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Fatal Light Aircraft Accidents in Ontario: A Five Year Study

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ABSTRACT: Fatal civil aviation crashes in Ontario from 1985 to 1989 were studied. Data regarding accident circumstances, injury patterns and medical factors (disease, alcohol/drugs) which could have contributed to accident causation was obtained from a review of the files of the Chief Coroner for Ontario in Toronto and the aviation occurrence reports of the Transportation Safety Board of Canada. Forty-seven crashes involving mainly general aviation type aircraft but also 2 gyroplanes, 2 ultralights and a glider were reviewed. About half occurred during the cruise phase of the flight. Seventy (40 pilots; 30 passengers) of the 98 occupants died. The bodies of 68 victims were recovered; 63 were dead at the scene and 5 survived up to ten hours after impact. Multiple trauma killed about half of all the victims (n = 34); 29% (n = 20) drowned; 16% (n = 11) and 3% (n = 2) died of head/neck injuries and coronary disease respectively. Neck trauma was observed mostly in pilots and was the most frequent major blunt trauma injury in drowning victims. Passengers sustained relatively more craniofacial fractures and abdominal/retroperitoneal trauma. Pilot error was the most frequent cause of crashes (55%; 26/47 impacts) followed by mechanical failure (15%; 7/47) and adverse weather/environmental conditions (11%; 5/47). Coronary artery disease incapacitated two pilots (4% of crashes) and ethanol intoxication was implicated in two other accidents. Other drugs did not appear to be a definite factor in accident causation.

KEYWORDS: forensic science, pathology and biology, light aircraft, fatality

In Ontario, fatal civil aircraft accident investigation is the joint responsibility of the Office of the Chief Coroner for Ontario and the Transportation Safety Board of Canada (TSB). If there is a criminal investigation, the police also become involved. The TSB, similar to the National Transportation Safety Board (NTSB) in the United States, is responsible for determining the cause of an aircraft accident (1,2). Board investigators in Canada and the United States have the authority to take custody of and examine all wreckage at the scene, to question witnesses and to study any pertinent information related to the crash with assistance from the police (1,2,3). The aim is to promote aviation safety and to recommend measures that could reduce safety deficiencies. Board investigators also have the legal right to order autopsies but this aspect of the investigation is usually handled by coroners or medical examiners (1,2,3). Although coroners, in Ontario, may seize and examine wreckage (4), generally this role is assumed by the Board examiner. Coroners or medical examiners and Board investigators will share information derived from their respective investigations.

During the investigation of mishaps involving fatal light aircraft (as defined in **Methods**), a coroner or medical examiner can order an autopsy to determine the cause of death. In Ontario, depending on the circumstances, a coroner can conduct an inquest (4). Post-mortem findings can be used to identify the deceased (5,6). The documentation of injuries can help in crash reconstruction (7) and their interpretation can be assisted by scene visitation by the coroner, medical examiner or pathologist. Observation of certain injuries can lead to improved structural and safety features which could prevent deaths (8,9). Knowledge of the trauma sustained in an aircraft impact can focus the clinical management of injured survivors (9). An autopsy performed on a light aircraft fatality should be complete as possible subject to the state of the body. Not only should injuries be accurately noted but also careful internal dissection (10,11) and collection of appropriate toxicologic samples (12) can address whether medical factors related to the pilot, such as incapacitation by disease or impairment by ethanol or drugs could have played a role in causing an accident (13,14,15). The interpretation of the significance of a post-mortem finding in accident causation should be integrated with other pertinent information gathered during the investigation (16). Communication between the various investigators is essential.

Methods

Fatal light aircraft accidents occurring in Ontario from 1985 to 1989 and investigated by the Chief Coroner's Office were reviewed. The aircraft crashes studied involved not only general aviation type planes but also gyroplane, ultralight and glider mishaps. General aviation craft included single and multi-engine, piston powered planes with a maximum take-off weight less than 5700 kg. (about 12500 lb.). Military accidents were excluded. "Mass" disasters (arbitrarily defined as more than six deaths) involving commercial airlines were not considered. Reports of autopsies, if done, coroners' findings, police investigations and TSB aviation occurrence reports, if investigated by this agency, gave the following information: the number of survivors and fatalities (pilots and passengers), type of pilot licence and licence validity, age and sex of the dead victims, method of identification, the state of body preservation, injuries observed in fatal cases, restraint system use, presence of disease and toxicology results in deceased pilots, cause of death, survival time, time and location of the crash,

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phase of flight during which crash occurred, witness accounts, flight purpose, occurrence of fire and accident cause. Toxicological analyses were done at either the Centre of Forensic Sciences (Toronto, Ontario) or the Civil Aviation Medical Unit (Downsview, Ontario).

Results

During the five year study period, 47 fatal crashes (mainly general aviation planes as well as 2 ultralights, 2 gyroplanes and 1 glider) occurred (Table 1). All of the aircraft had a single pilot with the exception of a pilot and co-pilot involved in a medical evacuation. Based on forty-three TSB investigations, the types of licences held by pilots involved in these crashes were either private (27), student (3), commercial (12, including co-pilot of medical evacuation flight) and airline transport (2, including pilot involved in medical evacuation). Three licences (2 private, 1 student) were invalid because of expiration of medical certification. Another private pilot's licence had been suspended for one year due to heart disease but was reinstated by a medical review panel. One private pilot was declared unfit because of a cardiac arrhythmia when he initially applied for a licence (see *Cause of Death, Disease*—Case 2). The licence of one commercial pilot had expired. Another, licensed in the United States, was not authorized to fly in Canada.

Of the 98 people involved in these impacts, 70 (40 pilots, 30 passengers) died. Five of the deceased pilots were in craft other than the usual general aviation type. All of the deceased pilots were males. Their age range was 25 to 63 years, average age 43 years. The age distribution of the dead pilots was: 25 to 34 years—9 victims; 35 to 44 years—12 victims; 45 to 54 years—14 victims and 55 to 64 years—5 victims. About two-thirds of the dead pilots were 35 to 54 years of age. The deceased passengers consisted of 25 men (aged 16 to 69 years) and 5 women (aged 24 to 57 years). Complete autopsies including craniocervical examination were done on 60 victims (autopsy rate = 86%) by 39 pathologists. Identification of the deceased was either by visual recognition (60 cases), dental examination (5 cases) or both (2 cases). Two bodies were not recovered. In one other case, only partial remains were found.

Four crashes occurred in winter (December/January/February); 11 in spring (March/April/May); 18 in summer (June/July/August) and 14 in the fall (September/October/November). Most of the crashes happened in the afternoon and early evening hours (Table 2).

Thirty-one crashes occurred on land (9 in remote or sparsely populated areas) and 16 in water, that is, lakes, rivers and swamps (9 in remote or sparsely populated locations). Recovery of bodies was delayed more than 24 hours in 10 crashes involving 16 victims. Nine of these incidents happened in remote areas. The other occurred in the Niagara River and body retrieval of the two victims was delayed 18 days and 22 days. Five victims, recovered 1 to 3 days following the crash, were not decomposed. Some of these accidents had occurred in autumn. Eleven individuals, found 2 to 22 days later, were minimally to severely decomposed.

At least 13 of the flights (28%) were related to work or business rather than personal or private use and involved 27 occupants, 23 of whom (33% of total fatalities) died. They occurred in the following situations: test flight to check repaired nose gear function; owner flying to open his lodge; plane pulling glider developed mechanical problem; plane preparing for glider towing activities; two crashes into lakes during charter flights; medical evacuation; engine failure during a chartered fishing trip; attending a meeting and a small commercial aircraft returning to home base. In three flights, the business purpose of the flight was not specified.

Cause of Death

Blunt trauma was the cause of death in about two-thirds of the cases (Fig. 1, addition of various percentages does not equal 100% because of rounding). About three-quarters of these trauma deaths were from multiple injuries; the remainder sustained major injury localized to the head and neck area. Drowning accounted for 29% of the fatalities (20 victims) followed by disease (3% or 2 victims). Most of the crash victims in light aircraft other than general aviation planes sustained serious blunt trauma (multiple injuries = 4, drowning = 1). Of the 68 victims examined, 63 were dead at the scene and 5 survived up to 10 hours after impact.

Injuries

Using information from reports of complete autopsies (n = 59; 1 report unavailable), specific injuries in all occupants were grouped according to cause of death (Table 3). The percentage of pilots

TABLE 1—*Crash statistics.*

Year	Crashes[1]	Occupants[1]	Deaths (Pilots:Passengers)	PM (% rate)[2]
1985	9 (U = 2, GL = 1)[3]	16	15 (9:6)	11[4][5]
1986	8	16	9 (7:2)	9
1987	8 (GY = 1)[3]	15	12 (8:4)	12
1988	8 (GY = 1)[3]	12	10 (7:3)	10
1989	14	39	24 (9:15)	18[6][7]
Total	47	98	70 (40:30)	60 (86%)

[1]Crashes (#) = # occupants each: (20) = 1; (15) = 2; (5) = 3; (3) = 4; (3) = 5; (1) = 6.

