Cognitive impairment and ultrasound (US)

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Dementia
a leading cause of memory impairment and disability
Dementia is a clinical syndrome presenting with acquired, progressive cognitive impairment that may be related to several ethiopathogeneses, the two main leading causes being represented by degenerative Alzheimer’s disease (AD) and vascular dementia (VaD).
Prevalence of Dementia-Clinical Subtypes

- PDD: 50%
- OTHERS: 22%
- MIXED: 10%
- VaD: 18%
Alzheimer’s Diseases: History

Presenile dementia versus “Senile cerebral arteriosclerosis”

Auguste D
Vascular Cognitive Impairment syndromes

• Multi-infarct dementia
• Lacunar state
• Single strategically-placed infarct
• Post-stroke cognitive deterioration
• Binswanger’s disease
• Genetic forms (e.g. CADASIL)
• Hypoxic-ischemic encephalopathy
DSM-IV criteria for VaD

1. Multiple cognitive deficits
   Memory + 1 (or more) additional problems:
   Aphasia/Apraxia/Object agnosia/Executive dysfunction

2. Each causing significant impairment in social/occupational functioning

3. Focal neurological signs/symptoms (DTR, ExPyr, Pseudobulbar palsy, gait impairment) OR Imaging evidence of CVD, which are judged to be etiologically related to the cognitive disturbance
The Vascular – AD Connection

• VaD patients frequently have an insidious onset and a progressive relentless course, mimicking PDD

• AD patients have vascular pathology, i.e. amyloid perivascular deposits
Brain infarction and the clinical expression of Alzheimer’s disease

The Nun Study

Snowdon et al, JAMA 277:813, 1997
Lessons from the Nun Study

Pure AD and pure VaD may represent the two extremes of the spectrum and a ‘mixed dementia’ is actually consistent with the majority of cases. As a matter of fact, at least one third of AD cases bear evidence of cerebral infarction on autopsy.
Vascular risk factors for dementia

- Hypertension
- Atherosclerosis
- Coronary artery disorder
- Smoking
- Hyperhomocysteinemia
- Diabetes mellitus
- Apolipoprotein E status
- High dietary saturated fat and cholesterol
- Midlife cholesterol
Dementia of Alzheimer type

Healthy control
DAT 60 year old male
Severe DAT 64 year old female
MID [define] 50 year old male
Pick's disease 69 year old female
DAT aphasia 59 year old female
VASCULAR DEMENTIA

Blood is delivered to the brain via the vascular system, a network of arteries and blood vessels. If the system has problems supplying blood, it can cause vascular dementia (the second most common form of dementia).

Two pairs of arteries supply blood to the brain. As the blood travels deeper into the brain it enters smaller blood vessels.

A healthy blood vessel's insides are smooth and the walls are flexible. This helps blood travel quickly and easily to where it needs to go.

Vertebral artery
Carotid artery

Problems arise when...
- The inside of the vessels becomes narrow, rigid or blocked.
- The blood vessels become damaged and blood leaks out of them.
- The heart doesn't pump forcefully enough.
- The blood thickens or clots too easily.

Searching for vascular dementia

A horizontal MRI scan of the brain in which brain regions with circulation problems appear as bright patches. This is typical of one type of vascular dementia in which multiple small vessels are blocked.

Source: Duke University,
Dr. Murali Doraiswamy
The News & Observer
Vascular dementia (VaD)

- Dementia is one of the common devastating sequels after stroke.
- 30% of post-stroke patients demonstrate the occurrence of cognitive impairment within 3 months following stroke.
- Patients with VaD have a higher mortality rate independently of the effects of age and co-morbidities.
- VaD diagnosed 3 months after stroke was also associated with an increased risk of stroke recurrence.
Four main difficulties in the definitions of VaD

