Medical Conditions and Driving:

A Review of the Literature (1960 – 2000)

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16. Abstract:

This report reviews the contribution of medical conditions and functional limitations (e.g., sensory, motor, or cognitive functioning) to motor vehicle crashes. It provides a comprehensive and up-to-date review of the international research literature on the effects of medical and functional conditions on driving performance. The report is divided into 15 sections (Introduction, Vision, Hearing, Cardiovascular Diseases, Cerebrovascular Diseases, Peripheral Vascular Diseases, Diseases of the Nervous System, Respiratory Diseases, Metabolic Diseases, Renal Diseases, Muscuoloskeletal Disabilities, Psychiatric Diseases, Drugs, The Aging Driver, and the Effects of Anesthesia and Surgery). Each section contains a brief overview of the condition/illness; prevalence information; a review of the medical, gerontological, and epidemiological literature relevant to the condition/illness, followed by current fitness to drive guidelines for the condition/illness from Canada and Australia. The Appendix presents preliminary guidelines for physicians to assess medical fitness-to-drive.

The report is a scholarly but practical compendium that can serve as a valuable resource for physicians, rehabilitation practitioners, other allied health care professionals and educators, Department of Motor Vehicle personnel, road and traffic safety personnel, transportation planners, highway safety researchers, and public policymakers. Its value is particularly relevant as the driving population increases in size and age.

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Foreword

This report provides a comprehensive review of past and current research on the effects of medical conditions on driving performance. It is divided into 15 sections (Introduction, Vision, Hearing, Cardiovascular Diseases, Cerebrovascular Diseases, Peripheral Vascular Diseases, Diseases of the Nervous System, Respiratory Diseases, Metabolic Diseases, Renal Diseases, Muscuoloskeletal Disabilities, Psychiatric Diseases, Drugs, The Aging Driver, and The Effects of Anesthesia and Surgery). Each section provides a brief overview of the condition/illness; prevalence information; review of the medical, gerontological, and epidemiological literature relevant to medical conditions and driving; followed by current fitness to drive guidelines from Australia and Canada for the condition/illness. An appendix contains preliminary guidelines developed to assist physicians in determining when patients have medical conditions that can affect fitness-to-drive.

This report is a scholarly but practical compendium that can serve as a valuable resource for physicians, rehabilitation practitioners, other allied health care professionals and educators, Department of Motor Vehicle personnel, road and traffic safety personnel, transportation planners, highway safety researchers, and public policymakers. Its value is particularly relevant as the driving population increases in size and age.

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Section 1: Introduction

Motor vehicle crashes are a leading cause of death in the United States. Data from the National Center for Health Statistics of the U.S. Department of Health and Human Services reveal that, in 1997, motor vehicle crashes resulted in 42,340 deaths, ranking eighth behind heart disease, cancer, and stroke as a leading cause of death (National Highway Traffic Safety Administration [NHTSA], 2000). The causes of motor vehicle crashes are varied, including road design, vehicle design, and traffic volume. However, it has been estimated that as much as 90 percent of highway crashes are due to human error (Tignor, 2000). Although data on the overall contribution of medical conditions to motor vehicle crashes are unavailable, it is reasonable to assume that medical conditions that affect functional capabilities (e.g., sensory, motor, or cognitive functioning) play a major role.

This report, entitled Medical Conditions and Driving: A Review of the Scientific Literature, provides a comprehensive and integrative review of past and current research (to the year 2000) on the effects of medical conditions on driving performance. The report is divided into 15 sections (Introduction, Vision, Hearing, Cardiovascular Diseases, Cerebrovascular Diseases, Peripheral Vascular Diseases, Diseases of the Nervous System, Respiratory Diseases, Metabolic Diseases, Renal Diseases, Musculoskeletal Disabilities, Psychiatric Diseases, Drugs, The Aging Driver, and The Effects of Anesthesia and Surgery). Each section is divided into subsections, with a brief overview of the condition/illness, information on prevalence, a review of the literature relevant to driving, followed by current fitness-to-drive guidelines from Australia and Canada for the condition/illness. The guidelines from Canada (Canadian Medical Association [CMA], 2000) and Australia (Austroads, 1998) have been reproduced with permission. Sincere appreciation is extended to both the CMA and Austroads for allowing the reproduction of their guidelines.

Medical Conditions that Serve as 'Red Flags' for Driving Impairment

A number of medical conditions may result in functional impairments that negatively affect driving performance. The effects can result in functional impairments that are either acute or chronic. The distinction between acute and chronic becomes critical in terms of assessment for fitness-to-drive and for licensing decisions (A. Dobbs, personal communication).

Acute Effects

With acute effects (e.g., an epileptic seizure, a hypoglycemic reaction), the event is, most often, sporadic and unpredictable. There is no question that when the event occurs, the individual is not competent to drive. The difficulty, in terms of licensing decisions, is that the occurrence of the event is unpredictable. This means that decisions about the individual's safety to drive cannot be based on direct measurement. Therefore, decisions about continued driving and/or potential restrictions on driving activities may need to be based on a consensus of estimated risk (e.g., expert panel decisions, calculated relative risk) to the person and society.

Chronic Effects

Unlike the acute effects of medical conditions, chronic effects are, by definition, more enduring. In addition, unlike acute effects, chronic effects are relatively predictable and stable. Importantly, the impact of chronic effects on an individual's driving ability is measurable. Thus, decisions about continued driving can be based on measures of individual performance rather than on estimates of risk. The challenge has been to operationalize performance in a manner that is valid, reliable, and defensible.

Acute and Chronic Effects

Some medical conditions can have both acute and chronic effects. For example, diabetes can have an acute effect (hypoglycemic reaction) and chronic effects (diabetic retinopathy, cardiovascular complications, diabetic neuropathy, etc.). Similarly, cardiovascular disease can be associated with acute effects (myocardial infarction) and with chronic effects (hypertrophic cardiomyopathy, congestive heart failure, etc.).

Research designed to increase our understanding of the effects of medical conditions on driving has increased substantially in recent years. Despite the methodological challenges of conducting research in this area, a substantial body of literature now exists on the relationship between many medical conditions and driving performance. In some instances, there is a clear link between the presence of a medical condition and impaired driving performance, allowing health care professionals to make evidence-based decisions. In other instances, the relationship is less clear.

The medical conditions listed below have been found, through research, to be associated with a higher risk of crash and/or have been associated with cognitive impairment and/or significant functional impairments (visual, motor). The list is confined to those conditions

that are chronic. Although there are likely to be other medical conditions that have the potential to adversely affect driving performance, those conditions may not appear on the list because of a lack of research into the effects of the condition on driving performance. Importantly, not everyone with an illness listed below would have their driving reduced to an unsafe level. Rather, the presence of one or more of the illnesses should serve as a 'red flag' that driving may be compromised, and that evaluation of driving competence is needed for both personal and public safety.

Summaries of current fitness-to-drive guidelines for medical practitioners from Australia (1998) and Canada (2000) are presented throughout this report. While these guidelines are not controlling on licensing authorities or physicians in the United States or endorsed by NHTSA, they are provided for informational and/or reference purposes.

Legal Limitations

In considering applications for fitness-to-drive guidelines, it should be noted that licensing activities of state and local motor vehicle agencies in the United States must comply with both the Americans with Disabilities Act of 1990 ('ADA') and the Rehabilitation Act of 1973 ('Rehabilitation Act'). Under these statutes and their implementing regulations, public entities may utilize neutral rules and criteria, such as medical guidelines, even if they screen out, or tend to screen out, individuals with specific medical conditions, provided the criteria are necessary for the safe operation of a program. However, the public entity must ensure that its medical standards are based on real risks, not on speculation, stereotypes, or generalizations about individuals with specific medical impairments. Consequently, the ADA and Rehabilitation Act typically require that a State Department of Motor Vehicles base its licensing decisions not on risk analyses, but on individual fitness-to-drive assessments that examine whether an applicant poses a direct threat to public safety, which cannot be eliminated through auxiliary aids or reasonable modifications of policies, practices, or procedures.

Medical Conditions that serve as 'Red Flags' that Driving Ability may be Compromised

A. Visual Conditions/Diseases

1. Low vision (vision ranging from 20/200 to 20/50)
2. Cataracts
3. Diabetic retinopathy
4. Glaucoma
5. Retinitis pigmentosa
6. Monocular vision (especially right eye blindness)
7. Macular degeneration
8. Nystagmus
9. Visual field defects

B. Cardiovascular Disease

1. Cardiac arrhythmias if associated with cerebral ischemia (e.g., paroxysmal arrhythmias such as non-sustained paroxysmal ventricular tachycardia, paroxysmal supraventricular tachycardia, paroxysmal atrial fibrillation/flutter; sinus node dysfunction)
2. Artificial cardiac pacemakers if associated with cerebral ischemia
3. Hypertrophic cardiomyopathy if associated with cerebral ischemia
4. Congestive heart failure if associated with cerebral ischemia
5. Valvular heart disease if associated with cerebral ischemia

C. Cerebrovascular Disease

1. Cerebrovascular accident (Stroke)
2. Transient ischemic attacks

D. Diseases of the Nervous System

1. Narcolepsy
2. Sleep apnea

E. Respiratory Diseases

1. Chronic obstructive lung disease if associated with respiratory failure resulting in cognitive impairment due to generalized hypoxia
2. Respiratory failure

F. Metabolic Diseases

1. Hypothyroidism if condition results in cognitive deficits
2. Diabetes - the chronic effects of diabetes (e.g., diabetic retinopathy, cardiovascular disease, etc.) are listed separately

G. Renal Disease

1. Chronic renal failure if associated with cognitive impairment

H. Dementia

1. Progressive dementia (e.g., Alzheimer's disease, Multi-infarct dementia)

I. Psychiatric Diseases

1. Schizophrenia
2. Personality disorder
3. Chronic alcohol abuse

J. Medications

Chronic use of the following medications:

1. Antidepressants (particularly the older tricyclics such as amitriptyline, imipramaine)
2. Antihistamines (particularly the older antihistamines)
3. Any drug that has prominent central nervous system effects (e.g., analgesics, some antihypertensives, sedatives, hypnotics, anxyiolytics, benzodiazepines, stimulants)

Section 2: Vision

The driving task is a highly visual one. It has been estimated that 90 percent of information used while driving is visual (Hills, 1980). Despite the apparent relationship between good visual function and safe driving performance, research has failed to find a strong relationship between the two. As noted a number of years ago by Burg (1971), there are at least five reasons for the reported weak relationship between visual functioning and driving performance. These include: (1) vision is but one factor affecting driver performance, (2) the disparity between an individual's visual capacities and the extent those capacities are used or needed in driving, (3) a lack of validity between tests used in research and the visual demands of driving, (4) the possibility of low reliability of the criterion measure of driving, and (5) methodological shortcomings of studies assessing the relationship between visual functioning and driving performance. A number of excellent reviews of the literature are available on vision and driving. The reader is directed to those for a general review of the literature (Charman, 1997; Owsley and McGwin, 1999), and for a review of visual changes with age (Kline, Kline, Fozard, et al., 1992; Kline and Scialfa, 1996: Owsley and Ball, 1993; Shinar and Schieber, 1991).

A number of conditions can affect visual functioning. The literature on those conditions is reviewed below. Additionally, a summary of the current fitness-to-drive guidelines (Visual Conditions/Diseases) for medical practitioners from Australia (1998) and Canada (2000) is presented in Table 1.

2.1 Acuity

2.1a. Static Visual Acuity

Errors of refraction are the most common type of visual disorder. An individual with 20/20 vision (or 6/6 metric) is classified as having normal visual acuity. An individual with 20/70 vision or less (even when wearing corrective lenses) is classified as visually impaired. A person is legally blind when their vision is 20/200 or less, even when wearing glasses.

On initial applications for a driver's license, all United States and Canadian jurisdictions require vision testing (Keeney, 1993). In most states in the United States (42 States or 84 percent), an unrestricted private driver's license requires visual acuity of 20/40 or better (corrected or uncorrected) in one eye alone. In other countries (e.g., Europe, Asia, Africa, the Middle East, Australia), the most prevalent acuity standard is 20/40 combined, or both eyes tested together (Keeney, 1993). Forty states (Shipp, 1998), the District of Columbia, and three Canadian provinces require vision testing at license renewal (Petrucelli and Malinowski, 1992). Recently, Shipp (1998) assessed the impact of vision-related re-licensing policies on traffic fatalities in 48 contiguous states and the District of Columbia. Data were obtained from the Fatal Accident Reporting System. Results of that investigation revealed lower vehicle occupant fatality rates of older drivers in those states with vision-related re-licensing policies. Thus, as noted by the author, state-level mandatory vision testing for re-licensure may enhance traffic safety and reduce the economic costs of fatal crashes.

Results from a large-scale study (Diller, Cook, Leonard, Dean, Reading, and Vernon, 1998) indicate that individuals who have a history of eye conditions that may affect driving have a higher risk of crashes compared to controls matched on age, gender, and county of residence. In 1979, the Utah Driver License Division implemented a program that restricts drivers with medical conditions according to their functional ability levels. Since the inception of the program, all licensing applicants are required to complete a questionnaire regarding their physical, mental, and emotional health. Individuals who self-report a medical condition are categorized by medical history (e.g., diabetes, neurologic, etc.) and functional ability. Based on results from the questionnaire, an applicant may immediately receive a license or be required to complete a more extensive health history form. On the basis of the screening process, applicants may be denied a license, or receive a fully unrestricted or restricted license. Data obtained from the state licensing agencies between 1992 and 1996 were subsequently linked to the Utah Department of Transportation Crash files. This allowed for the comparison of crash and citation rates of restricted and unrestricted drivers with medical conditions to controls matched on age group, gender, and county of residence. Relevant to this discussion,

drivers in the visual impairment category consisted of 13,075 drivers in the unrestricted category and 2,263 drivers in the restricted category. Drivers without restrictions had a significantly greater risk of crashes compared to controls (RR = 2.38, CI = 2.24 - 2.53). The relative risk for drivers with restrictions was 1.31 (CI = 1.10 - 1.56).

2.1b. Dynamic Visual Acuity

The act of driving primarily involves the ability to discriminate an object when there is relative movement between the object and observer. Therefore, tests of dynamic visual acuity rather than static visual acuity would seem to be more relevant for assessments of safe driving performance. In contrast to static visual acuity, dynamic visual acuity is a reliable predictor of crash probability (Fox, 1989; Graca, 1986; Hills and Burg, 1977; Reuben, Silliman, and Traines, 1988). In view of this, it is surprising that tests of dynamic visual acuity are seldom, if ever, included in traditional license renewal assessments. Importantly, declines in dynamic visual acuity and lateral motion detection start at an earlier age and accelerate faster, whereas deterioration in static visual acuity occurs later and progresses more slowly (Shinar and Schieber, 1991).

2.1c. Low Vision and Telescopic Lens

Individuals with low vision have impaired vision that cannot be fully corrected by ordinary prescription lenses, medical treatment, or surgery. Low vision is defined as vision ranging from 20/200 to 20/50, or when the corrected vision becomes a disability to the point at which one cannot function at his or her vocation (Fonda, 1986). Recent estimates suggest that approximately 14 million (one in twenty) Americans have low vision (Kupfer, 1999).

Low vision in older persons is most often the result of pathologies such as cataracts, macular degeneration, glaucoma, and diabetic retinopathy, or from a cerebrovascular accident. Individuals with low vision may experience one or more of the following: overall blurred vision, loss of central vision, and loss of peripheral vision. Telescopic spectacles and other low vision aids are used to assist individuals with low vision.

Despite the importance of research in this area, there has been little research on the use of telescopic lenses and driving performance. The literature that is available generally discusses the benefits and drawbacks of the use of telescopic lenses while driving. However, there is little in the way of data to support the pros or cons of their use. Limitations of telescopic lens systems have been documented by Lippman (1976) and Scott (1976). Those limitations include small central fields, ring scotomas, nearness illusion, movement of the image in the opposite direction of any head movement, reduced resolving power due to vibration, and altered head posturing. However, the papers are more than 25 years old and considerable improvements have been made in the technology of telescopic systems in recent years (Park, Unatin, and Hebert, 1993).

Advances in technology of telescopic systems include rear mounting, miniaturization, micro spiral galilean, vision enhancing systems, and bi-level telemicroscopic and behind the lens systems (Park et al., 1993). Research is needed, however, on the use of the new telescopic systems and driving performance. It is interesting to note that Park et al. (1993) have developed a driving program for visually handicapped telescopic drivers. The program is designed to ensure that every visually impaired telescopic driver meets the legal visual acuity and visual field requirements. In addition, the program is designed to improve the competency of telescopic lens use while driving. Details on the efficacy of the program are, however, lacking at this time.

2.2 Cataracts

A cataract is a clouding of the lens of the eye, which blocks light from reaching the retina of the eye. Cataracts can result in compromised visual acuity (Mantyjaravi and Tuppurainen, 1999; Rubin, Adamsons, and Stark, 1993), contrast sensitivity (Mantyjaravi and Tuppurainen, 1999; Rubin et al., 1993), and visual field sensitivity (Heuer, Anderson, Knighton, et al., 1988).

More than 12.9 million Americans age 40 and older have cataracts - about one in every seven persons (Vision Problems in the U.S., 2000). Cataracts may be due to a variety of causes: some are congenital, few occur during the early years of life, but the majority are the result of the aging process. Cataracts are a leading cause of vision impairment in older adults, affecting almost half of individuals 75 to 85 years of age (Klein, Klein, and Linton, 1992).

Treatment of cataracts involves the removal of the clouded lens either through phacoemulsification (phaco) or extracapsular surgery. With phaco, the clouded lens is removed by first breaking up the lens using ultrasound waves. The lens is then removed by suction. This technique requires an incision of only approximately three millimeters. With extracapsular surgery, the lens nucleus is removed in one piece through a larger incision on the side of the cornea. In most cataract surgeries, the removed lens is replaced by a clear, artificial intraocular lens. Research suggests that more than 85 percent of cataract cases achieve acuity of 20/40 or better following intraocular lens implant (Stark, Worthen, Holladay, et al., 1983). Superstein, Boyaner, and Overbury (1999)

evaluated visual acuity, spatial contrast sensitivity, and glare disability in cataract patients pre- and post-operatively. Pre-operatively, all patients had decreased visual acuity and spatial contrast sensitivity in the presence of glare, with statistically significant improvements in both following cataract surgery.

Talbot and Perkins (1998) have recently assessed the benefit of second eye cataract surgery in a sample of 50 patients. After the first cataract surgery, 88 percent had a visual acuity of 20/40. However, only 52 percent met the United Kingdom's Driving and Vehicle Licensing Agency (DVLA) driving standards for visual acuity and field. Following cataract surgery on the second eye, 60 percent of patients had improved binocular visual acuity. Stereo acuity improved from 32 percent after the first eye surgery to 90 percent after the second eye surgery. In addition, binocular horizontal field of vision improved by 20 degrees or more in 54 percent of the patients and binocular vertical vision improved in 36 percent of patients after the second eye surgery. Importantly, the proportion of patients meeting the DVLA standards improved from 52 percent after the first eye surgery to 85 percent after the second eye surgery. Results of this investigation suggest that second eye cataract surgery provides a significant improvement in binocular function and enables a substantially greater proportion of individuals to meet DVLA standards.

Although a number of studies reveal significant improvements in visual functioning following cataract surgery, there is little in the way of research to indicate when driving may safely resume following surgery. As noted by Munton (1997), there should be a post-operative period of adaptation before driving resumes. Munton suggests that, with current surgical methods, this period may be as short as one week. However, supporting data are unavailable.

Cataracts and Driving Literature Review

Results of a recent investigation indicate that individuals with cataracts have a higher risk of motor vehicle crashes. In 1999, Owsley, Sekuler, and Siemsen investigated the relationship between cataracts and crash risk in older community dwelling adults. Participants (aged 55-85) included 279 older adults with cataracts and 105 without cataracts who were legally licensed to drive. Crash data on all participants from the 5 years prior to study enrollment were obtained from state records. After adjustment for driving exposure, results revealed that drivers with cataracts were 2.5 times more likely to have a history of at-fault crashes in the previous five years compared to those without cataracts (RR = 2.48, 95 percent CI = 1.00 - 6.14). After adjusting for impaired health, the association between cataract and crash involvement remained significant (RR = 2.46, 95 percent CI = 1.00 - 6.16).

To date, there are no studies available that have examined the effects of cataract surgery on crash risk. Monestam and Wachtemeister (1997) examined the outcome of cataract surgery on the patients' self-estimation of visual functioning while driving. As reported by the authors, visual problems while driving decreased from 82 percent pre-operatively to 5 percent post-operatively. Visual problems included difficulty in driving in darkness and at twilight, problems with distance estimation, and difficulties with glare. Results also revealed that second eye cataract surgery should be performed, if necessary, in order to achieve good binocular vision and enhanced distance estimation, findings congruent with those reported by Talbot and Perkins (1998).

Although limited, the data suggest that individuals with cataracts are at significantly greater risk (2.5 times) for crashes than those without cataracts. The presence of a cataract can interfere with visual functioning by decreasing acuity, contrast sensitivity, and visual field sensitivity. Therefore, for those individuals who have not undergone surgical treatment for cataract, assessments of visual functioning should include not only tests of visual acuity but also of contrast, glare, and visual field sensitivity. Research reveals significant improvements in visual functioning following cataract surgery. Although the data are limited, second eye cataract surgery (if necessary) provides significant improvement in visual functioning and appears warranted. Future research is required to establish the parameters for safe resumption of driving following cataract surgery.

2.3 Color Vision Defects

Individuals with color vision defects lack a perceptual sensitivity to certain colors. There are three types of color receptors in our eyes: red, green, and blue. An inability to distinguish red from green is the most common form of color blindness, with blue deficiencies occurring very rarely. A color vision defect is the result of an inherited trait that occurs almost exclusively in males. Estimates suggest that eight percent of males and less than one percent of females have some difficulty with color vision (Gouras, 1991).

Color Vision Defects and Driving Literature Review

Surprisingly, there are few studies available with data relevant to color vision defects and crash rates. Individuals that are red colorblind (protanope) appear

to have almost twice as many rear-end collisions as those with both red and green color deficits (deutans) and normals (Verriest, Naubaauer, Marre, and Vvijls, 1980; Vingrys and Cole, 1988). Results from Steward and Cole (1989) suggest that individuals with color vision defects have difficulty, in general, when driving. In their study, Steward and Cole administered a questionnaire to 102 individuals with congenital color blindness. Twenty-nine percent of the sample reported having difficulty distinguishing the color of traffic signal lights, 32 percent to having confused traffic lights with streetlights, and 13 percent reported having difficulty in detecting brake lights on other cars.

Despite the reported difficulties with color vision discrimination while driving, it is unlikely that color vision impairments, in general, represent a driving hazard, particularly now that the position of traffic lights has been generally standardized (Canadian Medical Association, 1999). Nevertheless, it seems prudent to caution drivers regarding potentially hazardous situations (e.g., traffic lights, brake lights, parked cars).

2.4 Contrast Sensitivity

Contrast sensitivity is a measure of an individual's ability to perceive visual stimuli that differ in both contrast and spatial frequency. Age-related declines in static contrast sensitivity are most evident at intermediate and higher spatial frequencies (Crassini, Brown, and Bowman, 1988; Scialfia, Adams, and Givanetto, 1991). Deficits in static contrast sensitivity have been shown to be reduced, but not eliminated, when individuals are optimally corrected (Owsley et al., 1983). When dynamic targets are used in measures of contrast sensitivity (e.g., gratings that move across the screen), deficits in contrast sensitivity are much greater in elderly individuals compared to their younger counterparts (Scialfa, Garvey, Tyrrell, Goebel, Deering, and Leibowitz, 1988).

A number of studies have examined the relationship between measures of contrast sensitivity and driving performance. In a study evaluating the effects of cataracts on mobility, Owsley (2000) found contrast sensitivity to be a better predictor of crash involvement than measures of visual acuity. Results from that investigation revealed that individuals with cataracts pre-surgery with impairments in contrast sensitivity had a significantly higher risk of crash involvement than age matched controls without cataracts (RR = 2.70 with one eye impaired and RR = 5.78 with both eyes impaired). Decina and Staplin (1993) evaluated the relationship between several visual measures and selected crash categories over a 3.67-year period in 12,400 drivers in Pennsylvania. Neither visual acuity nor horizontal visual measures alone were related to crash involvement. However, the combination of visual acuity, horizontal visual fields, and broad contrast sensitivity criteria were associated with increased crash involvement for drivers aged 66-75, and 76 and older. Wood and Troutbeck (1995) report correlations of 0.71 between Pelli-Robson scores (based on the lowest contrast letter that can be recognized) and performance on a driving task in studies on the effects of various simulated visual impairments. Rubin, Roche, Prasada-Rao, and Fried (1994) found significant correlations between Pelli-Robson contrast sensitivity scores and self-reported difficulties in day and night driving.

In general, the research that is available suggests that impairments in contrast sensitivity are associated with higher rates of crashes. Results from Owsley (2000) suggest that measures of contrast sensitivity may be a more sensitive predictor of crash risk than current measures of visual acuity. Although more research is needed, it may be prudent to include measures of contrast sensitivity in assessments of older drivers.

2.5 Diabetic Retinopathy

According to recent statistics (Vision Problems in the U.S., 2000), 10 to 14 million people in the United States have diabetes. Nearly 40 percent, or 4 to 6 million, have diabetic retinopathy, which is the most common type of diabetic eye disease (National Eye Institute [NEI], 2000), and one of the leading causes of blindness in the United States (Richter, 1987). The incidence of diabetic retinopathy has increased with the increase in the long-term survival of diabetics (Richter, 1987). The prevalence increases with age and disease duration, with women affected more often than men (NEI, 2000).

There are two types of diabetic retinopathy: background and proliferative. Characteristic features of background retinopathy include micoraneurysms, venous dilation, exudates, hemorrhages, and retinal edema (Richter, 1987). Background retinopathy is often asymptomatic, but may result in decreased visual acuity. Proliferative retinopathy is the result of retinal hypoxia and carries a much graver prognosis (Richter, 1987). Proliferative retinopathy is characterized by a proliferation of new vessels in the retina or on the optic disc (neovascularization). Hemorrhage, retinal breaks, and retinal detachment can occur in the network of fragile vessels, resulting in vision loss and blindness.

Frequent eye examinations (every 6 to 12 months for diabetics) are an important step in the early detection and prevention of diabetic retinopathy. Laser surgery is the current treatment of choice for diabetic retinopathy. In proliferative retinopathy, the risk of severe vision loss has been reduced by 60 percent with the use of laser surgery (NEI, 2000). Vitrectomy, evacuation of hemorrhagic or fibrous tissue in the vitreous, may be the treatment of choice in individuals with advanced proliferative retinopathy or retinal detachment.

Diabetic Retinopathy and Driving Literature Review

The overwhelming majority of the literature on diabetic retinopathy and driving is concerned with the effects of panretinal photocoagulation (PRP) for proliferative diabetic retinopathy on visual fields. PRP reduces the risk of severe visual loss in proliferative diabetic retinopathy (The Diabetic Retinopathy Research Group, 1978). However, the procedure is associated with visual field loss and reductions in peripheral vision (Sieberth, Alexandrides, and Feng, 1987; Zaluski, Marcil, Lamer, and Lambert, 1986).

Buckley, Jenkins, and Benjamin (1992) studied the effects of PRP in 30 diabetic patients following full PRP. Fifteen of the patients failed the Humphrey binocular visual field test (a visual field less than 120 degrees along the horizontal with 20 degrees above and below the horizontal level). Patients who failed were more likely to be hypertensive and to have undergone treatment with a xenon laser. No differences were noted between those who passed and those who failed on a number of factors including age, sex, diabetic age, and number and size of burns. Hulbert and Vernon (1992) assessed the visual fields of 21 diabetics following PRP. In that investigation, 89 percent of the patients treated with laser alone met the United Kingdom Driver and Vehicle Licensing Agency (DVLA) visual field requirements. Similar to the findings reported by Buckley et al., treatment with xenon laser was associated with a higher risk of failure of DVLA requirements. However, unlike Buckley, et al., Hulbert and Vernon found that total burn area also was associated with a higher rate of failure. In a significantly larger sample, Mackie, Webb, Hutchinson, et al. (1995) evaluated 100 diabetic patients following bilateral PRP for proliferative diabetic retinopathy. Thirty percent of their patients failed to reach the DVLA standards following treatment. Finally, Pearson, Tanner, Keightley, and Casswell (1998) report the results of PRP in diabetic patients. Forty two percent of uniocular fields from treated eyes and 12 percent of binocular fields from those having bilateral PRP failed the United Kingdom's licensing requirements. In this investigation, Type II diabetes was associated with a significant increase in risk of failure. The large variation in failure rates across studies is likely due to variations in sample size and differences in the interpretations of minimum field requirements. For example, Pearson, Keightley, and Casswell (1998) reported on evaluations of visual field defects in 60 diabetic patients following PRP from the chairman of the Visual Standards Sub-Committee of the Royal College and separate evaluations from four consulting ophthalmologist. Significant discrepancies existed for both binocular and uniocular fields. In order to reduce the variability in assessments, guidelines are provided. In addition, Hulbert and Vernon (1992) provide guidelines for laser treatment in diabetic retinopathy aimed at preserving the driving visual field.

2.6 Glaucoma

It is estimated that between 2 and 3 million Americans aged 40 and older, or about 1 in every 30 people in that age group, have glaucoma (Vision Problems in the United States, 2000). Glaucoma is one of the leading causes of blindness, accounting for between 9 percent and 12 percent of all cases of blindness. The rate of blindness from glaucoma is between 93 and 126 per 100,000 population 40 years or older.

Glaucoma is a group of diseases characterized by increased intraocular pressure. The increased intraocular pressure (defined as pressure > 21 mm Hg) can lead to optic nerve damage, resulting in blindness (Richter, 1987). Types of glaucoma include adult primary glaucoma (chronic open angle, acute and chronic narrow angle, closed angle, and acute congestive), secondary, congenital, and absolute glaucoma. Open angle glaucoma is the most common, affecting 3 million Americans (NEI, 2000). It often is referred to as the 'silent blinder' because extensive damage may occur before the patient is aware of the disease (Richter, 1987). Those at increased risk for developing glaucoma include blacks, those over the age of 60, and individuals with a family history of glaucoma (NEI, 2000).

Early diagnosis and treatment are important for the prevention of optic nerve damage and visual field loss (primarily peripheral vision) due to glaucoma. Treatment includes medications that reduce aqueous fluid production or that facilitate the outflow of fluid. Laser surgery has been shown to be a safe and effective alternative to pharmacotherapy (NEI, 2000).

Glaucoma and Driving Literature Review

A number of studies have examined the relationship between peripheral visual function and driving performance. A number of earlier studies have reported a significant relationship between peripheral field loss and crash rates (Fishman, Anderson, Stinson, and Haque, 1981; Keeney et al., 1981). In one of the most extensive investigations, Johnson and Keltner (1983) studied the relationship between peripheral vision status and crash rates. Results from 8,767 volunteers (17,534 eyes) showed an incidence of visual field loss of 3-3.5 percent for persons age 16 to 60 years of age, with a four-fold increase to 13 percent for those 65 years of age and older. Visual field defect was noted in approximately 35 percent of individuals reporting the presence

of glaucoma. The authors noted that this value might be lower than expected for two reasons. First, the 'diagnosis' of glaucoma is based on self-report rather than medical examination, which may mean that many of the subjects have ocular hypertension rather than glaucoma. Second, a high target luminance was selected to minimize the number of false-positive results thereby increasing the likelihood of not detecting shallow defects. Nevertheless, the frequency of visual field loss in individuals with glaucoma was 10 times greater than for the general population examined. Results of the study also indicated that individuals with a family history of glaucoma had a higher incidence (5.6 percent) of visual field loss compared to the general population studied. Important to this review was the finding that individuals with visual field loss in both eyes had double the crash and conviction rates (per person per 160,000 km) as an age- and sex-matched control group with normal visual fields. The crash and conviction rates of those with monocular visual field loss were not significantly different than age- and sex-matched controls.

Fishman et al. (1981) studied 42 individuals with retinitis pigmentosa with significant visual field loss. Small differences were found between the crash rates of patients with pathology and a normal control group. A later study with 21 drivers with more severe visual field loss with retinitis pigmentosa found a significant relationship between the extent of visual field loss and motor vehicle crashes. These findings are congruent with results from Elkington and MacKean (1982). In that study, individuals with glaucoma with visual field loss also had significantly higher crash rates.

Results from Johnson and Keltner (1983) provide support for the feasibility of using automated visual field tests to perform mass visual field screening. In addition, the findings of significantly higher crash rates for individuals with visual field loss (Elkington and MacKean, 1982; Fishman et al., 1981; Johnson and Keltner, 1983; Keeney et al., 1981) suggest that screening tests for peripheral vision need to be considered for new drivers and for existing drivers at license renewal.

2.7 Loss of Vision in One Eye (Monocular Vision)

Research on the monocular driver is limited, with many of the studies conducted before the early 70's (Freytag and Sachs, 1969; Keeney, 1968; Kite and King, 1961; Liesman, 1973). Results of those investigations reveal that, in general, drivers with monocular vision have a greater number of crashes, more hazardous driving patterns, and a greater number of road problems compared to normal sighted drivers. Keeney et al. (1981) reported on the driving performance of 52 monocular drivers enrolled in Kentucky's Driver Limitation Program from 1976 through 1980. Crash and traffic violations were obtained from state driving records. Results indicated that monocular drivers have almost double the rate of crashes compared to the general motoring public. Monocular drivers also were found to have more reckless driving violations compared to their binocular counterparts. Side of monocularity was found to be a significant factor. Individuals with right eye blindness had significantly more traffic violations than those with left eye blindness. Interestingly, there were no significant differences between the crash or violation rates of drivers with license restrictions (mandatory left hand outside mirror) compared to those without license restrictions.

The most recent study was conducted by McKnight, Shinar, and Hilburn (1991). McKnight et al. evaluated the visual and driving performance of 40 monocular and 40 binocular tractor-trailer drivers. The monocular drivers exhibited impairments in contrast sensitivity, visual acuity under low illumination and glare, and binocular depth perception. Interestingly, there were no significant differences between the two groups of drivers on measures of visual search, lane keeping, clearance judgement, gap judgement, hazard detection, and information recognition. The authors concluded that despite the reductions in selective visual functions, monocular drivers are not significantly worse than binocular drivers in the safety of most day-to-day driving functions. Measures of crash rates were not included in the study.

Despite the evidence that monocular drivers have greater crash and traffic violation rates than binocular drivers, there are few restrictions placed on this category of drivers (see Table 1).

2.8 Macular Degeneration

Recent statistics suggest that more than 13 million people in the United States 40 years of age and older have signs of macular degeneration and more than 1.2 million have the later, vision-threatening stages of the disease. Age-related macular degeneration may account for up to 30 percent (230,000 cases) of all bilateral blindness among Caucasian Americans and is a leading cause of blindness in Americans 55 years of age and older (Steinert, 1987). The greatest risk factor for acquiring macular degeneration is age. Other risk factors include gender (females more at risk than males), race (whites more at risk than blacks), smoking, and family history (NEI, 2000).

Macular degeneration is a progressive and irreversible destruction of receptors in the central portion of the retina. This central portion, known as the macula, is responsible for focusing central vision in the eye. Destruction of the macula affects an individual's ability to read, to drive a car, to recognize faces or colors, and to see objects in fine detail.

Macular Degeneration and Driving Literature Review

Two recent studies have examined the effects of macular degeneration on driving performance. In the first study, Szlyk, Fishman, Severing, Alexander, and Viana (1993) compared the driving performance of 20 subjects with central vision impairment (juvenile macular dystrophies) to that of 29 individuals with normal vision. Mean age for the visually impaired group was 36.1 years and 38.9 years for the control group. Based on self-report, both groups of subjects had similar driving histories. Assessment of driving performance consisted of driving simulator performance and crash rates based on self-report and state records. Results revealed similar crash rates for the two groups. However, subjects with central vision loss who did not restrict their driving to daylight hours had a higher likelihood of being involved in nighttime crashes compared to the control group. Results from simulator performance revealed longer brake response times and a greater number of lane crossings for the central vision loss group. Visual function measures and simulator performance failed to predict crash performance for the visual function loss group.

The second study on central vision impairment (Szlyk, Pizzimenti, Fishman, Kelsch, Wetzel, and Kagan, 1995) compared the driving performance of 10 older subjects with age-related macular degeneration to that of 11 age-similar subjects with normal vision. Driving performance measures consisted of results from a driving simulator and an on-road driving test. Data also were obtained on crash and conviction rates (based on state- and self-report). Impaired simulator performance was noted for the age-related macular degeneration group as evidenced by delayed braking response times to stop signs, slower speeds, greater lane boundary crossings, and more simulator crashes. The age-related macular degeneration group also exhibited poorer overall on-road performance, with more points deducted for driving too slowly and for not maintaining proper lane position. A comparison of crash rates revealed a significantly higher rate of both state- and self-reported crashes for the control group than for the age-related macular degeneration group. However, differences in exposure may account for these findings. Finally, results of the study indicated that the age-related macular degeneration group compensated for their impairments in four ways: not driving in unfamiliar areas, traveling at slower speeds, self-restricting their night time driving, and taking fewer risks while driving (e.g., not changing lanes). Although often seen as a safety enhancing strategy, one of the compensatory strategies (e.g., driving at slower speeds) may in fact lead to crash involvement if the slower speed significantly differs from the speed of other motorists.

A review of the fitness-to-drive guidelines for medical practitioners from Australia (1998) and Canada (2000) reveals no recommendations for individuals with macular degeneration. Given the importance of central vision for driving, decisions about fitness-to-drive for individuals with macular degeneration should be determined on an individual level, with degree of central vision impairment a determining factor.

2.9 Nystagmus

Nystagmus is an involuntary, rapid, rhythmic movement of the eyeball. The rhythmic movements may be horizontal, vertical, rotary, or mixed. The types of nystagmus that occur before six months of age are called congenital or early onset, whereas those occurring after six months are labeled acquired nystagmus. Early onset nystagmus may be inherited, or the result of eye or visual pathway defects. In many cases, the cause is unknown (Royal Institute for the Blind, 2000). Causes of acquired nystagmus are many and may be a symptom of another condition such as stroke, multiple sclerosis, or even a blow to the head (Royal Institute for the Blind, 2000). Although the prevalence of nystagmus is not accurately known, the condition is believed to affect approximately 1 in 1,000 individuals (Royal National Institute for the Blind, 2000).

The majority of individuals with nystagmus have significant impairments in their vision, with many eligible to be registered as partially sighted or blind (Royal National Institute for the Blind, 2000). However, there is considerable variability in degree of visual impairment. Emotional and physical factors such as stress, tiredness, nervousness, or unfamiliar surroundings have been found to negatively affect visual functioning.

Given the considerable variability in visual impairment among individuals with nystagmus, decisions regarding fitness-to-drive should be determined on an individual basis.

2.10 Night Myopia

Night myopia is an increase in near-sightedness (ability to see near objects with blurring of distant vision) with declining levels of illumination. Night myopia is more common in younger individuals (Charman, 1997; Fejer and Girgis, 1992), possibly because of changes in accommodation in the youthful eye, changes that are absent after the age of about 50 (Charman, 1997). Results from Fejer and Girgis (1992) indicate that, of a sample of 380

randomly selected patients, 38 percent had night myopia of 0.75 dioptres, 23 percent had night myopia of 1.00 dioptres or more, and 4 percent night myopia of 2.50 dioptres (which is equivalent to an acuity of 20/265). Prescription of night myopic lenses is the treatment of choice for night myopia. However, results of trials with corrective lenses under night driving conditions are equivocal with some drivers reporting improved night vision and others reporting worsening of vision (Fejer, 1995; Owens and Leibowitz, 1976).

Because of the reported high prevalence of night myopia in younger drivers, it may be prudent to include questions regarding ability-to-see-at-night during licensing of younger drivers. Restrictions in nighttime driving may be warranted for those individuals without corrective lenses.

2.11 Post-Eye Surgery

Few studies have examined the relationship between the post-eye surgery period and driving competence. Results from Jude, Ryan, O'Leary, Gibson, and Dodson (1998) revealed a significant reduction in binocular visual acuity following pupillary dilation for fundoscopy in 61 diabetic drivers. As noted by the authors, studies are needed on the time course of the phenomena. In the meantime, Jude et al. conclude that patients should be warned not to drive for at least two hours following pupillary dilation for fundoscopic examination.

2.12 Visual Field Defects

The visual field is "the extent of visual space over which vision is possible with the eyes held in a fixed position" (Sekuler and Blake, 1985, p. 499). Visual field loss can result in significant functional impairments. However, studies examining the relationship between visual field loss and driving performance have been equivocal. In general, results from earlier studies (e.g., Burg, 1971; Council and Allen, 1974) failed to find an association between visual field loss and driving performance. More recent studies have, however, found significant relationships. As noted earlier, one of the most extensive investigations was conducted by Johnson and Keltner (1983). The authors examined the relationship between peripheral vision status and crash rates. Results from 8,767 volunteers (17,534 eyes) showed an incidence of visual field loss of 3 to 3.5 percent for people from the ages of 16 to 60, with a four-fold increase to 13 percent for those 65 years of age and older. Szlyk, Alexander, Severing, and Fishman (1992) compared the driving performance of 21 subjects with retinitis pigmentosa and varying degrees of peripheral field loss to that of 31 normally sighted control subjects. The two groups did not differ in terms of age, gender, years of driving experience, or miles driven per year. Individuals with retinitis pigmentosa had a significantly greater proportion of self-reported crashes and simulator crashes. Results of logistic regression revealed that binocular horizontal field extent and binocular field area differentiated between the no-crash and crash groups.

Recently, measures of the useful field of view (UFOV) have been found to be predictive of crash frequency. The UFOV is defined as "the total visual field area from which target characteristics can be acquired when eye and head movements are precluded" (Kline and Scialfa, 1997, p. 37). In essence, tests of the UFOV measure visual attention at the pre-attentive level. Considerable evidence exists to indicate that the UFOV is restricted in older individuals (Ball, Beard, Roenker, Miller, and Griggs, 1988; Sekuler and Ball, 1986). Ball and Owsley (1991) examined the performance of older drivers on a task measuring UFOV. Elderly individuals who failed the UFOV had 15.6 times more intersection crashes than those individuals who passed. Several studies since also have shown the UFOV measures to be related to crashes of older drivers (Ball, Owsley, Sloane, Roenker, and Bruni, 1993; Ball and Rebok, 1994). However, results from Brown, Greaney, Mitchell, and Lee (1993) did not find a significant relationship between UFOV and crash risk.

Table 1 Guidelines for Visual Conditions/Diseases (Reproduced with permission)

Guidelines for Visual Conditions/Diseases (Drivers of Private Vehicles)		
Conditions/ Diseases	**Austroads (1998)**	**CMA (2000)**
Acuity	Should not drive if binocular visual acuity is less than 6/12 (20/40). Visual acuity must be measured with both eyes open while wearing any corrective lenses usually worn for driving. More than one error in reading the letters of the 6/12 (20/40) line is a fail. Where a patient fails the test, eyesight must be corrected before the patient is fit to drive. Where corrective lenses are prescribed for the first time, the practitioner MUST advise the Driver Licensing Authority (DLA) which will endorse the license with the following: 'must wear corrective lenses when driving'.	Eye sight requirements: Not less than 20/50 (6/15) with both eyes open and examined together.
Aphakia	May drive if meets the acuity criteria. Specialist opinion recommended.	Not addressed.
Cataracts	Must meet the visual acuity criteria and other criteria and be aware that they may have difficulty with glare. Optometrist's or ophthalmologist's opinion recommended.	Listed as a medical condition that may require further assessment. If vision problem is suspected, the recommendation is for the individual to be referred to an ophthalmologist or optometrist for further assessment of visual function.
Color Vision Defects	No restriction. Patients with red color defects should be cautioned about hazardous situations - especially traffic lights, brake lights, and parked cars at night.	No required standard.
Conjunctivitis/ Other Anterior Eye Infections	Should not drive if severe and affecting eye comfort or vision.	Not addressed.
Contrast Sensitivity	Not addressed.	Individuals with reduced contrast sensitivity may experience difficulty with driving. However, it is unclear at this time what level of reduction in contrast sensitivity represents an unacceptable risk for driving. Individuals should be made aware of any significant reduction in contrast sensitivity.