[2]PM—number of complete autopsies; (%)—percentage of all deaths having complete autopsies.

[3]U = ultralight; GL = glider; GY = gyroplane.

[4]Autopsies were not performed on 3 passengers who sustained severe multiple traumatic injuries during a single aircraft crash in a remote area of Northern Ontario. Injuries were documented by external examination at the scene by the local coroner.

[5]Cause of death unknown in a female passenger whose autopsy was limited to examination of the right leg and portion of right pelvis recovered from water 18 days after the crash.

[6]Cause of death not determined in a pilot and passenger whose bodies were not recovered.

[7]Excludes limited autopsies on 4 passengers who died of multiple trauma.

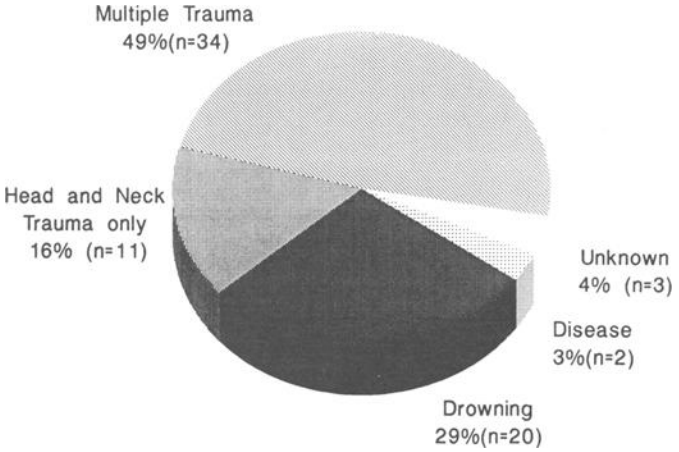
TABLE 2—*Time of crash.*

Time of Day (h)	No. of Crashes
2400–0359	1[1]
0400–0759	2
0800–1159	6
1200–1559	20
1600–1959	12[2]
2000–2359	6[3]

[1]One plane crashed into a lake after fuel supply was exhausted because of severe headwinds.

[2]Includes a plane which struck a utility line due to poor visibility. The pilot was not authorized for night flying.

[3]See Case 3 under *Ethanol* in *Results*. Poor visibility likely played a role in two other crashes.



compared to passengers dying of either drowning (58%; 11/19) or multiple trauma (63%; 17/27) was similar to the proportion of pilot deaths (57%; 40/70). A greater percentage of pilots (73%; 8/11) died of head and neck trauma. Cutaneous and soft tissue trauma of the head, neck and extremities was frequent in all of the fatalities regardless of the cause of death.

Multiple Trauma

At least about one-half of these deaths showed craniofacial, rib and extremity fractures, intracranial hemorrhage, hemothorax, pulmonary contusion/laceration, pericardial, cardiac, aortic and hepatic lacerations. Of those victims who had facial fractures, about one-half were multiple and the remainder were limited to either the nose or mandible or both. Aortic tears were observed either in the descending thoracic segment distal to the left subclavian artery (10 cases: 7 pilots, 3 passengers) or ascending aorta (1 pilot) or both (1 pilot). In one case, lacerations were observed in the thoracic and abdominal portions. The exact locations were not specified. Bronchial avulsion was noted in 2 cases (pilot and passenger). Passengers compared to pilots appeared to suffer a disproportionate number of craniofacial fractures and abdominal/retroperitoneal trauma. The latter included abdominal cutaneous/soft tissue injury, hepatic, splenic and mesenteric lacerations associated with hemoperitoneum, gastrointestinal trauma (gastric tears in a pilot, bowel lacerations in a pilot and a passenger and small bowel contusions in 1 pilot and 3 passengers) and renal/perinephric injury (cortical lacerations in a pilot of an ultralight; cortical and renal artery lacerations in a pilot; torn renal artery and vein in a pilot and a passenger and perinephric hematoma in 2 passengers). Other abdominal and retroperitoneal visceral injuries observed were pancreatic lacerations (2 pilots) and an adrenal tear (a pilot). Cervical spine fracture-dislocation, brainstem and cervical cord injuries were found almost entirely in pilots. In four cases, the level of cervical spine injury was specified (atlanto-occipital dislocation, two; C_1, C_2 combined with C_7, C_8; C_7). Fracture of the thoracic spine was infrequent. Two of these three cases had spinal cord injury (1 case of transection). Individuals dying as a result of light aircraft crashes other than general aviation type generally had a similar injury distribution.

Head and Neck Injuries

At least one-half of the victims who died solely of head and neck injuries suffered facial and lower extremity (usually multiple) fractures, cerebral contusions/lacerations with intracranial hemorrhage and pulmonary contusions/lacerations. Craniocerebral trauma was a major cause of death in pilots and passengers. Fracture-dislocation of the cervical spine with either brainstem or spinal cord damage was seen only in pilots. The level of vertebral column injury was either atlanto-occipital (2 cases) or atlanto-axial (2 cases). One 63-year-old male passenger died of aspiration due to facial fractures (left maxilla and nose). Facial fractures were multiple in most cases. Two pilots sustained only nasal fractures.

Drowning

Most of these victims were in planes which sank. Two restrained individuals were recovered in partly submerged aircraft which had flipped.

Other than cutaneous/soft tissue trauma, the most frequent blunt trauma injury was fracture-dislocation of the cervical spine (one case each of C_3–C_4, C_4–C_5, C_6–C_7, C_7–T_1 fracture-dislocation). One of these victims (C_4–C_5 injury) was found in shallow water. A high frequency of congested/edematous lungs was observed. At least one-half had froth in the upper respiratory tract. The findings in two cases were hindered by decomposition. Laryngospasm was considered the mechanism of death in a 48-year-old female passenger recovered four days after a crash. She was decomposed and her right and left lungs weighed 400 and 320 g respectively.

Pulmonary edema was also observed in a small number of multiple injury and head/neck trauma deaths. Two of the three multiple injury fatalities died 10 and 7 hours following separate ultralight plane crashes. The third victim suffered burns. Three individuals dying of head and neck trauma had pulmonary edema. One aspirated blood; the second received cardiopulmonary resuscitation for one hour and the other died in the operating room four hours after the crash.

Disease

Two pilots were incapacitated by disease.

Case 1

About 15 minutes after take-off of a plane carrying a 47-year-old pilot licensed in the United States and three passengers, one of the passengers radioed that the pilot had suffered a "heart attack." The plane then disappeared from the radar screen. The next day, the four deceased were found inside the wreckage. Investigation revealed that the pilot had been dyspneic prior to take-off and had a history of angina and hypertension. Autopsy of the pilot revealed multiple traumatic injuries (Table 3). Severe (>75% stenosis) tri-vessel coronary atherosclerosis was seen. The right coronary appeared occluded by subintimal hemorrhage. The antecedent cause of death was right coronary artery occlusion which had precipitated an air crash resulting in multiple injuries.

Case 2

A 39-year-old private pilot died following his fourth landing attempt when his plane collided with some trees. A 14-year-old passenger survived. Post-mortem examination revealed non-fatal injuries (Table 3), severe tri-vessel coronary atherosclerosis and an associated old myocardial infarct. The pilot had complained of chest pain prior to departure. Twelve years previously he had applied for a student licence and had been diagnosed with Wolf-Parkinson-White (WPW) syndrome during the medical examination. Coronary angiography done at this time was reported as

TABLE 3—*Crash injuries and cause of death.*[1]

