Neuroscience and Biobehavioral Reviews 115 (2020) 351–362

Contents lists available at ScienceDirect

Neuroscience and Biobehavioral Reviews

journal homepage: www.elsevier.com/locate/neubiorev

Functional neuroimaging of human postural control: A systematic review with meta-analysis

Bauke W. Dijkstra[a,*], Esther M.J. Bekkers[a], Moran Gilat[a], Veerle de Rond[a], Robert M. Hardwick[b,c], Alice Nieuwboer[a]

[a] Neuromotor Rehabilitation Research Group, Department of Rehabilitation Sciences, KU Leuven, Tervuursevest 101, Bus 1501, 3001, Leuven, Belgium
[b] Movement Control and Neuroplasticity Research Group, Department of Movement Sciences, KU Leuven, Tervuursevest 101, Bus 1501, 3001, Leuven, Belgium
[c] Institute of Neuroscience, Université Catholique De Louvain, Brussels, Belgium

ARTICLE INFO

Keywords:
Postural control
Posture
Balance
Functional neuroimaging
MRI
PET
Meta-Analysis

ABSTRACT

Postural instability is a strong risk factor for falls that becomes more prominent with aging. To facilitate treatment and prevention of falls in an aging society, a thorough understanding of the neural networks underlying postural control is warranted. Here, we present a systematic review of the functional neuroimaging literature of studies measuring posture-related neural activity in healthy subjects. Study methods were overall heterogeneous. Eleven out of the 14 studies relied on postural simulation in a supine position (e.g. motor imagery). The key nodes of human postural control involved the brainstem, cerebellum, basal ganglia, thalamus and several cortical regions. An activation likelihood estimation meta-analysis revealed that the anterior cerebellum was consistently activated across the wide range of postural tasks. The cerebellum is known to modulate the brainstem nuclei involved in the control of posture. Hence, this systematic review with meta-analysis provides insight into the neural correlates which underpin human postural control and which may serve as a reference for future neural network and region of interest analyses.

1. Introduction

Falls can lead to a wealth of clinical consequences, including troublesome injuries, fear of future falls, immobilization, decreased quality of life and even mortality (Hartholt et al., 2011; Burns and Kakara, 2018). Impairments in postural control are a major risk factor for falls (Ambrose et al., 2013) and become more prominent with aging (Boisgontier et al., 2017), thereby placing a high socio-economic burden on our aging society. Although critical for the development of fall prevention programs (Horak, 2006), the understanding of the neural control of posture in humans has, until recently, remained limited.

Animal research has shown that the brainstem contains key nodes for postural control, such as the vestibular nuclei, mesencephalic locomotor region and the pontomedullary reticular formation (Takakusaki, 2017; MacKinnon, 2018). These nodes receive input from subcortical structures (e.g. cerebellum and basal ganglia) and cortical areas, are interconnected and regulate posture through projections to premotor interneurons in the spinal cord as well as to alpha and gamma motoneurons (for comprehensive overview see: MacKinnon, 2018). StartReact and startle reflex paradigms provided evidence that also the human brainstem contains key nodes for postural control that are likely modified by cortical input (Nonnekes et al., 2015, 2014). The contribution of brain structures to postural control depends on the task at hand (Taube et al., 2015), which might involve various levels of automaticity, postural reflexes and task complexity.

A recent review addressing associations between structural magnetic resonance imaging (MRI) and postural control metrics provided evidence for the involvement of many regions across the entire brain and in particular the cerebellum in human postural control (Surgent et al., 2019). However, associations between structural brain metrics and behavior do not show which brain areas are activated during postural task performance. Whole-brain activity measurements across the entire brain obtained during actual postural task performance are needed to determine the brain networks activated during postural control. To date only one research group measured cortical and subcortical brain activity in humans during actual performance of postural tasks using a rare mobile gantry positron emission tomography (PET) system (Ouchi et al., 1999, 2001). Other, more common functional neuroimaging methods, such as electroencephalography or functional

* Corresponding author at: Department of Rehabilitation Sciences, KU Leuven, Tervuursevest 101 box 1501, 3001, Leuven, Belgium.
E-mail addresses: bauke.dijkstra@kuleuven.be (B.W. Dijkstra), esther_bekkers@hotmail.com (E.M.J. Bekkers), moran.gilat@kuleuven.be (M. Gilat), veerle.derond@kuleuven.be (V. de Rond), robert.hardwick@uclouvain.be (R.M. Hardwick), alice.nieuwboer@kuleuven.be (A. Nieuwboer).

https://doi.org/10.1016/j.neubiorev.2020.04.028
Received 15 October 2019; Received in revised form 7 April 2020; Accepted 23 April 2020
Available online 11 May 2020

near-infrared spectroscopy that allow measurements during upright postural tasks unfortunately lack the spatial resolution to study the involvement of subcortical structures in postural control. Magnetic resonance imaging and PET scanners offer sufficiently high spatial resolution, but require subjects to lie absolutely still and thus preclude measurements during actual postural tasks. Over the last decade, simulation of postural tasks during functional MRI, e.g. through motor imagery or action observation, has become increasingly popular to unravel the neural underpinnings of postural control. Recently, Hardwick et al. (2018) showed with an activation likelihood estimation (ALE) meta-analysis that motor simulation (303 experiments) and actual movement execution (142 experiments) activated subcortical structures in a similar fashion. Yet, no review of functional imaging evidence derived from actual posture or postural simulation paradigms has been conducted to identify the brain regions that are consistently activated during human postural control. This knowledge is essential to further our understanding of which brain regions can be targeted trough interventions aiming to reduce postural instability, which are especially important when pathology comes to the fore. We therefore performed a systematic review (qualitative analysis) with meta-analysis (quantitative analysis) on whole-brain functional MRI or PET studies assessing the neural control of posture in healthy adults.

2. Methods

2.1. Qualitative analysis

2.1.1. Search strategy and study selection for the qualitative analysis

PubMed was searched for relevant articles until July 12th 2019 without date restrictions using the following search terms: 'posture' or 'postures' or 'postural' or 'balance' or 'stance' or 'locomotor' in the title, in combination with 'magnetic resonance imaging' or 'MRI' or 'fMRI' or 'positron emission tomography' or 'PET' in the title or abstract. Only studies in which within-subject posture-related brain activations in healthy adults were measured through functional MRI or functional PET were selected. Tasks were deemed posture-related if subjects were required to be, either through actual performance or simulation (e.g. motor imagery or action observation), in an upright position without performance of stepping movements (i.e. non-gait tasks). The exclusion criteria were: 1) studies comparing functional neuroimaging outcomes before and after an intervention only, without reporting baseline data; 2) studies that did not include healthy adults; 3) studies not reporting brain coordinates in standard stereotactic coordinate space; and 4) studies not written in English. Studies focusing on associations between structural MRI metrics and postural behavior were not included, as this was recently reviewed (Surgent et al., 2019). The titles, abstracts and full-texts were subsequently screened independently by BWD and EMJB for inclusion. Discrepancies in article selection were discussed among the investigators until consensus was reached.

2.1.2. Data extraction qualitative analysis

The following data were extracted: number of subjects, age of the subjects, neuroimaging technique used, task performed in the scanner, correction for multiple comparisons (yes/no) and first-level contrast(s). In addition, BWD and VdR independently extracted study key findings, which comprised the postural activations reported in studies. In case of >1 contrast per study, the most consistent activations were selected. Discrepancies in key findings were discussed among investigators until consensus was reached. Only data of healthy subjects were extracted. As the brain structures involved in postural control are task-dependent (Taube et al., 2015), postural tasks were divided into static, dynamic and reactive postural control subdomains. Postural control was considered 'static' when unperturbed bilateral stance was performed. In contrast, upright stance tasks that involved a self-initiated action (e.g. standing on one foot or shifting weight from one leg to the other leg) were labeled as 'dynamic' postural control. Postural control was considered 'reactive' when a response to an external perturbation (e.g. a surface translation or visual field motion) was required.

2.2. Quantitative analysis: activation likelihood estimation meta-analysis

A coordinate-based ALE meta-analysis (Turkeltaub et al., 2002; Eickhoff et al., 2009, 2012) was performed on functional neuroimaging studies using actual postural or postural simulation tasks in healthy adults (i.e. the quantitative analysis). From here on, actual and simulated postural tasks are both labeled as 'postural tasks'.

2.2.1. Experiment selection and data extraction for the quantitative analysis

Posture-related neuroimaging contrasts (hereafter called experiments) included in studies of the qualitative review were selected for the ALE meta-analysis if coordinates from whole brain analyses were reported in standard stereotactic coordinate space (i.e. Montreal Neurological Institute (MNI) or Talairach). Experiments in which only an ROI analysis was used were excluded. Furthermore, only within-subject contrasts between a postural and a control task were selected. Experiments contrasting two postural tasks were included if the contrast task was considered to be more challenging than the control task, under the assumption that it would lead to greater activations across the postural control network. Deactivations were not considered. If no mention was made regarding the re-use of participants in studies coming from the same research group, we assumed that different samples were used. The following data were extracted: number of subjects, peak-voxel coordinates (called foci) and respective standard stereotactic coordinate space (i.e. MNI or Talairach), neuroimaging modality (i.e. fMRI or PET), field of view (i.e. whole brain or limited brain volume - see below for further details) and the contrasts of interest. Data extraction was performed by BWD and was checked by a second researcher (VdR), following the best-practice recommendations for neuroimaging meta-analyses (Muller et al., 2018). Coordinates reported in Talairach space were converted into MNI space using the Lancaster transform (Lancaster et al., 2007). Foci from studies that used small volume corrections were only included if the statistical threshold used in the rest of the brain was met, i.e. foci that did not meet the statistical threshold used in the rest of the brain were excluded (Muller et al., 2018). To prevent that subject groups involved in multiple experiments influenced the ALE values more than others, peak activations were organized by subject group (i.e. multiple experiments in one study were pooled; Turkeltaub et al., 2012).

