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**Literature search results**

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<td>11 February 2014</td>
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<td>Search completed by:</td>
<td>Marilyn Shaw</td>
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**Search details**

Basal ganglia stroke (right or left). Effect on cognitive/neuropsychological functioning in adults.

**Resources searched**

NHS Evidence; TRIP Database; Cochrane Library; MEDLINE; PsychINFO; Google Advanced Search

*Database search terms*: basal ganglia stroke; effect on cognitive and/or neuropsychological functioning

*Evidence search string(s):* 

*Google search string(s):* 

**Summary**

Not a great deal of articles on this subject have been found. Included articles are as close to the search terms that could be found.

**Guidelines and Policy**

**Evidence-based reviews**

R1. Changes in cognitive function after neuronal cell transplantation for basal ganglia stroke.
Citation: Neurology, October 2004, vol./is. 63/7(1320-1322), 0028-3878;1526-632X (Oct 2004)
Author(s): Stilley, C. S; Ryan, C. M; Kondziolka, D; Bender, A; DeCesare, S; Wechsler, L
Abstract: Reported is the change in cognitive function after neuronal cell transplantation as a treatment for basal ganglia stroke. Nine subjects (two controls, seven transplants), all over 2 years post stroke, completed a comprehensive neuropsychological test battery prior to and 6 months after treatment. Four transplanted subjects who had strokes in the nondominant hemisphere showed marked improvement on the Rey Complex Figure, a test of visuospatial/constructural ability and nonverbal memory. (PsycINFO Database Record (c) 2012 APA, all rights reserved) (journal abstract)
Publication Type: Journal; Peer Reviewed Journal
Source: PsycINFO

R2. Poulin V, Korner-Bitensky N, Dawson DR
Efficacy of executive functions after stroke: a systematic review (2014)
http://www.crd.york.ac.uk/crdweb/ShowRecord.asp?ID=12012019409

R3. Das Nair Roshan, Lincoln N
Cognitive rehabilitation for memory deficits following stroke
Cochrane Library, 2007
(May not be quite what you are after…..)

Published research – Databases
1. Cognitive deficits following stroke in the basal ganglia.
Citation: Clinical Rehabilitation, December 1998, vol./is. 12/6(514-520), 0269-2155;1477-0873 (Dec 1998)
Author(s): Hochstenbach, Jacqueline; van Spaendonck, Karel P. M; Cools, Alexander R; Horstink, Martin W. I. M; Mulder, Theo
Abstract: Examined the effect of a stroke confined to the basal ganglia on cognitive functioning in 12 patients aged 27-67 yrs. Different aspects of memory, attention and concentration, visuospatial and visuoconstructive functions, language, and arithmetic were assessed using neuropsychological tests. The data show a significant abnormality in cognitive functioning on all cognitive domains. The results stress the notion that subcortical damage may lead to substantial neuropsychological disorders and have clear implications for neurological rehabilitation. (PsycINFO Database Record (c) 2012 APA, all rights reserved)
Publication Type: Journal; Peer Reviewed Journal
Source: PsycINFO
Full Text: Available from EBSCOhost in Clinical Rehabilitation

