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Short-term mechanisms influencing volumetric brain dynamics

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ABSTRACT

With the use of magnetic resonance imaging (MRI) and brain analysis tools, it has become possible to measure brain volume changes up to around 0.5%. Besides long-term brain changes caused by atrophy in aging or neurodegenerative disease, short-term mechanisms that influence brain volume may exist. When we focus on short-term changes of the brain, changes may be either physiological or pathological. As such determining the cause of volumetric dynamics of the brain is essential. Additionally for an accurate interpretation of longitudinal brain volume measures by means of neurodegeneration, knowledge about the short-term changes is needed. Therefore, in this review, we discuss the possible mechanisms influencing brain volumes on a short-term basis and set-out a framework of MRI techniques to be used for volumetric changes as well as the used analysis tools. 3D T_1-weighted images are the images of choice when it comes to MRI of brain volume. These images are excellent to determine brain volume and can be used together with an analysis tool to determine the degree of volume change. Mechanisms that decrease global brain volume are: fluid restriction, evening MRI measurements, corticosteroids, antipsychotics and short-term effects of pathological processes like Alzheimer's disease, hypertension and Diabetes mellitus type II. Mechanisms increasing the brain volume include fluid intake, morning MRI measurements, surgical revascularization and probably medications like anti-inflammatory drugs and anti-hypertensive medication. Exercise was found to have no effect on brain volume on a short-term basis, which may imply that dehydration caused by exercise differs from dehydration by fluid restriction. In the upcoming years, attention should be directed towards studies investigating physiological short-term changes within the light of long-term pathological changes. Ultimately this may lead to a better understanding of the physiological short-term effects of pathological processes and may aid in early detection of these diseases.

1. Introduction

Nowadays, magnetic resonance imaging (MRI) is used to non-invasively investigate tissue loss overtime (Ashburner and Ridgway, 2013; Fox and Freeborough, 1997; Leung et al., 2012; Rudick et al., 1999; Smith et al., 2002), whereas in the early days it was predominantly investigated through post-mortem studies (Alzheimer, 1907; Braak and Braak, 1995, 1991; Dawson, 1916). Therefore, brain tissue loss can be assessed prior to death and changes can be monitored during life (Chetelat and Baron, 2003; Fox et al., 2000, 2014; Fox and Freeborough, 1997; Frisoni, 2001). Characterization of chronic brain atrophy by means of MRI is already well established and used widely in longitudinal studies (Fox and Freeborough, 1997; Rudick et al., 1999). The current brain imaging techniques and analysis tools are, however, so sensitive and precise that changes in brain volume can be measure up to around 0.5% (Caramanos et al., 2010; Chard et al., 2002; Rudick et al., 1999; Smith et al., 2001). These changes lie within the range of yearly brain changes in normal aging (Fisher et al., 2008) and may be short-term physiological fluctuations of the brain caused by fluid restriction (Biller et al., 2015; Dickson et al., 2005; Duning et al., 2005; Kempton et al., 2011, 2009; Meyers et al., 2016; Streitbürger et al., 2012) or use of medication, but could also be caused by pathological processes like diabetes, hypertension and Alzheimer's disease (AD) (Meusel et al., 2014; Thornton, 2014). In addition, it is though that during the night a redistribution of body fluids occurs, therefore brain volume even changes during the day, with a decrease in volume observed by the end of the day (Nakamura et al., 2015). For an accurate interpretation of brain volume measurements it is, therefore, essential to know to what extent the short-term changes – ranging from hours to a couple of days – are caused by physiological fluctuations or due to pathological differences. Furthermore for the interpretation of longitudinal data knowledge of the short-term changes is also crucial since they may introduce bias and additional corrections may be needed. Therefore, in this review, we discuss the possible mechanisms that can

Abbreviations: CVR, Cerebrovascular reactivity

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Table 1
Studies investigating mechanisms of brain volume fluctuations.

Mechanism	Author	MRI sequence	Analysis tool (version)	Study population	Number of patients	Effect on brain volume (numbers in %)	Effect on ventricular volume
Dehydration-*Fluid restriction*	Duning et al. (2005)	-3D T1-weighted	SIENA (FSL)	Healthy volunteers	20	↓0.55**–↑0.72*	n.a
	Streitbürger et al. (2012)	-3D T1-weighted	VBM8 (SPM), SIENAr (v5.0) and FreeSurfer (v4.5)	Healthy volunteers	6	↓1.7**–↑3.5*	↑2.6**
	Nakamura et al. (2014)	-3D T1-weighted -2D dual echo T2-weighted (for T2-relaxation)	BET, SIENAX & Jacobian integration method (all FSL v5.0)	Healthy volunteers	14	No effect of dehydration -↑0.36*	n.a
	Biller et al. (2015)	-3D T1-weighted -FLASH (T1-relaxation) -^{1}H-MR Spectroscopy (brain tissue fluid H_2O)	FreeSurfer (v5.3.0) – LCModel	Healthy volunteers	15	↓0.36**–↑0.87* No effect ↓1.63**–↑0.43*	n.a n.a.
	Meyers et al. (2016)	-3D T1-weighted -T2 relaxation	FLIRT, FAST, BET (all FSL v5.0) and ALVIN (v1.06)	Healthy volunteers	20	↓0.03 (ns)**–↑0.15 (ns)*	↑0.22 (ns)**–↓0.62 (ns)*
Dehydration-*Exercise*	Dickson et al. (2005)	-3D T1-weighted	Analyze (v7.0)	Trained healthy volunteers	6	No effect	No effect
	Kempton et al. (2009)	-3D T1-weighted	SIENA (v2.4), MEASURE	Trained healthy volunteers	7	No effect	↑(No percentages mentioned)**
	Kempton et al. (2011)	-3D T1-weighted	SPM5	Healthy volunteers	10	No effect	↑(No percentages mentioned)**
		-BOLD-fMRI				↑BOLD response	n.a
		-pcASL				↑Blood flow	n.a
Diurnal fluctuations	Nakamura et al. (2015)	-3D T1-weighted	BEaST (v1.15) and FAST (FSL v5.0)	MS and AD	1589	↓0.180–0.438	n.a

n.a. = not applicable.
* *after rehydration.*
** *after dehydration.*