ORIGINAL ARTICLE

Executive Function Deficits in Acute Stroke

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ABSTRACT. Zinn S, Bosworth HB, Hoenig HM, Swartzwelder HS. Executive function deficits in acute stroke. Arch Phys Med Rehabil 2007;88:173-80.

Objectives: To establish the frequency of executive dysfunction during acute hospitalization for stroke and to examine the relationship of that dysfunction to stroke severity and premorbid characteristics.

Design: Inception cohort study.

Setting: Inpatient wards at a Veterans Affairs hospital.

Participants: Consecutive sample of inpatients with radiologically or neurologically confirmed stroke. Final sample included 47 patients screened for aphasia and capable of neuropsychologic testing. Two nonstroke inpatient control samples (n=10 each) with either transient ischemic attack (TIA) or multiple stroke risk factors were administered the same research procedure and tests.

Interventions: Not applicable.

Main Outcome Measures: Composite cognitive impairment ratio (CIR), calculated from 8 scores indicative of executive function on 6 neuropsychologic tests by dividing number of tests completed into the number of scores falling below cutoff point, defined as 1.5 standard deviations below normative population mean.

Results: Stroke patients had a mean CIR of .61, compared with .48 for TIAs and .44 for stroke-risk-only. Analysis of variance revealed that CIRs of stroke-risk-only patients but not TIAs were lower than those of the stroke patients ($P=.02$). Impairment frequencies were at least 50% for stroke patients on most test scores. The Symbol Digit Modalities Test (75% impairment) and a design fluency measure distinguished stroke from nonstroke patients. CIR was not related to stroke severity in the stroke patient sample, but was related to estimated premorbid intelligence.

Conclusions: Executive function deficits are common in stroke patients. The data suggest that limitations in information processing due to these deficits may require environmental and procedural accommodations to increase rehabilitation benefit.

Key Words: Attention; Cognition disorders; Rehabilitation; Stroke.

PATIENTS WITH RECENT STROKE who are beginning rehabilitation often have cognitive impairment, either predating or acquired with the stroke. Deficits in the particular cognitive processes known as executive functions, which manage goal-oriented behavior, are common poststroke[1,2] and reduce stroke treatment effectiveness.[3,4] Current knowledge of executive function deficits is typically based on studies conducted 3 months or more poststroke. Prevalence of executive function deficits may be even higher early after stroke, when the initial rehabilitative training occurs, but this is unknown. No neuropsychologic studies of executive functions in acute stroke have been conducted, to our knowledge.

Although executive functions may broadly be said to manage goal-oriented behavior, several component processes have been identified. Important components of the executive functions include starting and stopping behavior at appropriate times, persisting at a task or switching tactics as needed, and selecting behaviors in novel situations based on context and higher-level or long-term goals. These components are also denoted (respectively) as initiation/perseveration, cognitive persistence and flexibility, self-monitoring, and abstract thinking (including planning). Working memory, the capacity for storing and manipulating data during problem solving, is dependent on frontal cortex (and other brain regions) and is often included as an executive function.

Combined deficits of these components create impairments that can compromise rehabilitation treatment in varying ways. For example, rehabilitation patients with deficits in initiation and persistence may have a reduced capacity to initiate, sequence, and sustain a series of exercises, due to their executive function impairment, and thus have reduced functional recovery after stroke.[3] Deficits of initiation and perseveration may also result in an impairment, producing compulsive repetition of a behavioral sequence.[5] Impairments of planning and/or problem-solving can lead to unsafe physical maneuvers and increase the risk of falls.[6,7] When there is a generalized deficit of attention and/or cognitive speed in addition to executive function deficits, the ability to process novel or complex information is curtailed, leading to limits in information processing capacity.[8] Impaired planning, reduced prospective memory (remembering to remember something), and reduced information-processing capacity may make it difficult for rehabilitation patients to remember and follow the complex treatment regimens, often provided at discharge, that are designed to promote functional gains and reduce their risk of stroke recurrence. Post-rehabilitation functional improvement has been related to executive function scores[9] and providing cognitive remediation has improved performance of activities of daily living (ADLs) in stroke patients.[10]

Poststroke cognitive impairment of any type has been repeatedly related to stroke severity,[11,12] but recent studies suggest that executive function decline may begin prior to completed strokes.[13-17] It appears that small vessel ischemic disease in white and subcortical gray matter leads to decrements in executive functioning[18-20] that primarily affect processing speed[21] and cognitive flexibility.[15] Functional decline, especially in instrumental ADLs, has been related to executive function impairment even in community-dwelling samples.[22-25] Thus any executive function deficit occurring as a

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sequelae of stroke may be amplified by prestroke executive function decline.

We believe that stroke outcomes can be improved if executive function deficits are identified and compensatory techniques are incorporated into treatment early. The study reported here was designed to establish the frequency and correlates of executive function deficits occurring during acute hospitalization for stroke. For this study, acute hospitalization was defined as the initial inpatient period (typically 3−7 days at our facility) during which symptoms were stabilized, diagnostic studies were completed, and rehabilitation was initiated.

