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Aquatic Therapy in Early & End-Stage Dementia

Learning Objectives

1) Understand the response of the end stage Alzheimer’s patient from the pre-published case review on Aquatic Therapy and End-Stage Dementia

2) Recognize the importance of an “enriched environment” and its crucial relationship to the continuum of care for all client populations.

3) Use clinical reasoning to defend the physiological, behavioral and cognitive responses of immersion in patients with moderate to severe cognitive impairments.

4) Identify aquatic treatment options for the minimally responsive patient population with focus on modifications of methods such as Halliwick, Water Specific Therapy, Watsu and Sensory Integration.
Aquatic Therapy in Early & End-Stage Dementia

Stacy Lynch, PTA, CLT, CPT, Aq, GAq
Director of Programming
Inertia Therapy Services

International AquaCongress
Comprehensive Aquatic Therapy put into Practice

Querétaro, Mexico
Saturday, October 29, 2016
Hippocrates
~ 400 B.C.

Galen
~ 200 A.D.

Kneipp
~ 19th Century
Hippocrates
~ 400 B.C.

“Water Cure”

Kneipp
~ 19th Century
Cerebral Palsy
Spastic conditions
Aquatic Therapy in Early & End-Stage Dementia

Stacy Lynch, PTA, CLT, CPT, Aq,Gaq
Director of Programming
Inertia Therapy Services
Aquatic Therapy in End-Stage Dementia

Stacy Lynch, PTA, CLT, CPT, Aq, GAq
Director of Programming
Inertia Therapy Services
Aquatic Therapy and Alzheimer’s Disease

Kent W. Myers, MD\textsuperscript{1,2} • Dina Capek, RN\textsuperscript{2} • Holly Shill, MD\textsuperscript{3} • Marwan Sabbagh, MD\textsuperscript{3,4}
Increased Responsiveness

Autism Spectrum Disorder
Cerebral Vascular Accident
Traumatic Brain Injury
Post Traumatic Stress Disorder
Dementia
DEMENTIA REHAB?

“We don’t treat dementia!?”
DEMENTIA REHAB?
The Link Between Physical Activity and Cognitive Dysfunction in Alzheimer Disease.

The Link Between Physical Activity and Cognitive Dysfunction in Alzheimer Disease

Cristy Phillips, Mehmet Akif Baktir, Devsmita Das, Bill Lin, Ahmad Salehi

Alzheimer disease (AD) is a primary cause of cognitive dysfunction in the elderly population worldwide. Despite the allocation of enormous amounts of funding and resources to studying this brain disorder, there are no effective pharmacological treatments for reducing the severity of pathology and restoring cognitive function in affected people. Recent reports on the failure of multiple clinical trials for AD have highlighted the need to diversify further the search for new therapeutic strategies for cognitive dysfunction. Thus, studies detailing the neuroprotective effects of physical activity (PA) on the brain in AD were reviewed, and mechanisms by which PA might mitigate AD-related cognitive decline were explored. A MEDLINE database search was used to generate a list of studies conducted between January 2007 and September 2014 (n=394). These studies, along with key references, were screened to identify those that assessed the effects of PA on AD-related biomarkers and cognitive function. The search was not limited on the basis of intensity, frequency, duration, or mode of activity. However, studies in which PA was combined with another intervention (eg, diet, pharmacotherapeutics, ovariectomy, cognitive training, behavioral therapy), and studies not written in English were excluded. Thirty-eight animal and human studies met entry criteria. Most of the studies suggested that PA attenuates neuropathology and positively affects cognitive function in AD. Although the literature lacked sufficient evidence to support precise PA guidelines, convergent evidence does suggest that the incorporation of regular PA into daily routines mitigates AD-related symptoms, especially when deployed earlier in the disease process. Here the protocols used to alter the progression of AD-related neuropathology and cognitive decline are highlighted, and the implications for physical therapist practice are discussed.

C. Phillips, PT, EdD, Department of Physical Therapy, Arkansas State University, PO Box 910, Jonesboro, AR 72467 (USA). Address all correspondence to Dr Phillips at: cphillips@astate.edu.

M. Akif Baktir, MD, Department of Physiology, School of Medicine, Erciyes University, Kayseri, Turkey; VA Palo Alto Health Care System, Palo Alto, California; and Cardiovascular Medicine, Department of Medicine, School of Medicine, Stanford University, Palo Alto, California.

D. Das, MD, MPH, VA Palo Alto Health Care System and Department of Psychiatry and Behavioral Sciences, School of Medicine, Stanford University.