Table 1 Guidelines for Visual Conditions/Diseases (*continued*)

Guidelines for Visual Conditions/Diseases (Drivers of Private Vehicles)		
Conditions/ Diseases	**Austroads (1998)**	**CMA (2000)**
Diplopia	Should not drive if has diplopia. The DLA may issue a conditional license if patch on eye and meets 'loss of vision in one eye' conditions.	Diplopia within the central 40 degrees (i.e., 20 degrees to the left, right, above, and below fixation) of primary gaze, is incompatible with safe driving. Individuals who have uncorrected diplopia within the central 40 degrees of gaze should be referred to an ophthalmologist or optometrist for further assessment. If the diplopia can be completely corrected with a patch or prisms to meet the appropriate standards for visual acuity and visual field, the individual may be eligible to drive. Prior to resuming driving, there should be an adequate adjustment period of 3 months or sufficient to satisfy the treating ophthalmologist or optometrist that adequate adjustment has occurred.
Glaucoma	May drive if meets the acuity criteria. Specialist opinion recommended.	Not addressed.
Monocular Vision	Should not drive for 3 months after loss of binocular vision. May then drive if vision in good eye is at least 6/12 (20/40). Should have mirrors on both sides of car or motorbike.	A driver who has recently lost the sight of an eye may require a few months to recover the ability to judge distance adequately.
Nystagmus	Should not drive if binocular visual acuity is worse than 6/12 (20/40).	Not addressed.
Poor Night Vision	Should not drive. The DLA may issue a conditional license for daylight driving. Specialist opinion recommended.	Currently there are no standardized tests or procedures that can be recommended for assessing dark adaptation and glare recovery.
Post Surgery	Should not drive for 4 weeks following surgery to the eye that will alter visual acuity of the eye unless cleared by an ophthalmologist.	Listed under medical conditions that may require further assessment for vision problems. If a vision problem is suspected, the recommendation is referral to an ophthalmologist or optometrist for further assessment of visual function.
Telescopic Spectacles	Not addressed.	The use of telescopic spectacle, hemianopia aids, and other low-vision aids is incompatible with safe driving.

Table 1 Guidelines for Visual Conditions/Diseases (*continued*)

Guidelines for Visual Conditions/Diseases (Drivers of Private Vehicles)		
Conditions/ Diseases	**Austroads (1998)**	**CMA (2000)**
Visual Field Defects	Hemianopia (total and partial): Should not drive if total hemianopia. A conditional license may be issued to a person with partial hemianopia or other defects provided on ophthalmologists or optometrist's report is obtained stating that the remaining visual field is no less that 120 degrees along the horizon meridian when measured with a Goldman IV4e target or its equivalent.	Recommended standard: 120 continuous degrees along the horizontal meridian and 15 continuous degrees above and below fixation with both eyes open and examined together. If a field defect is suspected, the patient should be referred to an ophthalmologist or optometrist for a full assessment. When a full assessment is required, the binocular visual field should be assessed using a III4e Goldman type target or the closest equivalent (e.g., the Esterman Functional Vision Test, the Humphrey Visual Field Analyzer, or kinetic perimetry on the Goldman perimeter) are recommended. Some automated testing devices used in driver testing centres are often insensitive to many types of visual field defects and thus may not be adequate for screening purposes.
Quadrantanopia	Should not drive. In the case of quadrantanopias, regardless of the extent of the remaining visual field, an ophthalmologist's report should be submitted to the DLA which may then consider a conditional license.	See Visual Field regulations.
Ptosis	Not addressed.	Not addressed.

DLA = Driver Licensing Authority

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Section 3: Hearing

Prevalence

According to recent statistics, more than 20 million people, or 8.6 percent of the total population in the United States 3 years of age or older, have hearing problems (National Center for Health Statistics, 1994). A similar rate is reported in Canada with the incidence of hearing loss in the Canadian population estimated to be 10 percent (Statistics Canada, 1996). As with visual deficits, impairments in hearing are strongly associated with age. The estimated prevalence of hearing impairments as a function of age is shown in Table 2.

Table 2 Estimate of the Prevalence of Hearing Impairments by Age Group, United States, 1990-1991
Source: National Center for Health Statistics, Data from the National Health Interview Survey, Series 10, Number 188, Table 1, 1994 (Reproduced with permission)

Age Group	Percent of Population
3-17 years	1.8
18-34 years	3.4
35-44 years	6.3
45-54 years	10.3
55-64 years	15.4
65 years and older	29.1
Total	8.6

As can be seen in Table 2, individuals 65 years of age and older are eight times more likely to have a hearing impairment than individuals 18-34 years of age. The prevalence of hearing impairment also differs as a function of gender. Males of all ages are more likely to have a hearing impairment (10.5 percent for males and 6.8 percent for females) and the gap widens after age 18 (National Center for Health Statistics, 1994). The gender differences in the prevalence of hearing impairment have, for the most part, been attributed to occupational differences in noise exposure. However, recent longitudinal research has revealed more rapid declines in hearing sensitivity in males not involved in occupations with high noise exposure (Pearson, Morrell, Gordon-Salant, et al., 1997).

Hearing and Driving Literature Review

Despite the importance of auditory information for driving (e.g., auditory feedback regarding operation of the motor vehicle, mechanical failure, awareness of other road users through detection of road noise, horn honking, etc.), there are few data to indicate that impairments in hearing affect driving ability. Results from an early study by Coppin and Peck (1963) indicated that deaf people, as a group, had poorer driving records than non-deaf people. However, more recent studies have failed to provide convincing evidence that individuals with hearing impairments are at a higher risk for motor vehicle crashes. In 1994, McCloskey, Koepsel, Wolf, and Buchner conducted a population-based case control study to determine whether sensory impairments place older drivers at risk for collision injuries. The cases were drivers who sought medical care, within 7 days, for injuries sustained in a police recorded motor vehicle crash. Controls were selected from a pool of eligible subjects who had not been injured in a police reported motor vehicle crash. Driving exposure, based on self-report, was similar for both groups. Sensory impairment data were extracted from medical records. Results of their investigation revealed no significant increase in risk of injury from motor vehicle collisions as a function of hearing impairment (See Table 3). However, those using hearing

Table 3 Risk of Injury from Motor Vehicle Crash Associated with Impaired Hearing (Re-produced in part, from McCloskey, L.W., Koepsell, T.D., Wolf, M.E., & Buchner, D.M. (1994). Motor vehicle collision injuries and sensory impairment of older drivers. Age and Ageing, 23, 267-273, by permission of Oxford University Press)

Condition	# with Valid Data		Percent with Condition		Risk Estimates	
	Cases	Controls	Cases	Controls	RR	CI
Hearing Impairment (ever diagnosed)	234	446	27.3	22.4	1.3	0.9-1.8
Hearing Aid:						
Prescribed	234	446	14.2	12.1	1.2	0.8-2.0
Owned	233	448	19.7	13.8	1.6	1.1-2.6
Owned and worn for driving*	215	423	13.0	8.7	1.9	1.1-3.3
Owned but not worn for driving*	204	409	8.3	5.6	1.7	0.8-3.6

* *versus non-owners*

aids were at increased risk of an injury collision. The authors speculated that feedback from the hearing aid while driving may create a distraction, placing the driver at an increased risk of crash involvement. However, an analysis was not conducted to determine who was at-fault.

Gresset and Myer (1994) conducted a case-control study investigating the relationship between impairments or chronic medical conditions and motor vehicle crashes. The sample consisted of 1,400 elderly male drivers (all aged 70) who had had a crash resulting in mild bodily injury or property damage between 1988 and 1989. Compared to same aged controls, cases with hearing impairments were not at increased risk for crashes (OR = 0.90, CI = 0.65-1.24). Importantly, male drivers involved in crashes causing death or causing severe bodily damage were excluded from the study. Exclusion of those cases may have led to an underestimation of the true relative risk of crashes, particularly for crashes associated with other medical impairments (e.g., cardio-vascular disease, diabetes mellitus).

More recently, Ivers, Mitchell, and Cumming (1999) examined the association between vision, hearing loss, and motor vehicle crashes in a cross sectional survey of 2,379 current drivers. Self-reports were used to assess hearing loss and motor vehicle crashes. Thirty-eight percent of the sample reported having hearing loss. Five point six (5.6) percent of individuals aged 49 to 79 reported being in a crash, with 9.1 percent of those 80 years of age and older reporting crash involvement. Moderate hearing loss (adjusted prevalence ratio PR = 1.9) and hearing loss in the right ear (PR = 1.8) were associated with an increased crash risk. Although not significant, those with severe hearing impairment also showed an increased risk for crash (PR = 1.5). It is important to note that indices of hearing loss and motor vehicle crashes used in this investigation were based on self-reports.

Conclusions

There are few studies that have examined the relationship between hearing impairment and risk of motor vehicle crash. Of those that are available, one study failed to find an association between hearing impairment and risk for injuries sustained in motor vehicle crashes (McCloskey et al., 1994). However, results of that investigation suggest that use of hearing aids by the hearing impaired while driving places the driver at an increased risk of motor vehicle crashes. Two studies have investigated the relationship between hearing impairment and risk of crash (Gresset and Myer, 1994; Ivers et al., 1999). Results from those investigations are mixed. Currently, therefore, there is little evidence to warrant driving restrictions for individuals with hearing impairments from operating private vehicles.

A summary of the current fitness-to-drive guidelines (Hearing Impairments) for medical practitioners from Australia (1998) and Canada (2000) is presented in Table 4.

Table 4 Guidelines for Hearing (Reproduced with permission)

Guidelines for Hearing (Drivers of Private Vehicles)		
Condition/lllness	**Austroads (1998)**	**CMA (2000)**
Totally Deaf	Not addressed.	No restriction.
Hearing Aids	Not addressed.	No restriction.
Some Hearing Loss	No restrictions. As greater reliance on vision is needed, external mirrors are required.	No restriction.
Vestibular Disorders	Acute labyrinthitis, Benign paroxysmal vertigo, Meniere's Disease, Recurrent Vertigo: Should not drive while symptoms persist.	Acute labyrinthitis Patients with acute labyrinthitis or positional vertigo with horizontal head movement should be advised not to drive at all until their condition has subsided or responded to treatment. Recurrent attacks of vertigo Patients who are subject to recurrent attacks of vertigo that occur without warning also should not drive until it is certain that their spells of dizziness have been controlled or abated.

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Section 4: Cardiovascular Diseases

4. Cardiovascular Diseases - Epidemiology

According to 1996 estimates, 58,800,000 Americans have one or more forms of cardiovascular disease (CVD): 50,000,000 are estimated to have high blood pressure, 12,000,000 coronary heart disease, 7,000,000 myocardial infarction, 6,200,000 angina pectoris, 4,400,000 stroke, and 1,800,00 rheumatic fever/rheumatic heart disease (American Heart Association, 1999).

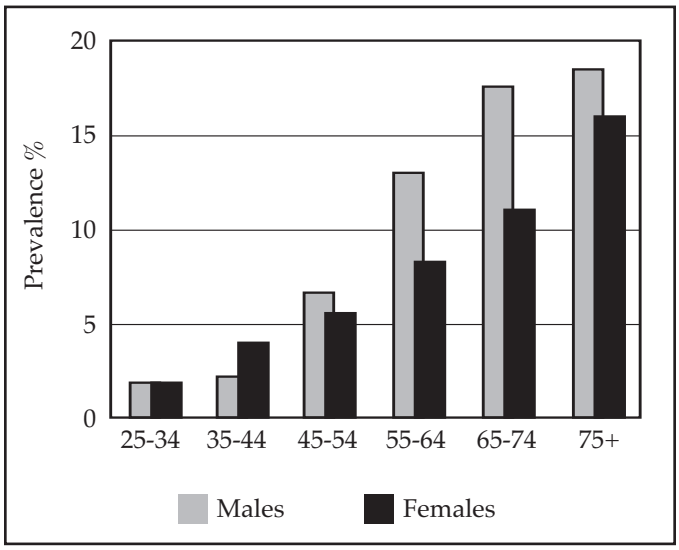
A summary of the current fitness-to-drive guidelines (Cardiovacular Diseases) for medical practitioners from Australia (1998) and Canada (2000) is presented in Table 9.

4.1 Coronary Heart/Artery Disease

4.1a. Epidemiology

Coronary heart disease (CHD) caused 1 of every 4.9 deaths in the United States in 1996 for a total of 476,124 deaths. Figure 1 shows the estimated prevalence in the United States of coronary artery disease by age and sex for the years 1988-1994 (National Health and Nutrition Examination Survey III [NHANES III], 1988-94, CDC/CHS and the American Heart Association).

Figure 1 Estimated prevalence of coronary artery disease by age and sex in the United States, 1988-1994.



Source: National Health and Nutrition Examination Survey III (NHANES III), 1988-94, CDC/CHS and the American Heart Association.

4.1b. Sudden Death at the Wheel

Crashes due to sudden death while driving represent one possible tragic outcome for individuals with coronary heart disease, and is a source of potential danger to other road users. A review of the literature suggests that sudden or natural death while driving due to cardiac and other illnesses is a rare event. Early reports suggest that 'sudden death while driving' is a causal agent in less than one percent of all crashes (Baker and Spitz, 1970; Grattan and Jeffcoate, 1968; Herner, Smedby, and Ysander, 1966; Peterson and Petty, 1962) and similar results are reported by Copeland (1987). In the Copeland study, case files from the Medical Examiner's office in Miami, Florida were examined from 1980-1984. Results reveal that less than one percent of all motor vehicle related accidental deaths were due to 'sudden natural death at the wheel'. More recently, Halinen and Jaussi (1994) investigated the incidence of fatal traffic crashes caused by sudden incapacity of the driver due to cardiac and other illnesses in Finland and Switzerland. Results, based on a retrospective analysis from traffic accident data files in Finland (1984-1989) and police records of crashes from Vaud, Switzerland (1986-1989), reveal that sudden driver incapacity caused 1.5 percent of all traffic deaths in Finland, and 3.4 percent in Vaud. According to the authors, the higher rate of traffic deaths in Vaud due to sudden driver incapacity is likely due to higher traffic densities in the county of Vaud, which place heavier demands on the driver, and the more advanced age of drivers in Switzerland compared to Finland.

Epilepsy and myocardial infarction (MI) are two of the most common causes of natural death while driving, with MI accounting for the majority of the deaths. In the Halinen and Jaussi (1994) study cited above, 68 percent of all driver deaths from the Finnish component of the study and 79 percent of all driver deaths from the Switzerland component were due to probable/possible cardiac arrest. These results are consistent with those from previous studies (See Table 5).

Table 5 Sudden Natural Death at the Wheel: Causes of Death *(Reproduced, in part, from Schmidt, P., Haarhoff, K., & Bonte, W. (1990). Sudden natural death at the wheel-a particular problem of the elderly. Forensic Science International 48, 155-162, with permission from Elsevier Science)*

Study	Sample Size	CVD (percent)	CAD (percent)
Tamaska (1961)	60	83.3	45
Peterson et al. (1962)	81	98.8	70.4
Gerber et al. (1966)	57	93	87
West et al. (1968)	155	94	86
Saternus et al. (1973)	91	88	—
Breitenecker (1976)	12	91.7	83.3
Krauland (1978)	433	91	83
Missliwetz et al. (1978)	76	92.1	85.5
Copeland (1987)	133	82	61.8
Ostrom et al. (1987)	126	96	88.9
Christina (1988)	64	87.5	76.8
Penttila et al. (1988)	60	—	75
Antecol and Roberts (1990)	20	—	80
Schmidt et al, (1990)	39	97	90

— = *Data not reported*
CVD = Cardiovascular Disease
CAD = Coronary Artery Disease

4.1c. Injury to Others

The data from early studies suggest that injury or death to others as a result of 'sudden death at the wheel' occurs rarely. For example, in studies by Peterson and Petty (1962) and Herner et al. (1966), no fatalities or serious injuries to others occurred as a result of a 'sudden death at the wheel' crash. Ostrom and Erikkson (1987) report that of the 31 'other persons at risk' in their study, only two suffered minor injuries. However, more recent studies suggest there may be cause for concern. In a study by Copeland (1987), 19 passengers were killed in the 133 'sudden death at the wheel' crashes reported in that study. More recent results from Halinen and Jaussi (1994) reveal somewhat higher figures. Eight passengers were killed in the 44 cases investigated in that study. It is important to note that the mean age of the drivers who died at the wheel has not changed between study periods. Thus, the mean age of the drivers in the early studies was mid to late 50's, an age range consistent with that found in later studies. Details regarding age of the passengers killed or speed of the vehicle prior to crash were not available. It may be that greater speeds were involved in the more recent crashes. The view that 'sudden death at the wheel' does not pose a serious risk to passengers or other road users may no longer be justified based on mortality rates from more recent studies. Future studies, with larger sample sizes, are clearly needed to determine if the patterns of morbidity and mortality will continue to escalate.

4.1d. Prodromal Symptoms

Although the data are limited, those that are available suggest that few individuals have premonitory symptoms before their crash. Results from Ostrom and Eriksson (1987) indicate that only 25 percent of the cases had prodromal symptoms prior to crashing, and 39 percent had not mentioned any symptoms before crashing. For the remaining individuals, the presence of prodromal symptoms was unknown. In the study by Halinen and Jaussi (1994), only two of the 44 cases showed a clear slowing of speed as indicated by signs of braking on the road surface, suggesting that prodromal symptoms were not present.

4.1e. Predictors of Sudden Death at the Wheel due to Coronary Heart Disease

i) Myocardial damage

Results from Copeland (1987) reveal that one third of the 'sudden death at the wheel' cases showed evidence of a healed infarction on autopsy. However, much higher figures have been reported in other studies. Sixty-seven percent of drivers dying suddenly in the study by Antecol and Roberts (1990) had healed myocardial infarcts (MI's). Similar results have been reported by Bowen (1973-56 percent), Hossack (1974-67 percent),

Ostrom and Erikkson (1987-56 percent), and Schmidt et al. (1990-66 percent). Results from Kerwin (1984) suggest that sudden cardiac death is seldom due to recent MIs. In that investigation, less than 20 percent of sudden cardiac deaths were due to recent infarction.

ii) Status of coronary arteries
A number of studies have examined the severity of the coronary artery disease (CAD) in victims of 'sudden death while driving'. The majority of the victims in the Copeland (1987) study had narrowing in one or more of their coronary arteries, and in 53 percent of the cases, luminal patency was less than 25 percent. Results from Antecol and Roberts (1990) reveal that in the 16 individuals who died suddenly from CAD, the mean number of coronary arteries narrowed greater than 75 percent was 2.3. Interestingly, none of the individuals in this study had a left main coronary artery narrowed more than 75 percent. Findings from Schmidt et al. (1990) indicate that in 33 out of the 35 cases of 'sudden death while driving', severe stenoses were evident (percent of narrowing is not reported). One quarter of the cases had one vessel disease, 25 percent had two-vessel disease, and approximately 50 percent of the cases had evidence of three-vessel disease.

iii) Age and gender
Results from research suggest that those most at risk for sudden death at the wheel due to cardiac involvement are older (mean age ~ 60) and male (Antecol and Roberts, 1990; Copeland, 1987; Ostrom and Eriksson, 1987; see also Schmidt et al., 1990 for a synopsis of earlier studies).

iv) Other risk factors
Left ventricular hypertrophy and intraventricular block have been shown to be positive predictors of sudden death in men with coronary heart disease (Schatzkin, Cupples, Heerne, Morelock, and Kannel, 1984). Thirty-two of the 35 (91 percent) sudden death cases while driving reported by Schmidt et al. (1990) had hypertrophied hearts.

v) Non-risk factors
Of those studies that investigated for the presence of alcohol, the majority of those studies report that alcohol was not a factor (Copeland, 1987; Hossack, 1980; Ostrom and Eriksson, 1987; Peterson and Petty, 1962).

vi) Conclusions
Crashes due to 'sudden death while driving' as a result of CAD are rare. Although statistics from early studies suggest that fatalities to passengers or other road users as a result of 'sudden death while driving' also are rare, more recent studies suggest there may be cause for concern. Nevertheless, given the rarity of 'sudden death while driving' crashes, the gains in traffic safety resulting from denial of licensing to those with CAD are likely to be minimal compared to the costs of loss of independence and mobility for this group of drivers.

However, driving a vehicle can be emotionally stressful. In individuals with CAD, the increased demands of driving could result in an increasing degree of myocardial ischemia. Results from Bellet, Roman, Kostis, and Slater (1968) provide support for this assumption. In that investigation, electrocardiogram (ECG) recordings were obtained from 65 young normal male subjects (25-29 years of age) and 66 individuals (primarily males ranging in age from 38 to 62) with clearly documented heart disease. The recordings were made during a 2-1/2 hour period of driving. No significant changes were observed in the normal subjects. Significant ECG changes were noted in 16.7 percent of the individuals with known heart disease. The ECG changes included ischemic type S-T depression, multifocal premature ventricular contractions (PVCs), and bigeminal and trigeminal rhythm. Importantly, the ECG changes occurred under relatively favorable driving conditions. It would seem prudent, therefore, for individuals with a known history of CAD, and particularly those who are older and male, to be advised as to the possible danger of 'sudden death while driving'. In addition, the presence of factors, such as three-vessel disease, 75 percent or greater narrowing of coronary arteries, and/or the presence of left ventricular hypertrophy are likely to place the individual at greater risk. Therefore, physicians should take these risk factors into consideration in determining whether an individual assessment is warranted.

4.1f. Current Licensing/Guideline Recommendations

Guidelines resulting from a Canadian Cardiovascular Society (CCS) Consensus Conference (1992; 1996), which are not binding on licensing entities or doctors in the United States or endorsed by NHTSA, recommend that all drivers with coronary heart disease should satisfy appropriate waiting periods, depending on their driving status (e.g., private versus commercial driving). Specific recommendations and waiting periods are provided based on: a) acute MI, unstable angina, b) stable angina, c) suspected asymptomatic CAD, d) coronary angioplasty, e) coronary bypass surgery and on the presence of left main coronary artery disease. In addition, the Canadian guidelines provide recommendations based on disturbances in cardiac rhythm, and the presence of other cardiac conditions (e.g., valvular heart disease, congestive heart failure, hypertrophic cardiomyopathy, congenital heart disease, and cardiac transplantations). The reader is referred to the CCS Consensus Conference (1992; 1996) guidelines for further information.

4.2 Disturbances of Cardiac Rhythm

Individuals with arrhythmia that induce symptoms of weakness, light headedness, and loss of consciousness may place themselves and others at risk if these symptoms occur while the individual is driving.

Estimating Risk of Harm

The CCS Consensus Conference (1996) presents an equation for mathematically estimating the risk of harm to other road users and/or innocent bystanders. The equation for risk of harm (RH) is:

RH = (TD) (V) (SCI) (Ac)

with:

TD = proportion of time individual spends driving during the year (estimated at four percent for private drivers and 25 percent for commercial drivers)

V = constant based on the type of vehicle driven (calculated to be 1.0 for a commercial vehicle, and 0.28 for a passenger car)

SCI = risk of sudden death or incapacity during the year (estimate may be provided by physician)

Ac = represents probability of death or injury to other road users

The following example, provided by the CCS Consensus Conference (1996), illustrates the calculation of risk of harm to other road users of a commercial driver returning to that occupation following an acute myocardial infarct:

> It may be assumed that the average commercial driver spends 25 percent of his/her time behind the wheel. Thus, in the formula above, TD = 0.25. V is assigned a value of 1 (commercial driver). Based on currently available data, an individual with an acute MI, who is a functional Class I with a negative exercise test at 7 METs, has no disqualifying arrhythmias on ambulatory ECG monitoring, and is at least three months post-infarct, would be assigned a risk of one (1). Available data, although limited, suggest that sudden cardiac incapacitation at the wheel poses a very small risk to public safety. Calculations, according to the CCS Consensus Conference (1996), estimate the probability of death or injury to others at 0.02. Thus, substituting in the formula above,
>
> **RH = (TD) (V) (SCI) (Ac)**
> **= (0.25) (1.0) (0.01) (0.02)**
> **= 0.00005**
>
> Allowing a driver with the described condition on the road is associated with a risk of death or injury to others of ~ 1 in 20,0000 (0.0005 = 1/20,000). According to the authors, this level of risk appears to generally be acceptable in Canada.
> (p. 412, CCS Consensus Conference, 1996)

Research on Arrhythmias

The research on ventricular fibrillation (VF), ventricular tachycardia (VT), supraventricular arrhythmias, and heart block is reviewed below. The literature on implantable cardioverter/ defibrillator devices [ICDs] and pacemakers also is reviewed.

Primary issues that need to be considered to help assess risk of death and injury to drivers and other road users as a result of disturbances of cardiac rhythm are:

1. Frequency and time course of arrhythmia
2. The likelihood of the arrhythmia resulting in loss of consciousness (LOC)
3. The risk of the event resulting in a crash
4. The risk to other road users
5. Predictors of arrhythmia

(Adapted from The Working Groups on Cardiac Pacing and Arrhythmia of the European Society of Cardiology; Jung, Anderson, Camm, et al., 1997).

4.2a. Ventricular Arrhythmias (Ventricular Fibrillation and Tachycardia)

Ventricular arrhythmias are the most common cause of sudden cardiac death, with ventricular fibrillation causing more than 300,000 sudden deaths each year in the United States alone (Chen, Kirsch, Zhang, et al., 1998). Variables known to be associated with the occurrence of VT/VF include older age, systemic hypertension, previous myocardial infarct, anterior infarct, and depressed ejection fraction (Newby, Thompson, Stebbins, Toepol, Califf, and Natale, 1998). For example, sustained VT and VF occur in up to 20 percent of patients with acute MI (Newby et al., 1998).

Primary issues for driving safety in individuals who have survived an episode of VT or VF are:

1. The likelihood of the arrhythmia recurring
2. The likelihood that the arrhythmia will result in loss of consciousness
3. The risk that the arrhythmia event will lead to a crash
4. The risk of harm to other road users

Primary Issues

1. The recurrence of an arrhythmia

Older studies reveal that the survivors of sudden death caused by VT or VF faced subsequent one and two year recurrence rates of 30 percent and 40 percent, respectively (Baum, Alvarez, and Cobb, 1974; Liberthson, Nagel, Hirschman, and Nussenfeld, 1974; Myerburg, Kessler, Zaman, Conde, and Castellanos, 1982). However, with the advent of ambulatory ECG monitoring, programmed electrical stimulation to guide anti-arrhythmic drug therapy, and the use of implantable cardioverter defibrillator devices, sudden death rates due to recurrent ventricular arrhythmias have declined significantly (Myerburg, Kessler, Zaman, et al., 1987).

Although the data are limited, those that are available suggest that the risk of recurrence of a ventricular arrhythmia is time-dependent with the highest risk occurring in the first 6 to 18 months following discharge from hospital following a first event (Furukawa, Rozanski, Nogami, Moroe, Gosselin, and Lister, 1989; Myerburg, Kessler, and Castellanos, 1992). To determine when survivors of VT and VF might safely return to driving, Larsen, Stupey, Walance, et al. (1994) followed 501 patients for 0 to 117 months (M = 26 months). Outcome events that could impair driving ability were analyzed. Those events included syncope, sudden death, recurrent VT, recurrent hemodynamically compromising VT, and ICD discharge. Results of that investigation revealed that the one-year outcome event rate for all patients was 17 percent. Importantly, three distinct periods of risk were identified. The monthly hazard rate was highest in the first month following hospital discharge (4.22 percent per month), intermediate in months 2 through 7 (1.81 percent per month), and lowest in months 8 through 12 (0.63 percent per month).

2. Likelihood that such episodes will result in loss of consciousness

-See literature on 'sudden death at the wheel' (Section 4.1b.).

3. The risk that the arrhythmia event will lead to a crash

-See literature on 'sudden death at the wheel' (Section 4.1b.)

4. The risk of harm to other road users

-See literature on injury to others (Section 4.1c.)

Recommendations from the CCS Consensus Conference (1996) for private drivers with VF or sustained VT are:

1. A waiting period of three months for individuals: a) with VT/VF non-inducible by electrophysiologic studies (EPS), with or without ICD, b) on EPS-predicted effective drug therapy, with or without ICD.
2. A waiting period of six months for individuals: a) on Holter-predicted effective drug therapy, with or without ICD, b) on empiric therapy with amiodarone, with or without ICD, c) on empiric therapy with other antiarrhythmic drugs, with ICD.
3. A waiting period of 12 months for individuals on empiric therapy with other antiarrhythmic drugs, without ICD.

4.2b. Atrial Fibrillation/Flutter

Atrial fibrillation (AF), one of the most common of the serious cardiac arrhythmias, is associated with substantial morbidity and mortality in the general population (Kannel, Abbott, Savage, and McNamus, 1983). The incidence and prevalence of AF increase with age. Based on data from the Framingham Heart study (cited in Kannel, Wolf, Benjamin, and Levy, 1998), the prevalence of AF doubles with each decade of age in those 50 and older, with an estimated prevalence of approximately 10 percent for those 80 years and older. The incidence of AF also doubles with each decade of age beyond age 50. Data from the Framingham Heart study (cited in Kannel et al., 1998) indicate that 10 percent of individuals 80 years and older acquire this rhythm disturbance. Data also indicate that the incidence of AF is greater for men than for women. After adjusting for age and other risk factors, the data from the Framingham Heart Study indicate that men were 50 percent more likely than women to develop AF (Kannel et al., 1998). A number of studies indicate individuals with AF are at increased risk for cardiac morbidity and mortality (Britton and Gustafson, 1985; Gajewski and Singer, 1981; Kannel, Abbott, Savage, and McNamara, 1982). Other risk factors for the development of AF are diabetes, left ventricular hypertrophy, coronary artery disease, valvular heart disease, heart failure, and stroke (Feinberg, Blackshearm, Laupacis, Kronmal, and Hart, 1995).

The most common symptoms associated with AF are palpitations, fatigue, dyspnea, and dizziness. Thus, the presence of AF has the potential to affect driving performance because of its hemodynamic consequences (e.g., cerebral ischemia). However, there are no data available on the effects of AF on driving performance. Because the presence of AF is strongly associated with

an increased risk for stroke, the most likely effects of AF on driving performance will be in terms of this complication. Epidemiological and clinical studies indicate that the presence of AF is associated with a four- to five-fold increased risk for stroke, after adjusting for other factors (Kannel et al., 1998). For the effects of stroke on driving performance, see Section 5 (Cerebrovascular Diseases).

Recommendations from the CCS Consensus Conference (1996) for private drivers with chronic AF or atrial flutter are:

1. No restriction for private and commercial drivers with no underlying heart disease and no associated cerebral ischemia.
2. No restriction for private drivers with underlying heart disease and no associated cerebral ischemia.

4.2c. Heart Block

Syncope is the primary symptom that places the driver with heart block at-risk. The syncopal symptoms are due to bradyarrhythmias secondary to structural conduction system disease. Cardiac pacing is highly effective for individuals with heart block (Miles, 1997).

Recommendations from the CCS Consensus Conference (1996) for private drivers with heart block are:

1. No restriction for private drivers with:
   a) Isolated first-degree AV block
   b) Isolated right-bundle branch block
   c) Isolated left-anterior fascicular block
   d) Isolated left-posterior fascicular block

2. No restriction for private drivers with no associated cerebral ischemia and:
   a) Left bundle branch block
   b) Bifascicular block
   c) Mobitz Type I AV block
   d) First-degree AV block and bifascicular block

3. Disqualification for private drivers with:
   a) Mobitz Type II AV block
   b) Trifascicular block
   c) First degree AV block and bifascicular block

4. No restriction for congenital third degree AV block for private drivers with
   a) no associated cerebral ischemia

4.2d. Pacemakers

Based on guidelines from the American College of Cardiology-American Heart Association task force (Dreifus, Fisch, Griffin, Gillette, Mason, and Parsonnet, 1991), indications for pacemakers are:

a) Complete Atrioventricular (AV) Block (acquired, surgical, or congenital)
b) Second degree AV block
c) Sick Sinus Syndrome
d) Carotid Sinus Hypersensitivity
e) Hypertrophic Cardiomyopathy

The use of pacemakers has grown since they were first introduced in 1958. In the United States, the number of pacemakers per million habitants has increased from 200 in 1985 to more than 400 in 1994 (Bjerregaard, 1997). Approximately 90 percent of all pacemakers are implanted because of either sinus node dysfunction or AV block, with equal frequency between the two abnormalities (Bjerregaard, 1997). Syncope is the most common symptom prior to pacemaker implantation, and is seen in 40 percent of individuals. The second most frequent symptom is dizziness (25 percent) followed by symptomatic bradycardia (e.g., fatigue, lassitude, weakness, visual disturbances) in 20 percent of individuals (Bjerregaard, 1997).

The risk of syncope is essentially eliminated with cardiac pacing. Abrupt pacemaker system failure is rare (Bjerregaard, 1997; Miles, 1997), although problems with pacing leads have been reported (Bjerregaard, 1997).

Recommendations from the CCS Consensus Conference (1996) for private drivers with artificial cardiac pacemakers are:

1. For all individuals (private and commercial drivers):
   a) A waiting period of one week
   b) No cerebral ischemia
   c) Normal sensing and capture on ECG
   d) Device performing within manufacturer's specifications

4.2e. Implantable Cardioverter/Defibrillator Devices (ICDs)

ICDs are used in the management of individuals with recurrent VT of VF that cannot be controlled with antiarrhythmic medications. ICDs treat symptoms of cardiac disease (e.g., VT/VF) that are notable because of their 1) unpredictability, 2) suddenness of onset, and 3) potential for rapid incapacitation of the individual.

The Working Groups on Cardiac Pacing and Arrhythmias of the European Society of Cardiology (Jung et al., 1997) suggest that the following issues be considered to help assess risk of death and injury as a result of ICDs:

i) Risk of recurrence

Data derived from individuals who have had cardioverter-defibrillator devices implanted to manage their ventricular tachyarrhythmias suggest that the risk of recurrence of a ventricular arrhythmia is highest in the first few months following discharge. Using those data, the recurrence of arrhythmias can be determined by examining delivery of appropriate shocks by the ICD. In the older models, "appropriate shocks are usually defined as shocks delivered during sustained VT or VF documented by ECG recordings or shocks delivered during a period of syncope or presyncope that resulted in restoration of consciousness" (Miles, 1997, p. 328). Newer devices provide telemetered electrograms during arrhythmia. Results from studies using telemetered electrograms suggest that appropriate shocks (i.e., shocks delivered for VT/VF) may occur in the absence of premonitory symptoms. Thus, the strict criteria of defining appropriate shocks based on significant warning symptoms may underestimate the true number of appropriate shocks. Other studies indicate that up to 47 percent of VT episodes may be preceded by no symptoms (Maloney, Masterson, Khoury, et al., 1991; Marchlinksi, Buxton, and Flores, 1990; Steinberg and Sugalski, 1991). Data from the newer devices, therefore, may represent a more accurate assessment of the recurrence of VT/VF compared to data from the older models.

Results from a number of studies (see Table 6) reveal that approximately 50 percent of patients will experience an appropriate shock during several years of follow-up (Fogoros, Elson, and Bonnet, 1989; Grimm, Flores, and Marchlinski 1993; Levine, Mellitis, Baumgardner, et al., 1991; Tchou, Axtell, Anderson, et al., 1991). Fogoros et al. investigated the pattern of occurrence of shocks in individuals following implantation of an ICD from one month to 71 months. The actuarial incidence of any shocks in this investigation was 43 percent at six months, with an 81 percent cumulative incidence of shocks at 48 months. The actuarial incidence of appropriate shocks during the same time periods was 28 percent and 64 percent, respectively. Appropriate shocks were defined as shocks that were preceded by symptoms of severe lightheadedness, presyncope, or syncope, and which were followed by immediate relief of those symptoms or documented VT or VF. Actuarial incidence rates of appropriate shocks reported by Grimm et al. based on the same criteria as used by Fogoros et al. were 13 percent, 42 percent, and 63 percent at 1, 3, and 5 years of follow-up, respectively.

ii) The likelihood that VT/VF episodes will result in loss of consciousness

The likelihood that episodes of ventricular arrhythmias will result in loss of consciousness is of primary consideration when assessing the driving risks of patients with ventricular tachyarrhythmias. Although the data

Table 6 Recurrence of Ventricular Arrhythmias in Individuals with Implantable Cardioverter Defibrillator Devices (ICDs)

Study	n	Follow-up (Months)	Percent Shocks	Definition of Shock
Fogoros et al. (1989)	65	25 ± 21	57	ICD
Gross et al. (1991)	1,281	0 - 60	37	S/R
Kou et al. (1991)	180	16 ± 12	59	S
Levine et al. (1991)	197	0 - 27.8	53	S/R
Maloney et al. (1991)	105	13 ± 8	44	E
Tchou et al. (1991)	184	24 ± 18.7	37	S/R
Grimm et al. (1993)	241	26 ± 22	43	S/R
Hook et al. (1993)	48	15.1 ± 7.8	60	E (P/S)
Freedberg et al. (1995)	145	18.3 ± 11.7	30	ICD
Ruppel et al. (1998)	40	23 ± 11	57	E

ICD = ICD discharge
S/R = Discharge accompanied by hypotensive symptoms or recorded arrhythmia
S = Discharge accompanied by hypotensive symptoms
E = Electrogram recordings
E(P/S) = Electrogram recordings (pacing or shock)

are limited, those that are available suggest that a significant number of individuals experience lightheadedness or syncope prior to receiving a shock, and a number of patients experience loss of consciousness prior to receiving a shock (See Table 7). Importantly, the absence of syncope during one episode does not always predict absence of syncope during subsequent shocks.

The findings from studies examining predictors of risk of syncope prior to receiving an ICD shock are mixed. For example, age, antiarrhythmic therapy, and left ventricular ejection fraction (LVEF) failed to distinguish between syncope and non-syncope groups in studies by Axtell and Akhatr (1990), and Kou, Calkins, Lewis, et al. (1991). However, individuals with a LVEF of < 40 percent had a significantly higher risk of syncope than patients with a LVEF > 40 percent in a study by Bansch, Brunn, Castrucci, Weber, Gietzen, Borggrefe, Breithardt, and Block, (1998). Using Cox regression, a one percent increase in LVEF implied a decrease of syncope by two percent. Individuals with an inducible fast VT during programmed ventricular stimulation showed a 2.2 fold increase in risk. Other predictors of risk of syncope included chronic atrial fibrillation (3.6 fold increase in risk). In addition, once patients had a recurrence of their VT/VF, syncope during the first VT and a high VT rate were the strongest predictors of future syncope. Schoels, Sarason, Beyer, and Brachmann (1995) also report that patients with lower LVEF are more at risk for syncope than individuals with a higher LVEF. However, as noted by the authors, the predictive value of LVEF was low because of an overlap between LVEF in patients with and without syncope.

iii. The risk that such an event will cause a crash
There are few data on the risk of crashes in individuals with ICDs. Results from Bansch et al. (1998) reveal that 14.7 percent of the patients in that study had syncope following ICD implantation (n = 421). Most of the episodes of syncope occurred while patients were at rest (43.6 percent). Of those experiencing syncope, one syncopal episode occurred while the patient was driving, with a crash prevented by the front seat passenger.

iv. The probability that such a crash will result in harm to other road users, bystanders, or passengers
Based on the formula provided by the CCS Consensus Conference (1996), Jung et al. (1997) have estimated that allowing an individual with an ICD to operate a private motor vehicle to be associated with an annual risk of harm of individuals to other road users or harm to bystanders or passengers to be 1 in 45,000.

Table 7 Percentage of Patients Experiencing Pre-Syncope or LOC Prior to Receiving an ICD Shock for Recurrent Ventricular Arrhythmias

Study	n	Follow-up (Months)	Percent Receiving Shocks	Percent Experiencing Lightheadedness or Syncope	Percent Experiencing LOC Prior to Shock
Fogoros et al. (1989)	65	25± 21	57%	49%	17%
Axtell & Akhtar (1990)	184	—	39%	79%	21%
Kou et al. (1991)	180	16±12	59%	—	15%
Maloney et al. (1991)	105	13±8	44%	VF = 100% VTs = 53% VTns = 65%	—
Grimm et al. (1993)	241	26±22	43%	Symptoms none (30%) mild (49%) severe (40%)	—
Bansch et al. (1998)	421	26±18	54.4%	—	14.7%
Ruppel et al. (1998)	40	23±11	33%	VT = 36%	VF = 100% VT = 3 %

— = *No data reported*
VTs = *Sustained Ventricular Tachycardia*
VTns = *Non-Sustained Ventricular Tachycardia*
LOC = *Loss of consciousness*

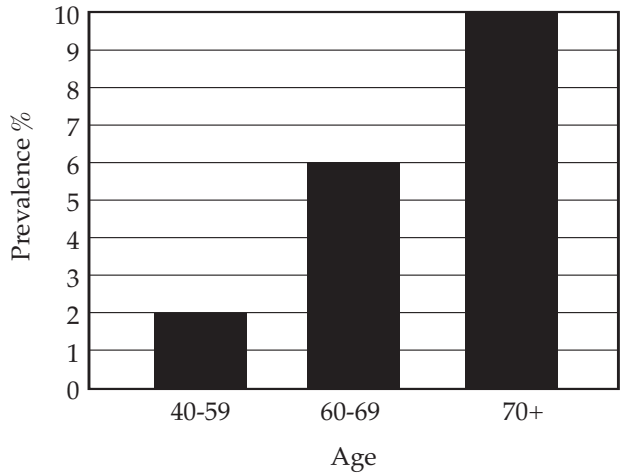
This estimate is based on the following calculations:

RH = TD x V X SCI x Ac
= 0.04 x 0.28 x 0.1 x 0.02
= 0.0000224 or 1 in 45,0000
(see Jung et al., 1997, p. 50 for a full description).

4.3 Congestive Heart Failure

Estimates suggest that approximately 4.8 million individuals in the United States have congestive heart failure (CHF), with 400,000 new cases each year (National Institute of Health, 2000 based on National Health & Nutritional Examination Survey, 1988-1991). The prevalence of CHF increases with age. As can be seen in Figure 2, the prevalence of CHF is five times greater in individuals 70 years of age or older compared to those aged 40-59 (National Health & Nutritional Examination Survey, 1988-1991).

Figure 2 Prevalence of CHF by Age.
Source: National Health and Nutrition Examination Survey (1998-1991). National Center for Health Statistics.



Importantly, the prevalence of CHF is increasing. Data from the National Health & Nutritional Examination Surveys in 1976-80 and 1988-91 indicate that the prevalence rates increased for every age group (35 through 75) between the 1976-80 and 1988-1991 surveys.

In a recent study, Senni, Tribouilloy, Rodeheffer, et al. (1999) compared the incidence of CHF in Rochester, Minnesota in 1981 with that observed in 1991. Results of that investigation revealed no significant differences in the incidence, after adjustment for sex and age, and survival rates between the two cohorts. Thus, these data suggest that recent advances in the management of cardiovascular disease have done little to affect the incidence or survival of individuals with CHF between 1981 and 1991.

Common symptoms of CHF include shortness of breath, fatigue, and exercise intolerance. A decline in mental status is a common manifestation in CHF, an effect that may adversely affect driving performance. Recently, Cacciatore, Abete, Ferrara, et al. (1998) investigated the relationship between CHF and cognitive impairment in an older population. Results of that investigation revealed a prevalence of CHF in subjects with cognitive impairment (e.g., Mini Mental State Examination [MMSE] below 24) of 20 percent compared to a prevalence of 4.6 percent in individuals with CHF with a MMSE > 24. Zuccala, Cattel, Manes-Gravina, et al. (1997) also report a positive relationship between CHF and cognitive impairment. Results from that study revealed significant correlations between left ventricular ejection fractions and MMSE scores, with ejection fractions < 30 percent associated with lower MMSE scores. Acanfora, Trojano, Iannuzzi, et al. (1996) report the results of a multicenter study investigating cognitive impairment and CHF. Results from preliminary data from 183 individuals with a diagnosis of CHF revealed significant differences in cognitive functioning between patients with CHF compared to those without CHF. Significant impairments were noted on the MMSE, verbal fluency, immediate and delayed recall (Rey test), and attentional measures. Importantly, MMSE scores for individuals without CHF were, on average, 24 (± 5), suggesting that the level of impairment for those with CHF may be even more severe when compared to unimpaired healthy controls.

CHF and Driving Literature Review

The effects of CHF on driving performance are, unfortunately, largely unknown due to a paucity of research in the area. Fitness-to-drive guidelines for individuals with CHF have, however, been published by the Canadian Cardiovascular Society (CCS). The CCS fitness-to-drive guidelines for individuals with CHF are summarized below.

