Federal Air Surgeon's Medical Bulletin

Aviation Safety Through Aerospace Medicine

Vol. 45, No. 3
2007-3

For FAA Aviation Medical Examiners, Office of Aerospace Medicine Personnel, Flight Standards Inspectors, and Other Aviation Professionals.

U.S. Department of Transportation
Federal Aviation Administration

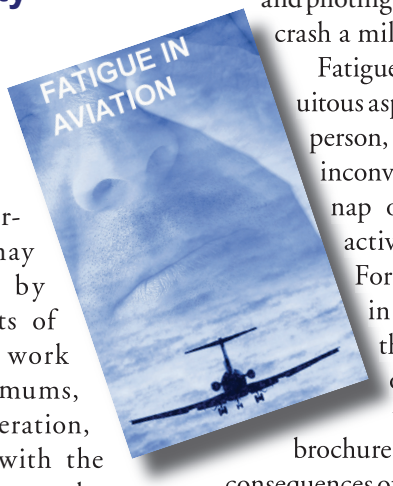
HEADS UP

OAM People Serving Airmen at Air Venture 2007

AME Resources

New Pilot Safety Brochure Published

Dangers of Fatigue Shown



A RECENT FATAL aircraft accident may have been caused by the cumulative effects of fatigue—a 16-hour work day, approach to minimums, stress, single-pilot operation, and over-familiarity with the airport area—all factors that contributed to the pilot's unfortunate decision to attempt a landing in a situation that called for heightened awareness and piloting skills but resulted in a fatal crash a mile short of the runway.

Fatigue is an expected and ubiquitous aspect of life. For the average person, fatigue presents a minor inconvenience, resolved with a nap or by stopping whatever activity that brought it on. For pilots and others involved in safety-related activities, though, the consequences of fatigue can be disastrous. A new pilot safety brochure describes the causes and consequences of fatigue, and the brochure provides methods to avoid it.

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QUICK FIX

IT'S HISTORY

By Richard 'Dick' Jones, MD

PROBLEM

THOSE OF US who review FAA medical examinations for which certificates have been issued by Aviation Medical Examiners (AMEs) have long felt that many medical histories provided by AMEs do not support the decision to issue the certificate. For instance, the most common deficiencies are (1) failure to address items airmen checked "yes" or left blank in blocks 13, 17, 18, or 19 of the examination form, and (2) the history given does not provide the information required by the *Guide for Aviation Medical Examiners.*

Please do not misunderstand, I am not trying to say that airmen who are certified despite these deficiencies are unsafe to fly, but I simply want to point out it is often difficult for our certification folks to determine whether or not it is safe for the airman to fly. These inadequate histories cause great consternation if a medically certificated airman has an aircraft accident that might be attributable to a medical cause.

RESULT

In November 2006, the Aerospace Medical Research Division of the Civil Aerospace Medical Institute (CAMI) completed a study on the adequacy of the medical histories provided by AMEs. Two physicians randomly selected 2,000 examinations for systematic review. They were classified into one of four categories: good history, issued erroneously, history inadequate

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Operation Safe Pilot Revisited

By Fred Tilton, MD

HELLO, EVERYONE. In my last editorial [Operation Safe Pilot, *Federal Air Surgeon's Medical Bulletin*, Vol. 45, No. 2, p. 2], I told you about *Operation Safe Pilot*. For those of you who did not have an opportunity to read the editorial, *Operation Safe Pilot* was a cooperative effort between the Inspectors General from the Department of Transportation and the Social Security Administration.

The purpose was to determine whether or not there were people who were fraudulently collecting total Social Security disability benefits and/or falsifying their Federal Aviation Administration (FAA) medical applications. It turns out that there were such people.

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The justice department took legal action against the offenders, and the FAA revoked both their airman certificates and their airman medical certificates. In that same issue of the *Bulletin*, **Dr. Jones** provided some feedback on the 2006 FAA Aerospace Medical Services Airman Customer Satisfaction Survey, which indicated that there are a few Aviation Medical Examiners (AMEs) who are not performing examinations up to FAA standards.

'Our whole system depends on the honesty of our applicants and your examinations'

I promise you that we will not make a habit of "talking" about the same subjects in consecutive bulletins. However, airman falsification of medical applications and substandard examinations strike at the heart of our medical certification program. We rely on you, the AME, to perform good examinations, and you rely on the airman to give you accurate and complete information. The first lesson, and probably the most important lesson we learned in medical school was, "listen to the patient and take a detailed history." Our whole system depends on the honesty of our applicants and your examinations.

Congress felt this issue was so important that the U.S. House of Representatives Committee on Transportation and Infrastructure held hearings on July 17th. The witnesses were Mr. **Nicholas Sabatini**, Federal Aviation Administration Associate Administrator for Aviation Safety (AVS-1), **Mitch Garber**, MD, from the National Transportation Safety Board, The Department of Transportation Inspector General, Mr. **Calvin L. Scovel III**, Mr. **Phil Boyer**, President or the Aircraft Owners' and Pilots Association, and I.

I will not go into a lot of detail regarding the hearings, but I do need to let you know that we agreed to some process changes. We are planning to:

- Change the FAA Form 8500-8 to include a question that asks the airman if he or she has ever received any form of disability compensation
- Add a notice similar to the National Driver Register notice on the current form, which authorizes the agency to compare the data on our 8500-8 with other agencies that might be providing disability benefits to the individual
- Enhance our quality control and AME oversight processes

The Chair of the Subcommittee on Aviation, Mr. **Jerry F. Costello**, has given us additional questions that we are addressing. I believe that Congress will ask us to take some additional steps to help assure we have a certification process that does everything it can to assure that our examination process is as robust as possible.

It is unfortunate that I have to write such an article, because most of you do an outstanding job for us, and most airmen are honest. However, the misadventures of a few are driving us to take these steps.

Remember, we all play an extremely important role in helping to assure the safety of the National Airspace. Please make sure you are completing FAA examinations according to standards, and that you remind your airmen of their responsibilities, and the consequences of falsification.

