The potential for DCS will persist as advances in aerospace technology continue to push the limits of high altitude flight. For example, the Air Force's newest fighter, the F-22 Raptor, can cruise at altitudes greater than 60,000 ft (18,288 m), while commercial companies are developing spaceflights for tourists. Future aviators would benefit from continued research as our understanding of DCS pathophysiology and treatment evolves.

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