



AMERICAN  
PSYCHOLOGICAL  
ASSOCIATION

# Division of Clinical Neuropsychology Newsletter 40

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## President's Message

Colleagues,

I have the privilege of writing to you about the issues that confront our specialty and the important work of the Division and of the APA. These are challenging times, with the economic downturn, the unknowns of health care reform, uncertain access to grant funding, and the increased stress and burden on the communities we serve. It has been a time of re-examining priorities and focusing efforts on the most critical issues for individuals and institutions. In my setting, the institution and my department have been taking stock and re-aligning goals. Similarly, this year, at the August 2009 meeting, the APA Council of Representatives adopted a set of goals and objectives for the first time in the organization's history. These are to: I. Maximize organizational effectiveness; II. Expand psychology's role advancing health; and. III. Increase recognition of psychology as a science. As neuropsychologists, we can certainly embrace these goals which are so consonant with our aspirations for our specialty. In preparing this column, I reviewed some of the goals developed by the Division almost ten years ago by a planning committee convened by then president, Gordon Chelune. This work resulted in a revised mission statement that we continue to endorse and a set of strategic goals for the future. The following goals were developed:

- Streamline the organizational structure and processes of the Division
- Enhance relationships between the Division and APA
- Improve the responsiveness of the Division to practice issues
- Increase public awareness of clinical neuropsychology
- Develop the science agenda of the Division
- Facilitate education about clinical neuropsychology within and outside the profession

I would add two additional goals that we have been advancing: increasing the number of neuropsychologist who represent diversity and integrating issues of diversity and health disparities in training, education, and research; and, advocating for education and training of future neuropsychologists. We have made substantial inroads in these areas and much work continues to be needed. We have benefited from hours of service and commitment from many individuals in the Division and our organizational structure continues to serve our needs. Attaining these goals requires leadership and members actively involved in APA as a whole, as well as in activities specific to clinical neuropsychology. We all need to advocate for the discipline and the profession of psychology. As neuropsychologists, we are uniquely poised to advance public awareness of psychology as a science and our invaluable role in addressing the health care needs of our population across the lifespan.

As a division, we are in a good position with respect to the financial downturn and are lucky to have very prudent management so our resources are used wisely. Nevertheless, continuing to expand and support our membership is critical. We have expanded our role in the APA as a whole, continuing to place neuropsychologists in task forces, committees, and boards to increase our voice within the organization. Just this week, we heard that Paula Shear was elected to the Commission on Recog-

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Past issues of the Division 40 Newsletter and Division 40 Executive Committee meeting minutes are now available online at the Division 40 Website. The URL address is:  
<http://www.div40.org/>.

## From the Editor

One down, a future to go! With your support, we successfully completed our first electronic dissemination of Newsletter 40 with the most recent issue in summer, 2009. To our knowledge, the response from Division 40 members on our transition from paper to the e-newsletter has been overwhelmingly positive. As noted previously, this transition will not only streamline the production of the Newsletter, save significant costs, and make for a more environmentally-friendly product, but also enable us to make further enhancements to the Newsletter's content and appearance in the future. For those who do not use email but have access to a computer, PDF copies of each Newsletter are archived for immediate access, 24/7, at [www.div40.org/newsletter.html](http://www.div40.org/newsletter.html).

I am delighted with our superb lineup of feature articles in the current issue of Newsletter 40, all focused on the topic of neurovascular neuropsychology. We are very fortunate to have contributions from Drs. Joanne Festa, Marykay Pavol, Emily Lantz, and Ronald Lazar, each with a slightly different perspective on the influence of cardiovascular and cerebrovascular disease on brain function, and all relevant to the practicing neuropsychologist in any setting. Once again, the work presented in these three feature articles showcases the role that neuropsychologists play everyday in advancing the science and clinical care for people affected by neurologic disease.

I am afraid this will be my final act as Editor of Newsletter 40. Division 40 is very fortunate to have Dr. Brian Yochim as our new Editor. Dr. Yochim is a board-certified neuropsychologist at the University of Colorado at Colorado Springs. He brings a strong commitment to our field and prior experience editing professional publications like Newsletter 40. I am certain that the Newsletter is in terrific hands under Brian's leadership. My only hope is that, like all those before me, I leave Newsletter 40 in slightly better shape than I found it.

I want to thank you, the Division 40 membership, for all your support of the Newsletter.

Sincerely,  
 Michael McCrea, Ph.D., ABPP-CN  
 Editor, Newsletter 40

## Cognitive Dysfunction in Heart Failure

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### Background

The American Heart Association estimates that approximately five million people in the United States are living with heart failure and more than 50,000 will die each year from the disease. Changes in cognition associated with heart failure were recognized as early as the 1970's when the term "cardiogenic dementia" was used to describe the cognitive effects of heart failure in older patients (Editor, 1977). Using conservative estimates, cognitive dysfunction due to heart failure (HFCD) is now considered to affect at least 1 million HF patients in the United States alone (Roman, 2004). Subsequent studies have shown that among advanced heart failure patients, neurocognitive dysfunction is common and worsens with increasing illness severity (Petrucci, et al., 2006; Trojano, et al., 2003). With the high prevalence of cognitive dysfunction in HF patients, it is important that clinicians be aware of the potential for these problems and the contributing factors in their patients with severe cardiac disease.

### Heart Failure Cognitive Dysfunction

There are many etiologies of heart failure. The most frequent among them are ischemic cardiac disease, non-ischemic cardiomyopathies, myocarditis, valvular disease, and congenital disorders. Often termed congestive heart failure (CHF), this condition involves cardiac output that is insufficient for the body's metabolic needs. Patients with heart failure are also at increased risk of cognitive dysfunction given their histories of prior cardiac surgery and myocardial infarction, medication regimen, co-morbidities of atherosclerotic disease, diabetes, metabolic disturbances, other organ failure, and mood disorders. The neuropsychological literature on HFCD dates back to the 1980's when multidisciplinary heart transplantation candidacy evaluations were being developed. As part of the evaluation team, neuropsychologists performed assessments ranging from cognitive screenings to comprehensive evaluations, and participated in the selection of appropriate transplant recipients. The early heart failure neuropsychological literature consists of clinical database analyses, while several later reports address methodological considerations, include comparison groups and controls, and examine medical predictor variables.

A summary of representative HFCD studies with larger samples is provided in Table 1. In the reported studies, advanced heart failure (AHF) patient samples are comprised predominantly of males being evaluated for transplant candidacy, which is representative of the demographics of heart transplant candidates. Several studies were omitted due to a lack of normative data or statistical comparisons that limit their interpretability. The patient samples examined in these studies typically exclude those with neurological disorders such as Alzheimer's disease as well as those with history of stroke, or major psychiatric disorder. Within the neuropsychological literature on heart failure patients, there is a selection bias that omits the most severe cases because the sickest patients are often too ill to undergo assessment. In addition, some studies employed briefer assessments, creating an analytic bias toward the neuropsychological functions assessed. Finally, several cardiac population studies often cited in the literature that have used cognitive screening tools such as the MMSE (Pulicino, et al., 2008) or the Hodkinson Abbreviated Mental Test (Zuccalà, et al, 1997; Zuccalà, et al., 2001) were omitted due to lack of specificity regarding neuropsychological functions most affected in this setting.

The neuropsychological literature on heart failure indicates that cognitive deficits are found in 30% to 80% of patients ranging from mild to severe, depending on the measures and methodology used (Bennett & Sauve, 2003). One of the abnormalities detected in the early stages of heart failure is in fine motor speed (Petrucci, et al., 2006; Putzke, et al., 2000). Attention and memory dysfunctions are frequently reported, followed by slowed processing speed and executive dysfunction (Bornstein, et al., 1995; Deshields, et al., 1996; Petrucci, et al., 2006; Putzke, et al., 2000; Schall, et al., 1989). These deficits, however, are also the neuropsychological functions that are most frequently assessed in briefer batteries.

Depression is a common comorbidity (Bornstein, et al., 1995; Deshields, et al., 1996) and is quite common in the cardiac patient population. Schall (1989) and colleagues published one of the earliest reports of cognition in end-stage heart failure, finding impairment in memory, information processing speed, and motor speed. In a group of 54 patients with a mean age of 46, analyses showed that the older group of patients, those over age 50 years, were more impaired than younger patients and that heart failure etiology was not predictive of cognition. Using standardized neuropsychological measures to examine the cognitive profile, Schall et al. were among the first to conclude that cognitive dysfunction arising from heart failure was a diffuse neurocognitive disorder.

Putzke's group at the University of Alabama has published several investigations in heart failure cognitive dysfunction using the largest dataset of heart failure patients (Putzke, et al, 1998; Putzke, et al., 2000; Putzke, et al., 1997; Temple, Putzke, & Boll, 2000). Their findings from 760 end-stage heart failure patients showed that up to one-third of their cohort was moderately impaired or worse on the majority of neuropsychological test outcomes. Verbal learning was the most severely

impaired function, along with psychomotor speed, mental speed, and cognitive flexibility. Widespread neurocognitive impairment was demonstrated in a proportion of the sample, with impairment defined as at least two standard deviations below normative means. They found that 35 percent of patients performed in the impaired range on five or more tests. Similar results were found in a case-controlled study using the same neuropsychological measures: performance on tests of psychomotor speed, executive function, as well as motor speed and dexterity was poorer in patients than in controls (Putzke, et al., 2000). In a subsequent analysis of 62 ESHF patients undergoing cardiac catheterization, neuropsychological performance was examined in relation to cardiac function parameters as well as demographic factors, psychiatric and surgical history, medication usage, and depressive and anxious symptoms. Increased hemodynamic pressure variables (pulmonary arterial pressure and right arterial pressure) were most related to declines in cognitive performances for attention, processing speed, and executive function. Surprisingly, decreased cardiac output was significantly related only to Trail Making Part B performance (Putzke, et al, 1998). Another study supported those findings, showing that hemodynamic pressure variables were related to cognitive tests but that cardiac index and cardiac output were not statistically related to neuropsychological functioning (Temple, Putzke, & Boll, 2000).