B. Lin, BS, VA Palo Alto Health Care System.

A. Salehi, MD, PhD, VA Palo Alto Health Care System and Department of Psychiatry and Behavioral Sciences, School of Medicine, Stanford University.


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Alzheimer disease (AD) is a chronic, neurodegenerative disorder that adversely affects neurons in the brain, ultimately resulting in loss of memory and language function, and dependence on caregivers. The strongest risk factor for AD is aging, and the risk doubles every 5 years after the age of 65 years. Increasing population, longevity, and economic prosperity have contributed to concern about a dementia epidemic in the aging population. Currently, 26 million people are affected by AD worldwide, the number of affected people is expected to approach 306 million by the year 2050, potentially burdening serious clinical, social, ethical, and economic problems.

The gradual decline in brain functioning caused by AD has been associated with several characteristic features, including changes in synaptic number and function, neurogenesis, and neurofibrillary tangles (NFTs), and amyloid plaques. The progression of these features is considered critical to the development of impairments in cognition, defined as the unique combination of attention, learning, memory, language, visuospatial skills, and executive function. Notably, many pathological features precede AD-related cognitive decline. However, despite the evidence for potential early markers, currently available pharmacotherapies (e.g., donepezil HCl [Aricept, Eisai Co Ltd, Tokyo, Japan], galantamine HBr [Razadyne, Janssen Pharmaceuticals Inc, Hormeg, Belgium],rivastigmine tartrate [Exelon, Novartis Pharmaceuticals Corp, Basel, Switzerland], and memantine HCl [Namenda, Forest Laboratories Inc, New York, New York]) offer transient symptomatic relief only. Given the lack of disease-modifying options, it is imperative to diversify the search for feasible and effective interventions. This realization has prompted great interest in the use of physical activity (PA) to attenuate the severity of neuropsychological features associated with cognitive decline in AD.

Physical activity is defined as a subcategory of PA that connotes purposeful, planned, and structured endeavors undertaken to improve skill or physical fitness level. Convergent evidence suggests that PA can alter the progression of AD-related neuropsychology and cognitive decline, leading to the incorporation of PA into basic clinical management protocols for AD. Because it is important that physical therapists understand the ways in which PA can be beneficial, from both self-education and patient education perspectives, the aims of this review are to discuss potential avenues for exploring the putative mechanisms by which PA might mediate these features, review protocols used to effectively implement PA in both animal and clinical studies, and highlight implications for physical therapists.

Pathological Features of AD

Amyloid plaques and NFTs are characteristic features of AD. Amyloid plaques comprise a potentially toxic protein formed by the aggregation of beta-amyloid (Aβ). In AD, the plaques are heterogeneously interposed throughout the brain. Neurofibrillary tangles are abnormal forms of twisted protein fibers found inside neurons comprising insoluble tau. For many years, it has been thought that plaques and NFTs may cause the disease, called amyloid beta (Aβ) theory. However, such an oversimplified concept has yielded to contemporary conceptualizations in which AD is viewed as a multifactorial disease arising from several abnormal compex features and processes (Fig. 1). We systematically reviewed how PA might be deployed as an intervention to address significant features of AD and, in turn, mitigate cognitive decline. Indeed, this discussion is integrated into an analysis of data derived from both rodent and human studies. This "mixed methods" approach is necessary given the obvious limits for experimental manipulation of brain tissue in living humans. Admittedly, evidence from rodents is not a substitute for human studies. Rather, the role of rodent investigations is to generate preclinical data to expedite the pace of discovery, bolster epidemiological research, and bridge the temporal gap between knowledge creation and clinical trials. With this caveat in mind, we present a summary of our preclinical data suggesting that PA benefits brain function and cognition in AD.

This review was designed and conducted in accordance with PRISMA (Preferred Reporting Items for Systematic Reviews and Meta-Analyses) guidelines.

Search Procedures

In an attempt to capture relevant data, we conducted a computer-based search of MEDLINE and performed manual searches of key references to identify studies that investigated the effect of PA on the progression of AD and putative mechanisms by which PA might mediate cognitive decline in AD.