	Multiple Trauma	Head/Neck Trauma	Drowning	Disease
	27 (17+ + 10*)[2]	11 (8+ + 3*)	19 (11+ + 8*)	2 (2+)
Head & Neck				
Skin/soft tissue trauma[3]	19 [70%][4] (13+ + 6*)	10 [91%] (8+ + 2*)	16 [84%] (11+ + 5*)	Case 1[5] Case 2[5]
Fracture(s) face	13 [48%] (7+ + 6*)	8 [73%] (6+ + 2*)	1+ [5%]	Case 1
Fracture(s) skull	16 [59%] (7+ + 9*)	4 [36%] (3+ + 1*)	1+ [5%]	Case 1
Intracranial hemorrhage[6]	14 [52%] (8+ + 6*)	7 [64%] (6+ + 1*)	3+ [16%]	...
Cerebral contusion/laceration	9 [33%] (6+ + 3*)	6 [55%] (4+ + 2*)	...	Case 1
Fracture/dislocation-cervical spine	7 [26%] (6+ + 1*)	4+ [36%]	4 [21%] (2+ + 2*)	Case 1
Brainstem laceration/transection	3+ [11%]	2+ [18%]	...	...
Cervical spinal cord laceration/ transection/contusion	4+ [15%]	2+ [18%]	2 [11%] (1+ + 1*)	Case 1 contusion
Chest				
Skin/soft tissue trauma[3]	16 [59%][7] (10+ + 6*)	6 [55%] (5+ + 1*)	3+ [16%]	Case 1 Case 2
Fracture(s) rib(s)	21 [78%] (14+ + 7*)	3+ [27%]	3 [16%] (2+ + 1*)	Case 1
Hemothorax	17 [52%] (10+ + 7*)	2+ [18%]	...	Case 1
Pulmonary contusion/laceration	17 [63%] (10+ + 7*)	8 [73%] (6+ + 2*)	2+ [11%]	Case 1
Pulmonary edema	3+ [11%]	3+ [27%]	16 [84%] (10+ + 6*)	...
Hemopericardium	3 [11%] (2+ + 1*)	...	...	Case 1
Parietal pericardial laceration	14 [52%] (10+ + 4*)	...	...	...
Cardiac contusion	1+ [4%]	...	...	...
Cardiac laceration	19 [70%] (14+ + 5*)	...	...	Case 1
Aortic laceration	13 [48%] (10+ + 3*)	...	...	...
Fracture thoracic spine, NOS[8]	3 [11%] (2+ + 1*)	...	1+ [5%]	Case 1
Other thoracic vascular lacerations[9]	8 [30%] (5+ + 3*)	...	...	...
Abdomen/Pelvis				
Skin/soft tissue trauma[3]	8 [30%] (4+ + 4*)	5+ [45%]	2 [11%] (1+ + 1*)	Case 1
Hemoperitoneum	9 [33%] (3+ + 6*)	...	...	...
Hepatic laceration	16 [59%] (9+ + 7*)	2+ [17%]	...	Case 1
Splenic laceration	11 [41%] (6+ + 5*)	...	...	Case 1
Mesenteric laceration/hematoma	5 [19%] (2+ + 3*)	...	1+ [5%] hematoma	...
G.I. tract trauma	7 [26%] (3+ + 4*)	...	1+ [5%] small bowel contusions	Case 2 small bowel contusions
Renal/perinephric injury	6 [22%] (3+ + 3*)	1+ [9%] renal vein laceration	...	...
Fracture(s) pelvis	6 [22%] (4+ + 2*)	2+ [18%]	1+ [5%]	Case 1
Fracture lumbar spine	1+ [4%]	1+ [9%]	...	Case 1

TABLE 3—*Continued.*

	Multiple trauma	Head/neck trauma	Drowning	Disease
	27 (17⁺ + 10*)[2]	11 (8⁺ + 3*)	19 (11⁺ + 8*)	2 (2⁺)
Extremities				
Skin/soft tissue trauma of:[3]				
Upper limbs	18 [67%] (12⁺ + 6*)	8 [73%] (7⁺ + 1*)	9 [47%] (7⁺ + 2*)	Case 1
Lower limbs	18 [67%] (13⁺ + 5*)	11 [100%] (8⁺ + 3*)	7 [37%] (3⁺ + 4*)	Case 1
Fracture(s) of:				
Upper limbs	14 [52%] (11⁺ + 3*)	2⁺ [18%]	1⁺ [5%]	Case 1
Lower limbs	20 [74%] (13⁺ + 7*)	7 [64%] (6⁺ + 1*)	2⁺ [11%]	Case 1

[1]Data derived from reports of 59 complete autopsies. Report on a drowning case unavailable.
[2]⁺ = pilot; * = passenger.
[3]Severe burning resulting from post-crash fires hindered assessment in 8 deaths; multiple trauma = 6 (3 pilots, 3 passengers); head/neck trauma = 1 pilot; drowning = 1 passenger.
[4][ ] = percentage based on no. of victims with specific injury/total victims by cause of death.
[5]Case descriptions in *Results.*
[6]Subarachnoid and/or subdural hemorrhage.
[7]Five victims had cutaneous injuries on back.
[8]NOS = not otherwise specified.
[9]Pulmonary artery, 3 cases = 2⁺ + 1*; pulmonary vein, 3 cases = 1⁺ + 2*; one case each (both pilots) of inferior vena cava and subclavian artery injury (repaired).

normal. One year later, a Civil Aviation Medicine cardiology conference in Ottawa decided that WPW afflicted individuals could pilot an aircraft if they did not exhibit signs of arrhythmia. As a result, this man was declared fit and licensed. Six months prior to the crash, he had chest pain and ECG abnormalities were noted. Five days prior to the accident, he denied any history of heart disease or chest pain during an examination by an aviation medical examiner who did not detect any evidence of cardiac abnormality. The cause of death was ischemic heart disease.

The presence and severity of coronary atherosclerosis in pilots was assessed on review of 35 autopsy reports (5 cases eliminated: 1 report unavailable; 1 body not recovered; 1 case, heart too fragmented; 2 cases, the severity of stenosis was not stated). The degree of luminal stenosis was graded as mild (≤25%), moderate (>25% to <75%) and severe (>75%); however, in only three reports was the actual percentage stated. The coronary arteries of nine pilots aged 25 to 34 years were described as "normal," "unremarkable," "patent throughout" or showing "no evidence of atheroma." The assessment of eleven in the 35 to 44 year age group, revealed a normal circulation in five, four with "mild," "minimal," "grade 1," "slight" atherosclerosis or having "some atheroma" and two classified as severe. Twelve aged 45 to 54 years were either normal (two) or had mild (six), moderate (two) or severe (two) atherosclerosis. Of three pilots in the 55 to 64 year age group described in detail, one had normal coronary arteries and two had mild stenosis. About 11% (4/35) of this small sample of pilots had severe stenosis. The prevalence of coronary atherosclerosis was 51% (18/35).

In 37 reports studied, 6 pilots were noted to have cardiomegaly or left ventricular hypertrophy or both. The descriptions of the heart varied: "left ventricular hypertrophy" in a 26-year-old (body weight = 80 kg, 400 g heart); "moderately enlarged" in a 31-year-old (body weight = 110 kg, 460 g heart); "flabby" in a 47-year-old (body weight not stated, 500 g heart); "subaortic hypertrophic stenosis" in a 59-year-old (body weight estimated 100 kg, heart weight not determined, left ventricular width = 2.2 cm); a 510 g heart (left ventricular width = 1.8 cm) in a 59-year-old weighing 70 kg and "moderately enlarged" in a 57-year-old (body weight = 110 kg; heart weight = 492 g).

Survivability—Aircraft Structure and Equipment

TSB investigators determined that the aircraft involved in the crashes studied were either "destroyed" or had "substantial" damage.

The use of a restraint system was unknown in 40 victims. Three individuals were unrestrained (one multiple trauma death, two drowning victims). Of the 27 restrained, 12 died of multiple trauma, 5 of head and neck injuries, 8 of drowning, 1 of ischemic heart disease and 1 of unknown cause. Four of the five short-term survivors, who died of multiple injuries or craniocerebral trauma, were restrained. The lack of shoulder harness use was cited by TSB investigators as contributing to the deaths of four people (a pilot whose face struck an instrument panel sustained acute subarachnoid hemorrhage and drowned; a pilot's head struck the throttle resulting in severe head injuries; another pilot suffered an atlanto-occipital dislocation; a passenger might not have drowned if he had not been incapacitated by a lower cervical spine injury). Possible restraint failure was seen in one crash in which a torn seat belt was found on the partial remains of a pregnant 24-year-old left rear passenger. There were three instances of seat mounting avulsion (2 pilots, 1 passenger). One of these pilots, traveling at an estimated speed of 160 km/hr (100 mph), died of a C_1–C_2 dislocation.

TSB investigators felt that two individuals drowned because their aircraft, which crashed following a medical evacuation, was not equipped with flotation devices. An amendment to existing regulations proposed that commercial aircraft flying over bodies of water should be equipped with a life jacket for each occupant.

One pilot who became entangled in ropes drowned because he could not escape his submerged aircraft.

Toxicology

Ethanol

Eliminating two decomposed cases, three of thirty-nine pilots (8%) had evidence of alcohol ingestion; two cases were above 40 mg% (.04) (Table 4). None of the decomposed cases had vitreous humor and urine samples.

Case 3

A 44-year-old pilot, who was not licensed for plane float operation, crashed into a lake following take-off at dusk. He drowned but his passenger survived. Postmortem analysis revealed a blood alcohol of 122 mg% and urine alcohol level of 177 mg%.

Case 4

A 40-year-old pilot was flying two passengers on a fishing trip when his plane stalled during a steep left turn. One passenger survived. The pilot suffered a transection of the upper cervical spinal cord. His liver was slightly fatty. The post-mortem blood alcohol concentration was 166 mg%; the urine alcohol level was 138 mg%. The pilot had a previous history of a transportation violation.