2.2.2. Exploratory ALE meta-analyses

The ALE meta-analyses were conducted using GingerALE version 3.0.2 (Eickhoff et al., 2009, 2012; Turkeltaub et al., 2012). The ALE approach models foci reported in neuroimaging studies as centers of 3D Gaussian probability distributions (Turkeltaub et al., 2002) in order to account for the inherent between-template and between-subjects variability in peak coordinates (Eickhoff et al., 2009). For every included experiment, a modeled activation map was created, which contained the probability of an activation for every voxel without allowing multiple foci from a single experiment to jointly influence a single voxel (Turkeltaub et al., 2012). The union of the modeled activations maps was then used to calculate voxel-based ALE scores (Eickhoff et al., 2012), but only for voxels with a $\geq$ 10 % grey matter probability based on the ICBM tissue probability maps (Evans et al., 1994). The ALE approach determines whether converging foci across different experiments occur at a level greater than chance by comparing them to an empirically determined null distribution based on a random spatial association between experiments (Turkeltaub et al., 2002). Coordinates of experiments in which SPM (version 99 or later) was used with normalization to the software's standard template and in which no transformation was reported were treated as MNI coordinates (Muller et al., 2018). The significance threshold in this study was set at $p < 0.05$ while applying a cluster-level family-wise error (FWE)

correction for multiple comparisons with a cluster-forming threshold at voxel level of $p < 0.001$ (Eickhoff et al., 2016). Results are provided with a voxel size of 2 mm^3.

Only studies with full brain coverage were incorporated in our main analysis (nine MRI and two PET). One of the PET studies measured brain activation related to actual stance (Schoberl et al., 2017), while all others investigated simulated postural control. Therefore, a sub-analysis was carried in which we excluded this PET-study to reduce this source of heterogeneity. Finally, meta-analyses per postural domain were not considered due to the small number of studies available in each domain.

Two additional PET-studies applied a limited field of view, namely from the middle frontal gyrus to the lower cerebellum (Ouchi et al., 1999) and from the lower superior frontal gyrus to the upper cerebellum (Ouchi et al., 2001). Therefore, these studies did not meet the inclusion criteria for a coordinate-based ALE meta-analysis and were not included in the main analysis. However, as the PET studies were conducted in the actual upright position, they can be considered as critical for unravelling the neural underpinnings of postural control (Bohnen and Jahn, 2013). As such, an exploratory supplementary meta-analysis was conducted including these studies (thus in total nine MRI and four PET), which is presented in the supplementary materials.

2.2.3. Robustness against publication bias

The generalizability of coordinate-based meta-analyses is hampered by the exclusion of studies that are not published, e.g. due to a lack of significant findings. Recently, the prevalence of the number of experiments without significant foci was estimated based on the number of foci per experiment in the BrainMap database (Samartsidis et al., 2019). It was estimated that 7.75 experiments per 100 published normal mapping experiments were missing, with a 95 % bootstrap confidence interval of 6.73 − 8.66. To determine the robustness of our main ALE meta-analysis against publication bias, we calculated per cluster the Fail-Safe N (FSN). The FSN represents the number of noise studies that can be added to an ALE meta-analysis before a cluster is no longer significant and was calculated based on the procedure proposed by Acar et al. (2018). We determined the minimum FSN based on the upper bound of the confidence interval of missing normal mapping studies reported by Samartsidis et al. (2019) as 8.66/100*11 = 0.95 and set it at 1. In addition, an upper boundary for the FSN was calculated to determine if clusters were highly influenced by a small amount of studies. Aiming for a contribution of at least 10 % of studies to the cluster of interest, the upper boundary of the FSN was calculated per cluster as: ((number of studies contributing to a cluster)/.1))-(total number of studies included in ALE meta-analysis). Noise studies were created using the R code provided by Acar et al. (2018). These noise studies were added to our main meta-analysis following the procedures proposed by Acar et al. (2018) to determine the FSN per cluster.

2.2.4. Labeling

Anatomic labels were assigned to significant clusters using the xjView toolbox (http://www.alivelearn.net/xjview). Coordinates are reported in Montreal Neurological Institute (MNI) space.

3. Results

3.1. Qualitative analysis

The systematic search resulted in 476 non-duplicate hits (see Fig. 1), of which 14 studies were included for review. Methodological features and key findings are summarized in Table 1. Fig. 2**A** illustrates foci activated during static, dynamic and reactive postural control. Fig. 2**B** shows a more posterior involvement of foci during actual compared to simulated postural control.

3.1.1. Static postural control

Nine studies measured brain activation during static postural control tasks of which seven corrected for multiple comparisons (Zwergal et al., 2012; Schoberl et al., 2017; Jahn et al., 2008; Ouchi et al., 1999, 2001; Karim et al., 2014; Malouin et al., 2003) and two did not (Jahn et al., 2009, 2004). Four studies were based on small groups (10 or less subjects). Four studies used PET neuroimaging, three of which measured actual bilateral stance with either a mobile gantry PET system that was able to measure brain activations during actual performance (Ouchi et al., 2001, 1999) or ligand-uptake after executing a postural task (Schoberl et al., 2017). Five studies used postural simulation during fMRI and one study used postural simulation during PET scanning.

Cerebellar involvement, especially in the vermis, was found to occur in six out of the eight studies that used whole brain analyses (the ninth study, Jahn et al., 2009, used an ROI analysis considering the hippocampus and parahippocampal gyrus). The studies which measured static postural control through actual stance (Ouchi et al., 1999, 2001; Schoberl et al., 2017) or motor imagery in combination with fMRI (Zwergal et al., 2012; Jahn et al., 2004, 2008) reported these activations in the cerebellar vermis. In contrast, the two studies in which PET in combination with motor imagery (Malouin et al., 2003) or stance simulation through dorsiflexor and plantarflexor activation was used, did not find cerebellar involvement (Karim et al., 2014). Motor imagery-based studies also revealed brainstem activations in the vestibular nuclei (Jahn et al., 2008), the pontomesencephalic junction (Zwergal et al., 2012) and the left midbrain (Jahn et al., 2004). Other subcortical activations, were captured mostly during motor imagery of stance and comprised the thalamus (Zwergal et al., 2012; Schoberl et al., 2017; Jahn et al., 2004, 2008), caudate nucleus, putamen or pallidum of the basal ganglia (Zwergal et al., 2012; Jahn et al., 2004; Karim et al. 2014) and the hippocampus (Jahn et al., 2009, 2004, 2008). Cortical activations were present in the right inferior occipital gyrus during actual standing (Ouchi et al., 1999, 2001). The left anterior cingulate gyrus and left paracentral lobule were active when PET-scans were administered after standing (Schoberl et al., 2017). Furthermore, actual stance deactivated the frontal lobe (Schoberl et al., 2017), whereas stance with eyes closed led to activations in the frontal lobe (Ouchi et al., 1999). Postural simulation studies also showed widespread activation in frontal, parietal, temporal and occipital cortical areas as well as in the cingulate cortex.

3.1.2. Dynamic postural control

Three studies measured dynamic postural control either through standing on one foot or tandem stance during actual postural performance (Ouchi et al., 1999) or motor imagery of swaying tasks (Ferraye et al., 2014; Slobounov et al., 2006a). Different kinds of control tasks were adopted. Control for multiple testing was applied in two studies (Ouchi et al., 1999; Ferraye et al., 2014), but not in the study of Slobounov et al. (2006a). Brainstem activation was reported in the red nucleus (Ouchi et al., 1999), the mesencephalic locomotor region (MLR) and pons (Ferraye et al., 2014). Importantly, the red nucleus activation was present in the tandem > bilateral stance contrast, but not in the tandem > supine contrast. Cerebellar activation, including foci in the vermis, was present across all studies. Actual postural performance resulted mainly in cerebellar activations, although cortical activity in the right inferior temporal and occipital gyri was also present. Motor imagery of dynamic postural control activated the basal ganglia, in particular the right putamen (Ferraye et al., 2014; Slobounov et al., 2006a). Furthermore, activations in the thalamus, frontal lobe (including the supplementary motor area (SMA)), parietal lobe, cingulate cortex, occipital lobe and insula were reported during motor imagery of a dynamic postural control task. Effective connectivity analyses showed that the right SMA increased its coupling with the right MLR, as did the left thalamus with the left lateral globus pallidus during motor imagery of dynamic postural control (Ferraye

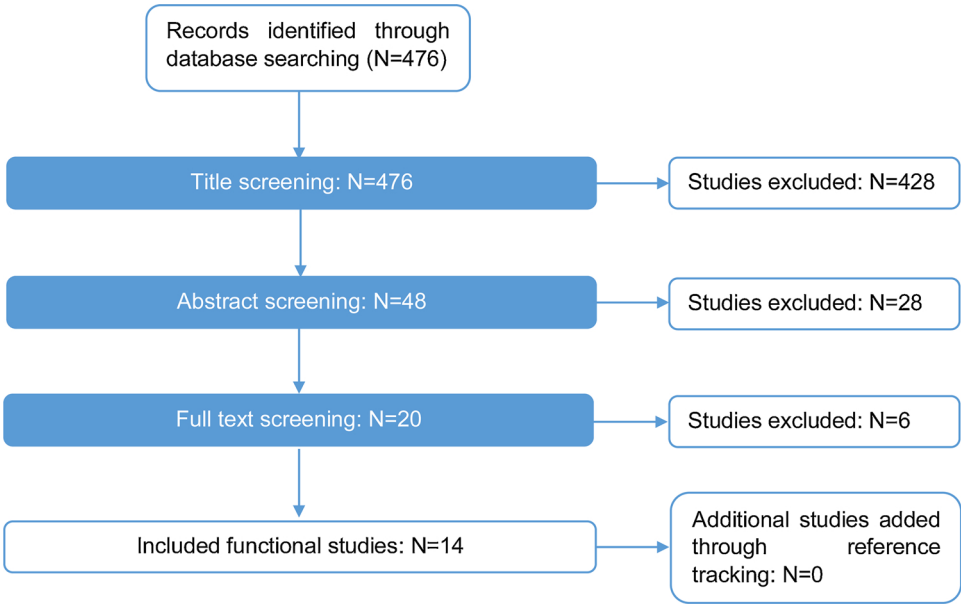

Fig. 1. Flow chart of the systematic search.

et al., 2014).

3.1.3. Reactive postural control

Three studies measured reactive postural control using fMRI in combination with postural simulation (either motor imagery or motor imagery plus action observation). These studies were statistically controlled for multiple comparisons. The perturbations used were visual field motion or a mechanical mediolateral perturbation while standing on an unstable wobble board. Importantly, all studies contrasted reactive against static postural control, thus the reported activations were specific to reactive postural control. Cerebellar activation was evident in all studies (Slobounov et al., 2006b; Mouthon et al., 2018; Taube et al., 2015). Additional subcortical activation was apparent in the thalamus (Mouthon et al., 2018). Consistent cortical activations across all three studies were found in the SMA and superior temporal gyrus. Furthermore, activations in the occipital and cingulate cortex were shown albeit at difference locations across studies. Lastly, the reactive postural control > rest contrast revealed activations in the bilateral SMA, bilateral putamen, cerebellum, and premotor cortex (Taube et al., 2015; Mouthon et al., 2018).