2. One step closer to understanding poststroke fatigue.
Citation: Neurology, October 2012, vol./is. 79/14(1414-1415), 0028-3878;1526-632X (Oct 2, 2012)
Author(s): Kutlubaev, Mansur A; Mead, Gillian E
Abstract: Comments on an article by N. Radman et al. (see record 2012-32888-006). Radman et al. report the results of their study of the associated factors of fatigue in patients at 6 and 12 months after minor ischemic stroke. The authors have shown, for the first time, that poststroke fatigue (PSF) is related to specific cognitive dysfunction, i.e., attentional and executive impairment. Although we do not know whether the relationship between fatigue and cognitive impairment is causal, and if so, whether fatigue causes cognitive impairment, or vice versa, the study results are consistent with the hypothesis that a stroke lesion affecting the neural circuits involved in regulation of attention and executive function may contribute to the development of tiredness and aversion to effort, and subsequently to the development of the behavioral phenomenon of fatigue. The authors found no association between lesion location and PSF. The neural circuits involved with attention and executive function are widely distributed across the entire brain, including the brainstem, thalamus, basal ganglia, and frontal and parietal lobes. The authors also reported the level of morning cortisol, adrenocorticotropic hormone (ACTH), thyroxin, and thyroid-stimulating hormone in plasma and found no association with PSF. These data suggest that PSF is unlikely to be associated with hypothyroidism. Finally, the results of this study support the previous findings that fatigue may be one of the reasons for a
reduction in professional activities. Hence treatment of fatigue is likely to improve the chances that survivors of minor stroke will return to work. The strengths of this article include the longitudinal design of the study, the detailed neuropsychological examination, and well-characterized participants. The study participants had no neurologic deficits, so it is unlikely that their fatigue could be attributed to physical disability. However, this also means that the results of this study cannot be generalized to all stroke survivors. This study by Radman et al. is an important step toward a better understanding of the mechanisms of PSF. Further research on neuropsychological and biological associates of PSF is needed. This is crucial for clarifying the etiology of PSF, and for the subsequent development of effective treatment strategies for this common and debilitating sequel of stroke. (PsycINFO Database Record (c) 2013 APA, all rights reserved)

Publication Type: Journal; Peer Reviewed Journal
Source: PsycINFO

3. Neuropsychological impairment after hemorrhagic stroke in basal ganglia.
Citation: Archives of Clinical Neuropsychology, May 2007, vol./is. 22/4(465-474), 0887-6177;1873-5843 (May 2007)
Author(s): Su, Chwen-Yng; Chen, Hui-Mei; Kwan, Aij-Lie; Lin, Yueh-Hsieh; Guo, Nai-Wen
Abstract: We aimed to determine the severity and pattern of cognitive dysfunction in patients with basal ganglia (BG) hemorrhage within the first 6 months after stroke and to identify its clinical correlates. The study samples consisted of 30 patients with BG hemorrhage and 37 healthy controls. A comprehensive neuropsychological battery including tests of attention, memory, language, visuospatial function, and executive function was administered to all participants. Relative to healthy controls, BG patients performed significantly worse across different cognitive domains after controlling for age, sex, and education. 96.7% of patients displayed defective performance on at least three neuropsychological tests. Discriminant function analysis showed that visuospatial function and memory were the best predictors of group membership (patient/control), with an overall classification rate of 95.5%. Only side of stroke and admission Glasgow Coma Scale (GCS) score correlated significantly with some of the cognitive domains. The widespread pattern of cognitive deficits seen in BG patients provides evidence for the substantial involvement of the BG in many neuronal pathways connecting cortical and subcortical brain areas responsible for various cognitive functions. (PsycINFO Database Record (c) 2013 APA, all rights reserved) (journal abstract)

Publication Type: Journal; Peer Reviewed Journal
Source: PsycINFO

Citation: Focus on cognitive disorder research., 2007(101-116) (2007)
Author(s): Uekermann, J; Daum, I
Abstract: (from the book chapter) Neuropsychological investigations in depression have revealed memory, executive and emotional processing deficits. The observed impairments are consistent with neuroimaging studies, in which structural and functional changes of the dorsolateral prefrontal cortex, the ventrolateral prefrontal cortex, the anterior cingulate cortex, the basal ganglia and the hippocampus have been observed. The neuropsychological impairment pattern has also been found to be associated with clinical variables such as number of depressive episodes. Depression is also a frequent symptom in neurological diseases such as Parkinson's and Huntington's Disease, epilepsy, multiple sclerosis and stroke. The occurrence of depression in neurological diseases has been interpreted as a maladaptive reaction to the neurological impairments and/or as a result of brain dysfunction. This chapter aims to give an overview of neuropsychological investigations of cognitive deficits in depression. The potential influence of depression on cognitive function in neurological disorders and the usefulness of cognitive assessment for the differential diagnosis of depression and dementia will also be addressed. (PsycINFO Database Record (c) 2012 APA, all rights reserved)

Publication Type: Book; Edited Book
Source: PsycINFO

5. Accumulation of MRI Markers of Cerebral Small Vessel Disease is Associated with Decreased Cognitive Function. A Study in First-Ever Lacunar Stroke and Hypertensive Patients.
Citation: Frontiers in aging neuroscience, 2013, vol./is. 5/(72), 1663-4365;1663-4365 (2013)
Author(s): Huijts M; Duits A; van Oostenbrugge RJ; Kroon AA; de Leeuw PW; Staals J