—Fred

Certification Update

Information About Current Issues

By Warren S. Silberman, DO, MPH

Update on FAAMedXPress

Our new **FAA**MedXPress computer system has been rolled out to the public with the quietest reaction I have seen yet. However, we have received phone calls from some of our aviation medical examiners who claim they know nothing about this system.

This amazes me as we mailed a letter out to all registered aviation medical examiners last winter, published several articles in the *Bulletin*, added a detailed explanation, a PowerPoint slide presentation of the screens that the airmen see, an instructional booklet provided by Northrop Grumman to the Aerospace Medical Certification Subsystem Web site, and sent postcards to all airmen announcing the system—some of them twice!

When the Aerospace Medical Certification Subsystem was activated, I asked you all to bookmark its information site and that you try to read it at least once per week. I am not going to belabor the point.

We initially rolled the system out on April 16th to Alaska and the entire western U.S. On May 16, we allowed the central USA to input examinations, and as of June 11, we permitted the remainder of the country and the rest of the world to participate. Our most recent statistics show that 10,625 airmen have completed the process to be able to electronically submit their medical history; 6,263 airmen have initiated filling out the front side of the 8500-8 (which is what the system is all about); 2,206 airmen have completed the exam history and submitted it for completion by an AME (an appointment date may be pending with an AME. The exam will sit in the netherworld until they give their confirmation number to the AME); and 1,090 airmen went to an AME who completed the physical examination section and then transmitted it to the Aerospace Medical Certification Division.

We urge you to encourage your airmen to participate in FAAMedXPress, as it will reduce our requirement to scan the hard copy 8500-8s into the Document Imaging and Workflow system (DIWS), thus reducing our workload.

Smoking Cessation Medications

The Federal Air Surgeon, under advisement from his Pharmacy and Therapeutics committee, accepted the use of Chantix (varenicline) tablets for use in smoking cessation. Varenicline binds with alpha4beta2 neuronal nicotinic acetylcholine receptors. The efficacy of varenicline is thought to be the result of its activity at a sub-type of the nicotinic receptor where its binding produces agonist activity, while preventing nicotine binding to the alpha4beta2 receptors.

Varenicline blocks the ability of nicotine to activate alpha4beta2 receptors and thus to stimulate the central nervous system dopamine structure, which may be the mechanism in reinforcing and rewarding that smokers experience. Varenicline, being highly selective, binds more potently to the receptors than to other common nicotinic receptors. It is metabolized in the liver and excreted 92% unchanged in the urine.

The dose is to titrate the 0.5 mg daily for three days, then 0.5 mg twice daily for four days. The maximum dose is 1 mg twice daily. The overall level of adverse reactions to varenicline was nearly the same as the level of reactions to placebo. The most common adverse reaction associated with taking the medication is nausea (32.4%). The majority of the nausea was reportedly mild, and only 3.9% of patients chose to discontinue the medication due to the nausea. Insomnia is the next most common adverse reaction (19.0%) associated with varenicline. Somnolence was also found to be a side effect (9.3%).

The likelihood of side effects is reduced by titrating the medication from 0.5 mg daily to 0.5 mg twice daily to 1 mg twice daily over a seven-day period. Smokers are supposed to quit on day eight of the medication. Most patients who make it through the titration period seem to do well.

The FAA recommends that the airman not fly until at least 72 hours at the maximum planned treatment dose. In other words, we recommend that airmen wait until the treating physician gets them to their maximal dose and then wait 72 hours more. Use of varenicline will not require a special issuance. We will require a statement from the treating physician or examining AME regarding the presence or absence of side-effects. The local AME may release the airman to flight duties if no side-effects are experienced, and the presence or absence of side-effects must be reported on the next FAA physical examination.

Note, the use of **Wellbutrin® and Zyban®** are unacceptable for smoking cessation at this time because they are antidepressant medications. However, **Nicorette®** (gum or patches) is acceptable for use in aviation.

Cardiology Teaching Case

Here is a great case that we managed recently. I thought that it was interesting enough to present it to you now and will update you in the next edition of the *Bulletin*. A 19-year-old student pilot suffered an abrupt onset of "excruciating" anterior chest pain while exercising. He was taken to a local emergency room where his initial electrocardiogram revealed marked anterior ST segment depression. His chest pain and electrocardiograph changes persisted, despite the administration of morphine sulfate and sublingual nitroglycerin.

His physical examination showed him to be in obvious distress. He appeared weak in diaphoretic. His blood pressure was 100/80 and pulse rate was 100 with respiratory rate of 20. Breath sounds were normal. There was no chest wall tenderness. Auscultation

Continued on page 4

Certification from page 3

of his heart showed a diffuse cardiac impulse. The first and second heart sounds were normal. There were no murmurs, rubs, or gallops. His cardiac rhythm was normal. All pulses were palpable and equal. Abdominal aortic pulsation was normal. The extremities were unremarkable, with no evidence of cyanosis.

Cardiac catheterization revealed that his ascending aorta was enlarged at 43mm, ejection fraction was 50%, a left sinus of valsalva origin of his left main coronary artery, and there was evidence of a dissection of his proximal circumflex artery! Ultimately, there was demonstrated evidence of an inferoposterior wall myocardial infarction.

He was taken to the operating room. Examination of the interior of his aorta revealed that the ascending aorta was not dilated. There was no evidence of dissection at either of the coronary cusps. The aortic valve was trileaflet and totally normal. An intraoperative cardiac catheterization was performed. The left circumflex artery showed staining of the epicardium and evidence of an acute dissection. A reversed saphenous vein bypass was performed, and a portion of the aorta was taken to look for cystic medial necrosis, a pathologic finding seen in Marfan's syndrome.

Marfan's syndrome, along with this pathologic finding, make it likely that the potential airman would be prone to dissection further down in his vascular system such as carotid arteries.

Pathologic examination did not demonstrate any cystic medial necrosis, nor did further studies demonstrate any evidence of a connective tissue disorder.

Recent Holter monitoring demonstrated over 17,000 premature ventricular contractions over the 24 hour monitoring period. There were periods of multifocal ventricular bigeminy and ventricular couplets. There was no sustained ventricular tachycardia.