3.2. Quantitative analysis

3.2.1. Experimental characteristics

Fourteen eligible experiments coming from 11 studies (out of the 14 studies included in the qualitative analysis) were identified. After pooling experiments per study (some studies contained multiple experiments), our main ALE meta-analysis involved 162 subjects with on average 14.7 subjects per experiment (for overview characteristics of the included experiments see **Supplementary** Table 1). Malouin et al. (2003) used a small volume correction and therefore the foci in this study that did not met the statistical threshold for the rest of the brain were excluded (inferior parietal lobule, dorsolateral prefrontal cortex, dorsal premotor cortex and posterior cingulate cortex). Two experiments used PET and nine used fMRI.

3.2.2. Main ALE meta-analysis: posture-related brain activations

Our main analysis revealed that a spatial convergence of foci was present in the right anterior cerebellar lobe (cluster 1) and the middle occipital gyrus (cluster 2, see Fig. 3 and **Supplementary Table 2)**. Four experiments contributed to cluster one, which was located in the cerebellar anterior lobe (culmen; Slobounov et al., 2006b; Ferraye et al., 2014; Jahn et al., 2004; Mouthon et al., 2018). Four experiments contributed to cluster 2 in the middle occipital lobe, which also comprised a small part of the inferior and middle temporal gyri (Slobounov et al., 2006a, b; Taube et al., 2015; Mouthon et al., 2018).

The addition of the two PET studies (in which actual postural tasks were performed) with a limited field of view in a supplementary analysis revealed consistent activation in two cerebellar clusters, situated in the anterior and posterior cerebellar lobes and in the middle occipital gyrus (see **Supplementary** Fig. 1 in the **Supplementary ALE meta-analysis)**. The new cerebellar cluster revealed in the supplementary analysis was located in the anterior and posterior cerebellar vermis.

3.2.3. Sub-analysis: simulation of posture

The sub-analysis, in which studies using simulation of posture were included only, revealed the same clusters as the main analysis, but with bigger volumes (anterior cerebellum cluster 1: 840 mm^3 with one peak at 14 −44 −20 (x y z coordinates); middle occipital lobe cluster 2: 1024 mm^3 with one peak at −44 −74 0 (x y z coordinates)). The studies that contributed to these clusters were the same as in the main analysis.

3.2.4. Robustness against publication bias

Considering that four studies contributed to each cluster from the main analysis, the FSN upper bound was set at 29. The minimum FSN was set at 1. The FSN for cluster 1 (anterior cerebellum lobe) was 1 and the FSN for cluster 2 (middle occipital gyrus) was 3.

4. Discussion

The aim of the present systematic review was to better understand the neural foundation for postural control in healthy adults by providing an overview of the posture-related functional neuroimaging literature published to date. Despite the heterogeneous methodologies employed, consistent activation was identified in the anterior cerebellum (cluster 1), likely reflecting the central role of the cerebellum in postural control (quantitative analysis). This is largely in agreement with a recently published systematic review on structural neuroimaging studies, showing that the cerebellum was also delineated as the area most highly associated with behavioral outcomes of postural control (Surgent et al., 2019). Our qualitative analysis further revealed that, in

Table 1
Functional neuroimaging studies of postural tasks in healthy adults.

Study	Subjects	Technique Method	Postural and control tasks	Contrasts	Multiple comparison correction	Key findings[a]
Static balance						
Ouchi et al. (1999)	N = 8 mean (SD) age = 31.8 (6.5) y	PET Stance	Static balance: actual bilateral stance during scanning. Control task: supine (lying).	Static balance > control task Bilateral stance eyes closed > eyes open	yes	Activations in the R cerebellar anterior lobe and anterior vermis and R lingual/inferior occipital gyrus. Additional activations in L+R middle frontal gyrus when eyes were closed. Note: field of view from middle frontal gyrus to lower cerebellum
Ouchi et al. (2001)	N = 8 mean (SD) age = 56.4 (10.8) y	PET Stance	Static balance: actual bilateral stance during scanning. Control tasks: supine and sitting.	Static balance > supine Static balance > sitting	yes	Cerebellar anterior vermis activation across both contrasts and in an additional ROI analysis. Static balance > supine → R lingual/inferior occipital gyrus Note: field of view from lower superior frontal gyrus to upper cerebellum
Malouin et al. (2003)	N = 6 mean (range) age = 55.9 (41-70) y	PET MI	Static balance: MI of bilateral stance during scanning. Control task: relax and think of nothing (i.e. supine).	Static balance > control task	yes	Activations in the L+R dorsal premotor area, L dorsolateral prefrontal cortex, L inferior parietal lobule, L+R precuneus and R posterior cingulate cortex.
Jahn et al. (2004)	N = 13 mean (range) age = 27.3 (21-35) y	fMRI MI	Static balance: MI static stance. Control task: MI lying.	Static balance > control task	no	Predominant activations in the basal ganglia (L putamen/pallidum) and thalamus. Additional activations in the L midbrain, cerebellar vermis and L+R cerebellum, R hippocampus, L superior and inferior frontal gyrus and L+R medial temporal gyrus.
Jahn et al. (2008)	N = 26 mean (range) age = 33.7 (21-61) y	fMRI MI	See Jahn et al. (2004)	See Jahn et al. (2004)	yes	Activation in the dorsal pons including the vestibular nuclei and lateral reticular formation. Additional activations in the cerebellar vermis, L+R cerebellum, L+R thalamus, R hippocampus, L+R superior and medial frontal gyrus, SMA, L +R insula, L+R anterior cingulum, L+R middle temporal gyrus, L+R supramarginal + angular gyrus, L+R precuneus and L medial occipital gyrus.
Jahn et al. (2009)	N = 26 mean (range) age = 33.7 (21-61) y	fMRI MI	See Jahn et al. (2004)	See Jahn et al. (2004)	no	ROI (hippocampus and L+R parahippocampal gyrus) analysis → R anterior hippocampus activation during imagined stance.
Zwergal et al. (2012)	N = 20, age range = 60-78 y (subgroup of a total group of N = 60, but only coordinates of this N = 20 subgroup are reported)	fMRI MI	See Jahn et al. (2004)	See Jahn et al. (2004)	yes	Activations in the cerebellar vermis and pontomesencephalic junction of brainstem, L caudate and L thalamus, L+R inferior, middle and superior frontal gyrus, R insula, R postcentral gyrus, L+R superior temporal gyrus, L fusiform gyrus, L+R lingual gyrus, L precuneus, R cuneus and L+R inferior occpital gyrus.
Karim et al. (2014)	N = 11 mean (SD) age = 75 (5) y	fMRI Stance simulation	Static balance: activate dorsiflexors and plantarflexors in a pattern similar to human upright stance. Elastic bands were aplied to simulate weight-bearing. Control task: dorsiflexor and plantarflexor activation	Static balance > control task	yes	Activations in R corpus callosum, R caudate, L frontal gyrus (superior, medial, middle and inferior) and L anterior cingulate, L superior and L middle temporal gyrus, R insula and L sublobar (extra-nucluear).
Schoberl et al. (2017)	N = 10 No specific age details, but age-matched for primary ortostatic tremor subjects with mean (SD, range) age = 69.9 (5.8, 56-75) y.	PET Stance	Static balance: actual bilateral standing, followed by a PET scan Control task: lying, followed by a PET scan	Static balance > control task	yes	Increased regional cerebal glucose metabolism in the L+R cerebellar (posterior lobes) and vermis, R posterolateral thalamus, L anterior cingulate gyrus and L paracentral lobule. Deactivations in the R secondary visual cortical areas, R lateral primary motor cortex and R prefrontal cortex (i.e. superior frontal gyrus).

(*continued on next page*)

Table 1 (*continued*)

Study	Subjects	Technique Method	Postural and control tasks	Contrasts	Multiple comparison correction	Key findings[a]
Dynamic balance						
Ouchi et al. (1999)	N = 8 mean (SD) age = 31.8 (6.5) y	PET Stance	Dynamic balance: unilateral stance and tandem stance during scanning. Control task: supine (lying).	Standing postures > control task Tandem stance > bilateral stance	yes	All contrasts showed activation in the cerebellar anterior vermis. Standing on one foot → increased activation in the cerebellar R anterior and R posterior lobes. Tandem stance > supine → posterior vermis and R inferior occipital and R inferior temporal gyrus. Tandem stance > bilateral stance → L midbrain (red nucleus) and posterior vermis (+ trend thalamus)
Slobounov et al. (2006a)	N = 18 age range = 21-25 y Stable (n = 12) and less stable (n = 6) sub groups were determined based on maximal AP sway in relation to feet length.	fMRI MI AO	Dynamic balance: MI+AO of an anteroposterior swaying computer-animated human body model. A button had to be pressed when the body model was in an unstable position. Control task: AO of the same anteroposterior swaying human body model while randomly pressing a button.	Dynamic balance > control task	no	Stable group → R caudate, R putamen, L+R parietal cortex, L+R cerebellum (including vermis), anterior cingulate and L occipital lobe. Less stable group → no brain activation.
Ferraye et al. (2014)	N = 20 mean (SD) age = 20.2 (1.8) y	fMRI MI	Dynamic balance: swaying in the anteroposterior direction on a balance board. Control task: matched visual control task.	Dynamic balance > control task	yes	Increased activity in the cerebellar vermis , L+R putamen, L lateral globus pallidus, L+R thalamus, L superior parietal lobe, R medial frontal gyrus (SMA), L precentral gyrus (dorsal premotor cortex), L+R middle cingulate cortex and L insula. ROI analysis → activation in the R MLR and R pons A psychophysiological interaction method with volumes of interest → increased coupling L thalamus with L lateral globus pallidus and R SMA with R MLR.
Reactive balance						
Slobounov et al. (2006b)	N = 12 age range = 21-25 y	fMRI MI	Reactive balance: viewing an animation a virtual room oscillating at various patterns (i.e. visual field motion) while pretending to stand in the room (i.e. MI). Control task: viewing stationary picture of virtual room while pretending to stand in the room (static balance).	Reactive balance > static balance	no	Brain activations were found in response to 3 patterns of low frequency visual field motion. Brain areas that were active in 2 out of 3 conditions → L+R cerebellum (including vermis), R middle temporal visual area, L+R superior temporal sulcus, L+R parietal cortex, L+R anterior prefrontal cortex, R precentral gyrus, SMA, L+R anterior cingulate cortex and L occipital lobe.
Taube et al. (2015)	N = 16 mean (SD, range) age = 27 (2.81, 20-37) y	fMRI MI AO	Reactive balance: mediolateral perturbation on a laterally tilting surface Control task: static stance Both tasks were performed during MI, AO, and AO+MI	Comparison of MI/AO/AO+MI Simple effects: reactive balance > rest Reactive balance > static balance	yes	Strongest activations during AO+MI. Simple effects (AO+MI) → activation in SMA, L+R cerebellum, L+R putamen, L+R ventral and dorsal premotor cortex, L+R superior temporal gyrus, L+R inferior frontal gyrus, L+R insula and L+R visual cortex. ROI M1: L M1. Reactive balance: (AO+MI) → activation in the L+R cerebellum, SMA, L inferior occipital gyrus, R insula, R middle temporal/fusiform gyrus and L+R superior temporal gyrus.