In addition to this pilot, there were eight others who were found to have mild to moderate fatty liver change at autopsy. Blood alcohol levels were either negative (three cases) or unavailable (five cases).

Carbon Monoxide

Eight victims (4 pilots; 4 passengers) were involved in six post-crash fires. All were charred except one drowned passenger who had extensive first/second degree burns. Carboxyhemoglobin saturation (COHb) was determined in three of the four pilots who died of multiple trauma. Their levels were 3%, 3% and 1.6%. None of the pilots had soot in the respiratory tract. COHb was not determined in the four passengers, three of whom died of multiple trauma and the fourth of drowning. Two of the trauma victims had soot in their airways. COHb was measured in fourteen other individuals (7 pilots; 7 passengers) not involved in fires. The results were negative with the exception of a pilot and a passenger who, each, had a level of 3%. The smoking history of these two individuals was unknown.

TABLE 4—*Alcohol in pilots.*

	# Tested/ # Recovered	Results			
		0	>0–39 mg%	40–79 mg%	≥80 mg%
BAC[1]	32/39[2] (82%)	27[3]	2[4]	0	3[5]

[1]BAC—blood alcohol concentration (mg%). Legal limit in Ontario for motor vehicle impairment is ≥80 mg%.

[2]One body not recovered. Three of the victims survived short time and not tested. Ethanol testing or results either not done or unavailable in four cases.

[3]Two of these tested victims survived 10 hours.

[4]13 mg% in one case, no decomposition; 37 mg% in another victim, slight decomposition.

[5]87 mg% in one case, moderate decomposition; 122 mg% and 166 mg% in two cases (*Case 3* and *Case 4* respectively—see *Results—Toxicology*), no decomposition.

Drug Screen

A screen was done on 19 of 39 pilots. In one case, caffeine was detected in blood and urine and quinine, consistent with ingestion of tonic water, was found in the urine. Another pilot had a therapeutic level of allopurinol. Codeine was detected but not quantitated in the blood of a fatally injured pilot who had been prescribed this analgesic following recent dental surgery. The presence of this medication was raised as a possible factor contributing to the pilot's fatigue which affected his flying performance.

Cause of Crash

The most frequent cause was pilot error (55%; 26/47 crashes) (Fig. 2).

Phase of Flight Take-Off

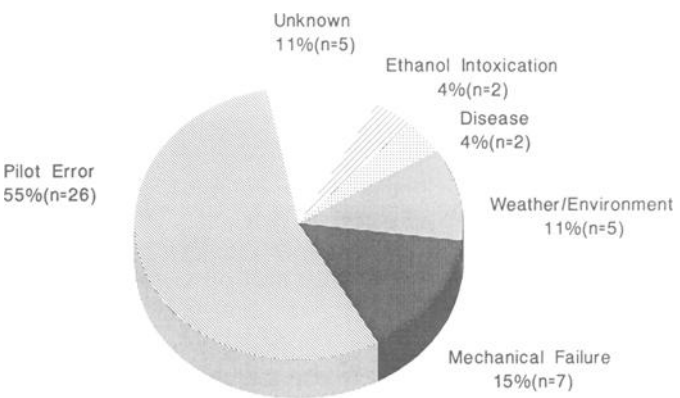
There were four crashes, including one ultralight, during this phase. Three were due to pilot error (inexperience in an ultralight; excess occupant weight; misjudged weather conditions). The cause of an unwitnessed crash was unknown.

Following Take-Off

Eleven crashes occurred. Four were due to pilot error (details were provided in three crashes—inexperience and excess passenger weight; poor judgment flying a plane with engine problems; air conditioner on resulting in engine stall). Three impacts were attributed to mechanical problems (elevator cable broke after repeated rubbing against battery terminal; engine problems due likely to water in fuel; pilot seat back failure due to metal fatigue resulting in seat falling back, jamming a rear control stick leading to a stall at low altitude). One crash was due to poor visibility during snow squalls. Another crash resulted from ethanol intoxication (Case 3, *Toxicology—Ethanol*). Two crashes were of unknown cause.

Cruise

There were twenty-three crashes, including four aircraft other than general aviation type. Twelve of the accidents occurred because of pilot error (inattention; misjudgment of weather; inexperience (2 cases); flying too low (8 cases)). The low flying cases included a pilot who was being photographed in his ultralight, three cases in which poor visibility due to darkness or weather contributed, two instances of pilots attempting to see a site on the ground and one pilot whose "buzzed" the home of a friend. Four



accidents were attributed to mechanical malfunction (ruptured fuel tank; fragmentation of a wooden propeller in a gyroplane; fuel exhaustion due to head winds; blockage of injection pump by tape resulting in engine failure). Four crashes were due to weather/environmental conditions (unable to maintain altitude due to icing; severe snowstorm; "poor weather conditions" in two cases). One impact was caused by ethanol intoxication (Case 4, *Toxicology—Ethanol*). Another accident resulted from pilot incapacitation due to disease (Case 1, *Cause of Death—Disease*). One crash was of unknown cause. In three instances, pilots, who encountered either poor weather or darkness, did not have an instrument flight or a night flying endorsement of their licences.

Landing

Nine crashes occurred. Seven arose because of pilot error (misjudgment of weather conditions, (2 cases); overflew air strip, (2 cases); crashed while preparing for landing; landing in lake with landing gear extended; lost control attempting to close canopy which opened on approach). One crash resulted from pilot disease (Case 2, *Cause of Death—Disease*). One crash was of unknown cause.

Discussion

In Canada, from 1985 to 1989, the fatal accident rate per one hundred thousand flying hours involving Canadian registered aircraft ranged from 1.2 to 2 (average 1.6) (17). During this time, there were a total of 270 fatal accidents and 17,136,000 flying hours were recorded (17). In the United States, the rate of fatal general aviation crashes per one hundred thousand hours flown decreased from 1.75 in 1978 to 1.53 in 1989 (18). During this period, there were 2310 fatal general aviation crashes and 137,733,000 hours flown (18).

Demographic Features

In the present series, the mean age of the deceased pilots, all men, was 43 years. In an NTSB study of alcohol and drug involvement in 2760 general aviation accidents (fatal to the pilot-in-command) from 1983 to 1988, the average age of the substance-free pilots, who comprised the majority (n = 2590; 94%) of the pilot victims, was 44 years (19). Two percent (51/2590) were women. The average age of the alcohol-involved group, all men, in the U.S. study was about 40 years (19). In the present study, the two significantly impaired pilots were 40 and 44 years of age. The NTSB review observed that about 44% of the substance-free deceased pilots were aged 25 to 44 years (19); whereas, the Ontario data revealed 53% (21/40) were in this age group. About one-half of the NTSB fatal accidents were fatal to the pilot only (19) compared to 57% (27/47) of the accidents studied in Ontario.

Aircraft Use

The majority (72%) of the Ontario accidents happened during personal use of these aircraft. NTSB data in the 1983–1988 study revealed that, of the substance-free accidents, 74% were known to be personal flights and 20% were work related; of the alcohol-involved accidents, 91% were personal and about 5% were work related (19). In a study of work related aviation fatalities in Colorado from 1982 to 1987, Wiant et al. concluded that because of the prominence of general aviation incidents, more attention should be directed to the safety of these workers (20).

Trauma

Blunt trauma was the leading cause of death. About two-thirds of all the fatalities died from injuries. A study of 57 accidental non-commercial aircraft deaths in Florida showed that 43 (75%) were due to trauma (21). Flailing of an unprotected individual during a crash is a major mechanism of injury (22,23). Restraint systems prevent this; however, they are ineffective if there is significant occupant compartment intrusion by the crushed air frame (23). Thirty-five of the trauma victims in this series were found inside the wreckage. Generally, immediately fatal injuries in belted and non-belted victims were associated with extensive damage to the aircraft. The use of lap/shoulder harnesses was not consistently documented. Of the thirteen whose restraint status was known only one was unrestrained. One pilot suffered a fatal neck injury and another severe head injuries because they did not wear the shoulder part of their restraints (9,23,24). Four individuals were ejected from the aircraft and at least three of these cases resulted from restraint or seat mounting failure (23). Seat failure compared to belt failure has been associated with more severe injury (25). A seat belt will absorb decelerative energy before failing reducing the degree of blunt force trauma. Lack of information regarding restraint use did not allow proper assessment of "survivable" injuries (23).

Although autopsy reports of aircraft fatalities are not standardized in Ontario (9), centralized files did allow an appreciation of injury patterns, that is, a compilation of injuries of individuals involved in a series of accidents (22) and comparison with other studies (8,26).