In a multicenter initiative, the Congestive Heart Failure Italian Study Investigators examined cognitive functioning in elderly heart failure patients (Trojano, et al., 2003). Unlike studies published by United States institutions, the average education level in this Italian sample was quite low, on average less than 6 years, although the sites were selected to be representative of Italy's socio-demographics and patients were excluded for complete illiteracy. The cognitive performance of 308 moderate to severe CHF patients' was compared to 207 cardiovascular disease patients without heart failure on a limited battery evaluating executive functioning, auditory and visual attention and memory. Attention and verbal memory were significantly worse in HF patients when compared to cardiovascular controls. A multivariate analysis included factors with known or suspected effects on cognition including alcohol addiction, coronary artery disease, hypertension, diabetes mellitus, respiratory disease without hypoxemia, atrial fibrillation, several co-morbid diseases, and reported depressive symptoms. Heart failure severity, depressive symptoms, and hypertension were predictive of abnormal performance on more than two of the seven cognitive tests. An abnormal performance on at least three of seven tests was found in 57.9% of severe heart failure patients, 43% of moderate HF patients and in 34% of the cardiovascular control group. Unfortunately, a healthy control group was not included in the analysis.

In a separate, large sample of advanced heart failure patients collected over 18 years, cases were potential candidates for heart transplantation and were classified by heart failure severity into three groups: group 1 was comprised of ESHF outpatients, group 2 was comprised of inpatients admitted for intravenous inotropic therapy, while

group 3 patients were considered unstable and possible recipients of a mechanical cardiac assist device (Petrucci, et al, 2006). In this atypical study, patients with neurological events were included: up to 24 percent of patients had histories that included stroke, TIA, seizure, head trauma, encephalitis and brain tumor. Results demonstrated that dysfunction increased with progressive heart failure severity. Motor functions were affected earlier in the disease, followed by memory and processing speed. Patients being considered for mechanical circulatory devices, those with the most severe heart failure, demonstrated the worst cognitive impairments. Notably, 37% of the potential sample was excluded being too ill to undergo a one-hour neuropsychological evaluation.

Although increased heart failure severity (often determined by the New York Heart Association Class) is frequently associated with poorer cognitive functioning, cardiac function variables such as ejection fraction, cardiac index, and cardiac output are inconsistently predictive of cognition, suggesting that concurrent hemodynamics may not be the sole mechanism of cognitive dysfunction. Depression and anxiety are frequent comorbidities in the heart failure population, present in more than 50% of these patients, (Koenig, 1998) but studies have not adequately or consistently accounted for the effects of these variables on cognitive functioning.

There are additional confounds in interpreting HF cognitive studies caused by the heterogeneity of the subject samples. Patients differ in terms of co-morbidities, age, degree of deterioration due to the nature, severity, and duration of their illnesses. Studies also vary in their levels of statistical control for age, education, depressive symptoms, and multiple comparisons. Older age and ischemic heart disease are particularly important confounds. Frequently occurring HF co-morbidities in the elderly, such as respiratory disorders, renal dysfunction, and depression in addition to polypharmacy have been shown to affect cognitive functioning (Lang & Mancini, 2007). Older patients are more likely to have unrelated neurodegeneration in the context of heart failure that may exaggerate cognitive dysfunction.

Little is known about the reversibility of these cognitive deficits with improved cardiac function post-transplantation. Reports of prospective cognitive assessment in heart-transplant patients have included small samples, ranging in size from 7 to 21 patients (Bornstein, et al., 1995; Deshields, et al., 1996; Roman, et al., 1997; Schall et al, 1989). While several investigators have demonstrated improvements in memory, processing speed, and executive functions, sample selection criteria differed. For example, Roman et al. (1997) selected only the healthiest patients with no neurologic or systemic illnesses and a history of 1 year post transplantation success. Post-transplant assessment intervals also vary across studies, ranging from three months to three years post transplant, further reducing interpretation of the data. Future studies should include consecutive cases, examine covariates and co-morbidities, and integrate neuroimaging to facilitate generalizability to the greater population of heart failure patients and identification of patients at greatest risk for irreversible cognitive dysfunction.

### Mechanisms of HFCD

Mechanisms for cognitive dysfunction in the heart failure population have been proposed and include cerebral hypoperfusion, microembolism (Pullicino & Hart, 2001), APOE e4, (Vogels, et al, 2007), endothelial dysfunction (Cohen, et al., 2009), increased cerebral white matter disease (Paul, et al., 2005), and cerebral atrophy (Paul, et al., 2005; Woo, et al., 2003). The emerging literature suggests factors both reversible and potentially irreversible may be related to HFCD. Siachos et al. (2005) found a 34% prevalence of asymptomatic ischemic brain infarction in a case series of 117 heart transplant candidates that excluded those with obvious risk factors for stroke. Age, gender, hypertension and diabetes were not predictive of strokes in this cohort. In a small study of selected patients with idiopathic, dilated cardiomyopathy and age-matched controls all under age 50 with no neurologic or systemic disease, the 20% rate of cerebral infarcts in patients was significantly higher than the 0% rate in controls (Schmidt, et al., 1991). Similarly, patients were found to have higher rates of cortical atrophy (50% vs 5% in controls) and semi-quantitative volumetric measures of ventricle-to-intracranial cavity ratio indicated significantly greater atrophy in patients. Patients with abnormalities on MRI had worse verbal memory, learning, and reaction time performance than patients and controls with normal imaging. Woo et al. (2003) have also assessed cerebral volume on MRI in patients with heart failure. Comparing nine advanced heart failure patients to 27 healthy controls' regional right-lateralized, gray-matter volume, they found tissue loss in the insula, basal ganglia, parahippocampal gyrus, dorsal midbrain, ventral and superior lateral frontal cortex and bilateral cerebellum. Medial temporal lobe atrophy was found to correlate with memory and executive functioning in heart failure patients but total white-matter hyperintensities were associated only with depressive and anxious symptoms not with cognitive measures (Vogels, et al., 2007). Studies of patients with cardiovascular disease are demonstrating that lower cardiac output is associated with increased cerebral white matter hyperintensities (Jefferson, et al., 2007) and subcortical hyperintensities are associated with executive functioning (Paul, et al., 2005). These initial studies suggest that neuropathological changes play a role in the cognitive functioning of patients with advanced heart failure. Unfortunately, imaging studies exclude a significant proportion of the HF population since those with end stage heart failure who are often implanted with mechanical devices such as pacemakers are not included due to the contraindications posed by these devices.

### Implications

Advanced heart failure patients are medically managed using pharmacological, mechanical, and lifestyle interventions. This multi-pronged approach may be complex, frequently modified, and cognitively demanding. Heart failure is more prevalent among older patients who may also be at greater risk for cognitive impairment. Cognitive dysfunction in heart failure patients can reduce or impair the ability to follow medical recommendations as well as

implement changes necessary for an optimal outcome. For example, research has demonstrated that cognitive functioning is linked to adherence with medical regimens and is an independent predictor of adherence to medications (Rosen, et al., 2003; Stillely, et al., 2004). Medication noncompliance was found to be two times greater in cognitively-impaired elderly subjects in the Rotterdam study (Salas, et al., 2001). The cognitive dysfunction demonstrated in heart failure patients may confer increased morbidity and mortality, independent of other medical complications. Early identification of neurocognitive impairment in heart failure patients can facilitate the timely implementation of compensatory systems to comply with medical protocols. Advances in research exploring the mechanisms of heart failure cognitive dysfunction may help identify patients in whom, with appropriate interventions, impairments are reversible as well as those in whom irreversible dysfunction will require sustained support to achieve an optimal outcome.

### Summary

Heart failure patients are a medically heterogeneous group with varying co-morbidities and often complex medical histories potentially contributing to cognitive dysfunction. Cardiac function variables are not consistently associated with cognition but evidence suggests that decreased cerebral perfusion, asymptomatic ischemic brain infarction, increased cortical atrophy, and increased cerebral white matter hyperintensities may be associated with cognitive dysfunction in heart failure. Medical management of heart failure often includes multiple interventions that can be complex, continually modified, and cognitively demanding. Identifying heart failure cognitive dysfunction can be important to optimal medical management of these patients.

