**Nucleus basalis of Meynert**

**Last updated: September 5, 2017**

**Articles to check**


**Nucleus basalis of Meynert**

- group of neurons in substantia innominata of basal forebrain which has wide projections to neocortex and is rich in acetylcholine and choline acetyltransferase.

**ATLAS OF THE NUCLEUS BASALIS OF MEYNERT**


**Hellyer G, Celano K, Cairns MJ (1993): Quantitation and three-dimensional reconstruction of ChAT nuclei in the human basal forebrain. Synapse (UK) 11, 1–10.**

**Gratwicke et al. / Neuroscience and Biobehavioral Reviews 37 (2013):**

**NBH is aflat, nearly horizontal structure extending from the olfactory tubercle anteriorly to the level of the uncal hippocampus at its most caudal extent, spanning a distance of 13–14 mm in the sagittal plane. It reaches its greatest cross-sectional diameter under the thalamus commissure in a region known as the substantia innominata, with a medio-lateral width of 16–18 mm (Mesulam and Geula, 1988). In its anterior portion the nucleus is limited inferiorly by the choroidal limb of the nucleus of the diagonal band of Broca, superb-medially by the ventral globus pallidus, and supero-laterally by the lateral extension of the anterior commissure (Figs. 1 and 2). In its posterior portion it abuts the ansa lenticularis superiorly, the puta-mena laterally, the posterior tip of the amygdala inferiorly, and the optic tract medially (Fig. 2) (Mesulam and Geula, 1988; Rossor et al., 1982).**

**Gratwicke et al. / Neuroscience and Biobehavioral Reviews 37 (2013):**

**Representation of the major anatomical structures and fibre tracts related to the nucleus basalis of Meynert (CH4, in red) in the human basal forebrain region. Overlying structures have been lifted upward to expose the NBH, as indicated by dashed grey lines. The major subregions of the NBH are shown within the nucleus; their approximate anatomical boundaries are indicated by dashed black lines. The diagram is based on anatomical observations in the human brain by Mesulam and Geula (1988) and Rossor et al. (1982). A = amygdala, AC = anterior commissure (lateral aspect), AL = ansa lenticularis, CH3 = horizontal limb nucleus of the diagonal band of Broca (cholinergic cell group 3 of the basal forebrain), GPs = globus pallidus internus; GPe = globus pallidus externus; OT = optic tract; P = putamen; uH = uncal hippocampus. Subregions of NBH as described in the main text, NSP = nucleus subparataudal.**

**Nucleus basalis of Meynert**

**ANATOMY**


**Hellyer G, Celano K, Cairns MJ (1993): Quantitation and three-dimensional reconstruction of ChAT nuclei in the human basal forebrain. Synapse (UK) 11, 1–10.**

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The efferent connectivity between individual subsectors of NBM and cortical areas displays a topographic specificity according to both retrograde tracer experiments in the primate and neuropathological studies in human AD patients (Fig. 3). Ch4am provides the major cholinergic projection to frontal, parietal and cingulate cortices situated along the medial wall of the hemisphere. Lesser projections are directed to the hypothalamus, hippocampal formation, ventral somatosensory cortex, amygdala, ventrolateral orbital, inferior parietal lobule. The Ch1d subsector is thirpontoparietal opercular regions and the amygdala. Additional projections are directed to the olfactory bulb, medial frontal pole, dorsal motor cortex, insular, inferotemporal area and parahippocampal regions. The Ch1d subsector has a more restricted major projection to the superior temporal gyrus and the temporal pole. Lastly the Ch4ps subsector has a more restricted major projection to the superior temporal gyrus and the temporal pole. Its lesser projections are confined to adjacent inferotemporal and posterior insular regions (Jones et al., 1976; Mesulam and Geula, 1988; Mesulam et al., 1983). Efferent cholinergic fibres from the human NSP course in the external capsule towards the inferior frontal gyrus, which lead Simić et al. to propose that it projects to the cortical speech area in man (Simić et al., 1999). The complex topographical arrangement of Ch4 efferent connectivity also contains considerable overlap between individual subsectors according to primate tracing studies. Some cortical areas, such as the ventrolateral orbital, insular, parahippocampal and ventral striatum, receive projections of comparable size from many different Ch4 subsectors (Mesulam et al., 1983). This may allow for some redundancy in the system, which could pre-vent these cortical areas from substantial cholinergic denervations should one Ch4 subsector be preferentially affected by disease. On the other hand, other cortical regions such as medial frontoparietal, superior temporal and temporopolar regions receive Ch4 projections from a much more restricted number of subsectors, and could therefore be much more vulnerable to cholinergic denervation following limited NBM cell loss in those areas. This is supported by observations in human post-mortem brain tissue which show that secondary degeneration in the nucleus basalis following temporal lobe lesions, but not after frontal or parietal lobe lesions (Kodama, 1929).
Immunohistochemical mapping in post-mortem brain tissue from healthy human subjects shows that efferent cholinergic projections from the NBM leave the nucleus in two highly discrete organized fibre bundles which form the lateral and medial cholinergic pathways (Fig. 1).