Craniofacial fractures were often seen in this series as noted in other studies (8,26) but the frequency of skull fractures in the Ontario data was lower. (Table 5) Various skull fracture mechanisms in aircraft impacts have been described (9,23). Associated facial and cranial fractures can indicate forces transmitted through the mandible and temporomandibular joints. Facial fractures can cause asphyxiation from aspirated blood (23). Such a case was documented among the Ontario victims. Cerebral trauma was less frequent than skull fractures supporting the observation that the latter does not always result in visible brain injuries (8). Concussion may occur in these cases (8). Spinal fractures were common. Cervical fractures were seen more often than those of the thoracolumbar spine. The former injury was noted almost exclusively in pilots. Rib and extremity fractures, particularly of the legs, were common as observed in previous studies (8,26).

Trauma to other viscera was as important in causing death as craniocerebral injuries. A higher frequency of pulmonary contusions/lacerations was seen compared to other studies (Table 5). Cardiovascular trauma was common. Cardiac injuries were evident in about one-half of cases and due either to penetration by fractured ribs or from the chest striking part of the aircraft cockpit/controls. Cardiac lacerations could have also resulted from chest compression leading to increased intracardiac pressure (9,22). Aortic tears were caused by either downward displacement of the compressed heart or deceleration (9,22). Relative to pilots, passengers sustained a greater proportion of craniofacial fractures and retroperitoneal/abdominal trauma including liver, spleen and mesenteric injuries. Liver injuries were more frequent than splenic trauma consistent with the more protected location of the spleen (8,9,26). Hepato/splenic injuries have been associated with severe head and chest trauma (27). Mason observed that the stomach almost never ruptures in an aircraft accident (9). One case of gastric laceration was seen in the present study. The bowel is more likely to be contused

TABLE 5—*Comparison of injury frequency in studies of light aircraft fatalities.*

	Ontario Study[1] % (no.)	Reals et al.[2] % (no.)	Stevens[3] % (no.)
Fracture(s) of			
Skull	53 (20)	70 (104)	67 (63)
Facial Bone(s)	55 (21)	...	48 (45)
Spine:			
Cervical	29 (11)	13 (19)	12 (11)
Thoracic	8 (3)	12 (18)	23 (21)
Lumbar	5 (2)	10 (15)	18 (17)
Rib(s)	63 (24)	56 (83)	63 (59)
Extremities:			
Upper	42 (16)	63 (94)	56 (52)
Lower	71 (27)	74 (110)	73 (68)
Pelvis	21 (8)	...	...
Visceral Trauma (laceration/contusion)			
Brain	39 (15)	53 (78)	56 (52)
Brainstem[4]	13 (5)	...	...
Cervical Cord[4]	16 (6)	...	...
Lungs	66 (25)	34 (50)	36 (33)
Heart	53 (20)	34 (51)	35 (33)
Aorta	34 (13)	23 (34)	39 (36)
Liver	47 (18)	49 (72)	35 (33)
Mesentery	13 (5)	...	...
Gastrointestinal Tract	18 (7)	...	...
Spleen	29 (11)	41 (61)	34 (32)
Kidney[5]	18 (7)	19 (28)	23 (21)

Based on autopsy findings seen in:
[1]38 trauma victims (25 pilots; 13 passengers). 27 died of multiple injuries and 11 of head and neck trauma only.
[2]148 fatalities (pilots/passengers not specified).
[3]93 deaths (70 pilots; 23 passengers).
[4]Includes transection.
[5]Includes perinephric hematoma, vessel lacerations.

and lacerated (9). Kidney lacerations have also been noted infrequently, the more common injury being perirenal or renal pelvic hemorrhage (9). The Ontario results showed that in multiple trauma fatalities, tears of the kidney or its major vessels or both occurred slightly more often than perinephric hematoma.

Drowning

Drowning was the second major cause of death resulting in more than one-quarter of the fatalities. By contrast, drowning was observed in only 5% of Florida aircraft accidents (21). The diagnosis of drowning is by exclusion of other causes of death through review of the circumstances and performance of a complete autopsy (28). Pulmonary congestion and froth in the upper respiratory tract are useful signs of drowning which were seen in most of the studied cases. Laryngospasm was considered a possible mechanism of death in one individual. Drowning associated with traumatic injuries was infrequent. The most common significant injury was fracture/dislocation of the cervical spine which could have prevented some injured victims from escaping the aircraft. At least eight of the twenty drowning victims were found restrained inside the wreckage. An association between non-fatal head injury, that is, concussion, and asphyxial death has been noted (23). In the present study, head injuries in drowning victims were infrequent. A pilot, who was not wearing his shoulder harness, sustained a closed head injury and subsequently drowned.

Post-Crash Fires

One of the questions raised during the medical investigation of fatalities recovered from aircraft fires is the determination whether burns were received post-mortem, that is, fatal injuries occurred prior to burning (29). Carbon monoxide determination is useful in reconstruction of certain aircraft crashes including fire-related accidents (14). Elevated levels could result from either smoking, equipment malfunction or fire before the accident or after impact. In the latter situation, death may not be immediate (14). Individuals killed on impact then recovered following a post-crash fire would have negligible levels of carboxyhemoglobin (COHb below 10%) (30) as observed in the present series in which three pilots had COHb levels less than 3% and no evidence of smoke inhalation. Two passengers who also died of injuries had soot in their airways indicating a short period of survival in the post-crash fire. COHb was not analyzed in these cases.

Disease

Almost all of the light aircraft, in this study, were single pilot operations. In this situation, incapacitation of the pilot can lead to a crash (31). Because cardiovascular disease, particularly coronary atherosclerosis, is prevalent and has the potential to cause sudden incapacitation or death, it has been well studied in relation to aircraft accidents and flight safety (13,32–35).

The prevalence of coronary atherosclerosis in private or civilian pilots is similar to that seen in the general population. In one study, 22% of private pilots killed in aircraft accidents (mean age 37.2 years) had significant atherosclerosis (more than 50% stenosis of the lumen or atheroma associated with degenerative changes) compared to 18.2% in a control group (mean age 29.9 years) (32). Booze et al. observed that the prevalence of coronary atherosclerosis, of any degree, varied from 50.9% to 69.1% (36,37). Severe stenosis (greater than 66% luminal restriction) was noted in 4.9% and 2.7% of general aviation pilot fatalities during two respective study periods (36,37). The Ontario data revealed 51% (18/35 pilots whose coronary artery status was known) had evidence of coronary artery disease; 11% (4/35) with "severe" stenosis. Although performance of an autopsy helps determine the prevalence of coronary atherosclerosis in deceased pilots, autopsy rates can depend on location and circumstances of the crash as well as the co-operation of local coroners or medical examiners in liaison with aircraft accident investigators in obtaining a post-mortem (37). Selection of cases for autopsy can be influenced by either publicity or a history of cardiac problems in the pilot (37). In the present study, almost all of the pilots were autopsied. The accurate determination of disease prevalence also depends on performance of a full autopsy (13). The complete post-mortem examination is necessary to ascertain whether coronary artery disease in a fatally injured pilot either caused, contributed to or was coincidental in an accident (13). The autopsy must include a thorough gross and microscopic study of the coronary arteries (11,38). The pathological grading of coronary atherosclerosis varies (36). In the present study, various terms were used by thirty-nine pathologists across the province to describe the severity of coronary stenosis. Luminal narrowing expressed as a percentage was infrequent. Despite the lack of centralized pathological investigation, the presence of coronary artery disease was properly interpreted when all the investigation findings were considered (13).

Difficulties in assessing the significance of coronary atherosclerosis in accident causation can arise because of trauma (13,39). Trauma can destroy evidence of disease. Extensive fragmentation

of the heart can hinder the examination of the coronary arteries (39). Trauma can interrupt the evolution of a disease (13,39). A myocardial infarct due to coronary atherosclerosis may not be evident because of immediately fatal injuries. Trauma can mimic a disease process (13,39). Hemorrhage into a coronary plaque is considered a useful finding indicating sudden incapacitation but this finding can result from trauma (13). In our series, a 47-year-old pilot (Case 1) was observed, at autopsy, to have fatal traumatic injuries. Severe coronary atherosclerosis with apparent right coronary artery occlusion by subintimal hemorrhage was also seen; however, plaque hemorrhage was not confirmed by microscopic examination. Nevertheless, coronary disease was considered a precipitating factor in this crash and the proximate cause of death. A passenger had radioed that the pilot had suffered a "heart attack" during the flight. Accounts of survivors and witnesses can link disease and crash causation (13). In the second case (Case 2) of inflight cardiovascular incapacitation, witnesses stated that the pilot had chest pain prior to the flight. His erratic landing attempts led to a crash. The injuries seen at autopsy were survivable. Coronary atherosclerosis can be compatible with normal cardiac function and is not necessarily the cause of unexplained accidents (13). The role of coronary artery disease found in a pilot should be supported by other evidence when determining accident cause (16).