(*continued on next page*)

Table 1 (*continued*)

Study	Subjects	Technique Method	Postural and control tasks	Contrasts	Multiple comparison correction	Key findings[a]
Mouthon et al. (2018)	N = 16 mean (SD) age: 72 (5) y	fMRI MI AO	See Taube et al. (2015)	See Taube et al. (2015)	yes	MI > AO → deactvation subcortical areas. Simple effects analysis (AO + MI) → L cerebellum (lobules I-IV and VI), L + R putamen, L + R SMA, L + R M1, R premotor cortex and L + R prefrontal cortex. Reactive balance (AO + MI) → avtivation in the L + R cerebellum (including vermis), L + R thalamus, L + R SMA, R precentral gyrus, R inferior and superior frontal gyrus, R middle cingulate cortex, R middle and superior temporal gyrus, L + R rolandic operculum, L middle occipital gyrus, L insula lobe, L paracentral lobule and L calcarine gyrus.

Abbreviations: PET = positron emmision tomography; fMRI = functional magentic resonance imaging; MI = motor imagery; AO = action observation; ROI = region of interest; y = years.
Abbreviations brain regions: SMA = supplementary motor area; MLR = mesencephalic locomotor region; M1 = primary motor cortex; R = right ; L = left; L + R = bilateral.
[a] Key findings result from whole brain analyses, unless stated otherwise.

accordance with animal work (Takakusaki, 2017), a wide variety of other brain regions were caught up in human postural control. Key nodes that were repeatedly activated included the brainstem, basal ganglia, thalamus and several cortical regions (qualitative analysis).

4.1. Cerebellar involvement in postural control

Although our main finding, that the cerebellum has a crucial role in postural control, is mostly based on neuroimaging conducted in a supine position, it is also extensively supported by animal work (Takakusaki, 2017) and cerebellar pathology in humans (Morton and Bastian, 2007; Van de Warrenburg et al., 2005). The cerebellum consists of different functional zones, which influence posture in a different manner. First, the vermis integrates spinal, vestibular and visual input and influences vestibulospinal and reticulospinal motor tracts important for postural control (Morton and Bastian, 2004). Second, the intermediate zone integrates spinal and cortical input and affects motor commands through projections to cortical motor areas and the red nucleus. A main function of the intermediate zone might be regulating precise movements (Morton and Bastian, 2004). Third, the lateral hemispheres are likely involved in adjusting and recalibrating motor behavior in novel or complex circumstances or when visual guidance is required through corticopontocerebellar loops (Morton and Bastian, 2004). The vermis seems to be the cerebellar zone most involved in postural control. A cluster in the anterior and posterior vermis was revealed in the supplementary meta-analysis. Although this analysis included two additional PET studies in which actual postural tasks were performed, these results should be interpreted with caution due to the limited field of view in these two PET studies. Furthermore, our qualitative analysis showed that 10 out of the 13 studies covering the cerebellum reported activation in the vermis. We will elaborate on the role of the cerebellar vermis in postural control below.

4.1.1. Cerebellar vermis projections to brainstem nuclei

Animal work has shown that the anterior and posterior cerebellar vermis project to the fastigial nucleus (Voogd and Glickstein, 1998; Courville and Diakiw, 1976), through which the vestibular nuclei, the pedunculopontine nucleus (PPN) and the pontomedullary reticular formation, which are involved the control of posture, can be modulated (Takakusaki, 2017; MacKinnon, 2018; McCall et al., 2017; Zhang et al., 2016). The anterior vermis also projects directly to the vestibular nuclei (Voogd, 2016). Of these direct and indirect brainstem projections from the vermis, the connections between the lateral vestibular nucleus and anterior vermis are probably the most pronounced. The lateral vestibular nucleus preferentially targets, via spinal premotor interneurons (McCall et al., 2017), the slow motoneurons of extensor axial muscles critical for maintaining upright posture (Basaldella et al., 2015). When posture is perturbed, the lateral vestibular nucleus activates extensor muscles followed by co-activation of antagonist muscles (Murray et al., 2018). Interestingly, the simultaneous activation of the vermis and lateral vestibular nucleus during imagined stance also highlighted the existence of a functional connection between these structures in humans (Jahn et al., 2008). The pontomedullary reticular formation and the PPN are involved in the regulation of muscle tone (Takakusaki, 2017) and as such are likely involved in the control of postural sway. Indeed, sway control systems returned to normal in patients with Parkinson's disease after receiving PPN deep brain stimulation (Perera et al., 2018). The MLR was activated during motor imagery of a dynamic postural control task (Ferraye et al., 2014). The MLR is a complex structure, which consists of the cuneiform nucleus and the PPN. Within the MLR, it is likely that functionally distinct but adjacent sub-regions exist that are responsible for the control of standing and locomotion (Takakusaki, 2017). Indeed, MLR activity during an imagined dynamic postural task was found nearby MLR activity generated during imagined gait (Snijders et al., 2011; Bakker et al., 2008; Ferraye et al., 2014). Bohnen et al. (2009) found an association between PPN

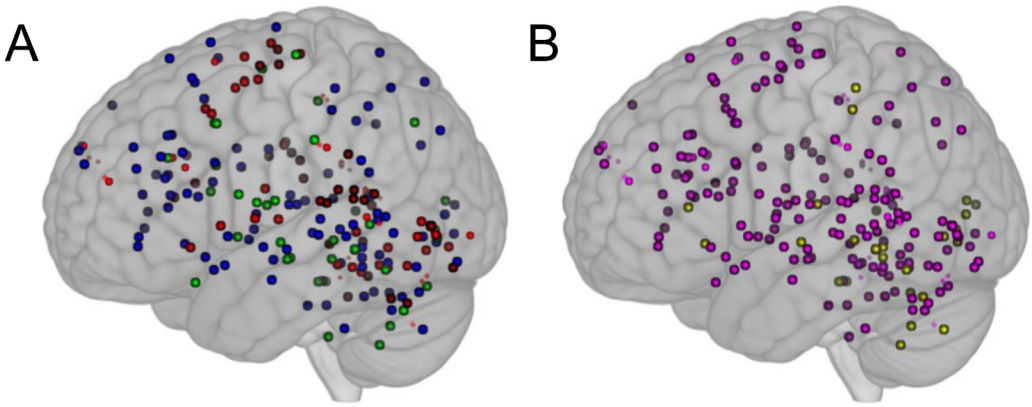

Fig. 2. Foci per postural domain and per method (actual postural task/postural simulation) Foci reported in the studies included in the qualitative analysis. A)● Static postural control; ● dynamic postural control; ●reactive postural control. B)●Actual postural control; ●simulation of postural control.

degeneration and falls in people with Parkinson's disease. In line, a connection was demonstrated between postural deficits and lesions of the cholinergic part of the PPN in macaques (Karachi et al., 2010). Finally, greater PPN volumes were associated with better regulation of postural control in healthy adults (Boisgontier et al., 2017). We speculate that one of the reasons that the cerebellar vermis came out as a key node in our analyses may be explained by the close and ongoing communication with the abovementioned brainstem nuclei.

Our qualitative analysis indeed revealed posture-related brainstem activation in the vestibular nuclei (Jahn et al., 2008), the MLR (Ferraye et al., 2014), the pontomesencephalic junction (Zwergal et al., 2012), and the red nucleus (Ouchi et al., 1999). Brainstem activity was not highlighted by our quantitative analysis. However, it should be noted that collecting reliable fMRI data from the brainstem network is difficult, due to the small size, close proximity and physiological noise coming from these structures (Beissner, 2015).

4.1.2. Cerebellar activation during postural tasks

Activity in the cerebellum was present during actual and simulated postural tasks. During actual postural tasks, the cerebellum may have been involved in integrating spinal and cortical input, derived from maintaining the standing position. However, during simulation while being in a supine position, upright posture-related sensory feedback was absent. Intriguingly, the cerebellar cluster 1, as well as the additional cerebellar cluster present in the supplementary meta-analysis which included the two PET studies with a limited field of view, fell within cerebellar functional regions shown to be involved in motor planning, based on a recent functional parcellation of the cerebellum (King et al., 2019). Motor imagery is indeed thought to pick up the neural correlates of the planning and preparation phase of movement (Cunnington et al., 1996). In monkeys, it was found that cortical motor areas project to the vermis (Coffman et al., 2011), indicating that the vermis receives motor-related information. In accordance, the cerebellar clusters could thus reflect activity generated during the planning