Identifying executive function deficits in acute stroke patients through neuropsychologic assessment is also needed because these deficits are more subtle than aphasia or neglect, and health care providers working with stroke patients have often been taught little about this area of cognitive functioning. Furthermore, identification of deficits is difficult because executive functions are multifaceted, and are most greatly activated in novel or unstructured situations. Most studies of any type of cognitive impairment in acute stroke involve brief screening tests such as the Mini-Mental Status Examination (MMSE),[26,27] yet the MMSE is relatively insensitive to executive function deficits.[28] Studies using more in-depth testing, thereby including some executive function measures, are typically conducted at 3 months[2] or more poststroke.[29] The only study we found that conducted neuropsychologic tests earlier than 3 months performed assessment at 1 month, but included only a single letter sorting task that might be considered an executive function measure.[30]

In general, executive function assessment in research studies of stroke has typically been conducted using 1 or 2 tests. If these are reported individually, often only the mean and standard deviation is presented. Rates of impairment are rarely reported, so it is difficult to determine whether a highly impaired minority or a broad reduction in scores are responsible for lowered means. Furthermore, the multiple aspects of executive functioning are not well captured by 1 or 2 test scores, especially in a stroke population who have broad variability among their deficits.

Therefore, we administered a battery of executive function tests to patients recently admitted for workup of acute stroke. We wanted to determine the rates of executive function impairment and to examine their relationship with the severity of the index stroke, prestroke functional decline, and patient characteristics such as age and intelligence. We examined both acute symptoms (stroke severity) and premorbid functioning as correlates of executive function deficits, and examined patients without stroke who may evidence this decline. Age, education, and intelligence are common correlates of many aspects of cognitive functioning, so they were examined as well. We predicted a strong relationship between rates of impairment and stroke severity. To better appreciate the context of our expected impairment rates and gain some understanding of possible prestroke decline and other factors, we administered the same neuropsychologic battery to small samples of patients with either transient ischemic attack (TIA) or stroke risk factors alone, who had not had a stroke. We hypothesized, however, that the stroke patients overall would have higher rates of executive function impairment than vascular disease patients without stroke.

METHODS

Design and Sample

We conducted a prospective cohort study of consecutive patients presenting to a Veterans Affairs medical center in the southeastern United States over a 2.5-year period with symptoms of stroke. All patients were evaluated by an admitting neurologist. We exluded patients with global aphasia, dementia or psychosis, or who were unconscious or otherwise too impaired to sign their own consent (when no proxy was available), or whose strokes had occurred more than 10 days prior to enrollment or were related to other brain disease. During screening for this study, patients were interviewed and screened for aphasia or confusion severe enough to hinder test administration, using the sequential commands and auditory verbal comprehension subtests from the Western Aphasia Battery.[31,32] Stroke was confirmed by diffusion-weighted magnetic resonance imaging (MRI), or in several cases where MRI was precluded or inconclusive, from clinical examination, chart review and computed tomography scan by a neurologist. Recruitment of the TIA sample was similar. Stroke-risk-only patients were recruited from the inpatient general medicine wards over a 2-month period; the screening and chart review process was the same. Both the stroke and risk factor protocols were approved by the medical center's institutional review board and all patients gave informed consent.

Patient Enrollment

Of 325 patients admitted with symptoms suggestive of stroke, 139 were evaluated by the admitting neurologist as having a diagnosis other than stroke. Of the 186 probable stroke patients, 83 were enrolled and 103 were not enrolled for the reasons noted in figure 1. Of the 83 enrolled, 23 patients were subsequently ruled out for stroke, which left 60 with confirmed stroke, and of these, 47 completed at least part of the neuropsychologic battery. Although our original target enrollment was 100 stroke patients, funding and administrative considerations prevented the extension of our enrollment period when recruitment rates fell below our predictions. Fifteen of the patients ruled out for acute stroke had a TIA; those who had no prior stroke became our TIA sample. The other 8 patients,

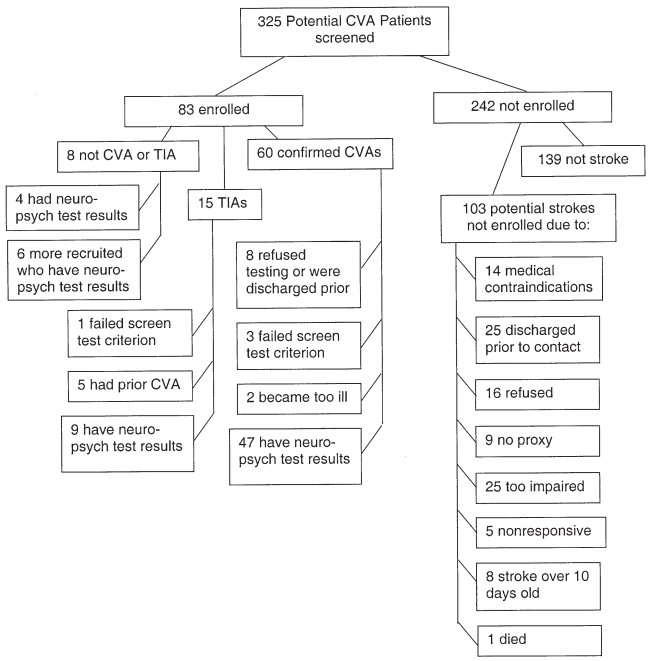

Fig 1. Recruitment flowchart. Abbreviations: CVA, cerebrovascular accident; neuro-psych, neuropsychologic.