INFORMATION TO USERS

This manuscript has been reproduced from the microfilm master. UMI films the text directly from the original or copy submitted. Thus, some thesis and dissertation copies are in typewriter face, while others may be from any type of computer printer.

The quality of this reproduction is dependent upon the quality of the copy submitted. Broken or indistinct print, colored or poor quality illustrations and photographs, print bleedthrough, substandard margins, and improper alignment can adversely affect reproduction.

In the unlikely event that the author did not send UMI a complete manuscript and there are missing pages, these will be noted. Also, if unauthorized copyright material had to be removed, a note will indicate the deletion.

Oversize materials (e.g., maps, drawings, charts) are reproduced by sectioning the original, beginning at the upper left-hand corner and continuing from left to right in equal sections with small overlaps. Each original is also photographed in one exposure and is included in reduced form at the back of the book.

Photographs included in the original manuscript have been reproduced xerographically in this copy. Higher quality 6" x 9" black and white photographic prints are available for any photographs or illustrations appearing in this copy for an additional charge. Contact UMI directly to order.
Neuropsychological deficits and health care use among patients at risk for cerebrovascular accidents: A retrospective analysis of cases referred for evaluation

Belán, Andrew Phillip, Ph.D.
The University of Arizona, 1992
Neuropsychological Deficits and Health Care Use Among Patients at Risk for Cerebrovascular Accidents: A Retrospective Analysis of Cases Referred for Evaluation

by

Andrew Phillip Belán

A Dissertation Submitted to the Faculty of the DEPARTMENT OF PSYCHOLOGY In Partial Fulfillment of the Requirements For the Degree of DOCTOR OF PHILOSOPHY In the Graduate College THE UNIVERSITY OF ARIZONA

1992
As members of the Final Examination Committee, we certify that we have read the dissertation prepared by Andrew Phillip Belan entitled NEUROPSYCHOLOGICAL DEFICITS AND HEALTH CARE USE AMONG PATIENTS AT RISK FOR CEREBROVASCULAR ACCIDENTS: A RETROSPECTIVE ANALYSIS OF CASES REFERRED FOR EVALUATION

and recommend that it be accepted as fulfilling the dissertation requirement for the Degree of Doctor of Philosophy

Alfred Kazniak 3/26/92
Richard Bootzin 3/26/92
Ronald Pool 3/26/92
James Comer 3/26/92
Mark Mennemier 3/26/92

Final approval and acceptance of this dissertation is contingent upon the candidate's submission of the final copy of the dissertation to the Graduate College.

I hereby certify that I have read this dissertation prepared under my direction and recommend that it be accepted as fulfilling the dissertation requirement.

Dissertation Director Alfred Kazniak 3/26/92
STATEMENT BY AUTHOR

This dissertation has been submitted in partial fulfillment of requirements for an advanced degree at the University of Arizona and is deposited in the University Library to be made available to borrowers under rules of the Library.

Brief quotations from this dissertation are allowable without special permission, provided that accurate acknowledgement of source is made. Requests for permission for extended quotation from or reproduction of this manuscript in whole or in part may be granted by the head of the major department of the Dean of the Graduate College when in his or her judgement the proposed use of the material is in the interests of scholarship. In all other instances, however, permission must be obtained from the author.

SIGNED: [Signature]
ACKNOWLEDGEMENTS

As in most important endeavors numerous individuals have aided me immeasurably in completing the doctoral program in general and the dissertation in particular. Without the understanding and support of the Psychology Department during very difficult periods it would have been impossible to continue in the program. Included among the individuals who guided and supported completion of the doctorate were James Comer, neuropsychologist at the Veterans Affairs Medical Center, George Hohmann, professor emeritus of psychology, and Alfred Kazniak, professor of psychology and dissertation director. Friends who were especially encouraging and helpful were Mary Newman, and William and Judith Passwaters. The person most responsible for my decision to pursue the doctorate in psychology is my wife, Betty Brummal. Throughout the long years of study and research Betty was unflagging in her encouragement and she accepted her role of "dissertation widow" with patience and aplomb. To these and to all of the people who assisted me in countless ways I am genuinely grateful.
# Table of Contents

List of Tables .......................................................... 6

Abstract ........................................................................... 7

Introduction ...................................................................... 8

  Definition of CVA ......................................................... 9
  Risk Factors ................................................................... 10
    Age ........................................................................... 11
    Gender ....................................................................... 13
    Prior CVA ..................................................................... 14
    Hypertension ................................................................. 16
    Heart Disease ................................................................ 18
    Arterial Disease ............................................................ 21
    Blood Disorders ............................................................. 25
    Diabetes ........................................................................ 27
    Other Medical Conditions ................................................ 28
    Substance Abuse ............................................................. 32
    Functional Severity of Disease .......................................... 36
    Present Study ................................................................. 37

Method ............................................................................. 38

  Subjects .......................................................................... 38
  Procedures ....................................................................... 40
    Psychological Tests ........................................................ 40
      HRNB ......................................................................... 41
      WAIS-R ....................................................................... 45
      WMS .......................................................................... 47
    Level of Risk .................................................................. 49
    Health Care Utilization .................................................... 50

Results ............................................................................. 50

Discussion ......................................................................... 51

References ......................................................................... 78
List of Tables

2. Characteristics of Cases Rejected for History of Head Injury with Loss of Consciousness and Non-CVA Central Nervous System (CNS) Disorders .................. 63
3. Characteristics of Cases Rejected for History of Substance Abuse and Psychiatric Disorders ............. 64
4. Characteristics of Missing and Incomplete Cases ...... 65
5. Comparison of Included and Rejected Cases ............ 66
6. Characteristics of CVA and non-CVA Patients ............ 67
7. Percent of Additional Diagnostic Procedures Performed on CVA and non-CVA Patients ..................... 68
8. Mean Raw Scores of CVA and non-CVA Patients on Halstead’s Neuropsychological Tests ..................... 69
9. Mean Raw Scores of CVA and non-CVA Patients on Additional HRNB Tests and the WMS ...................... 70
10. CVA Risk Due to Prior CVAs and Hypertension ........ 71
11. CVA Risk Due to Heart Disease .......................... 72
12. CVA Risk Due to Arterial (non-Cardiac) Disease ...... 73
13. CVA Risk Due to Blood Disorders and Diabetes ...... 74
14. CVA Risk Due to Other Medical Conditions ............ 75
15. CVA Risk Due To Substance Abuse ....................... 76
16. Regression Coefficients and ANOVA ..................... 77
Abstract

The association of risk for cerebrovascular accidents, severity of disease, and neurocognitive ability was examined in 58 veterans referred for neuropsychological assessment. Given the close association between health care utilization and cerebrovascular disease it was hypothesized that both health care use and stroke risk would account for a significant proportion of variation in scores on a battery of neuropsychological tests. Although age and education emerged as important predictors of neurocognitive ability, neither the use of medical resources nor the level of stroke risk added appreciably to the amount of variance explained by age and education alone. The failure to obtain the expected results may be interpreted as a result of the trend toward decreasing incidence of stroke, modification of stroke risk through effective medical care, the small sample size, possible insensitivity of the neuropsychological measures, fragmentary psychodiagnostic information, and incomplete or inaccurate medical records.
Introduction

Although neuropsychological measures have been shown to be sensitive to the behavioral sequelae of cerebrovascular accidents (CVAs), few research efforts have examined the relationship between neurocognitive impairment and real-world functioning in patients with mild to moderate cerebrovascular disease (Brown, Baird, & Shatz, 1986; Dull et al., 1982; Heaton & Pendleton, 1981). In most investigations of the neuropsychological consequences of CVAs, behavior outside the clinic or hospital ward has been largely ignored except among patients suffering from the most severe effects as in amnesia, aphasia, hemiplegia, and visual field deficits (Baird et al., 1987; Benson, Marsden, & Meadows, 1974). The impact of neurological disease on the day-to-day functioning of these patients has been evaluated almost exclusively by means of subjective quality-of-life measures (Baird, Adams, Ausman, & Diaz, 1985; Baird et al., 1987; Thames & McNeil, 1987; McSweeney, Grant, Heaton, Prigatano, & Adams, 1985) which correlate only modestly with neuropsychological test results (Baird et al., 1987). A noteworthy exception to the practice of relying on questionnaires to assess the everyday functioning of severely impaired CVA patients is found in the rehabilitation outcome literature (Anderson, Bourestrom, Greenberg, & Hildyard, 1970; Ben-Yishay, Gerstman, Diller, &
Haas, 1970; Bourestrom & Howard, 1968; Jongbloed, 1986; Lehmann et al., 1975; Loewen & Anderson, 1990; Lorenze & Cancro, 1962; Mysiw, Beegan, & Gatens, 1989; Novak, Haban, Graham, & Satterfield, 1987; Redig & McDowell, 1989; Sundet, Finset, & Reinvang, 1988). No study has been reported to date that compares the neuropsychological abilities of patients exhibiting a wide spectrum of CVA risk with their utilization of health care, a direct indicator of the functional severity of disease (Egan & Katon, 1987; Stein et al., 1987) that is sensitive to the long-term effects of brain damage (Carlsson, Svårdssudd, & Welin, 1987).

Definition of CVA

CVAs represent a continuum of pathology (Caplan, 1988) in which, according to the World Health Organization, there is "a focal disturbance of cerebral function ... with no apparent cause other than vascular" (Shinton, Gill, Zezulka, & Beevers, 1987, p.11). CVAs are classified by presumed etiology and/or symptom duration. Included among the causes generally cited in morbidity studies are CVAs due to subarachnoid hemorrhage, intracerebral hemorrhage (which often results from cardioemoblic ischemia), thrombotic acute brain infarction (also labeled "cerebral thrombosis"), cerebral embolism (which may be combined with the preceding category as "thromboembolic acute brain infarction"),
lacunar infarcts, and ill-defined, acute cerebrovascular accidents (Kurtzke, 1985; Ott, Zamini, Kleefield, & Funkenstein, 1986; Sobel et al., 1989). Depending on the criteria employed, stroke classifications may overlap. Categorized by symptom duration, CVAs are referred to as transient ischemic attacks (TIAs) when symptoms disappear within 24 hours, reversible ischemic neurologic disease (RIND) when symptoms persist for more than a day but clear within a week, and strokes when symptoms continue beyond 7 days (Levy, 1988). During the past 25 years, the latter classification which is based on the "tempo of illness" has been more dominant (Caplan, 1990).

Risk Factors

The focus of the majority of epidemiological investigations is on the incidence and prevalence of stroke, the symptoms of which are more likely to be reported to health care professionals and to be accurately diagnosed (Kurtzke, 1985). However, among cases of acute neurological disease requiring hospitalization, the most frequent source of treatment error results from the incorrect diagnosis of stroke (Dubois & Brooke, 1988). Approximately 20 percent of CVAs are misclassified as to location and/or etiology (Funkenstein, 1988) and the cause of infarction in as many as 40 percent of cases cannot be reliably determined (Sacco
et al., 1989; Stillman et al., 1987). Given the importance of accurate and consistent diagnosis in epidemiological studies (Angell, 1990), estimates of the incidence of CVAs are thus approximate at best. Factors contributing to CVA risk, stroke in particular, include permanent patient characteristics as in age, gender, and previous CVAs, and treatable conditions as in hypertension (HTN), diseases of the heart, vessels, and blood, other medical conditions, and drug abuse (Dyken et al., 1984).