In private aviation, the risk of inflight incapacitation from coronary atherosclerosis and consequent fatal accidents is low (13). Of 122 fatal general aviation accidents occurring in the western United States in 1965, none were definitely due to coronary disease (40). A United Kingdom study of 35 civilian pilots revealed that, in one case, coronary stenosis was the probable cause of a crash (26). From 1974 to 1975, the incidence of pilot cardiovascular incapacitation was 0.93% (13 pilot deaths in 1404 general aviation accidents) as reported by the NTSB (41). About half of these victims were between 50 to 59 years of age. Twelve were autopsied. All had severe coronary artery stenosis; five having recent coronary occlusion which was felt to be the cause of incapacitation and four with an old myocardial infarct. Among the Ontario pilots, two men aged 39 and 47 years, were incapacitated by cardiovascular disease resulting in accidents which killed five people. Of 47 fatal light aircraft accidents studied, at least two (4%) were caused by inflight pilot incapacitation due to coronary atherosclerosis. Although general aviation inflight incapacitation can happen during all phases of flight, the majority in one report occurred during the cruise phase (41). In the present study, one incident happened shortly after take-off (Case 1) and the other during attempted landing (Case 2).

In Canada and the United States, there are medical standards specifically related to cardiovascular disease which govern licensure of a civilian pilot (42,43). Certification is based on the level of flying activity and pilots who fly planes for personal use have the least strict standards. A history of myocardial infarct or angina can disqualify a pilot from flying (42,43). Cardiovascular disease has been noted as the leading cause for denial of medical certification (37) but heart disease is not always detected during routine medical screening (39). Although pilots are encouraged to report cardiac symptoms they may be non-compliant as seen in one of our cases (Case 2) and reported by others (33,38,41). Although there is an increased risk of an acute coronary event in older, affluent and presumably less fit private pilots (37,39,44), age is not a disqualifying factor (45). Older individuals are not necessarily prone to more accidents (46,47). The loss of older, more experienced pilots, particularly in commercial and military aviation, could adversely affect overall flight safety (33,48,49). Opinions regarding pilot disqualification based on detection of potentially incapacitating latent or subclinical disease have varied in the literature (45). The finding of even minor coronary atheroma has been considered a predictor of future cardiovascular events (50) although others have disagreed (48). Angiographically proven coronary artery disease would preclude unrestricted medical certification (50). Contrary to this view, other observers felt that, despite the increasing number and sophistication of screening tests, these additional measures may not be practical given the low prevalence of severe coronary atherosclerosis and incidence of inflight cardiovascular incapacitation (36,37,41).

Six cases of cardiomegaly were observed. There was no associated medical history reported. Heart weight was recorded in five of the six cases. In an autopsy analysis of military, professional and private pilot fatalities, heart weight was noted only in 26.7% (32). Myocarditis has not been implicated as a cause of inflight incapacitation (44) and no cases were found in our study. As in the assessment of coronary atherosclerosis, knowledge of the accident circumstances would assist in the determination of whether other types of cardiovascular disease precipitated an accident (44).

Ethanol

Early studies of alcohol and fatal general aviation accidents seemed to indicate significant alcohol usage in deceased pilots. In one study in the United States during 1960 to 1961, 35.4% of pilot fatalities (56/158 general aviation accidents) had a positive blood and/or tissue alcohol level (≥15 mg%) (51). This concentration was considered positive because it implied that, in an average person, one or more alcoholic drinks had been consumed within one hour (52). About 28% (n = 44) of these pilots had levels greater than 50 mg%. The average blood alcohol concentration (BAC) in the alcohol positive group was 145 mg% (51). This study was criticized because of the small sample size due to selection bias, that is, pilots were tested only in cases suspicious for alcohol use (53,54). A follow-up study in 1964–1965 during which autopsies and toxicological analyses were requested in all cases revealed 30.8% (36/117 pilot deaths studied) had a positive BAC (≥15 mg%) (53). Twenty-eight percent had a BAC greater than 50 mg%. Another report during this period revealed 20.5% of U.S. general aviation pilot fatalities (17 of 83 tested) had a BAC greater than 35 mg%. Thirteen (16%) had a BAC greater than 50 mg%; five with levels exceeding 250 mg% (40). From 1968 to 1974, 8.7% (n = 117) of pilots killed in 1345 fatal general aviation accidents in the United States had a BAC greater than 50 mg% (55). Another 10.8% (n = 145 pilots) had levels less than 50 mg%. Few cases of significantly elevated BAC were seen in pilots killed in light aircraft accidents in the United Kingdom from 1956 to 1966 (26). The incidence of alcohol positive cases was estimated to be 7 to 10% (26). Underwood Ground studied 86 fatal light aircraft accidents in the United Kingdom from 1964 to 1973 (56). Of 102 deceased pilots tested, 34 had detectable ethanol varying from trace amounts to 313 mg%. An Australian study of general aviation accidents from 1962 to 1965 showed that 18% (25 of 138 valid estimations corresponding to 133 accidents) of the deceased pilots had a BAC above 15 mg% and 10% (n = 14) had a level exceeding 50 mg% (54). Alcohol impairment of the pilot-in-command was a factor in 12 of these accidents (8.7% of the estimated BACs) based on investigation records. Inaccuracies in these early studies arising from alcohol generation due to bacterial contamination and other artifacts were raised (15,57–59). More recent studies

(19) have eliminated cases in which putrefaction may have altered the BAC. To address this problem, the post-mortem collection of not only blood but also vitreous humor and urine has been stressed (60) and is included as part of accident specimen collection protocols (61). Studies of alcohol in light aircraft fatal accidents do not reflect the prevalence of ethanol use in the entire pilot population during flights and the toxicological findings in all aircraft accidents (19,59).

The demands of flying an aircraft are greater than driving an automobile (53,58). Pilots generally recognize that ethanol can impair their performance and they have a more conservative attitude toward drinking and flying compared to driving (62). The adverse effects of alcohol, even in concentrations as low as 15 mg%, on pilot performance under simulated and actual flight conditions have been well described and summarized (63). For example, increased procedural errors during instrument assisted landing of a single engine Cessna were observed in pilots of varying experience even those having low alcohol levels (40 mg%) (64). The presence of low ethanol concentrations in a pilot cannot be discounted as a factor contributing to a crash (65). Interpretation of an ethanol result requires complete evaluation of all circumstances and factors related to the accident (15,58). Decreased performance due to ethanol can be exacerbated by night flying (66). In our study, 68% (32/47) of crashes occurred between noon and 1959 hours. About 56% of substance-free accidents in the NTSB series happened during this time (19). One of the Ontario alcohol related crashes (Case 3) occurred at dusk. NTSB data revealed that 52% of alcohol-involved accidents occurred under limited light conditions (dawn, dusk, night) compared to 26% of alcohol free accidents (19). Forty-three percent of the alcohol-involved incidents happened between 8:00 p.m. and 3:59 a.m., typical drinking hours. Hangover effects on the performance of complex tasks have been described (67).

In the United States, Federal Aviation Administration (FAA) regulations prohibit anyone acting as a crew member within 8 hours after the consumption of any alcoholic beverage, while under the influence of alcohol, while using any drug which could adversely affect the individual's faculties in any way contrary to safety or while having a BAC of .04 (40 mg%) (19,68). Canadian federal air regulations are similar except that there is no .04 blood alcohol concentration rule (69). Following the introduction of this FAA regulation in 1970 (with the exception of the 0.04 rule), the number of alcohol violations against U.S. general aviation pilots dropped in 1971 (52). The percentage of alcohol positive cases in fatal accidents stayed at about 20%; however, compared to the preceding year there was a greater proportion of victims who had a BAC exceeding 100 mg%. This suggested that the "eight hour" rule had less effect on heavy drinkers (52). A survey of private pilots showed that they underestimated the "safe" waiting time between drinking various amounts of alcoholic beverages and flying. About 30% of the respondents would have flown within a time period that would have resulted in a BAC of at least 15 mg% (65). In another American survey, many of the pilots lacked an understanding of the relation between the amounts of alcohol consumed and its metabolic rate (70). The majority of respondents were unaware of the 1985 FAA regulation change prohibiting individuals with a blood level of 40 mg% (0.04) acting as a crew member in a civil aircraft (70).