1. Undue reliance on memory impairment
2. Difficulty to decide whether, in a patient with CVD, the cognitive impairment is due to the vascular lesions
3. Difficulty to decide whether, in a stroke victim with physical incapacity, the cognitive decline would independently affect ADL
4. Difficulty in distinguishing slowness, lack of motivation and poor initiative from depression
### Accuracy of clinical diagnosis

<table>
<thead>
<tr>
<th></th>
<th>AD</th>
<th>VaD</th>
<th>Total</th>
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<tbody>
<tr>
<td>Todorov (’75)</td>
<td>28%</td>
<td>57%</td>
<td>30%</td>
</tr>
<tr>
<td>Molsa (’85)</td>
<td>71%</td>
<td>73%</td>
<td>65%</td>
</tr>
<tr>
<td>Victoroff (’95)</td>
<td>82%</td>
<td>45%</td>
<td>70%</td>
</tr>
<tr>
<td>Chiu (’96)</td>
<td>56%</td>
<td>18%</td>
<td>50%</td>
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Will imaging provide a solution?
Transcranial Doppler sonography

- TCD and more recently Transcranial Color Doppler and Power Imaging give detailed information about the flow velocity in brain arteries and veins.
- This hemodynamic information is routinely used in diagnosis of cerebrovascular disease.
Procedures using TCD

- Routine TCD
- Functional TCD
- TCD and microembolic detection
Multiinfarct and Alzheimer-type dementia investigated by transcranial Doppler sonography.

**Foerstl H, Biedert S, Hewer W.**

Zentralinstitut für Seelische Gesundheit, Mannheim, West Germany.

Primary degenerative dementia of the Alzheimer type and multiinfarct dementia exhibit differences in cerebrovascular blood flow velocity profiles, which were investigated by transcranial Doppler sonography. The pulsatility indices, as angle-independent parameters of peripheral vascular resistance, measured in middle cerebral and basilar arteries of patients with multiinfarct dementia were significantly increased (p less than 0.005) compared with cases of primary degenerative dementia of the Alzheimer type and with healthy age-matched controls.
Transcranial Doppler ultrasonography in senile dementia: neuropsychological correlations.

Caamaño J, Gómez MJ, Cacabelos R.
Department of Digital Diagnosis, Basic and Clinical Neurosciences Research Center, La Coruña, Spain.
Blood flow velocities were measured in right and left middle cerebral arteries (MCAs) and in basilar artery (BA) on rest conditions by transcranial Doppler ultrasonography in three groups of patients: a) Alzheimer's disease patients (AD, N = 12, age = 63.5 +/- 6.6 years), b) patients with multi-infarct dementia (MID, N = 12, age = 72.8 +/- 9.0 years), and c) control subjects (CS, N = 12, age = 57.20 +/- 7.5 years). TCD measures were taken through the temporal window for MCA recordings, and transforaminal approach for BA recordings, with a 2 MHz ultrasonic probe using a TC-2000S (EME). A significantly (p < 0.05) decrease in TCD measures was found in right and left MCA and BA of dementia patients with respect to controls. Data analysis included a significant correlation between blood flow velocities and Hachinski scores (p < 0.016) in multi-infarct dementia patients. These results appear to show a general hypoperfusion pattern in the brain of senile dementia patients.
OBJECTIVE: Recent post-mortem studies have reported that the severity of atheromatous deposits in the circle of Willis is significantly greater, relative to non-demented (ND) elderly persons, in subjects with neuropathologically diagnosed Alzheimer's disease (AD). Additionally, the severity of intracranial atherosclerosis correlates significantly with the densities of neuritic plaques and neurofibrillary tangles. In this study, we examine the arteries of the circle of Willis by transcranial Doppler (TCD) ultrasonography. METHODS: TCD was used to measure, in 25 AD patients and 30 ND elderly subjects, mean flow velocities and pulsatility indices in 16 different segments of the circle of Willis. The data were compared with and without adjustment for age, gender and systolic blood pressure. RESULTS: The AD patients had systematically higher pulsatility indices (p<0.005) than the ND group. Incremental increases of pulsatility indices in these segments had odds ratios ranging from 1.8 to 48 for the presence of AD when adjusted for age, gender and systolic blood pressure. The left internal carotid artery siphon and the left posterior cerebral artery were the two vessels that were strongly associated with AD diagnosis. Mean flow velocities were generally lower in patients with AD but the differences did not reach the significance level. DISCUSSION: The pulsatility indices of the arteries of AD patients were generally greater than those of similarly-aged ND subjects. This difference is most likely due to increased arterial wall rigidity imposed by atherosclerotic changes. Atherosclerotic disease of intracranial arteries may be a risk factor for AD.
Transcranial and extracranial ultrasound assessment of cerebral hemodynamics in vascular and Alzheimer's dementia.