The cholinergic axons in these bundles are mostly unmyelinated (Wainer and Mesulam, 1990).

The human post-mortem studies and MRI diffusion ten-sor tractography in healthy volunteers demonstrate that the medial/lateral pathway leaves the NBM anteriorly and joins the white matter of the gyrus rectus. It curves round the rostrum of the corpus callosum to enter the cingulum, travels posteriorly to the splenium and enters the retrosplenial white matter to merge with fibres of the thalamic pathway in the occipital lobe (Hong and Jang, 2010; Selden et al., 1998).

Individual axons radiate from this pathway to supply the medial orbitofrontal, subcallosal, cingulate, pericingular and dorsomedial prefrontal cortices. The lateral pathway subdivides into a capsular division, travelling within the external capsule, and a perisylvian division, travelling within the claustrum (Selden et al., 1998).

On leaving the lateral aspect of NBM the capsular division gives off a bundle of fibres ventrally which travel in the white matter of the uncinate fasciculus to supply the amygdala and temporal lobes. The rest of the capsular division ascends in the external capsule adjacent to the putamen and its individual fibres radiate out to supply the dorsal frontoparietal cortex, middle and inferior temporal gyri. From here its fibres radiate out to supply the frontoparietal opercular cortices, superior temporal gyms and the insula. The medial/lateral cholinergic pathways merge anteriorly in the white matter of the orbitofrontal gyrus. These cortical projections from the NBM have a weak contralateral component of fibres ventrally which travel in the white matter of the uncinate fasciculus to supply the amygdala and temporal lobes. The cap was used in the coronal plane through the anterior commissure - about 2 mm.

Probabilistic maps of compartments of the basal forebrain magnocellular system are now available as an open source reference for correlation with BMRI, PET, and structural MRI data of the living human brain (Zaborsky 2008).

**MR IMAGING**

- thin-section T2-weighted MR
- axial signal intensity of the gray matter
- inferior to globus pallidus
- in substantia innominata of anterior perforated substance

**Volumetric** normalised SI volume in normal subjects 1.68 ± 0.11 (Choi et al. 2012)

The volume of the basal forebrain complex in the human brain varies from 58 to 154 mm3 (Grinberg et al., 2008) as an abbreviation for 'cortex'. NBM = nucleus basal of Meynert (subsectors of NBM as described in main text); retic formation = brainstem reticular formation; SNpc = substantia nigra pars compacta; VTA = ventral tegmental area.
Our protocol for study: 4 mm anterior (2 AC widths) to AC; posterior – anterior border of mammillary body.