Echocardiography showed moderate left ventricular enlargement with decreased left systolic function. The ejection fraction was 41%. Regional wall abnormalities were present along with moderate mitral valve regurgitation. He also had mild left atrial enlargement.

Pertinent laboratory values showed a normal complete blood count, renal and liver function. Sedimentation rate was within normal range. Cholesterol was 122, triglyceride level was 60, high-density lipoprotein was 45 and low-density was 55.

Recently, the airman experienced palpitations but claimed to have had this symptom prior to the event. His medications are aspirin, Metoprolol and Lisinopril.

As you recall, the FAA policy is that an airman must wait six months from the event to be considered for medical certification, which this applicant has done. He must as yet provide us with a maximal stress test. Our inclinations are to deny certification to this airman, but this case will likely be presented to the FAA's cardiology consultant.

Have an enjoyable summer!

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Dr. Silberman manages the Civil Aerospace Medical Institute's Aerospace Medical Certification Division.

New Brochure from page 1

Fatigue in Aviation was written by Southwest Regional Flight Surgeon **Guillermo Salazar**, MD. Three copies of the 6-page informational brochure were recently sent to all aviation medical examiners to help answer questions that pilots may ask about fatigue.

Additional copies of the brochure are available from these sources:

- Download from the Federal Aviation Administration's Web site www.faa.gov/pilots/safety/pilotsafetybrochures/
- Phone: (405) 954-4831
- E-mail: Gail.Gentry@faa.gov

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Quick Fix from page 1

to support issuance, or incomplete examination. Approximately 1.8% of the histories were found to be incomplete, and 2.5% of the certificates were inappropriately issued.

A marked improvement in the quality of histories was noted in examinations performed after the implementation of the new Aerospace Medical Certification System (AMCS) in October 2005. Only 1.3% of the certificates issued after the new system was put in operation were judged to have been inappropriately issued. This extrapolates to more than 8,000 airmen in our system flying with questionable qualifications before and over 4,200 inappropriate issuances after October 2005. Surprisingly, 44% of the inappropriate issuances involved neurology (largely unconsciousness histories), followed by vision, cardiac, and cancer cases, each with 14% of the total.

SOLUTION

The Regional Flight Surgeons have volunteered to continue the same rigorous evaluations of randomly selected examinations done by AMEs in their regions to identify AMEs whose histories are poor, so they can help them improve. A full-time physician has been contracted to continue the same effort at CAMI. The information collected will be collated for use as part of each AME's annual performance report as judgment errors. It is hoped these and similar studies will result in greatly improved medical histories and, thereby, further increase the confidence that our AMEs contribute significantly to the safety of the National Airspace System.

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Dr. Jones manages the Aerospace Medical Education Division at CAMI.

Near-Ditching Proves Value of Training Course

INSTRUCTORS AT THE Civil Aerospace Medical Institute's Aerospace Medical Education Division (AMED) recently received a heartwarming "atta-boy!" in the form of a thank-you letter from their clients in the National Oceanic and Atmospheric Administration (NOAA). Two years ago, LCDR **Nancy Ash** of NOAA's Hurricane Research Group asked the AMED instructors to develop a training course for their mission scientists and other crewmembers who fly hurricane missions.

Since then, more than 250 managers, scientists, graduate students, and law enforcement agents have received potentially life-saving training at five locations.

The training's significance was tested early this year when engine problems developed on a weather research flight 300 miles offshore from St. John's, Newfoundland. The scientists aboard, who had been trained by the CAMI team, were told to prepare for ditching. Fortunately, the engine problems resolved, and the ditching wasn't necessary, but LCDR Ash says they were able to "calmly process what they needed to do as they prepared themselves to ditch" by relying on the training they had received, which "made the difference between panic and preparation." LCDR Ash stated that the "feedback from the scientists on the aircraft serves as testimony to the excellence and importance of the work performed by the [AMED] instructors."

Kudos for a job well done goes to the Oklahoma City-based instructors: **Eric Simson, Roger Storey, Don Demuth, Junior Brown**, and **Larry Boshers.**

AMED INSTRUCTORS SHOWN WITH THE VIRTUAL REALITY SPATIAL DISORIENTATION DEMONSTRATOR. (L-R) J.R. Brown, Eric Simson, Roger Storey, Don Demuth, Larry Boshers, and Team Lead Rogers Shaw, II.

Aviation Medical Examiner Seminar Schedule

2007		
August 17–19	Washington, D.C.	OOE (2)
August 27–31	Oklahoma City, Okla.	Basic (1)
September 14–16	Savannah, Ga.	CAR (2)
December 10–14	Oklahoma City, Okla.	Basic (1)
2008		
January 18– 20	Irvine, Calif.	OOE (2)
March 3–7	Oklahoma City, Okla.	Basic (1)
April 4– 6	Minneapolis, Minn.	N/NP/P (2)
May 12–15	Boston, Mass.	AP/HF (AsMA; 3)
June 2– 6	Oklahoma City, Okla.	Basic (1)
August 1–3	Washington, D.C.	CAR (2)
November 3– 7	Oklahoma City, Okla.	Basic (1)
November 14–16	Reno, Nev.	N/NP/P (2)

CODES

AP/HF Aviation Physiology/Human Factors Theme

CAR Cardiology Theme

N/NP/P Neurology/Neuro-Psychology/Psychiatry Theme

OOE Ophthalmology-Otolaryngology-Endocrinology Theme

(1) A 4½-day basic AME seminar focused on preparing physicians to be designated as aviation medical examiners. Call your regional flight surgeon.

(2) A 2½-day theme AME seminar consisting of 12 hours of aviation medical examiner-specific subjects plus 8 hours of subjects related to a designated theme. Registration must be made through the Oklahoma City AME Programs staff, (405) 954-4830, or -4258.

(3) A 3½-day theme AME seminar held in conjunction with the Aerospace Medical Association (AsMA). Registration must be made through AsMA at (703) 739-2240. A registration fee will be charged by AsMA to cover their overhead costs. Registrants have full access to the AsMA meeting. CME credit for the FAA seminar is free.

The Civil Aerospace Medical Institute is accredited by the Accreditation Council for Continuing Medical Education to sponsor continuing medical education for physicians.