Abstract: Background: White matter lesions (WMLs), asymptomatic lacunar infarcts, brain microbleeds (BMBs), and enlarged perivascular spaces (EPVS) have been identified as silent lesions due to cerebral small vessel disease (cSVD). All these markers have been individually linked to cognitive functioning, but are also strongly correlated with each other. The combined effect of these markers on cognitive function has never been studied and would possibly provide more useful information on the effect on cognitive function.

Methods: Brain MRI and extensive neuropsychological assessment were performed in 189 patients at risk for cSVD (112 hypertensive patients and 77 first-ever lacunar stroke patients). We rated the presence of any asymptomatic lacunar infarct, extensive WMLs, any deep BMB, and moderate to extensive EPVS in the basal ganglia. The presence of each marker was summed to an ordinal score between 0 and 4. Associations with domains of cognitive function (memory, executive function, information processing speed, and overall cognition) were analyzed with correlation analyses. Results: Correlation analyses revealed significant associations between accumulating cSVD burden and decreased performance on all cognitive domains (all p<0.001). Results remained significant for information processing speed ($r=-0.181$, $p=0.013$) and overall cognition ($r=-0.178$, $p=0.017$), after correction for age and sex. Testing of trend using linear regression analyses revealed the same results. Discussion: We tested a new approach to capture total brain damage resulting from cSVD and found that accumulation of MRI burden of cSVD is associated with decreased performance on tests of information processing speed and overall cognition, implying that accumulating brain damage is accompanied by worse cognitive functioning.

Publication Type: Journal Article
Source: MEDLINE
Full Text: Available from National Library of Medicine in Frontiers in Aging Neuroscience

6. Cognitive deficits following stroke in the basal ganglia.
Citation: Clinical Rehabilitation, December 1998, vol./is. 12/6(514-20), 0269-2155;0269-2155 (1998 Dec)
Author(s): Hochstenbach J; van Spaendonck KP; Cools AR; Horstink MW; Mulder T
Abstract: OBJECTIVE: To examine the effect of a stroke in the basal ganglia on cognitive functioning.
DESIGN: As part of a larger prospective study on the neuropsychological and psychosocial consequences of stroke, 12 patients with a stroke confined to the basal ganglia were examined.
SETTING: The patients were assessed in one of the three participating hospitals.
SUBJECTS: The results of 12 patients with a stroke in the basal ganglia (mean age 55 years), were compared to 24 controls (mean age 54.3 years).
MAIN OUTCOME MEASURES: Different aspects of memory, attention and concentration, visuospatial and visuoconstructive functions, language and arithmetic were assessed using neuropsychological tests.
RESULTS: The data show a significant abnormality in cognitive functioning on all cognitive domains.
CONCLUSIONS: The results stress the notion that subcortical damage may lead to substantial neuropsychological disorders and have clear implications for neurological rehabilitation.

Publication Type: Journal Article; Multicenter Study; Research Support, Non-U.S. Gov't
Source: MEDLINE
Full Text: Available from EBSCOhost in Clinical Rehabilitation

