A viation safety is dependent in part on the cardiovascular health of pilots. Hypertrophic cardiomyopathy (HC) is an often unpredictable, genetic cardiac disease associated with a risk for sudden cardiac death, or periods of impaired consciousness. A recent precedent-setting decision of the National Transportation Safety Board medically certified a commercial airline pilot with HC, despite the potential for sudden unexpected incapacitation in the cockpit implicit for this disease and the possible risk to public safety. Because airline pilots have a unique occupation which assumes professional responsibility for large numbers of passengers, this decision may be regarded as controversial.

Hypertrophic cardiomyopathy (HC) is a relatively common genetic heart disease, and an important cause of impaired consciousness and disability at any age. Sudden cardiovascular events in HC are known to be associated with a high degree of unpredictability. HC is recognized as the single most common cause of sudden death in young trained athletes and the risks associated with such extreme lifestyles have stimulated expert consensus criteria for disqualification from competitive sports.

The relation between underlying heart disease and unexpected sudden death and impaired consciousness has also been a concern for individuals with other unusual occupations or lifestyles such as commercial pilots. Commercial airline travel presents unique circumstances in which hundreds of passengers are dependent on the general medical condition and cardiovascular status of the pilot. Accordingly, the Federal Aviation Administration (FAA) has established medical standards for pilot certification in an effort to ensure aviation safety. Although sudden pilot incapacitation due to cardiovascular disease appears to be a relatively uncommon primary cause of air carrier accidents, such incidents are known to occur and public awareness has recently been heightened regarding the safety of air travel, including the medical histories of pilots.

A recent judicial proceeding under the jurisdiction of the National Transportation Safety Board (NTSB) involving a commercial airline pilot with HC has raised a number of issues related to airmen and cardiovascular disease, including whether it is safe and reasonable for airmen with obstructive HC to be medically sanctioned to operate commercial aircraft.

CASE REPORT

The 45-year-old airline captain for a major carrier was tentatively diagnosed with obstructive HC, based on a systolic heart murmur and an abnormal electrocardiogram during a routine examination by a FAA-designated aviation medical examiner. As a result, the examiner deferred issuing an airman medical certificate, thereby suspending his active flight status at that time. Subsequently, the pilot submitted to noninvasive testing including echocardiogram, 12-lead electrocardiogram and thallium-201 stress test under the supervision of 2 cardiovascular specialists. A definitive diagnosis of HC associated with left ventricular outflow obstruction was made for the first time.

Data assembled from all medical evaluations conducted over an 8-month period documented the following clinical profile. The pilot (height: 72 in; weight: 190 lbs) had always been asymptomatic, even during periods of stress associated with his duties as a pilot during the 20 years he held an unrestricted airman medical certificate. During high school he had participated in several competitive sports without difficulty. There was no history of systemic hypertension (blood pressure, 130 to 140/70 mm Hg), nor family history of heart disease or premature sudden death.

A grade 3/6 harsh systolic ejection murmur was audible periodically at the left lower sternal border and apex (and increased with standing). Electrocardiogram showed T-wave inversion in leads V1 and V2, abnormal Q waves in II, III and AVF, deep S waves in the right precordial leads consistent with left ventricular hypertrophy, and left atrial enlargement. Ambulatory 24-hour (Holter) electrocardiogram documented 38 isolated premature ventricular complexes (48-hour Holter showed 347 premature ventricular complexes and 2 couplets). On 3 occasions he performed treadmill exercise (Bruce protocol) for 8 to 12 minutes with normal blood pressure response, absence of arrhythmias, and peak oxygen consumption of 29 ml/kg/min (80% of predicted). Thallium-201 perfusion scan showed no evidence of myocardial ischemia or scarring.

Echocardiograms demonstrated asymmetric left ventricular hypertrophy limited to the anterior ventricular septum (thickness 20 mm). The left ventricle was hyperdynamic; end-diastolic cavity dimension was 44 mm. Left atrial dimension was reported variously.

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whether the pilot, as a result of HC, was disqualified from holding unrestricted airman medical certification. In the United States, such certification is necessary to exercise the privileges of a commercial airline transport pilot. FAA regulations provide that “. . . applicants for an airman medical certificate cannot have a disqualifying condition, including organic, functional or structural disease, defect or limitation that may reasonably be expected to make the applicant unable to perform piloting duties.” FAA regulations regarding airman medical certification are independent of whether another pilot (i.e., copilot) is present in the cockpit who would theoretically be capable of controlling the aircraft should the pilot become incapacitated.

The specific medical dispute focused on the likelihood that HC was of sufficient magnitude to cause cardiovascular-related sudden incapacitation in the petitioning pilot while in the cockpit. These considerations were not limited to sudden death, but included any transient or prolonged impairment in consciousness and any other event (such as sustained arrhythmia) that could cause substantial distraction or discomfort in the airman and reduce performance, judgment, and problem-solving capability while operating the aircraft.

The FAA presented the following arguments. In many respects HC is an unpredictable disease. Although this pilot would appear to be at relatively low risk for sudden death (due to the absence of risk factors such as family history of HC-related death, syncope, extreme left ventricular hypertrophy or non-sustained ventricular tachycardia on ambulatory Holter electrocardiogram), nevertheless, there were compelling reasons to regard him to be at an unacceptable risk for nonfatal sudden incapacitation.

This latter argument focused largely on the predisposition of the pilot to develop atrial fibrillation, based on marked left atrial enlargement (i.e., chamber dimension up to 53 mm). Because of the likelihood that AF would occur in the near future, HC presented a significant risk for syncope, near-syncope, and prolonged dizziness as well as acute onset of dyspnea, chest pain, palpitations, a peripheral embolic event, and possibly even sudden death; each of these adverse cardiovascular consequences frequently accompany the sudden onset of atrial fibrillation and would be incapacitating in the aviation environment.