Unruptured Intracranial Cerebral Aneurysms in Aviation

Case Report, by Marvin Jackson, MD, MS

This case report discusses the natural history of unruptured intracranial cerebral aneurysm and the potential impact of this diagnosis upon the individual and flight operations.

History

A 48 YEAR OLD flight engineer, currently holding a 1st class medical certificate, was awakened at 3 a.m. by a severe occipital headache. The pain was the most severe he ever experienced. Nevertheless, he spoke with his wife via telephone and took oral non-steroidal anti-inflammatory drugs (NSAIDs), which provided moderate relief and allowed him to continue his duties over the subsequent two days. He denied any systemic or neurologic symptoms concurrent with the headache. Upon return to the United States, the airman sought medical attention for the persistent headache. Medical history revealed a similar episode 19 years prior; however, there were no pertinent findings leading to a diagnosis. On occasion, he reported having occipital headaches, without migrainous signs or symptoms, which were relieved with NSAIDs. There was no report of hypertension or any other conditions. He had 18,400 total flight hours, with 300 hours in the six months prior to this event.

Lumbar puncture performed with this latest episode revealed a pleocytosis with 73% lymphocytes. Cerebral spinal fluid (CSF) protein content was normal at 47gm/dl. No organisms were seen and CSF culture was negative, suggesting aseptic meningitis, which resolved uneventfully over the next five days. Imaging included a non contrast computerized tomography (CT) scan of the head. This was read as normal; specifically, there was no evidence of acute hemorrhage, an inflammatory process, or a remote insult represented by hemosiderin, encephalomalacia, or hydrocephalus. Magnetic resonance imaging (MRI) and MR angiography revealed a 2-3mm saccular aneurysm at the A1-A2 junction of the anterior communicating artery. There was no suggestion of hemorrhage into the wall of the aneurysm-sentinel hemorrhage- or intraluminal clot. CT angiography one week later confirmed the location and size of the aneurysm. The patient remained neurologically intact and has petitioned to return to full flight status.

Aeromedical Concerns

All agree that an aneurismal SAH is a devastating insult to the central nervous system. The ISUIA retrospective arm reports a case fatality rate of 66% overall, 83% in the incidental group and 55% in the additional group. After 5 years in the prospective arm, 51 patients suffer their first SAH, with 33 (65%) going on to die.[1,2] Other work suggests that 30% of the remaining aneurismal SAH patients have poor neurologic outcomes.[6] The risk of sudden incapacitation after a SAH is tremendous. Of concern in the aerospace environment are 1) command and control of the aircraft; 2) Access to definitive medical care; 3) impact upon crew resources and performance capabilities.

In general aviation, such a hemorrhage could easily lead to a non-survivable accident, including the affected person, passengers, and those on the ground. This is more likely, if the affected person is also the pilot-in-command, flying alone, or with an inexperienced or non-pilot. Instituting definitive medical care is difficult, even in the best of circumstances. In such a situation, in-flight care would be prohibitive, and access to definitive neurosurgical care would be delayed, despite diversion, hastened landing, and emergency medical staff. Part 135 and 121 multi-crew cockpits, trained in resource management and in-flight emergency procedures, with supportive medical care available via on-board medical kits, contact with ground-based medical providers, and possibly medically trained passengers, have an advantage over the general aviation population. The potential for supportive care could preserve the possibility of surviving the primary insult and preventing secondary insults until the lesion is addressed. If the pilot-in-command was incapacitated and could not direct crew efforts, the task would fall to the first officer, who may not have the level of experience necessary to land the aircraft and direct in-flight medical emergency efforts. In all, the risk of SAH is small; however, the consequences of a hemorrhage, irrespective of aneurysm size, could be devastating in the flight environment. A safety margin could be maintained if the airman: 1) receives regular follow up neurological histories, physical exams, and appropriate imaging to reassess the aneurysm's dimensions and the rate of progression towards the 7mm cutoff; 2) flies only in a multi-crew capacity; 3) is restricted from pilot-in-command duties until the aneurysm is obliterated. If obliterated via surgery or endovascular therapy, one year follow-up imaging should confirm that the lesion remains occluded.

Outcome

The airman's medical certificate was initially denied. However, his neurosurgeon wrote that the risk of hemorrhage is negligible, and thus the risk of sudden incapacitation is not a legitimate concern if the entity seen on imaging studies is truly an aneurysm. The FAA neurology certification consultant concurred and advised restoring the airman's first-class medical certification, without restrictions, based

Continued on page 7

Dr. Jackson was a Wright State University resident in aerospace medicine when he wrote this case report at the Civil Aerospace Medical Institute. Currently, he is a flight surgeon in the FAA Great Lakes Medical Division in Des Plaines, Ill.

Natural History and Risk Assessment

Subarachnoid hemorrhage (SAH) is the most common presenting sign. Intraparenchymal extension occurs in 20-40%; and intraventricular hemorrhage, 13-20% of cases. Characteristic of anterior communicating artery aneurysm rupture is a "flame sign" on CT scan, created by hemorrhage into the anterior 3rd ventricle. Sentinel hemorrhage can lead to increased aneurysm size but not SAH. Cranial neuropathy may result from aneurysm expansion or hemorrhage, thus giving additional clues to the aneurysm's location. Transient ischemic attacks or cerebral vascular accidents due to emboli may produce infarcts in the distribution of the aneurysm's parent vessel. Finally, seizure activity due to gliosis helps localize the lesion but also may serve to precipitate a hemorrhage.[5]

Autopsy and long-term studies found the prevalence of unruptured intracranial cerebral aneurysms to range from 0.2-7.9%(mean 5%). Anterior circulation vessels produced 5-6 fold more aneurysms than the posterior circulation vessels. Multiple aneurysms accounted for 20-30% of cases. Familial aneurysms amounted to roughly 10%. The etiology most noted was a congenital defect in the muscular layer of the tunica media, a thinned adventitia, and thickened internal elastic lamina. Other possible causes are infectious diseases, trauma, and collagen vascular disorders, such as Ehlers-Danlos, type IV.