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Table 1. Neuropsychological Functioning in Advanced Heart Failure Patients

Author/ Date	Sample Size Population Mean Age (M)	Neuropsychological Measures		Neuropsychological Results	Additional Results
Schall, et al. 1989	54 AHF pts M=46	WAIS HRNB	WMS-R – LM WMS-R - VR	<ul style="list-style-type: none"> <li>• Impairments in memory, integrative processing speed, and motor speed</li> </ul>	<ul style="list-style-type: none"> <li>• Older patients showed poorer performance</li> <li>• HF etiology was not predictive of cognition</li> <li>• IQ not impaired</li> </ul>
Hecker, et al. 1989	64 AHF pts M=44	WAIS-R - BD WAIS-R - Voc AVLT	WMS CLAT MMPI	<ul style="list-style-type: none"> <li>• Mild dysfunction in sustained attention and short term retention</li> <li>• 26% perform below chance on abstract reasoning</li> </ul>	<ul style="list-style-type: none"> <li>• Average performance on estimated IQ and Memory Quotient</li> </ul>
Bornstein, et al. 1995	62 AHF pts M=45	WAIS-R WMS-R LM WMS -R VR VCAT WCST Trails	COWA Grooved Pegbd HRNB Knox Cube MMPI	<ul style="list-style-type: none"> <li>• Impairments in executive function, attention, and motor speed</li> <li>• 58% of patients had mild impairment on a summary measure of cognitive performance</li> <li>• 37% significant elevation on MMPI depression scale</li> </ul>	<ul style="list-style-type: none"> <li>• Higher cardiac stroke volume index and cardiac index, and lower right atrial pressure were associated with better cognition</li> <li>• HF etiology was not predictive of cognition</li> <li>• Summary cognitive performance associated with depression</li> </ul>
Deshields, et al. 1996	191 AHF pts M=47	WAIS-R – BD WAIS-R – Voc WMS-R LM WMS -R VR Trails	BDI BAI SCL-90	<ul style="list-style-type: none"> <li>• Impairment in verbal memory</li> <li>• 56% with mild to moderate depression</li> <li>• 52% with mild to mod anxiety</li> <li>• 9% with severe anxiety</li> </ul>	<ul style="list-style-type: none"> <li>• Anxiety and depression on BDI and BAI driven by physical symptoms</li> <li>• SCL-90 did not reveal depression or anxiety</li> </ul>
Putzke, et al. 1997	760 AHF pts M=49	SILS Digit span BNT COWA Grooved Pegboard Trails	SDMT Stroop WMS - LM WMS -VR WMS - PA SRT	<ul style="list-style-type: none"> <li>• 25-33% had moderate to severe impairment on 11 of 19 (58%) of neuropsychological tests</li> <li>• Worst performance was on verbal memory, psychomotor speed, mental speed, and executive functions.</li> <li>• 77% of pts impaired <math>\geq 1</math> tests</li> <li>• 35% of patients impaired <math>\geq 5</math> tests</li> </ul>	<ul style="list-style-type: none"> <li>• Better performance associated with younger age and higher education</li> </ul>
Putzke, et al. 1998	62 AHF pts having heart cath. M=50	SILS Digit span BNT COWA Grooved Pegbd Trails	SDMT Stroop WMS-LM WMS-VR WMS-PA SRT	<ul style="list-style-type: none"> <li>• Highest proportion of patients impaired on naming, verbal learning, speeded dexterity, verbal memory, processing speed, and executive function.</li> <li>• 34% of pts impaired on <math>\geq 5</math> tests.</li> <li>• 20-30% of patients had mod - severe impairment on 11 of 19 tests</li> <li>• 24% of pts had no impairments</li> </ul>	<ul style="list-style-type: none"> <li>• Right arterial mean pressure and pulmonary arterial diastolic pressure were associated with executive function and processing speed.</li> <li>• EF not related to cognition</li> </ul>

Table 1. continued

Putzke, et al. 2000	44 AHF pts M=49 44 matched cntrls M=45	SILS-AB SILS-Voc Grooved Pegbd Trails	WMS-LM CATS WRAT reading	<ul style="list-style-type: none"> <li>Compared to controls HF pts had slower speeded dexterity, psychomotor speed, and executive function.</li> <li>Pt scores were all in normal range</li> </ul>	<ul style="list-style-type: none"> <li>Cardiac function variables were not associated with cognitive performance.</li> </ul>
Callegari, et al. 2002	Age <65 years 64 AHF pts M=52	WAIS STNB Word list recall Story recall Cancellation test	Verbal Span Visual Span Verbal Logic Raven's Matrices Token Test	<ul style="list-style-type: none"> <li>Impairments in attention, memory and visual reasoning.</li> <li>9% - no impairment</li> <li>26% impaired on 1 function</li> <li>30% impaired on ≥ 4 functions</li> </ul>	<ul style="list-style-type: none"> <li>Cardiac function variables were not associated with cognitive performance.</li> </ul>
Trojano, et al. 2003	Elderly ≥ 65 years 149 Moderate HF M=75 159 Severe HF M=76 207 Cardiac cntrls M=74	Digit Cancel. Raven's Matrices Verbal fluency	Corsi blocks Verbal word span RAVLT	<ul style="list-style-type: none"> <li>Compared to controls, severe HF pts had worse attention and verbal memory.</li> <li>Subjects with &gt;2 tests impaired 58% severe HF group 43% moderate HF group 34% controls</li> </ul>	<ul style="list-style-type: none"> <li>All subjects &lt; 6 years education</li> <li>HF severity, depression and hypertension predicted cognitive impairment</li> </ul>
Petrucci, et al. 2006	252 AHF pts 113 Outpts M=46 83 Inpts M=50 56 MCAD Inpts M=52	WMS - LM WMS - VR BVRT RAVLT	Finger tapping Hand Dyn. Trails	<ul style="list-style-type: none"> <li>Impairments in memory, motor function, and processing speed.</li> <li>Earliest abnormality is motor</li> <li>Most impacted functions are processing speed and memory</li> </ul>	<ul style="list-style-type: none"> <li>Selection bias: the sickest 37% of potential sample could not withstand assessment</li> <li>Most severe impairments in sickest patients</li> </ul>
Vogels, et al. 2007	62 HF pts M=69 53 Cardiac cntrls M=69 42 Healthy cntrls M=67	RAVLT Digit span CANTAB	Trails Verbal fluency Stroop	<ul style="list-style-type: none"> <li>Compared to healthy controls HF pts performed worse in memory, executive, visuospatial, and processing speed functions.</li> <li>General cognitive impairment was found in 25% of HF patients and 4% of healthy controls</li> </ul>	<ul style="list-style-type: none"> <li>IQ, HF severity, ApoE e4 predicted cognitive impairment.</li> </ul>
Hoth, et al. 2008	Pts >55 years old 31 AHF pts Moderate- severe M=69 31 Cardiac cntrls M=69	RBANS Trails COWA	WAIS-III LN Stroop	<ul style="list-style-type: none"> <li>Normative data comparisons showed no impairments in pts</li> <li>Compared to controls, worse executive functioning and psychomotor speed</li> </ul>	<ul style="list-style-type: none"> <li>Lower EF associated with weaker global cognition and executive function</li> <li>Lower cardiac index associated with lower immediate recall</li> </ul>

AHF= advanced heart failure; Pts=patients; WAIS=Wechsler Adult Intelligence Test; WMS-R=Wechsler Memory Scale-Revised, LM=Logical Memory, VR=Visual Reproduction, PA=Paired Associates; HRNB=Halstead-Reitan Neuropsychological Battery; WAIS-R=Wechsler Adult Intelligence Test Revised, BD=Block Design, Voc=Vocabulary, LN=Letter Number Sequencing; AVLT=Auditory Verbal learning Test; CLAT= Conceptual Levels Analogies Test; VCAT=Verbal Concept Attainment Test; WCST=Wisconsin Card Sorting Test; Trails= Trail Making Tests; COWA=Controlled Oral Word Association Test; BDI= Beck Depression Inventory; BAI=Beck Anxiety Inventory; SCL-90=Symptom Checklist 90; SILS=ShIPLEY Institute of Living Scale, AB=Abstract Reasoning, Voc=Vocabulary; BNT=Boston Naming Test; SDMT=Symbol Digit Modalities Test; SRT=Selective Reminding Test; CATS=Short Categories Test; WRAT=Wide Range Achievement Tests; STNB=Spinnler and Tognoni Neuropsychological Battery; RAVLT=Rey Auditory Verbal Learning Test; BVRT=Benton Visual Retention Test; CANTAB=Cambridge Neuropsychological Test Automated Battery; RBANS=Repeatable Battery for the Assessment of Neuropsychological Status.

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**President's Message**

(continued from page 1)

In addition to the election of Specialties and Proficiencies in Professional Psychology, Adam Brickman was chosen for the highly selective and active APA Committee on Aging, Chris Loftus will be serving on the Policy and Planning Board, Sid O'Bryant was elected to the APA Committee on Rural Health, Dan Marson and Mieke Verfaellie have been appointed to the the APA Committee on Human Research, and Marsha Nortz Gragert will serve as liaison to the APA Presidential Task Force on Caregivers. Congratulations to them and all those who are serving in other positions and those who have paved the way for neuropsychology to be more involved.

Advocating for practice has been a strong focus of the Division. We have supported efforts to advocate against state laws restricting the use of technicians in neuropsychology practice. Jennifer Manly and Glenn Smith represented clinical neuropsychology at the Presidential Summit on the Future of Psychology Practice. You can view presentations from the Summit on the APA website. The issues raised at the meeting are similar to what we are hearing in higher education and health care reform: we need to focus on interdisciplinary collaboration, accountability, and effectiveness (outcomes), while serving an increasingly diverse population. Our next steps in clinical neuropsychology will need to focus on enhancing structures for integrated, interdisciplinary, primary care service delivery and evidence based care.

In science and education, we continue to foster our students and our early career neuropsychologists. We now have a science directory on our website to enhance communication and advocacy for science. In our training we need to focus on developing students' skills in advocating for science and the role of research in addressing public needs. Similarly, the focus in science is turning to collaborative, interdisciplinary, translational research and our students need to be poised to work effectively in this environment. Neuropsychologists should contribute and build on efforts to enhance funding for training, such as Graduate Psychology Education.