Recommendations from the CCS Consensus Conference (1996) for private drivers with CHF:

a) No restrictions for private drivers that are
Functional Class I-No functional limitations,
(Able to achieve 5-7 METS [1 MET equivalent to resting oxygen consumption in the seated position and equivalent to 3.5 ml/kg/min])
or
Functional Class II-Mild functional limitations
(Able to achieve 5-7 METS),
or
Functional Class III LV Class I-Ejection fraction 50 percent or more,
or

LV Class II-Ejection fraction 35 percent to 49 percent and Holter class II (No episodes of VT more than 3 beats in duration with an average cycle length 500 ms or less).

4.4 Abnormal Blood Pressure

4.4a. Hypertension

Hypertension is defined as systolic blood pressure of 140 mm Hg or greater, diastolic blood pressure of 90 mm Hg or greater, or taking antihypertensive medication (National Institutes of Health [NIH], 1997). Blood pressure classification criteria for individuals 18 years of age and older who are not taking antihypertensive medication and who do not have an acute illness have been provided by the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure (NIH, 1997). Those criteria are outlined in Table 8.

According to the American Heart Association (2000), one in four American adults have high blood pressure. Of those with high blood pressure, 14.8 percent are not on therapy (special diet or drugs), 26.2 percent are on inadequate therapy, and 27.4 percent are on adequate therapy. Individuals with lower educational and income levels that are obese and are physically inactive, have diabetes, who smoke, and/or have a family history of hypertension are at higher risk of developing hypertension. Also at increased risk are non-Hispanic blacks and Mexican Americans. Risk for hypertension also increases with age (Cressman and Gifford, 1990). Complications of hypertension include increased risk for stroke, coronary artery disease, myocardial infarctions, and kidney disease.

Table 8 Blood Pressure Classification Criteria for Individuals 18 years of Age and Older*
(Reproduced, with permission, from *The National Institutes of Health, Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure November, 1997)*

Category	Systolic (mm Hg)		Diastolic (mm Hg)
Optimal †	< 120	and	<80
Normal	<130	and	<85
High-Normal	130-139	or	85-89
Hypertension ‡			
Stage 1	140-159	or	90-99
Stage 2	160-179	or	100-109
Stage 3	>180	or	>110

** Not taking antihypertensive drugs and not acutely ill. When systolic (SBP) and diastolic blood pressures (DBP) fall into different categories, the higher category should be selected to classify the individual's blood pressure status. For example, 160/92 mm Hg should be classified as stage 2 hypertension, and 174/120 mm Hg should be classified as stage 3 hypertension. Isolated systolic hypertension is defined as SBP of 140 mm Hg or greater and DBP below 90 mm Hg and staged appropriately (e.g., 170/82 mm Hg is defined as stage 2 isolated systolic hypertension). In addition to classifying stages of hypertension on the basis of average blood pressure levels, clinicians should specify presence or absence of target organ disease and additional risk factors. This specificity is important for risk classification and treatment.*

† Optimal blood pressure with respect to cardiovascular risk is below 120/80 mm Hg. However, unusually low readings should be evaluated for clinical significance.

‡ Based on the average of two or more readings taken at each of two or more visits after an initial screening.

Hypertension and Driving Literature Review

As with many chronic medical conditions, there is a paucity of literature on the relationship between hypertension and motor vehicle crashes. For individuals with hypertension who experience complications from the disease, their risk for motor vehicle crashes would be related to the presence and/or severity of the disease (e.g., cerebrovascular accidents [CVA's], myocardial infarcts). The reader is directed to those sections of the review that are applicable (e.g., Cardiovascular, Cerebrovascular, Renal Diseases). In one study, Schmidt, Frerick, Kraft, Schenk, and Löw-Kröger (1992) examined cognitive and on-road performance of 20 hypertensive individuals pre-treatment (Mean systolic BP = 149 ± 15.8 mm Hg; diastolic BP = 101.4 ± 6.6), following antihypertensive medication (Mean systolic BP = 132.5 ± 13 mm Hg; diastolic BP = 91.7 ± 8.2), and 15 normotensive controls (Mean systolic BP = 127.7 ± 20.8 mm Hg; diastolic BP = 84.3 ± 8.3). Prior to treatment, there were significant differences between the hypertensives and controls on tests of attention and concentration. Untreated hypertensives made fewer correct responses and had an error rate double that of controls. Following treatment (with moxonidine), however, the hypertensive patients performed at a level comparable to the controls. Unfortunately, measures from the road test included only assessments such as lane positioning, maintenance of speed, and maintaining a safe distance.

There is a growing body of literature documenting the relationship between hypertension and cognitive impairment (see Waldstein, 1995 for an excellent review). As noted by Waldstein, hypertensive individuals of all ages generally show a consistent pattern of impairments

on tests of learning and memory, attention and mental flexibility, and abstract reasoning. Less consistent results have been reported on tests of visuospatial, visuoconstructive, psychomotor, and perceptual functioning (cf. Waldstein, 1995).

Hypertension is one of the primary risk factors for vascular dementia, the second leading cause of dementia in the elderly person (Marshall, 1993). Multiple infarct and ischemic white matter lesions are suggested causes of vascular dementia, both of which have been associated with hypertension (Strandgaard and Paulson, 1994). It is interesting to note that results from a 15-year longitudinal study in Goteborg, Sweden revealed that higher systolic blood pressure at age 70, and higher diastolic blood pressure at ages 70 and 75 predicted the presence of a dementia at age 79-85 (Skoog, Lernfelt, Palmertz, et al., 1996).

In summary, there is a large body of literature documenting the relationship between hypertension and impaired cognitive performance. Although there are no data available linking the presence of high blood pressure to an increased risk of motor vehicle crashes, it seems prudent, based on the available literature between hypertension and cognitive impairment, for licensing agencies to be alerted to the role that hypertension may play in motor vehicle crashes.

Consideration also must be given to the effects of antihypertensive medication on cognitive performance. For example, Larson, Kruskull, Buchner, and Reifler (1987) investigated the effects of medication on cognitive performance. Results of their investigation led the authors to conclude that antihypertensive agents are major culprits of cognitive deficits.

4.4b. Hypotension

Hypotension, or low blood pressure, is less common than hypertension. Hypotension may be caused by a number of factors: antihypertensive medications, pregnancy, diabetes, and endocrine disorders such as low thyroid or low adrenal gland functioning.

Hypotension and Driving Literature Review

Syncope (sudden and temporary loss of consciousness) is the major risk factor associated with hypotensive drivers. A review of the risks of syncope while driving is included in the review of the literature on sudden death at the wheel (Section 4.1b.). The major issue for individuals with hypotension who experience episodes of syncope is the identification and treatment of the cause of the condition.

Table 9 Guidelines for Cardiovascular Diseases (Reproduced with permission)

Guidelines for Cardiovascular Diseases (Drivers of Private Vehicles)		
Condition/Illness	**Austroads (1998)**	**CMA (2000)**
A. Coronary Artery Disease/Coronary Heart Disease		
1. Acute Myocardial Infarct (MI)	Should not drive for 2 weeks post uncomplicated acute MI. More than one acute MI needs cardiologist appraisal.	Waiting period of 1 month (post MI).
2. Stable Angina Pectoris	May drive if angina stable.	No additional restrictions. No waiting period.
3. Suspected Asymptomatic Coronary Artery Disease		No additional restrictions. No waiting period.
4. Percutaneous Transluminal Coronary Interventions for Revascularization (e.g., angioplasty, stenting, atherectomy)	Angioplasty Should not drive 1 week post angioplasty if stable.	Waiting period 48 hours.
5. Coronary Bypass Surgery	Should not drive for 4 weeks post coronary artery grafts. Specialist opinion recommended.	Waiting period one month.

Table 9 Guidelines for Cardiovascular Diseases (*continued*)

B. Disturbances of Cardiac Rhythm		
1. Ventricular Fibrillation (VF) and/or Sustained Ventricular Tachycardia (VT)		The following conditions apply with or without ICD: - Waiting period 3 months if: VT/VF non-inducible by EPS. On EPS predicted effective drug therapy. - Waiting period 6 months if: On Holter-predicted effective drug therapy. On empiric therapy with amio darone. On empiric therapy with other anti-arrhythmic drugs (with ICD). On empiric therapy with other anti-arrhythmic drugs (without ICD).
2. Nonsustained Paroxysmal VT, Paroxysmal Supraventricular Tachycardia, Paroxysmal Atrial Fibrillation/Flutter	<u>Paroxysmal Arrhythmia</u> Should not drive. DLA usually allows a conditional license on medical advice that patient has been symptom free for at least 3 months. Those with third degree heart block should not drive until a pacemaker is inserted - specialist opinion and annual assessment required. <u>Atrial Fibrillation/Flutter</u> Should not drive after acute episode which causes dizziness or syncope until condition is stabilized.	No restriction if: No associated cerebral ischemia and no underlying heart disease. With ventricular pre-excitation and no associated cerebral ischemia. Satisfactory control with associated with cerebral ischemia. Satisfactory control with underlying heart disease.
3. Chronic Atrial Fibrillation		No restriction if: No associated cerebral ischemia and no underlying heart disease. With underlying heart disease and no associated cerebral ischemia.
4. Sinus Node Dysfunction (Sick Sinus Syndrome, Sinus Bradycardia, Sinus Exit Block, Sinus Arrest)	See AV Block below.	No restriction if: No associated cerebral ischemia.

Table 9 Guidelines for Cardiovascular Diseases (*continued*)

5. Atrioventricular Block (AV) and Intraventricular Block	Those with third degree heart block should not drive until pacemaker inserted-specialist opinion and annual assessment required.	No restriction if: Isolated first degree block, Isolated right bundle branch block, Isolated left anterior fasicular block, Isolated left posterior fasicular block. No restriction if no associated cerebral ischemia with: Left bundle branch block, Bifasicular block, Mobitz Type 1 AV block + Bifasicular block. Disqualified if: Mobitz Type II AV Block, Trifasicular Block, Acquired third-degree AV Block. No restriction if no associated cerebral ischemia with: Congenital third-degree AV block.
6. Artificial Cardiac Pacemakers	Should not drive 2 weeks after insertion of a pacemaker.	Waiting period of one week if: No cerebral ischemia. Normal sensing and capture on ECG. Device performing within manufacturer's specification.
C. Congestive Heart Failure		
1. Congestive Heart Failure	May drive if asymptomatic on moderate exertion.	No restrictions if: Functional Class I Functional Class II Functional Class III if: LV Class I (i.e., Ejection fraction 50 percent or more) or LV Class II (i.e., Ejection fraction 35 percent to 49 percent and Holter Class II - No episodes of ventricular tachycardia more than 3 beats in an average cycle length of 500 ms or less).
2. Hypertrophic Cardiomyopathy		No restriction if no associated cerebral ischemia, Holter Class II.
D. Cardiac Transplantation		
Cardiac Transplantation		Waiting period 2 months. Annual reassessment.

DLA = Driver Licensing Authority

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Section 4.1 Coronary Artery Disease/ Coronary Heart Disease

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Section 5: Cerebrovascular Diseases

5.1. Transient Ischemic Attacks
5.2. Cerebrovascular Accidents

A summary of the current fitness-to-drive guidelines (Cerebrovascular Diseases) for medical practitioners from Australia (1998) and Canada (2000) is presented in Table 13.

5.1 Transient Ischemic Attacks

Transient ischemic attacks (TIAs) are brief episodes of stroke-like symptoms that last less than 24 hours. TIAs are thought to be caused by temporary dysfunction of a portion of the brain caused by transient ischemia and are more common in the older population (Earnest and Cohen, 1990). Permanent cerebral damage does not occur with TIAs. TIAs are an important warning symptom and are a risk factor for a cerebrovascular accident. Approximately 20 to 30 percent of individuals experiencing a first TIA will have a completed stroke within three years (Mohr and Pessin, 1986).

Prevalence

There are an estimated 30,000 to 150,000 TIAs each year but accurate estimates are difficult because of the likelihood of under reporting (American Academy of Neurology, 1997).

Transient Ischemic Attacks and Driving Literature Review

The symptoms of TIA depend on the vessel involved. Earnest and Cohen (1990) provide a description of symptoms based on carotid artery involvement and vertebrobasilar involvement. Those symptoms are presented in Table 10.

Many of the neurological sequelae of TIAs, clearly, can have important implications for driving. There are, however, few studies available on the relationship between TIAs and increased risk of motor vehicle crashes. Rehm and Ross (1995) prospectively evaluated drivers 60 years of age and older with unexplained motor vehicle crashes presenting to their trauma center over a one-year period. Of the 79 drivers (aged 60-98), 73 percent were at-fault in the crash. Of those patients with a syncope etiology, eight percent were deemed due to TIA. Although there is a paucity of literature investigating the relationship between TIAs and motor vehicle crash risk, most medical guidelines recommend driving cessation following a single TIA or recurrent TIAs until the cause has been identified.

Table 10 Summary of Transient Ischemic Attack Symptoms by Vascular Supply *(Reproduced from Earnest, M.P., & Cohen, J.A. (1990). Cerebrovascular disease. In R.W. Schrier (Ed.), Geriatric medicine (pp. 109-118), with permission from W.B. Saunders Company, Philadelphia)*

Carotid Artery Territory	Vertebrobasilar Territory
Hemiparesis	Vertigo
Hemisensory deficit	Auditory symptoms
Aphasia	Ataxia
Monocular blindness	Diplopia
	Dysarthria
	Dysphagia
	Bilateral facial or limb sensory symptoms
	Bilateral weakness
	Hemianopsia or total blindness
	Drop attacks
	Syncope

5.2. Cerebrovascular Accidents

Prevalence

Cerebrovascular disease is the third leading cause of death in the United States and Canada (American Stroke Foundation, 2000). Approximately 160,000 Americans die of stroke each year, and of the 570,000 Americans who survive a stroke each year, approximately 10 to 18 percent will have another stroke within one year (American Stroke Foundation, 2000). Statistics suggest that of the approximately 80 percent who survive the initial period, 75 percent will be left with residual perceptual-cognitive deficits (Bonita, Anderson, and North, 1987; Gillum, Gomez-Marin, Kottke, et al., 1985; Mayo, Hendlisz, Goldberg, et al., 1989). Strokes are the leading cause of chronic neurologic disability, with estimates that suggest there are four million Americans currently living with the effects of a stroke (American Stroke Foundation, 2000).

The risk of stroke increases with age, with the risk doubling per decade after age 55 (American Stroke Foundation, 2000). Two thirds of all strokes occur in individuals 65 years of age and over. Given the aging of

North American society, the incidence of stroke is expected to increase over the next few decades. African-Americans in the United States are at increased risk for stroke. Statistics reveal that not only are African-Americans one and a half times more likely to have a stroke (288 per 100,000 versus 179 per 100,000 for whites), they are twice as likely to die from the disease and are more likely to suffer more extensive physical impairments, with their impairments enduring longer than other racial groups (American Stroke Foundation, 2000).

There are multiple risk factors for stroke including aging, heart disease, diabetes mellitus, hypertension, smoking, contraceptive use, hyperlipidemia, excessive alcohol use, and transient ischemic attacks (Davis, Dambrosia, Schoenberg, et al. 1987).

Cerebrovascular Accidents and Driving Literature Review

Strokes have the potential to affect driving either from sudden driver incapacitation during the event (acute) or from the subsequent debilitation following a stroke (chronic). The motor, perceptual, and cognitive impairments resulting from CVAs vary as a function of the location and extent of cerebral damage.

Research by Legh-Smith, Wade, and Hower (1986) reveals that of the 144 individuals interviewed post-stroke who were living at home and had driven prior to their stroke, only 42 percent resumed driving post-stroke. Fisk, Owsley, and Pulley (1997) surveyed 290 stroke survivors three months to six years post-stroke. Results revealed that 30 percent of those who drove prior to their stroke resumed driving after the stroke. It is interesting to note that of those surveyed, 48 percent reported that they had not received advice about driving and an overwhelming majority (87 percent) reported not receiving any type of driving evaluation to assess driving fitness.

Acute Event

There is a paucity of literature on sudden driver incapacitation as a result of a cerebrovascular accident. As noted in the cardiovascular section, 'sudden death at the wheel' due to cardiac or other illnesses is a rare event. Results from early studies suggest that 'sudden death while driving' is a causal agent in less than one percent of all crashes (Baker and Spitz, 1970; Grattan and Jeffcoate, 1968; Herner, Smedby, and Ysander, 1968; Peterson and Petty, 1962), and similar results are reported by Copeland (1987). When 'sudden death at the wheel' does occur, the most common causes are myocardial infarction and epilepsy, with myocardial infarction accounting for the majority of the deaths. Thus, the acute effects of cerebrovascular accidents are unlikely to account for a substantial number of motor vehicle crashes.

More precise estimates of the contribution of acute cerebrovascular accidents to motor vehicle crashes come from two studies: The first, a study in Connecticut (Finelli and Lee, 1986), the second from Munich Germany (Büttner, Heimpel, and Eisenmenger, 1999). Finelli and Lee explored the relationship between stroke and automobile crashes by retrospectively reviewing hospital records of 2,844 ischemic stroke patients admitted to Hartford Hospital in Connecticut. Four drivers were identified as having a cerebral infarct associated with a motor vehicle crash, an incidence of 0.1 percent. In three of the crashes, stroke was the cause and in one, the result of the crash. When stroke preceded the crash, visual field defect, impaired consciousness, and impaired motor control were major contributing factors. In a larger study, Büttner et al. reviewed the files of individuals from the Institute of Legal Medicine in Munich, Germany. From a sample of 34,554 deceased persons, 147 cases of sudden death at the wheel were identified (representing an incidence of 2 percent of all autopsies of unnatural death at the wheel during a 15-year period, beginning in 1982 and ending in 1996). The main cause of 'sudden death at the wheel' was ischemic heart disease (77 percent). CVA's were deemed responsible for five percent of sudden deaths at the wheel.

Chronic Impairment

There are a number of ways of determining fitness-to-drive: a) determination of relative risk through the use of crash statistics[1], or b) the prediction of fitness-to-drive through the use of medical evaluations, neuropsychological testing, functional assessments (typically carried out by rehabilitation specialists), driving simulators, or actual on-road tests (specialized or regular). Each of the methodologies has their strengths and limitations. For a review, please see Ball and Owsley (1991) and Hakamies-Blomqvist (1998).

Despite the fact that cerebrovascular accidents (CVAs) are a leading cause of disability, there is a paucity of research on the relationship between the chronic effects of CVAs and motor vehicle crashes. That which is available comes from a study in Utah (Diller et al., 1998) (see Section 2.1 a., page 4 for details of the study) and from the State of Washington (Haselkorn, Mueller, and Rivara, 1998). Relevant to this discussion are results of

1 As noted above, disability laws may limit the use of risk analyses by State licensing authorities as a basis for making fitness-to-drive decisions with regard to categories of applicants with specific medical conditions.

the data available from the Diller et al. study for the neurological conditions category. Individuals with neurological conditions such as stroke, head injuries, cerebral palsy, multiple sclerosis, Parkinson's Disease, and progressive conditions (e.g., muscular atrophies, dystrophy, myasthenia gravis) and other brain and spinal cord diseases were included. Epilepsy was considered in a separate category. The sample consisted of 3,007 unrestricted drivers and 771 restricted drivers. Results revealed that drivers with neurologic impairments had a significantly increased risk of motor vehicle crash. For unrestricted drivers, the relative risk was 4.21 (CI = 3.86 - 4.60); for restricted drivers the relative risk for all crashes was 2.18 (CI = 1.72 - 2.78). Unfortunately, data on the relative risk of drivers with specific impairments within the neurological category are not available. Therefore, the relative risk of drivers with CVAs alone is not available.

Haselkorn et al. (1998) conducted a retrospective study comparing the driving records of four cohorts hospitalized with CVA, traumatic brain injury, fractured extremities, and appendicitis with driving records of age, gender-, and zip code- matched non-hospitalized controls. Hospitalized records for 1992 for the cohorts were linked with Department of Licensing records for 1991 to 1993. Measures included the occurrence of crashes or citations for moving violations. Linkages identified 1,917 patients with CVA, representing 39 percent of patients hospitalized with CVA. Data from individuals with more than one disease classification were excluded from the study, resulting in a sample of 1,910 patients with CVA, with the majority (73 percent) of the CVA sample 60 years of age and older. Results of estimates of relative risk (RR) for crash revealed that the CVA sample did not have an elevated risk relative to their comparison group (RR = 0.8, CI = 0.6-1.4). The results suggest, therefore, that individuals post-CVA do not have an increased risk of motor vehicle crashes during the 12 months following hospitalization for the event.

However, a number of methodological limitations are noted. First, driving exposure was not taken into consideration. It is likely that individuals experiencing a stroke will drive less frequently (particularly in the first several months following their stroke) compared to the non-hospitalized comparison group. If driving exposure is indeed less, then the results underestimate the risk of crash. Second, based on available data, the authors were unable to determine the severity of brain damage. Given that the CVA sample was representative of only 39 percent of patients hospitalized with CVAs, it is unknown if only those with mild impairments were included. Such an occurrence also would underestimate the risk of crashes. Finally, the data included only state-recorded crashes, data susceptible to errors of omission.

The majority of studies reporting on assessments of fitness-to-drive following a CVA have employed neuropsychological testing and some type of on-road assessment. A summary of the studies is presented in Table 11.

In general, results of fitness-to-drive assessments of individuals who have suffered a cerebrovascular reveal that, of those presenting for assessment, approximately 50 percent or more fail the assessment. Methodological differences, however, make comparisons across studies difficult. For example, some studies include subjects from different diagnostic categories; when similar diagnostic categories are used (e.g., left and right CVA), the exact location and extent of damage often is unknown, and testing time post-stroke frequently differs or is unknown. In addition, there often is considerable variability in neuropsychological assessment batteries and criteria used for subject classification based on neuropsychological performance. For example, often batteries are chosen to include tests that are sensitive to areas of impairment: language-oriented functions for individuals with left-hemisphere damage and visuospatial functions in individuals with right-hemisphere damage. Often neglected, however, is attention to the generalized cognitive deficits that may result as a consequence of a stroke. Finally, on-road assessments differ dramatically in terms of assessment procedures and criteria used for scoring. In most cases, criteria are not well defined or are lacking. As noted by Springle, Morris, Nowachek, and Karg (1995), criteria used for evaluation of results are based on subjective criteria or no criteria. What is needed is a standardized driving evaluation procedure that has been shown, through research, to be valid and reliable.

As noted earlier, attention to generalized cognitive functioning often is neglected in assessment of individuals post-CVA. A number of studies have investigated the effect of a CVA on *generalized* cognitive functioning. Horn and Reitan (1990) compared the performance, based on an extensive battery of neuropsychological tests, of 60 patients with lateralized or diffuse cerebrovascular lesions to 20 controls. Results indicated that the group with cerebrovascular lesions performed significantly worse than controls on measures of cognitive functioning. Important to this discussion is the finding that significant neuropsychological impairments were noted in the group with cerebrovascular lesions that extended beyond the expected lateralized dysfunctions or selected impairments associated with the damaged hemisphere. Tatemichi, Desmond, Stern, Paik, and Bagiella (1994) examined cognitive function in 227 patients three months following admission for ischemic stroke. Results were compared to 240 stroke-free controls. Like Horn and Reitan, the authors were interested in focusing on general cognitive impairments rather than on

Table 11 Summary of Studies Assessing Fitness-to-Drive in Individuals Post-CVA

Authors	Sample	Assessment Criteria	Results
Jones et al. (1983)	(L) CVA = 43 (R) CVA = 48	On-road assessment (ability to operate vehicle controls, perceptual and cognitive responses in demanding situations, knowledge of rules of the road, and general attitude). Criteria not specified.	(L) CVA = 42 percent failed the road test. (R) CVA = 48 percent failed the road test.
Lincoln & Fanthome (1994)	CVA = 36 (at least one year post stroke).	Stroke Drivers Screening Assessment (SDSA). -Dot cancellation (DC). -What's in the square? (WIS). -Road Sign Recognition (RSR). Algorithms for predicting likelihood of passing a road test and likelihood of failing a road test used. PASS = [(DC time x 0.012) + (DC false positives x 0.216) + (WIS x 0.049) + (RSR x 1.168)] -13.79. FAIL = [(DC time x 0.017) + (DC false positives x 0.035) + (WIS x 0.185) + (RSR x 0.813)] -10.04. Patients tested on two occasions 6 weeks apart.	<u>Initial Assessment</u> 8 passed. 28 failed. <u>Second Assessment</u> 6 passed. 30 failed. Significant practice effects observed on individual tests. However, patients who initially failed did not pass the second assessment as a result of practice.
Lings & Jensen (1991)	(L) CVA = 67 (R) CVA = 46 C = 109 LH: Disease duration, 2 yrs (median) RH: Disease duration, 2 yrs (median)	Mock Cars (Part-task driving simulators-tested sensori-motor performance). Neuropsychological testing.	Stroke patients performed signifi-cantly worse than controls on most all measures. Reaction times longer for paretic and contralateral extremities. Strength in unaffected side also reduced. More directional errors in (L) CVAs. Clinical examination did not predict Mock Car performance.

Table 11 Summary of Studies Assessing Fitness-to-Drive in Individuals Post-CVA *(continued)*

Mazer et al. (1998)	CVA = 84 (tested on average 4.5 months after stroke)	Battery of Perceptual Tests Complex Reaction Timer. Motor Free Visual Perception Test (MVPT). Cancellation Test (Single and Double letter). The Money Road Map Test. Trail Making Tests A & B. On-road evaluation (based on standard provincial testing procedures-43 item assessment form).	On-Road Evaluation Pass = 33 Fail = 51 As a group, those who failed the on-road evaluation performed more poorly on most perceptual tests. Logistic Regression Results: Study group as a whole The greatest odds of failing were predicted by the MVPT. Those who scored < were 8.7 times more likely to fail the road test than those who scored > 30. L Hemisphere Lesions Those who had 3 or more errors on Trails B were 11 times more likely to fail road test. R Hemisphere Lesions Those scoring < 30 on MVPT were 15 times more likely to fail road test.
Nouri et al. (1987)	(R) CVA = 23 (L) CVA =16 Tested 6 weeks-4 years post stroke	Cognitive Assessment Cube Copy Test. Dot Cancellation. Rey Figure Copy and Recall. Four Choice Reaction Time. What's In the Square?. What Else Is In the Square?. Pursuit Rotor. Token Test Part V. Road Sign Recognition Test. Titmus Vision Tester. Hand Sequencing Task. Recognition Memory Test-Faces. Hazard Recognition Task. Road Test Rated on 23 items (good or faulty) and graded as Pass (Good, Average) or Fail (Borderline, Below Standard). Criteria not defined.	Results of Road Test Pass = 22 Fail = 17 Discriminant Function Analysis used to determine best predictors. Results revealed that Dot Cancellation, Rey Figure, What Else Is In The Square, Pursuit Rotor, Token Test, Vision Testing, Recognition Memory test, Cube Copying, and Hazard Recognition were the best predictors. Algorithm developed which correctly classified 37 of the 39 patients (94.9 percent). (Misclassified 1 case who failed the road test as a pass (5.9 percent), and 1 case who passed the road test as a fail (4.5 percent)).

Table 11 Summary of Studies Assessing Fitness-to-Drive in Individuals Post-CVA *(continued)*

Sivak et al. (1981)	(L) CVA = 10 (R) CVA = 6 Diffuse brain damage = 7 Spinal cord damage = 8 Able bodied = 10	Neurocognitive Tests (Perceptual and cognitive). Visual acuity and stereodepth. On-Road Test. Closed Course (Number of knocked over cones, displaced cones, correct responses to a secondary task, and time). Open-Road Rated on an average of 144 actions, with rating based on a 2 point scale: well executed and not well executed.	Neurocognitive Tests Subjects with brain damage, as a group, performed worse than able-bodied subjects. On-Road Test. Closed Course. Subjects with brain damage performed significantly worse on several measures. Spinal cord damage subjects' performance did not differ from able-bodied subjects' performance. Open-Road. 4 subjects (1-CVA, 3-diffuse brain damage) were deemed unsafe for open-road assessment based on closed course performance (2) or open-road drive terminated shortly after commencing. In general, subjects with brain damage performed significantly worse on several on-road measures than able-bodied (No percentages of pass/fail as a function of group membership are provided). Those with spinal-cord damage did not differ from controls. No correlations were found between closed course measures an open road driving for brain damaged individuals. Several of the neuropsychological tests were correlated with open road Composite Driving Index, but tests for the brain damaged subjects were different than those for the non-brain damaged.

Table 11 Summary of Studies Assessing Fitness-to-Drive in Individuals Post-CVA *(continued)*

Sundet et al. (1995)	(L) CVA = 29 (R) CVA = 43 M. Infarct = 7	<u>Neuropsychological Testing</u> -visual perception. -spatial attention. -visuospatial processing. -language/praxis. Decisions re: driving suitability based on clinical judgement. Patients with CVA tested an average of 4 months post stroke.	Classified as unsafe to drive based on Clinical Global Ratings. (L) CVA's = 41 percent. (R) CVA's = 58 percent.
Wilson & Smith (1983)	CVA = 11 Older C = 11 (45-65 years) Young C = 8 (18-26 years)	<u>On-Road assessment.</u> (Mixture of city driving, motorway driving, and country driving). Scored on a 5 point scale.	Stroke drivers were significantly impaired on entering and leaving motorways and handling traffic at roundabouts. On private roads, stroke patients were relatively unaware of other vehicles, had difficulty in reversing, in doing two things at once in an emergency, and had difficulty in placing car accurately on the left (Great Britain study).

C = Controls
CVA = Cerebrovascular Accident
TBI = Traumatic Brain Injury
THI = Traumatic Head Injury
(L) = Left
(R) = Right
LH = Left Hemiparesis
RH = Right Hemiparesis

describing the circumscribed deficits associated with focal impairments. Compared to controls, impairments were noted on all 17 neuropsychological items. Cognitive impairment, defined as failure on any four or more items, occurred in 35 percent of stroke patients and four percent of controls. Cognitive domains most likely to be impaired were memory, orientation, language, and attention. Finally, results from the Copenhagen Stroke Study (Pederson, Jorgensen, Nakayama, Raaschou, and Olsen, 1996) indicated that, of those stroke patients completing the Mini Mental Status Examination (MMSE) one week after stroke onset, 42 percent scored below the cut-off level of 24. Further, MMSE scores one week post-stroke were significantly correlated with functional assessment at discharge. Results of these studies underscore the importance of assessing for generalized cognitive functioning in individuals post-stroke.

A number of investigators have recognized the need for a standardized procedure for assessing individuals following a cerebrovascular accident (Engum, Pendergrass, Cron, Lambert, and Hulse 1988a; Galski, Bruno, and Ehle, 1992; Korner-Bitensky, Sofer, Kaizer, Gelinas, and Talbot, 1994; Nouri, Tinson, and Lincoln, 1987). Results of studies aimed at enhancing the precision and rigor of fitness-to-drive assessments in stroke patients are summarized in Table 12. The approaches taken by Engum et al. (1988a, b, 1989, 1990) and Galski et al. (1990, 1992, 1993; 1996) are the most extensive and programmatic available. The approaches are ones that deserve special attention because they are likely to be instructive for future investigations in this area as well as in other areas.

Engum et al. (1988a) developed the Cognitive Behavioral Driver's Inventory (CBDI) specifically to provide rehabilitation professionals with a standardized battery for determining fitness-to-drive in individuals with a brain-injury (see Engum et al., 1988a for a description of the battery). Briefly, the battery consists of 10 tests measuring attention, concentration, rapid decision making, stimulus discrimination/response differentiation, visual scanning and acuity, and attention shifting. The criterion measure is on-road performance. The initial investigation provided data regarding internal consistency of the items in the battery (Cronbach's alpha = 0.949) and preliminary estimates of validity based on on-road driving performance. The battery was validated in 1989 on a sample of 175 brain-injury patients (Engum, Lambert, Scott, Pendergrass, and Womac, 1989). The correlation between outcomes on the CBDI (pass/fail) and road test outcome (pass/fail) was significant ($r = 0.81$, $p<.0001$). Overall, of the 42 patients receiving a favorable pass on the CBDI results, 40 passed the road test. However, 7 of the 39 patients receiving a fail on the CBDI results passed the road test. The sensitivity of the CBDI was further evaluated in 1990 (Engum, Lambert, and Scott, 1990). Results of that investigation show the CBDI to be highly sensitive in discriminating between healthy controls, brain-injured patients passing the road test, and brain-injured patients failing the road test (see Table 12 for a more detailed description).

The approach taken by Galski et al. (1990, 1992, 1993; 1997) was to first critically assess evaluations developed at their facility to determine fitness-to-drive (Galski, Ehle, and Bruno, 1990). Results of that investigation revealed a lack of internal and predictive validity of the driver evaluation. A model was then developed for evaluating fitness-to-drive (Galski, Bruno, and Ehle, 1992). The utility of the model was determined in terms of amount of variance of the criterion (on-road evaluation) explained by the predictor variables (neuropsychological tests, driving simulator, and parking lot driving scores). The results of that investigation revealed that 93 percent of the on-road assessment (in traffic) was explained by three predictor variables. In a follow-up investigation Galski, Bruno, and Ehle (1993), using a larger sample size, determined the effectiveness of the evaluation methods developed in the 1992 investigation by discriminant function analysis and measurements of sensitivity. The methods were found to be highly sensitive in predicting outcomes. In their 1997 study, Galski, Ehle, and Williams explored the dimensions underlying their pre-driver assessment (neuropsychological battery) and measures from a driving simulator. Results of factor analysis indicated the presence of five underlying factors (higher order visuospatial abilities, basic visual recognition and responding, anticipatory braking, defensive steering, and attentional measures), accounting for 66 percent of the variance. As noted by the authors, the factors can be used as a basis for understanding what is measured in off-road evaluations and in determining fitness-to-drive following cerebral injury. Details of the approach are provided in Table 12.

Table 12 Summary of Studies Assessing the Psychometric Properties of Fitness-to-Drive Evaluations in Patients Suffering from Cerebrovascular Accidents

Authors	Sample	Assessment Criteria	Results
Engum et al., (1988a; 1988b; 1989; 1990)	Patient samples consist of individuals with brain injury. 1988a n = 92 1998b n = 121 1989 n = 175 1990 n = 215 C = 41	Cognitive Behavioral Driver's Inventory 10 Neuropsychological tests: -Attention. -Concentration. -Rapid decision making. -Stimulus discrimination/ response differentiation. -Visual scanning and acuity. -Attention shifting. Yields 27 response measures. On-Road test Criterion measure.	1988a *Reliability* Good internal consistency (Cronbach's alpha = 0.949). *Validity* (based on General Driver's Index and on-road performance as criterion measure). Of 44 passing the CBDI, 42 passed road test. Of 48 failing the CBDI, only 6 were allowed to attempt the road test and all failed. 1988b Normative tables derived from 121 brain-injured patients provided, complete with decision-making rules. 1989 Double blind validity study. *Validity* (CBDI and on-road measures). Of 42 passing the CBDI, 40 passed road test. However, of 39 patients failing the CBDI, 7 passed the road test. 1990 109 brain injured passed the road test and 54 failed. 118 brain injured passed the CBDI and 97 failed. CBDI was sensitive to discriminating between controls, brain injured who passed, and those who failed road test. Importantly, age was a confounding factor.

Table 12 Summary of Studies Assessing the Psychometric Properties of Fitness-to-Drive Evaluations in Patients Suffering from Cerebrovascular Accidents *(continued)*

Galski et al. (1990)	CVA = 23 THI =14	Physical and Neuropsychological Tests (21 tests) -Attention. -Concentration. -Reaction time. -Memory. -Visual acuity. -Visuospatial skills. Scores converted to pass/fail by driver evaluator (criteria not defined). On-Road test -Consisted of 26 tasks believed to require an integration of basic driving skills. -Pass/fail if sufficient skill demonstrated to driving evaluator (criteria not specified).	Physical and Neuropsychological tests Only 4 of the tests predicted pre-driver evaluation outcome (Benton Visual Retention Test, cancellation test, visual acuity measure, and observations of inattention). Neither the pre-driver evaluation outcome nor any of the pre-driver evaluation tests predicted on-road evaluation outcome. On-Road Test Only six of the 26 on-road measures correlated with on-road evaluation outcome (caution, backing up in parking lot, on highway, parking on grade, lane use, and evaluating right of way). Conclusion Pre-driver evaluations must change from an attempt to measure abilities assumed to predict driving to an effort to screen out patients who are unsafe behind the wheel. Items used in driving evaluation, although high in face validity, were low in predictive validity. Test battery accounted for very small percentage of variance. Authors call for research involving the empirical evaluation of tests used in pre-driver evaluations and on-road

Table 12 Summary of Studies Assessing the Psychometric Properties of Fitness-to-Drive Evaluations in Patients Suffering from Cerebrovascular Accidents *(continued)*

Galski et al. (1992)	n = 13 (CVA) n = 21 (THI) Time since injury of stroke .08 to 17 years (mean = 1.8 + 3.6 years	<u>Neuropsychological Testing Battery</u> -sensory input. -scanning and attention. -calculation and construction. -general and specific driving knowledge tests. -resident diagnostic program (executive functions, awareness of deficits, etc.). -integration (from simulator and on-road testing- seat and mirror adjustment, signaling, steering and tracking, etc.). <u>Doron Simulator</u> -view and react to films from Doron Driving Analyzer (e.g., Threat Recognition and Crash Avoidance). -outcome measures were errors in braking and steering to escape danger or avoid disasters. <u>On-Road Evaluation</u> -criteria for ratings operationally defined prior to study. -pass/fail ratings used to assess performance on individual measures. -critical behaviors (impulsivity, distractability, confusion, anxiety, inattention, slowness, following directions, evaluation) scored as present or absent.	*Behind the wheel performance used as the criterion of fitness to drive.* Utility of measures determined by the amount of variance of the behind the wheel evaluation (street component) explained by a) the pre-driver evaluation, b) the simulator evaluation, and c) parking lot driving scores. <u>Pre-driver Evaluations</u> (Neuropsychological Tests) 64 percent of the behind-the wheel evaluation performance explained by tests that measured visual perception, visuomotor coordination, visuoconstructive abilities (planning, organizing, and executing test operations), and scanning and attention (selective and sustained). <u>Doron Simulator</u> Variance explained by simulator independently accounted for 63 percent of the variance. When simulator measures combined with pre-driver evaluation, the best simulator items enhanced the predictive ability of pre-driver evaluation by only 6 percent. *Most of the simulator measures (e.g., braking, reaction time, steering, and acceleration) were ineffective predictors of behind-the wheel performance.* Only two simulator items were significant predictors of driving outcome: appropriate use of signals on an introductory film and calculated percentage of valid attempts to steer out of potentially hazardous situations. <u>Parking Lot Driving Scores</u> Behind the wheel lot behaviors (e.g., following directions, slow response, inattention, distractability and lot index [lot behaviors scored and ranked, and then summed]) accounted for an additional 14 percent and 9 percent respectively of the outcome variance (raising the level of explained variance to 93 percent).

Table 12 Summary of Studies Assessing the Psychometric Properties of Fitness-to-Drive Evaluations in Patients Suffering from Cerebrovascular Accidents *(continued)*

Galski et al. (1993)	n = 48 (CVA) n = 58 (TBI)	Neuropsychological Testing Battery, Doron Simulator, and On-road Performance (see details in Galski et al., 1992).	Study designed to determine the discriminative power and measurements of sensitivity of the battery described in 1992 study. Results Methods of evaluation sensitive in predicting outcome: off-road and on-road sensitivities of 90 percent and 92 percent with the inclusion of behavioral measures were obtained. Importantly, results revealed that residual deficits in cognition per se did not render a person unfit to drive. The research underscores the importance of considering behaviors in determining fitness-to-drive.
Galski et al. (1997)	n = 106 (CVA and THI)	Neuropsychological Battery and Doron Simulator measures (see Galski et al., 1992).	Study objective was to determine the underlying factors of psychomotor testing and simulator evaluations useful in assessments of fitness-to-drive. Factor analysis identified 5 factors, accounting for 66 percent of the variance: 1. Higher Order Visuospatial Abilities. 2. Basic Visual Recognition and Responding. 3. Anticipatory Braking. 4. Defensive Steering. 5. Behavioral Manifestations of Complex Attention. More research needed to identify other relevant measures as 34 percent of the variance remains unexplained.

Table 12 Summary of Studies Assessing the Psychometric Properties of Fitness-to-Drive Evaluations in Patients Suffering from Cerebrovascular Accidents *(continued)*

Nouri & Lincoln (1992)	LH = 20 RH = 20 Mean time since stroke 33.3 + 40.7 months	Cognitive Assessment See battery in Nouri et al. (1987). Tasks excluded in this study were Choice Reaction test, Stereodepth Perception test, and the Hand Sequencing Task. Road Test Rated on 26 items (Correct or Fault) and graded into one of three categories: Pass, Borderline, and Fail (based on overall subjective impression). Different driving instructor and different route than 1987 study.	Purpose of study was to validate the cognitive battery developed in 1987 study. Results of Road Test Pass = 12 Borderline = 8 Fail = 20 Unable to validate equation from 1987 study because results of road tests between studies so discrepant (majority passed in 1987 study and majority failed in present study-may be due to change in instructor, route, or difference in severity of patient). New algorithm developed based on three tests: Dot Cancellation, What Else Is In The Square?, and Road Sign Recognition Test (Stroke Drivers Screening Assessment). Predictive values of algorithm ranged from 79 to 82 percent correct classification. No further details of classification provided.
Nouri & Lincoln (1993)	CVA = 27 (given SDSA) compared with general practitioner ratings for CVA = 25	All subjects given an on-road evaluation (criteria not defined). 27 subjects were given the Stroke Drivers Screening Assessment (SDSA). 27 subjects sought the advice of their General Practitioner re: driving fitness.	Comparison of predictive value of the SDSA and Advice from General Practitioner based on clinical assessment to results from road test. Results reveal that SDSA is better than General Practitioner assessment at predicting on-road performance. However, neither assessment is very accurate. For example, physicians misclassified 44 percent of the patients based on on-road criteria. The SDSA misclassified ~19 percent of the patients based on on-road criteria.

C = Controls
CVA = Cerebrovascular Accident
TBI = Traumatic Brain Injury
THI = Traumatic Head Injury
LH = Left Hemiparesis
RH = Right Hemiparesis

Table 13 Guidelines for Cerebrovascular Diseases (Reproduced with permission)

Guidelines for Cerebrovascular Diseases		
Illness	**Austroads (1998)**	**CMA (2000)**
Transient Ischemic Attacks (TIAs)	Should not drive for 6 weeks. Fitness-to-drive determined by cause of TIA. Should not drive for 2 years if multiple TIAs occur resulting in impaired consciousness or awareness, vertigo, or visual disturbances.	Patients who have experienced either a single or recurrent TIA should not be allowed to drive any type of motor vehicle until a complete assessment by a neurologist.
CVA's		
Aneurysms	Berry Aneurysms Should not drive after detection until assessed by a neurosurgeon and assessment confirms fitness-to-drive. Post Intracranial Surgery Should not drive for a minimum of 3 months post surgery and assessment by relevant specialist (Neurologist/ Neurosurgeon). Vascular Malformations of Brain Should not drive until assessed by specialist. The DLA may issue a license if risk of bleed is small and patient free of other conditions (e.g., epilepsy).	Untreated Absolute barrier to driving any class of motor vehicle. After Surgical Treatment Waiting period 3 months.
Strokes	Should not drive for 3 months post event. Medical and driving assessor* opinion recommended. Formal assessment of visual fields required.	Should not drive for at least one month. May resume driving if functionally able and if a neurologic assessment discloses no obvious risk of sudden occurrence and any underlying cause has been addressed with appropriate treatment. Residual loss of motor power-road test may be required. Physician should take particular care to note any changes in personality, alertness, or decision-making ability. Should remain under regular medical supervision.
Subarachnoid Hemorrhages	Should not drive for 3 months post event. Medical and driving assessor* opinion recommended. Formal assessment of visual fields required.	Not addressed.

** Driving assessor defined as a professional who assesses the fitness-to-drive of those with a medical condition.*
DLA = Driver Licensing Authority

Conclusions

Excepting for head injuries, the disability arising from stroke can be more varied than that associated with any other medical condition. The variability is not only in the severity of the disability, but also in the type of impairment. These impairments can range from paralysis without cognitive involvement to a wide range of cognitive impairments, including focal and generalized impairments. Combinations of physical, perceptual, motor, visual, and other senses can be impaired. Not surprisingly, then, the emphasis concerning fitness-to-drive cannot be on whether a stroke has occurred, but on mental and functional assessments of the consequences of the stroke for the individual. Future research is clearly needed so that advancements in the standardization of mental and functional assessments for fitness-to-drive for stroke individuals can continue.