In 1998, *The New England Journal of Medicine* published the results of a multi-center, international study of unruptured intracranial aneurysm (ISUIA). A total of 1,449 patients in the 7.5 year retrospective arm, along with 1,172 followed prospectively came from the United States, Canada, and Europe. In the former group, 727 subjects had never suffered a SAH (group 1, incidental). The remaining 722 individuals had suffered a SAH and subsequently underwent obliteration of the offending aneurysm (group 2, additional). The authors calculated an aneurismal rupture rate of 0.05%/year for anterior circulation aneurysms < 10mm in the incidental group. Also in this group, but with lesions >10mm, the rupture rate was 1%/year, similar to the additional group with aneurysm >10mm. The previous SAH group with aneurysm < 10 had a rupture rate of 0.5%/year.[1]

The prospective arm followed 4,060 subjects for 5 years: 1,692 had no surgery; 1,917 underwent surgery, and 451 received endovascular treatment. The annual rates of rupture for the no surgery group with anterior circulation lesions were as follows: < 7mm—0% (2 patients); 7-10mm—2.6% (5 patients); 13-24mm—14.5%; ≥25mm—40%. In all, the ISUIA reports conclude that the rates of SAH from aneurysm <7mm and located in the anterior circulation of the internal carotid artery were inconsequential. The 5-year prospective study went further in concluding that treatment of lesions < 7mm in patients <50 years old would be of no benefit.[2]

Controversy

The validity and power of the ISUIA have been questioned since first published. Regarding the retrospective arm, critics view the two groups as different, based upon the increased predilection for recurrent SAH by the additional group. Second, Ausman et al. regard the SAH and aneurysm prevalence rates as greater than the true general population. The Olmstead County population study followed patients for 30 years and found the prevalence of aneurysms to be 83.4/100,000 people and an incidence of 9/100,000 people.[3] Third, the ISUIA relocated posterior communicating aneurysms from the anterior circulation to the posterior circulation, thus increasing the rate in the posterior circulation groups and underestimating the true rate in the anterior circulation. Historical, histologic and pathophysiologic evidence has seen posterior communication artery aneurysms as an extension of the internal carotid artery system.[4] Finally, the ISUIA may not have captured the "active" lesions, but rather the "stable" lesions capable of being followed for 7.5 years.[3]

Alternative models produced different rupture rates. Clark et al. gathered 11 studies outside of the ISUIA data and conducted a meta analysis, including Juvela's 40-year follow up of Finnish patients with intracranial aneurysms, described by anatomic vascular group. The aggregate anterior circulation groups had an annual rupture rate of 0.49%/ patient year. The anterior communicating artery anatomic group, sans Juvela's patients to avoid bias, had an annual rupture rate of 1.9%/patient year. Vindlacheruvu et al. used a mathematical model, again focusing upon anatomic groups of the anterior circulation and calculated an annual rate of rupture of 0.08%/patient year, using the data from the 5-year prospective ISUIA incidental group.[4]

Aneurysms from page 6

upon the aneurysm's size (< 7mm) and location (in the anterior circulation) as negative predictors of rupture. Given the uncertainty about the lesion, the airman underwent a formal cerebral angiogram, which revealed a vessel loop of normal internal diameter and character. Thus, the airman was free of the diagnosis of a cerebral aneurysm and received a 1st class medical certification without restrictions or follow up.

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Hypertrophic Cardiomyopathy

Case Report, by Eveline F. Yao, MD

Hypertrophic cardiomyopathy (HCM) is a cardiac disorder affecting 1 in 500 persons. The cardinal feature is (inappropriate) left ventricular hypertrophy in the absence of another identifiable cause. Sudden cardiac death is the most devastating and feared complication and may be the presenting manifestation. The striking heterogeneity and unpredictability of HCM make risk stratification problematic and imprecise. Cases must be deferred to the FAA for third- or limited second-class special issuance consideration. In general, first-class and full second-class medical certification is not permitted.

History

A 43-year-old airline pilot with 9,900 flying hours was diagnosed with hypertrophic cardiomyopathy, HCM, after referral for a stable murmur of mitral regurgitation that had been present for four years. He denied palpitations, syncope, shortness of breath, and chest pain, but after further inquiry, also noted decreased exercise tolerance over the past year and mild dyspnea on exertion with lightheadedness/dizziness on extreme exertion. His systolic murmur was first discovered on a routine aviation medical exam, and his previous echocardiograms revealed only mild MR.

There was no significant family history; both parents were alive and well, and he had one healthy 41-year-old brother.

On physical exam, a grade II/VI harsh systolic murmur was auscultated at the left sternal border with radiation to the cardiac apex. Squatting diminished the murmur intensity, while standing accentuated the murmur to a grade III/VI. A premature ectopic beat was also heard on exam. An ECG showed normal sinus rhythm, left ventricular hypertrophy by voltage criteria, and repolarization abnormalities.

The diagnosis of HCM was made by 2-D echocardiogram study with Doppler. The interventricular septum thickness measured 21 mm. There was systolic anterior motion of the mitral valve leaflet and mild mitral regurgitation. An estimated subaortic outflow gradient was 64 mm Hg at rest, with a peak dynamic gradient of 134 mm Hg following exercise. There was no evidence of ischemia. Holter monitor results showed normal sinus rhythm with occasional multiform ventricular ectopics and one four-beat run of monomorphic ventricular tachycardia at 160 bpm.

Hypertrophic Cardiomyopathy

HCM is a relatively common genetic cardiac disorder with a prevalence of 0.2% in the general population),[1,2] caused by a mutation(s) in one of ten genes encoding proteins of the cardiac sarcomere. The condition exhibits striking heterogeneity in expression, resulting in a spectrum of clinical symptoms and outcome, but is defined by the presence of marked left ventricular hypertrophy (≥ 15 mm) in the absence of a detectable cause.[3] Approximately 25% of patients will exhibit a dynamic obstruction across the left ventricular (LV) outflow tract and this syndrome has previously been termed idiopathic hypertrophic subaortic stenosis (IHSS) or hypertrophic obstructive cardiomyopathy. Because the majority of hypertrophy occurs without stenosis, HCM is the preferred term. The most feared complication of HCM is sudden cardiac death and is the most common etiology of sudden death in young trained athletes.[4] The annual mortality rate of HCM patients is 1%. It may also manifest as near-syncope or syncope. There are no tests to reliable stratify the risk level in HCM patients. This is a condition with an obvious increased risk of sudden incapacitation and unpredictability.