It is clear that in these times, we all need to take on intentional, strategic roles in advocating for neuropsychology at the local, national, and international level. In our specialty, a schism between science and practice makes no sense. Clinical neuropsychologists are evidence based clinicians, actively integrating science in care. We need to bring the strong science foundation of our specialty not only as core members of interdisciplinary research collaborations, but also as a critical presence on health care and public policy teams. I look forward to working with you on these vital initiatives.

Sincerely,  
 Celiane Rey-Casserly, PhD  
 President, Division 40

### **Division 40 Education Advisory Committee (EAC)** Cindy Cimino, Ph.D., Chair

The Education Advisory Committee is composed of eight core continuing members as well as rotating representatives from six training/professional organizations in neuropsychology spanning the doctoral, internship and postdoctoral level. These training organizations include ADECN (Association for Doctoral Education in Clinical Neuropsychology), AITCN (Association for Internship Training in Clinical Neuropsychology), APPCN (Association of Postdoctoral Programs in Clinical Neuropsychology), ANST (Association of Neuropsychology Students in Training), ECP (Early Career Psychologist-Neuropsychology), CNS (Clinical Neuropsychology Synarchy).

Continuing Members include Rus Bauer, Cindy Cimino (Chair), Maureen Lacy, Joel Morgan, Neil Pliskin, Tony Stringer, Desiree White, John Woodard

Organizational Representatives: Catherine Mateer (ADECN), Beth Slomine (AITCN), Jacobus Donders (APPCN), John Strang (ANST), Deborah Weber (ECP), Celiane Rey-Casserly (CNS).

Web Listing of Training Programs Be on the lookout for changes coming to the Division 40 website listing of training programs in neuropsychology. We are currently working with the computer programmer who initially developed the site to effect some changes to the website within the first two months of 2010. These changes include 1) automatic renewal notices sent to programs annually to keep information on the site as up to date as possible 2) removal of sites that remain outdated after repeated notice 3) greater uniformity in site listings to more easily identify type, location and focus (i.e. adult, pediatric, combined) of the training program. The committee is also currently reviewing a more substantial overhaul to the website to take place over the next year with a target date of completion in January 2111. Changes in the type of information displayed and the search capabilities of the site are among many of the changes being considered.

Frequently Asked Questions To address the needs of trainees, the EAC is developing a FAQ section on the EAC portion of the Division 40 webpage. The purpose is to address some of the many questions that arise as individuals pursue their training goals and wade through the decision making process. ANST Chair, John Strang, provided questions from a brief survey of ANST members for the proposed FAQ section of the website. EAC members were also asked to survey doctoral, internship and post-doctoral trainees for additional questions to add to FAQ listing.

### **Division 40 Early Career Psychologist Committee** Deborah Weber Loftis, Ph.D.

Clinical neuropsychology is a rapidly expanding profession that today it is one of the largest specialty groups within psychology. In fact, ECP membership in Division 40 continues to grow despite overall declines in APA membership. After a low of 387 ECP members in 2006, we now have 599 ECP members in Division 40. This is the highest number of ECP in any Division in APA followed by Division 14 (459) and Division 38 (337).

One of the major goals of the ECP committee is to provide an ECP focused forum for professionals to come together to discuss issues relevant to ECPs in neuropsychology. Another area of focus is to provide informational resources and services for ECPs through conference programs. Lastly, the committee hopes to promote leadership development by mentoring and supporting ECPs in their professional activities and assisting them in advancing upward in the governance structure in order to communicate and advocate for the concerns of early career psychologists within Division 40 and APA. If you are interested in joining a committee please send a letter of interest and CV to [deb.loftis@yahoo.com](mailto:deb.loftis@yahoo.com) We look forward to hearing from you!

### **Early Career Psychologist (ECP) Survey Results**

The ECP Committee of Division 40 recently conducted a survey of ECP's regarding their experiences in obtaining training in neuropsychology, as well as establishing their professional practice. Sixty early career neuropsychologists completed the survey. Almost 2/3 of respondents had completed a two-year postdoctoral program, despite changes in Houston Guidelines that were captured based upon the graduation dates of our respondents. Significant challenges in postdoctoral training were noted in finding an accredited postdoctoral program or appropriate supervisor, in geographic or family limitations, as well as financial constraints. Following training, respondents indicated that financial pressures and board certification were significant challenges. Financially, ECP's seek guidance in clarification and resolution of billing issues with Medicare and insurance companies, as well as business strategies for beginning and running a private practice. Many ECP's reported challenges in establishing themselves within a professional or medical community, marketing their skills, and establishing a supportive and collegial consultation network. ECP's also expressed concern regarding the time and expense of board certification and the cost/benefit analysis of this step in professional development. Specifically, significant concerns were also raised regarding the protection of the term "neuropsychologist" given the additional time and financial constraints that formal training requires and the frequent use of the term by unqualified individuals. Overall, ECP's expressed a desire for additional networking and mentorship opportunities, financial and billing workshops, and seminars aimed at work and family balance following their training.

If you would like more information about the ECP Survey results, please contact Deborah Weber Loftis, Ph.D at [deb.loftis@yahoo.com](mailto:deb.loftis@yahoo.com)

### **Division 40 Members Elected to APA Committees**

We are proud to announce that Division 40 had a strong showing in members recently elected to APA committees. Please join us in congratulating the following members who were elected to these APA committees:

- Jennifer Manly** - Committee on the Future of Psychological Science
- Paula Shear** - Commission for the Recognition of Specialties and Proficiencies in Professional Psychology
- Sid O'Bryant** - Committee on Rural Health
- Adam Brickman** - Committee on Aging
- Chris Loftis** - Policy and Planning Board
- Dan Marson** - Committee on Human Research
- Mieke Verfaillie** - Committee on Human Research

Please accept our apologies if we omitted the success of other members! And, of course, thank you to everyone who ran for various offices.

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### **Division 40 Gains Additional Seat on APA Council of Representatives**

Dear Division 40 Members,

I am very pleased to share good news with you! We learned last month that due to your efforts and willingness to "Give all 10 to 40" on apportionment ballots, our division gained a fifth seat on the APA Council of Representatives. A reminder: the APA Council of Representatives has full power and authority over the affairs and funds of APA, and has the power to review the action of any of the boards and committees.

As a current Div 40 Council Rep, I am proud of the efforts of all four of our current reps. For example, we participate in the smaller Caucus meetings (such as the Coalition for Academic and Scientific and Applied Psychology, the Women's Caucus, and the Caucus on Ethnic Minority Affairs) which help to set the Council agenda and foster issue-based allegiances across council reps from Divisions and States/Provinces/Territories. Each representative gets a vote on the many issues that come before council, such as establishing the APA Mission and Strategic Plan, approving the budget, whether to establish new Divisions, or approve specialties within psychology.

Although the four of us have been active within Council, the addition of a fifth member will strengthen our influence and representation. Every vote counts; in my 3 years I have

been witness to several very close votes on the floor of Council. Every voice counts on Council, as each of us has the ability to speak on the floor, propose new action items, and to run for elected office within the Caucuses. Furthermore, Council votes on new members for several key spots on APA Boards and Committees, such as the Board of Scientific Affairs, the Commission for the Recognition of Specialties and Proficiencies in Professional Psychology, and the Committee for the Advancement of Professional Practice. Therefore, this additional Council seat represents not only more comprehensive representation of Division 40 on Council, but will have a positive rippling effect throughout the organization. As one of the largest Divisions in APA, it is time that our representation in the governance of the organization fully reflect our division's needs. Thanks to all of you for making that happen by focusing on the importance of the apportionment vote in the fall.

Now that we have gained another Council Representative, it will soon be time for the membership to vote on candidates for that seat. Candidates should be familiar with the expectations and commitments of the office. If any Division 40 member is interested in becoming a candidate for the Council of Representatives, please feel free to contact me at [jjm71@columbia.edu](mailto:jjm71@columbia.edu).

## Neuropsychology of Ischemic Stroke

Marykay Pavol, Ph.D.  
Assistant Professor of Clinical Neuropsychology  
Neurological Institute  
Columbia University Medical Center

A stroke is defined as a sudden, nonconvulsive, focal neurological deficit (Adams & Victor, 1993). Current definitions of stroke refer to ischemic (interruptions of blood flow) or hemorrhagic (bleeding) events involving the blood supply of the nervous system. Ischemic strokes make up the majority of new strokes, comprising 88% (Festa, Lazar & Marshall, 2008). Ischemic events may be described as “embolic” (usually from a cardiac source) or “atherothrombotic” when there is no clear source for emboli (Wolf, 2004). Ischemic stroke syndromes tend to be fairly consistent across patients as a result of similarities in the localization of behaviors as well as similarities in vascular distributions, although individual variability does occur (Kirshner & Mark, 2009). In his oft-quoted remark, C.M. Fisher observed that we learn neurology “stroke by stroke”; the focal ischemic lesion has led to some of the most important discoveries about the brain (Adams & Victor, 1993).