AGE. Health problems in general tend to increase with advancing age (Alavi, 1989; Charles & Stimson, 1987). The "price" that the average individual pays for each active year beyond age 65 is approximately 3.5 years of compromised mental, physical, and social functioning (Brody, Brock, & Williams, 1987). Abnormal neurological signs are prominent among even the relatively healthy elderly (Benassi, D'Alessandro, Gallasi, Morreale, & Lugaresi, 1990; Jenkyn et al., 1985). The incidence of CVAs increases logarithmically with each decade between the ages of 50 and 89 (Eriksson et al., 1987), the 40 year span during which 75 percent of all CVAs occur (Millikan, McDowell, & Easton, 1987). In the Honolulu Heart Program, for example, only 3 new cases of CVA per 100 patients occurred among those aged 45 to 49 years whereas about 13 new cases per 100 appeared in the group
aged 65 to 69 years (Abbott, Donahue, MacMahon, Reed, & Yano, 1987). Similar findings have been reported by a number of investigators (see, for example, Malmgren, Bamford, Warlow, Sandercock, & Slattery, 1989; Norrving & Löwenheim, 1988; Shaper, Phillips, Pocock, Walker, & Macfarlane, 1991). In addition, subcortical lesions generally attributed to microvascular atherosclerosis are more frequently visualized in patients over 50 (Awad et al., 1986).

Age is also an important factor in performance on neuropsychological tests (Bak & Greene, 1980; Ganguli et al., 1991; Heaton, Grant, & Matthews, 1986; Parsons & Prigatano, 1978; Vega & Parsons, 1967; Yeo, 1989). When compared with norms obtained from younger adults, the test scores of older normal individuals fall within the impaired range on many measures of neurocognitive ability (Brayne & Calloway, 1988). Verbal skills usually remain intact whereas noticeable declines in visuo-spatial integration and psychomotor speed appear beginning in the mid-thirties (Heaton et al., 1986; but see Stafford et al., 1988). Especially vulnerable to the effects of age is the performance of novel tasks requiring concept formation and hypothetico-deductive reasoning (Leckliter & Matarazzo, 1989). In general, however, intellectual ability as assessed by standardized psychometric instruments tends to
remain intact in healthy persons up to the ninth decade (Benton, Eslinger, & Damasio, 1981). As Welford (1985) succinctly summarized, while physical and mental agility declines with age, the fund of information tends to increase so that older persons are able to accomplish with greater efficiency those tasks that depend on accumulated knowledge.

**GENDER.** Gender differences are also apparent in stroke risk, with the ratio of men to women holding at about 1.4:1 until the age of 65 when the incidence of stroke among women begins to approximate that of men (Eriksson et al., 1987). The same gender-dependent trend is evident in the occurrence of TIAs (Lai et al., 1990). Both age and gender affect post-CVA prognosis with older patients and men faring worse than younger patients and women (Chambers, Norris, Shurvell, & Hachinski, 1987; Jette, Pinsky, Branch, Wolf, & Feinleib, 1988; Meissner, Whisnant, & Garraway, 1988). Below age 50, women are more likely than men to sustain ischemic infarcts (Bogousslavski, Van Melle, & Regli, 1988).

With respect to neuropsychological abilities, gender differences, which may reflect structural and metabolic cortical asymmetries (Gur & Gur, 1990; Rodriguez, Warkentin, Risberg, & Rosandini, 1988) that are evident even among fetal males and females (de Lacoste, Hovarth, & Woodward, 1991) do appear in specific skill areas (Ganguli et al.,
Women tend to achieve higher scores on measures of verbal ability whereas men characteristically perform better on visuo-spatial and quantitative tasks. On some test batteries like the Differential Aptitude Tests, gender differences have been declining (Feingold, 1988). The importance of these dissimilarities depends on the particular tests administered (Halpern, 1986, 1989). For example, no gender-specific differences have been noted in subtests of the Wechsler Adult Intelligence Scale or its revision (Snow & Weinstock, 1990; Wechsler, 1955, 1981). On the other hand, the average performance of men and women on both the Finger Tapping and Dynamometer tests of the Halstead-Reitan Neuropsychological Test Battery are significantly different, warranting separate norms (Fromm-Auch & Yeudall, 1983; Heaton et al., 1986; Russell, Neuringer, & Goldstein, 1970). Complicating the investigation of abilities among women are the intrasubject variations in performance associated with hormonal fluctuations of the menstrual cycle (Hampson & Kimura, 1988).

**PRIOR CVAs.** Age and gender aside, a history of at least one CVA increases the risk of a subsequent episode depending principally on the time since the last CVA, CVA duration, and the number of prior CVAs (Committee on Health Care
Issues, 1987; Dyken et al., 1984; Eriksson et al., 1987; Evans & Hayes, 1987; Golberg & Berger, 1988; Meissner et al., 1988; Millikan et al., 1987; Rogers, Meyer, & Mortel, 1987; Rubin, Goldstone, McIntyre, Malone, & Bernhard, 1986; Sobel et al., 1989; Taylor et al., 1987). The incidence of stroke following an episode of RIND is about 8 percent during the first 6 months and 5 percent for the remainder of the year (Sorensen, Marquardsen, Pedersen, Heltberg, & Munck, 1989). Between 5 and 11 percent of patients sustain a more serious CVA during the twelve months following a TIA (Alter et al., 1987; Dennis, Bamford, Sandercock, & Warlow, 1990; Sorensen et al., 1989). Although treatment of post-CVA patients with anticoagulants or antiplatelet agents reduces recurrence by as much as one-half (Sze et al., 1988), 8 percent sustain another CVA or die from other causes each year thereafter (Mohr, 1988). Even with negative angiographic findings, patients suffering a CVA of short duration remain at risk for subsequent neurological complications (Evans & Hayes, 1987) especially among those patients with cardiac comorbidity (Shuaib, Hachinski, & Oczkowski, 1988). The effects of stroke on cognitive abilities have been amply documented (Brown et al., 1986; Funkenstein, 1988; Meier, 1970). In addition, cognitive deficits have been reported among patients experiencing TIAs only (Delaney, Wallace, & Egelko, 1980; Ponsford, Donnen, &
Walsh, 1980; but see Sinatra et al., 1984).

**HYPERTENSION.** Without doubt the most prominent among the treatable predisposing factors in CVAs is hypertension (HTN) (Dyken et al., 1984; Scheinberg, 1988), accounting for as many as 36 percent of all CVAs (Bonita, Scragg, Stewart, Jackson, & Beaglehole, 1988) whether hemorrhagic or atherothrombotic (Weisberg, 1988). Even when other risk factors are absent or statistically controlled, HTN represents the most powerful predictor of CVAs (Dollery, 1987; Dyken et al., 1984; Himmelmann et al., 1988) regardless of age (Alter et al., 1987; Wolf & Kannel, 1986). Hypertensive patients experience from 2 to 6 times as many CVAs as normotensives (Alter et al., 1987; Bonita et al., 1986; Dollery, 1987; Gorelick et al., 1989; Henrich & Horwitz, 1989b; Shaper et al., 1991; Syrjänen et al., 1988; Wolf et al., 1988). Not only is HTN associated with reduced cerebral blood flow, especially in the territory irrigated by the middle cerebral artery (Naritomi, Meyer, Sakai, Yamaguchi, & Shaw, 1979), but also with increased microangiopathy (Weisberg, Elliott, & Shamsnia, 1990).

Both the degree of control and the means by which control is achieved are related to CVA risk (Chambers et al., 1987; Dollery, 1987; Eriksson et al., 1987; Millikan et al., 1987; Rogers et al., 1987; Rubin et al., 1986)
irrespective of comorbidity (Evans & Hayes, 1987; Goldberg & Berger, 1988; Lechner et al., 1988). It is generally believed that treatment of HTN at any age reduces the risk of a first CVA (Goldberg & Berger, 1988; Dollery, 1987; Dyken et al., 1984; Wolf & Kannel, 1986) and of CVA recurrence (Meyer, Judd, Tawalkna, Rogers, & Mortel, 1988; Mohr, 1986; Sze et al., 1988). However, other investigators have failed to confirm these effects (De Gaetano, 1988; Meissner et al., 1988; Ramirez-Lassepass & Cipolle, 1988).

A recent survey of randomized antihypertensive drug trials involving 37,000 patients followed for an average period of 5 years has provided strong evidence that at least 33 percent of strokes can be avoided by maintaining lower blood pressure, even in individuals without hypertension but with clinical evidence of cerebrovascular disease (Collins et al., 1990). Ironically, some routinely prescribed HTN medications may actually accelerate atherosclerotic disease thereby increasing CVA risk (Stark, 1988). Spence (1986) has cautioned that while management of HTN reduces the risk of hemorrhagic and lacunar CVAs, the CVA risk associated with atherosclerosis is not affected, a finding confirmed by Grotta (1987).

In many diseases, HTN exerts a significant and independent effect on neuropsychological performance (Grant et al., 1987). However, investigations of HTN and its
treatment reveal only mild neurocognitive impairment among hypertensive patients (Brown et al., 1986; Lezak, 1983) and only slight improvement with treatment (Elias, Robbins, Stretten, & Blakeman, 1986; Gengo, Fagan, DePadova, Miller, & Kinkel, 1988; Schultz, Elias, Robbins, Stretten, & Blakeman, 1986) that may in fact be due principally to practice alone or to the placebo effect (Goldstein et al., 1990).

HEART DISEASE. As with prior CVAs, the relative contribution of heart disease to CVA risk depends on the time elapsed since the last acute episode (Chambers et al., 1987; Dexter, et al., 1987; Millikan, 1985) and on the specific form of cardiac impairment (Alter et al., 1987; Dyken et al., 1984; Eriksson et al., 1987; Goldberg & Berger, 1988; Rubin et al., 1986) although any type of heart disease results in increased CVA risk (Lechner et al., 1988; Sherman, Hart, & Shi, 1987; Sobel et al., 1989). Regardless of the type of cardiac impairment, heart disease is associated with at least 58 percent of all CVAs (Barclay, Weiss, Mattis, Bond, & Blass, 1988) and with a less favorable post-CVA outcome (Chambers, Norris, Shurvell, & Hachinski, 1987; but see Fullerton, Mackenzie, & Stout, 1988). Even when there is no discernable neuropathology, the presence of heart disease is associated with a higher
incidence of "silent" cerebral infarcts (Petersen et al., 1987). Patients with known coronary artery disease are 5 times more likely to experience a CVA (Goldbeg & Berger, 1988). A history of myocardial ischemia increases the CVA risk by a factor of four (Shaper et al., 1991). Cardiogenic embolisms account for about 44 percent of CVAs in patients aged 80 years and older whereas among patients below the age of 26, only 17 percent of CVAs are referable to cardiac dysfunction (Norrving & Lowenhielm, 1988). Major ECG abnormalities indicative of left ventricular hypertrophy (LVH) are associated with a two- to fivefold increase in CVA risk (Knutsen et al., 1988; but see Sox, Garber, & Littenberg, 1989). Evidence of LVH results in as much as a tenfold increase in CVA risk (Millikan et al., 1987).

Other forms of cardiac anomalies which increase the probability of CVA include angina pectoris, chronic atrial fibrillation (AF), congestive heart failure, mitral valve prolapse, coronary artery bypass graft surgery (CABG), patent foramen ovale, prosthetic heart valves, rheumatic heart disease with or without AF, infective endocarditis, and valve disease which may be complicated by infective endocarditis (Acinapura et al., 1988; Burckhardt et al., 1988; Dyken et al., 1984; Goldberg & Berger, 1988; Hart & Easton, 1982; Kanter, 1990; Kelly, Pina, & Lee, 1988; Kopecky et al., 1987; Lechat et al., 1988; Loop et al.,...
1988; Lund, 1988; Millikan et al., 1987; Webster et al., 1988; Wolf, Dowber, Thomas, & Kannel, 1978; but see Dexter, Whisnant, Connolly, & O'Fallon, 1987). Replacement of a heart valve with a prosthesis increases CVA risk by about 2 percent (Burckhardt et al., 1988). As many as 36 percent of patients with chronic AF suffer silent infarcts which are usually found in subcortical white matter (Feinberg et al., 1990). Insertion of a cardiac pacemaker may not, however, reduce the risk of sustaining a CVA (Fisher, Case, Steele, & Miller, 1988). Anti-coagulant drug therapy may reduce by half the incidence of stroke and TIAs in patients with AF (Stroke Prevention in AF Study Group, 1990).