**Doepp F, Valdueza JM, Schreiber SJ.**

Transcranial and extracranial ultrasound permits analysis of cerebral hemodynamics and should help to differentiate between VD and AD. We compared multimodal ultrasound data between VD, AD and controls, and give an overview of the literature on this topic.

**METHODS:** Twenty VD and 20 AD patients were studied and compared with 12 age-matched controls. Transcranial color-coded ultrasound was performed to assess blood flow velocity ($V(\text{mean})$) and pulsatility indices (PI) of the middle cerebral artery (MCA). Extracranial duplex and Doppler ultrasound techniques were used to assess the blood volume flow (BVF) in the anterior circulation (both internal carotid arteries [ICA]) and posterior circulation (both vertebral arteries [VA]), the global cerebral blood flow ($\text{CBF} = \text{BVF(ICA)} + \text{BVF(VA)}$), the global cerebral circulation time ($\text{CCT} = \text{time delay of echo-contrast bolus arrival between ICA and internal jugular vein}$) and global cerebral blood volume ($\text{CBV} = \text{CCT x CBF}$).

**RESULTS:** MCA $V(\text{mean})$ in VD (36 +/- 8 cm/s) and AD (43 +/- 13 cm/s) were significantly lower than in controls (59 +/- 13 cm/s) but did not differ significantly between VD and AD groups. PI (1.1 +/- 0.2; 1 +/- 0.2; 0.9 +/- 0.2) only differed significantly between VD group and controls. CBF and CCT in VD (570 +/- 61 ml/min; 8.8 +/- 2.6 s) and AD (578 +/- 77 ml/min; 8.2 +/- 1.4 s) were similar but differed significantly from controls (733 +/- 54 ml/min; 6.4 +/- 0.8 s). DISCUSSION: Transcranial and extracranial ultrasound does not help to distinguish between VD and AD. However, our results add insight into the pathophysiology of dementia, arguing in favor of a common 'vascular' pathway in both conditions.
The cerebral vasculature has a unique ability to dilate during hypercapnia and to constrict during hypocapnia.

The differences between cerebral blood flow (CBF) at rest and after the induction of hypercapnia reflect the state of cerebral vasomotor reactivity (VMR) and, hence cerebrovascular reserve capacity.

VMR can be considered as a shift between cerebral blood flow velocity (BFV) before and after the administration of a potent vasodilatory stimulus test.

Assessment of VMR could provide the valuable information regarding hemodynamic patterns of cerebral autoregulation and collateral circulation.
Vasodilatory tests

• Apnea-breath holding test

• CO2 inhalation

• Diamox (acetazolamide) test

• L-Arginine test
During the past decade, transcranial Doppler sonography has widely been used to assess blood flow velocities in the basal intracranial arteries and cerebrovascular reactivity (CR) to various stimuli. Although numerous studies have shown a decline of cerebral blood flow velocity with age, the age dependency of CR, including cerebrovascular CO2 reactivity, however, is controversial.

**Conclusions**—There are no changes of CR during normal aging in men, whereas CR declines significantly from the 4th to the 5th decades in women. HRT in postmenopausal women appears to enhance CR.
The aim of this study was to explore the possible contribution of alterations in cerebral hemodynamics to the evolution of cognitive impairment in patients with Alzheimer disease (AD).