### Historical Background

Descriptions of stroke events date back thousands of years. Hippocrates (460-370 B.C.) referred to attacks of numbness and anesthesia as signs of “impending apoplexy”; apoplexy in Greek means “struck with violence” (Thompson, 1996). The conceptualization of the disease processes of strokes has evolved since Hippocrates’ time. The Greeks believed strokes were due to dry, cool, or phlegmatic disorders of the brain (Benton, 2000). Galen (A.D. 130-200) also believed that strokes were due to excessive phlegm. It was not until the anatomist Johann Jakob Wepfer’s 1658 paper that the association between stroke and cerebrovascular disease received strong support. This was followed by centuries of debates about whether the brain was made up of discrete areas responsible for specific abilities (localization), as argued by Franz Josef Gall (1758-1828) and Paul Broca (1824-1880) or whether all areas of the brain were equally involved in a behavior (equipotentiality), as argued by Rene DeCartes (1596-1650), Pierre Flourens (1794-1867), and Karl Lashley (1890-1958) (Kolb & Whishaw, 1990).

This debate leads to our current topic, the neuropsychology of ischemic stroke. Gall was a phrenologist and, as such, was a vocal proponent of localization. Jean Baptiste Bouillaud (1796-1881) presented a paper to the Royal Academy of France on Feb. 21 1825 which supported Gall’s theory that speech was governed by the frontal lobes. Bouillaud presented evidence from clinical studies that language was localized in the neocortex, specifically in the frontal lobes. In 1836, Marc Dax presented a series of cases which demonstrated that speech disorders were associated with lesions to the left hemisphere; this manuscript was published by his son in 1865. In 1861, Bouillaud’s son-in-law, Ernest Auburtin, presented a case to the Anthropological Society of Paris in which he reported the case of a patient who ceased speaking when pressure was applied to his anterior lobes. He also reported a patient who lost the ability to speak but who understood everything—Auburtin predicted that this patient’s lesion (“softening”) was in the anterior lobes. Paul Broca (1824-1880) was at this meeting and heard Auburtin’s assertion. Five days later Broca encountered a patient, Leborgne, who was virtually unable to speak and could only say “tan” (and recite an oath). Broca invited Auburtin to see Leborgne and they both predicted a frontal lesion. When Leborgne died later that year, Broca reported that the left anterior lobe was the site of Leborgne’s lesion. By 1863, Broca had collected eight more cases of patients with language disturbance who had lesions in the posterior third of the third frontal convolution. Although Broca has been criticized for not giving Dax sufficient credit, Broca was said to have acknowledged Dax’s work. Two important points in neuropsychology can be concluded from this series of events: 1) a behavior (language) is governed by a specific location; and 2) damage to this area will impair the behavior (Kolb & Whishaw, 1990). But the theory of localization had not yet been universally accepted. Carl Wernicke (1848-1904) and his colleague Theodore Meynert (1833-1892) reported evidence of important language areas more posterior in the brain, specifically the first temporal gyrus of the left hemisphere, now known as Wernicke’s area (Kolb & Whishaw, 1990). At the time, this was viewed as a challenge to the localization of function theory argued by Broca. In addition to describing this “fluent” aphasia, Wernicke also suggested a model of language in the left hemisphere which involved communication between the two distinct language areas, a model which continues to be influential.

Discoveries regarding the relationship between strokes and behavior were not, of course, limited to aphasia, as has been researched and described in detail by Benton (2000). Prosopagnosia (inability to recognize familiar faces) and achromatopsia (loss of color vision) were first described in 1867 by Italian ophthalmologists Antonio Quaglino and Giambattista Borelli. In 1892, H. Wilbrand reported a patient with prosopagnosia who had bilateral inferior occipitotemporal infarction. In 1892, J. Dejerine published his case of pure alexia associated with infarction in the left mesial occipital area and splenium of the corpus callosum. Foix and Hillemand (1925) provided detailed descriptions of disturbances associated with infarctions in the territory

of the anterior cerebral artery, including monoplegia (weakness of only the lower contralateral limb) and unilateral ideomotor apraxia of the left hand. Davidson (1934) conducted a study of 48 patients with single vascular lesions, confirming many previous findings regarding the effects of stroke on behavior. He concluded “cerebral localization on the basis of vascular supply, although difficult and necessitating caution in interpretation, can still be definitely established.” The remainder of this paper will present an overview of the various deficits associated with ischemic stroke, often influenced by the studies described above. Although not comprehensive, this review will cover many of the common stroke outcomes.

### **Ischemic Stroke Syndromes**

#### *Anterior cerebral artery (ACA) infarction:*

The anterior cerebral arteries originate at the anterior portion of the circle of Willis. The ACA perfuses the superior frontal gyrus, cingulate gyrus, and the premotor, motor, and sensory areas of the paracentral lobule (Lazar & Festa, 2009). Branches of the ACA also supply portions of the caudate, internal capsule, globus pallidus, olfactory regions and hypothalamus. The anterior cerebral arteries are joined by the anterior communicating artery. Infarction in the region of the ACA often results in impaired strength (monoplegia) and/or sensation of the contralateral lower extremity (Blumenfeld, 2001). The eyes may deviate toward the injured hemisphere (Miller, Benson & Johnson, 2003). Neglect may also be apparent in damage to the right frontal region and may be distinguished from parietal lobe damage by the finding of motor akinesia rather than visual or sensory inattention. Significant personality change or apathy may result and, if the damage is bilateral, the patient may appear akinetic and mute. Left arm apraxia may be found if the corpus callosum is affected (Bigler & Clement). Bird et al. (2004) studied a patient with a rare instance of ischemic damage to the mesial frontal lobes bilaterally; their patient showed impairments in planning and memory as well as a tendency to confabulate.

The anterior communicating artery (ACoA) is the most frequent site of aneurysms and thus the subject of a large body of research on the effects of aneurysm bleed and surgery (Vates et al., 2004). The cognitive profile associated with ACoA aneurysm bleed is the subject of some debate and, in a literal sense, not directly relevant to a discussion of ischemic stroke. Aneurysm bleeds are typically treated with surgery (clipping or coiling). The common impairments include memory (related to basal forebrain or septal region damage), reasoning, initiation and, at times, impulse control (Cullum et al., 2008; Pavol & Rastogi, in press). Abe et al. (1998) described a patient with a discrete lesion in the basal forebrain who demonstrated anterograde amnesia, possibly due to disconnection between the diagonal band of Broca and the hippocampus bilaterally. Confabulation, akinesia and alien hand syndrome have also been reported (DeLuca & Diamond, 1995). Many studies do not, however, support the existence of a particular cognitive profile associated with ACoA aneurysm bleeds and there is evidence that outcome in these

patients is related to surgery type, complications, and degree of basal forebrain involvement (Bauer, 2008; DeLuca & Prestigiacomo, 2009; Pavol & Rastogi, in press).

#### *Middle Cerebral Artery (MCA) infarction:*

The middle cerebral arteries are the largest branches of the internal carotid arteries and perfuse the lateral portions of the hemispheres (Lazar & Festa, 2009). The MCA is the most commonly affected artery in stroke and embolism is the most frequent source of ischemia (Mohr et al., 2004). Infarction involving the MCA can result in a wide range of impairments and the presentation often differs dramatically based on the laterality of the lesion. The MCA stroke may produce contralateral paresis and/or sensory disturbance with the face and upper extremity affected more than the lower extremities (Blumenfeld, 2001; Mohr et al., 2004). There may be a contralateral visual field deficit (homonymous hemianopia). With damage to the left MCA, language disturbance is common and may present as Broca’s, Wernicke’s, transcortical motor, transcortical sensory, conduction or global aphasia (Festa, Lazar & Marshall, 2008; Kirshner & Mark, 2009). The nature of the language disturbance will depend on the location of the ischemia (anterior vs. posterior). Apraxia and agnosia may also be apparent and are commonly associated with left parietal damage. Right neglect is less commonly noted but has been reported in left MCA infarctions (Kleinman et al., 2007). Although visualconstruction deficits are more typically associated with right-sided lesions, damage to the left hemisphere may impair drawing ability (Kirk & Kertesz, 1989; Lezak, 1995). These patients often show impairment in the drawing of details but are better with producing the outlines and proportions (Festa, Lazar & Marshall, 2008). Gerstmann syndrome (agraphia, right-left confusion, acalculia, and finger agnosia) may also occur with damage to the left parietal region (Kirshner & Mark, 2009). With damage to the right MCA, patients may show impairments in visual construction, with greater impairments in the creation of the gestalt and better preservation of details. Left neglect is common and may show rapid improvement (Bartolomeo, 2007; Heilman, Watson & Valenstein, 2003). The neglect may result from injury in either the posterior or anterior regions of the right hemisphere (Festa, Lazar & Marshall, 2008). Anosognosia, disturbances in prosody and what appear to be hallucinations or delusions may appear (Kirshner & Mark, 2009; Mohr et al., 2004; Ross, 2003). Patients may also show impairments in the ability to perceive or express emotional information, take turns in conversations, or other pragmatic communication skills (Cummings, 2007; Gainiotti, 2003; Lindell, 2006).