**medial** – anterior portion of hypothalamus (25 mm lateral from the midline).

**lateral** – lateral edge of GPe.

For NBM segmentation we are using the following anatomical borders:

- superior – inferior margin of subcommissural part of the globus pallidus
- inferior – pial (basal) surface of the brain.
- anterior – two sagittal widths of anterior commissure anterior from the center of anterior commissure
- posterior – anterior border of mammillary bodies
- medial – medial border of the globus pallidus
- lateral – lateral edge of globus pallidus but sometimes it extends as far as putamen

Zaborszky 2008

the reference space of the Montreal Neurological Institute (MNI) single subject brain:


**NBM SEGMENTATION ANATOMY**

- Ch1al region has the strongest connections to wide-spread cortical areas in the human brain [Mesulam and Geula, 1988; Selden et al., 1998].
- Teipel (2011) study suggests a sequence of atrophy from Ch1al to Chlam and Ch2/3;

**Anatomy of the basal forebrain complex**

3D-reconstruction of the basal forebrain complex (BFC – view from anterior) from the brain of a 29-year-old man who had died of pulmonary arrest [Grinberg and Heinsen, 2007]. The BFC is located within the substantia innominata that is delimited by the caudal rim of the ventral striatum, the ventral pallidum, the ventral parts of the internal capsule and the regions medial to the outlines of the anterior commissure. The BCF can be subdivided into four cell groups arranged in an arch-like path mainly beneath the anterior commissure:

- Ch1 - medial septal nucleus
- Ch2 - nucleus of vertical limb of the diagonal band of Broca
- Ch1–2 are called magnocellular cell groups within the septum.
- Ch3 - nucleus of horizontal limb of the diagonal band of Broca;
- Ch4 also called as the nucleus basalis of Meynert [Mesulam et al., 1983] or sublenticular part of the basal forebrain [Zaborszky 2008].

The nucleus subputaminalis, also called Ayala’s nucleus, has only been described in the human brain so far [Heinsen et al., 2006; Sica et al., 1999].

The volume of the BFC in the human brain varies from 58 to 154 mm³ [Grinberg and Heinsen, 2007, Halliday et al., 1993].
Talairach-Tournoux x/y/z coordinates

<table>
<thead>
<tr>
<th>Subnucleus</th>
<th>Right</th>
<th>Left</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ch2/3</td>
<td>-5/6/-8</td>
<td></td>
</tr>
<tr>
<td>Ch4km</td>
<td>12/4/-10</td>
<td></td>
</tr>
<tr>
<td>Ch4al (lateral subst. innominata)</td>
<td>22/3/-4/7/-10/-7</td>
<td></td>
</tr>
<tr>
<td>Ch4p (posterior subst. innominata)</td>
<td>24/-11/-8</td>
<td></td>
</tr>
<tr>
<td>Ch6 (medial subst. innominata)</td>
<td>14/2/-7</td>
<td></td>
</tr>
</tbody>
</table>

x, the medial to lateral distance relative to midline (positive = right hemisphere);
y, the anterior to posterior distance relative to the AC (positive = anterior);
z, superior to inferior distance relative to the AC-PC line (positive = superior).

Table 3

| Centers of gravity of cytoarchitectonic areas in the anatomical MNI space* |
|---------------------------------|-----------|-----------|
| Cytoarchitectonic area          | x⁰        | y⁰        | z⁰        |
| Ch1–2L                          | -18.4     | 3.7       | -2.0      |
| Ch1–2R                          | 3.0       | 4.3       | -2.5      |
| Ch3L                            | -6.0      | 2.0       | -6.7      |
| Ch3R                            | 7.5       | 2.1       | -6.0      |
| Ch4L                            | -17.2     | -2.3      | -7.1      |
| Ch4R                            | 18.2      | -1.5      | -6.3      |
| Ch4pL                           | -239.7    | -8.6      | -4.8      |
| Ch4pR                           | 24.0      | -9.3      | -4.5      |

* See definition in Materials and methods.
† In mm.
‡ LR left, right side.

Table 4

| Mean volumes of the basal forebrain structures in mm³ with standard deviations (N=10) using individual correction for shrinkage³ |
|---------------------------------|-----------|-----------|
| Structure                      | Left hemisphere | Right hemisphere |
| Ch1.2                          | 45.02±29.90  | 51.09±26.75 |
| Ch3                            | 23.31±16.15  | 27.75±13.88 |
| Ch4                            | 85.09±36.26  | 89.73±36.65 |
| Ch4p                            | 30.36±16.84  | 275.11±16.72 |
| Ch4all                          | 183.62±49.22 | 191.08±55.22 |

Neither interhemispheric or gender differences were significant (p>0.05).