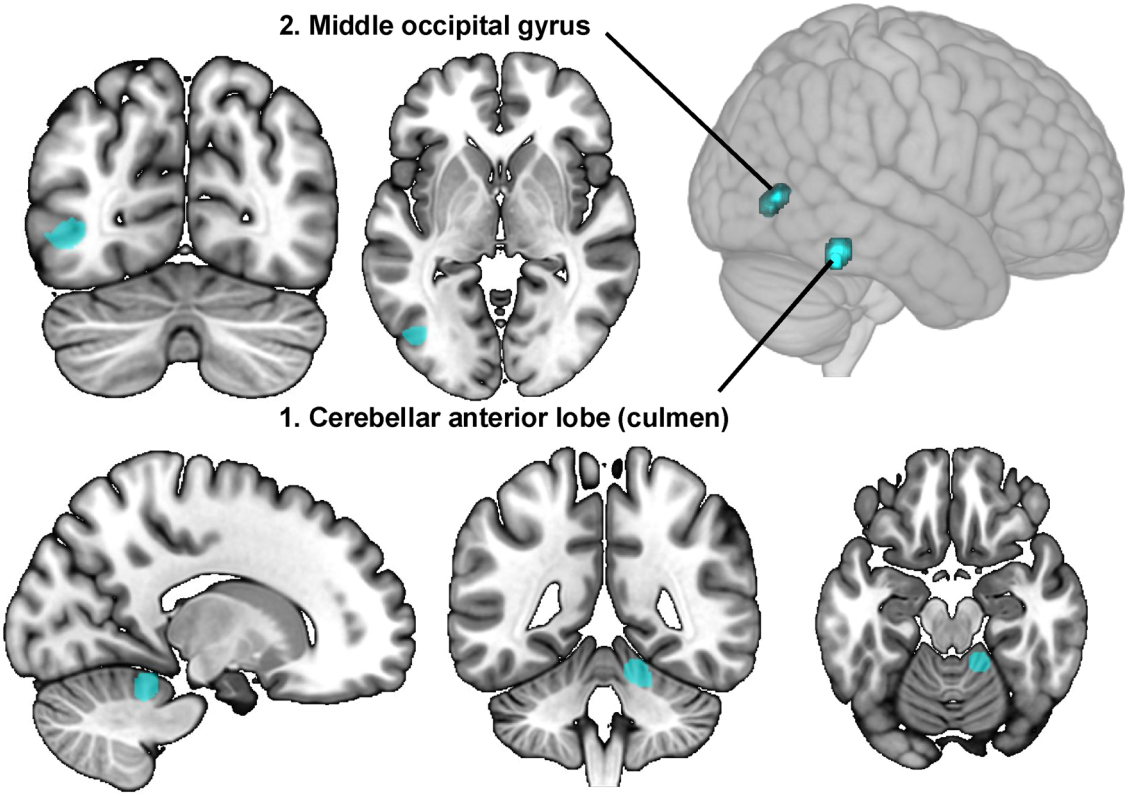

Fig. 3. Clusters main ALE meta-analysis.
Brain areas involved in postural control as determined by the main ALE meta-analysis.

phase of postural control. Alternatively, Kilteni et al. (2018) showed that during motor imagery, similar to overt movements, predictions about the sensory consequences of movements are formed. These so-called forward models embed a copy of the motor command (efference copy) as a reference for the ensuing motor performance (Wolpert et al., 1998; Therrien and Bastian, 2019). Although the precise neural foundation of this process is unknown, it is thought that forward modelling takes place in the cerebellum (Krakauer et al., 2019; Tanaka et al., 2019). However, an ALE meta-analysis on neuroimaging studies in which unexpected sensory feedback was induced, did not reveal consistent activations in the cerebellum (Johnson et al., 2019). Thus, the notion that the cerebellar vermis contribution during simulation of postural tasks may signify the prediction of sensory consequences of the imagined postural movements, remains speculative.

4.2. Occipital involvement in postural control

The quantitative analysis also indicated that the middle occipital gyrus (cluster 2) was activated consistently across postural tasks. This cluster was found to be robust against publication bias. This cluster may reflect the critical role of visual information processing for postural control (MacKinnon, 2018). However, the experiments that contributed to the cluster in the occipital cortex, involved contrasts in which watching static motion (e.g. person standing still) was compared against watching dynamic motion (e.g. a person counteracting balance perturbations). The cluster in the middle occipital gyrus also included voxels in the inferior and middle temporal gyrus, as part of the MT/V5 region (Sunaert et al., 1999; Dupont et al., 1994). Earlier work indicated that motion versus static visual input led to MT/V5 activation (Sunaert et al., 1999; Biehl et al., 2017). Therefore, it is plausible that this cluster was due to perceiving motion rather than being specific for postural control.

4.3. Interpretation of key findings in relation to postural domain

In the next sections, we will focus on structures involved in postural control reported in the qualitative analysis.

4.3.1. Static postural control

Static postural control was studied in nine studies through actual stance, motor imagery or stance simulation through plantar/dorsiflexion. One study used an ROI analysis and was not taken into account in the number of studies included in this section (Jahn et al., 2009). Static postural control activated the brainstem, cerebellum, thalamus, basal ganglia (caudate nucleus, putamen and pallidum), hippocampus and the cortex. Brainstem activations during imagined stance were found in the midbrain (Jahn et al., 2004), pons (including the lateral vestibular nucleus and lateral reticular formation: Jahn et al., 2008) and pontomesencephalic junction (Zwergal et al., 2012). These brainstem activations were accompanied by cerebellar vermis activations (see section 4.1.1 on the meaning of the communication between these structures).

Thalamus activation was reported in four of the eight studies in which a static postural task was performed (Jahn et al., 2004, 2008; Zwergal et al., 2012; Schoberl et al., 2017). Studies in people with Parkinson's disease and in people with progressive supranuclear palsy indicated that the influence of thalamic dysfunction is most prominent in quiet stance when sensory information is disturbed (Zwergal et al., 2011; Muller et al., 2013). In addition, Van Impe et al. (2012) found a relation between lower white matter integrity of the anterior thalamic radiation and poorer postural performance under sensory manipulation in older adults. These findings support the notion that the thalamus, considered a relay station for sensory information, also contributes to the integration of posture-related sensory input (Van Impe et al., 2012).

Putamen (Jahn et al., 2004) or caudate (Zwergal et al., 2012; Karim et al., 2014) activity within the basal ganglia was reported in three of the eight studies. The role of these nuclei in postural control are testified by the fact that people with Parkinson's disease, in whom these structures are affected, suffer not only from postural instability but also report frequent falling (Fasano et al., 2017). Also, animal work indicated that the basal ganglia output centers send strong inhibitory projections to the brainstem nuclei that regulate postural muscle tone (Takakusaki et al., 2003).

Three studies reported hippocampus activation during static postural control (Jahn et al., 2004, 2008; Jahn et al., 2009). Although the hippocampus is not typically associated with postural control, structural brain metrics of the hippocampus were also found to be frequently related to postural performance (Surgent et al., 2019). In another study, increased hippocampal volume was associated with situations when postural control relied more on vestibular and proprioceptive sensory information in healthy older adults (Beauchet et al., 2016). Finally, motor imagery of stance in blind and vestibular-deprived subjects indicated that the anterior hippocampus processed vestibular information and that the parahippocampal cortex and fusiform gyrus were involved in the utilization of visual information (Jahn et al., 2009). The hippocampus thus seems to be involved in the processing of sensory information critical for maintaining upright posture.

Static postural control has long been considered to be highly automated and heavily dependent on postural reflexes and thus requiring little cortical engagement (Morningstar et al., 2005). However, an electroencephalography study showed that cortical activity is temporally coupled to the maxima of natural static postural sway, even when standing still in healthy young adults (Varghese et al., 2015). Obviously, PET and MRI functional neuroimaging lack the temporal resolution to reveal such involvement. Indeed, Surgent et al. (2019) reported that structural brain metrics in the frontal cortex were more often associated with dynamic than static postural control outcomes. Brain activations during actual stance were found in the occipital (Ouchi et al., 1999), anterior cingulate and paracentral lobule (Schoberl et al., 2017), while deactivations were found in the occipital and frontal cortex (Schoberl et al., 2017). In contrast, postural simulation studies reported widespread cortical activation in frontal, parietal temporal and occipital regions as well as in the cingulate cortex. A recent ALE meta-analysis indicated that motor imagery activated more attention-related brain regions than movement execution, especially in the dorsolateral prefrontal cortex (Hardwick et al., 2018). We speculate that motor imagery does not adequately pick up the automatic postural control networks, but that a more conscious control of posture is at play during simulated postural tasks resulting in widespread cortical activation. Postural simulation studies also reported activated foci at the temporoparietal junction in close proximity to the parietal insular vestibular cortex (Jahn et al., 2004, 2008; Karim et al., 2014). Vestibular cortical areas are involved in perception of verticality and self-motion (Brandt and Dieterich, 1999). Activity in the temporoparietal junction may also reflect various aspects of self-consciousness, such as self-location and the first-person perspective, or dealing with conflicting vestibular and visual gravitational cues, e.g. lying supine in an MRI scanner while watching another person being upright (Ionta et al., 2011). Overall, activity in the temporoparietal junction indicates that this region is processing vestibular and multisensory information (Ionta et al., 2011), important for maintaining an upright posture.

4.3.2. Dynamic postural control

Three studies measured brain activation related to dynamic postural control (Ouchi et al., 1999; Slobounov et al., 2006a; Ferraye et al., 2014). Increased brainstem activation was reported in the red nucleus (Ouchi et al., 1999) and the MLR (Ferraye et al., 2014). The red nucleus, which constitutes the origin of the rubrospinal tract, was earlier found to be implicated during postural corrections in cats based on reflex mechanisms (Zelenin et al., 2010). In this review, red nucleus activity was demonstrated to be particularly evident during tandem stance using an upright PET scanner (Ouchi et al., 1999). Postural

corrections are expected to be more pronounced during more dynamic tasks such as standing on of foot or tandem stance compared to static stance or lying. Of note, red nucleus activity was only present when dynamic postural control was contrasted against static postural control and not when compared with lying (Ouchi et al., 1999). The role of the MLR in postural control has been discussed above (see *4.1.1 Cerebellar vermis projections to brainstem nuclei*). Cerebellar vermis and striatal activity also contributed to dynamic postural control (Slobounov et al., 2006a; Ferraye et al., 2014). Given the strong inhibitory projections to the brainstem nuclei (Takakusaki, 2017), suppressing muscle tone and locomotion, it is not surprising that basal ganglia nodes, such as the striatum, contribute to dynamic postural control. Indeed, people with Parkinson's disease, especially those experiencing freezing of gait, were found to have impaired dynamic postural control (Bekkers et al., 2018).

During dynamic postural control tasks, also cortical activations were noted in the frontal, cingulate and parietal lobules (Ferraye et al., 2014; Slobounov et al., 2006a). In particular, the SMA, which in recent reviews has been implicated in movement initiation (Takakusaki, 2017; MacKinnon, 2018) and gait-related dynamic posture (Wittenberg et al., 2017) was activated in both MRI studies (Ferraye et al., 2014; Slobounov et al., 2006a), while it was probably outside the axial field of view of the PET study. Involvement of the cingulate and parietal cortices in a neural network calibrating online postural responses were found with electroencephalography (Goel et al., 2019). The cingulate cortex is known to be responsible for the coordination of complex movements (Wenderoth et al., 2005) and could as such be involved in dynamic postural control. Importantly, the abovementioned results refer to activations without taking into account the functional connections between these brain regions. Only Ferraye et al. (2014) performed a connectivity analysis, showing that an integrated dynamic postural control network was present, which was determined by increased coupling between the thalamus and globus pallidus and between the SMA and MLR (Ferraye et al., 2014).