Citation: Journal of the Neurological Sciences, November 2012, vol./is. 322/1-2(176-83), 0022-510X;1878-5883 (2012 Nov 15)
Author(s): Lanna ME; Alves CE; Sudo FK; Alves G; Valente L; Moreira DM; Cavalcanti JL; Engelhardt E
Abstract: Strategic regions correspond to associative, limbic and paralimbic structures and related circuits, that underpin cognitive/behavioral functions. Strokes in these eloquent sites produce pictures of vascular dementia with syndromic features due to specific site lesion and/or interruption of their interconnections. This study aims at analysing subcortical strategic strokes that express similar cognitive/behavioral elements, by sharing common pathways. Patients (n=6) who attended in specialized ambulatory, were submitted to neuropsychological and neuroimaging assessments through MRI (GE Signa Horizon 1.5T) and brain SPECT (Millennium MG, ECD [TC-99m]). Stroke locations and respective main symptoms were: 1. anteromedian thalamus [L]: anterograde and retrograde amnesia (ARA), expression aphasia (EA), executive dysfunction (ED), apathy, and depression; 2. anterior thalamus [R]: ARA, inattention, apathy, and aggressiveness; 3.
dorsomedian thalamus [L]: inattention, ED, anosognosia, and aggressiveness; 4. central paramedian thalamus [R]: EA, visual perception deficits (VPD), ED, infantility, and personality disorder; 5. caudate nucleus (ventral-head) [L]: VPD, ED, delirium, visual hallucinations, and personality disorder; and 6. anterior capsule [L]: VPD, ED, apathy, and depression. Vascular strategic syndromes connote the predominantly impaired cognitive/behavioral symptom of each site. Temporal and frontal disconnection symptoms were produced by disrupted MTT/hippocampal and IML/amygdala circuits expressing amnesic syndrome associated with heterogeneous dysexecutive syndrome, in all the cases, by disrupting frontal-basal ganglia-thalamus-cortical net, in three different levels of their pathway. Copyright 2012 Elsevier B.V. All rights reserved.

Publication Type: Journal Article
Source: MEDLINE

8. Disrupted auto-activation, dysexecutive and confabulating syndrome following bilateral thalamic and right putaminal stroke.

Citation: Behavioural Neurology, 2008, vol./is. 19/3(145-51), 0953-4180;0953-4180 (2008)
Author(s): De Witte L; Engelborghs S; Verhoeven J; De Deyn PP; Marien P
Abstract: OBJECTIVE: Clinical, neuropsychological, structural and functional neuroimaging results are reported in a patient who developed a unique combination of symptoms after a bi-thalamic and right putaminal stroke. The symptoms consisted of dysexecutive disturbances associated with confabulating behavior and auto-activation deficits.BACKGROUND: Basal ganglia and thalamic lesions may result in a variety of motor, sensory, neuropsychological and behavioral syndromes. However, the combination of a dysexecutive syndrome complicated at the behavioral level with an auto-activation and confabulatory syndrome has never been reported.METHODS: Besides clinical and neuroradiological investigations, an extensive set of standardized neuropsychological tests was carried out.RESULTS: In the post-acute phase of the stroke, a dysexecutive syndrome was found in association with confabulating behavior and auto-activation deficits. MRI showed focal destruction of both thalami and the right putamen. Quantified ECD SPECT revealed bilateral hypoperfusions in the basal ganglia and thalamus but no perfusion deficits were found at the cortical level.CONCLUSION: The combination of disrupted auto-activation, dysexecutive and confabulating syndrome in a single patient following isolated subcortical damage renders this case exceptional. Although these findings do not reveal a functional disruption of the striato-ventral pallidal-thalamic-frontomesial limbic circuitry, they add to the understanding of the functional role of the basal ganglia in cognitive and behavioral syndromes.

Publication Type: Case Reports; Journal Article; Research Support, Non-U.S. Gov't
Source: MEDLINE
Full Text: Available from EBSCOhost in Behavioural Neurology

9. Neuropsychological impairment after hemorrhagic stroke in basal ganglia.

Citation: Archives of Clinical Neuropsychology, May 2007, vol./is. 22/4(465-74), 0887-6177;0887-6177 (2007 May)
Author(s): Su CY; Chen HM; Kwan AL; Lin YH; Guo NW
Abstract: We aimed to determine the severity and pattern of cognitive dysfunction in patients with basal ganglia (BG) hemorrhage within the first 6 months after stroke and to identify its clinical correlates. The study samples consisted of 30 patients with BG hemorrhage and 37 healthy controls. A comprehensive neuropsychological battery including tests of attention, memory, language, visuospatial function, and executive function was administered to all participants. Relative to healthy controls, BG patients performed significantly worse across different cognitive domains after controlling for age, sex, and education. 96.7% of patients displayed defective performance on at least three neuropsychological tests. Discriminant function analysis showed that visuospatial function and memory were the best predictors of group membership (patient/control), with an overall classification rate of 95.5%. Only side of stroke and admission Glasgow Coma Scale (GCS) score correlated significantly with some of the cognitive domains. The widespread pattern of cognitive deficits seen in BG patients provides evidence for the substantial involvement of the BG in many neuronal pathways connecting cortical and subcortical brain areas responsible for various cognitive functions.