In general, cardiac disease is associated with mild but often persistent impairment of neurocognitive functions (Brown et al., 1986). Neuropsychological status of patients prior to cardiac surgery is predictive of outcome, including the likelihood of a post-operative CVA (Kilpatrick, Miller, Allan, & Less, 1975). 38 percent of patients sustain moderate to severe impairment of neurocognitive performance following CABG surgery (Shaw et al., 1987). According to Barclay and colleagues (1988), even patients without manifest neurologic deficits or dementia tend to suffer persistent decrements in cognitive ability, especially in the domain of memory function, following myocardial infarction (MI) or congestive heart failure (CHF).
ARTERIAL DISEASE. Another major area of risk in CVA is arterial disease, most notably atherosclerosis. In addition to HTN and angina pectoris, medical conditions indicative of atherosclerosis include the presence of abdominal aortic aneurysms, carotid artery bruits, and intermittent claudication of the legs, each of which are associated with a yearly CVA incidence of 2 percent (Berkoff & Levine, 1988; Folger, 1987; Millikan et al., 1987; Nashitz, Ambrosio, & Chang, 1988). Unilateral carotid artery stenosis, which is associated with cerebral atherosclerosis, also results in an annual CVA rate of 2 percent, but only when the functional diameter of the artery is reduced by more than half (Autret et al., 1987; Gomez, 1989, 1990; Parnetti et al., 1988; Schoenfeld, Aronow, & Paul, 1988). Clinically demonstrable impairment of neurologic function may not appear, however, unless the stenosis reaches 80 to 90 percent (Fields & Lemak, 1989). Among patients with disturbed ocular blood flow, especially with evidence of retinal emboli or a history of amaurosis fugax, the annual rate of CVA is 5 percent (Kreshon, 1986; but see Merchat, Gupta, & Naheedy, 1988). Age does, however, appear to be an important cofactor as patients with ocular blood flow anomalies who are aged 45 years and younger do not appear to incur any greater CVA risk than their peers (Tippin, Corbett, Kerber, Schroeder, & Thompson, 1989). Patients with asymptomatic
arteriovenous malformations (AVMs) are also at risk with 2.2 percent sustaining CVAs annually (Brown et al., 1988). The risk of stroke from an unruptured AVM is about twice that of the general population (Brown et al., 1988). Though rare, primary cerebral angiitis results in some form of CVA in almost half of the cases (Calabrese & Mallek, 1987).

The link between cerebral atherosclerosis and elevated serum lipids or lipoproteins is becoming more firmly established as the use of non-invasive diagnostic procedures permits the inclusion of better control groups. Such diagnostic procedures also allow earlier identification of the effects of atherosclerosis on cerebral vasculature (Tell, Crouse, & Furberg, 1988). However, the significance of high plasma lipid or lipoprotein concentrations as CVA risk factors independent of atheromatous plaque formation remains equivocal (Folger, 1987; Medical Research Council, 1988; Schmitz-Huebner et al., 1988; Senin, Mannarino, & Ventura, 1983; Wolf & Kannel, 1986).

The effectiveness of pharmacological and surgical approaches to the treatment of cerebral atherosclerosis remains controversial. Vasoactive agents that are useful in treating myocardial ischemia do not appear to affect the course of cerebral ischemic disease (Cohn, 1988; Toole, 1985) or to improve cognitive functioning (Brown et al., 1986). Controlled clinical trials, for example, have not
produced any evidence to support the widespread use of aspirin and other antiplatelet pharmaceuticals to prevent recurrent TIAs (Marshall, 1990) but may be useful in preventing stroke recurrence (Barnett, 1990). According to Marder and Sherry (1988) thrombolytic therapy, which has not proven useful in treating occlusive retinopathy (Verstraete, 1989), is associated with a 20 to 40 percent failure rate. Such failure, though, may be due in large measure to diabetic and hypertensive comorbidity (del Zoppo, 1988).

Although surgical restoration of impaired cerebral blood flow should theoretically reduce CVA risk and improve cognitive functioning (Baker & Meier, 1970) the long-term neurobehavioral benefits of carotid endarterectomy (CE) have yet to be adequately demonstrated (Berkoff & Levine, 1987; Committee on Health Care Issues, 1987; Hertzer, 1988; Ratcheson, 1988; Winslow et al., 1988; but see Nunn, 1988). As many as one-third of patients undergoing CE fail to exhibit any demonstrable increase in cerebral perfusion (Maurer, Siegal, Comerota, Morgan, & Joshua, 1990). Among patients aged 65 and older the risk of CE appears to outweigh the potential advantages (Fisher et al., 1989). However, it has been demonstrated that among patients with a primary diagnosis of AF, a CE may substantially reduce the risk of stroke (Bernstein & Dilley, 1987). In patients with stenoses of 70 percent or greater, CE is more effective in
preventing strokes than medication but is of questionable benefit in the treatment of less severe occlusions (Call, 1991). Post-CE restenosis occurs in about 30 percent of patients after 7 years (Healy et al., 1989) but in only 5 percent is the treated vessel significantly occluded (Sundt, Whisnant, Houser, & Fode, 1990). In a carefully controlled, randomized series of clinical trials, extracranial-intracranial (EC/IC) arterial bypass surgery has been shown to be ineffective in preventing CVAs (EC/IC Bypass Study Group, 1985) and has consequently been performed much less frequently (Barnett, 1990).

At best only minor gains in neuropsychological ability have been reported following CE or EC/IC arterial bypass surgery (Ariel & Strider, 1983; Baird et al., 1985; Baird et al., 1987; Brown et al., 1986; Nielsen, Hojer-Pederson, Gulliksen, Haase, & Enevoldsen, 1985; Parker et al., 1983; Wilson & Polido, 1986; Younkin et al., 1985). Post-endarterectomy improvement on measures of neurocognitive ability may be due to the effect of practice (Casey, Ferguson, Kimura, & Hachinski, 1989). One possible exception is that of post-CE improvement in vigilance and motor function among patients with atheromatous obstruction of the right carotid artery (Greiffenstein, Brinkman, Jacobs, & Braun, 1988). Similarly, surgical treatment of peripheral vascular disease does not reduce CVA risk or
improve neurobehavioral status (Shaw et al., 1987). In only one study has the reduction of serum cholesterol resulted in improved cognitive ability, albeit minimally (Reitan & Shipley, 1963).

**BLOOD DISORDERS.** Blood dyscrasias as in hemophilia, leukemia, polycythemia vera, sickle cell disease, and thrombocytopenic purpura increase the risk of both embolic and hemorrhagic CVAs (Hart & Kanter, 1990; Millikan et al., 1987; Toole, 1984). During each of the first 5 years of illness, 7 percent of patients with polycythemia vera sustain CVAs (Berk et al., 1986; Najean, Mugnier, Dresch, & Rain, 1987). Up to 92 percent of patients with thrombotic thrombocytopenic purpura exhibit recurrent neurologic symptoms (Petitt, 1980) due principally to widespread hemorrhaging and occlusion of the cerebral microvasculature (Kwaan, 1987). Patients without patent brain lesions tend to attain full recovery (Kay, Solberg, Nichols, & Pettit, 1991).

The effect of other blood disorders on CVA risk is still controversial although evidence implicating hyperlipidemia in the etiology of heart disease, stroke, and dementia is accumulating (Zimetbaum, Frishman, & Aronson, 1991). Dyken and colleagues (1984) report that an elevated hematocrit level may not affect the likelihood of sustaining
a CVA but may increase CVA severity. Treatment of elevated hematocrit or hemoglobin does not appear to reduce the risk of a recurrent CVA (Goldberg & Berger, 1988). Reduced cerebral blood flow has been linked with elevated hematocrit and some investigators have identified serum fibrinogen as a possible independent factor in CVA (Wilhelmsen et al., 1984). Landi and colleagues (1987) report, for example, that survival following CVA is best predicted by the extent of structural brain damage as visualized in computed tomographic (CT) brain scan and the level of serum fibrinopeptide A. No other clinical indicators were found to be significant. About 17 percent of patients suffering from sickle cell anemia experience CVAs, many by the age of 9 years (Pavlakis et al., 1988). Treatment of blood disorders may not reduce either initial or subsequent CVA risk (Goldberg & Berger, 1988). An insufficient number of patients have been examined neuropsychologically to permit even preliminary conclusions regarding the effect of primary blood diseases on cognitive ability (Ariel & Strider, 1983).

**DIABETES.** It is now generally accepted that diabetes mellitus (DM) exerts an autonomous influence on CVA risk (Himmelmann et al., 1988; Stout, 1989; Tzagournis, 1989; Wolf & Kannel, 1986), especially in diabetic women (Dyken et al., 1988; Lapidus, 1985) among whom CVAs are half-again as
likely as among men (Goldberg & Berger, 1988). Even in non-insulin dependent diabetics, the occurrence of atherosclerotic vascular disease is markedly increased (Laakso et al., 1988). In several studies DM has emerged as one of the two or three most potent predictors of CVA incidence (Alter et al., 1987; Toole, 1984; Senin, Mannarino, & Ventura, 1983; but see Crouse et al., 1987). About 30 percent of patients with DM periodically exhibit focal neurological deficits (Ariel & Strider, 1983) and between 12 and 50 percent will develop some form of neuropathy (Masson & Boulton, 1980). Of patients sustaining CVAs, approximately 17 percent have positive histories for DM (Ericksson et al., 1987). When other factors are statistically controlled, glucosuria increases CVA risk two to three times above what ordinarily would be expected (Abbott et al., 1987; Goldberg & Berger, 1988; but see Rolfe, 1988). Many diabetics suffer from the effects of "silent" or lacunar infarcts, especially in the distribution of the posterior cerebral arteries, which may remain neurologically asymptomatic unless the thalamus is involved (Baxter, 1987; Soria, Fine, & Paroski, 1987). The clinical outcome in CVA patients with elevated serum glucose is less favorable (Berger & Hakim, 1986; Kushner et al., 1990; Stout, 1989). Treatment does not appear to modulate CVA risk (Goldner, Knatterud, & Prout, 1971).
Based on his comprehensive review of the literature, Ryan (1988) reports that virtually every study of adult diabetics has found an association between cognitive functioning and metabolic control. The most striking neurocognitive deficits apparently occur following episodes of hypoglycemia. Even in healthy volunteers, mild hypoglycemia significantly impairs performance on tasks sensitive to neurocognitive functioning (Stevens et al., 1989). Clinically, patients with poorly controlled blood sugar exhibit confusion, poor verbal comprehension, and impaired short-term memory (Bernstein, 1990). Establishing euglycemic serum levels in adult diabetics may improve cognitive abilities (Jangusch, Von Cramon, Renner, & Hepp, 1987) but the gains may not persist (Brown et al., 1986). In children with onset of DM prior to age 4, abilities generally associated with the right cerebral hemisphere are selectively impaired (Lacks, 1984).

