Integrative assessment and treatment of cognitive impairment

2005 U.S. Psychiatric Congress

James Lake M.D.
www.IntegrativeMentalHealth.net
Pacific Grove, CA.
Adjunct clinical faculty
Stanford

View Bibliography
Integrative mental health care—foundations

- Brief overview of steps
- History/assessment to formulation to Rx. Plan to assessment and so on until sx resolve and/or Rx exhausted
- Takes into account indiv preferences, values and resources, costs, etc.
Severe impairment: differential diagnosis

- Delirium—transient, normal baseline, global deficits, identified medical cause or acute substance intoxication
- Dementia—progressive deteriorating course, global deficits, no identified medical cause
- TBI or cerebrovascular accident (Stroke)
- Amnestic syndrome—transient deficit in capacity to learn or recall new information
Mild cognitive impairment

- Possible causes include
  - Normal aging
  - Chronic nutritional deficiencies
  - Less severe stroke injury
  - Chronic narcotic abuse
  - Adverse effects of prescribed drugs
  - Social isolation
Severe cognitive impairment

- TBI most common cause. 2 million new cases each year costing $48B
- Dementia is second most common cause and Alzheimer’s accounts for 60 to 70% of all dementia at annual cost of $100B
- Stroke is the third most common cause at 500,000 new cases each year costing $43B
Conventional assessment methods

- Careful history establishes course and symptoms
- Laboratory workup if indicated
- Functional brain imaging if evidence of CVA, TBI or infection
- Mini-mental state exam in dementia but not mild or moderate cases
- Co-morbid psychiatric symptoms and history
Limitations of conventional assessment methods

- Rigorous medical work-ups often not done
- Comprehensive medical-psychiatric history seldom done
- Insurance coverage limits work-ups
- Patients and relatives do not advocate for more extensive evaluation
- Consequences include missed or misdiagnosis, no or inadequate treatment, and reduced treatment cost-effectiveness
The integrative assessment of cognitive impairment

- Starts with a conventional medical, psychiatric, social and cultural history, MMSE, and appropriate laboratory studies
- Includes non-conventional approaches that potentially yield specific accurate information about causes of dysfunction
- Takes into account relative evidence for different approaches, cost, availability, and patient preferences
Rating evidence of non-conventional assessment and treatment approaches

- Based on modified EBM criteria
- Four levels of evidence
  - Substantiated—compelling evidence
  - Provisional—good evidence
  - Possibly effective/specific—inconsistent findings
  - Refuted—consistent negative findings
Non-conventional assessment approaches

- **Substantiated**
  - QEEG and neurometric brain mapping to evaluate cognitive impairment in TBI, post-stroke and dementia

- **Provisional**
  - QEEG mapping with VEP or AER in dementia and mild cognitive impairment
  - Virtual reality testing environments
  - Serum zinc and magnesium levels
Substantiated
assessment approaches
QEEG Brain mapping

- QEEG measures include power, left-right inter-hemispheric symmetry, and phase coherence of electrical brain activity in homologous regions in both hemisphere (Senf 1988)

- Shows correlations between cognitive impairment related to dementia or stroke (CVA) and diminished activity in the alpha and beta ranges
QEEG brain mapping

- Provides useful information when planning EEG biofeedback protocols addressing cognitive impairment.
- Shows changes at different stages of Alzheimer’s disease and other syndromes of severe cognitive impairment which are frequently not apparent in conventional CT or MRI imaging studies (Passero 1992).
- Can be used to evaluate response to both conventional and non-conventional cognitive enhancing treatments.
Neurometric Brain mapping

- Specialized QEEG approach that compares EEG characteristics of the individual being evaluated with normative databases for the same age.