Fifty-three patients with AD were investigated. The evolution of cognitive decline over 12 months was evaluated by means of changes in Mini Mental State Examination (MMSE) and AD Assessment Scale for Cognition (ADAS-Cog) scores.

These results show an association between impaired cerebral microvessels functionality and unfavorable evolution of cognitive function in patients with AD.
Top, Relationship between BHI and deterioration in MMSE scores according to a polynomial regression model that accounted for 43% of variance

Cerebrovascular Reactivity in Degenerative and Vascular Dementia: A Transcranial Doppler Study
Edoardo Vicenzini, Maria Chiara Ricciardi, Marta Altieri, Francesco Puccinelli, Novella Bonaffini Vittorio Di Piero, Gian Luigi Lenzi

Eur Neurol 2007;58:84–89
60 AD and 58 VaD patients and 62 nondemented controls. Both AD and VaD subjects showed lower flow velocities (FV) and higher pulsatility indices (PI) as compared with controls.

Lower total vasomotor reactivity and lower response to hypercapnia were observed in the AD and VaD groups as compared with controls.

AD and VaD patients did not show significant differences in FV, PI values or cerebral vasoreactivity.
**Table 3. Vasoreactivity in right and left MCA during hyper- and hypcapnic conditions in AD, VaD and controls**

<table>
<thead>
<tr>
<th></th>
<th>VaD</th>
<th></th>
<th>AD</th>
<th></th>
<th>Controls</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>right</td>
<td>left</td>
<td>right</td>
<td>left</td>
<td>right</td>
<td>left</td>
</tr>
<tr>
<td>Total VMR, %</td>
<td>62.1 ± 12.9*</td>
<td>61.8 ± 14.9*</td>
<td>63.8 ± 14.2*</td>
<td>60.1 ± 15.6*</td>
<td>81.4 ± 16.8</td>
<td>80.1 ± 15.7</td>
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<tr>
<td>RI CO₂</td>
<td>3.1 ± 1.7*</td>
<td>2.8 ± 1.5*</td>
<td>3.5 ± 1.9*</td>
<td>2.5 ± 1.5*</td>
<td>5 ± 1.4</td>
<td>4.8 ± 1.2</td>
</tr>
<tr>
<td>RI Hp</td>
<td>3.2 ± 1.6</td>
<td>3.1 ± 1.4</td>
<td>3.0 ± 1.6</td>
<td>2.9 ± 3.6</td>
<td>3.6 ± 0.8</td>
<td>3.6 ± 0.8</td>
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Values are means ± SD. Total VMR = Total vasomotor range; RI CO₂ and RI Hp = reactivity index to hypercapnia and hypcapnia, respectively, calculated as percentage per millimeter mercury column. * p < 0.0001 compared with controls.
Cerebral reactivity to apnea was significantly lower in the multi-infarct group.

These data suggest that cerebrovascular reactivity to apnea could be an additional criterion for discriminating between MID and DAT patients. Transcranial Doppler assessment during cognitive and motor tasks could provide useful complementary information for comprehension changes in cerebral activity in patients with dementia.
Can TCD to differentiate between VaD and AD?

Stroke. 1993 Feb;24(2):228-35

Differentiation of multi-infarct and Alzheimer dementia by intracranial hemodynamic parameters.

Ries F, Horn R, Hillekamp J, Honisch C, König M, Solymosi L.