**OTHER MEDICAL CONDITIONS.** CVA risk is also increased in patients with acquired immunodeficiency syndrome (AIDS), febrile infections, renal disease, late onset idiopathic seizures, migraine headaches, obesity, and oral contraceptive use. Evidence of a strong link between AIDS and CVAs is accumulating although the level of CVA risk has yet to be determined (Engstrom, Lowenstein, & Bredesen,
During the first month of febrile infection, the risk of stroke in young and middle aged patients is increased by a factor of 9 (Syrjänen, Valtonen, Iivanainen, Kaste, & Huttunen, 1988). About 10 percent of hemodialysis patients sustain hemorrhagic CVAs and renal patients in general are at greater risk for cerebral atrophy (Hutchison, Thomas, & McGibbon, 1982; Trompeter, Polinsky, Andreoli & Fennell, 1986). Uremic patients display generalized neuropsychological deficits which may partially be alleviated by hemodialysis. Successful renal transplantation is usually associated with improved cognitive functioning (Kasiske, 1988) although residual neurobehavioral impairment may persist (Ariel & Strider, 1983).

Among middle aged and older patients, idiopathic seizures with onset after age 30 precedes about 5 percent of initial and up to 9 percent of recurrent CVAs (Shinton et al., 1987; Kilpatrick et al., 1990). Cerebrovascular disease (CVD) accounts for almost 30 percent of epilepsies that occur in patients aged 60 years and older (Hauser & Kurland, 1975). Although infrequent, subcortical lesions of vascular origin result in seizures among individuals aged 40 years and under (Awad et al., 1986). Seizures develop in 17 percent of patients with carotid artery stenosis and in one-fifth of patients with middle cerebral artery occlusions.
(Cocito, Favale, & Reni, 1982; De Carolis et al., 1984). "Heralding seizures," those that precede CVAs, comprise just under 10 percent of all epilepsies associated with CVD (Daniele, Mattaliano, Tassinari, & Natalè, 1989). Neuropsychological effects of seizure disorders depend on a number of factors that include seizure type, location of the epileptic focus, total number of seizures experienced during a patient's lifetime, medications prescribed, and psychological tests administered (Andrews, Tomlinson, Elwes, & Reynolds, 1984; Dodrill, 1988; Loiseau et al., 1983; Hermann et al., 1988; Thompson & Trimble, 1982; Trimble & Thompson, 1982). Thus, as Haynes and Bennett (1990) have noted, there is no general pattern of neurocognitive dysfunction that applies to individuals suffering from epilepsy.

Migraine stroke, which is 3 times more common in women than men, accounts for 10.4 percent of CVAs in patients below 45 years of age (Bogousslavski, Regli, Van Melle, Payot, & Uske, 1988) especially when other CVA risk factors are present (Bevan, Sharma, & Bradley, 1990; Welch & Levine, 1990; but see Henrich & Horwitz, 1989a). Occurring during and immediately following classic migraines, CVAs are most likely associated with instability of regional cerebral blood flow (Lagrèze, Dettmers, & Hartmann, 1988) but may also reflect pre-existing undiagnosed vascular anomalies.
(Vasquez-Cruz & Alvarez-Sabin, 1991). Non-migraine headache with vomiting precedes as many as 86 percent of hemorrhagic CVAs (Gorelick, Hier, Caplan, & Langenberg, 1988). Up to half of patients suffering cerebral hemorrhages may experience severe headaches of sudden onset (Verweij, Wijdicks, & van Gijn, 1988).

Several studies have implicated obesity as an independent factor in CVA risk, among both women (Aronow, Gutstein, Lee, & Edwards, 1988; Folger, 1987; Lapidus, 1985; Medical Research Council, 1988) and men (Hoffmans, Kromhout, & Coulander, 1988). However, it is most likely the concomitant propensity to hypertension, glucosuria, and elevated serum lipids among the obese that accounts for the inclusion of body weight among CVA risk factors (Björntorp, 1983; Wolf & Kannel, 1986). Oral contraceptive (OC) use increases CVA risk by as much a 13 times especially in women above age 34 (Dyken et al., 1984; Xuereb & Pullicino, 1988) which accounts in large measure for the higher incidence of ischemic CVAs among women (Bogousslavski et al., 1988; Gerstman et al., 1991). One-fourth of cases of carotid artery stenosis among women may be attributed to OC use. On the other hand, among postmenopausal women estrogen replacement therapy appears to reduce CVA risk by half (Paganini-Hill, Ross, & Henderson, 1988). As with obesity, however, the effect of OC use may be due to the presence of
comorbid conditions (Bevan et al., 1990), most notably
cigarette smoking (Petitti, Wingerd, Pellegrin, & Ramcharan,
1979) which alone increases the risk of CVAs in older women
taking OCs fourfold (Wolf & Kannel, 1986).

SUBSTANCE ABUSE. Among the most prominent of the
"environmental" factors that contribute to the probability
of sustaining a CVA are abuse of tobacco, drugs and alcohol.
Results from the International Prospective Primary
Prevention Study in Hypertension indicate that among
hypertensive patients, smoking doubles the risk of CVAs
(Bühler, Vesanen, Watters, & Bolli, 1988; Shaper et al.,
1991). CVA risk among smokers is heightened even when HTN
is well-controlled (Henrich & Horwitz, 1989b; Rogers et al.,
1987). Normotensive smokers are also at greater risk of
sustaining a CVA (Wolf, D'Agostino, Kannel, Bonita, &
smoking is fourth after age, coronary artery stenosis, and
HTN as a risk factor for carotid artery atherosclerosis, a
condition commonly associated with TIAs and subsequent CVAs.
Taking HTN into account, tobacco use may contribute
independently to the etiology of up to 37 percent of CVAs
(Bonita et al., 1988; but see Folger, 1987; Senin,
Mannarino, & Ventura, 1983). Among men aged 62 to 97 years
followed by Aronow, Gutstein, Lee, and Edwards (1988),
cigarette smoking accounted for the largest percentage of new atherothrombotic CVAs. Discontinuing tobacco use following a CVA can reduce recurrence by more than 50 percent (Abbott, Yin, Reed, & Yano, 1986). Quitting smoking at any time appears to retard the development of carotid artery stenosis (Tell, Howard, McKinney, & Toole, 1989). Except in cases of chronic obstructive pulmonary disease (COPD) there is little or no evidence that heavy tobacco use independently impairs neurocognitive functioning (Brown et al., 1986) despite its adverse effect on cerebral blood flow (Meyer et al., 1986).

Cigarette smoking is so closely linked with alcohol consumption that some investigators discount the effect of alcohol use on CVA frequency (Gorelick et al., 1987; Gorelick, Rodin, Langenberg, Hier, & Costigan, 1989). Based on his review of the clinical literature, Hillbom (1987) concluded that CVAs do in fact occur more often during episodes of acute alcohol ingestion (but see Camargo, 1989). Controlling for hypertension, smoking, and medication, Gill and associates (1986) as well as Shaper and colleagues (1991) found that among males, heavy alcohol consumption (in excess of 330 grams per week) leads to a fourfold increase in CVA risk. Binge drinking (more than 80 grams consumed in a 24 hour period) results in a sevenfold increase in CVA risk (Syrjänen et al., 1988). Nevertheless, without other
risk factors present, excessive consumption of alcohol may not increase CVA risk appreciably (Bevan et al., 1990; Henrich & Horwitz, 1989c; but see Klatsky, Armstrong, & Friedman, 1989). Low levels of daily alcohol consumption may actually reduce the risk of atherothrombotic CVAs (Gill et al., 1991; Sleight, 1991; Stampfer, Colditz, Willett, Speizer, & Hemmekens, 1988).

There is a substantial age effect in the neuropsychological abilities of alcohol abusers which far exceeds the impact of alcohol consumption itself (Page & Cleveland, 1987). Although most studies reviewed by Parsons and Farr (1981) found that alcoholics were more impaired than non-drinkers on neurobehavioral measures, in only half of the investigations were the scores considered indicative of brain damage. Emmerson and associates argued based on their review of the literature and their own investigations that many if not all of the reported neurocognitive residua of alcohol abuse are due to differences in premorbid ability and the effects of withdrawal (Emmerson, Dustman, Heil, & Shearer, 1988). The principal effect of chronic, immoderate alcohol consumption emerges when the speed of performance is emphasized rather than accuracy (Bertera & Parsons, 1978; Glenn & Parsons, 1991; Goldman, 1983; Klisz & Parsons, 1977; York & Biederman, 1988). Both physiological and psychological improvement occur within the first 6 weeks of
abstinence (Lishman, Jacobson, & Acker, 1987). Some researchers contend, however, that a full restitution of abilities may not occur even after several years of sobriety (Tarter & Edwards, 1985). The principal factor in recovery appears to be the extent to which the patient exhibited signs of Wernicke's encephalopathy, viz. patients with signs of Wernicke's tended to experience an incomplete recovery (Carlen & Wilkinson, 1987).

Abuse of a number of sympathomimetic drugs such as cocaine, ephedrine, phenylpropanolamine, and amphetamines may also contribute to the onset of CVAs (Green, Kelly, Gabrielsen, Levine, & Vanderzant, 1990; Kase, 1986; Klonoff, Andrews, & Obana, 1989; Levine & Welch, 1988; Mody, Miller, McIntyre, Cobb, & Goldberg, 1988; Rothrock, Rubenstein, & Lyden, 1988). Vasospasm is the most likely cause of infarcts among chronic cocaine users (Volkow, Mullani, Gould, Adler, & Krajewski, 1988). Abuse of any drug increases CVA risk by a factor of six (Kaku & Lowenstein, 1990). Lezak (1983) and Parsons and Farr (1981) indicate that almost nothing is known about the long-term psychological effects of these substances or the extent to which neurobehavioral impairment may be ameliorated by abstinence.
Functional Severity of Disease

All diseases share a "final common pathway" that can be assessed with more or less precision: the ability to function in everyday life (Stein et al., 1987). The extent to which medical status affects everyday functioning can be measured by the level of health care utilization (HCU) (Egan & Katon, 1987). At the very least, HCU reflects the disruptions of daily activities that are occasioned by visits to health care providers. In addition, HCU measures the simultaneous effects of all symptoms (Stein et al., 1987) and the individual's response to them (Egan & Katon, 1987). Known factors which contribute to the level of HCU are age, education, gender, chronic disease, psychological distress, and history of neurologic impairment (Carlsson et al., 1987; Charles & Stimson, 1987; Egan & Katon, 1987; Hibbard & Pope, 1986; Pincus, Callahan, & Burkhauser, 1987). For example, older individuals suffer from more diseases and tend to utilize health care resources to a greater extent than younger persons (Clarke & Stimson, 1987; Callahan & Burkhauser, 1987).

Present Study

That the factors which contribute to CVA risk are numerous and interdependent (Alter et al., 1987) probably accounts for the paucity of neuropsychological studies that
attempt to control patient characteristics other than age, sex, education, and CVA history. For example, among the series of studies of cerebral revascularization candidates conducted by Baird and her colleagues, only two included a medical index of risk factors (Baird, Adams, Ausman & Diaz, 1985; Baird et al., 1985). The index of medical risk proved to be only marginally predictive of neurocognitive abilities, emerging as the third variable in a step-wise multivariate regression equation after type of CVA sustained and a global quality-of-life rating (Baird, Adams, Ausman, & Diaz, 1985). To explain this unanticipated result, the authors speculated that "possible confounding effects of premorbid and general medical factors" may have obfuscated the associations that should theoretically have emerged between neuropsychological status, quality-of-life, and severity of cerebrovascular disease (Baird, Adams, Ausman & Diaz, 1985, p. 153).

The present investigation focuses on cognitive ability and functional severity of illness in patients at risk for a CVA. In addition to replicating the well-known effects of age and education, the results of this study were expected to reveal significant associations between CVA risk, HCU, and neuropsychological test performance. It was expected that both CVA risk and HCU would account for a significant proportion of the variance in neuropsychological test
scores, the dependent variable, when entered following age and education into a regression equation. Although CVA risk and HCU were the "focal" independent variables, both age and education comprised potent "status" variables which predict not only performance on neuropsychological tests but also may be correlated with CVA risk and HCU. To ameliorate the effects of multicollinearity, hierarchical multiple regression analysis was utilized (see Cohen & Cohen, 1983). Placement of HCU before CVA risk in the model was somewhat arbitrary but appeared justified in that HCU reflects not only the presence of disease but also the subject's reaction to illness in general (Egan & Katon, 1987).