From 1975 to 1981, NTSB data revealed that 9.7% (336/3448 results), that is, 47 cases per year were alcohol-involved (19). From 1983 to 1988, the NTSB investigated 2760 general aviation accidents fatal to the pilot-in-command. Of this number, 71.8% (n = 1982) had conclusive toxicological results, that is, positive or negative for alcohol based on analysis of adequate samples. Although NTSB policy promotes alcohol testing of all fatally injured pilots, results were not available in every case because either an autopsy was not done, analysis was not requested or specimens were unsuitable or unavailable for testing because of decomposition (19). A positive ethanol result (range 30 mg% to 410 mg%) was observed in 133 (6.7% of the conclusive tests; 4.9% of all accidents) pilot fatalities, that is, 17 accidents per year. Toxicological analysis was not the only means of defining alcohol-involved ie. alcohol was cited by the NTSB as a cause or factor in a crash. Investigation revealed two additional pilots who, although not tested, were considered impaired based on witness accounts. Efforts by the FAA and various agencies to inform pilots of the deleterious effects of alcohol on flying appeared to have contributed to a decrease in alcohol involvement between the two time periods assessed by the NTSB (19). Nevertheless, 95% of the pilots testing positive in the 1983–88 study had levels above 40 mg%. The mean BAC was 150 mg% and 47% of the deceased pilots exceeded this level. In the present study, at least two (5%) of the forty accidents fatal to the pilot-in-command were alcohol-involved. One of these pilots had a record of an unspecified transportation violation. The NTSB observed that pilots in the alcohol-involved group had a greater disregard for aviation safety regulations than the substance-free group (19).

In North America and Europe, fatty liver is commonly associated with ethanol abuse. In a U.K. study, abnormal liver histology was found in 28% (45/160) of dead private pilots (71). Fatty change was observed in 21% (n = 34) but a history of alcoholism was available in few cases. The present series revealed nine pilots including an inebriated individual with this abnormality.

Drugs

Early studies have documented drugs in fatally injured pilots which may have contributed to general aviation accidents. Smith et al. noted 3.5% (7/202) of pilot fatalities had used potent therapeutic agents (perphenazine and amitriptyline, chlorpromazine, barbiturate, quinidine, pheniramine, chloroquine, morphine) (57). Stevens found that of 35 civilian aircraft accidents studied, two pilots had ingested drugs (antihistamines or amphetamine) which could have been an accident factor (26). Of 1345 U.S. fatal general aviation accidents reviewed over a seven year period, drugs were found in 1.2% (55). Barbiturates, antihistamines, tranquilizers and salicylates were the most frequently encountered. The NTSB classified an accident as drug-involved if either an illicit, prescription or over-the-counter drug was detected in a dead pilot-in-command and was cited by the Board as contributing to the crash (19). As in the United States, Canadian federal air regulations prohibit anyone acting as a crew member while using a drug that could jeopardize air safety (68,69). When drugs are detected, other factors including circumstances must be considered when determining accident cause. From 1983 to 1988 drug testing was infrequent and only 35 cases were determined by the NTSB to be drug-involved (19). As a result, no conclusions about drug involvement in general aviation accidents were made. The number of drug-involved accidents appeared to increase from 1983 to 1988, possibly due to increased drug testing and improved testing techniques. NTSB data revealed cocaine (12/35 pilots) and marijuana/metabolites (9/35 pilots) were the most frequently found drugs (19). An Armed Forces Institute of Pathology (AFIP) review of 377 general aviation fatalities occurring from 1988 to 1989 made similar observations (72). Cannabinoids and benzoylecgonine were the most

commonly detected illicit drugs, found in 1.3% and 1.6% respectively in the deaths analyzed. Non-narcotic analgesics (salicylate, acetaminophen) and nicotine were the most common legal substances. No consistent drug pattern use was evident. No conclusions about accident causation could be drawn because the history or circumstances of the accidents were unknown in the AFIP series. In the Ontario study about one-half of the pilots (19/39) were tested. Three had detectable legal substances or medications which, in two instances, did not contribute to any accidents. Although codeine was found in another pilot, no definite conclusion about its contribution to accident causation was reached.

Cause of Accident

In Ontario, 55% of the accidents from 1985 to 1989 were attributed to pilot error. Mechanical failure and factors related to the weather and environment were implicated in 15% and 11% respectively. Medical reasons, that is, disease or ethanol were observed in 8%. The cause was unknown in 11%. Pilot error has been observed as the leading cause of fatal general aviation accidents in other studies. The NTSB noted that flight crew factors, mainly planning decisions and aircraft handling, were the most common errors in general aviation accidents (substance-free group—61.1%; alcohol-involved—64.7%) (19). Environmental factors, mostly weather-related (substance-free—27.2%; alcohol-involved—30.7%) and aircraft related problems (substance-free—7.1%; alcohol-involved—2.9%) were less frequent. In one review of 485 fatal accidents, including 146 private operators, the cause of the accident was attributed to pilot error—35.5%; mechanical failure—37.5% and medical factors—5.8% (60). In another study, 65% of the accidents (33 civilian, 23 military) were due to pilot error (26). Copeland found at least 21% of non-commercial aircraft fatalities due to human error but the cause was not stated in 43.9% of his cases (21). Based on a sample of general aviation pilots, relatively low levels of risk and hazard awareness combined with a generally optimistic self-appraisal of abilities particularly in younger pilots (under 30 years), was observed (73).

Conclusions

In most cases, the purpose of the medical investigation of a fatal aircraft impact is not to determine the cause of the accident; however, the medical investigator (coroner, medical examiner or pathologist, if an autopsy is done) can assess whether disease or toxicologic findings could have precipitated the crash. Medical factors do play a small, but significant, role in accident causation and should be considered in a systematic and thorough manner in light of all the investigation findings.

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References

(1) Canadian Transportation Accident Investigation and Safety Board Act, 1990.
(2) Independent Safety Board Act of 1974 (United States). Statutes at Large, Vol. 88, sec. 304, 1974.
(3) McCormick MM. Medical investigator preparedness for aircraft accident investigations. Aviation, Space, Environmental Medicine 1977;48(10):932–36.
(4) Coroner's Act, Revised Statutes of Ontario, 1990, Chapt. C.37.
(5) Tarlton SW. Identification in aircraft accidents. In: Mason JK, and Reals WJ. (Ed.), Aerospace Pathology, College of American Pathologists Foundation, Chicago, 1973; Chapt. 7, pp. 53–63.
(6) Ford MA, Ashley KF. The role of the forensic dentist in aircraft accidents. In: Mason JK, and Reals, WJ. (Ed.), Aerospace Pathology, College of American Pathologists Foundation, Chicago, 1973, Chapt. 8, pp. 64–75.
(7) Germaniuk HD, Tate LR. Air crash! Are you ready?. The American Society of Clinical Pathologists Check Sample, Forensic Pathology 1990;32(1).
(8) Reals WJ, Davidson WH, Karnitschnig HH. Pathology of light-aircraft accidents. Aerospace Medicine 1964;34(2):133–35.
(9) Mason JK. Injuries sustained in fatal aircraft accidents. British Journal of Hospital Medicine 1973;9(5):645–54.
(10) Mason JK. Medicolegal aspects of aircraft accidents. In: Mason JK, and Reals WJ. (Ed.), Aerospace Pathology, College of American Pathologists Foundation, Chicago, 1973; Chapt. 6, pp. 44–52.
(11) Hemming FO. In-flight coronary occlusions: a short series of cases. Aerospace Medicine 1970;41(7):773–75.
(12) Blackmore DJ. Aircraft accident toxicology: U.K. experience 1967–1972. Aerospace Medicine 1974;45(8):987–94.
(13) Mason JK. Pre-existing disease and aircraft accidents. In: Mason JK, and Reals WJ. (Ed.), Aerospace Pathology, College of American Pathologists Foundation, Chicago, 1973, Chapt. 9, pp. 76–85.
(14) Blackmore DJ. The determination of carboxyhemoglobin and its importance in aviation accidents. In: Mason JK, and Reals WJ. (Ed.), Aerospace Pathology, College of American Pathologists Foundation, Chicago, 1973, Chapt. 20, pp. 190–202.
(15) Blackmore DJ. Ethanol and aircraft accidents. In: Mason JK, and Reals WJ. (Ed.), Aerospace Pathology, College of American Pathologists Foundation, Chicago, 1973, Chapt. 21, pp. 203–210.
(16) Stevens PJ. The search for the cause of an accident. Proceedings Royal Society of Medicine 1968;61:1076–79.
(17) 1993 Preliminary Transportation Occurrence Statistics, Transportation Safety Board of Canada Communique (TSB# 02/94), 1994.
(18) National Transportation Safety Board Statistics. Accidents, Fatalities, and Rates U.S. General Aviation 1982–1993.
(19) Alcohol and Other Drug Involvement in Fatal General Aviation Accidents, 1983 through 1988. National Transportation Safety Board (NTSB/SS-92/03), 1992.
(20) Wiant CJ, Baker SP, Marine WM, Vancil R, Keefer SM. Work-related aviation fatalities in Colorado 1982–1987. Aviation, Space, Environmental Medicine 1991;62(9):827–30.
(21) Copeland AR. Accidental non-commercial aircraft fatalities: the 7-year metro-Dade county experience from 1977–1983. Forensic Science International 1986;31(1):13–20.
(22) McMeekin RR. Patterns of injury in fatal aircraft accidents. In: Mason JK, and Reals WJ. (Ed.), Aerospace Pathology, College of American Pathologists Foundation, Chicago, 1973, Chapt. 10, pp. 86–95.
(23) Cullen SA. Death in general aviation accidents and its prevention. In: Mason JK, and Reals WJ. (Ed.), Aerospace Pathology, College of American Pathologists Foundation, Chicago, 1973, Chapt. 12, pp. 105–113.
(24) Hill IR. Inadequate harnesses as a cause of death in two light aircraft accidents. Aviation, Space, Environmental Medicine 1980;51(9): 1077–79.
(25) Pearson RG. Relationship between tie-down effectiveness and injuries sustained in lightplane accidents. Aerospace Medicine 1962; 33(1):50–59.
(26) Stevens PJ. Fatal accidents involving light aircraft: a review of UK experience, 1956–66. Aerospace Medicine 1968;39(6):622–27.
(27) Hill IR. Hepato-splenic injury in aircraft accidents. Aviation, Space, Environmental Medicine 1982;53(1):19–23.
(28) Thompson RL. Cause of death in aircraft accidents: drowning vs. traumatic injuries. Aviation, Space, Environmental Medicine 1977;48(10):924–28.