BACKGROUND AND PURPOSE: The differentiation between the Alzheimer and multi-infarct types of dementia may still be equivocal considering clinical criteria, neuropsychological tests, and imaging techniques. Cerebral microangiopathic alterations underlying multi-infarct dementia should allow the characterization of dementia subgroups. METHODS: Patients with a diagnosis of multi-infarct dementia (n = 17; mean age, 69.1 +/- 8.5 years) or Alzheimer dementia (n = 24, mean age, 65.8 +/- 9.0 years) according to standard testing criteria, clinical findings, and neuroimaging techniques (computed tomography and magnetic resonance imaging) were investigated prospectively by transcranial Doppler sonography and compared with a normal reference group (n = 64; mean age, 61.0 +/- 11.1 years). Transcranial Doppler sonography allows an indirect evaluation of peripheral flow resistance in the microcirculatory bed by quantifying pulsatility characteristics, as reflected in the effective pulsatility range (time-averaged mean blood flow velocity minus the peak-systolic to end-diastolic amplitude, in centimeters per second). RESULTS: A total of 204 vessels were investigated in 105 subjects. Mean and diastolic blood flow velocities as well as the effective pulsatility range were significantly lower in the multi-infarct dementia group compared with the Alzheimer and the normal reference groups (p < 0.001). By using receiver operating characteristic analysis, a cutoff point for effective pulsatility range values of -5 cm/sec gives a side-dependent sensitivity of 90.48-95.24% and a specificity of 64.71-70.59% in diagnosing Alzheimer-type dementia; the corresponding sensitivity and specificity for a value of -2 cm/sec are 82.35-88.24% and 80.95-90.48%, respectively. CONCLUSIONS: Pulsatility changes as reflected by the effective pulsatility range are a noninvasive additional criterion in the differential diagnosis of dementia.
Can TCD to differentiate between VaD and AD?

The value of transcranial Doppler sonography in the differential diagnosis of
Alzheimer disease vs multi-infarct dementia.

Biedert S, Förstl H, Hewer W.

Primary degenerative dementia of the Alzheimer type and multi-infarct dementia
exhibit differences in cerebrovascular blood flow velocity profiles, which were investigated
by means of transcranial Doppler sonography. The pulsatility indices, as angle-independent
parameters of peripheral vascular resistance, measured in middle cerebral and basilar
arteries of patients with multi-infarct dementia (MID), were significantly increased (p <
0.005) with respect to cases of primary degenerative dementia of the Alzheimer type and to
healthy age-matched controls. Approximately 75% of all MID patients exhibited small
vessel disease rather than thromboembolism from the extracranial arteries and the heart, as
judged by extracranial and transcranial Doppler sonographies, computerized cerebral
tomographies, EEGs, and, if necessary, 2-D echocardiographies.
Influence of galantamine on vasomotor reactivity in Alzheimer's disease and vascular dementia due to cerebral microangiopathy.

Bär KJ, Boettger MK, Seidler N, Mentzel HJ, Terborg C, Sauer H.

BACKGROUND AND PURPOSE: Recent reports suggest that vascular factors play a crucial role in the development and progression of Alzheimer's disease. We aimed to assess vasomotor reactivity in patients with Alzheimer's disease and vascular dementia due to microangiopathy using transcranial Doppler sonography and near-infrared spectroscopy during a CO(2) exposition task. METHODS: The normalized CO(2) reactivity assessed at the middle cerebral artery and the oxygenated and deoxygenated hemoglobin of the frontal cortex were obtained. To investigate the impact of cholinergic deficiency known for Alzheimer's disease on vasomotor reactivity, both groups were reinvestigated during treatment with the acetylcholine esterase inhibitor galantamine. RESULTS: Transcranial Doppler analysis revealed significantly reduced normalized CO(2) reactivity for Alzheimer's disease and vascular dementia. Vasomotor reactivity assessed by near-infrared spectroscopy was decreased in patients with vascular dementia, but not in Alzheimer's disease. Galantamine treatment showed a beneficial effect, normalizing these parameters close to age-matched control levels. CONCLUSIONS: Our results suggest that Alzheimer's disease is associated with a lack of vasomotor reactivity, which might be associated with disturbed autoregulation indicating a potential risk for a decreased protection of brain tissue against blood pressure changes. Additionally, a diminished increase of cortical oxygenated hemoglobin during the CO(2) test was apparent in patients with vascular dementia. Galantamine treatment influenced vascular reactivity in the CO(2) test, thus providing evidence for the cholinergic deficiency, thereby adding to vascular dysregulation in Alzheimer's disease, but also indicating an important role of cholinergic system dysfunction for vascular dementia.
Figure 2. NIRS. Differences for CR-O₂Hb between control subjects (old: control subjects matched with respect to age and sex, n=20; young: control subjects from a different age population, n=20) and patients with AD disease (n=17) and VaD (n=17) are depicted as box plots (A). Into NCR, no significant difference was obvious between old control subjects and AD, but between control subjects and VaD. The effect of galantamine treatment is depicted in (B) showing an increase of oxygenated hemoglobin in VaD to the level of patients with AD, but no significant change in AD itself. NIRS parameters showed significant correlations to Mini-Mental State Examination scores (C) for both patients with AD (empty squares) and patients with VaD (filled circles) as well as when including both in the analysis as shown here. Boxes in (A) and (B) indicate data between the 25th and 75th percentile with the horizontal bar reflecting the median (=mean; o=1st and 99th percentile; —=minimum and maximum of data; *P<0.05; **P<0.01; ***P<0.001).
CEREBRAL VASOMOTOR REACTIVITY AND COGNITIVE DECLINE IN PATIENTS AFTER ISCHEMIC STROKE. ISRAELI-TURKISH COLLABORATIVE STUDY