Zaborzsky 2008

HISTOLOGY

Several postmortem studies have found that total number of nucleus basalis Meynert neurons in the ninth decade was 20–30% below that in newborns (Lowes-Hummel et al., 1989; Mann et al., 1984; McGeer et al., 1984).

NBM in relation to globus pallidus (top of image):
These cholinergic neurons have a number of important functions in particular with respect to modulating the ratio of reality and virtual reality components of visual perception. Experimental evidence has shown that normal visual perception has two components. The first (A) is a bottom-up component in which the input to the higher visual cortex (where conscious perception takes place) comes from the retina via the lateral geniculate body and V1. This carries information about what is actually outside. The second (B) is a top-down component in which the input to the higher visual cortex comes from other areas of the cortex. This carries information about what the brain computes is most probably outside. In normal vision, what is seen at the center of attention is carried by A, and material at the periphery of attention is carried mainly by B. When a new potentially important stimulus is received, the Nucleus Basalis is activated. The axons it sends to the visual cortex provide collaterals to pyramidal cells in layer IV (the input layer for retinal fibres) where they activate excitatory nicotinic receptors and thus potentiate retinal activation of V1. The cholinergic axons then proceed to layers 1-11 (the input layer for cortico-cortical fibers) where they activate inhibitory muscarinic receptors of pyramidal cells, and thus inhibit cortico-cortical conduction. In this way activation of Nucleus Basalis promotes (A) and inhibits (B) thus allowing full attention to be paid to the new stimulus. Goard and Dan, and Kuo et al. report similar findings. Gerrard Reopit, in 1984, confirmed the reported findings in his research.
In Parkinson’s and Alzheimer’s diseases, the nucleus basalis undergoes degeneration. A decrease in acetylcholine production is seen in Alzheimer’s disease, Lewy body dementia, Pick’s disease, and some Parkinson’s disease patients showing abnormal brain function, leading to a general decrease in mental capacity and learning.

Most pharmacological treatments of dementia focus on compensating for a faltering NBM function through artificially increasing acetylcholine levels.

significant reductions of the substantia innominata in both AD and patients with Lewy bodies dementia, although the pattern of cortical atrophy is markedly different between both clinical populations [Whitwell et al., 2007].

significantly increased risk to develop dementia was found over 4 years follow-up in cognitively normal subjects with atrophy of the basal forebrain at baseline [Hall et al., 2008].

Cholinergic fibers innervating the cerebral cortex originate mainly from the NBM Ch4 region [Mesulam and Geula, 1988], and their spatial distribution was determined in one seminal study of postmortem sections [Selden et al., 1998].

Nucleus Basalis of Meynert/Substantia Innominata

The nucleus basalis of Meynert is a collection of large hyperchromatic neurons that occupy in part the substantia innominata of the basal forebrain (Kievit and Kuppers 1975; Dene 1975; Jones et al. 1976; Mesulam and Van Hoesen 1976) ventral to the anterior commissure and globus pallidus. Their cholinergic chemisty sets them apart (Mesulam et al. 1983) but also reveals that scattered groups of neurons extend into tentacles anteriorly into the diagonal bands of Broca and septal region, posteriorly toward the hypothalamus and midbrain and laterally to the globus pallidus and amygdala. The major output of the nucleus basalis of Meynert is to the cerebral cortex (Pearson et al. 1983; Mesulam et al. 1983; Wenk et al. 1980; Fibiger 1982) where it provides cholinergic innervation (Fig. 7). However, and importantly, this nucleus also projects to the thalamic reticular nucleus (Levy et al. 1987; Buzsaki et al. 1988; Asanuma 1989). This places the nucleus basalis of Meynert in a position to influence the cortex indirectly as well, because the thalamic reticular nucleus governs thalamic transmission via intrinsic thalamic inhibitory connections (Jones 1975).