- Helps to clarify functional brain correlates of cognitive impairment, and yields information that is useful for planning EEG biofeedback protocols addressing specific kinds of dysfunction.
Neurometric brain mapping

Neurometric brain mapping is an evolving diagnostic tool which is increasingly used in clinical settings to differentiate cognitive impairments that are due to head injuries, medical disorders, progressive dementia, alcohol or substance abuse, depressed mood, learning disorders or other underlying biological causes.
QEEG brain mapping

- The rate of deterioration in normal theta (3.5 to 7.5 Hz) activity in the early stages of dementia is highly predictive of long-term clinical outcomes (Rodriguez 1996)

- Abnormal decreases in slow-wave delta activity (1.5 to 3.5 Hz) take place after normal theta activity has deteriorated, and correspond to severe dementia
Provisional assessment approaches
Serum zinc

- Alzheimer’s risk may possibly be associated with chronic low blood levels of zinc and magnesium—findings inconsistent.

- High zinc levels are neurotoxic and may promote formation of amyloid plaques, however histopathology studies of the brains of Alzheimer’s patients reveal deficient zinc (Cuajungco 1997).

- Low plasma levels of zinc bound to thymulin in Alzheimer’s patients suggests that impaired zinc metabolism contributes to pathogenesis (Cuajungco 1997).
Serum magnesium

- Dysregulation of magnesium transport into neurons may be a metabolic abnormality of Alzheimer’s disease in spite of adequate magnesium intake in the diet (Glick 1990)

- Checking serum magnesium levels does not provide useful information and magnesium supplementation is probably not an effective intervention
QEEG brain mapping in TBI, post-stroke and dementia

- Emerging research findings suggest that quantitative EEG and EEG (auditory or visual) evoked potentials are useful assessment tools for the evaluation of mild cognitive impairment, early stages of Alzheimer’s disease, and symptoms of inattention and cognitive slowing following stroke or traumatic brain injury.
QEEG with evoked potentials

- Visual evoked potentials (VEP) may reliably differentiate patients with mild cognitive impairment (MCI) or frank Alzheimer’s disease from intact subjects (Benvenuto 2002).
- Abnormal auditory evoked potentials (AEP’s) provide specific markers of milder forms of cognitive impairment.
- Slowing of global alpha activity below 8 Hz, increased theta activity, and a decrease in the alpha to theta ratio are typical findings in Alzheimer’s disease.
- Vascular dementia is associated with normal EEG activity interspersed with regions of focal slowing.
QEEG brain mapping with VEP and AER in dementia and mild cognitive impairment

- Pre-frontal cortical dis-inhibition is a typical finding in AD and other forms of dementia.
- A significant percentage of mildly cognitively impaired adults eventually progress to frank dementia.
- Early QEEG evaluation of at-risk individuals may facilitate early aggressive treatment.
Virtual reality testing environments

- Virtual reality tools may enhance the diagnostic accuracy of conventional neuropsychological assessment methods used to evaluate cognitive impairment in degenerative neurological disorders, stroke, developmental disorders and traumatic brain injury (TBI) (Rizzo 2000).

- Virtual performance testing environments will lead to the development of novel methods for earlier and more accurate assessment of cognitive impairment.
Prototype VR environments have yielded promising results for assessment of memory, attention, executive functioning, sensorimotor integration, and activities of daily living.

Combining VR technology with fMRI and other functional brain imaging technologies is adding to our understanding of the causes of moderate and severe forms of cognitive impairment.

The use of VR environments to assess neuropsychological functioning will lead to individualized rehabilitation strategies that more effectively address performance deficits on a case by case basis.
Possibly specific
assessment approaches
Serum vitamin levels

- Malnutrition due to malabsorption of essential dietary factors may play a significant causative role in dementia.

- Chronic brain deficiencies of vitamins and certain trace elements may be causes of neurofibrillary tangles and other neuropathological changes that manifest as Alzheimer’s Disease or other degenerative disorders (Evans 1987).