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Section 6: Peripheral Vascular Diseases

Peripheral vascular disease (PVD) is a circulatory disorder involving any of the blood vessels outside the heart. Diseases of the lymph vessels are included in this classification. There are two types of PVDs: functional and organic. Functional PVDs are not due to organic causes and do not involve defects in the structure of the blood vessels. The effects are short-term. Raynaud's Disease is an example of a functional PVD. With Raynaud's Disease, exposure to cold or emotional stimulation results in intermittent attacks of pallor or cyanosis of the digits. In contrast to functional PVDs, organic PVDs are caused by structural changes, such as inflammation and tissue damage in the blood vessels (e.g., Buerger's disease). Buerger's disease is characterized by acute inflammatory lesions and occlusive thrombosis of the arteries and veins.

A summary of the current fitness-to-drive guidelines (Peripheral Vascular Diseases) for medical practitioners from Australia (1998) and Canada (2000) is presented in Table 14.

Prevalence

The prevalence of PVD is most commonly found in individuals 55 years of age and older, but prevalence increases with age (Hiatt and Regensteiner, 1990). For example, the prevalence of peripheral arterial disease in a population of retirees was 12 percent for those with an average age of 66, with an increase to 20 percent in those 75 and older (Criqui, Fronek, Barrett-Connor, et al., 1985).

Peripheral Vascular Diseases and Driving Literature Review

In essence, all PVDs are characterized by disturbances in blood flow through the peripheral vessels resulting, eventually, in damage to peripheral structures and loss of functional capacity. The majority of individuals suffering from vascular diseases also suffer from other diseases that affect their vascular system (e.g., heart disease, diabetes). Therefore, literature assessing the effects of peripheral vascular diseases on crash risk is often considered under other specific disease entities (coronary disease, cerebrovascular disease).

Despite the lack of available literature, current fitness-to-drive guidelines for medical practitioners from Australia (1998) and Canada (2000) have been developed for specific categories of peripheral vascular disease (e.g., aneurysms) or specific functional impairments as the result of peripheral vascular diseases (e.g., intermittent claudication). Those guidelines are summarized in Table 14.

Table 14 Guidelines for Peripheral Vascular Diseases *(Reproduced with permission)*

Guidelines for Peripheral Vascular Diseases (Drivers of Private Vehicles)		
Illness	**Austroads (1998)**	**CMA (2000)**
Arterial Aneurysms	Abdominal and Thoracic Aneurysms Untreated aneurysm over 5 cm need specialist examination. Should not drive 4 weeks post repair.	Aneurysms larger than 5 cm should be treated by surgery in order to be licensed to drive. No restrictions following surgical recovery.
Peripheral Arterial Vascular Diseases (e.g., Raynaud's Phenomena, Buerger's Disease, and Arterio-sclerotic occlusions)	Not addressed.	If of sufficient severity to cause claudication, may preclude driving, and always require careful evaluation and regular ongoing surveillance.
Diseases of the Veins	Deep Vein Thrombosis Should not drive for 2 weeks post event, subject to clinical assessment.	Acute Episodes of Deep Venous Thrombosis Should not drive because of danger of embolization and/or pulmonary infarction. May drive after appropriate treatment and with physician approval.

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Section 7: Diseases of the Nervous System

7.1 Syncope
7.2 Seizures
7.3 Sleep Disorders
  7.3.a. Narcolepsy
  7.3.b. Sleep Apnea

A summary of the current fitness-to-drive guidelines (Diseases of the Nervous System) for medical practitioners from Australia (1998) and Canada (2000) is presented in Table 21.

7.1 Syncope

See discussion and review of syncope in Section 4 (Cardiovascular Disease).

7.2 Seizures

A seizure is a paroxysmal uncontrolled abnormal discharge of electrical activity in the gray matter of the brain, causing clinical signs and symptoms that interfere with normal functioning. A seizure is not a disease but is a symptom of underlying pathology. Numerous conditions can induce a seizure (e.g., central nervous system infections, hyperpyrexia, intracranial tumors, cerebral hypoxia, cerebral trauma, alcohol or drug withdrawal, etc.).

According to McLachlan (1993), up to nine percent of the population will have at least one seizure during their lifetimes. Estimated risks of a recurrence following a single unprovoked seizure range from 23 percent (Pearce and MacKinstosh, 1979) to 71 percent (Elwes, Chesterman, and Reynolds, 1985). Recently, Berg and Shinnar (1991) explored the reasons for the variability in estimated risk reported in the literature. Results of their meta-analysis revealed that three methodological factors explained much of the variance: 1) study inclusion criteria, 2) retrospective versus prospective ascertainment of patients, and 3) the interval between the first seizure and the time at which the risk was assessed. The average recurrence risk across the studies included in the meta-analysis was 51 percent. Relevant to this review was the finding that the risk of seizure recurrence was highest in studies of children only (54 percent), compared to studies of adults only (43 percent). Predictors of seizure recurrence included seizure etiology and electroencephalogram (EEG) abnormalities. An abnormal neurological status was associated with a substantially increased recurrence risk compared to those with idiopathic seizures (e.g., without underlying brain pathology). EEG abnormalities were associated with a higher risk of seizure recurrence. Notably, patients with idiopathic seizures and normal EEG's were found to have a low recurrence risk of about 24 percent at two years. Finally, partial seizures were, in general, associated with an increased risk of seizure recurrence. Results of Berg and Shinnar's meta-analysis may be useful when making decisions about licensing decisions for individuals with first unprovoked seizures. Specifically, results from this meta-analysis suggest that two commonly assessed patient characteristics (seizure etiology and EEG results) are helpful predictors for seizure recurrence.

Epilepsy

Epilepsy, a common neurological disorder, is characterized by recurrent seizures that can involve loss of consciousness, convulsive movements or other motor activity, sensory phenomena, or behavioral abnormalities. Epilepsy has a prevalence rate of 0.7 percent (McLachlan, 1993) and an overall incidence of about 50 per 100,000 (Hauser, Annegers, and Rocca, 1996). The incidence of new-onset epilepsy is such that onset is highest in the first year of life, decreasing to a minimum during middle age (30's and 40's), and increasing again in individuals 60 years of age and older (Hauser, 1992).

Epileptic seizures often are classified into two broad groups, depending on etiology: 1) symptomatic or secondary epilepsy, and 2) idiopathic or primary epilepsy. In symptomatic or secondary epilepsy, the probable cause of the seizures often can be determined, whereas in idiopathic or primary epilepsy, specific causes cannot be found. Approximately two-thirds of epilepsy in young adults is idiopathic in origin. However, more than 50 percent of epilepsy in elderly individuals has a known cause (Hauser, Anderson, Lowenson, and McRoberts, 1992), including cerebrovascular disorders (~33 percent), brain tumors (~10-15 percent), and infections, trauma, or other secondary lesions (~23 percent) (Luhdorf, Jensen, and Plesne, 1986).

Classification of Epileptic Seizures

There are numerous classifications of epileptic seizures. The most widely accepted is the classification system advanced by the Commission on Classification and Terminology of the International League Against Epilepsy (1981). As can be seen from Table 15, within this system, seizures are classified as partial or generalized. The three most common types of seizures in adults are a) generalized tonic-clonic seizures (GTCSs), with convulsions (grand mal seizures), b) complex partial seizures (CPSs), with alterations of consciousness, and c) simple partial seizures (SPSs). Approximately one-third of all epileptic individuals have CPSs, with the prevalence increasing to approximately one-half in elderly epileptic individuals (Hauser, 1992).

Table 15 Classification System of Epileptic Seizures as Advanced by the Commission on Classification and Terminology of the International League Against Epilepsy (1981)

The International Classification of Epileptic Seizures
I. Partial (focal, local) seizure A. Simple, partial seizures (SPSs) Motor, somatosensory, autonomic, or psychic symptoms. B. Complex partial seizures (CPSs) 1. Begin with symptoms of simple partial seizure but progress to impairment of consciousness. 2. Begin with impairment of consciousness. C. Partial seizures with secondary generalization 1. Begin with simple partial seizure. 2. Begin with complex partial seizure (including those with symptoms of simple partial seizures at onset). II. Generalized seizures (convulsive or non-convulsive) A. Absence (typical and atypical) B. Myoclonic C. Clonic D. Tonic E. Tonic-clonic (GTCSs) (with convulsions, also known as grand mal seizures) F. Atonic/akinetic III. Unclassified

Source: from Commission on Classification and Terminology of the International League Against Epilepsy (1981). Available online at: http://neuroland.com/

Epileptic Seizures and Driving Literature Review

Epileptic seizures, which can result in abrupt loss of consciousness or loss of bodily control, place the individual at risk for motor vehicle crashes if the seizure occurs while driving. Because of the potential for rapid incapacitation of the driver, and of the unpredictability of the illness, epilepsy is one of the few medical conditions with driving restrictions that are enforced almost worldwide. The first report of a motor vehicle crash as a result of a seizure was in the early 1900's (Thakwitzer, 1906). Since then, a number of studies have reported an increased risk of crashes in individuals with epilepsy, with rates of crashes for individuals with epilepsy ranging from 1.5 (Crancer and McMurray, 1968; Keys, Martin, Barrow, and Fabing, 1961) to 1.95 (Waller, 1965) times greater than controls. More recent studies are equivocal: Taylor, Chawick, and Johnson (1996) suggest that the crash rates of individuals with epilepsy are no greater than the general population, after adjusting for age, gender, driving experience, and mileage, whereas results from Diller et al. (1998) suggest that crash rates are elevated for individuals with epilepsy (see Table 16). Table 16 provides a summary of results from older and more recent studies investigating the risk of crashes in individuals with epilepsy.

The majority, if not all, of the studies examining the crash rates of individuals with epilepsy have methodological limitations. For example, many studies are based on self-report, using questionnaire surveys or clinical interviews (Gastaut, and Zifikin, 1987; Hasegawa, Gastaut, and Zifikin, 1991; Stanaway, Johnson, and Lambie, 1983; Takeda, Kawai, Fukushima, and Yagi, 1991, Taylor, Chawick, and Johnson, 1996). In those studies, estimations of the crash rates of drivers with epilepsy may, in fact, be underestimations given the propensity for under-reporting of crashes in this population due to fear of license revocation (Andermann et al., 1988; Salinsky, Wegener, and Sinnema, 1992). In other studies, individuals with epilepsy were identified through state licensing authorities (Popkin and Waller, 1989) or crash rates of persons with epilepsy were determined through police identification following a crash; individuals were then referred for a medical evaluation on suspicion of epilepsy (Van der Lugt, 1975). In those instances, crash rates of drivers with epilepsy may be inflated because individuals with moderate to severe epilepsy are likely to be overrepresented and individuals with fully controlled or infrequent seizures are likely to be under-represented.

As noted previously, results from earlier studies (e.g., Crancer and McMurray; Keys et al., 1961; Waller, 1965) suggest that individuals with epilepsy have increased crash risks. However, recent advances have resulted in improved medications for controlling seizures. Moreover, increased understanding by the medical community and by patients of the causes and effects of epilepsy has, undoubtedly, resulted in improved seizure

Table 16 Summary of Studies Examining the Risk of Crash for Individuals with Epilepsy

Study	Sample Size	Methodology (Outcome measure)	Results
Waller (1965)	E = 580 C = 926	State recorded crashes. (crashes/million miles).	E = 1.95 higher crash rates than comparison sample.
Van der Lugt (1975)	E = 155 H C = all crashes in Netherlands in 1963	Police reports. (# epileptic crashes/ total crash).	1:10,000 crashes caused by epilepsy.
Stanaway et al. (1983)	Seizures = 103	Survey. (crashes/1,000/year).	S = 5.5/1000/yr. Pop = 4.3/1000/yr.
Gastaut & Zifikin (1987)	E = 82	Self-report. i. Seizures while driving. ii. Crashes as a result of seizure	17 percent - seizures while driving 52 percent - crash as a result of seizure.
Popkin & Waller (1989)*	E = 112 Known to DMV = 29 Unknown to DMV = 83	Driving records. (crashes/100 drivers/ year).	Crashes/100 drivers/year. Known = 8.6. Unknown = 6.7. Population = 6.0.
Hanostia & Broste (1991)	E = 241	Population based retrospective cohort study (SMR).	SMR = 1.33 crashes.
Hasegawa et al. (1991)	E = 72	Self-report. (crashes due to seizures).	25 percent of patients had one or more crashes due to seizures while driving.
Takeda et al. (1991)	Uncontrolled E = 858 Controlled E = 855 versus General Population	Prospective Questionnaire Survey. (percent crashes/year).	Uncontrolled = 9.6 percent crashes/yr. Controlled = 5.3 percent crashes/yr. Population = 14.4 percent crashes/yr.
Taylor et al. (1996)	E = 16, 958 Population = 8,888	Questionnaire Survey. (crashes/last 5 years).	E (Odds Ratio) = .95. (95 percent CI = .88 - 1.02).
Diller et al. (1998)	E** (unrestricted) = 33,499 E (restricted) = 1,112 Population = 921,774 (without medical conditions)	Probabilistic linkage of Utah DOT crash files, Utah Master Drivers License File, and Medical Condition Database.	Relative Risk (all crashes). RR (unrestricted) = 2.42. RR (restricted) = 1.74.

E = *Epilepsy*
C = *Controls*
H = *Identified by police following crash and referred for a medical evaluation on suspicion of epilepsy*
SMR = *Standardized Mishap Ratio (estimate of risk in the affected group relative to risk in comparison group).*
* = *Data collected in 1982*
** = *Epilepsy and other episodic conditions (syncope, cataplexy, narcolepsy, hypoglycemia, episodic vertigo)*

control for many individuals with epilepsy. .

Finally, few studies take into account factors such as sex, age, and driving exposure, factors known to affect crash rates in the general driving population. Noteworthy in this regard are the findings from Taylor et al. (1996) showing no differences in crash rates between individuals with epilepsy and those in the general population, once age, gender, driving experience, and mileage were controlled. However, results from that same study indicate that individuals with epilepsy have a 40 percent increased risk of more severe crashes than non-epileptic individuals.

Predictors of Seizure Recurrence

Few studies have investigated predictors in individuals with epilepsy most at-risk for crashes. Those that are available have identified a number of risk factors, which are reviewed below. It is important to note that the studies reviewed below suffer from many of the same methodological limitations discussed previously.

1. Age

Hansotia and Broste (1993) examined the association between a number of potential risk factors and risk of crashes in 241 individuals with a history of seizures. Age was a strong risk factor, with younger epileptic drivers (< 25) having 3.3 times the risk of crashes versus all other epileptic drivers combined.

2. Marital status

After adjusting for age, married epileptic drivers in the Hansotia and Broste (1993) study had one-third to one-half the risk of crashes as unmarried epileptic drivers.

3. Anti-epileptic drug treatment

In the Hansotia and Broste (1993) investigation, individuals receiving anti-epileptic drug treatment had a decreased risk for crashes.

4. Seizure recurrence

It is estimated that up to nine percent of the population will have at least one seizure in their lifetime (McLachlan, 1993), and the risk of convulsive seizure recurrence in individuals presenting with a first grand mal seizure is estimated to be between 23 percent and 84 percent (Berg and Shinnar, 1991; Chadwick, 1991; Hart, Sander, Johnson, and Shorvin, 1990; Hopkins, Graman, and Clarke, 1988). As noted earlier, results from a meta-analysis by Berg and Shinar (1991) indicate that the recurrence rate of seizures for adults is 43 percent.

The risk of seizure recurrence is increased if the previous seizure was focal in origin, if focal or neurologic deficits predated the seizure or following the seizure, or if the seizure is associated with chronic diffuse brain dysfunction, such as Alzheimer 's disease (McLachlan, 1993). A positive family history of epilepsy also increases the risk of seizure recurrence (Hauser, Rich, Annegers, and Anderson, 1990). Importantly, EEG abnormalities are not certain predictors of seizure recurrence. However, the presence of generalized spike waves or focal spikes on EEG recordings increases the risk for seizure recurrence up to 83 percent (Van Donselaar, Schimsheimer, Geerts, and Declerck, 1992).

Not surprisingly, individuals with recurring seizures are a greater risk of having a motor vehicle crash than individuals suffering from a single seizure. Individuals with a history of a single seizure were reported to have approximately one half the crash risk of those with multiple seizures in the investigation by Hansotia and Broste (1993).

A number of studies have investigated the risk of seizure recurrence following withdrawal from anti-epileptic medication, and estimates range from less than 10 percent to almost 70 percent (Berg and Shinar, 1994). Berg and Shinar (1991) performed a meta-analysis of the published literature to determine the risk of relapse at one and two years following discontinuation of anti-epileptic medication. Results of the meta-analysis revealed an overall risk of relapse of 0.25 (95 percent CI = 0.21-0.30) at one year, and 0.29 (95 percent CI = 0.24-0.34) at two years. Risk of relapse varied as a function of age-of-onset. Compared with epilepsy of childhood-onset, epilepsy of adolescent-onset had a relative risk of relapse of 1.79 (95 percent CI = 1.46-2.19). Adult-onset epilepsy was associated with a relative risk of 1.34 (95 percent CI = 1.00-1.81) compared to childhood-onset epilepsy. Individuals with remote symptomatic seizures were more likely to experience seizure relapse than individuals with idiopathic seizures (RR = 1.55, 95 percent CI = 1.21-1.98). Finally, an abnormal EEG was associated with a relative risk of 1.45 (95 percent CI = 1.18-1.79).

5. Type of seizure

There is some evidence to suggest that CPSs are more likely to be associated with a crash than other types of seizures. Gastaut and Zifkin (1987) reported higher crash rates with CPSs compared to nocturnal or idiopathic seizures. In that investigation, CPSs occurred in 81 percent of the epileptics studied and were responsible for 88 percent of the crashes. Van der Lugt (1975) reported that of those individuals involved in a crash due to epilepsy, 76 percent had CPSs. However, the high rate of crashes due to CPSs may be due, in part, to the greater frequency of CPSs in the epileptic population that is of driving age.

Licensing Guidelines for Epilepsy

At one time, most developed countries prevented individuals with epilepsy from holding a driver's license. Regulations, however, have gradually become less restrictive in many countries, such that individuals with epilepsy who have been seizure-free for a specified period of time are now granted driver's licenses. Most states in the United States have restrictions against driving for individuals with epilepsy. However, there is considerable variability in the length of time required for the individual to be seizure-free before license renewal is allowed. In general, the seizure-free requirements range from three months to two years, with one year the most frequent requirement.

In Canada (as of 2000), it is mandatory for physicians in Manitoba, New Brunswick, Newfoundland, Ontario, Prince Edward Island, and the two territories (Yukon and Northwest Territories) to report to the Department of Motor Vehicles individuals with seizures and other medical conditions that might impair one's ability to drive. Physicians may report in British Columbia, Alberta, Saskatchewan, Quebec, and Nova Scotia. In British Columbia, the physician must report only if the patient continues to drive against medical advice. Guidelines, established by the Canadian Medical Association (2000), recommend that driving privileges (private vehicles) be restored to individuals who are seizure-free for 12 months on medication, with the possibility of a reduction to six months on the recommendation of a neurologist.

Conclusions

A review of the extant literature suggests that, in general, individuals with epilepsy have an increased risk for crashes compared to the general driving population. However, it is difficult to determine an exact risk due to the methodological limitations of the current studies. Nevertheless, it is likely the case that certain members of the epileptic population are more at risk for crashes than are others. Research by Hansotia and Broste (1993) provides support for this assumption by identifying a number of factors associated with an increased risk of crashes in individuals with epileptic seizures. An important avenue for future research is to follow the lead of Hanostia and Broste. First, research is needed that would replicate Hansotia and Broste's findings. Second, research on other potential predictors and quantification of the predictive relationships is needed. In this way, decisions about licensing could move from those based on group membership to those based on individual characteristics. Individualized decision-making, based on empirically identified risk factors, could result in enhanced personal mobility for individuals with epilepsy without sacrificing personal and public safety.

Currently, most criteria for re-instating a driver's license in individuals with epilepsy involve extended seizure-free intervals. This, however, leaves unanswered questions of driving competence for those treated with anti-epileptic drugs. It may be that treatment with anti-epileptic drugs reduces the risk of crashes due to seizures, but increases crash risk due to drug-related impairments in mental abilities. Despite extensive research in the last 25 years, the relative effects of anti-epileptic drugs on cognition are controversial (Meador et al., 1995). In general, studies suggest that treatment with anti-epileptic drugs can impair cognitive performance (Dodrill, 1988; Gallassi et al., 1987, 1988; Meador et al., 1995; Pulliainen and Jokelainen, 1994). Recent research (Leach, Girvan, Paul, and Brodie, 1997; Martin et al., 1999) suggests that the newer anti-epileptics drugs (e.g., gabapentin, lamotrigine, and topirmate) produce fewer side effects than older anti-epileptic drugs (e.g., phenytoin, carbamazepine). The significance of the cognitive impairments is, however, unclear. Future studies are needed to examine the effects of anti-epileptic drug therapy on everyday activities such as driving. Although neuropsychological testing might be useful in identifying the presence or absence of cognitive impairments, a more direct approach to evaluating driving competence would be through the assessment of on-road performance.

7.3 Sleep Disorders

Sleep disorders are thought to be responsible for many motor vehicle crashes. However, it is difficult to establish reliable estimates of the contribution of sleepiness to motor vehicle crashes. The difficulty in identifying the role of sleepiness in crashes is due to the multifactorial nature of many crashes, and the lack of objective and reliable measures for assessing driver sleepiness (Pack, Pack, Rodgman, Cucchiara, Dinges, and Schwab, 1995). According to recent estimates, one to three percent of all highway crashes are caused by driver sleepiness (Knipling and Wang, 1994; 1995; Wang, Knipling, and Goodman, 1996; Webb, 1995). Narcolepsy and obstructive sleep apnea are two of the most common medical disorders that cause excessive daytime sleepiness, with obstructive sleep apnea the most common of the two disorders (Arbus, Tiberge, Serres, and Rouge, 1991; National Commission on Sleep Disorders Research Report, 1993). Both are believed to be associated with an increased risk of motor vehicle crashes. Literature relevant to both of these conditions is reviewed below.

7.3a. Narcolepsy

Narcolepsy is a chronic sleep disorder characterized by excessive daytime sleepiness, cataplexy, hallucinations, and sleep paralysis. Nocturnal polysomnograms and a Multiple Sleep Latency Test (MSLT) are used to confirm the diagnosis of narcolepsy. Current estimates suggest that 0.03 percent to 0.16 percent of the general population is affected, with men and women affected equally (Aldrich, 1990; Lyznicki, Doege, Davis, and Williams, 1998). The condition usually starts in adolescence or early adulthood. Treatment of narcolepsy includes the use of stimulants (methylphenidate HCl [Ritalin] or dextroamphetamine) for sleepiness and tricyclic antidepressants for cataplexy and sleep paralysis (Green and Stillman, 1998).

Excessive daytime sleepiness, which can affect driving performance, is generally believed to be the most debilitating of the symptoms (Green and Stillman, 1998). Cataplexy, a sudden episode of muscle weakness triggered by emotions (e.g., laughing, anger, surprise), also may affect driving performance. More than one quarter of all narcoleptics may suffer from cataplexy (Broughton, Ghanme, Hisikawa, Sugita, Nevismalova, and Roth, 1981).

Narcolepsy and Driving Literature Review

Despite the potentially negative impact narcolepsy may have on driving performance, there are few studies investigating the relationship between narcolepsy and driving performance (see Table 17). Aldrich (1989) compared self-reports of crashes from individuals with narcolepsy to those of controls. Results of that investigation revealed that patients with narcolepsy have higher self-reported rates of crashes due to sleepiness than controls. Self-reported crashes were 11 times greater in females with narcolepsy compared to controls and seven times greater in males with narcolepsy compared to controls.

Findley, Unverzagt, Suratt, Gabrizio, Guchyu, and Buckner (1991) compared the performance of nine individuals with narcolepsy with an age- and sex-matched control group on a computer program simulating long and monotonous driving (Steer Clear). The outcome measure was number of obstacles hit during a 30 minute testing session. Results revealed that the subjects with narcolepsy hit more obstacles (6.4 percent ± 3.2 percent) than the control group (0.8 percent ± 0.5 percent). Comparisons also were made between level of performance on Steer Clear and state recorded crash rates for a five-year period for individuals with narcolepsy and obstructive sleep apnea. The results revealed significantly higher crash rates in subjects with poorer performance on Steer Clear. The study, however, has a number of limitations. First, the sample size is small. Second, cut-off criteria for performance categories (normal, poor, and very poor performance) are not specified. Finally, it is unclear from the data the percentages of narcoleptic subjects and sleep apnea subjects exhibiting normal performance, poor performance, and very poor performance.

George, Boudreau, and Smiley (1996) investigated the performance of narcoleptics and controls on a divided attention driving test (DADT) involving tracking and visual search. Individuals with narcolepsy made signifi-

Table 17 Summary of Studies Examining the Relationship between Crashes and Narcolepsy and Simulator Performance and Narcolepsy

Study	n	Methodology	Results
Aldrich et al. (1989)	Narc = 56 C = 70	Self-report a. Crashes anytime in lifetime. b. Crashes due to sleepiness.	a) Males: 76 percent (Narc) at least one crash versus 79 percent (C). Females: 48 percent (Narc) at least one crash versus 74 percent (C). b) Males: 52 percent (Narc) versus 11 percent (C). Females: 74 percent (Narc) versus 6 percent (C).
George et al. (1996)	Narc = 16 C = 21	DADT a. Tracking Errors. b. Visual Search. i) Correct responses ii) RT (secs).	a) Narc = 196 (± 146) versus 71 (± 32) (C). b) i. Narc = 35.3 (± 6.2) versus 38.4 (± 2.5) (C). ii. Narc = 3.3 (± 1.1) versus 2.9 (± 0.8) (C).
Findley et al. (1991)	Narc = 9 C = 9	Steer Clear -obstacles hit	Narc = 6.4 percent ± 3.2 percent. C = 0.8 percent ± 0.5 percent.

Narc = Narcolepsy
C = Controls
DADT = Divided Attention Driving Test

cantly more tracking errors than controls. The differences between the two groups on the visual search test were less disparate. The pattern of findings suggests that individuals with narcolepsy have greater difficulty dividing attention compared to controls. However, baseline measures while performing each of the tasks separately were not available for either the narcoleptic group or the control group. Thus, the cost of performing both tasks together for each of the groups, and the possibility of cost differences between the two groups cannot be determined. Unknown also is the relationship between performance on laboratory based tests, such as the DADT, and on-road performance. Research exploring the relationship between the two is needed.

Despite the paucity of research in this area, most medical associations and driving agencies (Canadian Medical Association, 2000; The British Columbia Medical Association, 1997; Canadian Council of Motor Transportation Administrators, 1994) recommend that an individual who suffers from attacks of narcolepsy should not be allowed to drive. The Canadian Medical Association (2000) specifically recommends that individuals with a diagnosis of narcolepsy supported by a sleep study and with uncontrolled episodes of cataplexy during the past 12 months (with or without treatment) not drive any type of motor vehicle. It also is recommended that those with a diagnosis of narcolepsy supported by a sleep study and with uncontrollable daytime sleep attacks or sleep paralysis during the past 12 months (with or without treatment) not drive any type of motor vehicle.

7. 3 b. Sleep Apnea

Obstructive sleep apnea is a common disorder, affecting between two to four percent of the population (Young, Palta, Dempsey, Skatrud, and Badr, 1993). Prevalence rates are higher in middle aged and older adults, and obese individuals (National Heart, Lung, and Blood Institute Working Group on Sleep Apnea, 1996; Partinen, 1994; Strollo and Rogers, 1996).

With sleep apnea, the upper airway repetitively collapses during sleep, resulting in sleep fragmentation, nocturnal hypoxemia, and lack of slow wave sleep. Cognitive impairments are thought to be frequent (Guilleminault, Van den Hoed, and Mitler, 1978; Strohl, Saunders, and Sullivan, 1984), with attention and concentration difficulties and impairments in vigilance the most common (Bédard, Montplaisir, Richer, and Malo, 1991; Findley et al., 1986; Greenberg, Watson, and Depotual, 1987). The cognitive deficits are believed to be due to hypoxemia during sleep, disruptions during sleep, and/or abnormal brain blood flow during wakefulness (Bédard et al., 1991; Guillemineault, Partinen, and Quera-Salva, 1988; Orr et al., 1979; Poceta, Jeong, Ho, Timms, and Mitleer, 1990). However, more research is needed to identify the underlying mechanism(s) responsible for the cognitive impairments.

Nocturnal polysomnography is used to confirm the diagnosis of obstructive sleep apnea. With polysomnography, a number of physiological indices are monitored, including EEG, respiration, ECG, and oxygenation (American Thoracic Society, 1989). Generally, an individual is diagnosed with sleep apnea if they have greater than 10 apnea/hypopneas per hour of sleep (API). Apnea is defined as cessation of airflow lasting at least 10 seconds. Hypopnea is defined as a reduction in airflow lasting 10 seconds and is usually associated with a decline in blood oxygen level.

A number of treatments are available for sleep apnea including weight loss, alcohol abstinence, nasal continuous positive airway pressure (CPAP), and nasal and upper airway surgery (uvulopalato-pharyngoplasty). CPAP is the most common treatment. Reduction in daytime sleepiness often is reported immediately with CPAP treatment, although studies indicate that approximately six weeks of treatment are required for maximum improvement in symptoms (Lamphere, Roehers, Witteg, Zorik, Conway, and Roth, 1989). Compliance rates differ as a function of measurement: subjective rates of patient compliance are higher than objectively determined values. Based on objective measures, Kribbs et al. (1993) reported acceptable compliance rates of 46 percent in patients treated with CPAP. Acceptable compliance was defined as the use of the CPAP machine for more than four hours per night for more than 70 percent of the observed nights.

Sleep Apnea and Driving Literature Review

A number of studies (see Table 18) have investigated the relationship between obstructive sleep apnea and motor vehicle crashes with results revealing a two- to three-fold increase in crashes in individuals with sleep apnea compared to controls.

There are a number of methodological limitations to those studies. The sample size in one half of the studies (Findley et al., 1988; 1989; George, Nickersen, Hanly, Millar, and Kryger, 1987) is small. In those studies with a larger sample size, the data are based on retrospective self-reports (Aldrich, 1989; Gonzalez-Rothi, Foresman, and Block, 1988, but see Barbé et al., 1998). In one study (George et al., 1987), the diagnosis of sleep apnea was not confirmed by polysonography in seven of the study participants. According to the authors, if any of these seven patients had a condition such as narcolepsy or idiopathic hypersomnia, their results would lose significance. In the most recent study (Barbé et al., 1998), the sample size is relatively large, with reports of crashes

Table 18 Summary of Studies Examining the Crash Rates of Drivers with Obstructive Sleep Apnea

Study	n	Methodology	Results
George et al. (1987)	SA = 27 C = 270	Driving Records (time period not specified).	Two-fold higher crash rate for SA versus Controls. (Mean crash rate SA = 2.63, C = 1.28).
Findley et al. (1988)	SA = 29 NSA= 35 LD = 3.7 million	Driving Records (crashes/driver/5 years).	Seven-fold higher crash rate for SA (0.41) versus NSA (0.06). 2.6 fold higher crash rate for SA(0.06) versus LD (0.16).
Aldrich (1989)	SA = 228 C = 70	Self-report a. Crashes anytime in lifetime. b. Crashes due to sleepiness.	a) Males: 71 percent (SA) at least one crash versus 79 percent (C). Females: 68 percent (SA) at least one crash versus 74 percent (C). b) Males: 19 percent (SA) versus 11 percent (C). Females: 15 percent (SA) vs 6 percent (C).
Findley et al. (1989)	*$SA_{(mild)}$ = 16 $SA_{(mod)}$ = 17 $SA_{(severe)}$ = 13 LD = 3.7 million	Driving Records (crashes/driver/5 years).	LD = 0.16 $SA_{(mild)}$ = 0.13 $SA_{(mod)}$ = 0.24 $SA_{(severe)}$ = 0.46
Gonzalez-Rothi et al. (1988)	SA = 78 C = 28	Self-report (near miss or crash).	4.5 fold higher near miss or crash for SA (32 percent) versus C (7 percent).
Barbé et al. (1998)	SA = 60 C = 59	Insurance Company Crash Data (percent of drivers with at least one crash in last 3 years).	SA (Odds Ratio) = 2.3 versus Controls. SA (Odds Ratio) = 2.6 versus Controls (one or more crashes after controlling for mileage).

SA = Sleep apnea *NSA = Non-sleep apnea (subjects referred for evaluation of sleep apnea with normal sleep studies)*
LD = All licensed drivers in Virginia *C = Controls*
** Classified according to severity of nocturnal hypoxemia associated with apnea*

based on insurance company crash data. As noted by the authors, insurance company crash data are an objective source of data (although under-reporting may be a problem). However, in this study, data regarding motor vehicle crashes were included for a three-year period preceding the diagnosis of sleep apnea. Thus, crashes unrelated to sleep apnea may have been included in their statistics.

An additional limitation in studies investigating the relationship between sleep apnea and crashes is the lack of uniform diagnostic criteria. For example, in three of the studies listed in Table 18 (Findley et al., 1989; George et al., 1987; Gonzalez-Rothi et al., 1988), diagnostic criteria for sleep apnea are not specified. Barbé et al. (1998) defined sleep apnea as greater than 20 apnea/hypopnea episodes per hour of sleep as

measured by polysomnography. Individuals included in the study by Aldrich (1989) all underwent polysomnography, with a respiratory disturbance index calculated based on the number of apneas or hypopneas per hour of sleep. Participants were then classified into four diagnostic groups, ranging from sleep apnea to other disorders of excessive daytime sleepiness. However, the criteria used for group classification were unspecified. Lastly, subjects diagnosed as having sleep apnea in the study by Findley et al. (1988) met the criteria of at least five obstructive sleep apneas or hypopneas per hour of sleep, which resulted in a drop in baseline oxyhemoglobin saturation of four percentage points.

Finally, in the majority of studies, degree of driving exposure is not taken into account. Therefore, it is not clear if the elevated crash rates of sleep apnea individuals represent an overestimation or underestimation of risk compared to controls. In the one study where driving exposure is considered (Barbé et al., 1998), individuals with sleep apnea reported driving more kilometers per year (~27,000) than controls (~16,000). The reason for the significantly higher exposure per year in individuals with sleep apnea compared to controls is unclear. The patients and controls were matched for sex and age. The authors note that a selection bias is unlikely to account for the findings as an equal percentage of patients and controls came from areas surrounding the hospital. One possible explanation may be in terms of occupational differences. Although data on occupation were gathered in the Barbé study, those data were not reported. It may be that occupational differences account for the higher driving exposure of the sleep apnea patients. The findings from the Barbé et al. study are noteworthy in that the magnitude of the increased crash rates of sleep apnea patients persisted after controlling for number of kilometers driven per year.

The studies reported above suggest that individuals with sleep apnea are at increased risk of crashes. However, more research is needed using standardized diagnostic criteria, larger sample sizes, driving exposure, and objective measures of driving performance (e.g., crash data, on-road performance) gathered from time of diagnosis onward.

Sleep Apnea and Other Measures of Performance

Researchers have attempted to identify performance deficits associated with sleep apnea that may have relevance for driving. In that regard, studies have been done comparing the performance of individuals with sleep apnea to that of controls on driving simulators, computer programs simulating aspects of driving (Steer Clear), divided attention driving tasks (DADT), and the PVT (Psychometer Vigilance Test device). The results of those studies are summarized in Table 19.

In the first study listed in Table 19, Findley et al. (1989) compared the performance of patients with sleep apnea to that of age- and sex-matched controls. Performance was assessed by means of a computer program (Steer Clear). This program graphically displays a moving automobile, a two-lane highway, and intermittent obstacles in the highway. Subjects are to avoid hitting obstacles by changing lanes with a single computer key. The outcome measure is the number of obstacles hit during the 30-minute 'simulated drive'. Performance also was assessed by means of a Doran driving simulator, with number of steering, signaling, braking, accelerating, and speeding errors measured. The outcome measure was a percentage correct score. As can be seen in Table 19, subjects with sleep apnea performed significantly poorer in terms of obstacles hit (Steer Clear) and in terms of percentage correct (Doran Simulator score) compared to controls. In a later study, Findley et al. (1991) examined the relationship between performance on Steer Clear and crash rates. Results of that investigation revealed that individuals with very poor performance on Steer Clear had higher crashes/driver/5 years (0.38) than individuals with poor performance (crashes/driver/5 years = 0.20) or those with normal performance (crashes//driver/5 years = 0.05). However, as noted earlier, individuals with sleep apnea and narcolepsy were included in the sample. It is unclear from the data the percentage of sleep apnea patients and narcoleptic patients included in each of the above categories. Also unclear is the criterion used for performance classification. It is interesting to note that results from this investigation revealed that 31 percent of the patients with sleep disturbances (narcolepsy or sleep apnea) performed within normal limits on Steer Clear.

George et al. (1996) compared the performance of sleep apnea patients with that of age-matched controls on a divided-attention driving task (DADT). Tracking errors and visual search measures (correct responses and reaction time) were measured. There were significant differences between the two groups on tracking errors, with the sleep apnea patients exhibiting a three-fold increase in errors. Although the results from the visual search measures were statistically significant, there was considerable overlap in the measures between the two groups (see Table 20 below).

Barbé et al. (1998), in a retrospective controlled study, compared the performance of sleep apnea patients with age- and sex-matched controls on a vigilance task (PVT: Psychometer Vigilance Test) and on Steer Clear. As can be seen in Table 20, individuals with sleep apnea

Table 19 Summary of Studies Examining Differences in Performance on Simulators and Other Performance Measures Between Individuals with Sleep Apnea (SA) and Controls (C)

Study	N	Test	Measures	Results
Findley et al. (1989)	a. SA = 6 C = 7 b. SA =12 C = 12 c. $SA_{(UT)}$ = 6 $SA_{(T)}$ = 6 C = 12	a. Steer Clear. b. Driving Simulator. c. Steer Clear.	a. Obstacles hit (in 30 minutes). b. percent correct score. c. Obstacles hit (in 30 minutes).	a. SA = 44 C = 7. b. SA = 39 percent C = 58 percent. c. $SA_{(UT)}$ = 29 $SA_{(T)}$ =13 C = 9.
Findley et al. (1995)	SA & N = 68 <u>Steer Clear Perform:</u> Normal = 21 Poor = 25 V. Poor = 22	Steer Clear Crash rate (crashes/driver/ 5 years).	# obstacles hit used for categorization (Normal, Poor, V. Poor).	Crashes/driver/5 years Normal = 0.05 Poor = 0.20 V. Poor = 0.38.
George et al. (1996)	SA = 21 C = 21	DADT.	a. Tracking errors b. Visual Search i) correct responses ii) RT (secs).	a. SA = 228 (± 145) versus 71 (± 32) (C) b. i. SA = 36.2 ± 4.2 versus 38.4 ± 2.5 (C) ii. SA = 3.2 ± 0.8 versus 9 ± 0.8 (C)
Barbé et al. (1998)	SA = 60 C = 60	a. PVT. b. Steer Clear.	a. i. RT (msec). ii. RF (msec-1/min). b. Obstacles hit. c. i. PVT and crash rates ii. Steer Clear and crash rates.	a. i. SA = 283 (± 6) versus 262 (± 5) (C) ii. SA = -0.04 versus -0.03(C)$^{(NS)}$ b. SA = 0.4 (± 0.1) vs 2 (± 0.5) (C) c. i. NS ii. NS

SA = Sleep Apnea
PVT = Psychometer Vigilance Test device
$SA_{(T)}$ = Sleep apnea, Treated with CPAP
DADT = Divided Attention Driving Task
$SA_{(UT)}$ = Sleep Apnea, Untreated
RF = Reaction Fatigue
N = Narcolepsy
RT = Reaction Time
NS = Not Significant

performed significantly worse than controls (with the exception of the reaction fatigue measure). It is important to note, however, that despite the fact that the differences between the two groups were statistically significant, those differences are unlikely to be clinically meaningful because of the degree of overlap in the measures between the two groups. Importantly, the authors found no relationship between patient measures on Steer Clear and the vigilance task (PVT) or between Steer Clear measures and crash data from insurance companies.

In summary, although individuals with sleep apnea perform significantly worse than controls on laboratory based tests, it is unclear how those findings translate to real world driving performance. For example, Findley et al. (1991) found higher crash rates in individuals with either narcolepsy or sleep apnea who performed very poorly on Steer Clear compared to sleep apnea and narcoleptic individuals performing either poorly or normally on Steer Clear. However, sample sizes for each of the categories were small. Barbé et al. (1998), using a larger sample size, found no relationship between a

number of vigilance measures and crash rates. In both studies, the time periods for crash rate measurement were not assessed as a function of sleep apnea diagnosis. Future studies with larger sample sizes and more clearly defined diagnosis-crash parameters are needed.

Relationship Between Measures of Disease Severity and Crashes and/or Simulator Measure

A handful of studies have examined the relationship between measures of disease severity and crashes, and/or driving simulator measures in individuals with sleep apnea. The results of those studies are summarized in Table 20.

Researchers that have examined the relationship between the AHI (apnea/hypopexmia index) and performance have found little, if any, relationship between AHI and Steer Clear scores (Flemons, Remmers, and Whitelaw, 1993), tracking errors and visual search measures (George, Boudreau, and Smiley, 1996), or crashes (Barbé et al., 1998). Measures of daytime somnolence also have been found to be unrelated to crash rates (Aldrich, 1989; Barbé et al., 1998; Flemons et al., 1993). Other measures of disease severity (e.g., O2 desaturation) or clinical measures (e.g., anxiety, depression, etc.) also have been found to be unrelated to crash data or simulator measures (Barbé et al., 1998; Flemons et al., 1993). Taken together, the results reveal that measures commonly used to measure disease severity in sleep apnea are not very useful in discriminating between individuals who are likely to perform poorly on laboratory based measures putatively related to driving performance or who are at-risk for crashes.

Conclusions

The literature reviewed above suggests that both narcolepsy and obstructive sleep apnea may be associated with impaired driving performance. However, the limitations in the current literature are such that recommendations regarding licensure are likely to be based on an inadequate knowledge base. As noted by the

Table 20 Summary of Studies Examining the Relationship Between Measures of Disease Severity and Crashes and/or Simulator Measures

Study	n	Measure	Results
Aldrich (1989)	SA = 180	MSLT and self-reported crash rates. (Crashes due to sleepiness).	MSLT (no crash) = 8.2. MSLT (crash) = 7.8 (Not significant).
Flemons et al. (1993)	SA = 180	Correlation between Steer Clear Score and ___ a. AHI. b. ARI. c. O2 desaturation. d. Sleepiness scale. e. Daytime somnolenece. f. Age. g. Snoring. h. Self-reported crashes.	All measures uncorrelated with Steer Clear score.
George et al. (1996)	SA = 21	a. AHI and tracking errors. b. AHI and response time. c. AHI and correct targets. d. MSLT and tracking time.	a. r = .07. b. r = .05. c. r = .11. d. r = -.42.
Barbé et al. (1998)	SA = 60	Correlation between crash data (insurance records) and: a. AHI. b. SaO2. c. Reaction fatigue. d. Epworth Sleepiness Scale. e. Anxiety score. f. Depression score.	No relationship between measures and crash data.