Treatment and Clinical Course

The airman elected non-surgical septal reduction therapy with intracoronary ethanol (alcohol ablation), resulting in a reduction in the outflow tract gradient from 74 to 0 mm Hg. At his one-year follow-up, echocardiogram showed hypertrophic (concentric) cardiomyopathy with basal and anterseptal akinesis, mitral valve leaflet anterior motion with no resting outflow tract gradient. The patient remained asymptomatic with normal exercise tolerance.

Clinical Presentation

Most patients with HCM are asymptomatic or only mildly symptomatic. One of four patients in an HCM cohort achieved normal life span, but symptoms can manifest from infancy to old age.[4,5] The first manifestation of HCM may be SCM. The condition is most often diagnosed in adults in their 30s and 40s and may be an incidental finding. The most common symptom is dyspnea (up to 90% in symptomatic patients).[3] Exertional chest pain, fatigue, presyncope, and syncope are also common.[6] The overall annual mortality rate is estimated at 1%.[7,8]

In general, an adverse clinical course will proceed along four predicted pathways: 1) high-risk, premature, sudden and unexpected death; 2) progressive symptoms of exertional dyspnea, chest pain, and impaired consciousness (presyncope, syncope, dizziness/lightheadedness) with normal LV systolic function; 3) progression to heart failure and LV systolic dysfunction; and 4) complications attributable to atrial fibrillation.[5]

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Diagnosis

The physical exam may be unrevealing. The precordial impulse is often prominent and displaced laterally. The ausculatory hallmark of HCM is the typically harsh, crescendo-decrescendo, systolic murmur at the left sternal border from the sum of the outflow tract turbulence, with attendant mitral regurgitation. The murmur is characteristically labile and enhanced with maneuvers that reduce preload or such as Valsalva, standing, or hypovolemia. Squatting decreases the intensity of the systolic murmur.

ECG abnormalities include left atrial enlargement, ST segment and T-wave abnormalities, pathologic Q-waves (most commonly in the inferolateral leads), and LVH by voltage criteria.[3,4]

Echocardiography establishes the diagnosis of HCM. Unexplained left ventricular wall thickness greater than 15 mm (normal = 11 mm) is sufficient to make the diagnosis.[6] Most have a disproportionate increase in thickness in the interventricular septum. About 25% of patients have dynamic left ventricular outflow obstruction caused by contact with the anterior leaflet of the mitral valve (which may be elongated and abnormally large) and the hypertrophied septum during systole, which narrows the outflow tract, causing a pressure gradient.[6] Mitral regurgitation usually accompanies outflow obstruction.

Physiology

HCM pathology is characterized by diastolic dysfunction.[3] There is markedly limited diastolic distensibility due to a stiff left ventricle and impaired filling, resulting in elevated diastolic filling pressures that can cause shortness of breath. Once symptoms are present, therapy is directed toward improving diastolic filling. A cardinal feature of HCM is the lability of the outflow gradient. Increased contractility, decreased preload and decreased afterload reduce ventricular volume and accentuate the apposition of the mitral leaflet against the hypertrophied septum. Some patients may not have a resting outflow gradient, but a gradient may be provoked by any of these mechanisms.

Treatment

Medical management is directed to alleviate symptoms, prevent complications, and reduce the risk of sudden death. Prophylactic treatment of asymptomatic HCM remains a subject of controversy. First-line pharmacologic treatment is directed to slow the heart rate, enhance diastolic filling, and decrease myocardial oxygen demand. Beta-blockers are generally the initial choice. If they are not tolerated, the calcium channel blocker, Verapamil, can be used. The class IA antiarrhythmic agent disopyramide (negative inotropic properties) may be used, often in combination with a β-blocker.[6,8]

Drug refractory patients (persistent symptoms or resting gradient ≥ 50 mm) will need other invasive intervention. Surgery, septal myectomy involving resection of a small piece of basal septum, is the "gold standard." Surgery will reduce the gradient to < 10 mm Hg. in 90% of patients with long-lasting improvement in symptoms and no recurrence in obstruction. Alternatives to surgery include dual-chamber pacing and alcohol ablation (percutaneous transcoronary septal ablation). Randomized clinical trials have shown that improvement (short-term) with dual-chamber pacing is due largely to a placebo effect, since residual gradients remain 30-50 mm Hg.[5,8] Alcohol ablation (introduced in 1995) involves infusion of absolute alcohol into targeted septal perforating artery or arteries producing a controlled myocardial infarction. Alcohol septal ablation produces results similar to surgical myectomy in short- and medium-term follow-up. The primary disadvantage of alcohol ablation is the potential long-term mortality due to arrhythmia-related cardiac events. The MI and scarring adds a permanent arrhythmogenic substrate.[4,8]

Risk Stratification

All patients, once diagnosed, should be evaluated with a history, Holter monitoring, and maximal exercise testing.

The positive predictive value of any single factor is low (secondary to low event rates); however, the absence of multiple factors is a better predictor of a good prognosis and identifies a lower-risk group.

A high negative predictive value (at least 90%) suggests that the *absence* of risk factors can stratify adults into *low risk* if they demonstrate 1) no or

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Table 1. Risk Factors for Sudden Death in HCM (adapted from 5,8,9,10)

Prior cardiac arrest (ventricular fibrillation)
Spontaneous sustained ventricular tachycardia
Family history of HCM related death*
Otherwise unexplained syncope
Nonsustained ventricular tachycardia (Holter)^
Extreme left ventricular hypertrophy (> 30 mm)
Left ventricular outflow pressure gradient (> 30 mm Hg)
Abnormal blood pressure response to exercise†
High-risk specific mutations in sarcomeric proteins
Microvascular obstruction/limited myocardial flow reserve§

*2 or more first-degree relatives

^3 consecutive beats or more and of at least 120 beats/min

†Failure of BP to rise 25 mm from baseline or a fall in BP during exercise

§Perfusion defects on nuclear imaging

Eveline F. Yao, MD, was a resident in aerospace medicine when she wrote this case report at the Civil Aerospace Medical Institute.