Method

Subjects

All cases referred for neuropsychological evaluation from 1983 through 1986 at the Tucson Veterans Affairs Medical Center (VAMC) were examined retrospectively to identify patients at risk for initial and recurrent CVAs at the time of assessment. 264 cases were initially identified. The pool of potential cases was somewhat older and included more non-whites but did not differ significantly in education or percentage of females from the groups of VAMC patients referred for neuropsychological evaluations reported by Russell (1987, 1988; see Table 1).
206 cases were excluded for one or more of the following reasons: history of head injury involving loss of consciousness, cerebral impairment due to neurological trauma or disease other than that associated with CVAs, drug and/or alcohol abuse within 12 months prior to evaluation, a history of psychiatric illness (excepting affective disorders secondary to CVAs), and incomplete or missing medical records (see Tables 2, 3, and 4). Patients whose medical records were incomplete or missing were significantly older than patients excluded for other reasons \((p < .001)\). The 58 patients who were included in the present study were significantly older and better educated than those who were excluded (see Table 5). There were fewer non-whites among the selected cases but the percentage of females was comparable (see Table 5). 23 of the patients had sustained at least one CVA prior to assessment. There was no evidence of amnesia, aphasia, hemiplegia, or visual field deficits among the CVA cases. Patients with a history of CVA were older (62.6 years) than non-CVA patients (53.4 years) but were similar in years of education and measured intellectual ability (see Table 6). Among the CVA patients there were two females and one Black male. One female, one Black male, and one Hispanic male were in the non-CVA group. More of the non-CVA patients were employed at the time of evaluation but otherwise, both groups were equivalent in the
number of disabled and retired veterans. Three-fourths of the patients in both groups were married and living with their spouses. Eligibility for VAMC medical care was also comparable. Three of the CVA patients were left-handed. One non-CVA veteran was in the second trimester of an uncomplicated pregnancy. All of the patients were evaluated by VAMC neurologists who, based on clinical observations and anamnestic material, ordered additional diagnostic procedures when medically indicated. Substantially fewer of the non-CVA patients were given CT scans, carotid angiograms, or electroencephalographs (EEGs), but the proportion of non-CVA patients given electrocardiographs was not significantly different from that of patients who had experienced a CVA (see Table 7).

Procedures

Psychological Tests. Each record contained the results of a standard battery of psychological tests consisting of the Halstead-Reitan Neuropsychological Test Battery (HRNB), the Wechsler Adult Intelligence Scale - Revised Edition (WAIS-R), portions of the Wechsler Memory Scale (WMS), and responses to a structured interview. Several records also included the results of the Minnesota Multiphasic Personality Inventory (MMPI) completed at the time of the evaluation. All procedures were administered and scored by
trained technicians.

HRNB. The compilation of tests and procedures that form the core of the Halstead-Reitan Neuropsychological Test Battery (HRNB) comprises the most widely used set of measures in neurocognitive assessment (Boll, 1981). Apart from the validation studies conducted by Reitan and his associates (see Reitan & Wolfson, 1985) the clinical efficacy of the HRNB has been confirmed in several independent investigations (Filskov & Goldstein, 1974; Goldstein, Duysach, & Kleinkecht, 1973; Klonoff, Fibiger, & Hutton, 1970; Matthews, Goldfader, & Snow, 1976; Stuss & Trite, 1977; Vega & Parsons, 1967).

Seven of Halstead's original measures of brain function (Halstead, 1947) are contained in the HRNB: Category Test, Tactual Performance Test (Time, Memory, and Location), Seashore Rhythm Test, Speech-sounds Perception Test, and Finger Oscillation Test. A measure of abstracting ability (Lezak, 1983), the Halstead Category Test challenges the patient to formulate and maintain a fixed concept against a background of changing stimulus configurations. The principles range in complexity from the correspondence of Arabic and Roman numerals to stimulus proportion. Following the patient's response to each of the 208 items that are divided into 7 subtests immediate feedback is given in the
form of a bell for correct answers or a buzzer for incorrect choices. The Category Test is not only a measure of abstract concept formation but also of non-verbal learning (Boll, 1981). Performance is measured by the total number of incorrect responses.

The Tactual Performance Test is a modification of the Seguin-Goddard form board which is presented to the blindfolded patient at 70 degrees to the horizontal. Using only the dominant hand on the first timed trial the patient is instructed to fit all the blocks into the spaces on the board. The procedure is repeated with the non-dominant hand and then with both hands. Following removal of the board and blocks the blindfold is removed and the patient is given a blank sheet of paper on which the form board is to be drawn from memory (Halstead, 1947). Three primary scores are derived from this procedure: total time required for all three trials, the number of blocks recalled, and the number of blocks correctly "juxtaposed" (Boll, 1981) on the drawing. Successful completion of the Tactual Performance Test requires manual dexterity, intact stereognosis, constructional praxis, and tactile-spatial recall.

Auditory discrimination ability is examined in two of Halstead's tests, the Seashore Rhythm Test and the Speech-sounds Perception Test. In the first procedure, which was borrowed from the Seashore Measures of Musical Talent
(Seashore, 1919; Seashore, Lewis, & Saetvith, 1960), the patient listens to a tape-recording of pairs of non-meaningful rhythmic "beeps" and indicates on an answer sheet which pairs are the same and which are different. The number of correct responses is recorded. The Seashore Rhythm Test demands nonverbal auditory perception (Lezak, 1983), attention and sustained concentration (Boll, 1981), and adequate short-term auditory memory. Patients taking the Speech-sounds Perception Test are presented with an answer form containing 60 multiple choice items consisting of monosyllabic variations of the "ee" digraph (Halstead, 1947). One of the nonsense syllables is spoken by a pre-recorded voice and the patients select the corresponding lexical representation. The number of erroneous choices reflects the ability of patients to match meaningless phonemes to lexemes and to maintain attention in what most patients consider a very boring task (Lezak, 1983).

On Halstead's Finger Oscillation Test the patient is required to press the lever of a mechanical counter with the index finger of the dominant hand as rapidly as possible for 10 seconds on 5 successive trials (Halstead, 1947). Also known as the Finger Tapping Test (Meier, 1974), this procedure has been expanded to include 5 consecutive trials with the index finger of the non-dominant hand (Boll, 1981). According to Lezak (1983) the Finger Oscillation Test is
probably the technique most widely used to gauge manual dexterity.

Additional procedures have been added to Halstead's tests to form the current HRNB. Reitan and his colleagues modified the Halstead-Wepman Aphasia Screening Test (Halstead & Wepman, 1948) reducing the number of items to 32 (Lezak, 1983). A cross section of language disabilities may be identified by the Reitan-Indiana Aphasia Screening Test including dysnomia, dyslexia, dysgraphia, body dysgnosia, ideokinetic dyspraxia, dyscalculia, dysarthria, and auditory-verbal dysgnosia (Boll, 1981). The patient also demonstrates constructional ability by copying simple geometric designs and a key. Set rules for scoring the test were deliberately omitted and the results are presented descriptively (Heimburger & Reitan, undated). A scoring system has been devised by Russell and colleagues (Russell et al., 1970) to facilitate computerized patient classification.

The Trail Making Test was adapted from the version contained in the Army Individual Test Battery (1944). On the first part of the test the patient is instructed to draw a continuous line as quickly as possible connecting circles numbered sequentially. The total time required to correctly complete the task reflects manual dexterity, visuospatial organization, and motor execution under time pressure. Task
complexity is increased in the second part of the Trail Making Test by requiring that a continuous line be drawn connecting numbered or lettered circles alternating numerical and alphabetic sequences. The total time required to complete the second part of the test provides information about the patient's cognitive flexibility (Lezak, 1983).

The Reitan-Klove Sensory-Perceptual Examination standardizes the assessment of visual, auditory, and tactile sensory perception by presenting unilateral and bilateral stimuli. Results not only reflect the gross acuity of sensory perception in these modalities but also reveals suppression of sensory inputs. Finger gnosis, finger graphesthesia, and stereognosis are evaluated in the Tactile Finger Recognition, Fingertip Number Writing, and Tactile Form Recognition tests. In all of these procedures, the number of errors on the right and left sides are recorded (Boll, 1981; Reitan & Wolfson, 1985).

WAIS-R. Wechsler's tests of adult intelligence are the most frequently used in neuropsychology (Hartlage, Chelune, & Tucker, 1982) and have been administered as part of the HRNB since the early 1950's (Reitan & Wolfson, 1985). The WAIS-R is gradually replacing the previous edition in neuropsychological evaluations (Bornstein, 1987.) The WAIS-R incorporates eleven subtests divided into the Verbal and
Performance Scales (Wechsler, 1981). The six subtests of the Verbal Scale are Information, Digit Span, Vocabulary, Arithmetic, Comprehension, and Similarities. The Information subtest assesses the patient's general knowledge of the world and events of the present century. In first part of the Digit Span subtest the patient repeats series of numerical digits of increasing length until two trials at the same level of difficulty are missed. The patient is then required to repeat number series in reverse. Verbal definitions of individual words presented both visually and auditorially are elicited from the subject in the Vocabulary subtest. The Arithmetic subtest challenges the patient to solve word problems within a specified time limit. The Comprehension subtest presents the subject with both practical and abstract questions testing common-sense judgement and reasoning. Abstraction ability is more directly assessed in the Similarities subtest in which the patient describes the most fundamental feature shared by two items or concepts (Lezak, 1983; Lindemann & Matarazzo, 1984).

The Performance Scale includes the Picture Completion, Picture Arrangement, Block Design, Object Assembly, and Digit Symbol subtests. Identification of an essential missing component in several line drawings is the task assigned to the patient in the Picture Completion subtest.
In the Picture Arrangement subtest the patient is presented with an array of cartoon-like cards which must be placed in the proper sequence within a limited period of time. The Block Design subtest is another timed procedure and involves reproducing geometrical patterns of increasing difficulty with colored cubes. Four jigsaw-type items are put together as quickly as possible by the patient in the Object Assembly subtest. In the Digit Symbol subtest, the subject is required to copy geometrical symbols into a series of squares which correspond to numerals printed above each box (Lezak, 1983; Lindemann & Matarazzo, 1984).

In addition to scaled scores for each subtest, the WAIS-R yields composite indices of cognitive ability. The scaled scores of the Verbal Scale and Performance Scale subtests are summed and provide standardized Verbal and Performance Intelligence Quotients (VIQ & PIQ). A global estimate of intelligence is obtained from the combination of VIQ and PIQ (Wechsler, 1981). Factor analytic studies of both the WAIS-R and its predecessor have usually identified 3 components: verbal comprehension, perceptual organization, and memory and freedom from distraction (Chelune, Ferguson, & Moehle, 1986).

WMS. The Wechsler Memory Scale (Wechsler, 1945) is the most frequently administered clinical test of memory (Erickson &
Scott, 1977). Even though the Wechsler Memory Scale suffers from psychometric deficiencies including poor norms, lack of standardized scores for subtests, inadequate scoring criteria for the Logical Memory subtest, and an outdated conceptualization of memory (Erickson & Scott, 1977; Prigatano, 1978), it has been found to be a useful component of neuropsychological evaluations (Chelune et al., 1986). Based on Russell's (1975) revision of the WMS, only the Logical Memory and Visual Reproduction subtests were routinely administered to patients at Tucson VAMC. In the Logical Memory subtest the patient repeats verbatim the content of two stories presented in order. Four stimulus cards on which are printed geometric designs are shown to the patient one at a time in the Visual Reproduction subtest. The patient draws the figures from memory onto a standardized answer form. Following a 20 minute delay, during which the Halstead Speech-sounds and Seashore Rhythm Tests are administered, the patient's recall of both the meaningful verbal material and the geometric designs is obtained. Thus, for both the verbal and non-verbal components of the WMS three scores are obtained reflecting immediate and delayed recall as well as the percentage of content lost over time (Chelune et al., 1986).