#### *Posterior Cerebral Artery (PCA) infarction:*

The posterior cerebral artery comes from the basilar artery (which connects to the vertebral arteries) (Lazar & Festa, 2009). The PCA perfuses much of the occipital lobes and (importantly for memory) the lower/mesial surfaces of the temporal lobes, which include the hippocampus. Disturbances in vision are common with PCA infarction, including contralateral hemianopia (Binder & Mohr, 2004; Blumenfeld,

2001). Left PCA strokes may cause reductions in verbal memory, alexia without agraphia, and color naming deficits (Kirshner & Mark, 2009). Right PCA strokes may affect nonverbal memory. Bilateral PCA strokes may result in visual agnosia, disturbed ability to recognize familiar faces (prosopagnosia) or perceive colors (achromatopsia) (Farah & Feinberg, 2003; Kirshner & Mark, 2009; Tranel, 2003). Bilateral PCA stroke has also been implicated in cases of severe memory deficits, apparently from damage to the hippocampus or related structures (Binder & Mohr, 2004; Zola-Morgan, Squire & Amaral, 1986). The memory impairments are characterized by deficient encoding and do not benefit from cueing or recognition formats. Bilateral involvement may also produce cortical blindness or impair the ability to synthesize visual information, as is seen in Balint's syndrome (optic ataxia, optic apraxia, simultagnosia) (Coslett & Chatterjee, 2003).

### *Thalamic Infarction:*

The thalamus is located near the midline of the each cerebral hemisphere and is separated from the basal ganglia structures by the internal capsule. Each thalamus is comprised of many nuclei and is served by several small branches of the posterior cerebral artery (e.g., geniculothalamic artery, posterior choroidal artery, tuberothalamic artery) and internal carotid artery (e.g., anterior choroidal artery) (Crosson, 1992; Lazar & Festa, 2009). Aphasia (hypophonia, paraphasias, impaired comprehension) has been associated with infarction of the left anterior thalamus (Kirshner & Mark, 2009). Speech may be nonfluent with poor comprehension but relatively preserved repetition (Crosson, 1992). Hemianopia and hemisensory impairment have been associated with the lateral geniculate and ventroposterolateral regions, respectively. Deficits in memory and attention have been attributed to paramedian thalamic infarction. Right thalamic stroke has been associated with left neglect and anosognosia (Karussis, Leker & Abramsky, 2000). Bilateral thalamic stroke may result in impairments in arousal, initiation, executive function, and memory (Kirshner & Mark, 2009; Mori, 2002). The memory impairment associated with thalamic stroke may be related to the connections between the midline thalamic nuclei and the limbic system (Crosson, 1992) or disruptions in the mammillothalamic tract (Bauer, 2008). The executive impairment is likely related to disruptions in the circuitry connecting the thalamus and frontal lobes (specifically, the dorsolateral prefrontal circuit and the lateral orbitofrontal circuit) (Tekin & Cummings, 2002).

### *Basal Ganglia Infarction:*

The basal ganglia include the caudate, putamen, and globus pallidus. The structures are supplied by branches off of the ACA (e.g., Heubner's artery, medial striate artery) and MCA (e.g., lateral striate artery) (Crosson, 1992; Lazar & Festa, 2009). Infarction of the basal ganglia has been associated with executive dysfunction and, following left-sided lesions, language disturbance similar to Broca's aphasia (but often with better comprehension and repetition) (Crosson, 1992; Kirshner & Mark, 2009; Mori, 2002). Hypophonia,

delayed speech initiation, and writing impairments have also been reported. Basal ganglia lesions have been associated with poor learning curves on memory tasks (Crosson, 1992). Infarction specific to the caudate has been associated with abulia, executive dysfunction, aphasia (with left lesions), and neglect (with right lesions) (Caplan et al., 1990; Kumral, Evyapan, & Balkir, 1999; Kumral, Evyapan, & Balkir, 1999). As with the thalamus, the impairments in executive function in basal ganglia infarction appears to result from disturbances in frontal-subcortical pathways (Tekin & Cummings, 2002).

### *Cerebellar infarction:*

In recent decades, the role of the cerebellum in cognition and behavior has been an increasing focus of study. The role of the cerebellum in the production of smooth, coordinated movement has long been appreciated. Recent research has established a complex network of connections between the cerebellum and the frontal, parietal, temporal and occipital lobes. These connections are responsible for the role of the cerebellum in a wide range of non-motor activities including attention, executive control, language, working memory, learning, pain, emotion, and addiction (Glickstein & Doron, 2008; Strick, Dum & Fiez, 2009).

## **Discussion**

The information provided in this review is based on study of the natural history of patients with ischemic stroke. Strokes have often been observed to be "experiments of nature". The approach of studying a patient's clinical presentation and correlating the findings with neuropathology (via autopsy or imaging) has proved invaluable in our appreciation of structure-function relationships. This approach, however, may be affected by developments in stroke treatment. Significant treatment advances have recently been made, namely the availability of tissue plasminogen activator (tPA). tPA is a clot-dissolving drug that is used to restore blood flow to the brain following an ischemic stroke; it is highly effective in reducing stroke deficits but carries the risk of hemorrhage (Davalos, 2005; Wardlaw et al., 2009). Guidelines had initially recommended that tPA should be administered within 3 hours of stroke onset but the time frame has recently been extended to 4.5 hours for some patients (Hacke et al., 2008). Nonetheless, many patients do not arrive in time for treatment and some physicians are wary of the medicolegal risks (Bambauer et al., 2006; Weintraub, 2006). It remains to be seen how the use of this "clot busting" treatment will affect the study of the relationship between ischemic stroke and cognition.

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### APA Convention News

The APA 118th Annual Convention will be held in San Diego August 12th – 15th, 2010. Invited speakers for the Division 40 program will include: Charles Hoge (mild TBI), Vilayanur Ramachandran (neurology of human nature), Keith Yeates (TBI in children), Helena Chui (vascular cognitive impairment), and many others! Mark your calendar and plan to attend!

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### Women In Neuropsychology (WIN) The WINner's Box

Chris Morrison, Ph.D., ABPP  
Women in Neuropsychology Committee Chair

WIN would like to honor Dr. Kyle Brauer Boone in this issue's "WINner's Box." Dr. Boone earned her doctoral degree from the California School of Professional Psychology and was a postdoctoral fellow at the Neuropsychiatric Institute and Hospital at the University of California Los Angeles. She is currently Clinical Professor in the Department of Psychiatry and Biobehavioral Sciences at the David Geffen School of Medicine at University of California, Los Angeles and is also a Professor in the California School of Forensic Studies at Alliant University.

She is an ABPP/ABCN Diplomate and a fellow of both the Division 40 of the American Psychological Association and the National Academy of Neuropsychology. In 2004, she was voted "Psychologist of the Year" by the Psychological Services Development Committee, Department of Mental Health, Los Angeles County.

Dr. Boone has contributed to neuropsychology in many ways. She is on the board of directors for the American Academy of Clinical Neuropsychology and recently served as one of only two female members of the advisory committee for the first AACN Consensus Conference on the assessment of malingering and response bias in neuropsychology. She serves on the editorial boards of /The Clinical Neuropsychologist, Journal of the International Neuropsychological Society, Journal of Clinical and Experimental Neuropsychology, Psychological Assessment, and Archives of Clinical Neuropsychology, and is on the University of Minnesota Press MMPI-2/MMPI-2-RF Advisory Board.

Dr. Boone is also an accomplished researcher, with well over 100 peer-reviewed publications to her name. She is coauthor of two books, including both editions of /Handbook of Normative Data for Neuropsychological Assessment/ (Oxford, 1999 and 2005) and /Neuropsychology Casebook / (Springer, 1988) and recently edited the volume /Assessment of Feigned Cognitive Impairment/ (Guilford, 2007). In addition, she has published two measures of noncredible responding, the b Test and the Dot Counting Test. While she may be best known for her work in neuropsychological assessment of noncredible responding, she has contributed to scientific knowledge in multiple areas of neuropsychological assessment.

Dr. Boone is also a mother of 19-year-old boy/girl twins in their first year of college, and has been married for 28 years to another Dr. Boone (Rodney Boone), also a psychologist. Dr. Boone serves as a deeply admired role model for her neuropsychological colleagues.

By Julie Suhr, Ph.D.

**If you would like to highlight your work or that of a colleague, please make your submission to Chris Morrison, Ph.D., ABPP at [chris.morrison@nyumc.org](mailto:chris.morrison@nyumc.org)**

## Cerebrovascular Anomalies: Brain AVMs and Cerebral Aneurysms&

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### BRAIN ARTERIOVENOUS MALFORMATIONS Pathophysiology and Epidemiology

Arteriovenous malformations of the brain are lesions composed of coiled batches of blood vessels which can be located in any part of the brain (cortical, sub-cortical, dural, or brain stem) and are defined primarily by abnormal hemodynamics. They are made up of a complex tangle of abnormal veins and arteries ( The Arteriovenous Malformation Study Group, 1999; Stapf et al., 2001) with a missing capillary bed, and instead are connected by fistulas and characterized by shunting of blood from artery to vein (Bambakidis et al., 2001; Klimo, Rao, & Brockmeyer, 2006; O'Brien, Neyastani, Buckley, Chang, & Legiehn, 2006). This shunting mechanism causes hypertension within the AVM and in the draining vein (Group, 1999; Iwama, Hayashida, Takahashi, Nagata, & Hashimoto, 2002; Loring, 1999) and hypotension in the surrounding and feeding vessels (Group, 1999), with little apparent clinical effect (Diehl, Henkes, Nahser, Kuhne, & Berlit, 1994; Fogarty-Mack et al., 1996; Mast et al., 1995; Murphy, 1954). Unlike normal blood vessels in the brain, it has been suggested that blood flow through the AVM is not autoregulated, and thus it is impacted by fluctuations in blood pressure (Guglielmi 2008). In addition, blood flow through the AVM has been found to be 200-300% higher than normal controls due to the pressure differential between the feeding arteries and normal brain tissue (Grzyska & Fiehler, 2009; Quick, Hashimoto & Young, 2001).