Publication Type: Journal Article
Source: MEDLINE

10. Cognitive dysfunction in patients with cerebral microbleeds on T2*-weighted
gradient-echo MRI.

Citation: Brain, October 2004, vol./is. 127/Pt 10(2265-75), 0006-8950;1460-2156 (2004 Oct)

Author(s): Werring DJ; Frazer DW; Coward LJ; Losseff NA; Watt H; Cipolotti L; Brown MM; Jager HR

Abstract: Gradient echo T2*-weighted MRI has high sensitivity in detecting cerebral microbleeds, which appear as small dot-like hypointense lesions. Microbleeds are strongly associated with intracerebral haemorrhage, hypertension, lacunar stroke and ischaemic small vessel disease, and have generated interest as a marker of bleeding-prone microangiopathy. Microbleeds have generally been considered to be clinically silent; however, since they are located in widespread cortical and basal ganglia regions and are histologically characterized by tissue damage, we hypothesized that they would cause cognitive dysfunction. We therefore studied patients with microbleeds (n = 25) and a non-microbleed control group (n = 30) matched for age, gender and intelligence quotient. To avoid the confounding effects of coexisting cerebrovascular disease, the groups were also matched for the extent of MRI-visible white matter changes of presumed ischaemic origin, location of cortical strokes, and for the proportion of patients with different stroke subtypes (including lacunar stroke). A battery of neuropsychological tests was used to assess current intellectual function, verbal and visual memory, naming and perceptual skills, speed and attention and executive function. Microbleeds were most common in the basal ganglia but were also found in frontal, parieto-occipital, temporal and infratentorial regions. There was a striking difference between the groups in the prevalence of executive dysfunction, which was present in 60% of microbleed patients compared with 30% of non-microbleed patients (P = 0.03). Logistic regression confirmed that microbleeds (but not white matter changes) were an independent predictor of executive impairment (adjusted odds ratio = 1.32, 95% confidence interval 1.01-1.70, P = 0.04). Patients with executive dysfunction had more microbleeds in the frontal region (mean count 1.54 versus 0.03; P = 0.002) and in the basal ganglia (mean 1.17 versus 0.32; P = 0.048). There was a modest correlation between the number of microbleeds and the number of cognitive domains impaired (r = 0.44, P = 0.03). This study provides novel evidence that microbleeds are associated with cognitive dysfunction, independent of the extent of white matter changes of presumed ischaemic origin, or the presence of ischaemic stroke. The striking effect of microbleeds on executive dysfunction is likely to result from associated tissue damage in the frontal lobes and basal ganglia. These findings have implications for the diagnosis of stroke patients with cognitive impairment, and for the appropriate use of antihypertensive and antiplatelet treatments in these patients.

Publication Type: Journal Article; Research Support, Non-U.S. Gov't

Source: MEDLINE

Full Text: Available from Highwire Press in Brain

11. Asymptomatic ischemic cerebrovascular disorders and neuroprotection with vinpocetine.

Citation: Idegggyogyaszati Szemle, May 2003, vol./is. 56/5-6(166-72), 0019-1442;0019-1442 (2003 May 20)