Key word search criteria combined the terms "Alzheimer disease" and "exercise." This search was used to generate a list of relevant studies conducted between January 2007 and September 2014 (n=364).
<table>
<thead>
<tr>
<th>Rodent Model</th>
<th>Targeted Gene</th>
<th>Age (mo)</th>
<th>Modality</th>
<th>Frequency and Duration</th>
<th>Cognitive Assessment and Outcome</th>
<th>AD-Related Neurobiological Outcome</th>
<th>Study</th>
</tr>
</thead>
<tbody>
<tr>
<td>ICV injection of Aβ</td>
<td>NA</td>
<td>2–4</td>
<td>Resistance exercise (swimming with tail weights)</td>
<td>5 d/wk × 8 wk</td>
<td>↑ Exploratory behavior (open field)</td>
<td>↓ Inflammatory markers</td>
<td>Souza et al, 85, 2013</td>
</tr>
<tr>
<td>SAMP8</td>
<td>NA</td>
<td>10</td>
<td>Running wheel</td>
<td>Free access × 24 wk</td>
<td>NA</td>
<td>↑ BDNF gene expression</td>
<td>Alvarez-Lopez et al, 117, 2013</td>
</tr>
<tr>
<td>APP695/PS1A246</td>
<td>APP/PS1</td>
<td>24</td>
<td>Treadmill</td>
<td>5 d/wk × 5 wk</td>
<td>↑ Spatial learning (MWM)</td>
<td>↓ Aβ deposition</td>
<td>Ke et al, 21, 2011</td>
</tr>
<tr>
<td>NSE/hTAU23</td>
<td>TAU</td>
<td>16</td>
<td>Treadmill</td>
<td>1 h/d × 5 d/wk × 12 wk</td>
<td>NA</td>
<td>↓ Inflammatory markers</td>
<td>Leem et al, 84, 2011</td>
</tr>
<tr>
<td>ICV injection of streptozotocin</td>
<td>NA</td>
<td>3</td>
<td>Treadmill</td>
<td>Daily × 5 d/wk × 5 wk</td>
<td>↑ Spatial learning (MWM)</td>
<td>↑ Antioxidant molecules; ↓ oxidative stress</td>
<td>Rodrigues et al, 118, 2010</td>
</tr>
<tr>
<td>APP23</td>
<td>APP</td>
<td>6 and 18</td>
<td>Running wheel</td>
<td>Free access × 10 d</td>
<td>NA</td>
<td>↓ Aβ deposition in 18-mo group; ↑ neurogenesis in 18-mo group</td>
<td>Mirochnic et al, 119, 2009</td>
</tr>
<tr>
<td>ICV injection of Aβ23-35</td>
<td>NA</td>
<td>2</td>
<td>Running wheel</td>
<td>Free access × 12 d</td>
<td>↑ Spatial learning (Y maze)</td>
<td>↑ Synaptogenesis</td>
<td>Wang et al, 120, 2013</td>
</tr>
<tr>
<td>APP/PS1</td>
<td>APP/PS1</td>
<td>3</td>
<td>Treadmill</td>
<td>30 min/d × 5 d/wk × 20 wk</td>
<td>NA</td>
<td>↓ Aβ deposition; ↓ Tau phosphorylation</td>
<td>Liu et al, 121, 2013</td>
</tr>
<tr>
<td>PS2 mutant</td>
<td>PS2</td>
<td>24</td>
<td>Treadmill</td>
<td>60 min/d × 5 d/wk × 12 wk</td>
<td>↑ Spatial learning (MWM)</td>
<td>↓ Aβ deposition; ↓ Aβ42; ↓ inflammatory markers</td>
<td>Kang et al, 20, 2013</td>
</tr>
<tr>
<td>ICV injection of Aβ1-42</td>
<td>NA</td>
<td>Adult (age not specified)</td>
<td>Treadmill</td>
<td>2 sessions/d (15 min each) × 2/wk progressing to 3 sessions/d × 5 d × 2 wk</td>
<td>↑ Spatial learning (RAWM)</td>
<td>↑ BDNF protein</td>
<td>Dao et al, 122, 2013</td>
</tr>
<tr>
<td>CRND8</td>
<td>APP</td>
<td>2.5</td>
<td>Running wheel</td>
<td>Free access × 10 wk</td>
<td>↔ Spatial learning (Barnes maze); ↓ stereotypic behavior (eg, jumping, climbing, and bar chewing)</td>
<td>↔ Aβ deposition; ↔ stress hormones</td>
<td>Richter et al, 123, 2008</td>
</tr>
<tr>
<td>Tg2576</td>
<td>APP</td>
<td>15–19</td>
<td>Running wheel</td>
<td>Free access × 3 wk</td>
<td>↑ Spatial learning (RAWM)</td>
<td>↔ Aβ deposition; ↑ levels of immune system–related protective molecules</td>
<td>Parachikova et al, 22, 2008</td>
</tr>
<tr>
<td>Damaged cholinergic neurons</td>
<td>NA</td>
<td>Age not specified</td>
<td>Treadmill</td>
<td>60 min/d × 7 d/wk × 8 wk</td>
<td>↑ Spatial learning (MWM)</td>
<td>NA</td>
<td>Hoveida et al, 124, 2011</td>
</tr>
<tr>
<td>Tg2576</td>
<td>APP</td>
<td>5</td>
<td>Running wheel</td>
<td>60 min/d × 5 d/wk × 12 wk</td>
<td>↓ Aβ plaques</td>
<td>↓ Aβ plaques</td>
<td>Yuede et al, 125, 2009</td>
</tr>
<tr>
<td>3×AD</td>
<td>APP, TAU, PS1</td>
<td>1 and 9</td>
<td>Running wheel</td>
<td>1 mo old: free access × 4 or 36 wk; 9 mo old: free access × 44 wk</td>
<td>↔ Spatial learning (MWM)</td>
<td>↔ BDNF; ↔ Aβ; ↔ pTau</td>
<td>Marlatt et al, 52, 2013</td>
</tr>
</tbody>
</table>