Arteriovenous malformations are an uncommon vascular phenomenon occurring in approximately 4.3% of the population based on 4,530 consecutive autopsies (W. F. McCormick & Rosenfield, 1973). Pathology data have shown that approximately 12% of AVMs are symptomatic (Hashimoto, Iida, Kawaguchi, & Sakaki, 2004; W. McCormick, 1978) and researchers report that approximately 0.1 to 1% of the population will have a symptomatic AVM annually (Brown, Wiebers, Torner, & O'Fallon, 1996; Group, 1999; Hofmeister et al., 2000; J. H. Mohr, A.; Mast, H.; Pile-Spellman, J.; Schumacher, H.C. & Stapf, C. , 2004; Redekop, TerBrugge, Montanera, & Willinsky, 1998; Singer, Ogilvy, & Rordorf, 2006). More often, arteriovenous malformations are asymptomatic and go undetected unless there is a clinical event (such as hemorrhage or seizure). Unlike other brain lesions, the AVM itself often does not cause cognitive dysfunction. Lazar and his colleagues proposed that brain reorganization, due to the chronic nature of the AVM, explained this phenomenon (Lazar et al., 2000; Lazar et al., 1997). Rather, it is usually a hemorrhage that is responsible for functional/cognitive changes seen in patients with AVM.

The central point of abnormal development in the AVM is the nidus (Loring, 1999) which is the densest region of arteries and veins. The feeding arteries can include branches off the main cerebral arteries (e.g., middle cerebral artery, posterior cerebral artery, and anterior cerebral artery), carotid or vertebral arteries, or the choroidal arteries from the subcortical regions (Group, 1999). Both parenchymal (brain) and dural AVMs have the potential to cause a more focal deficit (similar to that seen in stroke) while dural AVM's have also been shown to cause a more global dementia-like syndrome (Lantz et al, 2009; Festa et al., 2004; Hurst et al., 1998; Ito, Sonokawa, Mishina, & Sato, 1995; Matsuda et al., 1999; Tanaka, Morooka, Nakagawa, & Shimizu, 1999). Subcortical (deep) AVMs have been associated with deficits such as neglect and memory disturbance (Buklina, 2001, 2002).

#### Neurological/Neuropsychological Deficits

Neurological deficit is reported in the AVM population with varying frequencies (1.3% to 48%), with reversible deficits significantly more common than persistent deficits (Hofmeister et al., 2000; Mast et al., 1995; Wenz et al., 1998). Hemorrhage related to AVMs account for approximately 1 to 2% of all strokes (Furlan, Whisnant, & Elveback, 1979; Gross, Kase, Mohr, Cunningham, & Baker, 1984; Hashimoto et al., 2004; Perret & Nishioka, 1966). Non-hemorrhagic seizures co-occur in 16 to 53% of the AVM population (Group, 1999; Hofmeister et al., 2000) and are the second most common presentation.

Some researchers have demonstrated significant deficits in neuropsychological functioning of patients, some studies of which unfortunately combine ruptured and unruptured AVMs (Baker, McCarter, & Porter, 2004; Mahalick, Ruff, Heary, & U, 1993; Marshall, Jonker, Morgan, & Taylor, 2003; Steinvorth et al., 2002; Wenz et al., 1998). Wenz et al. found that AVM patients (both with and without hemorrhage) demonstrated below normal performance on tests of general IQ (24% of patients), attention (34% of patients), and memory (48% of patients). Mahalick et al. also demonstrated significantly lower performance on tests of neuropsychological functioning for AVM patients as compared to normals, but again did not co-vary for prior

hemorrhage. In another study combining ruptured and unruptured AVM, it was found that AVM patients were again significantly below normals on test of intelligence, memory, and attention (Steinvorth et al., 2002).

Researchers have demonstrated improvement in neuropsychological functioning post-surgical resection of the AVM (Malik, Seyfried, & Morgan, 1996; Wenz et al., 1998). Cognitive improvements after AVM treatment have been attributed to improved cerebral blood flow and the presumed reduction of the “steal effect” (Malik et al., 1996; Steinvorth et al., 2002; Wenz et al., 1998). The steal effect refers to the assumption that shunting and hypertension through the AVM decreases cerebral perfusion, thus causing cerebral ischemia and ultimately neurological deficits (Mast et al., 1995). Iwama et al. (2000) demonstrated that intracranial steal and venous hypertension and not decreased neurological activity or mass effect, are responsible for the hemodynamic changes seen in high-flow arteriovenous malformations.

Other researchers disagree with the “steal” hypotheses (Mast et al., 1995; Stabell & Nornes, 1994). Stabell and Nornes reported that AVM patients performed the same as normal controls on cognitive assessment. While some significant improvement was observed, Stabell and Nornes dispute the “steal” hypothesis as an explanation for cognitive improvement after AVM surgery. Mast et al. (1995) were unable to replicate the “steal effect” using their prospective database. They demonstrated, for example, that AVM patients with chronic cerebral hypotension in Wernicke’s area did not have any functional cognitive impairment. In addition, they used positron emission tomography (PET) to study 14 AVM patients and demonstrated that while these patients did have hypoperfusion in surrounding tissue, they did not have any parenchymal volume loss and metabolism was normal (Mast et al., 1995). Developmental learning disorders have been found in 66% of adults with AVMs (Lazar et al., 1999). In this study, AVM patients reported four times the rate of learning disability than the normal population (17%). Lazar et al. reported that perhaps these disorders of higher intellectual functioning (e.g., learning) may serve as a marker for subtle developmental cerebral dysfunction in AVM patients.

### Treatment

Because only 0.1 to 1% of the population will have a symptomatic AVM, there is extensive research and discussion within the field regarding whether to perform preemptive surgery on unruptured AVM’s (Stapf, Mohr, Choi, Hartmann, & Mast, 2006). Natural history risk must be weighed with surgical risks in order to determine the risk-to-benefit ratio of treating unruptured AVMs. Because the risk of re-bleeding after initial intracranial hemorrhage (ICH) is significant, most researchers agree that treating a ruptured AVM is worth the risk. Unruptured AVMs, however, are more controversial.

Treatment decisions are determined by appearance (size, location, etc.) of the AVM and the Spetzler-Martin grading system has been adopted as a standardized system to rate AVMs according to their size, location (functional importance, often referred to as “eloquence”, of surrounding brain tissue) and venous drainage (Riina & Gobin, 2001). The

current treatments for AVM are embolization, radiosurgery (such as gamma knife), and craniotomy and resection. Each treatment can be given on its own, but is more often administered in combination. One goal of embolization and radiosurgery is to minimize the size of the AVM, thereby lowering the grade and improving treatment outcome after craniotomy. Due to the natural disease course of AVM’s, often non-symptomatic but still significant *risk* for morbidity or mortality, careful treatment planning is crucial.

Mahalick et al. (1993) reported that surgical excision of the AVM resulted in significant improvements in tasks involving short and long-term verbal memory, long-term visual-spatial memory, verbal learning, verbal and non-verbal intelligence. Overall, they found 60% of AVM patients performed in the normal range on neuropsychological examination post-surgery (Mahalick et al., 1993). When an AVM is inoperable due to size and/or location or a patient chooses not to undergo surgery given the natural history risk of bleeding, stereotactic gamma-knife radiosurgery is the treatment of choice.

Superselective Wada testing and electrocortical stimulation mapping are in vivo procedures used to help interventional neuroradiologists and neurosurgeons to identify eloquent cortex that may be surrounding the AVM, and helps to predict neurological and cognitive changes that would occur as an adverse effect of treatment. Currently, the Spetzler-Martin grading system aids surgeons by establishing a risk model by assessing the size and location of the AVM. The treating physician traditionally assumed eloquence from the location of the AVM (i.e., if the AVM is located in Broca’s area, the surgeon will assume this area is crucial for expressive language). However, because brain reorganization has now been associated with AVM (Lazar et al. 1997, 2000), the grading system without empirical data from in vivo testing of eloquence is felt no longer capable of establishing true treatment risk (Lazar, 2001).

Based on techniques used in epilepsy, superselective Wada testing is a clinical procedure that is used to help the interventional neuroradiologist determine prior to embolization whether a feeding artery to an AVM also supplies blood necessary for eloquent function in nearby brain areas. With the microcatheter in place delivery of embolic material, the neuroradiologist injects a short acting anesthetic (usually amobarbital or a combination of amobarbital and lidocaine) that lasts for approximately three to four minutes. During the testing period a neuropsychologist (who has previously collected baseline neurocognitive data for this particular patient) performs tests of cognitive functioning typically associated with brain region supplied by the feeding vessel. The neuroradiologist will use the results of Wada testing to help determine course of treatment. If a deficit is apparent during Wada testing, the radiologist may choose to embolize closer to the AVM nidus to diminish neurocognitive damage, or perhaps decide not to embolize at all. A negative Wada test result allows the neuroradiologist to embolize the feeding vessel with diminished risk for significant neurological damage. Evaluation for cognitive function during superselective Wada is typically adapted from well-

known neuropsychological tests (i.e., Boston Diagnostic Aphasia Tests, Wechsler Memory Scale) with well-established norms (Fitzsimmons, Marshall, Pile-Spellman, & Lazar, 2003; Goodglass & Kaplan, 1983; Wechsler, 1987).

Electrocortical stimulation mapping is performed intraoperatively during an awake craniotomy. While the patient performs different tasks, the brain is stimulated and essential versus nonessential cortical site can be mapped out (Cannestra et al., 2004).