A.Y. Gur,¹ D. Gücüyener ², N.Üzüner ², Y. Gilutz ¹. Özdemir ², A.D. Korczyn ¹, N.M. Bornstein¹

Departments of Neurology, Tel Aviv University, Tel Aviv, Israel ¹; Osmangazi University, Eskisehir, Turkey ²
• To assess and compare VMR findings in patients with and without dementia after suffering IS.
Eleven patients with first-ever acute (within 72 hours of stroke onset) IS (AIS).

20 patients within 3-36 months after IS (PIS).

Israeli-Turkish collaborative study: Tel Aviv Sourasky Medical Center (TASMC).

Department of Neurology, Osmangazi University, Eskisehir, Turkey.
• TCD and the Diamox test (1g acetazolamide IV).
• VMR is defined as a shift between cerebral blood flow (CBF) or cerebral blood flow velocity (BFV) before and after administration of a potent vasodilatory stimulus test.
• Dementia was diagnosed based on DSM-IV and the MMSE scale.
• The VMR% values of patients with and without dementia were compared using the ANOVA test.
Diamox test

- Full cooperation is not essential
- Easy performing
- Blood gases monitoring is not essential
VMR% on the side of brain infarct in the AIS group with and without dementia after 6 months of follow-up.

<table>
<thead>
<tr>
<th>AIS group</th>
<th>Mean VMR%</th>
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<tbody>
<tr>
<td>without dementia</td>
<td>40.7±29.1%</td>
</tr>
<tr>
<td>n=9</td>
<td>P=NS</td>
</tr>
<tr>
<td>with dementia</td>
<td>33.3±21.5%</td>
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<tr>
<td>n=3</td>
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VMR% on the side of brain infarct in the PIS group with and without dementia

<table>
<thead>
<tr>
<th>PIS group n=20</th>
<th>Mean VMR%</th>
</tr>
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<tbody>
<tr>
<td>without dementia n=10</td>
<td>43.7±29.8%</td>
</tr>
<tr>
<td>with dementia n=10</td>
<td>36.4±28.5</td>
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P=NS
Conclusions

VMR values assessed in the acute phase or late phases of stroke neither predict nor correlate with cognitive decline.
Cerebral microembolus detection

[Diagram showing a vessel with blood flow and Doppler power measurement]
Fig. 2. A cerebral microembolus (arrow) passing through the middle cerebral artery during one heart cycle. Colour scale in decibels (top). Embolus (red), red blood cells (blue), Horizontal axis = time. Vertical axis = velocities (cm/sec).
The value of TCD with microemboli detection to investigate potential causes of vascular dementia

Stroke. 1998 Sep;29(9):1821-6. Links

Microemboli in cerebral circulation and alteration of cognitive abilities in patients with mechanical prosthetic heart valves.