Author(s): Hadjieva D

Abstract: The asymptomatic ischemic cerebrovascular disorders (AICVD) is an early manifestation of cerebrovascular disease. It is also known as latent insufficiency of the cerebrovascular circulation or as asymptomatic cerebrovascular disorders. Recently, the term subclinical disease, detected noninvasively, has been introduced by American Heart Association. The diagnosis is based on the following criteria: evidence of vascular risk factors; episodic nonspecific complaints without any focal cerebral symptoms; mild cognitive deficit, detected by neuropsychological tests; carotid ultrasonography often shows intimal-medial thickening. atherosclerotic plaques and carotid stenosis; CT and MRI occasionally reveal silent cerebral infarctions, white matter hyperintensities or cerebral atrophy; regional hypoperfusion above the ischemic threshold is also seen by rCBF measurements. Treatment of the AICVD, modifying the vascular risk factors and using neuroprotective agents, should be the cornerstone of primary prevention of ischemic stroke and cognitive decline, caused by cerebrovascular disorders. Vinpocetine has been found to interfere with various stages of the ischemic cascade: ATP depletion, activation of voltage-sensitive Na(+) and Ca(++)-channels, glutamate and free radicals release. The inhibition of the voltage-sensitive Na(+) -channels appears to be especially relevant to the neuroprotective effect of vinpocetine. Pronounced antioxidant activity of the drug could also contribute to the neuroprotection. PET studies in primates and man showed that 11C
labelled vinpocetine passes the blood-brain barrier rapidly. Heterogeneous brain distribution of the compound was observed mainly in the thalamus, basal ganglia, occipital, parietal and temporal cortex, regions which are closely related to the cognitive functions. PET studies in chronic ischemic stroke patients revealed favourable effects of vinpocetine on rCBF and glucose metabolism in the thalamus, basal ganglia and primary visual cortex. It seems, vinpocetine, affecting the multiple mechanisms of the AICVD, could be of benefit for the treatment in this early stage of cerebrovascular disease. Vinpocetine may also become a new therapeutic approach to prophylactic neuroprotection in patients at high risk of ischemic stroke.

Publication Type: Journal Article; Review
Source: MEDLINE

Citation: Acta Neurologica Scandinavica, November 2002, vol./is. 106/5(309-13), 0001-6314;0001-6314 (2002 Nov)
Author(s): Sartor H; Loose R; Tucha O; Klein HE; Lange KW
Abstract: We report on a patient with long standing, full-blown mitochondrial encephalomyopathy, lactic acidosis and stroke-like episodes (MELAS). In contrast to earlier publications, detailed neuropsychological assessment revealed no dementia but a pattern of distinct cognitive deficits with marked impairment of visuo-constructive and executive functions. Focal lesions and progressing atrophy mainly of the basal ganglia and the temporo-parieto-occipital area with preservation of hippocampal and entorhinal structures were present. Furthermore, a 4-year follow-up assessment revealed an increasing deterioration of distinct cognitive functions, including phasic alertness, tactile functions and the discrimination of tone pitch and rhythm. This may be because of chronic regional metabolic disturbances, as there was no further stroke-like episode in that period of time.
Publication Type: Case Reports; Journal Article
Source: MEDLINE
Full Text: Available from EBSCOhost in Acta Neurologica Scandinavica

13. Rule-abstraction deficits following a basal ganglia lesion.
Citation: Neurocase, 2001, vol./is. 7/5(433-43), 1355-4794;1355-4794 (2001)
Author(s): Swainson R; Robbins TW
Abstract: The cognitive profile of a patient, PM, who had damage to the right basal ganglia as the result of a stroke was investigated. Whilst most cognitive functions were intact, she showed specific neuropsychological deficits, most notably a difficulty in developing, through abstraction of the relevant information, a higher-level rule by which to guide behaviour. The types of rule affected were those based upon an attentional set (attending to a particular dimension of stimulus features, such as 'shape') or a response strategy (continuing to apply a previously successful pattern of responses). The learning of lower-level rules based on stimulus-reward values was spared, as was the ability to apply an instructed rule and to discontinue use of rules which were no longer appropriate. These data provide evidence for the dissociability of cognitive functions within the basal ganglia.
Publication Type: Case Reports; Journal Article; Research Support, Non-U.S. Gov't
Source: MEDLINE

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From 1st fifty results:
G1. Brown LL
Sensory and cognitive functions of the basal ganglia
Current Opinion in Neurobiology, 1997, 7, 157-163
G2. Sachdev PS
The neuropsychological profile of vascular cognitive impairment in stroke and TIA patients
Cognitive impairment after stroke
Current Opinion in Neurology, 2002, 15, p79 – 84

Providing explicit information disrupts implicit motor learning after basal ganglia stroke
Learn Mem, 2004, 11, 388 -396

Early depressive symptoms after stroke: neuropsychological correlates and lesion characteristics
Jnl of the Neurological Sciences, 2005, 228, p27 – 33

Vascular cognitive impairment
Nature Clinical Practice Neurology, 2006, 2, p538-547