- Clear correlations have not been established between the risk of developing AD and abnormal low serum levels of vitamins or trace elements (Scileppi 1984).
Serum DHA levels

- Case studies suggest that abnormal low levels of serum DHA (an Omega-3 fatty acid) may be correlated with the severity of cognitive impairment in demented patients (Tully 2003).
- Other studies contradict this finding (Laurin 2003).
- Testing serum DHA levels in demented patients should not be a specific assessment approach pending more conclusive findings.
Integrative formulation

- **Includes**: biological, psychological, psychosocial, mind-body, and possible energy-information causes or meanings of cognitive impairment

- Suggests integrative treatment plan including biological, psychological, mind-body and energy-information treatments addressing identified causes or meanings
Conventional treatments of cognitive impairment

- Acetylcholinesterase inhibitors
- MAOIs
- Estrogen replacement therapy
- Naloxone
- On-going research: neuropeptides—somatostatin, vasopressin; vaccines, secretase inhibitors, NSAIDS, statins
Non-biological conventional treatments

- Address behavioral and cognitive symptoms of dementia, TBI, and stroke
  - Behavioral interventions
  - Environmental enrichment
  - Social support
Limitations of conventional treatments

- Acetylcholinesterase inhibitors are the only FDA approved treatments
  - Limited clinical benefits generally in early and moderate AD only
  - Few or no demonstrated benefits in other forms of severe cognitive impairment
- Toxicities and adverse effects
- High cost and limited availability
Substantiated non-conventional treatments

- Dietary changes
- Regular exercise
- Ginkgo biloba
- Huperzine A
- Phosphatidyl serine
- CDP-choline
- Idebenone
Dietary modification

- Moderate wine consumption, reduced saturated fats, and reduced total caloric intake are correlated with reduced risk of developing Alzheimer’s.

- Excess caloric intake and high fat intake promote formation of damaging free radicals that cause many neuropathological changes in the brain.

- A meta-analysis of findings from 18 community-wide studies concluded that the risk of Alzheimer’s disease increased linearly at a rate of 0.3% with every 100 calorie increase in daily intake (Grant 1997).
Dietary modification

- Average daily fat consumption was highly correlated with increased risk of developing dementia.

- Fish consumption was the **only specific dietary factor** associated with a measurable reduction in the risk of developing Alzheimer’s disease.

  - **Caution:** Consuming over 2-4 glasses/day increases the risk of dementia

  - **Caution:** Elderly people should be cautioned about excessive alcohol consumption
Regular physical exercise in healthy elderly individuals significantly reduces the risk of all categories of dementia.

*Note:* Exercise is probably not an effective intervention after the onset of dementia.

*Caution:* Individuals who have been sedentary or who have a chronic pain syndrome or a major medical illness should be evaluated by a physician before starting a rigorous exercise program.
Exercise

- Physical exercise increases levels of brain-derived neurotrophic factor (BDNF), probably enhancing neural plasticity and new synapse formation (Cotman 2000).

- Over 2,000 physically non-impaired men aged 71 to 93 years were followed with routine neurological assessments at two year intervals starting in 1991 (Abbott 2004). At the end of the study period, men who walked less than a quarter of a mile daily had an almost two-fold greater probability of being diagnosed with any category of dementia compared with men who walked at least two miles each day.
Exercise

- Bi-annual mailed surveys over ten years showed that elderly women aged 70 to 81 years who engaged in regular vigorous physical activity were significantly less likely to have been diagnosed with dementia compared to women with more sedentary lifestyles (Weuve 2004).

- A randomized controlled trial showed that regular daily exercise in moderately demented individuals receiving in-home care reduces depressed mood, but does not improve cognitive functioning (Teri 2003).
**Ginkgo biloba**

- Standardized extracts of *Ginkgo biloba* are effective treatments of mild to moderate symptoms of Alzheimer’s disease and vascular dementia.

- Most commercially available Ginkgo preparations are standardized to two bioactive constituents: 24% flavone glycosides, and 6% terpenoids.
Ginkgo biloba

- The flavonoid constituent functions as a strong antioxidant and is believed to have a general neuroprotective effect (Seif-El-Nasr 1995).