Curiously, the nucleus basalis of Meynert receives cortical projections from only a small fraction of the cortex it projects to (Fig. 8). Notable sources of input are from the anterior insular, medial frontal, temporal polar, orbitofrontal and entorhinal cortex (Mesulam and Geula 1988). A strong input is also received from the amygdala (Price and Amaral 1991), and the hippocampal formation projects strongly to septal and diagonal band cholinergic neurons (Fig. 9). These are all endstations for multynaptic streams of cortical axons. It could be argued that the nucleus basalis of Meynert influences all levels of cortical processing, either directly or indirectly via the thalamic reticular nucleus, but is influenced only by cortical endstations after the whole sequence of cortico-cortical processing streams has been traced.

In Alzheimer’s disease, the cortical areas that project to the nucleus basalis of Meynert and the other cholinergic neurons of the basal forebrain are heavily damaged (Arnold et al. 1991; Braak and Braak 1991). Likewise, the cholinergic enzymes in the cortex are diminished (Davies and Maloney 1976) and the nucleus basalis of Meynert is damaged (Whitehouse et al. 1981, 1982; Arendt et al. 1983, 1985; Wilcock et al. 1983; Tourtellotte et al. 1989, Geula and Mesulam 1996). Thus, like the entorhinal/hippocampal cortex and the amygdala, the nucleus basalis of Meynert and its input/output relationships provide another example in Alzheimer’s disease where the endstations of cortical feedforward axons and the origin of cortical feedback axons are damaged heavily.

Telenchic Structures

Among subcortical telencephalic areas, those that form the basal forebrain are clearly implicated in AD. They include the gray matter masses located deep to the neostriatum and globus pallidus and those that converge along the midline at the base of the septum pellucidum. Structures such as the septum, the nuclei of the horizontal and vertical limbs of the diagonal bands of Broca, the substantia innominata and its associated nucleus basalis of Meynert, and the amygdala are major parts. Anatomically these are characterized by reciprocal connections with at least one part of the preoptic-hypothalamic area and reciprocal connections with at least one part of the cerebral cortex. In this sense they serve as intermediaries between those parts of the brain that largely subserves and interact with the internal environment and those that more prominently interact with the external environment.
Nucleus Basalis of Meynert

The nucleus basalis of Meynert is a dispersed group of hyperchromatic neurons that lies in a position ventral to the striatum and globus pallidus. The neurons are multipolar and fusiform in shape and are distributed broadly in both medial-lateral and anterior-posterior directions. In the medio-lateral plane they occupy a position a few millimeters from the midline anteriorly to a position lying beneath the temporal limb of the anterior commissure posteriorly, where the neurons are situated dorsal to the amygdala and ventral to the globus pallidus. In the anterior-posterior plane they extend from the septum anteriorly to the substantia nigra posteriorly. At some levels, scattered neurons of the nucleus basalis may be noted within the internal and external medullary laminae of the globus pallidus and within the lateral hypothalamic area. Although the neurons of the nucleus basalis of Meynert are dispersed, clustering does occur and can be observed in certain loci (Mesulam, Mufson, Levey, & Wainer, 1983). Some of the larger clusters lie within the so-called substantia innominata.

The nucleus basalis of Meynert has attracted attention because of several factors. During the 1980s, with the advent of retrograde tracing procedures, several investigators observed that the neurons that form the nucleus basalis send axons to much of the cerebral cortex and especially to the somatomotor cortices, including Brodmann’s areas 6, 4, 3, 1, 2, and 5 (Divac, 1975; Jones, Burton, Saper, & Swanson, 1976; Kievet & Kyppers, 1975; Mesulam & Van Hoesen, 1976; Pearson, Gauter, Brutal, & Powell, 1983). Using combined retrograde labeling and histochemical methods, Mesulam and Van Hoesen (1976) demonstrated that many of the neurons that form the nucleus basalis of Meynert nerve cells similar to if not identical to those that form the nucleus basalis of Meynert (Jones et al., 1976). As is the case for the closely related cholineric neurons of the medial septum, they have projections directed not to the isocortex but preferentially to the prefrontocortical, periaqueductal, and allocortical areas of the limbic lobe. An especially strong input is directed toward the hippocampal formation, the frontostriatal loop and forms the well-known septohippocampal cholinergic system. Evidence suggests that this pathway is affected in AD (Arendt et al., 1983; Nakano & Hiramo, 1982, 1983), and in such cases the hippocampal formation would be deprived of another major afferent source. This is of special interest because infarcts in this region cause a specific impairment of memory (Damasio, 1985; Volpe & Hirxt, 1983).