(Continued)
Table 2. Effects of Physical Activity on Cognitive Outcomes in People With Alzheimer Disease (AD)\(^a\)

<table>
<thead>
<tr>
<th>AD Intervention</th>
<th>Sample (n)</th>
<th>Dementia Severity as Determined by MMSE</th>
<th>Age (y)</th>
<th>Sex</th>
<th>Modality</th>
<th>Frequency and Duration</th>
<th>Assessment</th>
<th>Study</th>
</tr>
</thead>
<tbody>
<tr>
<td>Randomized control</td>
<td>14</td>
<td>14.3</td>
<td>76.8</td>
<td>5 men, 9 women</td>
<td>Walking, strengthening, flexibility, balance training, agility</td>
<td>1-h session × 3 sessions/ wk × 24 wk</td>
<td>Severity of sleep disturbances</td>
<td>Nascimento et al,(^98) 2014</td>
</tr>
<tr>
<td>Randomized control</td>
<td>32</td>
<td>&lt;23</td>
<td>81.8</td>
<td>4 men, 12 women</td>
<td>Walking</td>
<td>1-h session × 3 sessions/ wk × 15 wk</td>
<td>Positive correlation with ERFC</td>
<td>Kemoun et al,(^101) 2010</td>
</tr>
<tr>
<td>Randomized control</td>
<td>27</td>
<td>≥10</td>
<td>74–76.5</td>
<td>8 men, 19 women</td>
<td>Walking</td>
<td>Single session daily × 12 wk</td>
<td>MMSE</td>
<td>Steinberg et al,(^105) 2009</td>
</tr>
<tr>
<td>Randomized control</td>
<td>21</td>
<td>5–15</td>
<td>84.0</td>
<td>30 men, 5 women</td>
<td>Walking</td>
<td>30-min session × 4 sessions/ wk × 24 wk</td>
<td>Rate of decline in MMSE</td>
<td>Venturelli et al,(^106) 2011</td>
</tr>
<tr>
<td>Randomized control</td>
<td>40</td>
<td>10–28</td>
<td>51–89</td>
<td>16 men, 24 women</td>
<td>Walking, strengthening, balance training</td>
<td>1-h session/wk × 6 wk</td>
<td>MMSE; ADAS-cog</td>
<td>Vreugdenhil et al,(^102) 2012</td>
</tr>
<tr>
<td>Randomized control</td>
<td>27</td>
<td>12–29</td>
<td>72.0</td>
<td>11 men, 16 women</td>
<td>Brain Gym Exercise Protocol</td>
<td>1-h session/wk × 6 wk</td>
<td>Cantab-Expedio</td>
<td>Yaquez et al,(^103) 2011</td>
</tr>
<tr>
<td>Nonrandomized control</td>
<td>30</td>
<td>19.4</td>
<td>77.0</td>
<td>6 men, 24 women</td>
<td>Aerobic training, strengthening, flexibility, balance training</td>
<td>1-h session × 5 sessions/ wk × 16 wk</td>
<td>MCE</td>
<td>de Andrade et al,(^104) 2013</td>
</tr>
<tr>
<td>Randomized control</td>
<td>27</td>
<td>19</td>
<td>77.5</td>
<td>Not reported</td>
<td>Aerobic training, strengthening, flexibility, balance training, agility, cognitive activities</td>
<td>1-h session × 3 sessions/ wk × 16 wk</td>
<td>Executive functions, including FAB</td>
<td>Coelho et al,(^72) 2013</td>
</tr>
<tr>
<td>Randomized control</td>
<td>21</td>
<td>Not applicable</td>
<td>76.0</td>
<td>Not reported</td>
<td>Treadmill</td>
<td>1 session starting at 4 km/h with slope of 3% and increasing slope by 1% every 3 min until heart rate reached 85% of maximum capacity</td>
<td>Plasma BDNF in controls and cases with AD</td>
<td>Coelho et al,(^129) 2014</td>
</tr>
</tbody>
</table>