4.3.3. Reactive postural control

Three eligible studies elicited brain activations during postural reactions through simulating mechanic (i.e. surface translation) or visual (i.e. visual field motion) perturbations (Slobounov et al., 2006b; Taube et al., 2015; Mouthon et al., 2018). The SMA, which is thought to play a role in the release and timing of postural responses (Jacobs et al., 2009), was activated in all three studies. This finding is underscored by recent work showing that preparatory activity in this region, as measured with electroencephalography, increased with higher postural demands (Solis-Escalante et al., 2019). Although the earlier phases of postural reactions (i.e. short latency and medium latency components) likely depend on the spinal cord and brainstem circuits, SMA input can influence the long latency component of postural reactions, e.g. by controlling step placement (Jacobs and Horak, 2007; Bolton, 2015). A systematic review focusing on electroencephalography studies also found that SMA-activity was modulated during postural challenges (Wittenberg et al., 2017). Besides the SMA, the cerebellum was active in all three studies, as well the temporal and occipital lobe, probably reflecting the sensory processing required for ensuring the response adequacy. A cerebellar-cortical loop was proposed to be involved in adapting postural responses based on prior experience (Jacobs and Horak, 2007). Recently, it was shown with PET imaging that the cerebellum and SMA were operational during the adjustment and fine-tuning of locomotor patterns during split-belt perturbations, which also contain a postural reaction component (Hinton et al., 2019).

4.4. Strengths and limitations of the ALE meta-analysis

To the best of our knowledge, this is the first meta-analysis in which functional neuroimaging studies addressing postural control were brought together. Meta-analyses provide an objective approach to pool data from previous studies leading to superior statistical power over individual studies. However, their robustness is determined by the number of included studies and their methodological coherence. The present meta-analysis should be labeled as exploratory, as 11 experiments were included which is below the recommended minimal number of experiments (17–20) required to achieve sufficient power for detecting moderate statistical effects (Eickhoff et al., 2016). Indeed, the main analysis did not reveal posture-related activation in frontal or parietal cortical areas, basal ganglia, thalamus or brainstem, which would be expected based on animal research (Takakusaki, 2017) and our qualitative analysis. As only one out of 11 experiments reported deactivations in the whole brain, we also limited ourselves to an activation rather than a deactivation meta-analysis. Future meta-analyses including a greater number of studies with less heterogeneity across study paradigms can provide further insight in the neural control of posture by revealing possible deactivation patterns and by removing false positives and revealing false negative clusters. Finally, this study was not pre-registered in Prospero, but robustness against publication bias was assessed according to recent guidelines (Acar et al., 2018).

4.5. Recommendations for future research

This review delineated posture-related brain activations in healthy young and older adults. We did not compare between age groups, as we aimed to characterize the neural underpinnings of healthy postural control in a wide age range of subjects. Studying posture-related brain activations in clinical and older populations prone to falling will strengthen the clinical impact of these findings (Zwergal et al., 2011; Schoberl et al., 2017; Jahn et al., 2009). Identifying abnormal brain activity, with reference to the present findings, may inform future rehabilitation programs targeting falls and postural instability. In line, it was recently demonstrated that intermittent θ-burst stimulation over the cerebellum improved postural control performance in people with hemiparesis due to stroke (Koch et al., 2019).

The studies included in the present review used a variety of postural simulation tasks. Interestingly, combined action observation and motor imagery showed more widespread brain activations compared to action observation or motor imagery alone (Mouthon et al., 2018; Taube et al., 2015; Vogt et al., 2013). Especially, older adults prone to falling were found to be more dependent on visual input to elicit an internal representation of the postural task compared to young subjects (Mouthon et al., 2018). Therefore, action observation plus motor imagery appears to be the most optimal method for future studies to investigate postural control network in a lying position.

In this review, only one study used an effective connectivity analysis (Ferraye et al., 2014) enabling interpretation of the underlying brain networks. To fully uncover the connectivity between brain regions which underlie postural control, a next step would be to utilize functional connectivity and network-based analyses, such as graph theory, dynamic causal modelling or granger causality mapping (Nackaerts et al., 2019). The outcomes of the present review may serve as a guide for selecting appropriate regions of interest for those future studies.

Finally, because most brain activations were derived from postural simulations, it was not possible to establish relationships between brain activity and postural outcome measures. However, interpretation of imaging-behavior associations derived from simulation will benefit from also collecting sensitive behavioral outcomes outside the scanner in static, dynamic and reactive postural contexts. By calculating associations between neural and actual behavioral outcomes, further insight may be gained into the specific nature of postural abnormities, albeit indirectly. As such, we recommend future studies to capture postural control measures using posturography. Furthermore, multimodal neuroimaging techniques combining MRI in supine, with electroencephalography or PET findings that can measure brain activation while standing, will move this field further forward. Studies which combine mobile techniques such as electroencephalography or functional near-infrared spectroscopy with electromyography or

posturography, will also help to unravel brain-body interactions during actual postural tasks (Zandvoort et al., 2019). The influence of a cognitive dual-task on postural control can also be studied using these mobile neuroimaging techniques (Marusic et al., 2019).

5. Conclusion

This systematic review revealed key nodes of the human postural control network, identified by being repeatedly activated during brain imaging of various postural tasks. Nodes included the brainstem, basal ganglia, thalamus and several cortical regions. The meta-analysis highlighted that the anterior cerebellum was most consistently involved. The central role of the cerebellum in conjunction with other supraspinal centers may reflect the extremely dynamic processing required for constant adaptation to and modulation of external demands to maintain a stable upright position. Future studies that combine imaging and posturography are needed to further elucidate how the brain interacts with the complex demands of postural control.

Declaration of Competing Interest

None.

Acknowledgements

BWD was supported by the Research Foundation Flanders (FWO) (grant number G.0867.15). MG was supported by a KU Leuven Internal Funds Postdoctoral Mandate. This project has received funding from the European Union's Horizon 2020 research and innovation programme under the Marie Skłodowska-Curie grant agreements No. 838576 (MG), No. 702784 (RMH) and No. 721577 (VdR).

Appendix A. Supplementary data

Supplementary material related to this article can be found, in the online version, at doi:https://doi.org/10.1016/j.neubiorev.2020.04.028.

References

Acar, F., Seurinck, R., Eickhoff, S.B., Moerkerke, B., 2018. Assessing robustness against potential publication bias in Activation Likelihood Estimation (ALE) meta-analyses for fMRI. PLoS One 13, e0208177. https://doi.org/10.1371/journal.pone.0208177.

Ambrose, A.F., Paul, G., Hausdorff, J.M., 2013. Risk factors for falls among older adults: a review of the literature. Maturitas 75, 51–61. https://doi.org/10.1016/j.maturitas.2013.02.009.

Bakker, M., De Lange, F.P., Helmich, R.C., Scheeringa, R., Bloem, B.R., Toni, I., 2008. Cerebral correlates of motor imagery of normal and precision gait. Neuroimage 41, 998–1010. https://doi.org/10.1016/j.neuroimage.2008.03.020.

Basaldella, E., Takeoka, A., Sigrist, M., Arber, S., 2015. Multisensory signaling shapes vestibulo-motor circuit specificity. Cell 163, 301–312. https://doi.org/10.1016/j.cell.2015.09.023.

Beauchet, O., Barden, J., Liu-Ambrose, T., Chester, V.L., Szturm, T., Allali, G., 2016. The relationship between hippocampal volume and static postural sway: results from the GAIT study. Age Dordr. (Dordr) 38, 19. https://doi.org/10.1007/s11357-016-9883-4.

Beissner, F., 2015. Functional MRI of the brainstem: common problems and their solutions. Clin. Neuroradiol. 25 (Suppl 2), 251–257. https://doi.org/10.1007/s00062-015-0404-0.

Bekkers, E.M.J., Dijkstra, B.W., Heremans, E., Verschueren, S.M.P., Bloem, B.R., Nieuwboer, A., 2018. Balancing between the two: Are freezing of gait and postural instability in Parkinson's disease connected? Neurosci. Biobehav. Rev. 94, 113–125. https://doi.org/10.1016/j.neubiorev.2018.08.008.

Biehl, S.C., Andersen, M., Waiter, G.D., Pilz, K.S., 2017. Neural changes related to motion processing in healthy aging. Neurobiol. Aging 57, 162–169. https://doi.org/10.1016/j.neurobiolaging.2017.05.018.

Bohnen, N.I., Jahn, K., 2013. Imaging: What can it tell us about parkinsonian gait? Mov. Disord. 28, 1492–1500. https://doi.org/10.1002/mds.25534.

Bohnen, N.I., Muller, M.L., Koeppe, R.A., Studenski, S.A., Kilbourn, M.A., Frey, K.A., Albin, R.L., 2009. History of falls in Parkinson disease is associated with reduced cholinergic activity. Neurology 73, 1670–1676. https://doi.org/10.1212/WNL.0b013e3181c1ded6.

Boisgontier, M.P., Cheval, B., Chalavi, S., van Ruitenbeek, P., Leunissen, I., Levin, O., Nieuwboer, A., Swinnen, S.P., 2017. Individual differences in brainstem and basal ganglia structure predict postural control and balance loss in young and older adults. Neurobiol. Aging 50, 47–59. https://doi.org/10.1016/j.neurobiolaging.2016.10.024.

Bolton, D.A., 2015. The role of the cerebral cortex in postural responses to externally induced perturbations. Neurosci. Biobehav. Rev. 57, 142–155. https://doi.org/10.1016/j.neubiorev.2015.08.014.

Brandt, T., Dieterich, M., 1999. The vestibular cortex. Its locations, functions, and disorders. Ann. N. Y. Acad. Sci. 871, 293–312. https://doi.org/10.1111/j.1749-6632.1999.tb09193.x.

Burns, E., Kakara, R., 2018. Deaths from falls among persons aged ≥65 years - United States, 2007-2016. MMWR Morb. Mortal. Wkly. Rep. 67, 509–514. https://doi.org/10.15585/mmwr.mm6718a1.

Coffman, K.A., Dum, R.P., Strick, P.L., 2011. Cerebellar vermis is a target of projections from the motor areas in the cerebral cortex. Proc Natl Acad Sci U S A 108, 16068–16073. https://doi.org/10.1073/pnas.1107904108.

Courville, J., Diakiw, N., 1976. Cerebellar corticonuclear projection in the cat. The vermis of the anterior and posterior lobes. Brain Res. 110, 1–20. https://doi.org/10.1016/0006-8993(76)90205-5.

Cunnington, R., Iansek, R., Bradshaw, J.L., Phillips, J.G., 1996. Movement-related potentials associated with movement preparation and motor imagery. Exp. Brain Res. 111, 429–436. https://doi.org/10.1007/bf00228732.

Dupont, P., Orban, G.A., De Bruyn, B., Verbruggen, A., Mortelmans, L., 1994. Many areas in the human brain respond to visual motion. J. Neurophysiol. 72, 1420–1424. https://doi.org/10.1152/jn.1994.72.3.1420.