Unlike many neural systems of the cortex, direct reciprocity of connections does not characterize the nucleus basalis. Although the nucleus has extremely widespread and topographically organized cortical projections, it receives input from only a subset of the cortical areas to which it projects. These inputs are largely derived from those parts of the limbic lobe that are located in the temporal lobe, anterior insula, and medial and orbital parts of the frontal lobe (Mesulam & Mufson, 1984). Thus, for instance, nucleus basalis output, or feedback, to the visual cortex is reciprocated only after the entire feedforward sequence of corticocortical outflow from sensory cortex to the limbic lobe is traced.

Role in Cognitive Dysfunction in PD

PDD - PD with dementia.
- prevalence of PDD - studies indicating a range of 19%–78% (Biggins et al., 1992; de Lau et al., 2005; Hobson and Meara, 2004; Levy et al., 2002).
- neural basis for cognitive dysfunctions in PD remains unknown.
- PET study using imaging of cerebral acetyl cholinesterase demonstrated that cholinergic dysfunction occurs even in the early course of PD and is more widespread and profound in PDD (Hilker et al., 2005; Shimada et al., 2009).
- basal forebrain pathology occurs simultaneously with nigral pathology (Braak et al., 2003, in a staging study of PD pathology).

**HANYU**


- thickness of the substantia innominata was measured on the coronal T2-weighted image obtained through the anterior commissure: 1. 39 healthy control subjects (age range, 25–86 y; mean age, 62 y) - thickness of the substantia innominata significantly decreased with age 2. 39 patients with AD 3. 36 patients with non-AD dementia, including vascular dementia, frontotemporal dementia, and Parkinson disease with dementia.

- compared with age-matched control subjects, both patients with AD and patients with non-AD dementia had significant atrophy of the substantia innominata:

**Thickness of the substantia innominata in elderly control subjects and patients with dementia**

<table>
<thead>
<tr>
<th>Patient Group</th>
<th>Substantia Innominata Thickness (mm)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Elderly control subjects (n = 21)</td>
<td>2.57 ± 0.19</td>
</tr>
<tr>
<td>AD (n = 39)</td>
<td>1.78 ± 0.28</td>
</tr>
<tr>
<td>Vascular dementia (n = 23)</td>
<td>1.94 ± 0.22</td>
</tr>
<tr>
<td>Frontotemporal dementia (n = 5)</td>
<td>1.79 ± 0.38</td>
</tr>
<tr>
<td>Parkinson disease (n = 8)</td>
<td>1.93 ± 0.19</td>
</tr>
</tbody>
</table>

* P < .0001, compared with thickness in elderly control subjects. Probably “cm” (not “mm”) but still – rostrocaudal thickness is about 2 cm

- thickness of the substantia innominata significantly correlated with scores from the Mini-Mental State Examination in patients with AD but not in patients with non-AD dementia
MR imaging features in this structure may not be specific to AD.

- mean normalized SI volume was significantly decreased in patients with PD-IC (1.54 ± 0.12, p < 0.001), PD-MCI (1.49 ± 0.12, p < 0.001), and PDD (1.39 ± 0.12, p < 0.001) compared with that of control subjects (1.68 ± 0.11).

- normalized SI volume did not differ between patients with PD-IC and PD-MCI; however, the normalized SI volume was significantly decreased in patients with PDD compared with those with PD-IC (p < 0.001) or PD-MCI (p = 0.016).

- normalized SI volume was significantly correlated with general cognitive status (r = 0.51, p < 0.001) as well as with performance in each cognitive subdomain, with a particularly significant independent association with attention (β = 0.33, p = 0.003) and object naming (β = 0.26, p = 0.017).