## **CEREBRAL ANEURYSM**

### **Pathophysiology and Epidemiology**

Intracranial aneurysms are bulges in the arterial wall, with most in the brain being thin-walled sacs arising out of the circle of Willis or its major branches (Vates, Zabramski, Spetzler, & Lawton, 2004). Those that arise from defects in the media of the blood vessel wall are divided in those which are saccular (berry-shaped are the most common) or fusiform in shape, but other classifications include those which come about from an infectious (mycotic), traumatic or neoplastic etiology (J.P. Mohr & Gautier, 1995). Because of their weakened state, the walls of these vascular anomalies are vulnerable to rupture, producing spontaneous subarachnoid hemorrhage that is the predominant reason that aneurysms come to clinical attention. As with cerebral AVM's, once an aneurysm is identified the treating physicians must weigh the risk factors when determining whether or not to treat the unruptured aneurysm with some studies revealing higher mortality and morbidity with treatment than with spontaneous rupture while other studies have revealed better outcomes with treatment of unruptured aneurysms (Towgood, Odgen & Mee, 2005).

Cerebral aneurysms are the fourth leading cause of a cerebrovascular event, accounting for approximately 5-10% of all strokes (Adams & Davis, 2004), with most unruptured aneurysms detected incidentally and a small proportion found after seizures or III-nerve palsy. It has been estimated, based largely on autopsy findings, that the prevalence of intracranial aneurysm is about 2% of the general population (Hassler, 1961). In the United States, the most common age period for aneurysmal rupture is between 40 and 60 years old (Kassell & Torner, 1982), with occurrence in children and adolescents considered rare. Although SAH can present with different symptoms, it is the presence of the "worst headache of my life" that frequently occurs, along with nausea and vomiting. Among the most common clinical tools for rating syndrome severity are the Hunt and Hess Scale (Hunt & Hess, 1968) and Fisher Grade (Fisher, Kistler, & Davis, 1980). SAH is associated with a significant mortality rate so that 12% have sudden death, and 40% die in the first week after hospitalization (Huang & van Gelder, 2002) and 50% in the first six months (Schievink, 1997). Risk factors associated with SAH include the size of the aneurysm, female gender and pregnancy, smoking, excessive alcohol consumption and hypertension (J. P. Mohr, 1984; Weir, 2002). In terms of brain imaging, a non-contrast CT is currently the first step for the detection of SAH. The identification of the underlying aneurysm is still most reliability made via cerebral

angiography, but now CT angiograms appear capable of high sensitivity in the detection of these lesions (Papke et al., 2007).

The deleterious effects of SAH can be made worse with a number of possible complications. Vasospasm, for example, refers to the narrowing of the intradural subarachnoid arteries that occurs between 4 and 10 days after SAH, and can result in cerebral infarction, with ensuing behavioral and cognitive changes typical of those seen in ischemic stroke. In addition, there can be hydrocephalus, seizures, recurrent hemorrhage, electrolyte imbalance, and cardiopulmonary dysfunction (Stern, Chang, Odell, & Sperber, 2006).

### **Treatment**

The major treatment goal is geared towards disrupting the blood supply from the parent artery to the aneurysm thereby eliminating the risk of hemorrhage (Burns & Brown, 2009). Size and location are the two most important factors when deciding on treatment options. Aneurysms >10mm and in the posterior circulation have the highest rates of rupture (ISUIA Investigators, 1998). Additionally, morphological characteristics such as daughter sacs and surface irregularities, as well as individual hemodynamic features have been associated with increased risk of aneurysmal hemorrhage (Lall, Eddleman, Bendok & Batjer, 2009; Sadatomo, Yuki, Migita et al 2008).

There are two principle interventions for the treatment of the ruptured aneurysm designed to reduce the occurrence of re-hemorrhage. Microsurgical placement of a clip at the neck of the aneurysm takes place after craniotomy (Hop, Rinkel, Algra, & van Gijn, 1997), often occurring during the acute clinical period. Endovascular treatment became an attractive alternative with the development of microcatheters which could place detachable coils within the body of the aneurysm and induce thrombosis (Guglielmi, Vinuela, Dion, & Duckwiler, 1991). Despite the recent outcomes of the ISAT trial that showed lower morbidity and complication rates for coiling than with clipping (Molyneux et al., 2002), superiority with regard to long-term effectiveness between these two treatment has not been well established. Generally, coiling is the preferred initial treatment option due to a high likelihood of aneurysmal occlusion and the reduced risk of re-bleeding (Burns & Brown, 2009, Ries & Groden, 2009).

### **Neuropsychological Findings**

Improvements in treatment of ruptured aneurysms have increased survival rates so that quality-of-life concerns have gained increasing relevance in the assessment of patient outcomes. It is now well-accepted that despite improvements in physical status, patients who survive SAH can suffer from significant neurobehavioral deficits, even with excellent outcomes as defined by the Glasgow Outcome Scale (Haug et al., 2007). Residual deficits have been reported in patients with so-called "good recovery" even up to seven years after initial hospitalization (Hellawell & Pentland, 2001). Studies have largely confirmed that aneurysmal clipping of unruptured aneurysm produces few, if any, neuropsychological deficits, and that it is the presence of

hemorrhage that appears responsible for the neurocognitive sequelae in most patients (Hillis, Anderson, Sampath, & Rigamonti, 2000; Tuffiash, Tamargo, & Hillis, 2003). What is not yet clear, however, is the nature, extent and time course of the neuropsychological and emotional changes that arise after hemorrhage and intervention.

There have been a wide range of impairments associated with aneurysmal SAH. The spectrum of disorders measured after hemorrhage has included verbal memory deficits (DeLuca, 1993; Irle, Wowra, Kunert, Hampl, & Kunze, 1992), visual disorders (Ogden, Mee, & Henning, 1993) and defects in information processing (Bellebaum et al., 2004). Frontal-lobe dysexecutive syndromes are also commonly reported (Bellebaum et al., 2004). But, as has been pointed out by Haug et al (Haug et al., 2007), follow-up of patients up to one year after SAH has demonstrated that various cognitive functions have different courses of recovery. The time point of assessment after SAH therefore appears to be an important factor in the constellation of residual deficits, especially in patients with poor SAH grades (Mocco et al., 2006).

One of the complicating factors is determining the cognitive consequences of SAH is that the majority of patients have also received either surgical or endovascular treatment. Early studies in which patients underwent either intervention and were assessed at one time point showed marginally fewer effects after coiling than post-clipping (e.g. Hadjivassiliou et al., 2001). Bellebaum and his colleagues compared two groups of 16 patients, assigned either to clipping or coiling treatment (Bellebaum et al., 2004). Both patient groups showed deficits in verbal and visual memory, with the clipping group demonstrating slightly greater impairment, especially in frontal executive function. But, as pointed out by Haug et al above, serial measurement of cognitive function can yield other outcomes. Individuals undergoing coiling or clipping were assessed within two weeks of treatment and again at six months by Frazer et al. (Frazer, Ahuja, Watkins, & Cipolotti, 2007) They found acute changes in both groups in memory, executive function, and information-processing speed. At follow-up, deficits in both groups were less severe, with residual dysfunction in memory, frontal executive dysfunction, naming skills and information-processing speed. Interestingly, there was no difference in functioning between the treatment groups at both time points. Koivisto et al found no differences in neuropsychological function across three measurement points, and patients undergoing either coiling or clipping demonstrated improvement over time (Koivisto et al., 2000).

One of the presumed, determining factors has been the location of the bleed. As the most common site of SAH, rupture of the anterior communicating artery has been proposed by some investigators to be associated with the triad of an amnesic disorder, confabulation and alteration of personality, collectively labeled the "ACoA syndrome" (Damasio, Graff-Radford, Eslinger, Damasio, & Kassell, 1985; DeLuca & Diamond, 1995). Comparisons of neurocognitive outcomes of patients with ruptured aneurysms in this and other regions, however, have not yielded consistent behavioral differences. Results of executive function tests,

for example, have not uniformly discriminated between patients with anterior and posterior SAH sites (Papagno, Rizzo, Ligori, Lima, & Riggio, 2003).

The inconsistency of syndromal findings in patients with similar regions of SAH, similar behavioral manifestations in the context of hemorrhage in different brain areas, and differing long-term outcomes in patients with comparable initial cognitive profiles does suggest the presence of other important factors that affect neurocognition in this setting. It was proposed almost 20 years ago that local bleeding in the subarachnoid space causes a more diffuse, global toxic effect on the brain (Laiacona et al., 1989). This notion has received increasing support from clinical outcome studies. Kreiter et al prospectively evaluated 113 of 248 consecutively admitted nontraumatic SAH patients alive at 3 months (Kreiter et al., 2002). Predictors of cognitive dysfunction in two or more domains in a multivariate model showed that global cerebral edema had the highest predictive value associated with residual cognitive impairment. It is therefore not surprising that the severity of the hemorrhage in other studies as seen on imaging is correlated with more diffuse neuropsychological functions, even when the precise origin of the hemorrhage is unknown (Hutter, Gilsbach, & Kreitschmann, 1994). Another potential global factor, the presence of the apolipoprotein  $\epsilon 4$  allele which has been found important in other neurological conditions, does not appear to be a global risk factor for late cognitive impairment, even 10 years after hemorrhage (Louko, Vilkki, & Niskakangas, 2006).

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## Newsletter

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## Newsletter