Eickhoff, S.B., Laird, A.R., Grefkes, C., Wang, L.E., Zilles, K., Fox, P.T., 2009. Coordinate-based activation likelihood estimation meta-analysis of neuroimaging data: a random-effects approach based on empirical estimates of spatial uncertainty. Hum. Brain Mapp. 30, 2907–2926. https://doi.org/10.1002/hbm.20718.

Eickhoff, S.B., Bzdok, D., Laird, A.R., Kurth, F., Fox, P.T., 2012. Activation likelihood estimation meta-analysis revisited. Neuroimage 59, 2349–2361. https://doi.org/10.1016/j.neuroimage.2011.09.017.

Eickhoff, S.B., Nichols, T.E., Laird, A.R., Hoffstaedter, F., Amunts, K., Fox, P.T., Bzdok, D., Eickhoff, C.R., 2016. Behavior, sensitivity, and power of activation likelihood estimation characterized by massive empirical simulation. Neuroimage 137, 70–85. https://doi.org/10.1016/j.neuroimage.2016.04.072.

Evans, A.C., Kamber, M., Collins, D.L., MacDonald, D., 1994. An MRI-based probabilistic atlas of neuroanatomy. In: Shorvon, S.D., Fish, D.R., Andermann, F., Bydder, G.M., Stefan, H. (Eds.), Magnetic Resonance Scanning and Epilepsy. Springer US., Boston, MA, pp. 263–274.

Fasano, A., Canning, C.G., Hausdorff, J.M., Lord, S., Rochester, L., 2017. Falls in Parkinson's disease: a complex and evolving picture. Mov. Disord. 32, 1524–1536. https://doi.org/10.1002/mds.27195.

Ferraye, M.U., Debu, B., Heil, L., Carpenter, M., Bloem, B.R., Toni, I., 2014. Using motor imagery to study the neural substrates of dynamic balance. PLoS One 9, e91183. https://doi.org/10.1371/journal.pone.0091183.

Goel, R., Nakagome, S., Rao, N., Paloski, W.H., Contreras- Vidal, J.L., Parikh, P.J., 2019. Fronto-parietal brain areas contribute to the online control of posture during a continuous balance task. Neuroscience 413, 135–153. https://doi.org/10.1016/j.neuroscience.2019.05.063.

Hardwick, R.M., Caspers, S., Eickhoff, S.B., Swinnen, S.P., 2018. Neural correlates of action: comparing meta-analyses of imagery, observation, and execution. Neurosci. Biobehav. Rev. 94, 31–44. https://doi.org/10.1016/j.neubiorev.2018.08.003.

Hartholt, K.A., van Beeck, E.F., Polinder, S., van der Velde, N., van Lieshout, E.M., Panneman, M.J., van der Cammen, T.J., Patka, P., 2011. Societal consequences of falls in the older population: injuries, healthcare costs, and long-term reduced quality of life. J. Trauma 71, 748–753. https://doi.org/10.1097/TA.0b013e3181f6f5e5.

Hinton, D.C., Thiel, A., Soucy, J.-P., Bouyer, L., Paquette, C., 2019. Adjusting gait step-by-step: brain activation during split-belt treadmill walking. NeuroImage 202, 116095. https://doi.org/10.1016/j.neuroimage.2019.116095.

Horak, F.B., 2006. Postural orientation and equilibrium: what do we need to know about neural control of balance to prevent falls? Age Ageing 35 (Suppl 2), ii7–ii11. https://doi.org/10.1093/ageing/afl077.

Ionta, S., Heydrich, L., Lenggenhager, B., Mouthon, M., Fornari, E., Chapuis, D., Gassert, R., Blanke, O., 2011. Multisensory mechanisms in temporo-parietal cortex support self-location and first-person perspective. Neuron 70, 363–374. https://doi.org/10.1016/j.neuron.2011.03.009.

Jacobs, J.V., Horak, F.B., 2007. Cortical control of postural responses. J. Neural Transm. Vienna (Vienna) 114, 1339–1348. https://doi.org/10.1007/s00702-007-0657-0.

Jacobs, J.V., Lou, J.S., Kraakevik, J.A., Horak, F.B., 2009. The supplementary motor area contributes to the timing of the anticipatory postural adjustment during step initiation in participants with and without Parkinson's disease. Neuroscience 164, 877–885. https://doi.org/10.1016/j.neuroscience.2009.08.002.

Jahn, K., Deutschlander, A., Stephan, T., Strupp, M., Wiesmann, M., Brandt, T., 2004. Brain activation patterns during imagined stance and locomotion in functional magnetic resonance imaging. Neuroimage 22, 1722–1731. https://doi.org/10.1016/j.neuroimage.2004.05.017.

Jahn, K., Deutschlander, A., Stephan, T., Kalla, R., Wiesmann, M., Strupp, M., Brandt, T., 2008. Imaging human supraspinal locomotor centers in brainstem and cerebellum. Neuroimage 39, 786–792. https://doi.org/10.1016/j.neuroimage.2007.09.047.

Jahn, K., Wagner, J., Deutschlander, A., Kalla, R., Hufner, K., Stephan, T., Strupp, M., Brandt, T., 2009. Human hippocampal activation during stance and locomotion: fMRI study on healthy, blind, and vestibular-loss subjects. Ann. N. Y. Acad. Sci. 1164, 229–235. https://doi.org/10.1111/j.1749-6632.2009.03770.x.

Johnson, J.F., Belyk, M., Schwartze, M., Pinheiro, A.P., Kotz, S.A., 2019. The role of the cerebellum in adaptation: ALE meta-analyses on sensory feedback error. Hum. Brain Mapp. 40, 3966–3981. https://doi.org/10.1002/hbm.24681.

Karachi, C., Grabli, D., Bernard, F.A., Tande, D., Wattiez, N., Belaid, H., Bardinet, E., Prigent, A., Nothacker, H.P., Hunot, S., Hartmann, A., Lehericy, S., Hirsch, E.C., Francois, C., 2010. Cholinergic mesencephalic neurons are involved in gait and postural disorders in Parkinson disease. J. Clin. Invest. 120, 2745–2754. https://doi.org/10.1172/jci42642.

Karim, H.T., Sparto, P.J., Aizenstein, H.J., Furman, J.M., Huppert, T.J., Erickson, K.I.,