- Animal studies have confirmed that the terpenoid fraction antagonizes platelet activating factor (PAF), facilitating post-stroke recovery through vascular reperfusion by decreasing thrombosis and nerve cell death associated with cerebral ischemia (Smith 1996).
Ginkgo biloba

- Therapeutic doses range from 40mg three times daily up to 600mg/day in divided doses, and sustained improvement in cognitive functioning is usually observed following three month of treatment.
Ginkgo biloba—safety issues

- Informed consent should be documented in the patient's chart at the time of the consultation when *G. biloba* is discussed.

- Because of its strong anti-platelet aggregation factor (PAF) profile, *G. biloba* extract increases the risk of bleeding, and concurrent use should be avoided in patients taking aspirin, warfarin, heparin or other medications that interfere with platelet activity and increase bleeding time.

- *G. biloba* preparations should be discontinued at least two weeks prior to surgery.

- *G. biloba* preparations have been reported to result in elevation of hepatic enzymes.

- Case reports of possible serotonin syndrome when *G. biloba* is taken with SSRIs.

- Mild transient adverse effects include upset stomach, dizziness and headaches.
**Ginkgo biloba**

- Systematic reviews and meta-analyses of double blind studies show that standardized preparations of Ginkgo biloba in doses between 120 and 600mg/day taken over durations of several weeks to one year result in consistent modest improvements in memory, general cognitive functioning, and activities of daily living in mild to moderate cases of both Alzheimer’s dementia and multi-infarct dementia that are equivalent to improvements seen with donepezil™, a conventional cholinesterase inhibitor (Wong 1998; Emst 1999; Kanowski 1997; Oken 1998; Le Bars 1997).
Ginkgo biloba

- A more recent meta-analysis pointed out inconsistent findings of three trials based on more rigorous methodologies and commented on research design problems in those studies and earlier trials including the absence of standardized preparations and the use of different dementia rating scales across studies (Birks 2004).
Huperzine-A

- Huperzine-A (Huperzia serrata) may be more effective than conventional cholinesterase inhibitors for age-related memory loss and dementia.

- Important ingredient of many compound herbal formulas used in Chinese medicine to treat cognitive impairment related to normal aging.
Huperzine-A

- Huperzine-A reversibly inhibits acetylcholinesterase, and may also slow production of nitric oxide in the brain, reducing age-related neurotoxicity (Zhao 2002).

- Findings from animal studies suggest that Huperzine-A may be a more potent and more specific inhibitor of acetylcholinesterase compared to available conventional cholinesterase inhibitors.
Huperzine-A

- Controlled trials show consistent beneficial effects in both age-related memory loss (ie benign senescent forgetfulness) and Alzheimer’s disease at doses between 200 and 400 micrograms/day (Wang 1994; Zucker 1999).
- Infrequent adverse effects include transient dizziness, nausea and diarrhea.
Phosphatidyl serine

- Phosphatidyl serine is beneficial for age-related cognitive decline and Alzheimer’s disease.
- Phosphatidyl serine is one of the most important phospholipids in the brain and is an essential component of nerve cell membranes.
- Believed to enhanced fluidity of nerve cell membranes, indirectly increasing brain levels of many important neurotransmitters (Pepeu 1996).
Phosphatidyl serine

- The findings of many large double-blind placebo-controlled studies confirm improved global functioning and memory in Alzheimer’s disease and age-related cognitive decline at typical doses of 300mg/day (Cenacchi 1993; Crook 1991; Villardita 1987; Palmieri 1987; Amaducci 1988).
Phosphatidyl serine—sources and safety

- Brain-derived phosphatidyl serine is probably more effective than the soy-derived product (Hibbeln and Salem, 1995) possibly due to the higher content in DHA, an Omega-3 fatty acid.