In contrast, the medical expert for the pilot argued that he had a “benign form of HC” and the risk for either sudden death or nonfatal impaired consciousness and incapacitation was very low . . . “and not dissimilar to that of a well-matched normal male ex-smoker who does not have HC.” Furthermore, the opinion was advanced that all complications of HC (including sudden death and impaired consciousness) are predictable with a high degree of reliability. Offered in support of this view was the pilot’s generally excellent physical fitness and asymptomatic state, the likelihood that left ventricular wall thickness would not progress at his age, the absence of traditional sudden death risk factors or arrhythmias with noninvasive testing, and the contention that the pilot’s left atrial size did not represent a risk marker for atrial fibrillation.

Five months after the evidentiary hearing, the NTSB administrative law judge issued a decision and order affirming FAA denial of the pilot’s application for an unrestricted medical certification, stating that the pilot did not establish that his risk for experiencing an incapacitating event due to HC was within acceptable limits and, therefore, that he had not “. . . proved by a preponderance of the substantial, reliable and probative evidence that he is qualified for the certificate.”

The pilot appealed the decision of the administrative law judge to the 5 member NTSB appellate board. Four members reversed the order of the judge, concluding that the pilot had “. . . carried out his burden of demonstrating that he is medically qualified to hold an unrestricted medical certificate.” In reversing the law judge’s decision, the appellate board found that the medical testimony presented for the pilot was “. . . as a whole. . . more persuasive” because it was “. . . more focused on the [pilot’s] actual morphology and symptoms.” Also, it was more “. . . cogent and persuasive . . .” not to relate the pilot’s medical data to that of large HC patient populations for estimating risk for sudden incapacitation (as the FAA had argued). One board member filed a dissenting opinion stating that HC “. . . could well present an unacceptable risk of sudden incapacitation while at the controls of an aircraft” and that “the pilot was not medically qualified for an unrestricted first-class medical certificate.”

**DISCUSSION**

The NTSB decision in this case established an important precedent, by rejecting the position taken by the FAA (and affirmed by the administrative law judge) that a pilot with HC and a clinical profile similar to this case is ineligible for the medical certification necessary to pilot commercial aircraft. Moreover, the NTSB disputed the substantial concern raised that sudden and unexpected incapacitation
could well occur in the cockpit when a pilot has HC. A cardiovascular event related to HC could, of course, represent a significant risk to commercial airline passengers, who would be completely uninformed regarding the medical status of their pilot.

Sudden death due to ventricular fibrillation is relatively uncommon within the general HC population. Both sides in this dispute agreed that the pilot was generally at relatively low risk for sudden death, given his particular clinical profile. In contrast, transient or prolonged (but nonfatal) incapacitation may occur in up to 50% of patients with HC at some point in their clinical course due either to syncope, near-syncope, or prolonged dizziness. Such clinical events can, however, also be associated with sudden onset of primary ventricular tachycardia/fibrillation or on occasion when supraventricular tachyarrhythmias (e.g., atrial fibrillation) trigger ventricular fibrillation. The potential importance of HC-related impaired consciousness is obvious when associated with the unique stresses incumbent upon airline pilots, particularly during the critical phases of take-off, final approach and landing, as well as with emergencies and other unexpected contingencies.

Acute onset of atrial fibrillation would appear to be the most likely potential trigger for sudden incapacitation in this pilot, given his substantial left atrial chamber enlargement of up to 53 mm. The average left atrial dimension in HC patients experiencing paroxysmal or chronic atrial fibrillation has been reported to be only 43 mm, or 51 mm and there is a direct relation between left atrial size and the likelihood that atrial fibrillation will develop.

Furthermore, data presented as part of the NTSB hearing showed that the risk of atrial fibrillation occurring in an asymptomatic HC patient with a left atrial size of 53 mm (such as in the pilot) is 20% over 3 years and 30% over 5 years.

Left ventricular outflow obstruction is a strong and independent predictor of HC mortality. Therefore, the documentation of an outflow gradient of 45 mm Hg under resting conditions in the pilot strongly suggests that even more marked subaortic gradients would be easily provoked with stress (similar to the result of the dobutamine infusion in this case), potentially predisposing to a deleterious sequence of pathophysiologic events including sudden incapacitation. Furthermore, the association of outflow obstruction and atrial fibrillation has been shown to be particularly unfavorable in HC.

Clinical data addressing the level of risk for all forms of sudden incapacitation are not presently available for each possible scenario within the broad and heterogenous HC disease spectrum. However, the present case in which medical certification was conferred on a pilot with HC sets a precedent and standard for future eligibility decisions. Consequently, other pilots with similar or different expressions of this disease (presumably including those with even greater risk for sudden incapacitation) may be viewed as an acceptable risk for certification, equivalent to pilots free of any medical problems.

The present discussion focuses on commercial pilots responsible for large passenger and cargo-carrying aircraft. However, we believe that these issues and related medical standards regarding certification should similarly apply to all pilots with HC, including those of higher risk (single pilot) small private aircraft.

**Conclusions**

The precedent-setting administrative decision on the part of the NTSB to medically certify commercial airplane pilots with a cardiovascular disease such as HC, harboring the potential for sudden and unexpected incapacitation in the cockpit, may be regarded as controversial. Because of the unique professional responsibilities attributable to airline pilots, a measure of caution—and conservative and prudent eligibility decisions—are in the best interests of the public and aviation safety, particularly when involving diseases with a large measure of unpredictability such as HC.

24. Title 14 of the Code of Federal Regulations, sections 67.113(b), 67.213(b) and 67.313(b).