Loughlin, P.J., 2014. Functional MR imaging of a simulated balance task. Brain Res. 1555, 20–27. https://doi.org/10.1016/j.brainres.2014.01.033.
Kilteni, K., Andersson, B.J., Houborg, C., Ehrsson, H.H., 2018. Motor imagery involves predicting the sensory consequences of the imagined movement. Nat. Commun. 9, 1617. https://doi.org/10.1038/s41467-018-03989-0.
King, M., Hernandez-Castillo, C.R., Poldrack, R.A., Ivry, R.B., Diedrichsen, J., 2019. Functional boundaries in the human cerebellum revealed by a multi-domain task battery. Nat. Neurosci. 22, 1371–1378. https://doi.org/10.1038/s41593-019-0436-x.
Koch, G., Bonni, S., Casula, E.P., Iosa, M., Paolucci, S., Pellicciari, M.C., Cinnera, A.M., Ponzo, V., Maiella, M., Picazio, S., Sallustio, F., Caltagirone, C., 2019. Effect of cerebellar stimulation on gait and balance recovery in patients with hemiparetic stroke: a randomized clinical trial. JAMA Neurol. 76, 170–178. https://doi.org/10.1001/jamaneurol.2018.3639.
Krakauer, J.W., Hadjiosif, A.M., Xu, J., Wong, A.L., Haith, A.M., 2019. Motor learning. Compr. Physiol. 9, 613–663. https://doi.org/10.1002/cphy.c170043.
Lancaster, J.L., Tordesillas- Gutierrez, D., Martinez, M., Salinas, F., Evans, A., Zilles, K., Mazziotta, J.C., Fox, P.T., 2007. Bias between MNI and Talairach coordinates analyzed using the ICBM-152 brain template. Hum. Brain Mapp. 28, 1194–1205. https://doi.org/10.1002/hbm.20345.
MacKinnon, C.D., 2018. Sensorimotor anatomy of gait, balance, and falls. Handb. Clin. Neurol. 159, 3–26. https://doi.org/10.1016/b978-0-444-63916-5.00001-x.
Malouin, F., Richards, C.L., Jackson, P.L., Dumas, F., Doyon, J., 2003. Brain activations during motor imagery of locomotor-related tasks: a PET study. Hum. Brain Mapp. 19, 47–62. https://doi.org/10.1002/hbm.10103.
Marusic, U., Taube, W., Morrison, S.A., Biasutti, L., Grassi, B., De Pauw, K., Meeusen, R., Pisot, R., Ruffieux, J., 2019. Aging effects on prefrontal cortex oxygenation in a posture-cognition dual-task: an fNIRS pilot study. Eur. Rev. Aging Phys. Act. 16, 2. https://doi.org/10.1186/s11556-018-0209-7.
McCall, A.A., Miller, D.M., Yates, B.J., 2017. Descending influences on vestibulospinal and vestibulosympathetic reflexes. Front. Neurol. 8, 112. https://doi.org/10.3389/fneur.2017.00112.
Morningstar, M.W., Pettibon, B.R., Schlappi, H., Schlappi, M., Ireland, T.V., 2005. Reflex control of the spine and posture: a review of the literature from a chiropractic perspective. Chiropr. Osteopat. 13, 16-16. https://doi.org/10.1186/1746-1340-13-16.
Morton, S.M., Bastian, A.J., 2004. Cerebellar control of balance and locomotion. Neuroscientist 10, 247–259. https://doi.org/10.1177/1073858404263517.
Morton, S.M., Bastian, A.J., 2007. Mechanisms of cerebellar gait ataxia. Cerebellum 6, 79–86. https://doi.org/10.1080/14734220601187741.
Mouthon, A., Ruffieux, J., Mouthon, M., Hoogewoud, H.M., Annoni, J.M., Taube, W., 2018. Age-related differences in cortical and subcortical activities during observation and motor imagery of dynamic postural tasks: an fMRI study. Neural Plast. 2018, 1598178. https://doi.org/10.1155/2018/1598178.
Muller, M.L., Albin, R.L., Kotagal, V., Koeppe, R.A., Scott, P.J., Frey, K.A., Bohnen, N.I., 2013. Thalamic cholinergic innervation and postural sensory integration function in Parkinson's disease. Brain 136, 3282–3289. https://doi.org/10.1093/brain/awt247.
Muller, V.I., Cieslik, E.C., Laird, A.R., Fox, P.T., Radua, J., Mataix-Cols, D., Tench, C.R., Yarkoni, T., Nichols, T.E., Turkeltaub, P.E., Wager, T.D., Eickhoff, S.B., 2018. Ten simple rules for neuroimaging meta- analysis. Neurosci. Biobehav. Rev. 84, 151–161. https://doi.org/10.1016/j.neubiorev.2017.11.012.
Murray, A.J., Croce, K., Belton, T., Akay, T., Jessell, T.M., 2018. Balance control mediated by vestibular circuits directing limb extension or antagonist muscle Co-activation. Cell Rep. 22, 1325–1338. https://doi.org/10.1016/j.celrep.2018.01.009.
Nackaerts, E., D'Cruz, N., Dijkstra, B.W., Gilat, M., Kramer, T., Nieuwboer, A., 2019. Towards understanding neural network signatures of motor skill learning in Parkinson's disease and healthy aging. Br. J. Radiol., 20190071. https://doi.org/10.1259/bjr.20190071.
Nonnekes, J., Oude Nijhuis, L.B., de Niet, M., de Bot, S.T., Pasman, J.W., van de Warrenburg, B.P., Bloem, B.R., Weerdesteyn, V., Geurts, A.C., 2014. StartReact restores reaction time in HSP: evidence for subcortical release of a motor program. J. Neurosci. 34, 275–281. https://doi.org/10.1523/jneurosci.2948-13.2014.
Nonnekes, J., Carpenter, M.G., Inglis, J.T., Duysens, J., Weerdesteyn, V., 2015. What startles tell us about control of posture and gait. Neurosci. Biobehav. Rev. 53, 131–138. https://doi.org/10.1016/j.neubiorev.2015.04.002.
Ouchi, Y., Okada, H., Yoshikawa, E., Nobezawa, S., Futatsubashi, M., 1999. Brain activation during maintenance of standing postures in humans. Brain 122 (Pt 2), 329–338.
Ouchi, Y., Okada, H., Yoshikawa, E., Futatsubashi, M., Nobezawa, S., 2001. Absolute changes in regional cerebral blood flow in association with upright posture in humans: an orthostatic PET study. J. Nucl. Med. 42, 707–712.
Perera, T., Tan, J.L., Cole, M.H., Yohanandan, S.A.C., Silberstein, P., Cook, R., Peppard, R., Aziz, T., Coyne, T., Brown, P., Silburn, P.A., Thevathasan, W., 2018. Balance control systems in Parkinson's disease and the impact of pedunculopontine area stimulation. Brain 141, 3009–3022. https://doi.org/10.1093/brain/awy216.
Samartsidis, P., Montagna, S., Laird, A.R., Fox, P.T., Johnson, T.D., Nichols, T.E., 2019. Estimating the prevalence of missing experiments in a neuroimaging meta-analysis. bioRxiv. https://doi.org/10.1101/225425.
Schoberl, F., Feil, K., Xiong, G., Bartenstein, P., la Fougere, C., Jahn, K., Brandt, T., Strupp, M., Dieterich, M., Zwergal, A., 2017. Pathological ponto-cerebello-thalamocortical activations in primary orthostatic tremor during lying and stance. Brain 140, 83–97. https://doi.org/10.1093/brain/aww268.
Slobounov, S., Wu, T., Hallett, M., 2006a. Neural basis subserving the detection of postural instability: an fMRI study. Motor Control 10, 69–89. https://doi.org/10.1123/mcj.10.1.69.
Slobounov, S., Wu, T., Hallett, M., Shibasaki, H., Slobounov, E., Newell, K., 2006b. Neural underpinning of postural responses to visual field motion. Biol. Psychol. 72, 188–197. https://doi.org/10.1016/j.biopsycho.2005.10.005.
Snijders, A.H., Leunissen, I., Bakker, M., Overeem, S., Helmich, R.C., Bloem, B.R., Toni, I., 2011. Gait-related cerebral alterations in patients with Parkinson's disease with freezing of gait. Brain 134, 59–72. https://doi.org/10.1093/brain/awq324.
Solis-Escalante, T., van der Cruijsen, J., de Kam, D., van Kordelaar, J., Weerdesteyn, V., Schouten, A.C., 2019. Cortical dynamics during preparation and execution of reactive balance responses with distinct postural demands. Neuroimage 188, 557–571. https://doi.org/10.1016/j.neuroimage.2018.12.045.
Sunaert, S., Van Hecke, P., Marchal, G., Orban, G.A., 1999. Motion-responsive regions of the human brain. Exp. Brain Res. 127, 355–370. https://doi.org/10.1007/s002210050804.
Surgent, O.J., Dadalko, O.I., Pickett, K.A., Travers, B.G., 2019. Balance and the brain: a review of structural brain correlates of postural balance and balance training in humans. Gait Posture 71, 245–252. https://doi.org/10.1016/j.gaitpost.2019.05.011.
Takakusaki, K., 2017. Functional neuroanatomy for posture and gait control. J. Mov. Disord. 10, 1–17. https://doi.org/10.14802/jmd.16062.
Takakusaki, K., Habaguchi, T., Ohtinata-Sugimoto, J., Saitoh, K., Sakamoto, T., 2003. Basal ganglia efferents to the brainstem centers controlling postural muscle tone and locomotion: a new concept for understanding motor disorders in basal ganglia dysfunction. Neuroscience 119, 293–308. https://doi.org/10.1016/S0306-4522(03)00095-2.
Tanaka, H., Ishikawa, T., Kakei, S., 2019. Neural evidence of the cerebellum as a state predictor. Cerebellum 18, 349–371. https://doi.org/10.1007/s12311-018-0996-4.
Taube, W., Mouthon, M., Leukel, C., Hoogewoud, H.M., Annoni, J.M., Keller, M., 2015. Brain activity during observation and motor imagery of different balance tasks: an fMRI study. Cortex 64, 102–114. https://doi.org/10.1016/j.cortex.2014.09.022.
Therrien, A.S., Bastian, A.J., 2019. The cerebellum as a movement sensor. Neurosci. Lett. 688, 37–40. https://doi.org/10.1016/j.neulet.2018.06.055.
Turkeltaub, P.E., Eden, G.F., Jones, K.M., Zeffiro, T.A., 2002. Meta-analysis of the functional neuroanatomy of single-word reading: method and validation. Neuroimage 16, 765–780. https://doi.org/10.1006/nimg.2002.1131.
Turkeltaub, P.E., Eickhoff, S.B., Laird, A.R., Fox, M., Wiener, M., Fox, P., 2012. Minimizing within-experiment and within-group effects in Activation Likelihood Estimation meta-analyses. Hum. Brain Mapp. 33, 1–13. https://doi.org/10.1002/hbm.21186.
Van de Warrenburg, B.P., Bakker, M., Kremer, B.P., Bloem, B.R., Allum, J.H., 2005. Trunk sway in patients with spinocerebellar ataxia. Mov. Disord. 20, 1006–1013. https://doi.org/10.1002/mds.20486.
Van Impe, A., Coxon, J.P., Goble, D.J., Doumas, M., Swinnen, S.P., 2012. White matter fractional anisotropy predicts balance performance in older adults. Neurobiol. Aging 33, 1900–1912. https://doi.org/10.1016/j.neurobiolaging.2011.06.013.
Varghese, J.P., Beyer, K.B., Williams, L., Miyasike-daSilva, V., McIlroy, W.E., 2015. Standing still: is there a role for the cortex? Neurosci. Lett. 590, 18–23. https://doi.org/10.1016/j.neulet.2015.01.055.
Vogt, S., Di Rienzo, F., Collet, C., Collins, A., Guillot, A., 2013. Multiple roles of motor imagery during action observation. Front. Hum. Neurosci. 7, 807-807. https://doi.org/10.3389/fnhum.2013.00807.
Voogd, J., 2016. Deiters' Nucleus. Its Role in Cerebellar Ideogenesis : The Ferdinando Rossi Memorial Lecture. Cerebellum 15, 54–66. https://doi.org/10.1007/s12311-015-0681-9.
Voogd, J., Glickstein, M., 1998. The anatomy of the cerebellum. Trends Neurosci. 21, 370–375.
Wenderoth, N., Debaere, F., Sunaert, S., Swinnen, S.P., 2005. The role of anterior cingulate cortex and precuneus in the coordination of motor behaviour. Eur. J. Neurosci. 22, 235–246. https://doi.org/10.1111/j.1460-9568.2005.04176.x.
Wittenberg, E., Thompson, J., Nam, C.S., Franz, J.R., 2017. Neuroimaging of human balance control: a systematic review. Front. Hum. Neurosci. 11, 170. https://doi.org/10.3389/fnhum.2017.00170.
Wolpert, D.M., Miall, R.C., Kawato, M., 1998. Internal models in the cerebellum. Trends Cogn. Sci. (Regul. Ed.) 2, 338–347.
Zandvoort, C.S., van Dieën, J.H., Dominici, N., Daffertshofer, A., 2019. The human sensorimotor cortex fosters muscle synergies through cortico-synergy coherence. NeuroImage 199, 30–37. https://doi.org/10.1016/j.neuroimage.2019.05.041.
Zelenin, P.V., Beloozerova, I.N., Sirota, M.G., Orlovsky, G.N., Deliagina, T.G., 2010. Activity of red nucleus neurons in the cat during postural corrections. J. Neurosci. 30, 14533–14542. https://doi.org/10.1523/jneurosci.2991-10.2010.
Zhang, X.-Y., Wang, J.-J., Zhu, J.-N., 2016. Cerebellar fastigial nucleus: from anatomic construction to physiological functions. Cerebellum Ataxias 3, 9-9. https://doi.org/10.1186/s40673-016-0047-1.
Zwergal, A., la Fougere, C., Lorenzl, S., Rominger, A., Xiong, G., Deutschenbaur, L., Linn, J., Krafczyk, S., Dieterich, M., Brandt, T., Strupp, M., Bartenstein, P., Jahn, K., 2011. Postural imbalance and falls in PSP correlate with functional pathology of the thalamus. Neurology 77, 101–109. https://doi.org/10.1212/WNL.0b013e318223c79d.
Zwergal, A., Linn, J., Xiong, G., Brandt, T., Strupp, M., Jahn, K., 2012. Aging of human supraspinal locomotor and postural control in fMRI. Neurobiol. Aging 33, 1073–1084. https://doi.org/10.1016/j.neurobiolaging.2010.09.022.

Web References

http://www.alivelearn.net/xjview/, last visit on 16/05/2019.