There are over 11 million silent strokes in the United States every year, affecting up to 30% of the population over age 65. Silent strokes affect areas of the brain involved with thinking, but not areas of the brain involved with motor functions. Thus, the physical symptoms are not so problematic as a traditional stroke; however, damage to the brain does occur and can be seen as a trail of scar-tissue damage in brain regions with current imaging technology. A new study* shows that silent strokes are a significant factor in memory loss. (*See next page)

This new study involving 658 patients ages 65 and older, without dementia or Alzheimer’s, used high-resolution MRI to study their brain for the signs of silent stroke as well as to measure the size of key brain parts such as the hippocampus, which is known for its vital role in memory and learning. Additionally, the participants were given numerous types of cognitive and memory tests, and those results were compared to the MRI findings.

The researchers found evidence of silent stroke in 174 of the participants (26%). As expected, a smaller sized hippocampus was correlated to poorer scores on memory. And those with smaller hippocampus were more likely to have had a silent stroke. However, even when the size of the hippocampus was accounted for as a variable, those with silent stroke had worse memory. This means that both the size of the hippocampus and the condition of the vascular system within the brain predicted memory problems.

It is noteworthy that a person does not go from optimal brain health to silent stroke, like night and day. Rather, it is a sliding scale of thousands of shades of gray. It is common, especially under high stress, to have mini regions of your brain deprived of oxygen and to have micro-strokes that would not show up as accumulated damage reflecting a larger silent stroke. Many such micro-strokes occurring over time would lead to decreased mental capacity, loss of computational ability, and memory loss. I would expect such problems would be far more common than not and will be easy to see as imaging technology continues to improve.

The key finding of this study is that memory issues relate to more than what has been considered the key memory structure in your brain, the hippocampus. This is consistent with other emerging brain science showing that as brain cell damage accumulates brain speed slows down, brain plasticity is compromised, and cognitive ability is reduced. For example, inflammation from micro-strokes in the brain’s circulatory system would be highly inflammatory to glial cells and result in impaired function.

Thankfully, the new science is also showing that nutrients can protect your brain from stroke of all types. Top choices include DHA, tocoptrienols, grape seed extract, resveratrol, lipoic acid, camosine, acetyl-l-carnitine, pantethine, blueberries, curcumin, vitamin C, folic acid – just to name a few. If these nutrients are present in your brain and there is a problem, there is less damage and faster recovery. It is reasonable to assume that the same nutrients help prevent such problems in the first place.

In addition to nutrients, aerobic exercise, including walking, has been shown to boost brain size as well as increase oxygen flow within your brain, not to mention helping to stimulate brain rejuvenation. With an aging baby boomer population facing an epidemic of dementia and Alzheimer’s, this information is of the utmost value to any person wishing to not go down the wrong path.

http://www.wellnessresources.com/health/articles/baby_boomers_beware_silent_strokes_linked_to_memory_problems/
Memory after Silent Stroke: Hippocampus and Infarcts both Matter

Study Title:
Memory after Silent Stroke: Hippocampus and Infarcts both Matter

Study Abstract:

Objective:
Memory decline commonly occurs among elderly individuals. This observation is often attributed to early neurodegenerative changes in the hippocampus and related brain regions. However, the contribution of vascular lesions, such as brain infarcts, to hippocampal integrity and age-associated memory decline remains unclear.

Methods:
We studied 658 elderly participants without dementia from a prospective, community-based study on aging and dementia who received high-resolution structural MRI. Cortical and subcortical infarcts were identified, and hippocampal and relative brain volumes were calculated following standard protocols. Summary scores reflecting performance on tasks of memory, language, processing speed, and visuospatial function were derived from a comprehensive neuropsychological battery. We used multiple regression analyses to relate cortical and subcortical infarcts, hippocampal and relative brain volume, to measures of cognitive performance in domains of memory, language, processing speed, and visuospatial ability.

Results:
Presence of brain infarcts was associated with a smaller hippocampus. Smaller hippocampus volume was associated with poorer memory specifically. Brain infarcts were associated with poorer memory and cognitive performance in all other domains, which was independent of hippocampus volume.

Conclusions:
Both hippocampal volume and brain infarcts independently contribute to memory performance in elderly individuals without dementia. Given that age-associated neurodegenerative conditions, such as Alzheimer disease, are defined primarily by impairment in memory, these findings have clinical implications for prevention and for identification of pathogenic factors associated with disease symptomatology.

Study Information:

Taub Institute for Research on Alzheimer's Disease and the Aging Brain at Columbia University Medical Center in New York

http://www.wellnessresources.com/studies/memory_after_silent_stroke_hippocampus_and_infarcts_both_matter