AD Alzheimer’s disease

- 21 patients with AD + 16 subjects with MCI + 20 healthy elderly subjects
- deformation-based morphometry of MRI scans.

ROI - square aligned relative to the anterior commissure.

DTI imaging was performed with an echo-planar imaging sequence (field-of-view: 256 mm; repetition time: 9,300 ms; echo time: 102 ms; voxel size: 2 x 2 x 2 mm3; four repeated acquisitions, b-value = 1,000, 12 directions, 64 slices, no overlap).

The volume of the right antero-lateral NbM nucleus was correlated with intracortical projecting fiber tract integrity.
**DBS of NBM**

Gratwicke et al. *Neuroscience and Biobehavioral Reviews 37 (2013)*

Nucleus Basalis Deep Brain Stimulation for Thinking & Memory Problems in Parkinson's.

**consent. These considerations will reduce the risks of n**

**cholinesterase inhibitors, have minimal cortical atrophy on imaging, lack significant co**

**electrical stimulation of other brain targets (particularly basal ganglia targets in PD patients). Dementia is a progressive di**

**the clinical efficacy of low frequency DBS of the NBM in dementia and to identify the specific patient characteristics that mig**

**caution since the long line with t**

**results showing that this intervention may markedly improve cognitive functioning. That the patient received benefit across sev**

**communication of the patient with NBM DBS was more impressive than the testing of individual cognitive faculties and critically**

**implantation and motor speed). In addition to these impairments he also displayed poor attention, rigid thinking, psychomotor slowing and ap**

**visual spatial delayed conditions of the test (AVLT (recall) and (recog) tested for multiple co**

**problems with attent**

**et al. (2009)**

**patients with mild AD**

**performed safely in individuals with advanced dementia and also provides preliminary**

**approximately with some co**

**features within the memory circuit of Papez. However, no significant clinical benefit at the gro**

**performed longitudinal analysis of these metabolic changes must be guarded given the**

**dysfunction in dementia. However its clinical value is limited by the fact that it does not allow other cognitive deficits in the disease state**

**the patient was also able to perform AVLT (recog) for the first time, recognizing six words, demonstrating some amelioration of**

**hypothetical: LFS electrophysiological correlates of NBM influence and MMSE score correlation between atrophy and fiber tract changes was**

**measures of cleaning phase resetting in the ipsilateral hippocampal EEG (which**

**entorhinal cortex stimulation could represent one strategy for improving memory function in demen**

**neuronal efficacy with transmission and receive single and pseudo random stimuli (NBM theta modulation), suggesting single and pseudo**

**particular stimulus cycle chosen was unusual by today’s standards given that stimulation was only**

**and none were spared (from atrophy).**

**phase resetting in the ipsilateral hippocampal EEG (which**

**occipital, and uncinate fasciculus (P < 0.05, **

**LFS to neighbouring entorhinal cortex enhances spatial memory in cognitively normal human subjects when applied during the learn**

**visual perceptual abilities increased**

**s) in six patients with mild AD**

**functional imaging was considered as a non invasive way to investigate putative functional circuits that are involved in memory function in humans**

**-month period compared to the baseline assessment. Conclusions regarding the relevance of these metabolic changes remain guarded given the**

**failure to show clinical improvement. Possible methodological factors limiting the clinical effect include unilateral short**

**baseline stimulation only was delivered for a total of 90 minutes each day (a following**

**DBS of NBM is based on a novel therapeutic hypothesis that NBM provides an alternative target for deep brain stimulation therapy for**

**Please visit website at www.NeurosurgeryResident.net**

**TRIALS**

Nucleus Basalis Deep Brain Stimulation for Thinking & Memory Problems in Parkinson's.

https://www.clinicaltrials.gov/ct2/show/NCT01701544?term=NCT01701544&rank=1

Nucleus Basalis Deep Brain Stimulation for Thinking & Memory Problems in Parkinson's.

https://www.clinicaltrials.gov/ct2/show/NCT01701544?term=NCT01701544&rank=1

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