Raw scores for each of the neuropsychological tests are listed in Tables 8 and 9. A summary score of
neuropsychological performance was calculated by first converting raw scores into T-scores using the means and standard deviations for veterans reported by Russell (1987, 1988). The T-scores of each patient were averaged to yield a single measure of neurocognitive ability.

**Level of Risk.** Each medical record was reviewed twice to insure both the accuracy and comprehensiveness of the information extracted. For the sake of convenience, the medical data were divided into categories representing the major variables contributing to CVA risk as identified by previous researchers: prior CVA, hypertension, heart disease, clinical or laboratory evidence of atherosclerosis or other forms of arterial disease (excluding cardiac disease), diabetes, blood disorders, other medical conditions, and substance abuse (including abuse of alcohol and tobacco). Reported differences in the effect of a particular factor were averaged. In order to include potential sources of CVA risk which remain controversial or for which no specific risk or incidence data have been published, a risk level of 0.2 per hundred population, the lowest level established for any factor, was arbitrarily assigned. Similarly, a "ceiling" risk level of 12 per hundred population was assigned for those medical conditions in which the likelihood of sustaining a CVA is substantial.
(see Tables 10 through 15). The CVA risk for each patient was determined by adding the highest level of risk attained in each risk category. For example, a patient might have a history of MIs, ECG evidence of LVH, and chronic AF. Only the highest risk level, 5.0, would be used in determining the total risk.

Health Care Utilization. HCU was defined as the total number of medical visits, including hospitalizations, during a specified period of time (Carlsson et al., 1987; Stein et al., 1987). In order to insure that HCU figures for each patient were not artificially inflated by the effects of acute illness at the time of evaluation (Egan & Katon, 1987), data were gathered for two periods: the 24 months preceding and the 24 months following neuropsychological assessment. A potential weakness of HCU was the possibility that a particular veteran might elect to obtain medical care from non-VAMC providers. It was assumed that veterans residing outside of Tucson would be more likely to rely on local rather than VAMC medical services. No significant difference was found in HCU among veterans residing outside of Tucson.

Results

As expected, the hierarchical multiple regression
analysis performed with SYSTAT (Wilkinson, 1986) using the
default alpha of 0.15 and the force option, showed that both
age and education accounted for a significant amount of
variance (R-Squared = .291, p < .001) in neuropsychological
test performance. Neither HCU or CVA risk were significant
(see Table 16).

Discussion

The failure to confirm the predicted relationships
between neuropsychological performance, CVA risk, and
functional status may be less reflective of the true
association between these variables than of the current
state of epidemiological and neurocognitive research.
Despite the noteworthy gains in methods of risk estimation
and neuropsychological assessment that have been realized
during the last four decades, more accurate and precise
procedures continue to evolve. In addition to the
deficiencies of current neuroepidemiologic and
neurobehavioral techniques, the present study may have been
hampered by an inadequate measure of functional impairment,
an incomplete and/or inaccurate pool of medical records, and
an insufficient number of suitable cases.

Among the challenges to the estimation of CVA risk are
changes in the incidence of CVAs, differences in the
etiology of CVA subtypes, the presence of "confounders," the
complex interrelationships among risk factors, and the relatively infrequent occurrence of CVAs in the general population. The incidence of stroke has been steadily declining since 1915 (Wolf, 1990). In the city of Rochester, Minnesota in which one of only three ideal time-trend studies is being conducted (Malmgren et al., 1989), stroke incidence has fallen by almost half between 1950 and 1980 (Broderick et al., 1989). Although it is generally believed that the decrease in CVA incidence is due to improvements in the detection and treatment of HTN, it appears that the decline in HTN pre-dated by several years the introduction of effective antihypertensive medication and the wide-spread concern with ancillary therapeutic regimen (Fields & Lemak, 1989). Nevertheless, the effect of changing frequency of disease necessitates continual revisions of risk levels (Brody, 1986). As in other attempts to organize lists of risk factors and their respective levels (see, for example, Ebrahim, 1990), epidemiologic data incorporated in the present study was collected at different points in time. Although care was taken to utilize risk levels established during the years in which the veterans were tested, the values reported in Tables 10 through 15 may not reflect the true, possibly lower risk faced by the subjects included in this study.

Related to the influence of changing levels of risk are
the improvements that have occurred in the recognition and classification of CVAs. Since 1980 CVAs are being diagnosed more frequently owing to improved neurodiagnostic imaging techniques (Broderick et al., 1989). More refined detection and discrimination of CVA types has led to a greater appreciation of differences in etiology. Risk factors for thromboembolic stroke may differ widely for those associated with hemorrhagic infarcts (Alter et al., 1985; Bamford & Warlow, 1988; Bogousslavski et al., 1988). Beghi and colleagues, for example reported several etiologic distinctions in their extensive case-control study (Beghi et al., 1989). The etiology of intracerebral hemorrhages may include factors not ordinarily identified as in exposure to severe cold weather, dental pain, and heart surgery (Caplan, 1988a). Both age and gender differences have also been found in the incidence of cerebral hemorrhage and infarction. In patients under 50, men are more likely than women to suffer intracerebral hemorrhage (Bogousslavski et al., 1988). Older patients sustain cardioembolic infarcts more frequently than younger individuals (Norrving & Löwenstein, 1988). There is, moreover, growing evidence of an etiologic as well as prognostic distinction between small- and large-vessel disease (Lodder, Bamford, Sandercock, Jones, & Warlow, 1990).

Another difficulty encountered in establishing levels
of risk is the presence of "confounders," variables that confer risk independently and that interact with other risk factors (Schlesselman & Stolley, 1982). In Table 9, for example, the independent effect of oral contraceptive medication is listed along with the combined effect of cigarette smoking which, in Table 10 is listed as an independent risk factor. In the present study, confounders may not only have complicated the assignment of overall CVA risk (see below) but may also have introduced an "input bias" (Kurtzke, 1984) which led to the inclusion of patients whose medical care reduced their likelihood of sustaining a CVA (Wolf & Kannel, 1986). For example, Sleight (1991) has reported that a reduction of as little as 5 mm Hg in diastolic blood pressure may lessen a patient's risk by 35 to 40 percent. Only for diseases with comparatively high rates of incidence have methods of weighting co-morbid conditions been developed (Charlson, Pompei, Ales, & MacKenzie, 1987).

Unmeasured intervening variables also plague epidemiological research (Angell, 1990; Thompson, 1991). For example, in this study the influence of heavy alcohol consumption on CVA risk was acknowledged. However, deficient performance on neuropsychological tests results less from the direct effect of drinking history than to the extent of liver disease. Cirrhosis, irrespective of cause,
impairs neurocognitive abilities (Tarter, Van Thiel, Arria, Carra, & Moss, 1988). Even among alcoholics, the serum level of gamma-glutamyl transferase predicts more precisely than drinking history the degree of neuropsychological impairment (Irwin et al., 1989).

Adjusting risk indices to account for confounders and unmeasured intervening variables requires more than the collection of additional data (Roos, Sharp, Cohen, & Wajda, 1989). It has become apparent that better mathematical models are needed to represent the relationships among the increasing number of variables that have been identified (Kreiner, 1989). As Golberg (1991) has indicated, the linear models typically employed do not provide a satisfactory representation of the complex interrelationships among risk factors and biologic function. The use of more complex analyses is constrained, however, by the number of available cases. For example, at least 200 cases are needed to satisfy the assumptions of structural equation modeling (Francis, 1988).

A small sample size may have led to the failure to realize the anticipated results due to the low incidence of CVAs in the general population. Actual CVA probabilities are relatively small, rarely exceeding 10 percent per year and, as noted above, are declining (Barnett, 1990; Broderick et al., 1989). For the patients included in this research,
the average odds of suffering a CVA within the study period were 4 in 100 (SD = 3.4). In fact, none of the patients sustained a CVA. As Angell (1990) noted, small levels of risk, however prominent in a large population, may have little or no meaning for the individual patient. It has been suggested that the best predictor of future disease may be the presence of a minor disease (Fletcher, Fletcher, & Wagner, 1988). For ischemic infarcts, a better measure of risk might have emphasized the actual degree of stenosis in the peripheral and central vasculature (Blombery, 1987). However, as Autret and colleagues cautioned, such data may be misleading (Autret et al., 1987).

In a recent editorial concerning stroke rehabilitation, Hachinski (1989) wrote that the behavioral sequelae of CVAs are heterogeneous and complex. Given the complexity of the relationships between infarcts and behavior (Mazziotta, 1986), the use of a global measure of neurocognitive status may have obscured more subtle deficits associated with increments in CVA risk. Scores for individual tests and for tests representing specific cognitive domains (e.g., memory, visuo-constructive ability) were substituted in the multiple regression equation. Neither level of risk nor health care utilization were more predictive of these measures than of global psychological ability. The small sample size may have contributed to the
failure to confirm the expected results as well.

The resolution of the measures employed in this study may not have been sufficient to detect the effects of increased CVA risk in so small a number of cases (Johnston, Findley, DeLuca, & Katz, 1991). Designed by Ward Halstead to reveal the effects of principally cortical lesions (see, for example, Halstead 1939, 1947), the battery of tests compiled and refined by Ralph Reitan has been applied to the study of other neurologic conditions (Reitan & Wolfson, 1985). There are, however, indications that some of the procedures routinely administered as part of the HRNB contribute little to the differentiation of brain-damaged individuals from pseudo-neurologic controls (see Sherer, Parsons, Nixon, & Adams, 1991).

It was assumed in this as well as other studies (e.g. Baird, Adams, Ausman, & Diaz, 1985) of the relationship between CVA risk, functional status, and neurobehavioral proficiency that increased CVA risk is associated with decreased blood flow and hence compromised metabolism. It has long been established that cerebral blood flow (CBF) declines with age (Leenders et al., 1990; Hagstadius & Risberg, 1989). Ewing and associates have also demonstrated that CBF is inversely related to stroke risk (Ewing et al., 1989; see also Yoshii et al., 1988). However, metabolic activity is not always compromised as individuals age nor is
it permanently suppressed in TIA or RIND (Duara et al., 1984; Ewing et al., 1989; Sokoloff, 1975; Herscovitch, 1989). Moreover, even though CBF may be reduced with advancing age or increased stroke risk, behavioral activation of blood flow may not be affected (Warren, Butler, Katholi, & Halsey, 1985). The vascular bed may remain sufficiently flexible to respond to behavioral challenges (Ewing et al., 1989).

Health care utilization may not represent the best index of functional status. Two factors confound HCU as an appropriate measure: gender and social class. With the exception of medical visits associated with accidental injury, women use medical care to a greater degree than men (Hibbard & Pope, 1986; Rice, Hing, Kovar, & Prager, 1984). Social class also influences health seeking behavior. Not only are members of the lowest socioeconomic stratum more likely to become ill, they are more likely to sustain CVAs (Shaper et al., 1991). At the same time, they are less apt seek medical advice (Liberatos, Link, & Kelsey, 1988). Although other measures of socioeconomic status have been devised (see Stevens & Featherman, 1981), educational attainment continues to be the most relevant to health care issues (Liberatos et al., 1988).