MSLT = Multiple Sleep Latency Test *ARI = Arousal Index*
AHI = Apnea/Hypopnea Index *SA = Sleep Apnea*

Table 21 Guidelines for Diseases of the Nervous System (Reproduced with permission)

Guidelines for Diseases of the Nervous System (Drivers of Private Vehicles)		
Condition/ lllness	**Austroads (1998)**	**CMA (2000)**
Seizures		
Auras	Not addressed.	Drive if there is no impairment in level of consciousness and cognition, the seizures are unchanged for more than one year, and a neurologist approves.
Single, Unprovoked Seizure Before a Diagnosis	Isolated Seizure Should not drive for 3-6 months. Consultant opinion recommended.	Not drive for at least 3 months and until a complete neurological examination including EEG and CT have been conducted to determine cause.
After Epilepsy Diagnosis	Recently Diagnosed Epilepsy: 1. Should not drive until seizure free for 3-6 months from start of therapy. 2. Consultant opinion recommended. A conditional license may be issued by the DLA on medical advice.	Drive if: 1. 12 months seizure free and on medication* 2. Physician has insight into patient compliance. 3. Physician cautions against fatigue, alcohol use. **The 12- month period may be reduced to 6 months on the recommendation of a neurologist.*
After Surgery to Prevent Epileptic Seizure	Not addressed.	Resume driving if 12 months seizure free after surgery* **The 12-month period may be reduced to 6 months on the recommendation of a neurologist.*
Chronic Epilepsy (History of Previously Controlled Seizures)	1. Should not drive for up to 2 years. 2. Consultant opinion recommended. A conditional licence may be issued by the DLA on medical advice.	See 'After Epilepsy Diagnosis' section.
Medication Withdrawal or Change a) Initial Withdrawal or Change b) If Seizures Recur After Withdrawal or Change c) Long-Term Withdrawal and Discontinuation of Medication	a) When consultant opinion suggests significant risk of recurrent seizure, driving should cease during with drawal and for at least 3 months thereafter. b) Should not drive for 1 month after resuming previously effective medication or for 2 years if refusing to resume medication. c) Not addressed.	a) Not drive for a period of 3 months from the time medication has been discontinued or changed. b) Resume driving if takes medication according to the physician's instructions and is seizure free for 6 months (can reduce period if neurologist agrees). Drive any vehicle if seizure-free off medication for 5 years.
Alcohol Withdrawal Induced Seizures	Not addressed.	a) May drive if individual: remains alcohol-free and seizure-free for 12 months, and b) Completes a recognized rehabilitation program for substance dependence.

Table 21 Guidelines for Diseases of the Nervous System (*continued*)

Syncope	Unpredictable, spontaneous loss of consciousness is incompatible with safe driving. A single episode of syncope/fainting of unknown cause renders an individual unable to drive for at least 4 weeks after the event. Recurrent events require a specialist opinion.	A single occurrence that is fully explained and unlikely to recur may require no more than careful observation. Individuals who have a history of a number of fainting spells or repeated unexplained falls should not drive until the cause has been determined and corrective measures taken.
Sleep Disorders		
Narcolepsy	Should not drive until disorder fully investigated and treated and report considered by DLA.	Patients with a diagnosis of narcolepsy supported by a sleep study and with an episode of cataplexy in the past 12 months (with or without treatment) *should not drive any type of motor vehicle.*
Sleep Apnea	Should not drive. DLA may issue conditional licence if condition stable. Annual review and neurologist opinion recommended.	Obstructive Sleep Apnea: Patients with obstructive sleep apnea documented by a sleep study that are compliant with CPAP and UPPP treatments should be safe to drive any type of motor vehicle. Patients with moderate to severe obstructive sleep apnea documented by sleep study who are non-compliant with treatment should not drive any type of motor vehicle. Patients with obstructive sleep apnea who are believed compliant with treatment and who are subsequently involved in a motor vehicle crash should not drive for at least 1 month after the motor vehicle crash. During this one- month period, their compliance must be reassessed. After the one-month period, they may or may not drive depending on the results of reassessment.

CPAP = Continuous Positive Airway Pressure *UPPP = Uvulopalato-pharyngoplasty* *DLA = Driver Licensing Authority*

American Thoracic Society (1994), sleep apnea is "undoubtedly a risk factor, but is not invariably linked with impaired driving" (p. 1466). Thus, "efforts to reduce excessive driving risk should most sensibly be directed at selected patients with excessive daytime sleepiness, rather than categorically applied to anyone with apnea or with a certain number of sleep apnea events" (p. 1466). For example, results from Findley et al. (1988) reveal that more than two thirds of individuals with sleep apnea were crash free during a five-year study period. Currently used measures of disease severity are inadequate in identifying those individuals with sleep apnea most at-risk for motor vehicle crashes. Research is needed that will identify those individuals with sleep apnea who are most at-risk for motor vehicle crashes. Finally, research examining the relationship between simulated driving performance and on-road performance is needed. Currently, that relationship is not well defined.

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7.1 Syncope (See Section 4: Cardiovascular Diseases)

7.2 Seizures

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7.3 Sleep Disorders

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Section 8: Respiratory Diseases

8.1 Asthma
8.2 Chronic Obstructive Pulmonary Disease
8.3 Other Pulmonary Conditions

A number of respiratory diseases may interfere with the safe operation of a motor vehicle by causing reduced oxygen flow to the brain and subsequent cognitive impairment (e.g., impairments in judgment, decision making, attention). Respiratory diseases pertinent to driving include asthma, chronic obstructive pulmonary disease, and carcinoma of the lung to name but a few.

A summary of the current fitness-to-drive guidelines (Respiratory Diseases) for medical practitioners from Australia (1998) and Canada (2000) is presented in Table 23.

8.1 Asthma

Asthma is a chronic lung disease characterized by recurrent breathing problems (e.g., wheezing, shortness of breath, chest tightness). Asthma is caused by inflammation of the lower airways. Triggers of asthma attacks include irritants in the air, respiratory infections, exercise, and changes in weather.

Prevalence

Recent statistics suggest that more than 14.6 million Americans suffer from asthma (Vital and Health Statistics, December 1995). Approximately 10 million of those with asthma are 18 years of age or older. In 1994, 5.4 percent of Americans reported having asthma, a 75 percent increase since 1980 (Morbidity and Mortality Weekly Report, 1998).

Asthma and Driving Literature Review

There are no studies known to the author examining the relationship between asthma and motor vehicle crashes.

8.2 Chronic Obstructive Pulmonary Disease

Chronic obstructive pulmonary disease (COPD) refers to diseases involving obstructed airflow, such as peripheral airway disease, emphysema, and chronic bronchitis. Emphysema and chronic bronchitis frequently coexist and the term COPD is often applied to individuals suffering from these two disorders. Importantly, asthma typically is not included under the COPD categorization. Complications of COPD include chronic hypercabia, cor pulmonale (hypertrophy of the right ventricle), supraventricular and ventricular tachyarrhythmias, sleep hypoexmia, and acute respiratory failure (King, Jr., 1990).

Prevalence

Recent statistics suggest that approximately 16.4 million Americans suffer from COPD, which is the fourth leading cause of death in America (National Institute of Health, 2000). It is the only leading cause of death in America that is increasing in prevalence (Higgins, 1989). Based on the National Health Interview Survey, 1982-1995, the estimated number of individuals reporting they have chronic bronchitis has increased dramatically in recent years: from 7.7 million in 1982 to 14.5 million in 1995, an increase of 88 percent (NIH, 2000). Overall, the reported prevalence has increased 64 percent between 1982 and 1995: from 33.9 per 1,000 to 55.5 per 1,000 (NIH, 2000).

Chronic bronchitis affects individuals of all ages. Emphysema, on the other hand, is more common among elderly individuals. In 1995, based on the National Health Interview Survey 1982-1995, the reported prevalence rate for emphysema was 7.1 per 1,000 persons, a 30 percent decrease over that reported in 1982 (NIH, 2000).

COPD and Driving Literature Review

As with asthma, there are no studies available that have examined explicitly the relationship between COPD and motor vehicle crashes. Because of the paucity of research regarding COPD and crashes, decisions regarding driving competence for individuals with COPD, as with many other chronic conditions, must be based on an evaluation of the effects of the condition on driving competence (e.g., direct driving performance measures) rather than on relative risk data.

Cognitive impairment, resulting from chronic hypoxemia, is the primary issue for driving competency in individuals with COPD. A number of researchers have examined cognitive performance in individuals with COPD (Fix, Golden, Daughton, Kass, and Bell, 1982; Grant, Prigatano, Heaton, McSweeny, Adams, and Timms, 1982; Huppert, 1982; Incalzi et al., 1997; Isoaho, Puolijoki, Huhti, and Laippala, 1996; Incalzi, Chiappini, Fuso, Torrice, Gemma, and Pistelli, 1998; Kozora, Filley, Julian, and Cullum, 1999; Prigatano et al., 1983; Stuss, Peterkin, Guzman, Guzman, and Troyer, 1997). In general, the overwhelming majority of studies have found cognitive deficits in individuals with COPD, with impairments greater on the more complex and demanding cognitive tasks.

Two studies worthy of further description are those conducted by Grant et al. (1982) and Prigatano et al. (1983). Both studies were large multicenter trials in United States and Canada. Each study included extensive neuropsychological and pulmonary function testing in patients with COPD and a group of non-patient demographically matched controls. The first study (Grant et al., 1982), termed the Nocturnal Oxygen Therapy Trial (NOTT), included 203 patients who were moderately to severely hypoxic (mean age = 64 years, mean PaO2 = 51 mm Hg). Results revealed that patients with COPD performed significantly worse than controls on almost all neuropsychological tests. Forty two percent of the patients exhibited moderate to severe neuropsychological test performance compared to 14 percent of controls. Higher cognitive functions were the most severely affected. Correlations between neuropsychological test performance and pulmonary function measures were, however, disappointingly low. As noted by the authors, a restriction of range on some of the key medical variables most likely accounts for the findings.

The second multicenter investigation was conducted by Prigatano et al. (1983) and was called the Intermittent Positive Pressure Breathing (IPPB) Trial. One hundred patients with COPD (mean age = 62, mean PaO2 = 66 mm Hg) underwent extensive neuropsychological and pulmonary function testing (identical with those of the Grant et al., 1982 investigation), with results compared with demographically matched controls tested on the same measures. The COPD patients in this investigation were classified as more mildly impaired than those in the Grant et al. (1982) investigation. Nevertheless, the mildly impaired patients were found to have selective neuropsychological impairments compared to controls. Again, the correlations between neuropsychological test performance and pulmonary function measures were disappointing.

In a more recent report, Grant, Prigatano, Heaton, McSweeny, Wright, and Adams (1987) merged the databases from these two large multicenter trials. Importantly, the merged database addressed some of the limitations inherent in the independent databases: notably the restriction of range in key medical variables of interest. Thus, the merged database allowed for the analysis of results from a much larger and more representative database. The sample consisted of 86 mildly hypoxic (PaO_2 > 60 mm Hg), 155 moderately hypoxic (PaO_2 = 50-59 mm Hg), and 61 severely hypoxic (PaO_2 <50 mm Hg) COPD patients and 99 age- and education-matched non-patients. Results indicated that the rate of neuropsychological deficits rose from 27 percent in those with mildly hypoxemia to 61 percent in those with severe hypoxemia. One category of neuropsychological tests (based on factor analysis) was the most effective in discriminating between the study groups. Specifically, tests reflecting perceptual learning and problem solving were effective in separating the groups: Controls and mildly hypoxic patients were similar in performance, moderately hypoxic patients were significantly worse than controls or mildly hypoxic patients, and severely hypoxic patients were the most impaired. Results from multiple regression indicated that age and PaO2 were significant predictors of the perceptual learning and problem scores. Finally, logistic regression results indicated that there were three predictors of neuropsychological impairment versus non-impairment. Those predictors were age, education, and PaO_2. Higher age, lower levels of education, and lower PaO_2 levels were associated with impairment.

Results from Grant et al. (1987) suggest that categorization of COPD patients in terms of disease severity is an important factor for determining the presence or absence of cognitive impairment. In an earlier study, Grant et al. (1982) developed a summary index of medical disease for the patients with COPD. The Severity of Disease Index is based on five key variables (forced expiration volume, maximum exercise tolerance, heart rate, mean pulmonary artery pressure, and resting arterial oxygen saturation), and rated on a four point scale with a higher rating (4) indicative of a severely abnormal result. The overall disease severity index has a range of 5 to 20, with the upper end of the scale reflective of more severe disability. The psychometric properties of the Severity of Disease Index were not provided and perhaps are not available. This is unfortunate as this type of scale may have considerable utility for evaluating disease severity. Important next steps would be to validate the scale against the type and extent of cognitive impairment, and, for fitness-to-drive goals, to validate it against defensible driving performance measures.

Criteria for the evaluation of respiratory impairment/disability also have been published by the American Thoracic Society (1986). In their statement, the American Thoracic Society recommends that evaluation of respiratory impairment include both forced vital capacity (FVC) and forced expiratory volume in the first second (FEV1). Categorization of patients as to degree of impairment is listed in Table 22. As with the Severity of Disease Index, psychometric properties of the Ratings of Impairment were not reported. Important next steps would be to evaluate the utility of the ratings in predicting cognitive impairment, and to validate the ratings against defensible driving performance measures.

Table 22 Ratings of Impairment in Individuals with Respiratory Disorders (based on information from *The American Thoracic Society (1986). Evaluation of impairment/disability secondary to respiratory disorders. American Review of Respiratory Disorders, 133, 1205-1209)*

Rating	Pulmonary Function Testing
Normal	FVC ≥ 80 percent of predicted *and* FEV_1 ≥ 80 percent of predicted, *and* FEV_1/FVC x 100 ≥ 75 percent *and* D_{LCOsb} ≥ 80 percent of predicted
Mildly Impaired. (Usually not correlated with diminished ability to perform most jobs).	FVC ≥ 60 to 70 percent of predicted, *or* FEV_1 ≥ 60 to 79 percent of predicted, *or* FEV_1/FVC x 100 60 to 74 percent *or* D_{LCOsb} 60 to 79 percent of predicted.
Moderately Impaired. (Progressively lower levels of lung function correlated with diminished ability to meet the daily demands of many jobs).	FVC 51 to 59 percent of predicted *or* FEV_1 41 to 59 percent of predicted, *or* FEV_1/FVC x 100 41 to 59 percent *or* D_{LCOsb} 41 to 59 percent of predicted.
Severely Impaired. (Unable to meet the physical demands of most jobs including travel to work).	FVC 50 percent or less of predicted *or* FEV_1 40 percent or less of predicted, *or* FEV_1/FVC x 100 ≥ 40 percent or less *or* D_{LCOsb} ≥ 40 percent or less of predicted.

FVC = *Forced vital capacity*
FEV_1 = *Forced expiratory volume in first second*
FEV_1/FVC x 100 = *Using the previously selected values for FVC and FEV1, compute the ratio and express as percentage*
D_{LCOsb} = *Single breath diffusing capacity*

In conclusion, although the literature is sparse, that which is available suggests that individuals with COPD are at higher risk of cognitive impairment compared to age matched controls. Clearly, the presence of cognitive impairment places the individual at-risk for motor vehicle crashes. Future research, focusing on predictors of cognitive decline in this population, is needed. For the present, decisions regarding fitness-to-drive should be made on an individual basis, with determinations of driving competence based on cognitive and/or on-road assessments.

8.3 Other Pulmonary Conditions

Evidence from a study conducted in Utah indicates that individuals with pulmonary conditions (conditions not specified) are at a higher risk for crashes (Diller et al., 1998-see Section 2.1 a., page 4 for details of the study). With respect to pulmonary conditions, the driving records of 5,055 drivers with unrestricted licenses and 572 drivers with restricted licenses were compared to controls. Unrestricted drivers had a significantly higher relative risk for crashes (RR = 1.96, CI = 1.80 - 2.14). The results for the restricted drivers were non-significant (RR = 0.65, CI = 0.39 - 1.10).

Table 23 Guidelines for Respiratory Diseases *(Reproduced with permission)*

Illness	Austroads (1998)	CMA (2000)
Asthma	No restrictions if well controlled and no significant side effects from medications. Should not drive for 2 weeks following admission to an Intensive Care Unit or if experienced Loss of Consciousness.	Not addressed.
Carcinoma of Lung	Not addressed.	Not addressed.
Chronic Obstructive Pulmonary Disease (COPD)	Can drive if well-controlled and no significant side effects from the condition or medication.	No or Mild Impairment Can usually drive. Moderate or Severe Impairment Driving permitted. Moderate Impairment requiring supplemental O_2. Road test with supplemental O_2. Remain under close and regular supervision.
Oxygen Therapy	Not addressed.	See Moderate Impairment with O_2 guidelines above.
Post-Thoracotamy	Should not drive for 4 weeks post-surgery.	Not addressed.
Recurrent Pneumothorax	Should not drive for 2 weeks post pneumothorax unless cleared by physician.	Not addressed.
Respiratory Failure	Should not drive if becomes significantly dyspneic when walking on level surface.	See COPD guidelines for degree of impairment.
Tracheostomy	May drive if clinically stable.	Should be able to drive if individual has no difficulty keeping the opening clear of mucus provided that the medical condition necessitating the tracheostomy does not preclude driving.
Tuberculosis	May drive.	Not addressed.

DLA = Driver Licensing Authority

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Section 8.1 Asthma

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Section 8.3 Other Pulmonary Conditions

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Section 9: Metabolic Diseases

9. 1 Diabetes Mellitus
9. 2 Thyroid Disease
    9.2.a. Hyperthyroidism
    9.2.b. Hypothyroidism

A summary of the current fitness-to-drive guidelines (Metabolic Diseases) for medical practitioners from Australia (1998) and Canada (2000) is presented in Table 29.

9.1 Diabetes Mellitus

Prevalence

Diabetes mellitus, one of the most common endocrine diseases, affects approximately 16 million Americans (Centers for Disease Control [CDC], 1998). Prevalence rates in the United States range from two percent to six percent (CDC, 1998; National Institutes of Health [NIH], 1995). The number of people diagnosed with diabetes increased five-fold between 1958 and 1993 (NIH, 1995). Statistics reveal that the prevalence of diabetes increases with age, and recent estimates are that 18 percent to 20 percent of those 65 and over in the United States have diabetes (CDC, 1998).

Typically, the disease is categorized into two forms: insulin-dependent diabetes mellitus (IDDM or Type I diabetes) and non-insulin-dependent diabetes mellitus (NIDDM or Type II diabetes). IDDM may occur at any age, but it primarily appears before age 30. NIDDM, on the other hand, usually occurs in individuals over the age of 40. The diseases also differ in severity, underlying deficit, and type of therapeutic control. IDDM usually is more severe and is characterized by impairment in the ability to produce insulin. Daily insulin injections are required to manage the disease. NIDDM, on the other hand, typically is less severe, and is marked by an impaired ability to recognize and utilize insulin. Therapeutic control often is achieved by diet alone or in combination with oral hypoglycemic agents. Some individuals with NIDDM, are, however, treated with insulin. Of the two, NIDDM is the most common, with IDDM comprising only five percent to 10 percent of the total diabetic population (Canadian Diabetic Association, 2000; NIH, 1995).

The problems associated with diabetes which may affect driving competency can be classified as either acute or chronic. Chronic effects of diabetes include cardiovascular disease (coronary artery disease, hypertension, cerebrovascular accidents, microangiopathy), neuropathy, and diabetic retinopathy. The effects of the chronic complications of diabetes mellitus on driving are discussed under their respective headings (e.g., cardiovascular disease, peripheral neuropathy, etc.). Hypoglycemic reactions among diabetic drivers represent the most acute risk and are a primary factor of concern for traffic safety. Importantly, hypoglycemia does not occur in NIDDM treated only with diet and is unlikely to occur in those individuals with NIDDM treated with oral hypoglycemics. For example, six percent of individuals treated with sulfonylurea-derivatives in a study by Jennings, Wilson, and Ward (1989) experienced hypoglycemic symptoms monthly, and only 14 percent experienced hypoglycemic symptoms less frequently. Hypoglycemic reactions are most likely to occur in insulin treated individuals with IDDM, particularly those who are under tight glycemic control (The DCCT Research Group, 1987). Because of the importance for traffic safety, a more detailed discussion of hypoglycemic reactions in the diabetic driver is provided following the general review of research on diabetes and driving given below.

Diabetes Mellitus and Driving Literature Review

The following section provides a review of the studies examining the influence of diabetes mellitus on driving behaviour. The studies can be roughly grouped into two time periods: the early studies, which were conducted in the mid to late 60's, and more recent studies, published between 1988 and 1991. A summary of the studies is provided in Table 24.

As can be seen, some studies fail to find a significant increase in crash risk for individuals with diabetes mellitus, others report a significant increase, whereas others show no difference. Reasons for the discrepant results are proposed below.

One of the first studies to examine the risks associated with driving a motor vehicle by individuals with diabetes was conducted by Waller in 1965. In a retrospective study, he compared the driving records of 257 individuals known to the California Department of Motor Vehicles with the records of 922 randomly selected controls. For both samples, information was obtained, through direct interviews or written questionnaires, regarding age, sex, marital status, occupation, and number of miles driven annually. Results revealed that drivers with medical conditions had significantly higher crash and traffic violation rates at all ages than did those in the comparison sample. In those individuals with diabetes (type not specified), there was a reported 78 percent increase in crash rates (8.7/106 miles versus 15.5/106 miles), and a 39 percent increase in traffic violations (3.3/105 miles versus 4.6/106 miles). In Sweden, Ysander (1966) examined the driving records of individuals with chronic diseases during a

Table 24 Summary of Studies on the Risk of Crash for Drivers with Diabetes Mellitus

Study	n	Method	Controls	Diabetics	Exposure taken into account	Diabetes
Waller (1965)	257	State Records	8.7/105 mi	15.5/105 mi	Yes	NS
Ysander (1966)	243	State Records	7.7 percent	5 percent	Yes	IDDM-90 percent
Crancer & McMurray (1968)	7646	State Records	26.5/100 drivers	31.5/100 drivers	—	NS
Davis et al. (1973)	108	State Records	7.4100 drivers	7.1/100 drivers	?	?
Songer et al. (1988)*	127	Self-Report	7.1/100 drivers	14.2/100 drivers	Yes	IDDM
Eadington & Frier (1989)**	166	Self-Report	10.0/109 miles	5.4/109 miles	Yes	IDDM
Stevens et al. (1989)	354	Self-Report	25 percent	23 percent	Yes	IDDM
Chantelau (1991)	257	Self-Report	0.07/driver/yr	0.06/driver/yr	?	Insulin treated
Hansotia & Broste (1991)***	484	State Records	1.00	1.32	—	IDDM-10 percent NIDDM-90 percent

* *Differences not significant*
** *Rate for control group based on estimated general population crash rates based on DOT statistics*
*** *Standardized mishap ratio (estimate of the risk of mishap in the affected group relative to the risk in the comparison group)*
– *No Data*
? *Not reported*

ten-year period. Frequency of crashes and serious traffic offenses was compared with a control group matched for sex, age, and duration-of-license holding. In contrast to Waller's results, Ysander's results revealed a lower rate of crash and violation involvement for drivers with chronic medical conditions compared to controls. Reported crashes for individuals with IDDM were 5 percent compared to 7.7 percent for the whole control series. The rates for reported serious driving offences were 12 percent for IDDM individuals compared to 15.3 percent for controls. It is interesting to note that the average number of kilometers driven per year was substantially less for individuals with chronic diseases 26 years and older compared to their same age counterparts. The medically impaired group aged 26-50 drove a reported 3,600 fewer kilometers per year and the 50 years of age and older group drove 13,300 fewer reported kilometers per year than their same aged peers. In addition, 21 percent of those in the medically impaired group did not drive during the study period due to illness, lowering the risk for this portion of the group. Therefore, exposure may have been a factor in the reduced rate of crashes and violations for the medically impaired group in this investigation.

Using the same methodology, Crancer and McMurray (1968) compared the crash and violation rates of medically restricted drivers to 1.6 million non-medically restricted licensed drivers. Individuals with diabetes (type not specified) had statistically higher crash rates (31.45 per 100 drivers), compared to age- and sex-matched controls (26.5 per 100 drivers). Amount of driving exposure was not measured in this investigation. Finally, Davis, Wahling, and Carpenter (1973) examined the driving records of individuals who had been granted driver's licenses following a review by the Oklahoma Medical Advisory Committee in 1969. The crash rates of 108 diabetic drivers the year following the review were compared to 1.65 million age- and sex-matched controls. There were no significant differences between the two groups in incidence of crashes per 100 drivers (7.4 crashes per 100 diabetic drivers versus 7.1 for the control group).

Fifteen years later, Songer, LaPorte, Dorman, et al. (1988) examined one-year self-reported crash rates of 127 IDDM individuals and their non-diabetic siblings. Although the overall crash risks of the two groups (7.1/100 drivers versus 14.2/100 drivers) were not

significantly different, female diabetic drivers showed a marked increase for crashes. Compared to the female controls, female diabetic drivers had a five-fold increase in reported crashes. Overall, when adjusted for mileage driven, the number of crashes was higher in the IDDM population compared to the non-diabetic controls (10.4 versus 3.9 crashes per 100 drivers per 1 million miles), but this difference was not significant.

The following year, based on the results of a questionnaire mailed to individuals with Type 1 (IDDM) diabetes, Eadington and Frier (1989) compared the crash rates of 166 respondents to that of drivers in the general population. Mileage-adjusted crash rates for males were 4.9 crashes per million miles driven and 6.3 crashes per million miles for females, with an overall rate of 5.4 crashes per million miles driven. The authors concluded that the diabetics' crash rate was comparable to that of the overall population crash rate of 10 crashes per million miles driven (based on 1986 DOT statistics).

Stevens, Roberts, McKane, et al. (1989) compared the self-reported crash rates of 354 diabetic drivers treated with insulin to 302 non-diabetic outpatient drivers. The two groups showed similar characteristics in terms of annual distance driven and usual driving area. Crash rates were similar between the two groups with 23 percent of the diabetic drivers and 25 percent of the non-diabetic drivers reporting crashes in the previous five years. Importantly, as noted by the authors, the diabetic drivers included in this study, and those of Eadington and Frier (1989), were a select group of diabetic drivers. That is, individuals who had diabetic complications or difficulties with diabetes had often stopped driving, a consideration that may have contributed to the favourable crash record of the diabetic group. In Germany, a recent survey among 257 diabetic individuals treated with insulin revealed that study individuals reported a total of 27 severe car crashes during the previous two years (Chantelau, 1991). This translates into a rate of 0.06 severe crashes per driver per year, compared with approximately 0.07 such crashes per driver per year in the average population. Again, the major limitation is that risk exposure (e.g., annual mileage driven) was not taken into consideration in this investigation.

Finally, in a population-based, retrospective cohort study of 30,420 individuals (16 to 90 years of age), with and without diabetes mellitus or epilepsy, Hansotia and Broste (1991) compared the standardized rates of moving violations and crashes during a four-year period in affected and unaffected cohorts. The size of the final cohort of persons with diabetes included for study was 484. Standardized mishap ratios for individuals with diabetes for moving violations were non-significant, but the standardized crash ratio was 1.32 versus 1.00. Of interest, 90 percent of the diabetic individuals were considered to have Type II diabetes (NIDDM), although more than one third of the individuals of the NIDDM cohort took insulin. Again, the study is limited by the lack of information on the number of miles driven annually by study participants.

A summary of the available literature reveals a lack of consensus on the risk of crashes for diabetic drivers. Generally, the results from the earlier studies are based on crash and violation data obtained from state records while those from later years are, for the most part, based on data obtained from self-reports. Despite the limitations of each of the methodologies, there are, however, no clear-cut patterns either for time period or methodology. That is, two of the earlier studies (Crancer and McMurray, 1969; Waller, 1965) reported an increase in crashes among diabetic drivers, while Ysander (1966) and Davis et al. (1973) found no significant differences. Among the later studies, Hansotia and Broste (1991) found significant increases for diabetic drivers and Songer et al. (1988) found increases for female diabetic drivers. Eadington and Frier (1989), on the other hand, reported decreases in crash frequencies, while Stevens et al. (1989) found no differences. Five of the investigations utilized state records for determining crash risk, and in three of those (Crancer and McMurray, 1968; Hansotia and Broste, 1991; Waller, 1965), significant increases for diabetic drivers were found. However, a decrease was noted in Ysander's (1966) investigation, and no differences were reported in Davis et al.'s (1973) investigation.

Studies relying on self-report are equally disparate. One investigation found a decrease in frequency of crashes for diabetic drivers (Eadington and Frier, 1989), results from another indicated no difference (Stevens et al. 1989), while the third (Songer et al., 1988) reported an increase but for female diabetic drivers only. The pattern of results is equivocal even when distinguishing between drivers with IDDM versus those in which type of diabetes is unspecified. Similarly, no clear-cut pattern emerges when amount of driving exposure is taken into consideration. However, when one considers country of origin, a pattern emerges. That is, studies done in the United States generally show either an increased crash risk or a trend toward increased crashes for individuals with diabetes mellitus (Crancer and McMurray, 1968; Hansotia and Broste, 1991; Koepsell, Wolf, McCloskey, et al., 1994; Songer et al., 1988; Waller, 1965). The European studies, on the other hand, fail to show a significant difference in crash rates. Thus, differences in licensing requirements may account for the differences in crash rates for the different countries. It may be that the European countries have had more restrictive licensing requirements and/or there may be more

awareness in European countries of the associated risk than in America. Therefore, diabetic drivers in European countries may be compensating for their illness by driving less.

Hypoglycemic Reactions

As noted earlier, of all the metabolic complications of diabetes, hypoglycemia represents the most acute risk for traffic safety concerns. Hypoglycemia is common in diabetic individuals treated with insulin and also can occur in individuals treated with oral hypoglycemic sulfonylurea agents. Severity of hypoglycemia can range from very mild lowering of glycemia (60-70 mg/dl) with minimal or no symptoms to severe hypoglycemia with very low glucose levels (<40 mg/dl) and neurologic impairment (Gerich, Mokan, Veneman, and Korytkowski, 1991).

A typical hierarchy of responses to decreases in plasma glucose concentrations has been described by Gerich et al. (1991). The initial response, occurring at approximately 70 mg/dl, involves an increase in the secretion of counterregulatory hormones (glucagon, epinephrine, growth hormone, and cortisol) and a concomitant increase in norepinephrine and acetylcholine release. If the initial responses are ineffective and further decreases in plasma glucose concentrations occur, the autonomic symptoms of sweating, tremor, hunger, anxiety, and palpitations occur, typically at blood glucose concentrations of 60 mg/dl. These autonomic symptoms usually act as a warning to the experienced individual to undertake protective measures (e.g., the intake of food) to ward off an impending hypoglycemic reaction. If the autonomic warning symptoms are ignored or unrecognized (hypoglycemic unawareness), with subsequent reductions in plasma glucose concentrations to around 50 mg/dl, symptoms of neuroglycopenia (weakness, lethargy, blurred vision, confusion, dizziness) and signs of cognitive dysfunction usually occur. Results from Pramming, Thorsteinsson, Bebdtson, and Bionder (1986) reveal deteriorations in cognitive performance in IDDM individuals at blood glucose concentrations just below subnormal levels (54 mg/dl). An important finding in this investigation is that for all but one of the neuropsychological tests (finger tapping), there was a gradual deterioration in cognitive performance with decreasing blood glucose concentrations. Based on outcomes, the authors concluded that performance on everyday tasks that entail planning and control would be adversely affected even at subnormal blood glucose concentrations, concentrations that are usually not considered to be hypoglycemic. Significant disruptions in simulated driving behaviors during moderate hypoglycemia (2.6 + 28 mM, ˜50 mg/dl) have been reported by Cox, Gonder-Frederick, and Clarke (1993). Disrupted behaviors included more swerving, spinning, time over midline, time off road, and apparent compensatory slowing with an increase in 'very slow' driving.

Despite the fact that hypoglycemia is the most common complication of insulin therapy in individuals with diabetes mellitus, the actual incidence of hypoglycemia is, however, difficult to ascertain. Ward, Stewart, Cutfield, et al. (1990) examined the prevalence of hypoglycemia in individuals randomly selected from outpatient clinics in Auckland, Australia. The authors found that the majority (98 percent) of those surveyed had experienced hypoglycemia, with 73 percent reporting having had at least one mild episode of mild hypoglycemia monthly. Episodes of minor hypoglycemia have been estimated to occur twice per week in individuals with IDDM (The DCCT Research Group, 1991). Although short-term effects of mild hypoglycemia are troublesome for the individual, it is unlikely that episodes of mild hypoglycemia, if circumvented, pose much of a danger for individuals operating a motor vehicle. This is because signs of cognitive dysfunction generally begin to occur at plasma glucose concentrations around 50 mg/dl, which are below plasma glucose concentrations that initiate warning signs (Blackman, Towle, Lewis, Spire, and Polobsky, 1990; Ipp and Forster, 1987; Mitarkou, Ryan, Veneman et al., 1991; Wideom and Simonson, 1990). Severe hypoglycemic reactions, on the other hand, represent the most significant short-term danger for the diabetic individual and particularly if the episode occurs during driving.

Definitions of severe hypoglycemia vary and include hypoglycemia resulting in a seizure or a coma, reactions that require the intervention of another person, or a reaction that requires the administration of intravenous glucose, intramuscular glucagon, or hospitalization. Table 25 presents a summary of studies that have investigated the incidence of severe hypoglycemia in insulin treated diabetics. As can be seen, there is considerable variation among studies in the reported frequency of severe hypoglycemia, with estimates ranging from 0.04 to 1.7 episodes per patient per year.

The disparity can be attributed to a number of factors, including differences in study population, criteria for severe hypoglycemia, and degree of metabolic control. Generally, those studies employing the most restrictive criteria for hypoglycemic reactions tend to report the lowest incidence (Casparie and Elving, 1985; Goldstein, England, Hess, Rawlings, and Walker, 1981; Mulhauser, Berger, Sonnenberg, et al., 1985; Nilsson, Tideholm, Kalen, and Katzman, 1988; The DCCT Research Group, 1987). The very low rate (0.04 episodes per patient per year) reported by Goldstein et al. is most likely attrib-

Table 25 Summary of Studies on the Incidence of Severe Hypoglycemic Reactions in Individuals with Diabetes Mellitus

Study	n	Rx	Criteria	Reactions/Person/ Year	
				IDDM	NIDDM
Goldstein et al. (1981)*	147	C	Altered Consciousness/ Prolonged CNS Symptoms	0.04	
Potter et al. (1982)	204	C	Not Specified	0.14	
Casparie & Elving (1985)	400	C	External Intervention	0.12	0.05
Mulhauser et al. (1985)	384	C	Loss of Consciousness or IM Glucagon or Assistance from Physician or Hospitalization	0.19	
	50	I			
The DCCT Research Group (1987)	817	C	Coma/Seizure/ IV Glucose/ IM Glucagon	0.17	
		I		0.54	
Nilsson et al. (1988)	˜900		IV Glucose/ IM Glucagon	0.07	
Bergada et al. (1989)*	350	C	Seizure or Loss of Consciousness or External Assistance	0.07	
Pittsburgh (EDC) (1991)**	—	C	Loss of Consciousness	0.31	
Pramming et al. (1991)	411		External Intervention***	1.6	
McLeod et al. (1993)	600	C	External Intervention*** External Intervention****	.46 1.7	0.73
Study	**n**	**Rx**	**Criteria**	**Percent Hypoglycemic**	
Goldgewicht et al. (1983)	172	C/I	External Intervention or Leading to Hospitalization	26 percent	
Ward et al. (1990)	158	C/I	External Intervention	17 percent	

** Children and adolescents only* *** Cited in Songer et al. (1993)*
**** Including oral CHO* ***** Excluding CHO*
C = Conventional Therapy *I = Intensive Therapy*

utable to their unusual criterion measure in which severe hypoglycemia was defined as those episodes of hypoglycemia characterized by altered central nervous system function or prolonged autonomic symptoms. The incidence of severe hypoglycemia reported in the MacLeod, Hepburn, and Frier study of 1.7 episodes/ person/year is similar to that reported in the 1991 study by Pramming and collegeaues (1.6 episdoes/person/ year). Both studies used similar definitions of severe hypoglycaemia, which included episodes that involved external help including the sole administration of oral carbohydrates. Excluding those individuals from the analysis, the incidence rate in the MacLeod et al. investigation was 0.46 episodes per patient per year, a rate similar to that reported by the DCCT Research Group. Many of the reported rates shown in Table 25 are based on individuals from medical centers or clinics. Thus, it may be that the rates overestimate the incidence of severe hypoglycaemia, given that individuals attending clinics or medical centres may have more problems with hypoglycemia than the general diabetic population. Results from Songer, Lave, and LaPorte (1993) support this assumption. Songer et al. (1993), using unpublished population-based data from the Pittsburgh Epidemiology Diabetes Complication Study, place the incidence of severe hypoglycemia in the insulin-dependent diabetic population at 0.31 episodes per person per year. Severe hypoglycemia in the Pittsburgh investigation was defined as loss of consciousness. Extrapolating from the available data, the best estimate for the incidence of severe hypoglycemia in insulin-dependent diabetes is around 0.30 incidences/person/year.

It is important to note that differing types of treatment regimes also may affect incidence rates. In recent years, management of IDDM has included efforts to achieve

near normal glucose levels as a means of controlling or delaying chronic complications. However, tighter glycemic control has not been without adverse consequences. As can be seen from Table 25, results from the DCCT Research Group's one year feasibility study (1987) revealed a three-fold increase in the occurrence of hypoglycemia in individuals receiving intensive therapy (an insulin pump or three of more insulin injections per day) compared with conventional therapy (one or two insulin injections per day). Analysis of data from the first 45 months of the DCCT revealed that the incidence of severe hypoglycemia ranged from two to six times that observed with conventional therapy (The DCCT Research Group, 1991). As noted by this group:

> *The substantially increased risk for severe hypoglycemia that accompanies intensive therapy re-emphasizes the importance of determining the potential benefits and risks of efforts to maintain blood glucose at near normal levels in persons with IDDM. Although long-term benefits of these intensive efforts remain unproven, the present study indicates that efforts to maintain pre-meal and bedtime glucose levels between 70 and 120 mg/dl, using treatment methods employed in the DCCT, will at least double the risk of hypoglycemia with temporary neurologic impairment sufficient to preclude self-treatment.*
>
> **The DCCT Research Group, 1991, pp. 458-459.**

If future trends are for stricter glycemic control, the present estimate rates may seriously underestimate the incidence for severe hypoglycemia in the diabetic population. More importantly, doubling the risk of hypoglycemic reactions as a result of tighter glycemic control could have important implications for traffic safety.

The effect of hypoglycemia on driving is an important issue. However, information on the frequency and severity of hypoglycemic reactions while driving is scarce. Clark, Knight, Wiles, et al. (1982), in a retrospective questionnaire of 94 insulin treated diabetic drivers, found that 49 percent of the men and 19 percent of the women interviewed had, at some time, experienced symptoms of hypoglycemia while driving. In a survey by Stevens et al. (1989), approximately 30 percent of diabetic drivers receiving insulin reported recognising hypoglycemic symptoms while driving. Forty percent of randomly selected patients with IDDM attending outpatient clinics in Auckland, Australia reported experiencing hypoglycemia while driving, and 13 percent attributed a crash to hypoglycemia (Ward, Stewart, and Cutfield, 1990). Eadington and Frier (1989) estimate that 15 percent of crashes involving diabetic patients may be attributable to hypoglycemia. In 12 percent of the sample studied by Stevens and his colleagues, hypoglycemia was felt to be the cause of a crash. Forty-six percent of those drivers reported experiencing hypoglycemic events two to five times during the year, and 13 percent of the sample reported having had a hypoglycemic event more than five times. Importantly, the number of hypoglycemic episodes while driving during the past year was associated with the total number of crashes experienced by drivers during the past five years ($p = 0.03$). Drivers with two or more hypoglycemic events in the last year were almost twice as likely to incur one or more crashes during a five-year period as compared to those diabetic drivers without a hypoglycemic episode.

Frier, Matthews, Steel, and Duncan (1980) surveyed 250 individuals with IDDM currently licensed to drive. Thirty-eight percent of the individuals who admitted to being involved in a crash since starting insulin treatment attributed the causal factor to hypoglycemia. The role of hypoglycemia was reported to play a substantially greater role in motor vehicle crashes in a study by Chantelau (1991). His data revealed that 60 percent of severe car crashes, based on self-reports of diabetic individuals treated with insulin, were most probably related to hypoglycemia according to the patient's own monitoring of blood glucose levels, monitoring of the emergency department immediately after the crash, or both. Therefore, despite the lack of data from large samples indicating an increased risk for motor vehicle crashes as a result of hypoglycemia, the available evidence from smaller studies reveals a positive relationship between hypoglycemic reactions and motor vehicle crashes.

Predictors of Hypoglycemia

The major morbidity associated with hypoglycemia is temporary neurologic deficit and coma, seizures with central nervous system injury, and permanent neurologic impairment if treatment is absent or delayed. Given the potential seriousness of hypoglycemic reactions for individuals with diabetes, it is not surprising that a number of studies have attempted to find reliable predictors of hypoglycemia. Table 26 presents a summary of studies that have examined potential risk factors for hypoglycemia. In general, the majority of studies have examined the relationships between demographic and medical factors using severe hypoglycemia as the dependent or criterion variable. Most, if not all of the studies, have relied on self-report for documentation of hypoglycemic reactions. Despite the obvious limitations in the methodology, it is noteworthy that research suggests that most diabetic individuals are able to recall and retrospectively report severe episodes with considerable accuracy (Pramming et al., 1991).

Generally, the associations between age or insulin dosage and severe hypoglycemic reactions have been insignificant. Higher levels of insulin have been significantly associated with an increased risk of severe episodes in some studies (Casparie and Elving, 1985; The DCCT Research Group, 1991) but not in others (Goldgewicht et al., 1983; MacLeod et al., 1993; Nilsson et al., 1988; Ward et al., 1990). Significant differences have been found in mean hemoglobin A1c (HbA_{1c}) levels, with lower recent levels reported in the hypoglycemic groups (Casparie and Elving, 1983; The DCCT Research Group, 1991), although Nilsson et al. (1988) failed to find significant differences.

A previous history of severe hypoglycemic episodes is a consistent and significant predictor of future episodes. Results from Nilsson et al. (1988) revealed that 78 percent of individuals in the severe hypoglycemia group (n = 46) had experienced episodes of severe hypoglycemia prior to the study compared with 22 percent in the control group (n = 22 for those individuals not experiencing a severe hypoglycemic reaction during study). A history of severe hypoglycemia was a significant predictor of severe hypoglycemia in intensively treated subjects in the DCCT Research Group study (1991), with almost a three-fold increase in relative risk (RR = 2.54, 95 percent CI = 1.67-3.88). Significant risk factors for severe hypoglycemia in the MacLeod et al. (1993) investigation included a history of severe hypoglycemia, a history of hypoglycemia related injury, or hypoglycemia related convulsion. A retrospective review of driving records of insulin-dependent diabetic and non-diabetic drivers revealed that the number of hypoglycemic episodes while driving during the past year was significantly related to total number of crashes during a five-year period for 354 diabetic drivers. Individuals reporting two or more hypoglycemic episodes were twice as likely to be involved in one or more crashes compared to those diabetic drivers reporting no hypoglycemic episodes.

In summary, a previous history of severe hypoglycemic reactions is significantly associated with future episodes of severe hypoglycemia. Thus, a history of severe hypoglycemia could serve as a medical red flag for licensing decisions. A review of the driver licensing regulations in the United States reveals, however, that few of the states currently take history of hypoglycemic reactions into consideration when making licensing decisions. For example, summary guidelines of licensing regulations for diabetic drivers are available for 19 of the states in the United States (U.S. Department of Transportation, 1992). For 11 of the 19 states, there is no specific mention of diabetes in the summary guidelines. For 8 of the 19 states, the specific guidelines for diabetes range from Medical Report requirements (District of Columbia, Florida, West Virginia) to functional ability profiles (Maine, Utah). For seven of the states, there is no specific mention of diabetes mellitus in the summary guidelines. Presumably, acute complications of diabetes mellitus (e.g., hypoglycemic episodes leading to temporary neurological impairment, including seizures and/or coma) would be covered in guidelines for altered states of consciousness. Finally, for those states that have specific licensing guidelines for diabetes mellitus, a review of those guidelines reveals considerable variability between states. Utah provides the most comprehensive guidelines for the licensure of diabetic drivers and the reader is encouraged to review those guidelines for more details.

Table 26 Summary of Studies Examining Predictors of Severe Hypoglycemic Reactions in Individuals with Diabetes Mellitus

Study	Predictors						
	Age	Illness Duration	Insulin Dose	HbA1c level	Hx of HE*	Hx of HUA**	Insulin Regime
Goldgewicht et al. (1983)	NS	±	—	—	—	—	—
Casparie & Elving (1985)	NS	NS	±	±	—	—	—
Nilsson et al. (1988)	NS	NS	NS	NS	±	—	±
Eadington & Frier (1989)	—	—	—	—	±	±	—
Ward et al. (1990)	—	—	NS	—	—	—	—
The DCCT Group (1991)	NS	±	±	±	±	—	±
MacLeod et al. (1993)	NS	3	NS	—	±	±	—

**History of Hypoglycemc Episodes* ***History of Hypoglycemic Unawareness*
NS = Relationship not significant *± = Relationship found* *— = Relationship not examined/reported*

In addition to a history of severe hypoglycemia, recent research suggests that hypoglycemia unawareness may be an important risk factor for severe hypoglycemic episodes. The following section reviews the literature on hypoglycemic unawareness and its relevance for driving.