Deklunder G, Roussel M, Lecroart JL, Prat A, Gautier C.

BACKGROUND AND PURPOSE: It has been shown previously that cerebral microemboli may occur frequently in patients with a normal mechanical heart valve (MHV) without prior history of stroke. Some arguments strongly suggest that these microemboli have a gaseous origin. In other circumstances such as extracorporeal circulation or decompression in divers, it has been demonstrated that cerebral microbubbles could lead to some deterioration in cognitive functions. Therefore, we have studied attention and memory, which are among the most impaired cognitive functions as demonstrated in previous studies, in patients with an MHV.

METHODS: Three groups of 12 volunteers each were composed of patients with an MHV and embolic signals in the cerebral circulation (group 1), patients with biological prostheses (group 2), and healthy subjects (group 3). Groups were carefully matched for age and verbal intellectual abilities. For each group, a transcranial Doppler examination was performed and a set of cognitive tests assessing sustained and selective attention and episodic and working memory was administered.

RESULTS: The mean embolic rate was 29 per hour in patients with an MHV. No embolus was detected in the other 2 groups. Episodic memory was significantly modified in both groups 1 and 2 compared with the control group for tasks that required high-processing resources. Working memory performance was significantly decreased in MHV patients. No between-groups differences were observed for the other parameters. CONCLUSIONS: Alteration of episodic memory can be attributed to a long-term effect of the surgical procedure. Deterioration of working memory can be related to the presence of cerebral microemboli in MHV patients.
The value of TCD with microemboli detection to investigate potential causes of vascular dementia

Methods of detecting potential causes of vascular cognitive impairment after coronary artery bypass grafting.

Russell D, Bornstein N.
Coronary artery bypass grafting (CABG) is the most common major surgical procedure performed worldwide. Neuropsychological deficits are frequent following CABG occurring in up to 80% in the early postoperative period, 20-50% at 6 weeks and 10-30% of patients at 6 months. Transcranial Doppler monitoring is well suited for monitoring the brain during surgery. It has been shown that both solid and gaseous microemboli are frequent during, surgery especially during clamping and declamping of the aorta. This method can also monitor cerebral hemodynamics during surgery and alert the surgical team when a fall in perfusion pressure occurs. Magnetic resonance imaging (MRI) studies have found evidence which suggests increased water content in the brain following "on-pump" CABG. New postoperative cerebral lesions have also been found in many patients using diffusion-weighted MRI.
Cerebral microemboli and cognitive impairment
David Russell

Transcranial Doppler Ultrasound (TCD) may be used to detect cerebral microemboli in patient groups with an increased stroke risk and during invasive cardiovascular examinations and operations. Although these microemboli do not cause immediate symptoms, there is growing evidence which suggests that they may cause cognitive impairment if they enter the cerebral circulation in significant numbers. This has been studied in detail in patients who have had coronary artery bypass surgery. In these patients, an association has been found between the number of intraoperative cerebral microemboli detected by transcranial Doppler and postoperative neuropsychological outcome. It is also possible that cerebral microemboli may be the cause of cognitive impairment in patients with cerebrovascular disease. Cerebral microemboli are often found in patients with atherosclerosis, especially of the carotid arteries and aortic arch, and in patients with heart disease. There is also an increased risk for silent strokes and cognitive impairment in these patients. Prospective clinical studies are therefore required to determine if continuous cerebral microembolization to the brain will lead to progressive cognitive impairment.
IN SUMMARY

• The potential usefulness of US (TCD) in the diagnosis and management of patients with dementia has been clearly shown.

• The value of TCD in the differential diagnosis of AD and VaD is still controversial.

• Cerebral microemboli detection by TCD may be used to determine potential causes of cognitive impairment.