- However, recent concerns have been raised over the risk of slow viruses in infected bovine tissue.
CDP-choline

- Cytidinediphosphocholine (CDP-choline) has beneficial effects on memory and behavior in post-stroke patients and possibly also in TBI and Alzheimer’s disease.

- Like Acetyl-L-carnitine, CDP-choline increases mitochondrial energy production and in many parts of the world is used to treat cognitive impairments resulting from neurodegenerative diseases.
CDP-choline

- CDP-choline 500mg to 1000mg/day improves overall energy metabolism in the brain, increases dopamine and norepinephrine (Secades 1995) and enhances short-term memory in Alzheimer’s patients (Alvarez 1997).

- Two Cochrane systematic reviews concluded that CDP-choline has consistent positive effects on the rate of recovery in post-Stroke patients, and in elderly individuals who are cognitively impaired due to cerebrovascular disease (Mitka 2002; Fioravanti 2004).
CDP-choline

- There is preliminary but promising evidence of a beneficial effect following traumatic brain injury (Spiers 1999).
- The findings of one small study suggest a possible effect of CDP-choline 1000mg/day in the early stages of Alzheimer’s disease (Alvarez 1999).
Idebenone

- Idebenone may be more effective than conventional treatments of Alzheimer’s disease

- Naturally occurring substance that is related to ubiquinone (coenzyme Q10), and like Co-Q10 also increases intra-cellular energy production in mitochondria.

- Idebenone 360mg/day may be more effective than tacrine (Gutzmann 2002) and possibly other cholinesterase inhibitors for cognitive impairment in mild to moderate cases of Alzheimer’s dementia (Gutzmann 1998).

- Preliminary evidence from animal studies suggests that combining Idebenone with vinpocetine accelerates recovery following Stroke (Ishihara 1989).
Provisional non-conventional treatments

- Dietary modification
- Acetyl-L-carnitine
- Kami-untan-to (KUT)
- Rhododendron rosea
- Choto-san
- DHEA
- B vitamins
Dietary modification

- Evidence from epidemiologic studies suggests that regular intake of foods rich in Omega-3 fatty acids may be inversely related to cognitive impairment or the rate of overall cognitive decline in non-demented elderly individuals. However, findings to date are inconclusive.

- Lifestyle choices confound findings.
Acetyl-L-carnitine

- Acetyl-L-carnitine (ALC) occurs naturally in the brain and liver.
- ALC may stabilize nerve cell membranes, stimulate synthesis of acetylcholine, and increase the efficiency of mitochondrial energy production.
- Commonly used to treat cognitive impairments related to dementia or other neurodegenerative diseases.
- Findings of human clinical trials are inconsistent (Pettegrew 2000).
Kami-Utan-Utan-to (KUT)

- Kami-Utan-Utan-to (KUT) is probably an effective treatment of age-related cognitive decline and early or mild Alzheimer’s disease.

- Compound herbal formula consisting of 13 different herbs used in Japanese traditional healing (Kampo) to treat cognitive impairment and frank dementia, as well as other psychiatric symptoms.

- KUT increases brain levels of both nerve growth factor (NGF) and choline acetyltransferase (ChAT), the enzyme that makes acetylcholine (Yabe 1996; Yabe 1996/1997).

- KUT protects against cognitive impairment due to thiamin deficiency in mice suggesting possible beneficial effects in delirium tremens and other syndromes of cognitive impairment related to thiamin deficiency (Nakagawasai 2004).
Rhodiola rosea

- Golden Root (Rhodiola rosea) may enhance memory in healthy adults and speed up recovery following traumatic brain injury.
- Subject of intense research interest in the former Soviet Union because of its use as an adaptogen or performance enhancer in athletes, soldiers and cosmonauts.
**Rhodiola rosea**

- Psychiatric benefits are probably related to increased dopamine, serotonin and norepinephrine (Petkov 1990), and include improved memory, increased mental stamina, and a general calming effect.