Sarcoidosis

Case report, by Trey Milligan, MD, MPH

In the United States, sarcoidosis is 10 times more prevalent in African-Americans than in Caucasians; people of Scandinavian descent also have a high incidence of the disease. More women than men contract the disease, and the onset is most often between the ages of 20 and 45. Although many patients with sarcoidosis are asymptomatic, diverse and clinically important syndromes do result. Sarcoid can present clinical features and symptoms that are incompatible with safely operating an airplane or performing airman duties.

History

In February 1996, a 34-year-old African-American airman with a first-class medical certificate, was working for a commercial air cargo company when he experienced cough, dyspnea, weight loss, myalgia, and malaise of six weeks' duration. The illness was refractory to several regimens of antibiotic treatment prescribed by his primary care physician. He was referred to a pulmonary specialist for consultation. His physical exam was remarkable for bibasilar rales but was otherwise within normal limits. Specifically, the cardiac exam revealed a regular rate and rhythm, with no abnormal sounds. There were no acute skin lesions. Echocardiogram was within normal limits, with an ejection fraction of 60%. ECG was normal. Chest X-ray revealed hilar lymphadenopathy and parenchymal pulmonary infiltrates. Fiberoptic bronchoscopy and transbronchial biopsy revealed noncaseous granulomas with negative special stains for fungi and acid-fast bacillus, consistent with pulmonary sarcoidosis. Initial pulmonary functions tests (PFTs) showed a forced vital capacity (FVC) of 2.4 liters (51% of predicted) and an FEV1 of 2.63 liters (58% of predicted). Prednisone 40mg a day provided almost immediate cessation of cough and a progressive decrease in dyspnea on exertion.

Over the next 4 months, the airman was maintained on oral prednisone, with gradual tapering as compatible with symptomology. Repeat PFTs in June 1996 revealed a FVC of 4.76 liters (63% predicted) and an FEV1 of 3.84 (70% of predicted). Room air oxygen saturation was 97% with very little, if any, dyspnea on exertion. One year later, after full disclosure to the FAA regarding his pulmonary condition, he received a special issuance second-class medical certificate with restriction to flight engineer duties only, valid for six months. Repeat application in November 1996 required completion of repeat PFTs. At that time, FVC was 2.67 liters (56% of predicted) and FEV1 was 2.36 (62% of predicted). Another second-class special issuance medical certificate was granted six months later, and yet another one six months after that.

In March 1998, the airman applied for a second-class medical renewal. The results of PFTs included with his application revealed a FVC of 2.38 liters (51% of predicted) and FEV1 of 2.09 (55% of predicted). This represented an overall decline since initial diagnosis, in spite of oral prednisone treatment ranging from 10-20mg a day and long-acting beta-agonist bronchodilator therapy. He was denied a

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Sarcoidosis

is a disease characterized by the formation of noncaseating granulomas, particularly in the lungs, but also throughout the rest of the body. The precise cause remains unknown, but activation of T-cell lymphocytes in the lung plays an important role in the pathogenesis of granulomas. In the United States, sarcoidosis is 10 times more prevalent in African-Americans than in Caucasians; people of Scandinavian descent also have a high incidence of the disease. More women than men contract the disease, and the onset is most often between the ages of 20 and 45.

Although many patients with sarcoidosis are asymptomatic, diverse and clinically important syndromes do result. Granuloma formation in the lung can be especially damaging, as can involvement in a number of other organ systems (e.g., eye, gastrointestinal tract, and heart). Once the diagnosis is established, the prime management medication is corticosteroid therapy.

Clinical Manifestations

The most common presentation, especially in young adults, is bilateral hilar adenopathy, which occurs in 50% of patients and is often detected on routine chest radiography. About 25% present with bilateral hilar adenopathy and pulmonary infiltrates, and 15% present with infiltrates alone. Disease in the hilum is not associated with invasion or compression of the bronchi or nodal calcification. Erythema nodosum or uveitis (manifested by red, watery eyes) may accompany hilar adenopathy. Some patients exhibit cough, shortness of breath, and wheezing or chest discomfort, in addition to constitutional symptoms of fever, malaise, and fatigue, as did the airman in this case study.

Although pulmonary symptoms are the most frequent, sarcoidosis may present with extrathoracic disease, including hepatomegaly, splenomegaly, or uveitis. Other presenting manifestations include fever of unknown origin, granulomatous hepatitis, salivary and lacrimal gland enlargement, arthritis, peripheral adenopathy, and skin lesions.

Treatment

The goals in the treatment of sarcoid include relief of symptoms and prevention of significant impairment of organ function. Principal treatment is systemic corticosteroid therapy. The view that emerges from available studies is that treatment should be reserved for patients who are symptomatic and show evidence of active pulmonary disease (dyspnea on exertion, abnormal pulmonary function studies, or infiltrate on chest X-ray films). The airman in this study fit this criterion and, therefore, was maintained on oral prednisone 10-20mg a day, as needed to abate symptoms.

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special issuance medical certificate under 14 Code of Federal Regulations (CFR) Section 67.401 of the Federal Aviation Regulations.

Disposition

Sarcoid presents clinical features and symptoms that are incompatible with safely operating an airplane or performing airman duties. Myocardial features of sarcoid that may adversely affect an airman's ability to perform required tasks while in flight include incapacitation and sudden death. To illustrate, in a series of 250 sarcoid patients followed for several years, the following complications were noted: complete heart block (49), premature ventricular contractions and ventricular tachycardia (48), myocardial disease (43), sudden death (37), bundle branch block (33), supraventricular arrhythmia (23), valvular lesions (21), and pericarditis (6).[1]

Pulmonary manifestations of sarcoid may also limit an airman's effectiveness. Reductions in FVC and FEV1 may accompany sarcoid, despite medical treatment. Current FAA medical standards require FVC and FEV1 values to be no less than 50% of predicted. Additionally, an absolute upper limit of oral prednisone therapy is set at 20mg per day. Many patients with sarcoid are disqualified from airman medical certification for these factors.