(29) Dunne MJ, McMeekin RR. Medical investigation of fatalities from aircraft—accident burns. Aviation, Space, Environmental Medicine 1977;48(10):964–68.
(30) Dominguez AM. Problems of carbon monoxide in fires. J Forensic Sci 1962;7(4):379–92.
(31) Chaplin JC. In perspective—the safety of aircraft, pilots and their hearts. European Heart Journal 1988;9(Supplement G):17–20.
(32) Underwood Ground KEA. Prevalence of coronary atherosclerosis in healthy United Kingdom aviators. Aviation, Space, Environmental Medicine 1981;52(11):696–701.
(33) Bennett G. Aviation accident risk and aircrew licensing. The First United Kingdom Workshop in Aviation Cardiology, European Heart Journal 1984;5(Supplement A):9–13.
(34) Cardiovascular problems associated with aviation safety. Eighth Bethesda Conference, American College of Cardiology, The American Journal of Cardiology 1975;36:573–628.
(35) The First United Kingdom Workshop in Aviation Cardiology, European Heart Journal 1984;5(Supplement A):1–164.
(36) Booze CF, Pidkowicz JK, Davis AW, and Bolding FA. Postmortem coronary atherosclerosis findings in general aviation accident pilot fatalities: 1975–77. Aviation, Space, Environmental Medicine 1981;52(1):24–27.
(37) Booze CF, Staggs CM. A comparison of postmortem coronary atherosclerosis findings in general aviation pilot fatalities. Aviation, Space, Environmental Medicine 1987;58(4):297–300.
(38) Reighard HL, Mohler SR. Some aspects of sudden incapacitation in airmen due to cardiovascular disease. Aerospace Medicine 1967;38(12):1273–75.
(39) Mason JK. Previous disease in aircrew killed in flying accidents. Aviation, Space, Environmental Medicine 1977;48(10):944–48.
(40) Dille JR, Morris EW. Human factors in general aviation accidents. Aerospace Medicine 1967;38(10):1063–66.
(41) Mohler SR, Booze CF. U.S. Fatal general aviation accidents due to cardiovascular incapacitation: 1974–75. Aviation, Space, Environmental Medicine 1978;49(10):1225–28.
(42) Engelberg AL, Gibbons HL, Doege TC. A review of the medical standards for civilian airmen. Synopsis of a two-year study. The Journal of the American Medical Association 1986;255(12): 1589–99.
(43) Guidelines for the assessment of cardiovascular fitness in Canadian pilots, 1988. November 1988, Civil Aviation Medicine, Health Advisory Services, Medical Services Branch, Department of National Health and Welfare.
(44) Underwood Ground KE. Occurrence of pre-existing disease in aircrew killed in flying accidents. Aviation, Space, Environmental Medicine 1981;52(11):672–76.
(45) Froom P, Benbassat J, Gross M, Ribak J, Lewis BS. Air accidents, pilot experience, and disease-related inflight sudden incapacitation. Aviation, Space, Environmental Medicine 1988;59(3):278–81.
(46) Mohler SR, Bedell RHS, Ross A, Veregge EJ. Aircraft accidents by older persons. Aerospace Medicine 1969;40(5):554–56.
(47) Booze Jr, DF. Epidemiologic investigation of occupation, age, and exposure in general aviation accidents. Aviation, Space, Environmental Medicine 1977;48(11):1081–91.
(48) Kelly HB. Coronary artery disease in aviation. Journal of the Royal Society of Medicine 1979;72:374–77.
(49) Bennett G. Pilot incapacitation and aircraft accidents. European Heart Journal 1988;9(Supplement G):21–24.
(50) Joy M. Introduction and summary of principal conclusions. The First United Kingdom Workshop in Aviation Cardiology, European Heart Journal 1984;5(Supplement A):1–7.
(51) Harper CR, Albers WR. Alcohol and general aviation accidents. Aerospace Medicine 1964;35(5):462–64.
(52) Ryan LC, Mohler SR. Intoxicating liquor and the general aviation pilot in 1971. Aerospace Medicine 1972;43(9):1024–26.
(53) Gibbons HL, Ellis Jr, JW, Plechus JL. Medical factors in 1964/65 fatal aircraft accidents in the southwest. Aerospace Medicine 1966;37(10):1057–60.
(54) Brown TC, Lane JC. Post-mortem blood alcohol in general aviation pilots. Aviation, Space, Environmental Medicine 1977;48(8): 771–75.
(55) Lacefield DJ, Roberts PA, Blossom CW. Toxicological findings in fatal civil aviation accidents, fiscal years 1968–1974. Aviation, Space, Environmental Medicine 1975;46(8):1030–32.
(56) Underwood Ground KE. Alcohol associated with fatal light aircraft accidents, United Kingdom—1964–1973. Aviation, Space, Environmental Medicine 1975;46(10):1275–79.
(57) Smith PW, Lacefield DJ, Crane CR. Toxicological findings in aircraft accident investigation. Aerospace Medicine 1970;41(7):760–62.
(58) Davis GL. Postmortem alcohol analyses of general aviation pilot fatalities, Armed Forces Institute of Pathology, 1962–1967. Aerospace Medicine 1973;44(1):80–83.
(59) Hill IR. Toxicological findings in fatal aircraft accidents in the United Kingdom. The American Journal of Forensic Medicine and Pathology 1986;7(4):322–26.
(60) Canfield DV, Kupiec T, Huffine E. Postmortem alcohol production in fatal aircraft accidents. J Forensic Sci 1993;38(4):914–17.
(61) Instructions re: Aviation Accident Specimen Collection, Preservation and Continuity, Civil Aviation Medical Unit (Health and Welfare Canada), October, 1989.
(62) Ross LE, Ross SM. Pilots' attitudes toward alcohol use and flying. Aviation, Space, Environmental Medicine 1988;59(10):913–19.
(63) Modell JG, Mountz JM. Drinking and flying—the problem of alcohol use by pilots. New England Journal of Medicine 1990;323(7): 455–61.
(64) Billings CE, Wick Jr, RL, Gerke RJ, Chase RC. Effects of ethyl alcohol on pilot performance. Aerospace Medicine 1973;44(4): 379–82.
(65) Damkot DK, Osga GA. Survey of pilots' attitudes and opinions about drinking and flying. Aviation, Space, Environmental Medicine 1978;49(2):390–94.
(66) Gilson RD, Schroeder DJ, Collins WE, Guedry Jr, FE. Effects of different alcohol dosages and display illumination on tracking performance during vestibular stimulation. Aerospace Medicine 1972; 43(6):656–60.
(67) Yesavage JA, Leirer VO. Hangover effects on aircraft pilots 14 hours after alcohol ingestion: a preliminary report. American Journal of Psychiatry 1986;143(12):1546–50.
(68) General Operating and Flight Rules, (United States), 14 CFR 91.17, Alcohol or Drugs.
(69) Section 409. Federal Air Regulations (Canada), 1992.
(70) Ross SM, Ross LE. Pilots' knowledge of blood alcohol levels and the 0.04% blood alcohol concentration rule. Aviation, Space, Environmental Medicine 1990;61(5):412–17.
(71) Underwood Ground KE. Liver pathology in aircrew. Aviation, Space, Environmental Medicine 1982;53(1):14–18.
(72) Kuhlman JJ, Levine B, Smith ML, Hordinsky JR. Toxicological findings in Federal Aviation Administration general aviation accidents. J Forensic Sci 1991;36(4):1121–28.
(73) O'Hare D. Pilots' perception of risks and hazards in general aviation. Aviation, Space, Environmental Medicine 1990;61(7):599–603.

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