- Results from open studies suggest that Golden root 500mg improves overall mental performance and stamina in normal individuals (Spasov 2000), and may accelerate return to normal cognitive functioning following traumatic brain injury.
Choto-san

- **Choto-san**, a Kampo herbal formula, may be beneficial for vascular dementia.

- Choto-san is a kampo herbal formula used to treat symptoms associated with cerebro-vascular disease.

- Putative mechanisms of action include activating effects at serotonin and dopamine receptors, and strong antioxidant effects.

- Two controlled double-blind studies showed consistent beneficial effects in the treatment of vascular dementia, including improved global functioning, improved sleep, reduced psychotic symptoms, and improved cognitive functioning (Terasawa 1997; Shimada 1994).
DHEA

- DHEA is a precursor of testosterone and other hormones.
- Widely used in Europe and North America to self-treat declines in cognitive functioning associated with normal aging.
- Binds to both GABA receptors and NMDA receptors but it is unclear whether these receptor affinities are related to its putative cognitive enhancing role (Friess 1995).
Preliminary evidence from anecdotal reports and pilot studies suggests that DHEA 25-50mg/day improves memory and enhances general cognitive functioning in healthy adults (Wolkowitz 1997).

Research findings are inconsistent, and negative findings have been reported at doses lower than 90mg/day (Wolf 1997).

DHEA 200mg/day may improve symptoms of cognitive impairment in multi-infarct dementia (Azuma 1999).

Mild insomnia is an infrequent side effect of DHEA. There is no evidence that DHEA replacement doses of 25 to 50mg/day increase the risk of prostate cancer in middle aged men.
B vitamins

- Folic acid, B-12 and thiamin improve cognitive functioning in some cases of dementia
- Many B vitamins are essential enzyme co-factors in the synthesis of neurotransmitters.
- Low serum levels of folic acid, niacin and thiamin are associated with cognitive impairment in general (Hassing 1999).
- A diet low in folic acid and B12 leads to elevated blood levels of homocysteine and decreased synthesis of S-adenosyl methionine (SAMe), resulting in reduced synthesis of several neurotransmitters critical for normal cognitive functioning.
B vitamins

- Chronic dietary deficiencies of folate and B-12 ultimately manifest in moderate to severe degrees of cognitive impairment.

- Clinical trials results suggest that healthy adults who take a B vitamin supplement experience improvements in overall cognitive functioning (Benton 1997).
B vitamins

- A daily B vitamin supplement may slow deterioration of cognitive functioning in mild to moderate dementia (Pelka 1995).

- Findings from case studies and controlled trials suggest that a large daily dose of folic acid reduces the severity of dementia in some cases.

- In a double-blind placebo-controlled study depressed demented patients treated with 5-MTHF (a form of folate) 50mg/day experienced significant improvements in both mood and memory after four weeks of therapy (Passeri 1993).
Possibly effective treatments

- Ashwagondha
- Lemon balm (*Melissa officinalis*)
- Common sage (*Salvia officinalis*)
- Ginseng
- Alpha-lipoic acid
- Centrophenoxine (CPH)
- Trasina™ and Mentat™
- DHED
- Regular massage therapy
- Bright light exposure
- EEG biofeedback
- Qigong
Ashwagondha

- Ashwagondha 50mg/kg may improve short term memory, long term memory and executive functioning in cognitively impaired individuals (Bhattacharya 1995)

- **Note:** few human trials on Ashwagondha have been published
Lemon balm and Sage

- Extracts of Lemon balm and Common sage (60 drops/day) may slow the rate of decline in Alzheimer’s disease (Akhondzadeh 2003)
- Teas made of both herbs are traditionally used to enhance general cognitive functioning and to treat dementia
Panax ginseng

- Ginseng may enhance memory and overall cognitive performance in healthy adults (Sorensen 1996; Vogler 1999)

- **Note:** some negative trials with Ginseng, and at least one reported case of mania induction
**Alpha-lipoic acid (ALA)**

- Animal studies suggest that ALA may enhance recovery following stroke. Human case reports suggest that ALA may accelerate return of cognitive function after traumatic brain injury.