Gender and social class aside, HCU does not necessarily correlate highly with the severity of disease. The most
noteworthy exception to the generally weak association
between HCU and functional impairment is among patients with
CVD (Pope, 1988). Nevertheless, a more direct measure of
social adaptation and social support may have been more
useful (Brown et al., 1986; Stein et al., 1987; Tompkins,
Jackson, & Schulz, 1990).

Since 30 to 65 percent of stroke victims exhibit
symptoms of depression (Eastwood, Rifat, Nobbs, & Rudder- 
aman, 1989; Koenig & Studentski, 1988; Starkstein & Robinson,
1989) regardless of lesion location (House, Dennis, Warlow,
Hawton, & Molyneux, 1990; but see Bolla-Wilson, Robinson,
Starkstein, Boston, & Price, 1989), assessment of affect and
mood are particularly relevant (Cushman, 1988; Robinson,
Lipsey, & Price, 1985). In the domain of self-report
personality tests the MMPI (Dahlstrom, Welsh, & Dahlstrom,
1972) is the most widely used and researched instrument
(Butcher & Keller, 1984). Although the MMPI does not
effectively identify neuropsychological deficits (Chelune et 
al., 1986) or distinguish post-CVA emotional disturbances
from those resulting from other serious medical illnesses
(House, 1988), it does provide important information about
the patient's level of adjustment in the face of
neurocognitive dysfunction (Filskov & Leli, 1981). The MMPI
has been used routinely in conjunction with the HRNB (Reitan
and Wolfson, 1985). Among those for whom MMPI data were
available, no associations between neuropsychological test performance, health care utilization, or CVA risk and individual MMPI scale elevations or the average MMPI scores were significant. These findings must be interpreted cautiously as only half of the CVA group (N = 11) completed MMPIs.

Whenever patient records are used to conduct a retrospective study there is always a risk that the sample may not reflect the true population (Williams, Kingham, Morgan, & Davies, 1990). Although the pool from which subjects were selected for this investigation is representative of patients served by Veterans' Hospitals, it is recognized that important segments of the general population of individuals at risk were excluded. Not only were women under-represented but so also were individuals who elect to obtain health care elsewhere or who choose not to seek care. Consequently, subjects with greater risk were probably excluded from the present study. There is, moreover, a growing concern among physicians that the information contained in medical records is inadequate because of patient access and the classifications of care that have been imposed by third party reimbursers (Burnum, 1989). Of course, patients themselves do not always furnish accurate information regarding current symptoms or medical history. This is especially vexing when health care
professionals ask patients about their use of psychoactive substances (Carlsson et al., 1987).

While this study did not produce significant results, the necessity to investigate the impact of risk on health care use and functional impairment continues to grow as the population ages. It is clear that an investigation into the relationship between neuropsychological performance, functional severity of disease, and health care utilization requires a larger and more representative sample of subjects than were used in this study. A cross-sectional design, for example, in which patients who are not referred for neuropsychological evaluation are also studied might yield stronger associations between behavior and disease risk. Larger subject pools might be obtained through multi-center efforts. More sensitive measures of neurocognitive deficit may be needed, especially with smaller samples. It may be necessary to target a more specific CVA type and to focus on the etiologic factors that are specific to it (Caplan, 1988b).
Table 1: Comparison of VAMC Patients Reported by Russell (1987, 1988) and the Present Study

<table>
<thead>
<tr>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>N</td>
<td>732</td>
<td>690</td>
<td>261</td>
</tr>
<tr>
<td>Age (SD)</td>
<td>47.7 (14.2)</td>
<td>48.3 (14.1)</td>
<td>51.8* (14.6)</td>
</tr>
<tr>
<td>Education (SD)</td>
<td>11.9 (3.3)</td>
<td>12.0 (3.2)</td>
<td>12.0 (3.0)</td>
</tr>
<tr>
<td>Percent Female</td>
<td>5.2</td>
<td>4.5</td>
<td>4.6</td>
</tr>
<tr>
<td>Percent Non-white</td>
<td>9.7</td>
<td>9.0</td>
<td>12.0**</td>
</tr>
</tbody>
</table>

*p<.05, t-test of differences between means

**p<.05, X² test of comparisons between proportions
Table 2: Characteristics of Cases Rejected for History of Head Injury with Loss of Consciousness and Non-CVA Central Nervous System (CNS) Disorders

<table>
<thead>
<tr>
<th></th>
<th>Head Injury</th>
<th>CNS Disorders</th>
</tr>
</thead>
<tbody>
<tr>
<td>N</td>
<td>71</td>
<td>27</td>
</tr>
<tr>
<td>Age, yrs (SD)</td>
<td>48.4 (13.7)</td>
<td>50.1 (15.3)</td>
</tr>
<tr>
<td>Education, yrs (SD)</td>
<td>11.9 (2.9)</td>
<td>11.7 (3.9)</td>
</tr>
<tr>
<td>Percent Female</td>
<td>2.8%</td>
<td>10.7%</td>
</tr>
<tr>
<td>Percent Hispanic</td>
<td>9.8%</td>
<td>3.4%</td>
</tr>
</tbody>
</table>
Table 3: Characteristics of Cases Rejected for History of Substance Abuse and Psychiatric Disorders

<table>
<thead>
<tr>
<th></th>
<th>Substance Abuse</th>
<th>Psychiatric</th>
</tr>
</thead>
<tbody>
<tr>
<td>N</td>
<td>47</td>
<td>19</td>
</tr>
<tr>
<td>Age, yrs (SD)</td>
<td>47.4 (13.5)</td>
<td>47.7 (14.9)</td>
</tr>
<tr>
<td>Education, yrs (SD)</td>
<td>11.9 (2.6)</td>
<td>11.5 (3.0)</td>
</tr>
<tr>
<td>Percent Female</td>
<td>0%</td>
<td>0%</td>
</tr>
<tr>
<td>Percent Hispanic</td>
<td>21.3%</td>
<td>3.4%</td>
</tr>
</tbody>
</table>
Table 4: Characteristics of Missing and Incomplete Cases

<p>| | |</p>
<table>
<thead>
<tr>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>N</td>
<td>38</td>
</tr>
<tr>
<td>Age, yrs (SD)</td>
<td>58.0 (15.8)</td>
</tr>
<tr>
<td>Education, yrs (SD)</td>
<td>11.3 (3.2)</td>
</tr>
<tr>
<td>Percent Female</td>
<td>10.5%</td>
</tr>
<tr>
<td>Percent Hispanic</td>
<td>7.9%</td>
</tr>
</tbody>
</table>
Table 5: Comparison of Included and Rejected Cases

<table>
<thead>
<tr>
<th></th>
<th>Included</th>
<th>Rejected</th>
</tr>
</thead>
<tbody>
<tr>
<td>N</td>
<td>58</td>
<td>203</td>
</tr>
<tr>
<td>Age, yrs (SD)</td>
<td>57.5 (12.0)</td>
<td>50.4 (14.2)*</td>
</tr>
<tr>
<td>Education, yrs (SD)</td>
<td>12.9 (3.0)</td>
<td>11.7 (2.9)</td>
</tr>
<tr>
<td>Percent Female</td>
<td>5.9</td>
<td>4.3</td>
</tr>
<tr>
<td>Percent Non-white</td>
<td>7.8</td>
<td>12.9</td>
</tr>
</tbody>
</table>

*p<.05, t-test of differences between means
Table 6: Characteristics of CVA and non-CVA Patients

<table>
<thead>
<tr>
<th></th>
<th>CVA</th>
<th>non-CVA</th>
</tr>
</thead>
<tbody>
<tr>
<td>N</td>
<td>23</td>
<td>35</td>
</tr>
<tr>
<td>Age, yrs (SD)</td>
<td>62.6 (8.9)</td>
<td>53.4 (13.1)*</td>
</tr>
<tr>
<td>Education, yrs (SD)</td>
<td>12.6 (2.6)</td>
<td>12.8 (2.8)</td>
</tr>
<tr>
<td>Intelligence** (SD)</td>
<td>98.4 (13.0)</td>
<td>99.7 (12.1)</td>
</tr>
<tr>
<td>Employment:</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Employed</td>
<td>0</td>
<td>9</td>
</tr>
<tr>
<td>Retired</td>
<td>10</td>
<td>13</td>
</tr>
<tr>
<td>Disabled</td>
<td>13</td>
<td>13***</td>
</tr>
<tr>
<td>Married:</td>
<td></td>
<td></td>
</tr>
<tr>
<td>No</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>Yes</td>
<td>16</td>
<td>26</td>
</tr>
<tr>
<td>Sep/Div</td>
<td>4</td>
<td>7</td>
</tr>
<tr>
<td>Widow/er</td>
<td>2</td>
<td>1</td>
</tr>
<tr>
<td>Disability:</td>
<td></td>
<td></td>
</tr>
<tr>
<td>0%</td>
<td>9</td>
<td>9</td>
</tr>
<tr>
<td>1-49%</td>
<td>7</td>
<td>18</td>
</tr>
<tr>
<td>50-100%</td>
<td>7</td>
<td>8</td>
</tr>
<tr>
<td>Percent Female</td>
<td>10%</td>
<td>3%</td>
</tr>
<tr>
<td>Percent Non-White</td>
<td>4%</td>
<td>9%</td>
</tr>
</tbody>
</table>

*p<.05, t-test for difference between means

**WAIS-R Full-scale IQ

***p<.05, Kruskal-Wallis One-Way ANOVA
Table 7: Percent of Additional Diagnostic Procedures Performed on CVA and non-CVA Patients

<table>
<thead>
<tr>
<th>Procedure</th>
<th>CVA</th>
<th>non-CVA</th>
</tr>
</thead>
<tbody>
<tr>
<td>Head CT Scan</td>
<td>82.6</td>
<td>52.3*</td>
</tr>
<tr>
<td>Carotid Angiogram</td>
<td>52.2</td>
<td>14.3*</td>
</tr>
<tr>
<td>Electroencephalogram (EEG)</td>
<td>78.3</td>
<td>40.0*</td>
</tr>
<tr>
<td>Electrocardiogram (ECG)</td>
<td>47.8</td>
<td>40.0</td>
</tr>
</tbody>
</table>

*p<.05, Kruskal-Wallis one-way ANOVA
Table 8: Mean Raw Scores of CVA and non-CVA Patients on Halstead’s Neuropsychological Tests

<table>
<thead>
<tr>
<th></th>
<th>CVA</th>
<th>non-CVA</th>
</tr>
</thead>
<tbody>
<tr>
<td>Category Test (errors)</td>
<td>72.3 (35.1)</td>
<td>64.3 (23.6)</td>
</tr>
<tr>
<td>Tactual Performance Test</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Total Time (sec)</td>
<td>28.3 (17.1)</td>
<td>19.3 (11.2)*</td>
</tr>
<tr>
<td>Memory (correct)</td>
<td>5.6 (2.5)</td>
<td>7.2 (1.6)*</td>
</tr>
<tr>
<td>Localization (correct)</td>
<td>2.0 (2.0)</td>
<td>3.2 (2.4)*</td>
</tr>
<tr>
<td>Rhythm Test (errors)</td>
<td>6.5 (4.4)</td>
<td>4.8 (3.6)</td>
</tr>
<tr>
<td>Speech Perception (errors)</td>
<td>9.9 (6.6)</td>
<td>8.7 (6.4)</td>
</tr>
<tr>
<td>Finger Tapping (number)</td>
<td>42.1 (9.2)</td>
<td>43.7 (9.4)</td>
</tr>
</tbody>
</table>

*p<.05, t-test of difference between means
Table 9: Mean Raw Scores of CVA and non-CVA Patients on Additional HRNB Tests and the WMS