Hypoglycemia Unawareness

Hypoglycemic unawareness is commonly defined as an inability to recognize the autonomic symptoms (sweating, tremor, hunger, anxiety, and palpitations) of decreased plasma glucose concentrations or a failure of the warning signs to occur before development of neuroglycopenia (Gerich et al., 1991). In some European countries, unawareness of hypoglycemia is considered the most important reason for denying driving privileges to individuals with diabetes mellitus (Veneman, 1996). A review of the literature suggests that hypoglycemic unawareness is a frequent phenomenon among insulin treated diabetics. Table 27 provides a summary of those studies that have investigated the frequency of hypoglycemia unawareness in individuals with diabetes. Despite the varying methodologies (e.g., populations, categorization of hypoglycemic unawareness, retrospective versus prospective surveys, etc.), the best current estimate is that hypoglycemia unawareness occurs in about 25 percent of individuals with IDDM, with estimates ranging from eight percent to 70 percent. Bergada, Suissa, Dufresne, and Schiffrin (1989) report the highest rate (70 percent). However, their total study population consisted of 350 diabetic children and some of the episodes not preceded by warning symptoms occurred during the night. The higher rates (50 and 51 percent, respectively) reported by Arias, Kernerm Zier, Navacues, and Pfeiffer (1985) and the DCCT Research Group (1991) may be the result of therapeutic regime. In both investigations, the samples consisted of IDDM individuals undergoing intensive insulin therapy.

Predictors of Hypoglycemia Unawareness

A number of studies have examined factors that may be associated with hypoglycemic unawareness. A summary of the literature is provided in Table 28. As can be seen, factors that may be associated with hypoglycemic unawareness include age, duration of diabetes, presence or absence of autonomic neuropathy, species of insulin, degree of metabolic control, and hypoglycemia itself. To date, most studies that have examined hypoglycemic unawareness in individuals with diabetes have relied on questionnaires to classify subjects, a methodology which may limit the findings. Gerich et al. (1991) suggest that in future investigations, subjects should be categorized on the basis of prospectively obtained objective criteria such as glycemic thresholds for development of autonomic symptoms obtained during a standardized insulin infusion test.

Although a number of factors have been proposed to be associated with hypoglycemia unawareness, there is a paucity of literature addressing this issue. More research is needed before any definitive statements can

Table 27 Summary of Studies Investigating the Frequency of Hypoglycemia Unawareness (Reproduced from *Spinger-Verlag Diabetologica, Hypoglycemic reactions in 172 Type 1 [Insulin Dependent] diabetic patients, Gerich et al., 24, 95-99, 1991* with permission from Springer-Verlag)

Study	Subjects (n)	Unawareness (n)	Percent
Arias et al. (1985)	19	10	50
Bergada et al. (1989)	24	17	70
Berlin et al. (1987)	37	12	33
Collier et al. (1987)	49	12	25
Fui et al. (1986)	11	4	35
Goldgewicht et al. (1983)	180	36	20
Grimaldi et al. (1990)	151	26	17
Hepburn et al. (1990)	302	69	23
Mokan et al. (1991)	34	9	26
Moses et al. (1985)	52	6	12
Orchard et al. (1991)	628	126	20
Potter et al. (1982)	120	10	8
Pramming et al. (1991)	411	111	27
Ryder et al. (1990)	23	5	22
The DCCT Research Group (1991)	216	110	51
Overall Average	**2257**	**563**	**25**

Table 28 Summary of Studies Examining Possible Predictors of Hypoglycemia

Factor	Study
Age	Hepburn et al. (1990)
Duration of Diabetes	Hepburn et al. (1990) Mokan et al. (1994) Ward et al. (1990)
Autonomic Neuropathy	Cryer & Gerich (1983) Heller et al. (1987) Hoeldtke et al. (1982) Sussman et al. (1963)
Species of Insulin	↑ Risk: Teuscher & Berger (1987) No Difference: Egger et al. (1991) Gale & Tattersal (1979) Jones et al. (1991) Muhlhasser et al. (1991) Orchard et al. (1991) Schwarz et al. (1990)
Increased Metabolic Control	Mokan et al. (1994)
Hypoglycemia itself	Banting et al. (1923) Goldfein et al. (1961) Heller & Cryer (1991) Joslin et al. (1924) Maddock & Trimble (1928) Widom & Simonson (1990)

be made regarding risk factors for hypoglycemia unawareness. Nevertheless, hypoglycemia unawareness is a major risk factor for the development of severe hypoglycemia and therefore should be a major concern for individuals with diabetes who drive.

9.2. Thyroid Disease

9.2.a. Hyperthyroidism
9.2.b. Hypothyroidism

9.2a. Hyperthyroidism

Hyperthyroidism is the clinical expression of a group of disorders that produces elevated levels of free thyroxine and/or triiodothyronine (Gorroll, May, and Mulley, 1987). Disorders include toxic goiter (Grave's disease), toxic multinodular goiter, and toxic uninodular goiter. Although the prevalence of hyperthyroidism is not known precisely, community-based studies report prevalence rates of 1.9 percent in women and 0.16 percent in men (Gorroll, May, and Mulley, 1995). Approximately 15 percent of recognized cases occur in people older than 60 (Gorroll et al., 1995).

Clinical symptoms of hyperthyroidism include nervousness, tremor, muscle weakness, increased appetite, weight loss, and heat intolerance. In the elderly individual, symptoms may be atypical, with the patient presenting with apathy, weight loss, and cardiovascular dysfunction (unexplained atrial fibrillation). A number of therapeutic agents are available for the treatment of hyperthyroidism.

9.2b. Hypothyroidism

Of the thyroid disorders, hypothyroidism is the more common. The condition is most often caused by some disorder of the thyroid gland that causes decreased thyroid hormone production and secretion (Barnes, 1990). Iodine deficiency is the most common cause worldwide of hypothyroidism. In regions where iodine intake is adequate, the most common causes are chronic autoimmune thyroiditis (Barnes, 1990).

Clinically, the patient with hypothyroidism presents with the following symptoms: fatigue, lethargy, sleepiness, dry skin, cognitive impairment, intolerance to cold, and weight gain. Fatigue, sleepiness, and cognitive impairment are the symptoms with the greatest relevance for driving. Once the diagnosis is established, treatment consists of thyroid hormone replacement therapy.

As with hyperthyroidism, hypothyroidism affects more women than men. The prevalence of hypothyroidism increases with age. According to Goroll et al. (1987), as much as five percent of the elderly population show evidence of hypothyroidism. Subclinical hypothyroidism, on the other hand, is estimated to affect between four percent and 14 percent of people older than 60, with more females than males affected.

Hypothyroidism and Driving Literature Review

As noted previously, cognitive impairments, sleepiness, and fatigue associated with hypothyroidism have direct relevance for driving. In terms of research literature, the effects of hypothyroidism on cognitive functioning have received the most attention. Cognitive deficits associated with hypothyroidism include impairments in general intelligence (Haggerty, Evans, and Pringe, 1986; Mennemeier, Garner, and Heillman, 1993), attention and concentration (Osterweill, Syndulko, Cohen, et al., 1992), memory (Haggerty et al., 1986; Mennemeier et al., 1993), perceptual and visual functioning (Mennemeier et al., 1993; Osterweill et al., 1992), and executive/frontal lobe functioning (Mennemeier et al., 1993). It is interesting to note that many of the cognitive deficits associated with hypothyroidism do not show consistent improvement following treatment with thyroid hormone replacement therapy. It may, therefore, be important to test for cognitive deficits in individuals with hypothyroidism once they have been stabilized on thyroid hormone replacement therapy.

Table 29 Guidelines for Metabolic Diseases *(Reproduced with permission)*

Guidelines for Metabolic Diseases (Drivers of Private Vehicles)		
Illness	**Austroads (1998)**	**CMA (2000)**
Diabetes		
Non-Insulin Treated Diabetes	Should not drive. The DLA will normally issue a conditional license if condition stable. Reviews of driving status will normally be required at intervals no greater than 5 years.	Can usually drive if: 1. Has good understanding of condition. 2. Follows instructions about diet, medication, and prevention of complications. 3. Remains under regular supervision.
Insulin-Treated Diabetes	Should not drive. Normally, the DLA will issue conditional licence on certificate of physician caring for the patient on the premise that the condition is stable and all other criteria (as per guidelines) have been met. Review of condition required at maximum period of 2 years.	Can drive if : 1. Under regular medical supervision. 2. Individual understands their diabetic condition and the close interrelationship between insulin demand, and diet and exercise. 3. Follows physician's advice 4. No history of impairment due to alcohol or drug abuse. 5. No history of severe hypoglycemic episodes in last 6 months.
Hyperthyroidism	May drive if stable and eligible under general vision criteria.	Patients with hyperthyroidism complicated by cardiac or neurologic symptoms should not drive any type of motor vehicle until the condition has been controlled.
Hypothyroidism	May drive if stable.	Patients with symptomatic hypothyroidism should not drive any type of motor vehicle until the condition has been brought under satisfactory control.

DLA = Driver Licensing Authority

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9.1 Diabetes Mellitus

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Section 10: Renal Diseases

10.1 Chronic Renal Failure
10.2 End Stage Renal Disease

A summary of the current fitness-to-drive guidelines (Renal Diseases) for medical practitioners from Australia (1998) and Canada (2000) is presented in Table 30.

10.1 Chronic Renal Failure

Chronic renal failure is a progressive disease involving deterioration and destruction of renal nephrons, with progressive loss of renal function. There are numerous causes of chronic renal failure such as chronic glomerulonephritis, polycystic kidney disease, obstruction, and repeated bouts of pyelonephritis. End stage renal disease is the final stage of chronic renal failure and is characterized by distinctive cardiovascular (e.g., hypertension, anemia), gastrointestinal (e.g., anorexia, nausea, vomiting), metabolic (e.g., increased blood urea nitrogen and serum creatinine levels), musculoskeletal (e.g., diffuse bone pain and bone abnormalities), and neurologic (e.g., peripheral neuropathy, cognitive impairment) symptoms.

Often, symptoms of chronic renal failure are absent in the early stages of the disease. As the disease progresses, however, signs and symptoms of chronic renal failure appear. Symptoms include abnormal urinalysis, hypertension, weight loss, fatigue, malaise, and decreased mental acuity. As noted above, if chronic renal failure progresses to end-stage kidney disease, complications may include hypertension, congestive heart failure, anemia, bone disease, gastrointestinal problems, urinary infection, and dementia (Mayo Clinic, 1997).

For individuals with irreversible kidney disease, management of the illness may include dialysis. Dialysis also is used as a means of managing individuals awaiting a kidney transplant. There are two types: peritoneal dialysis and hemodialysis. Peritoneal dialysis involves instillation of dialysis fluid into the peritoneal cavity. The dialysis fluid is left in the cavity for a short period of time which enables the removal of waste products such as urea and creatinine. The dialysis fluid is then removed. Continuous ambulatory peritoneal dialysis involves infusing and draining dialysis solution from the peritoneal cavity several times a day. This technique allows for greater mobility and independence of the individual. Hemodialysis, on the other hand, consists of creating a shunt between an artery and a vein (e.g., radial artery and cephalic vein). When connected to a dialysis machine, this shunt allows blood to pass from the patient's body to the dialysis machine, through the filter, and back to the patient. Waste products are removed from the body and restoration of fluid and electrolytes occurs during the dialysis procedure.

The 1995 prevalence rate for kidney conditions, in general (e.g., infection, kidney stones, cancer, missing kidney, other), is 3.022 million conditions in the civilian non-institutionalized population (National Center for Health Statistics, 1998).

10.2 End Stage Renal Disease Prevalence

The annual prevalence of End Stage Renal Disease (ESRD) in the United States is 361,031 (based on 1997 data, United States Renal Data Systems [USRDS] 1999 Renal Data Report). The prevalence rates for ESRD have increased substantially (e.g., from 219,255 in 1991, to 314,364 in 1995, to 361,031 in 1997), most likely because of improved survival rates among high-risk populations (e.g., patients with diabetes, hypertension), improvements in management of ESED, and the aging of the population. In general, ESRD is the result of three primary diseases: diabetes, hypertension, and glomerulonephritis.

End Stage Renal Disease and Driving Literature Review

To this author's knowledge, there are no studies that have investigated the relationship between chronic renal failure and risk of motor vehicle crashes. There is a small body of literature, however, indicating that ESRD is associated with diminished perceptual motor-coordination, impairments in intellectual functioning including decreased attention and concentration, and memory impairments (Baker, Brown, Byrne, et al., 1989; Ginn, Tescahn, Walker, et al., 1975; Hart, Pederson, Czerubski, and Adams 1983; Hagberg, 1974; McGee, Burnett, Raft, Batten, and Bain, 1982; Ryan, Souheaver, and DeWolfe, 1980; but see Kramer, Madl, Stockenhuber et al., 1996; Pliskin, Yurk, Ho, and Umans, 1996; Umans and Pliskin, 1998). It is interesting to note that the earlier studies are more likely to report the presence of cognitive impairment in individuals with ESRD compared to more recent studies. It may be that more effective management of ESRD in the last decade or so has led to substantial improvements in cognitive functioning in this patient population.

There is some suggestion that the effects of ESRD on cognitive functioning may differ as a function of type of dialysis program the patient is on. For example,

Buoncristiani, Gubbiotti, Mazzotta, et al. (1993) investigated the relationship between cognitive functioning in patients undergoing either peritoneal or hemodialysis and healthy controls. The sample included 18 patients on continuous ambulatory peritoneal dialysis (CAPD), 15 on hemodialysis comparable in terms of age and time on dialysis, and normal controls. P300 event related potentials were used as an objective marker of cognitive brain function. Results showed that the latencies of the P300 in CAPD patients were comparable to normal controls and to those obtained in postdialytic patients on hemodialysis. However, the results of the predialytic values were significantly different from the postdialytic values, and from the values of the CAPD patients and controls. These results suggest that hemodialysis may restore the cognitive functioning of patients only transiently in the postdialytic stage. On the other hand, results of this research suggest that cognitive functioning is maintained close to the normal range in patients on CAPD.

Not surprisingly, improvements in cognitive performance have been reported in individuals who have undergone a kidney transplant. Recently, Kramer et al. (1996) reported on the effects of renal transplantation on cognitive performance. Cognitive functioning was measured by the P300, the Trailmaking Test, and the Mini-Mental State Examination. The tests were administered to 15 chronic hemodialysis patients pre- and post-transplant, and 45 matched healthy controls. Consistent with the results from Buoncristiani et al. (1993), the patients receiving hemodialysis (pre-transplantation) showed significantly impaired P300's, along with deficits on the Trailmaking Test and the MMSE compared to controls. Following transplant, there were no significant differences between the two groups on measures of cognitive performance. Results of this investigation suggest that cognitive impairments that may be present prior to transplant can be successfully reversed following transplant.

Table 30 Guidelines for Renal Diseases *(Reproduced with permission)*

Illness	Austroads (1998)	CMA (2000)
Chronic Renal Failure	**Renal Failure** No restrictions. May need to assess other problems individually (such as hypertension, medication).	Patients with chronic renal failure are often able to continue to drive with the advent of hospital- and home-based intermittent hemodialysis programs, and the development of portable equipment for continuous peritoneal dialysis. **Intermittent Dialysis** An individual requiring intermittent hemodialysis who wishes to drive more than 1 or 2 days distance from home cannot safely do so without making firm arrangements for dialysis at a conveniently located hospital. Patients must be warned never to venture beyond the range of their customary hospital- or home-dialysis based unit without first making a firm appointment for dialysis elsewhere. **Continuous Peritoneal Dialysis** Individuals who are able to manage (by themselves or with the assistance of others) can probably drive more or less as they wish, limited only by their ability to carry or obtain a continuing supply of fresh dialysis fluid. The individual must be knowledgeable about his/her dialysis procedures and seek immediate assistance if problems should arise.
Renal Transplant	Should not drive for 4 weeks post-surgery. Specialist opinion recommended*.	No restrictions following successful recovery. Ongoing medical supervision a pre-requisite.

** Defined as a professional who assesses fitness-to-drive of those with a medical condition.*
DLA = Driver Licensing Authority

Conclusions

There is little in the way of empirical literature to assist in fitness-to-drive decisions in individuals with renal failure. There are no studies available, to this author's knowledge, assessing the relationship between chronic renal failure and risk for motor vehicle crashes. Nevertheless, there is a small body of literature suggesting that cognitive impairment is associated with untreated renal disease. A number of more recent studies (Buoncristiani et al., 1993; Pliskin et al., 1996; Umans and Pliskin, 1998), however, have reported a lack of significant differences between dialyzed patients and healthy controls on various measures of cognitive performance. Improvements in the last decade or so in the management of ESRD may account for these findings. Importantly, results from Buoncristiani et al. (1993) suggest that the beneficial effects of CAPD may be more enduring than with hemodialysis. Future research with larger sample sizes and a more sensitive battery of cognitive testing would be instructive in this regard.

Given the improvements in the quality of life of renal patients in recent years due to advances in patient care, it is likely that many patients with chronic renal disease can drive safely. However, directives from licensing agencies regarding the necessity for strict adherence to dialysis schedules may be prudent to ensure safety while driving.

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Section 11: Musculoskeletal Disabilities

The driver of a motor vehicle must be able to perform complex muscular movements in order to safely operate a motor vehicle. Driving tasks requiring musculoskeletal function include steering, braking, reversing, accelerating, and maneuvering the vehicle. The reader is directed to the following articles for a review of motor functioning as it relates to driving: Eby, Trombley, Molnar, and Shope, 1998; Marottoli and Drickamer, 1993; Roberts and Roberts, 1993; Sabo and Shipp, 1989; Stelmach and Nahom, 1992.

Prevalence

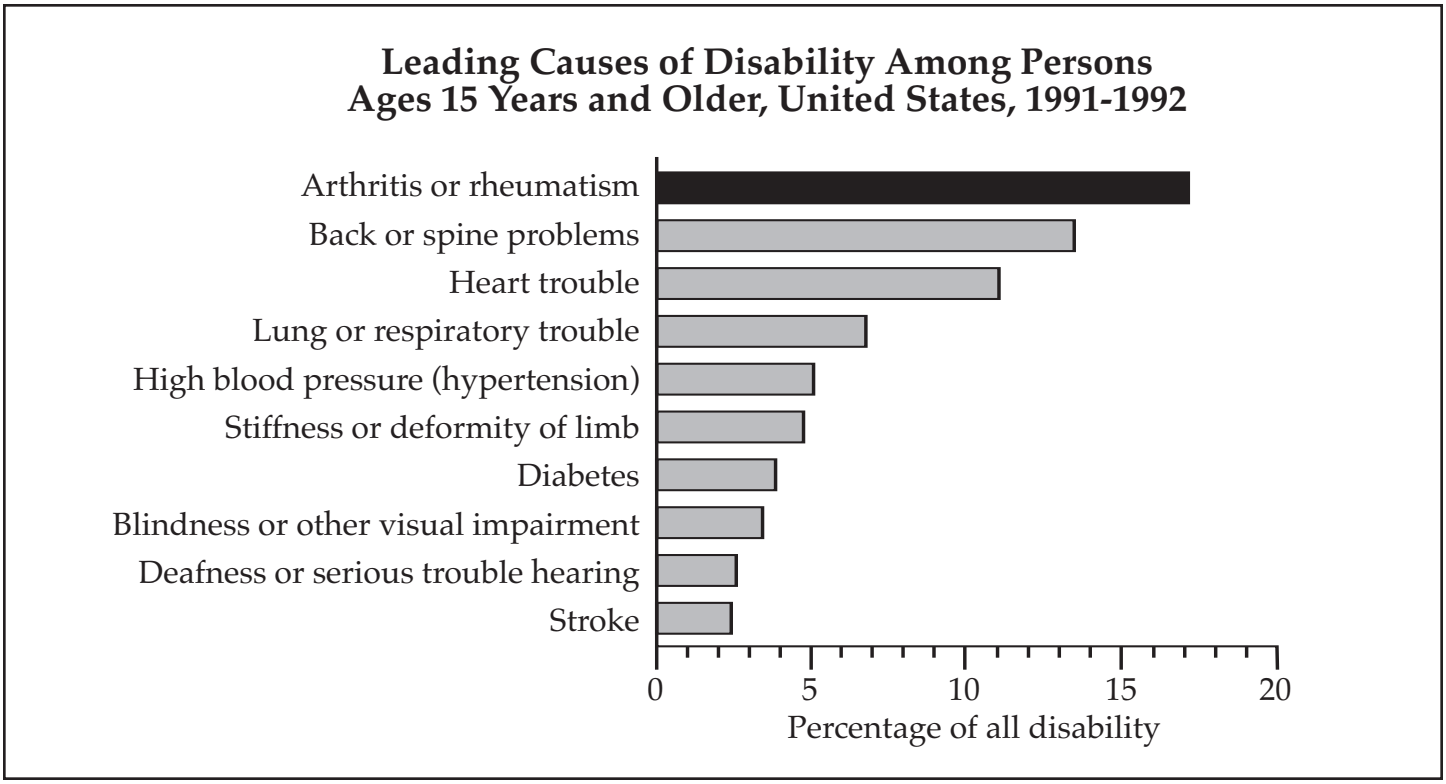
Prevalence estimates on musculoskeletal impairments, as a whole, are difficult to obtain. Data from the Iowa 65+ Rural Health Study reveal that 13.6 percent of men and 17.4 percent of women report impaired upper limb flexibility. Thirty percent of men and 43 percent of women from the same study report impaired lower limb flexibility. In addition, 32 percent of males reported gross physical functional impairment compared to 48 percent of females (Cormoni-Huntley, Brick, Ostfeld, et al., 1986). Gross functional impairments were defined as an inability to do heavy housework, walk half a mile, or climb stairs, but not Activities of Daily Living limitations. Reasons for the gross functional impairments are not specified and are likely to include causes other than musculoskeletal dysfunction.

Figure 3 depicts the leading causes of disability among persons aged 15 years and older in the United States, 1991-1992 (Centers for Disease Control, 1994). As can be seen, arthritis and rheumatism are the leading causes of disability with back and spine problems the second most frequent cause. Stiffness or deformity of limbs was the sixth leading cause of disability during the same time period.

Literature Review

There is a host of musculoskeletal disabilities/impairments that could impair an individual's ability to safely operate a motor vehicle (e.g., arthritis, limb amputations, paraplegia). Perhaps surprisingly, there are few studies that have examined the relationship between musculoskeletal disabilities, in general, and motor vehicle crashes. One study investigating that relationship comes from Utah (Diller et al., 1998-see Section 2.1 a., page 4 for details of the study). There were two categories of medical conditions in the Utah investigation with relevance to this discussion: A category of drivers with musculoskeletal abnormality or chronic medical debility and a category with functional motor impairment.

Figure 3: Leading causes of disability among persons aged 15 years and older in the United States, 1991-1992 (Centers for Disease Control, 1994).



The former category consisted of 1859 drivers without restrictions and 260 restricted drivers. Results reveal that individuals with musculoskeletal abnormality or chronic medical debility have a higher risk of crashes compared to controls (Unrestricted Relative Risk [RR] = 4.02, CI = 3.57 - 4.53: Restricted RR = 3.07, CI = 2.14 - 4.40). Unfortunately, in addition to those with musculoskeletal abnormalities, this category included individuals with active disease, including HIV.

The second category included drivers with a history of functional motor impairments including difficulties with muscular strength, coordination, range and motion, spinal movement and stability, amputations or the absence of body parts, and/or abnormalities affecting control. The sample included 1387 unrestricted drivers and 202 restricted drivers. The relative risk for crashes in the unrestricted category was 3.59 (CI = 3.16 - 4.08) and 2.60 (CI = 1.78 - 3.78) for those in the restricted category. Overall, these results suggest that individuals with functional motor impairment and musculoskeletal abnormalities or chronic medical debilities have a much greater risk for at-fault crashes.

Crash rates and relative risk for crashing for several indicators of health status were determined using data from the Iowa 65+ Rural Health Study (Foley, Wallace, and Eberhard, 1995). Results indicated that drivers with functional impairments (see prevalence section for a description) did not have a significantly increased risk for crashes compared to drivers without physical limitations. However, as noted above, the criteria used to define functional impairments limit the findings.

Gresset and Meyer (1994), using case-control methodology, examined the risk of motor vehicle crashes associated with chronic medical conditions in men 70 years of age and older in the Canadian province of Quebec. The sample included 13 cases and 29 controls with amputations, and 50 cases and 80 controls with paralyses. Cases were not found to have a significantly higher risk of crashes compared to controls. Methodological limitations of the study include small sample size per condition, data based on self-report, and the inclusion of only those cases having had a crash resulting in only mild bodily injury or property damage. Excluded from the study were male drivers involved in fatal crashes and in crashes causing severe bodily damage.

Musculoskeletal impairments (foot abnormalities) were associated with the occurrence of automobile crashes, moving violations, and being stopped by police in a study conducted by Marottoli, Cooney, Wagner, Doucette, and Tinetti (1994). The investigation, a prospective cohort study, included 283 community living individuals 72 years of age and older living in New Haven, Connecticut. Data were collected by means of structured interviews. Adverse driving events (crashes, violations, and being stopped by police) were based on self-report. People with three or more foot abnormalities were more likely to have adverse driving events (RR = 2.2, CI = 1.2 to 3.9). Impaired left-knee flexion also was associated with adverse driving events (RR = 2.9, CI = 1.2 to 6.7). Limitations of the study include data based on self-report and unknown driving exposure, which precludes the adjustment for adverse events based on exposure.

Results of an investigation by Tuokko, Beattie, Tallman, and Cooper (1995) reveal that the presence of arthritis was a predictor of motor vehicle crashes. In that investigation, the authors reviewed official driving records of patients (n = 249) seen in a dementia clinic in Vancouver, British Columbia. Logistic regression revealed that presence of arthritis (Odds Ratio = 3.23, p = .009) and female gender (Odds Ratio = 0.53, p = .04) predicted the occurrence of a motor vehicle crash. It is interesting to note that, in further analyses using data from the arthritis group and those taking non-steroidal anti-inflammatory drugs (NSAIDS), the combination of arthritis and the use of NSAIDS was associated with increased crash involvements. As noted by the authors, further investigation of the relationship between arthritis and NSAID-use, and crash involvement is needed.

The final study of relevance to this discussion was conducted by Jones, McCann, and Lassere in 1991. The authors evaluated 94 individuals with a variety of musculoskeletal disorders (e.g., rheumatoid arthritis [RA], osteoarthritis [OA], low back pain and/or sciatica [LBP], fibromyalgia [FM], and ankylosing spondylitis [AS]) in terms of driving difficulties. Results revealed that individuals with RA exhibited difficulties in all areas of driving performance evaluated. One half of those with RA (total n = 37) experienced difficulties with steering and cornering, and use of the hand brake. Almost 40 percent had difficulties with reversing. An even greater percentage of individuals with OA (total n = 23) had difficulties with reversing. In addition, those with OA had difficulties with steering and cornering, and with lower limb functions. Individuals with LBP (total n = 16) had the most difficulty with use of the foot petal, whereas 50 percent of those with FM (total n = 6) had difficulty with steering and cornering, reversing, and use of the handbrake. Seventy-seven percent of the 94 patients were deemed to be safe drivers with or without minor modifications to the vehicle. Unfortunately, criteria used to determine safe driving were not specified.

Conclusions

As with many chronic diseases, the issue regarding licensing of individuals with musculoskeletal impairments concerns the effects of the impairment on functional ability with respect to driving. It should therefore not be surprising that guidelines regarding evaluation of fitness-to-drive recommend that, for many musculoskeletal disorders, assessments at the individual level be conducted (Austroads, 1998; Canadian Medical Association, 2000). In the absence of cognitive impairment, fitness-to-drive evaluations of individuals with musculoskeletal disabilities need to be directed toward assessing physical functioning. Those assessments are, for the most part, conducted by occupational therapists. However, as noted by Korner-Bitensky, Sofer, Kaizer, Gelinas, and Talbot (1994), survey results of occupational therapists across Canada reveal that "there is no well defined procedure for the assessment of drivers with, not only motor involvement, but also perceptual-cognitive disorders such as those seen following a stroke or head injury" (p. 142). The authors call for the development of a standardized driving evaluation battery to "ensure a thorough and comprehensive evaluation of the individual, to provide consistent evaluations across centres and regions of the country and to strengthen the scientific justification for revoking the license" (p. 147).

A similar call has been made by Springle, Morris, Nowachek, and Karg (1995) following an assessment of the evaluation procedures of drivers with disabilities. In their investigation, the authors sent surveys to 403 driver evaluators and trainers throughout the United States whose clientele included persons with disabilities. One hundred and thirty eight responses (38 percent) were received from 44 states. The authors state that most of the respondents were experienced evaluators (criteria not defined) and 62 percent were occupational therapists. Survey responses revealed that measurement of specific driving characteristics (e.g., brake reaction time, steering force) was thought to be more important than measuring non-specific physical characteristics (e.g., range of motion, grip strength). However, only half the respondents reported measuring those characteristics deemed most important. Most of the characteristics were measured through observation or by using a functional test but the overwhelming majority of evaluators used subjective criteria or no criteria in assessing the results of the test. The authors conclude that research is needed to assist in developing a standardized evaluation procedure.

Many musculoskeletal disabilities may be accommodated through the use of vehicle modifications (see Shipp, 1989 for an overview). These modifications may allow individuals with physical disabilities to drive competently and safely. However, individuals with musculoskeletal disabilities may need to be restricted to driving only those vehicles with the appropriate modifications.

A summary of the current fitness-to-drive guidelines (Musculoskeletal Impairments) for medical practitioners from Australia (1998) and Canada (2000) is presented in Table 31.

Table 31 Guidelines for Musculoskeletal Disabilities *(Reproduced with permission)*

Guidelines for Musculoskeletal Disabilities (Drivers of Private Vehicles)		
Disability/ Illness	**Austroads (1998)**	**CMA (2000)**
Disability of Cervical Region	Some loss of movement of head and neck allowable if vehicle fitted with adequate outside mirrors.	Some degree of loss of movement of head and neck permitted but driver restricted to driving vehicles equipped with right and left outside mirrors, and must have the ability to shoulder check. People wearing a neck brace or cast should not be approved for driving until pain and restriction of movement are minimal, and external support is no longer required.
Disability of Thoracic Region	Persons with interscapular pain, which prevents free movement of shoulder joints, should not drive. Persons wearing braces or body casts should not drive without specialist recommendation.	Persons with marked deformity or painfully restricted motion in thoracic vertebrae can best be determined to drive by a driver examiner. Persons wearing braces or body casts must be evaluated on the basis of their ability to move free of pain, operate the controls, and observe approaching vehicles.
Disability of Lumbar Region	Persons with severe pain, reduced mobility, or neurological impairment should not drive. Persons with moderate lumbar pain should use vehicle with power brakes, steering, and automatic transmission.	May need to be restricted to driving vehicles with power-assisted brakes.
Inflammatory Arthritis	Should not drive if permanent damage of joints has occurred which limits ability to drive. Conditional license may be issued. DLA should be notified.	Not addressed.
Joint Replacement	Driving assessor opinion recommended.	Not addressed.
Loss of Limbs	Should not drive if both upper limbs are missing. All cases need to be individually assessed. Conditional license may be issued. License should be restricted to modified vehicle. DLA should be notified.	May drive provided they demonstrate their ability to drive to the satisfaction of the driver examiner.

Table 31 Guidelines for Musculoskeletal Disabilities *(continued)*

Loss of Thumbs and Fingers	Digit losses to be assessed with regard to spinner knobs. Driving assessor opinion recommended.	Can drive any type of motor vehicle provided they demonstrate ability to the satisfaction of the driver examiner.
Paraplegia or Quadriplegia	Not addressed.	May receive a learner's license on the basis of favorable recommendation from medical consultant in physical medicine and rehabilitation. With permit, may then take driving lessons in specially modified vehicle.
Painful Joints	Should not drive if condition directly affects ability to drive. May drive once condition stabilized. Driving assessor opinion may be needed.	Not addressed.
Muscle and Movement Disorders	See specific impairments.	See specific impairments.
Post surgery	Should not drive for 6 weeks post major orthopedic surgery. Specialist opinion recommended.	Not addressed.
Prostheses	Driving assessor opinion required.* Driving test may be necessary.	Persons with amputations of arms or legs and who have been fitted with an adequate prosthesis may drive any class of motor vehicle provided they have demonstrated their ability to the satisfaction of a driver examiner.

** Defined as a professional who assesses fitness-to-drive of those with a medical condition.*
DLA = Driver Licensing Authority

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Section 12: Psychiatric Diseases

Mental illness is relatively common, with recent studies suggesting that roughly one-third of the general population exhibits signs of mental illness sometime during their lifetime (Weiten, 1998). Earlier estimates were much lower, with indications that one-fifth of the population would exhibit signs of mental illness sometime during the lifespan (Neugebauer, Dohrenwend, and Doherenwend, 1980). The current estimates are much higher due to the recent inclusion of substance abuse disorders as a category of psychiatric illness. Before 1980, substance abuse disorders were vaguely defined in the first two editions of the Diagnostic and Statistical Manual of Mental Disorders (DSM), manuals published by the American Psychiatric Society and used to categorize psychiatric disorders. However, in 1980, with the advent of the DSM-III, explicit criteria for substance abuse disorders were introduced, resulting in more effective recording of substance abuse (American Psychiatric Association, 1980).

A summary of the current fitness-to-drive guidelines (Psychiatric Diseases) for medical practitioners from Australia (1998) and Canada (2000) is presented in Table 33.

As noted above, psychiatric disorders are relatively prevalent in the general population. Some of the more common psychiatric disorders include mood disorders (depression, bipolar disorder), anxiety disorders, schizophrenia, and other psychotic disorders, such as delusional disorder, delirium, dementia (including Alzheimer's disease), and substance abuse disorders (National Institute of Mental Health, 1999). Recent statistics indicate that of adult Americans 18 and older, more than 19 million suffer from a depressive illness each year, more than 2.3 million Americans suffer from bipolar disorder, more than two million are affected by schizophrenia, and more than 16 million adults (ages 18 to 54) suffer from anxiety disorders (National Institutes of Health, 1999).

Despite the prevalence of psychiatric disorders in the general population, there have been few investigations into the relationship between psychiatric illness and motor vehicle crashes. Surprisingly, the majority of the research that is available was conducted, on average, more than 30 years ago. Because of the paucity of recent literature, the older literature will be described below, followed by a review of the most recent literature. A summary of the results is provided in Table 32.

In one of the earliest studies, Waller (1965) distributed questionnaires to individuals with known psychiatric conditions based on reports from the California Department of Motor Vehicles. Questionnaire data also were obtained from a random sample of drivers seeking driver's license renewal. Results revealed that those with reported psychiatric disorders drove fewer miles per year but had double the crash rate compared to the comparison sample. Methodological limitations of this study include a biased patient group (i.e., only those patients reported to the Department of Motor Vehicles by physicians), unknown diagnostic criteria (e.g., not standardized DSM criteria), and crash rates based on self-report.

Four years later, Crancer and Quiring (1969) compared the driving records of individuals admitted to a county mental hospital with the driving records of the remaining county population. The patient category included those with a diagnosis of schizophrenia, personality disorder, and those with psychoneurotic disorders. It is unknown if patients met the DSM criteria for psychiatric disorders. Results of that investigation revealed that individuals with a personality disorder had a crash rate twice that of the control group. Those individuals in the psychoneurotic group had a 50 percent higher crash rate. However, the crash rates for individuals with schizophrenia were no higher than the controls. The amount of driving exposure was not considered in this investigation. Additionally, results of the study are from data that were collected from 1961 through 1967. The patient population consisted of individuals admitted to King County Hospital in Seattle, Washington. However, the data are not adjusted for the length of time individuals spent in hospital, nor is there any indication of length of hospital stay for each of the individuals. It is reasonable to assume that patients were not driving during their hospitalization, thus reducing their overall exposure during the study period. If this assumption is correct, then the higher crash rates of the patient population may, in fact, be an underestimation of crash rates compared to the overall driving population.

In 1970, Elkema, Brosseau, Koshnick, and McGee compared the driving records of psychiatric patients pre- and post-hospitalization with a control group matched for age, sex, and area of residence. The data were analyzed in terms of crashes per hundred driver years, with time spent in hospital subtracted from the total driving experience. Five diagnostic categories were included in the study: male alcoholics, male psychotics, female psychotics, male psychoneurotics, and male personality disorders. Results reveal that all diagnostic groups had higher crash rates than their matched

Table 32 Summary of the Research Literature on Psychiatric Conditions and Motor Vehicle Crashes

Study	Sample Size	Methodology (Outcome measure)	Results
Waller (1965)	Psychiatric = 292 Controls = 926	State recorded crashes (Crashes /million miles).	Psychiatric = 2 fold higher crash rates than comparison sample despite reduced exposure of psychiatric group.
Crancer & Quiring (1969)	Psychiatric = 271 Controls = 687,228 (remaining drivers in county)	State recorded crashes (Mean crashes per group).	Personality disorders = 2 fold higher than controls. Psychoneuroses = 1.5 fold higher than controls. Schizophrenics = no difference. **Exposure not controlled.*
Elkema et al. (1970)	Psychiatric = 238 Controls = 290 (matched for age, sex, area of residence)	State driving records (Crashes per hundred driver-years). (Ratio between experimental and control groups, pre- and post-hospitalization).	Pre / Post Alcoholics (M) 1.59 / 1.15 Personality (M) 35.10 / 6.09 Psychoneur (M 2.22 / 0.51 Psychotics (F) 1.94 / 0.35 Psychotics (M) 1.35 / 0.89 Exp. (Total M) 1.72 / 1.23 Exp. (Total F) 2.69 / 0.33 **Exposure not taken into consideration.*
Armstrong & Whitlock (1980)	Psychiatric = 100 Physically Ill Controls = 100	Self-report interviews. Crashes a) 6 mos pre-admission. b) 2-3 yrs pre-admission. c) Yrs driving experience.	Psychiatric* / Physically a) 14 / 11 b) 33 / 30 c) 70 / 63 **Unadjusted for reduced driving exposure in the psychiatric group.*
Edlund et al. (1989)	Schizophrenia = 70 Controls = 122 (age matched)	Self-report questionnaires. Crude incidence of crashes.	Schizophrenia = 10 percent* Controls = 9 percent* **Unadjusted for exposure. When adjusted for miles driven, crash rate for schiz. was double that of controls.*
Cushman et al. (1990)	Psychiatric = 17 Controls = 17 (matched for age, sex, and marital status)	Retrospective review of medical records and police accident reports.	No significant differences between the two groups for a) head-on crashes b) roll-overs c) single car crashes. **Exposure not taken in to consideration.*
Diller et al. (1998)	Psychiatric/Emotional Disturbance <u>Unrestricted</u> = 8,791 <u>Restricted</u> = 475	Questionnaire data on medical conditions. Probabilistic linkage of different state databases.	<u>Unrestricted</u> RR = 2.11 (CI = 1.99-2.23). <u>Restricted</u> RR = 1.74 (CI = 1.45-2.10).

comparisons pre-hospitalization. Male alcoholics, males with personality disorders, and males with psychoneuroses had the highest crash rates. Post-hospitalization, males with personality disorders continued to have higher crash rates compared to controls. All diagnostic groups had reduced crash rates post-hospitalization versus pre-hospitalization, irrespective of diagnosis. However, male alcoholics and males with personality disorders continued to show higher crash rates post-hospitalization compared to controls, perhaps indicating the difficulty in treating these conditions. Similar to previous studies, driving exposure was not considered in this investigation. In addition, the small sample size for a number of the diagnostic categories is a limiting factor for generalization of the findings.

In 1980, Armstrong and Whitlock compared self-reported crash rates of 100 psychiatric patients with 100 physically ill patients matched for age, sex, and social background. Psychiatric diagnoses were schizophrenia (n = 12), manic-depression (n = 34), neuroses (n = 28), personality disorders (n = 8), alcoholism (n = 15), and drug abuse (n = 2). One individual had a diagnosis of epilepsy (n = 1). A description of the illnesses for the physically ill group is not provided. There was no significant difference in self-reported crashes between the two groups. However, driving exposure for the mentally ill group was substantially less than the physically ill group, suggesting that risk for crashes in the psychiatric group, based on exposure, is substantially greater than their physically ill counterparts. It is interesting to note that 60 percent of the psychiatric patients reported increases in driving problems since becoming ill compared to reports of the same from 23 percent of the physically ill group. The authors also investigated the effects of prescription drugs on crash rates. Neither the physically ill nor the psychiatrically ill patients who reported crashes were taking more drugs than the no-crash group. Not surprisingly, there was a significant difference between the two groups in terms of psychotropic drug use, with the psychiatric group taking a substantially greater number of psychotropic drugs. As noted earlier, limitations of the study include small sample size per diagnostic group, and the use of data based on self-report. In addition, details of the physically ill participants were not included.

In a 1989 study, Edlund, Conrad, and Morris (1989) compared the incidence of crash rates for 70 outpatient schizophrenic patients with 122 age-matched controls. The psychiatric patients met DSM-III-R criteria for schizophrenia of at least one year's duration. There were no significant differences between the two groups for road crashes based on crude accident rates by self-report for the previous 12 months (10 percent for psychiatric patients versus nine percent for controls). There were, however, considerable differences between the two groups in terms of active licensed drivers and number of miles driven. Only 68 percent of the schizophrenics reported driving at all compared to 99 percent of the controls. In terms of driving exposure, only 40 percent of the patients reported driving more than 100 miles per year, whereas 98 percent of the controls reporting driving 100 or more miles per year. Only 27 percent of the psychiatric patients reported driving 5,000 or more miles per year compared to 67 percent of the controls. Thus, when adjusted for exposure, the crash rates of the drivers with schizophrenia are double that of controls. Methodological limitations of the study include data based on self-report and the potential for selection bias for the psychiatric patients. In addition, 20 of the psychiatric outpatients approached for inclusion in the study refused to participate. Chart reviews, conducted for 15 of the 20 patients refusing participation, revealed that three patients had incurred major motor vehicle crashes in the last year - a crash rate of 20 percent.

Cushman, Good, and State (1990) investigated the relationship between psychiatric disorders and motor vehicle crashes. The authors reviewed crash data from all motor vehicle crash victims admitted to 10 Rochester, New York area hospitals between 1983 and 1986. Of the 1,775 cases investigated, 25 individuals were identified as having a psychiatric diagnosis, with eight subjects eliminated because of an additional diagnosis of substance abuse. Comparison of the 17 psychiatric patients with controls matched on age, sex, and marital status revealed no significant differences between the two groups for single-car crashes, head-on crashes, or roll-overs. As with many of the previous studies, limitations of this study include a small sample size and lack of data on driving exposure. In addition, the study was a retrospective review of medical/police reports, and confirmation of diagnoses was lacking. Recency of diagnoses and severity of illness were not established. In addition, the effects of medications on crash rates were not examined.

Finally, results from Utah (see Section 2.1 a., for details of the study) reveal that individuals with psychiatric or emotional conditions have a higher risk of crashes compared to controls (Diller et al., 1998). Results of that study indicate that restricted drivers with psychiatric or emotional conditions had a higher relative risk for all crashes compared to controls (RR = 1.74, CI = 1.45-2.10), and unrestricted drivers with psychiatric or emotional conditions had a relative risk almost two and half times greater than controls (RR = 2.42, CI = 2.23-2.63).

Conclusions

In general, the data that are available suggest that individuals with a psychiatric illness are at increased risk for crashes. Individuals with personality disorders (untreated or treated), untreated psychotics and psychoneurotics, untreated alcoholics, and individuals with schizophrenia appear to be at-risk. However, as noted above, there are a number of methodological weaknesses that limit the findings. For example, the use of self-report data or data obtained from medical records and/or police reports are likely to result in an underestimation of crashes. In addition, the use of different diagnostic categories across studies makes comparisons difficult. Sample sizes per diagnostic category often are small. Importantly, few studies failed to consider amount of driving exposure. It is not unreasonable to expect that individuals with a psychiatric disorder drive substantially less than age- and sex-matched controls in the general population. Thus, available estimates of crash risk are likely to be underestimations.

An important consideration when examining the crash rates of psychiatric patients is the role of suicidal motivation. A number of studies have examined this relationship with some studies reporting higher crashes rates for psychiatric patients with suicidal ideation and suicide attempts (Cushman et al., 1990; Elkema et al., 1970; Selzer and Payne, 1962). However, others suggest that suicide plays a limited role in motor vehicle crashes (Isherwood, Adam, and Hornblow, 1982; Schmidt, Shaffer, and Zlotowitz, 1977). It is important to note that the majority of studies in this area were completed before 1983, which may limit the findings. Even with that limitation, it may be that single-vehicle crashes involving drivers with psychiatric disorders could be used as a red flag for suicidal ideation in this population.