- **Note:** A 2004 Cochrane review identified no controlled trials; findings on ALA in dementia are largely from case studies (Sauer 2004).
Centrophenoxine

- CPH may improve global functioning and cognitive performance in Alzheimer’s disease (Pek 1989)
- Benefits of CPH are possibly enhanced when combined with vitamins.
- **Note**: beneficial findings for CPH in dementia are still very preliminary
Trasina™ and Mentat™

- Trasina™ and Mentat™ are compound herbal formulas used in Ayurvedic medicine to treat symptoms that resemble Alzheimer’s disease and age-related cognitive impairment (Battacharya 1995, 1997).

- Trasina has beneficial effects on experimental animal models of Alzheimer’s disease (Bhattacharya 1997).
Dehydroevodiamine (DHED)

- Dehydroevodiamine (DHED) may be more effective than cholinesterase inhibitors in Alzheimer’s disease.
- DHED is a biologically active constituent of *Evodia rutaecarpa*, a plant used in traditional Korean medicine, and may prove to be more effective in the treatment of age-related cognitive decline and Alzheimer’s disease than cholinesterase inhibitors (Park 1996).
Massage therapy

- Regular massage therapy may reduce agitated behavior in demented individuals
- Skilled nursing homes frequently provide regular massage therapy to demented residents.
- Anecdotal evidence of calming effects and reduced agitation following massage.
- Findings from open trials suggest that massage decreases agitated behavior in demented individuals (Snyder 1995; Fraser 1993).
Bright light exposure therapy

- Regular exposure to bright light may improve sleep and reduce sun-downing in demented patients.
- Bright light exposure may improve sleep and reduce sun-downing in Alzheimer’s patients (Satlin 1992).
- However, findings of a Cochrane meta-analysis of studies on bright light therapy in the management of sleep, behavior and mood in demented individuals were inconclusive (Forbes 2004).
EEG biofeedback training may enhance recovery following stroke and traumatic brain injury.

EEG biofeedback training, also called neurotherapy, employs light or sound as feedback with the goal of entraining changes in brain electrical activity that result in improved cognitive functioning.
EEG biofeedback

- Findings of a case report suggest that EEG biofeedback training improves the speed of cognitive rehabilitation following stroke, including enhanced word finding, attention, speech and concentration (Rozelle 1995).

- Findings from one small open study suggest that EEG biofeedback training may improve memory following traumatic brain injury (TBI) (Thornton 2000).
Qigong

- Regular Qigong practice may normalize EEG activity and slow the rate of cognitive decline in Alzheimer’s disease.
- 3-dimensional positron emission tomography (PET) and EEG were used to examine relationships between brain electrical activity and changes in regional cerebral blood flow in a qigong practitioner (Manabu 1996).
Qigong

- Findings included significant increases in high frequency (alpha and beta) EEG domains and reduced slow frequency (delta) activity following qigong.

- Enhanced beta activity in the frontal lobes following qigong practice was correlated with increased cerebral blood flow in the same region and relatively decreased regional blood flow in posterior brain regions.
Summary

- Preliminary findings suggest that many non-conventional biological and energy-information therapies reduce the severity or rate of cognitive decline, and improve global functioning in dementia, post-stroke recovery and traumatic brain injury.

- Larger and better studies are needed to further substantiate efficacy claims.
resources


Rizzo, A Wiederhold B. Applications and issues for the use of virtual reality technology for cognitive-behavioral/neuropsychological assessment and intervention; A workshop at the 34th annual convention of the Assoc. for Advancement of behavior therapy New Orleans, Louisiana U.S. Nov 16-20, 2000.


Weuve J Kang J Manson J Breteler M Ware J Grodstein F Physical activity, including walking, and cognitive function in older women JAMA 2004;292:12 1454-1461.