The airman in this case study was denied special issuance of a second-class medical certificate under 14 Code of Federal Regulations (CFR) Section CFR 67.401 by the Aerospace Medical Certification Division, citing that his medical condition precludes the safe performance of airman duties under any condition that could reasonably be prescribed. Specifically, his deteriorating FVC/FEV1 measurements and reliance on maximum allowable oral prednisone doses were emphasized in his denial for medical certification.

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Dr. Trey Milligan was a resident is aerospace medicine when he researched this case report at the Civil Aerospace Medical Institute.

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only mild symptoms of chest pain or exertional dyspnea (NYHA functional classes I and II); 2) absence of family history of premature death from HCM; 3) absence of syncope judged to be HCM-related; 4) absence of nonsustained VT during ambulatory (Holter) ECG; 5) outflow tract gradient at rest of less than 30mm Hg; 6) normal or relatively mild increase in left atrial size (less than 45 mm); 7) normal blood pressure response to upright exercise; 8) mild LVH (wall thickness less than 20 mm).[5] Younger patients, previously asymptomatic, are at higher risk of SCD. Older patients, by surviving, declare themselves to be at lower risk.[4]

Patients at increased risk of SCD should be considered for implantation of an automatic defibrillator, the treatment of choice for prevention of sudden death.[10]

Aeromedical Disposition

The FAA is duly cautious in issuing medical certificates in patients with HCM,[11,12] since it is an unpredictable disease. The negative predictive value (at least 90%) of the *absence* of the above-listed risk factors can identify patients at low risk of SCD[5]; however, there may still be an unacceptable risk for nonfatal, but transient and prolonged, sudden incapacitation related to an altered level of consciousness (near-syncope, syncope). In this illustrative case, alcohol ablation of septal hypertrophy may obliterate the outflow gradient. However, alcohol ablation is a newer treatment option and has not yet sustained the test of long-term post-procedural follow-up and/or randomized controlled studies. The potential for seeding an already pro-arrhythmic heart with permanent, electrically unstable scar tissue is also an area of concern in long-term risk assessment.[1,4,6] An airman is ineligible for medical certification under 14 CFR 67.111(b), 67.113(b), 67.213(b) and 67.313(b). However, 14 CFR 67.401 provides authority for special issuance medical certification.[13] Current FAA guidelines have been developed for third- and limited second-class special issuance medical certification.

Case Outcome

The airman was denied special issuance of a first-class airman medical certificate.

This is a commercial pilot with risk factors not amenable to low-risk stratification. Despite elimination of outflow tract obstruction with alcohol ablation (unproven in long-term studies), he remains at risk for sudden incapacitation.

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CAMI Researchers Receive Awards From AsMA

Shown (R-L) are Drs. Fred Tilton (Federal Air Surgeon), Eduard Ricaurte, William Collins, Carol Manning, Estrella Forster, and Melchor Antuñano (CAMI Director and AsMA Past-President)

The FAA Office of Aerospace Medicine (OAM) was well represented at the awards ceremony of the Aerospace Medical Association (AsMA) during its 78th Annual Scientific Meeting. Dr. **Carol Manning**, Manager of the Training and Organizational Research Laboratory at the Civil Aerospace Medical Institute's (CAMI's) Aerospace Human Factors Research Division received the **Raymond F. Longacre Award** in recognition of her outstanding accomplishments in the psychological aspects of aerospace medicine. Dr. **Estrella Forster**, Aeromedical Research Scientist at CAMI's Aerospace Medical Research Division received the **Eric Liljencrantz Award** for her excellence as an educator in aerospace medicine. Dr. **Eduard Ricaurte**, Contract Scientist at CAMI's Aerospace Medical Research Division received the **John A. Tamisiea Award** for his outstanding contributions to the art and science of aviation medicine in its application to the general aviation field. Dr. **William Collins**, former CAMI Director, received the **Louis H. Bauer Founder's Award** for the most significant contribution in aerospace medicine. In addition, Dr. Forster was elected President of the Life Sciences and Biomedical Engineering Branch of AsMA, and Dr. **Lawrence Bailey**, Personnel Research Psychologist at CAMI's Aerospace Human Factors Research Division was elected President of the Aerospace Human Factors Association.

Air Venture 2007 Medical Booth Well Attended

WORKING THE BOOTH. (L-R) Drs. Silberman, Tilton, Kowalsky, and Jackson.

OAM medical staff, including the Federal Air Surgeon, supported a medical information booth at the Experimental Aircraft Association's 55th annual convention and fly-in at Oshkosh, Wis.

The OAM booth featured the popular presence of laptop computers configured for online remote access to the FAA's medical certification system, thereby allowing 900-plus booth visitors the opportunity to obtain a personalized interview and review of real-time information regarding the status of their medical certification application, as well as briefings on the recently introduced MedExpress feature of airman certification.

The Civil Aerospace Medical Institute-based Airman Education program, represented by **Rogers Shaw II** and his crew, offered hundreds of pilots a "flying experience" in their spatial disorientation demonstrators. Federal Air Surgeon Dr. **Fred Tilton** visited the booth, participated in the "Meet the Administrator" session, and also gave a presentation on "Hot Aeromedical Issues."

Dr. **Warren Silberman**, Manager of the Aerospace Medical Certification Division in Oklahoma City, assisted the information booth staff and also spoke on "Problem Medicals – How to Prepare."

The OAM booth was sponsored and staffed by FAA Great Lakes Region Medical Office staff.

This year, airshow officials welcomed about 560,000 visitors from 36 nations and 10,000 aircraft over the course of the 7-day convention. The event featured 784 commercial exhibitors and nearly 1,000 forums, workshops, educational seminars, and special activities for aviation enthusiasts. In addition to displays of aircraft with the latest flight innovations and legendary aircraft of aviation's first 100 years, visitors enjoyed a daily airshow, a U-2 spy plane on display, and a flyby of USAF F-22 Raptors.

—Thanks to Dr. Nestor Kowalsky, Great Lakes Regional Flight Surgeon, for contributing this report.