Future Research Considerations

A number of variables need to be considered in future research including: 1) diagnosis and criteria used to establish a diagnosis, 2) duration and severity of illness, 3) prescription medications-type of medication and compliance, and 4) amount of driving exposure. A major limitation in the existing literature is the lack of uniformity in diagnostic criteria across studies. Use of standardized diagnostic criteria (e.g., DSM-IV) in future studies would help to alleviate this limitation. In addition, comparison between the earlier and later studies is difficult because of methodological differences including differences in duration and severity of illness, use of control groups, and adjustments for driving exposure. The effects of psychotropic drugs (e.g., anti-psychotics, antidepressants, benzodiazepines, etc.) on driving performance are an important consideration when assessing crash risk. Unfortunately, there are few epidemiological field studies investigating this relationship, and those that are available have methodological limitations. Those studies that are available are examined in Section 13 (Drugs) of this review. In future studies, the inclusion of data on psychotropic drug use and the use of statistical control for drug use would be beneficial in advancing our knowledge of the effects of psychotropics on crash risk.

Table 33 Guidelines for Psychiatric Diseases *(Reproduced with permission)*

Illness	Austroads (1998)	CMA (2000)
Anorexia Nervosa and Bulimia Nervosa	No restrictions if condition stable.	Not addressed.
Anxiety or Depression	Psycho-neurosis (Anxiety or Panic Disorders) May drive if condition stable. Side effects of medications need to be assessed. Depression May drive if condition stable. Should not drive if being stabilized on medications. Those with severe depression, and impaired concentration and agitation should not drive. Need to assess all patients on medications carefully.	Emotional Disorders If disturbance severe enough to produce symptoms such as uncontrollable crying, severe depression, slowed psychomotor activity, preoccupation, or loss of sense of caution and good judgement, these persons should be warned not to drive until solution to problem is found. Side effects of drug therapy should be kept in mind.
Behavioral and Learning Disabilities	Not addressed.	Mental Disability, ADD, ADHD, Tourette's Syndrome Licensure for ADD/ADHD patients should be based on clinical assessment, where indicated, and positive response to treatment. Evaluation for those with behavioral and learning disabilities is probably best carried out using a road test by a professional driving instructor. Conditional licenses may be recommended for those who can drive in uncongested slower rural traffic but unable to drive safely in heavy city traffic or high-speed expressways. Individuals with difficulties with emotional control or attention span be referred for psychological testing.
Dementia	See Section 14 (The Aging Driver) for a review of dementia and guidelines.	
Personality Disorders	Personality Disorders No restrictions if condition stable and patient capable of safe and responsible driving. DLA should be informed.	Anti-Social Personality Disorder Those who show a complete disregard for accepted social values or who have a history of erratic, violent, aggressive, or irresponsible behavior *should never be approved* as medically fit without the most careful consideration.
Psychiatric Illness (Chronic)	No restrictions if condition stable.	Not addressed per se.
Psychiatric Illness (Past)	No restrictions if condition stable.	Not addressed.

Table 33 Guidelines for Psychiatric Diseases *(continued)*

Psychotic Illness	Acute: Should not drive during active phase. Non-Acute No restrictions if condition not acute and capable of safe and responsible driving.	Acute Should not drive. Recurrent Psychotic Episodes May drive during periods of remission if a consultant's assessment is favorable. Must file an annual report from their physician for 5 years following favorable consult.
Manic-Depressive Illness	No restrictions if condition stable Should not drive if in acute phase of mania.	Not addressed.

ADD = Attention Deficit Disorder ADHD = Attention Deficit Hyperactivity Disorder DLA = Driver Licensing Authority

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Section 13: Drugs

Introduction

A number of prescription and over-the-counter medications negatively affect cognitive performance (Bruera, Macmillan, Hansaon, et al., 1989; Golombok, Moodley, and Lader, 1988; Larson, Kukull, Buchner, et al., 1987; Salzman, Fisher, Nobel, et al., 1992; Tune and Bylsma, 1991). Relevant to this discussion are the findings that the use of medications, including those suspected of adversely affecting driving performance, increases with age (Ray, Gurwitz, Decker, and Kennedy, 1992). According to these authors, in the United States in 1988, the 65 and over age group comprised 12 percent of the population yet received 29 percent of all prescriptions, with approximately 11 prescriptions per person per year. Alberta seniors, representing 10 percent of the population, use more than 25 percent of all prescriptions and submit an average of 17 prescription claims annually (D.U.E. Quarterly, 1994). Other statistics on drug use in the elderly population reveal that the older individual uses an average of four prescriptions and two over-the-counter medications at any one time (D.U.E. Quarterly, 1994).

Medications commonly used by ambulatory elderly individuals include analgesics (opiods), antihypertensives, tranquillizers, antidepressants, antihistamines, and hypoglycemics (Colsher and Wallace, 1993; Ellinwood and Heatherly, 1985; Seppala, Linnoila, and Matilla, 1979). As noted by Ray, Purushottam, and Shorr (1993), benzodiazepines and cyclic antidepressants can impair the safety of the older driver, with sufficient data to raise concerns for opiods, antihistmaines, and sulfonlyureas.

Although many laboratory and experimental driving studies have documented drug-induced impairments on driving performance (see Janke, 1994, and Ray et al., 1993 for full reviews), medication use on its own is not an acceptable criterion for determining individual driving competence. The lack of clearly defined criteria for individual drugs is compounded by the presence of multiple drug regimes (polypharmacy), a common occurrence in this population. Moreover, the overall risk rating of polypharmacy cannot be determined by simply summing the risks of taking individual drugs (Wallace, 1997), making the task that much more daunting, if not impossible.

In addition to the effects of specific drugs and possible interactions of multiple drugs, consideration must be given to the pharmacokinetics (the absorption, distribution, metabolism, and elimination of drugs), and pharmacodynamics (the actions of drugs on the body) in the elderly population (see Catterson, Preskorn, and Martin, 1997 for a review). Both are likely to be altered in the elderly population because of normal aging, the presence of intercurrent illnesses, and the likely possible of drug-drug interactions. There have been calls for the development of a compendium of age-related medical conditions and drug interventions that could be used to inform physicians about which medical conditions and the severity levels, and which drugs and the dosages sufficiently impair driving abilities to warrant recommendations about driving cessation (Association for the Advancement of Automotive Medicine, 1996). However, the potential number of combinations of medical conditions, severity levels, drugs, dosages, and interactions is truly daunting and may in fact be the ultimate limiter of this approach. An alternative approach would be to evaluate the current status of the person, regardless of the medical condition(s), drug(s), or interactions that may be the causal agents of driving impairment, an approach that could be accommodated with the development and implementation of an empirically validated competence screen.

A review of the studies examining the effects on specific categories of drugs on driving performance is provided below.

A summary of the current fitness-to-drive guidelines (Drugs) for medical practitioners from Australia (1998) and Canada (2000) is presented in Table 34.

13.1 Antidepressants

Recent statistics indicate that of adult Americans 18 and over, more than 19 million will suffer from a depressive illness each year (National Institute of Health, 1999). antidepressants are the cornerstone of treatment for major depression, with tricyclic antidepressants and related newer compounds (Selective Serotonin Reuptake Inhibitors) the treatment of choice. There are currently a large number of tricyclic and other heterocyclic antidepressants on the market. In general, the major differences among compounds are in the degree of anticholinergic and sedative side effects, the major side effects associated with tricyclic use. Of the tricyclic antidepressants, amitriptyline and doxepin are highly sedating, with imipramine classified as moderately sedating. All of the tricyclic secondary amines (desipramine, protriptyline, and nortriptyline) are classified as having relatively low sedative effects. Trazadone is classified as being highly sedating, with

Table 34 Guidelines for Drugs *(Reproduced with permission)*

Guidelines for Drugs (Drivers of Private Vehicles)		
Illness	**Austroads (1998)**	**CMA (2000)**
Anticonvulsants	Once stabilized and cleared to drive patients should be warned about dosage changes and using other medication.	Patients should be closely observed and warned not to drive if drowsiness persists.
Anti-Infective Agents	Anti-inflammatory: Medication should be checked carefully for possible side effects.	Patients should be told of all possible reactions and warned about the danger of driving if they occur.
Analgesics	Codeine and other Opiates, Narcotics, Propoxyphene: Patients should be cautioned about driving if using these medications due to sedative side effects. Synthetic Narcotics (Methadone): May drive if patient under regular review and stable. Warn patient of dosage changes.	Opiates: After assessment for frequency and habituation, patients who use these opiates may warrant disqualification from operating any class of motor vehicle. Synthetic Narcotics (Methadone): Patients on a formal maintenance program of methadone prescribed by a physician are eligible for a license if recommended by the prescribing physician.
Antidepressants	The newer antidepressants should be used in preference if driving is an important issue. All patients should be cautioned when commencing these medications.	Patients should be carefully observed during the initial phase of dosage adjustment and advised not to drive if they show any evidence of drowsiness or hypotension. Patients who are stabilized on maintenance doses can usually drive any class of motor vehicle if they are symptom free.
Antihistamines	The newer antihistamines should be used in preference. Patients should be cautioned when starting these drugs.	Patients should be told of all possible reactions and warned about the danger of driving if they occur.
Anti-Emetics	Warn patients that this may affect ability to drive.	Not addressed.
Antihypertensives	Newer antihypertensives are a better choice but all drivers should be cautioned when starting new medication.	Not addressed.
Hallucinogens	Other Illicit Drug Use: Should not drive if there is clear evidence of abuse or dependence. Could be advised not to drive until cleared by specialist drug and alcohol unit.	Drugs such as cannabis and its derivatives (LSD, MDA) impair driving ability because they can drastically alter perception.
Sedatives	Chronic long term use of these drugs does impair ability to drive and all patients should be cautioned. Should not drive while being stabilized.	Mild Sedation: Can usually drive any type of motor vehicle without difficulty (if no drowsiness experienced). Heavy sedation: Should not drive any type of motor vehicle.

Table 34 Guidelines for Drugs *(continued)*

Stimulants	Other Illicit Drug Use: Should not drive if there is clear evidence of abuse or dependence. Could be advised not to drive until cleared by specialist drug and alcohol unit.	Patients who take stimulants must always be informed about the hazards of initial and prolonged use.

maprotoline moderately sedating, and amoxapine low in sedative properties.

Single doses in healthy volunteers of the more sedating antidepressants (e.g., amitriptyline, imipramine, and doxepin) have been shown to impair attention, memory, motor coordination, and open road driving (cf. Ray et al., 1993). As reported by Ray et al., amitriptyline and doxepin result in impairments in open-road driving comparable to a blood alcohol level of 0.10 percent.

In contrast to the older tricyclic antidepressants, the newer selective serotonin reuptake inhibitors (e.g., femoxetine, fluoxetine, fluvoxamine, sertraline, paroxetine, celexa, etc.) produce little or no impairments in performance (cf. Ray et al., 1993). O'Hanlon, Robbie, Vermeeren, van Leeuwen, and Danjou (1998) compared the effects of venlafaxine (Effexor), a selective serotonin and norepinephrine reuptake inhibitor to that of mianserin, a cyclic antidepressant, on simulated driving, psychomotor, and vigilance performance. Results from 37 healthy volunteers revealed that venlafaxine had no significant effects on psychomotor performance or on the primary driving parameter (standard deviation of lateral position). Mianserin, on the other hand, profoundly and consistently impaired both psychomotor and simulated driving performance. Vigilance, however, was significantly impaired with both venlafaxine and mianserin. Similar results have been reported by van Laar, van Willigenburg, and Volkerts (1995). Simulated driving and psychomotor performance of 24 healthy subjects were examined following the administration of nefazodone (a selective serotonin reuptake inhibitor) and imipramine (a cyclic antidepressant). Using double-blind, crossover, placebo-controlled methodology, impairments were noted on lateral position control in the simulated driving test with single doses of imipramine compared to no impairments following single doses of nefazodone. Minor impairments in psychomotor performance were noted with imipramine compared to no impairments with nefazodone.

Results from two epidemiologic studies on elderly drivers have shown an increased crash risk among those using antidepressant medications. In the Ray, Gurwitz, Decker, and Kennedy (1992) study, users of tricyclic antidepressants were found to have a 2.2 fold increase in crash rate compared to matched controls. A similar association was found in the Leveille, Buchner, Koepsell, et al. (1992) study. In that investigation, users of tricyclic antidepressants had a 2.3 fold increase in crash risk compared to matched controls. In conclusion, significant impairments in psychomotor and driving performance have been noted with the cyclic antidepressants. On the other hand, few impairments are evident with the newer selective serotonin reuptake inhibitors. For those individuals receiving cyclic antidepressants, assessments of driving performance may be warranted. At this time, there is little in the way of evidence to recommend assessments for those on selective serotonin reuptake inhibitors.

13.2 Antihistamines

Antihistamines are commonly prescribed to alleviate the symptoms of allergic reactions (American Medical Association, 1986). The older antihistamines (e.g., tripolidine, dephenhydramine, clemastine, terfenadine), which cross the blood-brain barrier and affect the central nervous system, are associated with pronounced sedation and impairments in psycho-motor performance, effects likely to negatively affect driving performance (Gengo and Mechtler, 1990; O'Hanlon, Vemeeren, Uiterwijk, van Vegel, and Swijgman, 1995). The newer generation of antihistamines (e.g., loratadine, fexofenadine, zyrtec) which do not cross the blood-brain barrier has been shown to have potent anti-allergic effects, but, in therapeutic doses, are largely free from the sedating effects of the older antihistamines (Gengo and Mechtler, 1990; Meltzer, 1990; Ray et al., 1992).

There is a small body of literature that suggests that the older antihistamines are associated with increased crash risk. In an epidemiologic study, Warren, Simpson, Hilchie, et al. (1981) found that drivers killed in automobile crashes attributed to their own error were one and a half times more likely to have been using one of the older, sedating antihistamines. Ray et al. (1992) found a 20 percent increase in crash involvement in older drivers using antihistamines, but this increase was not statistically significant.

In healthy volunteers, sedating antihistamines are associated with impaired psychomotor performance and impairments in simulator and open-road driving conditions (cf. Ray et al. 1993). In contrast, a number of studies have not found these impairments with the newer non-sedating compounds (cf. Ray et al., 1993).

In general, significant impairments have been noted with the older antihistamines, with few, if any, impairments noted with the second-generation antihistamines. As a result, warnings about the adverse effects of the older antihistamines on driving performance are warranted. However, for individuals taking therapeutic doses of the newer antihistamines, those warnings appear unnecessary.

13.3 Benzodiazepines

Benzodiazepines are the most commonly used medications for the treatment of anxiety and insomnia (Ray et al., 1993), and one of the most frequently used classes of drugs by elderly people (Baum, Kennedy, Knapp, et al., 1988; Ray et al., 1993). Benzodiazepines can be divided into those with a short half-life (e.g., lorazepam, alprazo, triazolam, oxazepam, temazepam) and those with a long half-life (e.g., clonazepam, clordiazepoxide, diazepam, halazepam, prazepam, clorazepate, flurazepam). In general, the duration of action for those with a short half-life is two to four hours, and six to eight hours for those with a long half-life. Side effects that may adversely affect driving performance include sedation, drowsiness, prolonged psychomotor times, incoordination, memory loss, vertigo, dizziness, and double vision (see Ray et al., 1993 for a complete review).

O'Hanlon and colleagues have conducted a number of studies examining the relationship between benzodiazepines and road tracking behavior in non-elderly populations (O'Hanlon et al., 1982; O'Hanlon and Volkerts, 1986; Brookius, Volkerts, and O'Hanlon, 1990; O'Hanlon et al., 1995). Significant impairments in lane tracking behavior were evident in the drug condition compared to performance in the placebo condition. In the most recent study, O'Hanlon et al. (1995) examined the effects of benzodiazepines (diazepam and lorazepam), beonzidiazeoine-like anxyiolytics (alpidem and suriclone), and a 5-HT agonist (ondanstron) on a standardized simulated road-tracking test. Study participants consisted of healthy young controls (22 to 43 years) and patients with anxiety (24 - 64 years). In a double blind, placebo-controlled design, the participants were tested on the simulated road-tracking test two to three times after taking one of the drugs for eight to 15 days. There were no significant differences in simulated driving performance between the two groups in the baseline, placebo, and ondanstron conditions. However, significant impairments in simulated driving performance were noted in the benzodiazepine and benzodiazepine-like drug conditions.

Analyses of studies using crash data also suggest a relationship between benzodiazepine use and impaired driving performance. Skegg, Richards, and Doll (1979), in a prospective study, identified 57 drivers involved in motor vehicle crashes resulting in death or hospital admission. Minor tranquilizer use was five times greater in the motor vehicle crash group compared to a control group matched for age, sex, and medical group practice. Honaken, Ertama, Linnoila, et al. (1980), using case-control methodology, studied 203 survivors of motor vehicle crashes attending an emergency department within six hours of the crash. Motorists stopped at service stations were used as controls. Cases and controls were matched for weekday, hour, and location of crash. Crash victims were significantly more likely than controls to have benzodiazepines in their blood (Cases: 10 of 203; Controls, 7 of 325). Oster, Russell, Huse, Adams, and Imbimbo (1987) examined the health utilization of 7,271 benzodiazepine users and 65,439 non-users. Results revealed higher accident-related health care utilization among benzodiazepine users. Based on prescription and driving records of 16,262 seniors (age 65 and older), Ray et al. (1992) found that current users of benzodiazepines had injurious crash rates 1.5 times higher than individuals with no psychoactive drug use. Results also revealed a dose-dependent relationship. The crash rates of benzodiazepine users at the lowest therapeutic level were approximately equal to that of the controls. However, those at the highest therapeutic level had crash rates 2.4 times higher than controls.

Using Saskatchewan health administrative databases on hospital admission, Neutel (1995) compared the risk of becoming involved in an injurious motor vehicle crash for 148,000 patients receiving a prescription for benzodiazepines to that of 98,000 controls. In the first week after their prescriptions were filled, patients had a 13.5 fold increase in risk for injurious crashes compared to controls. That risk decreased to 2.6 after four weeks, with no measurable effects after the four-week period, suggesting tolerance after long-term drug use. Similar findings were reported by Van Larr et al. (1992). In a study of elderly drivers in Quebec, Hemmelgarn, Suissa, Huang, Boivin, and Pinard (1997) compared injurious crash rates of 5,579 older drivers using benzodiazepines to a group of 13,256 controls. After adjusting for age, sex, chronic disease score (derived from drug use), and exposure to other drugs, the rate ratio for those with benzodiazepine use was 1.28. For those

individuals on long-acting benzodiazepines, the risk was the highest in the first week and remained higher than controls for continuous use of longer duration up to a period of one year. There was, however, no evidence of increased crash risk for those on the short-acting benzodiazepines.

Unlike the results of Hemmelgarn et al. (1997), Leveille et al. (1994) failed to find a relationship between benzodiazepine use and motor vehicle crashes resulting in injuries. In this investigation, injurious crash rates of 234 elderly drivers were compared to those of 447 controls matched for age, sex, and county of residence. Differences in the results between the two studies may, in part, be due to the fact that the most widely used benzodiazepine used in the Leveille study was triazolom, a short acting benzodiazepine, whereas both short- acting and long-acting benzodiazepines were examined in the Hemmelgarn study. In fact, the results from Leveille are consistent with those of Hemmelgarn when only short-acting benzodiazepines are considered. In general, despite the methodological strengths and weaknesses of the numerous studies, the findings suggest that benzodiazepine use is associated with an increased risk for motor vehicle collisions, particularly at higher doses and with long half-life compounds.

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Section 14: The Aging Driver

14.1 Sensory Decline
14.2 Motor Decline
14.3 Cognitive Decline
14.4 Dementia

A summary of the current fitness-to-drive guidelines (The Aging Driver) for medical practitioners from Australia (1998) and Canada (2000) is presented in Table 36.

14.1 Sensory Decline

Several biological changes occur in the sensory organs with age. Hearing impairments begin to increase around the age of 40 and show sharp increases after age 60. Approximately 20 percent of people aged 45 to 54 experienced some difficulties with hearing compared to 75 percent of those between 75 and 79 years of age (Fozard, 1990). Although common in elderly populations, it is unclear what effects hearing loss has on driving ability. Research suggests that individuals with hearing loss tend to compensate for their disability with increased visual attentiveness (National Center for Health Statistics, 1986). The results from a population-based case-control study by McCloskey, Koepsell, Wolf, and Buchner (1994) revealed no significant association between impaired hearing and injury collision in a sample of individuals 65 and over. However, individuals who used hearing aids while driving had approximately twice the risk of age- and sex-matched controls. Given the paucity of data, many jurisdictions do not prohibit individuals with hearing loss from driving private vehicles (British Columbia Medical Association, 1997; State of Maine, 1994; State of Utah Functional Ability in Driving, 1992).

Visual capacities also become less efficient with advancing age and would appear to be more relevant to the driving process. It has been estimated that approximately 90 percent of the information required for driving is acquired visually (Hills, 1980). Changes in visual function with age include decreased static and dynamic visual acuity, decreased temporal fields, decreased resistance to glare, and reduced low luminescence vision (see Klein, 1991; Owsley and Ball, 1993; Reuben, 1993).

Several state and federally sponsored studies also have examined the relationship between vision and crashes in large samples (N >1000) of older adults (Henderson and Burg, 1974; Hills and Burg, 1977; Shinar, 1978). Somewhat surprisingly, most studies report only weak associations between static visual acuity and elderly driver crash rates, with the correlations accounting for less than 5 percent of the variance. McCloskey et al. (1994) examined the relationship between visual impairments and the risk for motor vehicle collision injuries. Study subjects were 235 licensed drivers 65 years of age and older who had sustained injuries in a police-reported crash occurring in 1997 or 1998. Controls were 448 drivers who experienced no such injury during the same time period and who were matched to the study cases by age, sex, and county of residence. In addition, the two groups were similar in terms of number of miles driven per year, percentage of driving done at night, and frequency of driving alone. Results revealed no significant increase in the risk of an injury collision with impaired visual acuity.

Owsley, Ball, Sloane, Roenker, and Bruni (1991) examined the relationship between visual functioning and state-recorded crashes in 53 individuals 57 years of age and older. The strongest predictor of vehicle crashes was the size of the useful field of view (UFOV)-a test of higher order cognitive functioning. Pertinent to this discussion, the authors found no direct relationship between eye health or visual functioning and crashes. As noted by Owsley and Ball (1993), methodological considerations may play a role in why studies have failed to establish a link between vision and driving performance (e.g., the statistical burden of predicting an improbable event, the problem of underreporting with self-reported crash data, etc.). In addition, measures of visual function (e.g., visual acuity, contrast sensitivity) used in previous studies most likely fail to measure the visual complexity required for everyday driving performance (Ball and Owsley, 1991). However, static visual acuity continues to be the primary vision-screening test for driving in North America and many parts of Europe (Bailey and Sheedy, 1988).

Because the act of driving primarily involves the ability to discriminate an object when there is relative movement between the object and observer, tests of dynamic visual acuity rather than static visual acuity would seem to be more relevant for assessments of safe driving performance. In contrast to static visual acuity, dynamic visual acuity is a reliable predictor of crash probability (Fox, 1989; Graca, 1986; Reuben, Silliman, and Traines, 1988). In view of this, it is surprising that dynamic visual acuity is seldom, if ever, included in traditional license renewal assessments. Importantly, declines in dynamic visual acuity and lateral motion detection start at an earlier age and accelerate faster, whereas deterioration in static visual acuity occurs later and progresses more slowly (Shinar and Schieber, 1991).

Reductions in visual acuity under levels of low luminescence occur with age. Sixty-five has been suggested as the critical average age after which visual acuity becomes significantly impaired under conditions of reduced illumination (Sturr, Kline, and Taub, 1990). Interestingly, several reports cite visual problems as frequently motivating a change in driving patterns (Kosnick, Sekuler, and Kline, 1990; Retchin, Cox Fox, and Irwin, 1988; Stewart, Moore, Marks, May, and Hale, 1993) although there is evidence that older drivers have little or no insight into their vision problems (Flint, Smith, and Rossi, 1988). Despite the obvious role of vision for driving, the evidence is that the visual deficiencies are only weakly related to crashes (Kline, Kline, and Fozard, 1992; McCloskey et al., 1994).

14.2 Motor Decline

Key motor factors in driving, as identified by Marottoli and Drickamer (1993), include trunk and neck mobility, range of motion of extremities, strength, and proprioception. Declines in the first three factors are associated with increasing age, with conflicting reports regarding the effects of age on proprioception. McPherson, Michael, Ostrow, et al. (1988) report a positive correlation between joint range of motion and physical driving ability. Specifically, older people with less flexibility had reductions in driving abilities compared to those with more flexibility.

Reaction time and motor response times are known to increase with age (Carr, Jackson, Madden, and Cohen, 1992; Graca, 1986). Marottoli and Drickamer (1993) suggest that although there are age-related decrements in the sensory, central processing, and motor components of reaction time, central processing changes appear to be the major contributor to slowing.

14.3 Cognitive Decline

Within the information-processing framework, the driver is conceptualized as an information processor who actively and selectively processes different sources of information associated with the driving task, usually under temporal constraints (Shinar, 1978). Previous investigators have noted the relationship between higher order processes and safe driving records (Ball and Rebok, 1994; Parasuraman and Nestor, 1991; Shinar and Schieber, 1991). In particular, requirements for safe driving include the ability to understand and remember traffic rules and signs, follow directions, develop and utilize problem-solving skills, and maintain concentration and attentional skills.

The most commonly occurring traffic violations leading to increased crashes of older drivers include failure to obey signs including stop signs and red lights (Gloag, 1991; Graca, 1986; Keltner and Johnson, 1987), unsafe left turns (Keltner and Johnson, 1987; Kline et al., 1992; Staplin, Breton, Haimo, Farber, and Byrnes, 1987), inappropriate turns (Keltner and Johnson, 1987; Kline et al. 1992), unsafe passing (Graca, 1986), and failure to yield (Cerelli, 1989; Graca, 1986; Kline et al, 1992). In fact, 'failure to yield the right of way' is listed as the primary cause of older driver crashes as early as age 50 (Gebers, Romanowich, and McKenzie, 1993). It is reasonable to argue, however, that the source of traffic violations are due not to an 'obedience' problem (e.g., failure to obey stop signs) but rather to attentional errors.

The specific increase in elderly driver crossing accidents (one vehicle crossing the path of another) has been attributed to deficiencies in attentional capacity (Hakamies-Blomqvist, 1993), with attentive errors proposed as a major cause of elderly driver crash rates (Hakamies-Blomqvist, 1994; Keltner and Johnson, 1987). Studies of divided attention suggest that strategies for allocation of attention are similar between young and old (Brouwer, Waterink, Van Wolffelaar, and Rothengatter, 1991). However, older adults show greater performance deficits on a variety of divided attention tasks, particularly when task demands are high (Crook, West, and Larabee, 1993; Guttentag, 1989, Kausler, 1991; Ponds, Brouwer, and Van Wolfelaar, 1988; Salthouse, Rogan, and Prill, 1984). For example, Staplin et al. (1987) report comparable performance in terms of accuracy between young and old on divided attention tasks involving simple target detection. However, deficits on more complex divided attention tasks begin to appear in middle age.

One of the better predictors of crash frequency is a test that is said to measure visual attention at the preattentive level. The test is based on the useful field of view (UFOV) or that area in the visual field over which information can be acquired during a brief glance (Sanders, 1970). Ball and Owsley (1991) examined the performance of older drivers on a task measuring UFOV. Utilizing a pass/fail criterion, measures on a number of UFOV subtests included subjects' performance on a central task alone, the combination of a central task with an uncluttered peripheral task (divided attention) and finally, a dual task with distracters in the peripheral field (distractibility). Elderly individuals who failed the UFOV had 15.6 times more intersection accidents than those individuals who passed. Of relevance to aging driving research is the reported reduction in the size of the UFOV with age (Ball, Beard, Roenker, Miller, and Griggs, 1988).

Selective attention, or the ability to selectively attend to a target stimulus in the presence of distracters, also is important to driving. A number of different paradigms have been used to assess changes in selective attention as a function of age (e.g., visual card sort, dichotic listening task, embedded figures test). Results from a visual search card-sorting task revealed significant age differences in sort time with older adults being disproportionately affected by the presence of irrelevant stimuli (Rabbitt, 1965).

In a review of the selective visual attention literature, Plude and Hoyer (1986) proposed that age decrements in selective attention are due to a decline in the ability to locate task relevant information in the visual field. A number of studies lend support to Plude and Hoyer's spatial localization hypothesis. In particular, when targets are easily discriminable from distracters, as when target and distractor differ in color (Nebbes and Madden, 1983), or when target locations are specified a priori (Plude and Hoyer, 1986), age differences in search performance are slight or nonexistent.

Attention switching is the process of alternately monitoring two or more sources of input. Despite its importance in the driving situation, little work has been done on this aspect of attentional functioning. In an early selective attention/driving study of young professional bus drivers, Kahneman, Ben-Ishai, and Lotan (1973) reported significant correlations between omission, intrusion, and attentional shifting errors. It is of particular interest that the highest correlations were obtained for the switching measure of selective attention. Attentional switching, as conceptualized by Parasuraman and Nestor (1991), involves the processes of engagement or the "initial adoption of a focused attentional state" and disengagement or the "ability to re-orient attention to another channel" (p. 545). Studies cited in Parasuraman and Nestor reveal that patients with both mild and moderate Dementia of the Alzheimer Type show an impaired ability to disengage, although their ability to engage attention is relatively spared. The relationship between attentional switching and normal aging requires further study.

Early research on aging and driving focused on sensory and motor changes (Burg, 1967; Cox, Fox, and Irwin, 1988; Fox, 1989; Planek, 1972). However, other research (Brouwer et al., 1991; Crook et al., 1993; Owsley et al., 1991; Parasuraman and Nestor, 1991) has focused more on higher level functioning of the cognitive apparatus (e.g., perceptual rather than sensory processing and attention rather than reaction times or motor speed). Evidence suggests that the skills putatively relevant to safe driving do deteriorate with age (Graca, 1986). However, except for very elderly persons, the effects of age, per se, are unlikely to account for many crashes. An important and often overlooked consideration is that changes due to age are both complex and variable across time and among individuals (Blanchard-Fields and Hess, 1996). Indeed, many older adults retain the ability to drive competently and safely, and basing decisions to drive on chronological age is neither practical nor warranted. Recent and earlier evidence confirms that medically compromised drivers make up a substantial subset of drivers who crash, especially when the crash involves an older driver. Identifying those older adults whose abilities have been compromised to an unsafe level, rather than targeting the entire older population, is both appropriate and more likely to be effective.

14.4 Dementia

A potentially high-risk group of older drivers are those with a dementing illness (Dobbs, 1997; Dubinsky, Williamson, Gray, and Glatt, 1992; Gilley, Wilson, Bennett, et al., 1991; Kaszniak, Keyl, and Albert, 1991). Prevalence estimates from the Canadian Study on Health and Aging (1994) suggest that eight percent of all Canadians aged 65 and older met the criteria for dementia, increasing to a staggering 34.5 percent for those age 85 and older. Alzheimer's disease, the most common form of dementia, currently has a prevalence rate of 5.1 percent overall or 161 cases per one hundred thousand with a projected prevalence rate of 509 cases per 100,000 by the year 2031 (Canadian Study on Health and Aging, 1994).

Alzheimer's disease (AD) is a progressive, degenerative brain disorder characterized by impairments in multiple cognitive functions. The earliest cognitive symptoms include difficulties in recent memory, word finding, confrontation naming, orientation, and concentration. Slowed rates of information processing, attentional deficits, disturbances in executive functions, impairments in language, perception, and praxis characterize the cognitive changes in later stages. The impairments in cognitive functioning increasingly interfere with social and occupational functioning, including the driving abilities of affected patients. Because of the progressive nature of the disease, at some point in the course of their illness, all individuals with a progressive dementia will become incapable of driving safely and will eventually stop driving. However, many patients continue to drive after the onset of their illness. An early study of retirement community residents indicated that 31 percent of those driving were suffering from a dementia (Waller, 1967), and 28 percent of patients (192) referred to a dementia clinic were active drivers (Odenheimer, 1993). Moreover, research

suggests that many patients continue to drive for as long as four years following initial diagnosis (Friedland et al., 1988, Gilley et al., 1991; Lucas-Blaustein et al., 1988).

The problems associated with driving in this population are receiving increasing attention, and research findings using crash data suggest there is cause for concern. Results from one of the earliest studies examining the crash risks associated with dementia were reported by Waller (1967) who compared the driving records of 83 normal older drivers to those of 82 older drivers described as 'senile', 80 drivers with cardiovascular disease, and 199 drivers diagnosed with dementia and cardiovascular disease. The comparisons revealed crash rates of 12.1, 19.3, 14.7, and 36.2 crashes per million miles driven for the four groups, respectively. More than twenty years later, Friedland et al. (1988) compared the driving history of 30 patients with dementia of the Alzheimer's type (DAT) and 20 healthy age-matched controls. Results from this investigation revealed that individuals with DAT were nearly five times more likely to have had a crash than healthy elderly controls. Thirty percent of the dementia patients studied by Lucas-Blaustein et al. (1988) had at least one crash and another 11 percent were reported to have caused a crash since the onset of the disease.

Recent research corroborates these early findings of increased rates of crashes for dementia patients compared to age-relevant population rates and/or elderly controls. As shown in Table 35, the most well documented finding from retrospective studies is that patients with dementia have crash rates that far exceed those of non-dementing seniors (Cooper et al., 1993; Drachman and Swearer, 1993; Dubinsky et al., 1992; Friedland et al., 1988; Gilley et al., 1991; Lucas-Blaustein et al., 1988; O'Neill et al., 1992; Tuokko et al., 1995). Despite the variation in the retrospective methodology, the majority of the evidence provides a clear indication that, as a group, patients with dementia who continue to drive pose a considerable public safety risk. It is interesting to note that all studies find a small but significant subset of dementia patients competent to drive, a point that will be addressed shortly.

In addition to retrospective surveys and driving record examinations, a number of studies have examined the driving ability of patients with dementia using on-road assessments (Cushman, 1992; Dobbs, 1997; Fitten et al., 1995; Hunt et al., 1993; Kapust et al., 1992; Odenheimer et al., 1994; Shemon and Christensen, 1991). As evidenced from the results in Table 35, dementia patients, in general, perform less well than their counterparts without dementia on tests of on-road performance.

Of particular note to this discussion is the use of the Mini Mental Status Exam [MMSE] (Folstein, Folstein, and McHugh, 1975) as a predictor of driving competency in individuals with a dementia. The MMSE is a short screening instrument of cognitive status consisting of questions and tasks designed to assess orientation to time and place, registration of verbal information, attention and calculation, recall, language and visual construction. Importantly, a number of organizations, including medical ones, have suggested the use of the MMSE to screen drivers. In fact, according to results from Miller and Morley (1993), the majority of physicians surveyed felt the MMSE was the best available mental status examination for fitness-to-drive evaluation. However, the evidence to date indicates that the MMSE is of questionable utility for identifying individual driving competency.

A number of retrospective studies compared the MMSE scores of patients with dementia, who reported involvement in at least one crash, with patients who were not involved in car crashes. In many cases, the difference in MMSE scores between the two groups was less than one point (Friedland et al., 1988; Gilley et al., 1991; Lucas-Blaustein et al., 1988). No differences in MMSE scores between patients with diminished driving ability and patients with preserved driving ability have been reported by O'Neill et al. (1992). Using a global severity score (rather than MMSE), Drachman and Swearer (1993) found no difference between patients who had a collision and those who had not.

The data from studies that have examined on-road performance provide a somewhat different picture, with reliable correlations reported between MMSE scores and on-road performance. Odenheimer et al. (1992) reported a substantial correlation ($r = .72$) between the MMSE score and their in-traffic driving score. Similarly, Hunt et al., (1993) found that a combined score from the Clinical Dementia Rating scale was related to driving outcome on a road test (pass/fail) using Kendall's Tau Coefficient, a measure of association between ranked data ($\tau = .50$). Fitten et al. (1995) reported that drive scores from their specially designed road test were strongly correlated with a transformed MMSE score ($r = -.63$). However, in the Fitten et al. study, mental status scores did not correlate with drive scores at the upper end of the MMSE scale. Although the correlations reported above show a clear relationship between mental status as measured by the MMSE and driving performance over a group of individuals, they do not indicate that the MMSE score is a sufficient predictor of the driving performance of individuals.

Table 35: Summary of the Research Literature on Driving and Dementia

Study	Type	Sample Size	Diagnosis	MMSE	Criterion	Results
Dobbs et al. (1997)	Prosp.	P = 115 C_o = 35 C_Y = 23	Dementia (Mostly AD)	P = 23.4 C_o = 28.5 C_Y = 29.6	Road Test Neuropsych. Battery Neurocog. Battery	Isolated 3 categories of driving errors 1. Non-Discriminating-made by all drivers. 2. Discriminating-rarely by young, most often by old, frequently by cognitively impaired. 3. Hazardous or Catastrophic-made only by cognitively impaired.
Fitten et al. (1995)	Prosp.	P_{AD} = 15 P_{MID} = 12 C_{DIAB} = 15 C_{HO} = 26 C_{HY} = 16	AD/MID	P_{AD} = 23.2 P_{MID} = 25.4 C_{DIAB} = 27 C_{HO} = 29.2 C_{HY} = 29.9	Road Test Neurocog. Tests C/G Crash Reports	Dementia patients significantly worse on road test. Collisions/Moving violations/10^4 miles driven: P_{AD} = .214 P_{MID} = .156 C_{DIAB} = .014 C_{HO} = .028
Hunt et al. (1993)	Prosp.	P = 65 C = 38	AD_{VMild} (12) AD_{Mild} (13)	CDR Scale	Road Test Neuropsych. Tests	40 percent of mild AD failed road test. All controls and AD_{VMild} passed the road test
Harvey et al. (1995)	Prosp.	P = 13	AD (10) Focal Dementia (3)	--	Driving Simulator	54 percent patients had 'normal' performance. 46 percent patients had 'poor' performance.
Rizzo et al. (1997)	Prosp.	P = 21 C = 18	AD	--	Driving Simulator	29 percent of AD 'crashed' versus 0 percent for controls. AD 2 times as likely to experience close calls compared to controls.
Drachman & Swearer (1993)	Retros Survey	P = 83 C = 83	AD M_{dur} = 4.18 yrs (± 2.71)		C/G Reported Crashes/Year	M_{AD} = .091 c/d/y M_c = .040 c/d/y

Table 35: Summary of the Research Literature on Driving and Dementia (*continued*)

Friedland et al. (1988)	Retros. Survey	P = 30 C = 20	AD M_{dur} = 5.5 yrs (± 2.0)	P = 19.9 (+ 6.3) (At time of first crash)	C/G Reported Crashes/Year	AD = 14 crashes for period of study. C = 2 crashes for period of study.
O'Neill et al. (1992)	Retros. Survey	P_{AD} = 43 P_{MID} = 7 P_{MIXED} = 5 P_o = 2	AD MID Mixed	18.7 (± 5.4)	C/G Reported Crashes/Year	29 percent of patients involved in crash.
Cooper et al. (1993)	Retros. Survey	P = 165 C = 165	AD	—	State Records-Crashes and Violations	AD = 61 versus Controls = 25 c or c/d/y. AD = .15 c/d/yr. C = .06 c/d/yr.
Trobe et al. (1997)	Retros. Survey	P = 143 C = 715 (Matched)	AD M_{dur} = 2.57 yrs (±1.59)	14.8 (±6.4)	State Records-Crashes and Violations Neuropsych Tests	AD = 39 crashes. Controls = 199 crashes. AD rate = .05-.08. Controls = .05-.08.
Tuokko et al. (1995)	Retros. Survey	P = 165 C_{ill} = 84	M_{dur} = 4.54 yrs (± 3.23)	Mild	State Records-Crashes and Violations	Dementia patients 2.2 times the crash rate of matched controls. Multiple medical problems 2.2 times the crash rate as matched controls.

P = Patient
AD = Alzheimer Disease
C = Controls
C_o = Old Control
C_y = Young Control
DPT = Driver Performance Test
DAS = Driver Advisement System
C_{HO} = Healthy Older Controls
C_{HY} = Healthy Younger Controls
**c or c/d/y = crashes or crashes /driver/year equivalent*
Prosp = Prospective Study
C/G = Caregiver
CDR = Clinical DementiaRating
RT = Reaction Time
MMSE = Mini Mental State Examination

It is generally well accepted that dementia patients who drive, as a group, pose substantial safety problems. However, clear cut guidelines for the identification and evaluation of individual at-risk drivers are lacking. In some reports, the investigators have argued that as soon as a diagnosis of dementia is made, a recommendation be given not to drive (Friedland et al., 1988; Lucas-Blaustein et al., 1988). Others, however, have argued that diagnosis alone is insufficient for revocation of a driver's license (Drachman, 1988; Drachman and Swearer, 1993; Fitten et al., 1995). One of the problems with using diagnosis is that severity does not correlate sufficiently with driving performance to be a valid criterion for determining driving cessation (Johannson et al., 1997). According to Drachman (1988) "the limitation of the privilege to drive should be based on demonstration of impaired driving competence rather than a stigmatizing label, such as AD" (p. 787). This is a strong argument given the fact that, although many drivers with dementia as a group do have high crash rates, a significant minority of patients with early dementia show no evidence of deterioration of driving skills. Importantly, recent empirical evidence suggests that almost one third of drivers with a dementing illness are competent to drive in the early stages of their illness (Dobbs et al., 1997, Fitten et al., 1995; O'Neill, 1992). In light of this evidence, restrictions in driving or de-licensing based on a medical diagnosis of dementia alone not only unfairly penalizes the patient, it can limit their independence and mobility in the early stages of the disease when they may still be competent to drive.

Table 36 Guidelines for the Aging Driver *(Reproduced with permission)*

Guidelines for the Aging Driver (Drivers of Private Vehicles)		
Illness	**Austroads (1998)**	**CMA (2000)**
Age	Advanced age is not in itself a barrier to driving. Therefore, in assessing an older person's ability to drive safely, it is important to consider his or her functional ability, rather than chronological age.	Although the rate of physical and mental decline varies greatly from person to person, the physiologic changes that accompany aging eventually affect everyone's driving ability. The borderline is often hazy between a hazardous deterioration and a decline that can be compensated for by long experience and voluntary limitation of driving. Many drivers, as they grow older, find it increasingly difficult to cope with the power and speed of the modern automobile and the progressively increasing traffic congestion on both urban and rural roads. Keeping in step with the traffic on high-speed freeways requires the utmost concentration.
Mental Deterioration	Adequate cognitive functioning is important to the driving task Ability to carry out the following processes should be gauged in assessing driving competence: 1. Attention. 2. Concentration. 3. Thought processing. 4. Problem solving skills. 5. Memory.	Slowed reaction time, lack or attentiveness, poor judgment, and faulty attitudes are responsible for many crashes at all ages. These factors assume an increasing importance with advancing years. An older driver who is physically fit may be quite unable to drive safely on today's crowded streets because of mental deterioration.
Multiple Physical Defects	Frequently an older driver has several minor physical defects, each of which taken separately may not affect driving ability very much. However, when taken together these defects may make driving potentially dangerous, particularly if the defects are accompanied by some slowing of ability to convert perception and judgement into timely action.	An older person often has several minor physical defects, each of which if taken separately may not affect driving ability very much, but when taken together may be dangerous. The hazards increase if these physical defects are accompanied by some slowing of the ability to convert perception and judgement into timely action.
Progressive Dementia	<u>Intellectual Impairment</u> Should not drive. The DLA will require a test by a driving assessor before considering issue of a license or conditional license.	With documented dementia, the operation of any motor vehicle is risky. Individuals identified with possible dementia must have an assessment of their cognitive function. Individuals scoring less than 24 on the Mini-Mental State Examination (MMSE) are ineligible to hold a driver's license of any class pending complete neurological assessment. Individuals suspected of showing poor judgement, poor reasoning ability, poor abstract thinking, and poor insight also should be evaluated for driving ability even if they have a MMSE score of 24 or higher.

DLA = Driver Licensing Authority

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Section 15: The Effects of Anesthesia and Surgery

15.1 General Anesthesia
15.2 Outpatient surgery
15.3 Major Surgery

A summary of the current fitness-to-drive guidelines (Anesthesia and Surgery) for medical practitioners from Australia (1998) and Canada (2000) is presented in Table 39.