<table>
<thead>
<tr>
<th>Test</th>
<th>CVA</th>
<th>non-CVA</th>
</tr>
</thead>
<tbody>
<tr>
<td>Aphasia Screening Test*</td>
<td>1.2 (1.8)</td>
<td>1.8 (2.3)</td>
</tr>
<tr>
<td>Sensory Perception*</td>
<td>14.2 (8.9)</td>
<td>14.5 (17.0)</td>
</tr>
<tr>
<td>Tactile Form Recognition</td>
<td>33.8 (16.7)</td>
<td>28.3 (13.1)</td>
</tr>
<tr>
<td>Trail Making Test</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Part A</td>
<td>54.6 (17.8)</td>
<td>38.7 (15.5)***</td>
</tr>
<tr>
<td>Part B</td>
<td>177.9 (106.3)</td>
<td>109.7 (62.1)***</td>
</tr>
<tr>
<td>WMS-Logical Memory**</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Immediate</td>
<td>14.9 (8.0)</td>
<td>16.9 (7.2)</td>
</tr>
<tr>
<td>Delayed</td>
<td>11.1 (8.0)</td>
<td>12.8 (7.2)</td>
</tr>
<tr>
<td>WMS-Visual Reproduction**</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Immediate</td>
<td>7.2 (3.5)</td>
<td>9.2 (2.9)***</td>
</tr>
<tr>
<td>Delayed</td>
<td>5.6 (3.7)</td>
<td>7.3 (3.9)</td>
</tr>
</tbody>
</table>

*Error scores (Russell, 1987)

**Number of correct elements (Wechsler, 1945; Russell, 1988)

***p<.05, t-test of difference between means
<table>
<thead>
<tr>
<th>Chronicity/Severity</th>
<th>Risk/100 Pop.</th>
</tr>
</thead>
</table>

**Prior CVA**

- **Stroke or RIND**
  - 0-6 mos: 8.5
- **TIA**
  - 0-6 mos: 5.0
- **Stroke + RIND/TIA**
  - 7-12 mos: 5.0
  - 13-60 mos: 3.0
  - 61-120 mos: 0.8

**Diagnosed Hypertension**

- NA: 0.5

*Note. Risk represents average of differing findings.*

Alter et al. (1987); Bonita et al. (1986); Dennis et al., 1990; Dollery (1987); Gorelick et al. (1989); Henrich & Horwitz (1989b); Meissner et al. (1988); Shaper et al. (1991) Sorensen et al. (1989); Syrjänen et al. (1988); & Wolf et al. (1988)
Table 11: CVA Risk Due to Heart Disease

<table>
<thead>
<tr>
<th>Heart Disease</th>
<th>Chronicity/Severity</th>
<th>Risk/100 Pop.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Atrial Fibrillation</td>
<td>Chronic</td>
<td>5.0</td>
</tr>
<tr>
<td></td>
<td>Paroxysmal</td>
<td>2.0</td>
</tr>
<tr>
<td>Coronary Artery Stenosis</td>
<td>Greater than 50%</td>
<td>2.2</td>
</tr>
<tr>
<td></td>
<td>Less than 50%</td>
<td>0.3</td>
</tr>
<tr>
<td>Heart Valve Prosthesis</td>
<td>Chronic</td>
<td>2.0</td>
</tr>
<tr>
<td>Any form of Heart Disease</td>
<td></td>
<td></td>
</tr>
<tr>
<td>with MI</td>
<td>Chronic</td>
<td>1.2</td>
</tr>
<tr>
<td>without MI</td>
<td>Chronic</td>
<td>1.0</td>
</tr>
<tr>
<td>Angina or LVH</td>
<td>Any</td>
<td>0.5</td>
</tr>
<tr>
<td>Mitral Valve Prolapse</td>
<td>Any</td>
<td>0.5</td>
</tr>
<tr>
<td>Patent Foramen Ovale</td>
<td>Chronic</td>
<td>0.2</td>
</tr>
</tbody>
</table>

Note. Risk represents average of differing findings.
Alter et al. (1987); Autret et al. (1987); Dexter et al. (1987); Goldberg & Berger (1988); Hart & Easton, 1982; Henrich & Horwitz (1989b); Kannel et al. (1983); Knutsen et al. (1988); Lechat et al. (1988); Lund (1988); Petersen et al. (1987); & Wolf et al. (1987)
### Table 12: CVA Risk Due to Arterial (non-Cardiac) Disease

<table>
<thead>
<tr>
<th>Arterial Disease</th>
<th>Chronicity/Severity</th>
<th>Risk/100 Pop.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Primary Cerebral Angiitis Acute</td>
<td>Acute</td>
<td>12.0</td>
</tr>
<tr>
<td>Amaurosis Fugax Acute</td>
<td>Acute</td>
<td>5.0</td>
</tr>
<tr>
<td>Retinal Emboli Acute</td>
<td>Acute</td>
<td>5.0</td>
</tr>
<tr>
<td>AVM Chronic</td>
<td>Chronic</td>
<td>2.2</td>
</tr>
<tr>
<td>Abdominal Aortic Aneurysm Chronic</td>
<td>Chronic</td>
<td>1.8</td>
</tr>
<tr>
<td>Carotid Artery Bruits Acute</td>
<td>Acute</td>
<td>1.8</td>
</tr>
<tr>
<td>Carotid Artery Stenosis &gt;50%</td>
<td>Chronic</td>
<td>1.8</td>
</tr>
<tr>
<td>Intermittent Claudication Chronic</td>
<td>Chronic</td>
<td>1.8</td>
</tr>
</tbody>
</table>

**Note.** Risk represents average of differing findings.

Autret et al. (1987); Berkoff & Levine (1988); Brown et al. (1988); Calabrese & Mallek (1987); Folger (1987); Gomez (1989, 1990); Kreshon (1986); Millikan et al. (1987); Nashitz et al. (1988); Parnetti et al. (1988); Schoenfeld et al. (1988)
Table 13: CVA Risk Due to Blood Disorders and Diabetes

<table>
<thead>
<tr>
<th>Chronicity/Severity</th>
<th>Risk/100 Pop.</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Blood Disorders</strong></td>
<td></td>
</tr>
<tr>
<td>Thrombocytopenic Purpura</td>
<td>Acute</td>
</tr>
<tr>
<td>Sickle Cell Disease</td>
<td>Chronic</td>
</tr>
<tr>
<td>Polycythemia Vera</td>
<td>1-5 yrs</td>
</tr>
<tr>
<td></td>
<td>6-10 yrs</td>
</tr>
<tr>
<td><strong>Elevated Serum Levels</strong></td>
<td></td>
</tr>
<tr>
<td>Fibrinogen</td>
<td>Chronic</td>
</tr>
<tr>
<td>Hematocrit</td>
<td>Chronic</td>
</tr>
<tr>
<td>Hemoglobin</td>
<td>Chronic</td>
</tr>
<tr>
<td>Lipids/lipoproteins</td>
<td>Chronic</td>
</tr>
<tr>
<td><strong>Diabetes</strong></td>
<td></td>
</tr>
<tr>
<td>Diabetes Mellitus</td>
<td>Chronic</td>
</tr>
<tr>
<td>Glucosuria</td>
<td>Chronic</td>
</tr>
<tr>
<td>Glucose Intolerance</td>
<td>Chronic</td>
</tr>
</tbody>
</table>

Note. Risk represents average of differing findings.
Abbott et al. (1987); Alter et al. (1987); Goldberg & Berger (1988); Henrich & Horwitz (1989b); Najean et al. (1987); Pavlakis et al. (1988); Pettit (1980); Wolf et al. (1988)
Table 14: CVA Risk Due to Other Medical Conditions

<table>
<thead>
<tr>
<th>Chronicity/Severity</th>
<th>Risk/100 Pop.</th>
</tr>
</thead>
<tbody>
<tr>
<td>AIDS</td>
<td>n/a</td>
</tr>
<tr>
<td>Hemodialysis</td>
<td>Chronic</td>
</tr>
<tr>
<td>Oral Contraceptive Use</td>
<td></td>
</tr>
<tr>
<td>with smoking</td>
<td>Current</td>
</tr>
<tr>
<td>without smoking</td>
<td>Current</td>
</tr>
<tr>
<td>Febrile Infections</td>
<td>0-30 days</td>
</tr>
<tr>
<td>Migraine Headaches</td>
<td></td>
</tr>
<tr>
<td>Classic</td>
<td>Chronic</td>
</tr>
<tr>
<td>Other</td>
<td>Chronic</td>
</tr>
<tr>
<td>Epilepsy (post age 30)</td>
<td>Acute</td>
</tr>
<tr>
<td>Obesity</td>
<td>Chronic</td>
</tr>
</tbody>
</table>

Note. Risk represents average of differing findings.
Bonita et al. (1986); Engstrom et al. (1989); Goldberg & Berger (1988); Gerstman et al. (1991); Gorelick et al. (1989); Henrich & Horwitz (1989a & 1989b); Petitti et al. (1979); Rosengren et al. (1988); Trompeter et al., (1986)
Table 15: CVA Risk Due To Substance Abuse

<table>
<thead>
<tr>
<th>Substance</th>
<th>Chronicity/Severity</th>
<th>Risk/100 Pop.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Alcohol Use</td>
<td>80 grams/24 hours</td>
<td>0.8</td>
</tr>
<tr>
<td></td>
<td>330 grams/week</td>
<td>0.5</td>
</tr>
<tr>
<td>Tobacco Use</td>
<td>20+ cigarettes/day</td>
<td>0.3</td>
</tr>
<tr>
<td>Sympathomimetics</td>
<td>Acute/Chronic</td>
<td>0.2</td>
</tr>
</tbody>
</table>

Note: Risk represents average of differing findings.
Gill et al. (1986); Kaku & Lowenstein (1990); Shaper et al. (1991); Syrjänen et al. (1988)
Table 16: Regression Coefficients and ANOVA

<table>
<thead>
<tr>
<th></th>
<th>Standardized Coefficient</th>
<th>R-Squared</th>
<th>T</th>
<th>P (2 Tail)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Constant</td>
<td>0.00</td>
<td></td>
<td>5.86</td>
<td>0.000</td>
</tr>
<tr>
<td>Age</td>
<td>0.39</td>
<td>0.18</td>
<td>2.65</td>
<td>0.004</td>
</tr>
<tr>
<td>Education</td>
<td>-0.35</td>
<td>0.29</td>
<td>-2.95</td>
<td>0.004</td>
</tr>
<tr>
<td>Health Care</td>
<td>-0.17</td>
<td>0.32</td>
<td>-1.43</td>
<td>0.151</td>
</tr>
<tr>
<td>CVA Risk</td>
<td>-0.02</td>
<td>0.32</td>
<td>-0.13</td>
<td>0.893</td>
</tr>
</tbody>
</table>

Full Model Test

<table>
<thead>
<tr>
<th>Sum-of Squares</th>
<th>DF</th>
<th>Mean-Square</th>
<th>F-Ratio</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Regression</td>
<td>844.4</td>
<td>4</td>
<td>211.1</td>
<td>6.19</td>
</tr>
<tr>
<td>Residual</td>
<td>1808.3</td>
<td>53</td>
<td>32.2</td>
<td></td>
</tr>
</tbody>
</table>
References


Caplan, L. R. (1988b). TIAs: We need to return to the question, 'What is wrong with Mr. Jones?' *Neurology, 38*, 791-793.


Henrich, J. B., & Horwitz, R. I (1989c). Evidence against the association between alcohol use and ischemic stroke. Archives of Internal Medicine, 149, 1413-1416.


Pincus, T., Callahan, L. F., & Burkhauser, P. V. (1987). Most chronic diseases are reported more frequently by individuals with fewer than 12 years of formal education in the age 18-64 United States population. *Journal of Chronic Disease, 40*, 865-874.