\(^a\) Among the cognitive assessments used in these studies were Cantab-Expedio, which measures sustained attention and visual memory; the Montreal Cognitive Assessment (MCE), which assesses frontal cognitive function, attention, and language; and the Mini-Mental State Examination (MMSE), which is an objective measure of cognitive function that examines several parameters (including orientation with regard to time and place, registration [eg, repeating prompts], attention and calculation [eg, backward spelling task], recall, language, and repetition [eg, verbalizing names of objects and complex commands]). Mini-Mental State Examination scores ranging from 0 to 9 indicate severe cognitive impairment, those ranging from 10 to 18 indicate moderate impairment, and those ranging from 19 to 24 suggest mild cognitive impairment. The Brain Gym Exercise Protocol consists of fine motor, balance, and eye-hand coordination activities. ERFC=Rapid Evaluation of Cognitive Functions Test, ADAS-cog=Alzheimer’s Disease Assessment Scale–cognitive subscale, FAB=Frontal Assessment Battery, BDNF=brain-derived neurotrophic factor.
<table>
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<td>Souza et al, 2013</td>
</tr>
<tr>
<td></td>
<td>(swimming with tail weights)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Cognitive Assessment and Outcome</strong></td>
<td></td>
<td></td>
<td>↑ Exploratory behavior (open field)</td>
<td></td>
</tr>
</tbody>
</table>
Enriched Environment
1947 - Donald O. Hebb
Rats raised as pets solved problems better than rats raised in cages

1960 - Harry Harlow
Recognized changes in the behavioral and cognitive development of Rhesus monkeys due to maternal and social deprivation

1960 - Mark Rosenzweig
Began studying cognitive benefits of using toys, ladders, and tunnels over being isolated in cages

1974 - Rudolf Jaenisch
First genetically engineered mouse
Physical Activity ↓ Social Isolation

< Cause > < Effect >
Physical Activity ↓ Social Isolation

< Effect > < Cause >
Dr. Alois Alzheimer

1864-1915
Mrs. Auguste D

Auguste Deter
“I have lost myself!”

Auguste Deter
"I have lost myself!"

Vicki
VIDEO: Case Review Intro
Dementia

The behavioral component
Oliver Augustine Adrian
Aquarobics group’s response:

Significant increases in:
Cardio-respiratory endurance, muscle endurance, flexibility, balance, nimbleness.

Significant reductions in:
Tension, Depression, Anger, Fatigue, and Total Mood Disturbance (TMD)

Functional Assessment Staging of Alzheimer’s disease (FAST) Moderate to severe dementia range

Psychological Well-Being in Cognitively Impaired Persons Scale (PW-BCIP)
Patients showed a statistically significant increase in their psychological well-being

Revised Memory and Behavior Problems Checklist (RMBPC)
Significant decrease in psychological symptoms of dementia towards other residents as well as staff and family
Family Involvement & Compliance
Family Involvement & Compliance
VIDEO: Case Review Aquatic Enrichment
Becker, Bruce MD & Cole, Andrew MD (2011)

Comprehensive Aquatic Therapy

3rd edition.

Pullman, WA

Washington State University Publishing
Circulatory System

- 60% in thoracic blood volume
- 35% in cardiac stroke volume

**REST** = Land-based cardiovascular activity
Structure of Endothelial Nitric Oxide Synthase. Delker SL, Xue F, Li H, Jamal J, Silverman RB, Poulos TL.
Nitric Oxide
Nitric Oxide
Land Treadmill participants had no increase in eNOS

Aquatic Treadmill participants had an average of 31% increase in eNOS 24 hours following their sessions.

Aquatic treadmill training reduces blood pressure reactivity to physical stress.
doi: 10.1161/CIRCRESAHA.110.234450
Exercise = \[\uparrow\] Nitric Oxide
Neurotrophic Factors

Nitric Oxide = \[\downarrow\] Amyloid

Aquatic Exercise =

\[\uparrow\uparrow\uparrow\uparrow\] Nitric Oxide
Halliwick/Water Specific Therapy

Applications to the severe dementia patient
VIDEO:
Case Review
HAT/WST
Modifications
Halliwick/Water Specific Therapy

Progression and carryover with the severe dementia patient
VIDEO:
Case Review
Patient Response