15.1 General Anesthesia

General anesthesia involves depression of the central nervous system. In the late 1950's and early 1960's, it was believed that the drugs used to produce anesthesia continued to exert effects on cognitive function for a period of time following anesthesia (Bedford, 1955). Since then, a number of researchers have investigated the effects of anesthesia on cognition by comparing cognitive performance in patients having either general or regional anesthesia (See Table 37). There is, however, considerable variability among the studies in age of sample, type of surgical procedures (cataract, total hip/knee replacement), the tests used to assess cognitive functioning, type of pre-medication and sedation used, and time of assessment. In general, however, the results have failed to find differences in cognitive functioning in the post-operative period between patients receiving either a general or regional anesthetic.

Despite the lack of significant differences in cognitive performance in patients receiving either a general or regional anesthetic, there is considerable evidence that declines in cognitive functioning occur in the post-operative period. A number of factors have been proposed to account for the changes in cognition following surgery. Factors include physiological effects of the anaesthetic such as hyperventilation, hypotension, and/or hypoxia, and changes in the role of catecholamines or cholinergic transmission within the central nervous system (See Dodds and Allison, 1998 for a complete review). Regardless of the causal agent, there is considerable evidence that declines in cognitive functioning occur following surgery (See Table 38 for a summary of the findings).

Research suggests that the elderly population is particularly at risk. Ritchie, Polge, deRoquefeuil, Djakovic, and Ledesert (1997) recently reviewed the literature in order to describe post-operative cognitive impairment in elderly individuals. Results of the review indicate that significant cognitive impairment was a common finding in elderly persons 1 to 3 days post-surgery.

Table 37 Summary of Studies Examining Differences in Cognitive Impairment between Patients Receiving Either a General or Regional Anesthetic

Study	n	Surgery	Age (Range/Mean)	Results
Hole et al. (1980)	60	Total Knee Replacement.	56-84 years Mean = 71	↓ Mental function in 7/31 general anesthetic group. No change in epidural group.
Riis et al. (1983)	30	Total Hip Replacement.	> 60 years	↓ Learning and retention scores day 2 post-operatively, normal on day 7 post-op for both general and regional anaesthetic groups.
Jones et al. (1990)	146	Total Hip Replacement. Total Knee Replacement.	> 60 years	No change on tests of choice reaction time and critical flicker fusion threshold between individuals receiving general or regional anaesthesia.
Campbell et al. (1993)	169	Cataracts.	65-98 years	↓ at 24 hrs in verbal recall, verbal learning, psychomotor speed, and tactile naming in both general and regional anaesthesia groups. General anaesthesia group decrease was greater but results not significant. Recovery in both groups by 2 weeks post-operatively.
Williams-Russo et al. (1995)	262	Total Knee Replacement.	> 40 years Mean = 69	Generalized decline at 1 week followed by a return to baseline or improvement by 6 months in both general and regional anaesthesia groups.

Table 38 Summary of Studies of Effects of General Anesthesia on Cognitive Functioning Post-Operatively (*from Dodds, C., & Allison, J. (1998). Postoperative cognitive deficit in the elderly surgical patient, British Journal of Anaesthesiology, 81, 449-462, ©The Board of Management and Trustees of the British Journal of Anaesthesia.* Reproduced by permission of Oxford University Press/*British Journal of Anaesthesiology*)

Study	n	Surgery	Age (Range /Mean)	Results
Hole et al. (1980)	60	Total Knee Replacement.	56-84 years. Mean = 71.	↓ mental function in 7/31 General Anaesthesia group. No change in epidural group.
Kaarhunen et al. (1982)	60F	Cataract Surgery.	> 65 years.	↓ in Wechsler Memory Scale and Luria tests at 1 week post-op.
Riis et al. (1983)	30	Total Hip Replacement.	> 60 years.	↓ in learning and retention scores day 2 post-op, normal on day 7 post-op for both general and regional anaesthetic groups.
Bigler et al. (1985)	40	Acute Hip Replacement.	> 60 years. Mean = 78.9.	Abbreviated Mental Test at one week.
Smith et al. (1986)	85	Orthopedic. Gynecological. General.	Young (50). Old (69).	↓ memory (all ages). ↓ orientation & concentration in older patients.
Chung et al. (1987)	44	Transurethral Prostatectomy.	60-93 years. Mean = 72 years.	↓ at 6 hours on recall, attention, calculation (MMSE). No differences at day 5 post-op.
Ghoneim et al. (1988)	105	Hysterectomy. Prostate. Joint.	25-86 years.	↓ Paired Associate Learning post-operatively. Marked improvement at 3 months post-op.
Hughes et al. (1988)	30	Total Hip Replacement.	50-80 years.	No change in recall. ↓repeat recall and repeat recognition.
Ashborn et al. (1989)	40	Transurethral Prostatectomy.	60-80 years. Mean = 68.8 years.	No change in verbal memory. ↓ Paired Associate Learning day 4 ↓ visual memory and delayed visual recall.
Chung et al. (1990)	40	Cholecystectomy.	25-83 years. < 60 (39.7 percent). > 60 (67 percent).	↓ Digit Symbol Test day 1 (all patients). ↓ Trail Making Test Day 1 (Older patients).

Table 38 Summary of Studies of Effects of General Anesthesia on Cognitive Functioning Post-Operatively *(continued)*

Jones et al. (1990)	146	Total Hip Replacement. Total Knee Replacement.	> 60 years.	No change on tests of choice reaction time and critical flicker fusion threshold between individuals receiving general or regional anaesthesia.
Smith et al. (1991)	112	Transurethral Procedure.	48-78 years.	↑ variability in choice reaction time at 24 hours.
Campbell et al. (1993)	169	Cataracts.	65-98 years.	↓ at 24 hrs in verbal recall, verbal learning, psychomotor speed, and tactile naming in both general and regional anaesthesia groups. General anaesthesia group decrease was greater but results not significant. Recovery in both groups by 2 weeks post-operatively.
Williams-Russo et al. (1995)	262	Total Knee Replacement.	> 40 years. Mean = 69 years.	Generalized decline at 1 week followed by a return to baseline or improvement by 6 months in both general and regional anaesthesia groups.

15.2 Outpatient Surgery

Changes in cognitive functioning after surgery occur in patients of all ages. The majority of studies summarized in Table 38 involve older individuals. However, cognitive deficits have been noted in younger populations as well (Smith, Roberts, Rodgers, and Bennett, 1986). Although there is an absence of literature on the effects of surgery on driving performance, the broad spectrum of cognitive deficits recorded suggests that driving performance is likely to be impaired in the immediate post-operative period. In addition, for patients undergoing outpatient surgery, the administration of narcotics in the immediate post-operative period is likely to result in greater impairments in performance.

15.3 Major Surgery

See sections 15.1 and 15.2.

Table 39 Guidelines for Anesthesia and Surgery *(Reproduced with permission)*

Guidelines for Anesthesia and Surgery (Drivers of Private Vehicles)		
Area/Domain	**Austroads (1998)**	**CMA (2000)**
General Anesthetic	Should not drive for 24 hours after a general anesthetic.	Patients undergoing outpatient surgery under general anesthesia should not drive for at least 24 hours. The pain and discomfort following even minor surgical procedures may extend this prohibition for several days.
Local Anesthetic	Should not drive if anesthetized region impairs motor or cognitive functioning.	See general anesthetic guidelines.
Major Surgery	Not addressed.	Necessary to evaluate on an individual basis.
Outpatient Surgery	Not addressed.	See general anesthetic guidelines.

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Appendix A
Preliminary Guidelines for Physicians: Assessing Medical Fitness-to-Drive
June 2000

Contents

Acknowledgements

The enclosed guidelines are the result of a collaborative effort of a group of individuals with expertise in medical conditions and driving. The process involved:

a) An integrative review of the relevant scientific literature on the specific medical conditions and driving;

b) Drafting of preliminary medical fitness-to-drive guidelines based on the integrated literature review and current medical fitness-to-drive guidelines from Australia[1], Ireland[2], and Canada[3];

c) Distribution of the draft preliminary guidelines to representatives of the AAAM and NHTSA* for initial evaluation and feedback;

d) A meeting of AAAM and NHTSA representatives in New York in March of 2000, to discuss the preliminary guidelines; *

e) A revision and final draft of the preliminary guidelines based on feedback and revisions from the New York meeting.

**AAAM and NHTSA representatives who participated in this effort include: B.M. Dobbs, PhD, D. Carr, MD, J. Eberhard, PhD, R. Marottoli, MD, M. Malinowski, D. O'Neill, MD, G. Odenheimer, MD, E. Petrucelli, & R. Raleigh, MD.*

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Additionally, the reader should note that the June 2000 preliminary guidelines that follow formed the bases for the September 2003 recommendations set forth in Chapter 9, entitled Medical Conditions and Medications That May Impair Driving, of the American Medical Association's *Physician's Guide to Assessing and Counseling Older Drivers* (Wang, Kosinski, Schwartzberg, Shanklin. Washington, DC: National Highway Traffic Safety Administration, 2003).

[1] *Austroads (1998). Assessing fitness to drive. Austroads guidelines for health professionals and their legal obligations. Sydney, Australia: Superline Printing.*

[2] *Northern Ireland Driver and Vehicle Licensing Agency (1998). For medical practitioners At a glance guide to the current medical standards of fitness to drive. Northern Ireland: Drivers Medical Unit DVLAS, Swansea.*

[3] *Canadian Medical Association (1999, draft guidelines). Guide for physicians in determining medical fitness to drive. Ottawa, Canada.*

Section 1: Introduction

The following preliminary guidelines were developed to assist physicians in determining when patients have medical conditions that can affect fitness-to-drive. The recommendations are for drivers of private motor vehicles.

Responsibilities of the Individual Driver

In North America, as elsewhere, driving is legally a privilege. In practice, however, driving is often viewed as a right. In instances where a medical condition or conditions(s) affect(s) fitness-to-drive, and the physician has informed the individual that the condition(s) may affect driving, the primary onus usually is on the individual to adhere to the physician's recommendation regarding driving. Moreover, typically, it is the responsibility of the individual to report to his/her physician and to the state/province any change in condition or in a treatment that may negatively affect his/her driving.

Responsibilities of the Physician

There is considerable variation in state/province/territory/county policy regarding the responsibilities of physicians in evaluating and counseling patients about fitness-to-drive. Physicians are encouraged to contact their state/provincial Department of Motor Vehicles to obtain legal or voluntary requirements for reporting medically unsafe drivers.

In December of 1999, the American Medical Association released a policy statement articulating physicians' responsibility with regard to physical and mental impairments that might adversely affect driving (Current Opinions of the Council on Ethical and Judicial Affairs (CEJA), Rep1, 1-99).

The policy statement is summarized below. More detailed information can be obtained from the American Medical Association web site: http://www.ama-assn.org/apps/pfonline/pfonline

(1) Physicians should assess patients' physical or mental impairments that might adversely affect driving abilities. Cases must be evaluated individually. In making evaluations, physicians should consider the following factors: (a) the physician must be able to identify and document physical or mental impairments that clearly relate to the ability to drive, and (b) the driver must pose a clear risk to public safety.

(2) Before reporting, there are a number of initial steps physicians should take, including a tactful but candid discussion with the patient and family about the risks of driving.

(3) Physicians should use their best judgment when determining when to report impairments that could limit a patient's ability to drive safely. In situations where clear evidence of substantial driving impairment implies a strong threat to patient and public safety, and where the physician's advice to discontinue driving privileges is ignored, it is desirable and ethical to notify the Department of Motor Vehicles.

(4) The physician's role is to report medical conditions that would impair safe driving as dictated by his or her state's mandatory reporting laws and standards of medical practice. The determination of the inability to drive safely should be made by the state's Department of Motor Vehicles.

(5) Physicians should disclose and explain to their patients this responsibility to report.

(6) Physicians should protect patient confidentiality by ensuring that only the minimal amount of information is reported and that reasonable security measures are used in handling that information.

(7) Physicians should work with their state medical societies to create statutes that uphold the best interests of patients and community, and that safeguard physicians from liability when reporting in good faith.

Classes of Motor Vehicles

The preliminary guidelines provided herein are for drivers of private motor vehicles. A license for a private motor vehicle permits the operation of a motor vehicle not exceeding a certain weight (the definition varies among the individual states/provinces. Therefore, individuals should consult the requirements for their individual states/provinces for more specific definitions of private motor vehicle operation). The operation of an ambulance, a taxicab, a bus, or a semi-trailer is not considered private vehicle operation.

Section 2: Vision

1. Acuity

2. Visual Field
   a. Hemianopia / Quadrantanopia
   b. Monocular Vision

3. Miscellaneous Conditions
   a. Aphakia
   b. Cataracts
   c. Glaucoma
   d. Color Blindness
   e. Poor Night Vision
   f. Conjunctivitis and other Anterior Eye Infections
   g. Diplopia
   h. Nystagmus
   i. Ptosis
   j. Telescopic Lens

4. Contrast Sensitivity-see Section 16-Areas Under Investigation

Preliminary Guidelines for Medically-At-Risk Drivers **Section 2: Vision**	
Function/Condition	
1. Acuity	Visual acuity must be measured with both eyes open while wearing any corrective lenses usually worn for driving. <u>Eye sight requirements:</u> Not less than 20/40 with both eyes open and examined together.
2. Visual Field	The minimum field of vision for safe driving is defined as a field of at least 120 degrees on the horizontal plane. Note: Visual field assessment using the automated vision testers typical at Department of Motor Vehicles may be insensitive to some visual field defects.
a. Hemianopia/Quadrantanopia	Typically should not drive, however, should be assessed by the driver licensing authority on an individual basis. An ophthalmologist's report should be submitted to the driver licensing authority, which may then consider a conditional license.
b. Monocular Vision	No restrictions for monocular drivers if standards for visual acuity and field of vision are met.
3. Miscellaneous Conditions:	
a. Aphakia	May drive if meets the acuity criteria. Specialist opinion recommended.
b. Cataracts	<u>With contact lens or intraocular lens following cataract removal:</u> May, after full recovery, qualify for a license if able to wear contact lenses or have had an intraocular lens transplant. The surgeon should advise the patient when it is safe to resume driving.
c. Glaucoma	May drive if an optometrist's or ophthalmologist's report is obtained stating that the visual acuity and visual field criteria are met. Must be subject to annual evaluation of vision and visual fields by an eye care specialist, with the report forwarded to the licensing agency.
d. Color Blindness	No restrictions for deficits in color vision if standards for visual acuity and field of vision are met.
e. Poor Night Vision	Should not drive at night or under other low light daytime conditions.
f. Conjunctivitis and other Anterior Eye Infections	Physician should advise re: driving if condition is severe enough to interfere with eye comfort or vision.

Preliminary Guidelines for Medically-At-Risk Drivers **Section 2: Vision** (*continued*)	
g. Diplopia	Should not drive in the early stages of diplopia. If the diplopia can be completely corrected with a patch or prisms to meet the standards for visual acuity and visual field, the driver may be eligible to drive on specialist recommendation.
h. Nystagmus	No restrictions if standards for visual acuity and field of vision are met.
i. Ptosis	Individuals with fixed ptosis can drive provided lids do not obscure the pupil of both eyes and the applicants are able to meet the standards for visual acuity and field without having to hold their head in an extreme position.
j. Telescopic Lens	The ability to drive safely using bioptic lenses should be demonstrated by a road test.

Section 3: Hearing

1. Hearing

Preliminary Guidelines for Medically-At-Risk Drivers **Section 3: Hearing**	
Function	
Hearing	No restrictions. The driver who wears a hearing aid should be advised that feedback from hearing aids may create a distraction and thus pose a hazard when driving.

Section 4: Cardiovascular Diseases

The recommendations are from the Canadian Cardiovascular Society Consensus Conference (1996). The general guidelines recommend that all drivers with coronary heart disease should satisfy appropriate waiting periods. Specific recommendations and waiting periods are provided based on:
a) acute MI, unstable angina,
b) stable angina,
c) suspected asymptomatic coronary artery disease,
d) coronary angioplasty,
e) coronary bypass surgery and on the presence of left main coronary artery disease.

In addition, the guidelines provide recommendations based on disturbances in cardiac rhythm and the presence of other cardiac conditions (e.g., valvular heart disease, congestive heart failure, hypertrophic cardiomyopathy, congenital heart disease, and cardiac transplantations).

1. Angina
2. Angioplasty
3. Myocardial Infarction (Acute)
4. Cardiac Bypass Surgery
5. Atrial Flutter / Fibrillation
6. Atrio-Ventricular / Intra-Ventricular Block
7. Non-Sustained Paroxysmal Ventricular Tachycardia/ Paroxysmal Supra Ventricular Tachycardia/ Paroxysmal Atrial Flutter or Fibrillation
8. Sick Sinus Syndrome, Sinus Bardycardia, Sinus Exit Block, Sinus Arrest
9. Sustained Ventricular Tachycardia, Ventricular Fibrillation
10. Implantable Cardioverter /Defibrillator Devices (ICDs)
11. Pacemaker
12. Aortic Aneurysms
13. Cardiac Arrest
14. Hypertrophic Cardiomyopathy
15. Congenital Heart Disease
16. Congestive Heart Failure
17. Deep Vein Thrombosis
18. Heart Transplant
19. Hypotension
20. Hypertension
21. Valvular Heart Disease

Preliminary Guidelines for Medically-At-Risk Drivers **Section 4: Cardiovascular Diseases**	
Condition	
1. Angina	Stable angina pectoris No additional restrictions. No waiting period. Unstable angina pectoris Waiting period one month before resuming driving.
2. Angioplasty	Waiting period 48 hours before resuming driving.
3. Myocardial Infarction (Acute)	Uncomplicated MI Waiting period of 2 weeks before resuming driving. Complicated MI (e.g., arrhythmia, CHF, dizziness, recurrent MI's) Waiting period of one month before resuming driving. Complete assessment by a cardiologist before resuming driving.
4. Cardiac Bypass Surgery	Waiting period of one-month post surgery (using current open-heart surgery techniques). For those individuals undergoing coronary artery grafts using the minimally invasive surgery technique, the waiting period may be considerably shorter.
5. Atrial Fibrillation/Flutter	Should not drive after acute episode, which causes dizziness or syncope until condition is stabilized.
6. Atrio-Ventricular/Intra-Ventricular Block	If block isolated No restriction. If LBBB, Bifascicular Block, Mobitz Type 1 AV Block, First Degree AV Block and Bifascicular Block No restrictions if no associated signs of cerebral ischemia. Mobitz Type II AV Block, Trifascicular Block, Acquired Third Degree Block Should not drive unless satisfactorily treated. Congenital Third Degree AV Block No restrictions if no associated signs of cerebral ischemia.
7. Non-sustained Paroxysmal VT/Paroxysmal Supra Ventricular Tachycardia/Paroxysmal Atrial Fibrillation or Flutter.	No restrictions: 1. With no associated signs of cerebral ischemia and no underlying heart disease. 2. With ventricular pre-excitation and no associated cerebral ischemia. 3. If satisfactory control with resolved signs of cerebral ischemia. 4. If satisfactory control with underlying heart disease.
8. Sick Sinus Syndrome, Sinus Bardycardia, Sinus Exit Block, Sinus Arrest	No restrictions if no associated signs of cerebral ischemia.
9. Sustained Ventricular Tachycardia, Ventricular Fibrillation	The following conditions apply with or without an Implantable Cardioverter / Defibrillator Device (ICD): 1. Waiting period 3 months if: VT/VF non-inducible by EPS, on EPS predicted effective drug therapy. 2. Waiting period of 6 months if: On Holter-predicted effective drug therapy, on empiric therapy with other anti-arrhythmic drugs (with ICD only, on empiric therapy with other anti-arrhythmic drugs [without ICD]). Recommendations re: 'firing free' time interval (e.g., > 6 months).

10. Implantable cardioverter/ defibrillator devices (ICDs)	See ventricular fibrillation/sustained ventricular tachycardia guidelines.
11. Pacemaker	Artificial cardiac pacemakers Conditions: Waiting period 1 week. May resume driving if: a. No cerebral ischemia. b. Normal sensing and capture on ECG. Device performing within manufacturer's specifications.
12. Aortic Aneurysms	See guidelines in Section 6: Peripheral Vascular Disease.
13. Cardiac Arrest	After recovery, the individual requires a certificate from appropriate specialist before permitting a return to driving when underlying etiology treated and other relevant criteria in this table met.
14. Hypertrophic Cardiomyopathy	No restrictions if no associated signs of cerebral ischemia and Holter classification Class 2.
15. Congenital Heart Disease	Assessment should be based on the presence or absence of myocardial ischemia, left ventricular dysfunction, valvular lesions, and/or disturbances of cardiac rhythm, and should adhere to the relevant guidelines in the preceding sections.
16. Congestive Heart Failure	No restrictions if: 1. Functional Class I (no functional limitations, able to achieve 7 METS without developing symptoms or objective evidence of cardiac dysfunction). 2. Functional Class II (mild functional limitations, able to achieve 7 METS). 3. Functional Class III (moderate limitations, working capacity < 2 METS, symptoms at rest) if no signs of cerebral ischemia (e.g., dizziness, palpitations, lightheadedness, loss of consciousness) or dyspnea. 4. Functional Class IV (severe impairment, working capacity < 2 METS, symptoms at rest) DO NOT DRIVE . Recommend reassessment every two years.
17. Deep Vein Thrombosis	See guidelines in Section 6: Peripheral Vascular Disease.
18. Heart Transplant	Waiting period of 2 months before resuming driving. Waiting period may be shortened at the discretion of specialist. Annual re-assessment recommended.
19. Hypotension	Not a contraindication to driving unless it has caused episodes of loss of consciousness (syncope) or confusion caused by cerebral ischemia or hypo-perfusion. If cerebral ischemia or hypoperfusion has occurred, the individual should discontinue driving. If it is possible to prevent further attacks by treatment, the patient may resume driving.
20. Hypertension	No driving restrictions for any type of hypertension other than uncontrolled malignant hypertension.
21. Valvular Heart Disease	Medically treated/untreated valvular heart disease No restrictions if no associated cerebral ischemia. Surgically treated valvular heart disease (e.g., mechanical prostheses, mitral bioprostheses, or valvuloplasty with non-sinus rhythm). Waiting period 6 weeks. No restrictions if no thromboembolic complications.

Section 5: Cerebrovascular Diseases

1. Stroke
2. Transient Ischemic Attacks (TIA) / Syncope
3. Aneurysms of Brain and Malformations
4. Subarachnoid Hemorrhage
5. Post Intacranial Surgery

Preliminary Guidelines for Medically-At-Risk Drivers **Section 5: Cerebrovascular Diseases**	
Condition	
1. Stroke	Should not drive for at least one month following a stroke unless specialist recommends that driving may resume earlier. Individuals with sensory loss or neglect, inattention, cognitive impairment, visual field defects, and/or motor deficits should be referred for an assessment.
2. Transient Ischemic Attacks (TIA)/Syncope	Individuals who have experienced either a single TIA or recurrent TIA's or syncope should not be allowed to drive until they have undergone a complete medical assessment and should refrain from driving for at least one month following the last event.
3. Aneurysms of Brain and Malformations	<u>Brain Aneurysm</u> Should not drive following detection of a brain aneurysm until assessed by a neurosurgeon. Driving may resume if risk of bleed is small and/or individual is free of other medical contraindications to driving such as uncontrolled seizures or significant perceptual or cognitive impairments. <u>Arterio-Venous Malformations</u> Should not drive until assessed by a neurosurgeon. May resume driving if risk of bleed is small and/or individual is free of other medical contraindications to driving such as uncontrolled seizures or significant perceptual or cognitive impairments.
4. Subarachnoid Hemorrhage	Should not drive for at least 3 months post-event. Driving may resume following medical assessment.
5. Post Intracranial Surgery	Should not drive for a minimum of 3 months post surgery. Driving may resume following assessment and recommendation by neurosurgeon or neurologist.

Section 6: Peripheral/Systemic Vascular Disease

Individuals with Peripheral Arterial Vascular Diseases (e.g.. Raynaud's Phenomena, Buerger's Disease, and Arteriosclerotic Occlusions), may be precluded from driving if the condition is of sufficient severity to cause claudication. Individuals with Peripheral Arterial Vascular Diseases always require careful evaluation and regular ongoing surveillance.

1. Peripheral Arterial Aneurysms
2. Aortic Aneurysms including Marfan Syndrome
3. Deep Vein Thrombosis

Preliminary Guidelines for Medically-At-Risk Drivers **Section 6: Peripheral/Systemic Vascular Disease**	
Condition	
1. Peripheral Arterial Aneurysms	No restrictions to driving unless other disqualifying conditions are present. Individuals whose aneurysm appears to be at the stage of imminent rupture based on size, location, and/or recent change should not drive.
2. Aortic Aneurysms including Marfan Syndrome	No restrictions to driving unless other disqualifying conditions are present. Individuals whose aneurysm appears to be at the stage of imminent rupture based on size, location, and/or recent change should not drive.
3. Deep Vein Thrombosis	Individuals with acute deep venous thrombosis may drive after one week of appropriate treatment. The physician should advise individuals with a prior history of deep venous thrombosis to take frequent 'mobilization breaks' when driving long distances.

Section 7: Diseases of the Nervous System

1. Auras and Focal Seizures
2. Single Unprovoked Seizure
3. Withdrawal or Change of Anti-Epileptic Drug Therapy
   a. If seizures recur after withdrawal or change of medication
4. Seizures while Asleep
5. Sleep Disorders
   a. Narcolepsy
   b. Sleep Apnea
6. Other Neurologic Conditions
   a. Dementia
   b. Parkinson's Disease
   c. Multiple Sclerosis
   d. Migraines
   e. Brain Tumors
   f. Peripheral Neuropathy

Preliminary Guidelines for Medically-At-Risk Drivers **Section 7: Diseases of the Nervous System**	
Condition	
1. Auras and Focal Seizures	No driving until seizure free for 6 months. Time period may be shortened upon approval of specialist (see unfavorable/favorable modifiers below).
2. Single Unprovoked Seizure	Should not drive for 6 months. Time period may be shortened with specialist approval. Predictors of recurrent seizures that would preclude shortening of time interval are: 1. If previous seizure was focal in origin. 2. If focal or neurologic deficits predated the seizure. 3. If seizure is associated with chronic diffuse brain dysfunction. 4. A positive family history of epilepsy. 5. The presence of generalized spike waves or focal spikes on EEG recordings. Favorable modifiers: 1. Seizures during medically directed medication changes (see below). 2. Simple partial seizures that do not interfere with level of consciousness and/or motor control. 3. Seizures with consistent and prolonged auras. 4. Established pattern of pure nocturnal seizures. 5. Seizures secondary to acute metabolic or toxic states not likely to recur. 6. Sleep deprived seizures. 7. Seizures related to reversible acute illness. Unfavorable modifiers: 1. Non-compliance with medication or medical visits and/or lack of credibility. 2. Alcohol and/or drug abuse in the past 3 months. 3. Increased number of seizures in past year. 4. Prior bad driving record. 5. Structural brain lesion. 6. Non-correctable brain functional or metabolic condition. 7. Frequent seizures after seizure free interval. 8. Prior crashes due to seizures in the past 5 years.
3. Withdrawal or Change of Anti-Epileptic Drug Therapy a. If seizures recur after withdrawal or change of medication	When physician suggests significant risk of recurrent seizure, driving should cease during withdrawal or change and for at least 3 months thereafter. Should not drive for 1 month after resuming previously effective medication or for 6 months if refusing to resume medication and individual is seizure free during that time period.

4. Seizures while Asleep	A person who has suffered an attack while asleep should refrain from driving for 6 months from the date of the attack. However, the time period may be shortened at the advice of a specialist (i.e., 3 months) if an established pattern of pure nocturnal seizures is evident.
5. Sleep Disorders:	
a. Narcolepsy	Should cease driving on diagnosis. Driving may be permitted when satisfactory control of symptoms achieved.
b. Sleep Apnea	Driving permitted when satisfactory control of symptoms achieved.
6. Other Neurological Conditions	
a. Dementia	Recommendations from the Canadian Consensus Conference on Dementia: 1. Physicians should consider risks associated with driving in every patient for whom they treat dementia or cognitive impairment. 2. Focused medical assessments that include history of driving difficulty from a family member or friend, and an exam focused on cognitive abilities such as memory, attention, reaction time, judgment, and visuospatial abilities is recommended. Physicians should be alerted that driving difficulties may indicate other cognitive/functional problems that need to be addressed. 3. Physicians should encourage patients with AD and related dementias, along with their caregivers, to plan early for eventual cessation of driving privileges and develop transportation support to those who lose their capacity to drive. 4. Physicians are advised to notify their driver licensing agency regarding the patient's competence to drive, even in those provinces/states that have not mandated reporting by physicians, unless the patient gives up driving voluntarily. 5. Physicians should advocate for the establishment and access to affordable, validated performance-based driving assessments and transportation programs. Serial evaluations are recommended every 6-12 months because of the progressive nature of disease.
b. Parkinson's Disease	Driving may be permitted based on outcome of assessment for level of symptom involvement, response to treatment, and likelihood of freezing or dyskinesias. Serial evaluations are recommended every 6-12 months because of progressive nature of disease.
c. Multiple Sclerosis	Assessment by specialist and driving assessor recommended.
d. Migraines	Individuals with recurrent migraines should be cautioned about driving when experiencing neurologic manifestations (e.g., visual disturbances, dizziness).
e. Brain Tumors	Driving recommendation will depend on type of tumor, prognosis, rate of growth, type of treatment, seizures, cognitive or perceptual impairments.
f. Peripheral Neuropathy	If difficulty with proprioception or sensation is identified, a driver rehabilitation specialist can assist in selecting appropriate vehicle controls so that driving can be maintained.

Section 8: Respiratory Diseases

1. Asthma
2. Chronic Obstructive Pulmonary Disease (COPD)
3. Sleep Apnea

Preliminary Guidelines for Medically-At-Risk Drivers **Section 8: Respiratory Diseases**	
Condition	
1. Asthma	No restrictions. Driver licensing agency need not be notified unless attack associated with fainting or loss of consciousness.
2. Chronic Obstructive Pulmonary Disease (COPD)	No restrictions if well controlled and no significant side effects from the condition or medication. (Physicians should be alert to the possibility of cognitive impairment in individuals with COPD with respiratory failure).
3. Sleep Apnea	See guidelines in Section 7: Diseases of the Nervous System.

Section 9: Metabolic Diseases

Individuals in the acute phase of a metabolic disease (e.g., Diabetes, Cushing's Disease, Addison's Disease, hyperfunctioning of the adrenal medulla, thyroid disorders) may experience signs and symptoms that are incompatible with safe driving. Physicians should advise those individuals to refrain from driving until the symptoms have abated.

Diabetes Mellitus
- a. Insulin Dependent (IDDM)
- b. Non-Insulin Dependent Diabetes (NIDDM)
- c. Gestational Diabetes

Preliminary Guidelines for Medically-At-Risk Drivers **Section 9: Metabolic Diseases**	
Condition	
1. Diabetes Mellitus	
a. Insulin Dependent (IDDM)	Must demonstrate satisfactory control, recognize warning symptoms of recurrent hypoglycemia*, and meet required visual standards. For individuals experiencing *recurrent* hypoglycemic or hyperglycemic attacks, those individuals should not drive until they have been free of recurrent hypoglycemic or hyperglycemic attacks for 3 months. Individuals with extremes of hypoglycemia or hyperglycemia should not to drive. For individuals with IDDM who drive, food, fruit, or candy must be within their reach while driving at all times. *Hypoglycemia is defined as reactions that require the intervention of another person or a reaction that requires the administration of intravenous glucose, intramuscular glucagon, or hospitalization.
b. Non-Insulin Dependent Diabetes (NIDDM)	<u>Managed by Diet and Tablets:</u> No restrictions unless individual develops relevant disabilities (e.g., diabetic eye problems). <u>Managed by Diet alone:</u> No restrictions unless individual develops relevant disabilites (e.g, diabetic eye problems).
c. Gestational Diabetes	If the individual develops permanent diabetes, see guidelines for IDDM or NIDDM.

Section 10: Renal Diseases

1. Chronic Renal Failure
2. Renal Transplant

Preliminary Guidelines for Medically-At-Risk Drivers **Section 10: Renal Diseases**	
Condition	
1. Chronic Renal Failure	No restrictions unless subject to symptoms (e.g., sudden disabling attacks, fainting, impaired psychomotor function, or impaired cognitive function). If symptoms are present, driving cessation may be recommended upon the advice of the physician.
2. Renal Transplant	Driving may resume 4 weeks following successful transplant on the advice of a nephrologist.

Section 11: Musculoskeletal Disabilities

Musculoskeletal Disabilities often affect range of motion and can cause pain. If there is any question of an individual's ability to perform the required movements for driving accurately and repeatedly without undue pain, that individual must be assessed by an appropriately trained assessor. Individuals with significant musculoskeletal disabilities should be considered for remediation and be referred to a specialized disability assessment centre if possible. If vehicle adaptation should be required, the individual should be assessed and trained by the appropriate specialists. If vehicle adaptation is needed, the driver will be restricted to driving vehicles so equipped.

If the physician has concerns about the individual's ability to safely operate a motor vehicle, that individual must demonstrate his/her ability to drive to an appropriately trained driving examiner.

1. Limitation of Cervical Movement
2. Limitation of Thoracic and Lumbar Spine.
3. Severe Arthritis
    a. Painful Joints
4. Loss of Extremities
    a. Prostheses
5. Paraplegia or Quadriplegia
6. Muscle and Movement Disorders

Preliminary Guidelines for Medically-At-Risk Drivers **Section 11: Musculoskeletal Disabilities**	
Condition	
1. Limitation of Cervical Movement.	Some degree of loss of movement of head and neck is acceptable but vehicles should be equipped with right and left outside mirrors.
2. Limitation of Thoracic and Lumbar Spine.	Persons with marked deformity, wearing braces or body casts or painfully restricted motion in thoracic or lumbar vertebrae should be evaluated by a driver examiner for recommendations to maximize driving safety (e.g., power brakes, steering automatic transmission, cruise control).
3. Severe Arthritis	
a. Painful Joints	Vehicle adaptation may be necessary. Modification must be noted on license. Should not drive if condition directly affects ability to drive. May drive once condition stabilized. Driving assessor opinion may be needed.

4. Loss of Extremities	
a. Prostheses	<u>Arms/Hands</u> When fitted with a prostheses, can drive a motor vehicle (must demonstrate ability before a driver examiner). If special controls necessary, restricted to driving vehicles so-equipped. <u>Legs/Feet</u> Usually able to drive a motor vehicle provided individual has adequate strength and movement in back, hips, and knee joints and a properly fitted prostheses or hand controls. Must demonstrate ability to drive to a driver examiner. <u>Fingers/Toes</u> Can drive a motor vehicle provided they demonstrate ability to a driver examiner. Persons with amputations of arms or legs who have been fitted with adequate prostheses may drive a motor vehicle provided they have demonstrated their ability to the satisfaction of a driver examiner.
5. Paraplegia or Quadriplegia	May receive a learner's license on the basis of favorable recommendation from medical consultant. With permit, may then take driving lessons in specially modified vehicle.
6. Muscle and Movement Disorders	Referral to rheumatologist/specialist in driving assessment for determination if these disorders are significantly disabling. Vehicle may require adaptation. If restrictions required, these must be noted on license. May require a shorter licensing period.

Section 12: Psychiatric Diseases

Readers are referred to the Position Statement on the Role of Psychiatrists in Assessing Driving Ability (American Journal of Psychiatry, 1995, 1S2:5, p. 819) regarding the psychiatrist's role in assessing driving ability and advising patients about fitness-to-drive.

1. Anxiety/Panic Disorder or Depression
2. Attention Deficit Disorder, Attention Deficit Hyperactivity Disorder, Tourette's Syndrome
3. Personality Disorders
4. Psychotic Illness
5. Manic-Depressive Illness
6. Substance Abuse

Preliminary Guidelines for Medically-At-Risk Drivers **Section 12: Psychiatric Diseases**	
Condition	
1. Anxiety/Panic Disorder or Depression	<u>Psychoneurosis (Anxiety or Panic Disorders)</u> May drive if condition stable. Side effects of medications need to be assessed <u>Depression</u> May drive if condition stable. Should be cautioned re: driving if being stabilized on medications. Those with severe depression and impaired concentration and agitation should not drive. Physicians should warn their patients who drive that the prescription of any new psychotropic medication has the potential to affect their driving skill. Caution should be given about driving during the titration phase of the drug is needed.
2. Attention Deficit Disorder, Attention Deficit Hyperactivity Disorder, Tourette's Syndrome	An individual assessment may be required for persons with mental and/or learning disabilities, depending on the severity of the disability. Individuals with difficulties with emotional control or attention span should be referred for psychological testing.
3. Personality Disorders	No restrictions for those individuals with personality disorders who have no history of risky driving behaviors and psychiatric review is favorable. For those who have a history of erratic, violent, aggressive, or irresponsible driving behavior, licensing approval requires the most careful consideration.
4. Psychotic Illness	<u>Acute</u> Should not drive during active phase. <u>Chronic</u> No restrictions if condition not acute and the individual is capable of safe and responsible driving. Licensing may be conditional upon compliance with medication.
5. Manic-Depressive Illness	No restrictions if condition stable. Should not drive if in acute phase of mania or if actively suicidal.
6. Substance Abuse	See guidelines in Section 13: Drugs.

Section 13: Drugs

Alcohol Abuse

Alcoholism is a primary, chronic disease with genetic, psychosocial, and environmental factors influencing its development and manifestations. The disease is often progressive and fatal. It is characterized by continuous or periodic: impaired control over drinking, preoccupation with the drug alcohol, use of alcohol despite adverse consequences, and distortions in thinking, most notably denial (JAMA, 1992).

A variety of resources are available to assist physicians in the assessment and treatment of patients with substance abuse disorders. Physicians are encouraged to contact local or state/provincial medical associations for further information.

Prescription and Over-the-Counter Medications

A number of prescription and over-the-counter medications can negatively affect cognitive performance. Laboratory and experimental driving studies have documented drug induced impairments on driving performance (see Janke, 1994 and Ray, Purushottam, and Shorr, 1993). In general, any drug that has a prominent central nervous system effect has the potential to adversely affect driving performance. Physicians should advise their patients regarding the potential effects of medications on driving performance.

Physicians also should be alert to the potential dangers of polypharmacy on driving performance and advise their patients accordingly. Alcohol can potentiate the cognitive effects of medications. Physicians should warn their patients that combining alcohol with their medication may negatively affect driving performance.

1. Alcohol
2. Anticonvulsants
3. Narcotic Analgesics
4. Antidepressants
5. Antiemetics
6. Antihistamines
7. Antihypertensives
8. Sedatives, Hypnotics, Anxyiolytics, Benzodiazepines
9. Stimulants

Preliminary Guidelines for Medically-At-Risk Drivers **Section 13: Drugs**	
1. Alcohol	Individuals suspected of alcoholism should be instructed not to drive while under the influence of alcohol and be referred to alcohol and drug abuse centers for treatment.
2. Anticonvulsants	Once stabilized and cleared to drive patients should be warned about dosage changes and using other medication or alcohol.
3. Narcotic Analgesics	Patients should be cautioned about driving if using narcotic analgesics due to sedative side effects. Patients should be warned about the potential for impairment with dosage changes.

4. Antidepressants	The older tricyclics (e.g., amitriptyline, imipramine) have more sedating side effects than the newer antidepressants. When treating depression, physicians should consider using the more recent compounds. Although side effects are minimized with the newer antidepressants, side effects do occur and the physician should warn patients who drive that the prescription of any psychotropic medication has the potential to affect their driving skill. Individuals should be advised not to drive during the initial phase of dosage adjustment(s) if they show evidence of drowsiness, hypotension, or other side effects that may affect safe driving performance. Note: MAO inhibitors can be associated with hypertensive crisis if the patient has exposure to tyramines in the diet. Physicians should warn patients of the potential for this side effect.
5. Antiemetics	Individuals should be advised that side effects of the medication (e.g., drowsiness, dizziness) may affect their ability to drive.
6. Antihistamines	The earlier antihistamines have more sedating side effects than the newer antihistamines. Physicians should consider using the more recent compounds and use the lowest possible dosage for the shortest period of time. Individuals taking antihistamine medication should be advised not to drive during the initial phase of dosage adjustment(s) if they show evidence of drowsiness or other side effects that may affect safe driving performance.
7. Antihypertensives	Individuals should be warned by their physicians about possible side effects of any drug that has a prominent central nervous system effect. In addition these agents can cause orthostatic hypotension and syncope and the sudden cessation of some antihypertensives can lead to hypertensive crisis. Physicians should warn patients of the potential for this side effect.
8. Sedatives, Hypnotics, Anxyiolytics, Benzodiazepines	Physicians should avoid prescribing any long-acting benzodiazepine if possible. Physicians should use the more recent compounds and use the lowest possible dosage for the shortest period of time. Individuals taking sedatives/hypnotics etc. should be advised not to drive during the initial phase of dosage adjustment(s) if they show evidence of drowsiness or other side effects that may affect safe driving performance.
9. Stimulants	Should not drive if there is clear evidence of abuse or dependence. Withdrawal from stimulants also may impair behavior.

Section 14: The Aging Driver

A number of cognitive (e.g., attention, judgment, decision making) and motor skills important for competent driving have been shown to decline with normal aging. However, it is unlikely that age per se or even these 'normal' changes affect driving in a substantial way. Rather, it is likely that common medical conditions that affect cognitive and/or motor abilities interfere with safe driving performance. Physicians need to be alert to medical conditions and medications that can lead to cognitive impairment (e.g., dementias, chronic renal failure, COPD, etc) when assessing the older driver.

1. Age
2. Multiple Medical Conditions
3. Polypharmacy

Preliminary Guidelines for Medically-At-Risk Drivers **Section 14: The Aging Driver**	
1. Age	There is no specific age at which a driver becomes unsafe. Thus, advanced age is not in and of itself a barrier to driving. Therefore, when assessing an older person's ability to drive, it is most important to consider functional ability rather than chronological age. However, medical conditions that may interfere with driving performance are more likely to occur as one gets older. Physicians should be aware that many medical conditions may impair driving abilities (e.g., Alzheimer's Disease, Mulit-Infarct Dementia, glaucoma, diabetic retinopathy). Physicians should be aware of and monitor their patients for medical conditions that may adversely affect driving. (See the appropriate section for recommendations for specific medical conditions).
2. Multiple Medical Conditions	An older person often has several medical conditions, each of which if taken separately may not substantially affect driving ability, but when taken together may impair driving performance. The hazards increase if these medical conditions are accompanied by slowing of perception and judgment and/or by medications used to treat the disorder. Individualized assessment for driving fitness is recommended using assessments that have been developed and validated for older drivers.
3. Polypharmacy	An older person often has several medical conditions requiring drug treatment. The interaction between medical condition and drug(s) may impair driving performance. Individualized assessment for driving fitness is recommended using assessments that have been developed and validated for older drivers.

Section 15: The Effects of Anesthesia and Surgery

Physicians must be alert for peri- and post-operative risk factors that may affect cognitive functioning post-surgery, placing the individual at risk for impairments in driving performance.

1. Type of Anesthesia
   a. General/Spinal/Epidural

Risk factors include:
a) Pre-existing cognitive impairment;
b) Duration of surgery;
c) Age (older than 60);
d) Altered mental status post-surgery;
e) The presence of multiple co-morbidities; and
f) Emergency surgery

Preliminary Guidelines for Medically-At-Risk Drivers **Section 15: The Effects of Anesthesia and Surgery**	
1. Type of Anesthesia	
a. General/Spinal/Epidural	The surgeon and the attending physician must make certain that patients are warned against driving for at least 24 hours after a general anaesthetic. Longer periods of driving cessation may be recommended if pain persists or complications occur. For regional and spinal anesthetics, the patients should not drive if anaesthetized region impairs motor or cognitive functioning.

Section 16: Conditions under Investigation

1. Contrast Sensitivity

Preliminary Guidelines for Medically-At-Risk Drivers **Section 16: Conditions under Investigation**	
Condition	
1. Contrast Sensitivity	Binocular measures of contrast sensitivity have been found to be a better predictor of crashes than visual acuity.

Section 17: Miscellaneous Conditions

1. Cancer

Preliminary Guidelines for Medically-At-Risk Drivers **Section 17: Miscellaneous**	
Condition	
1. Cancer	Those individuals with motor weakness or cognitive impairment from direct effect of cancer, metastases, cachexia, anemia, and/or chemotherapy should not drive until or when their condition improves and stabilizes.

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