Supplementary figures

Figure e-1: Overlap of lesion-network maps for 30 lesions causing asterixis. Maximal network overlap was 22/30 and included sites in the left thalamus, MNI coordinates x=-12, y=-6, z= 8 (VA nucleus) and x=-10, y=-18, z=2 (CM nucleus) and right thalamus, MNI coordinates: x=10, y=-18, z=2 (CM nucleus).

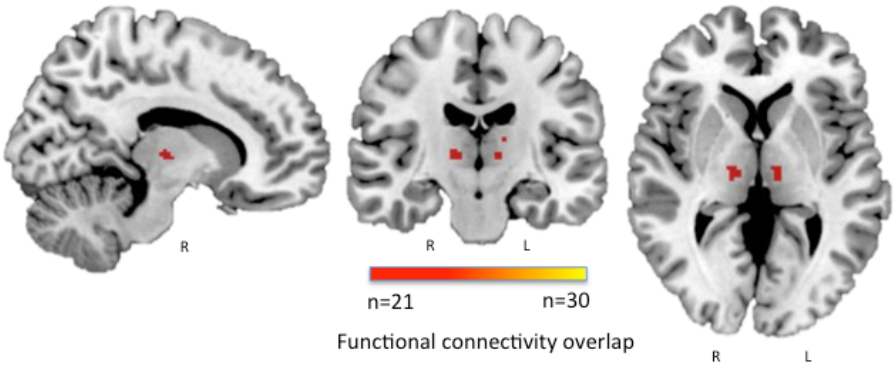
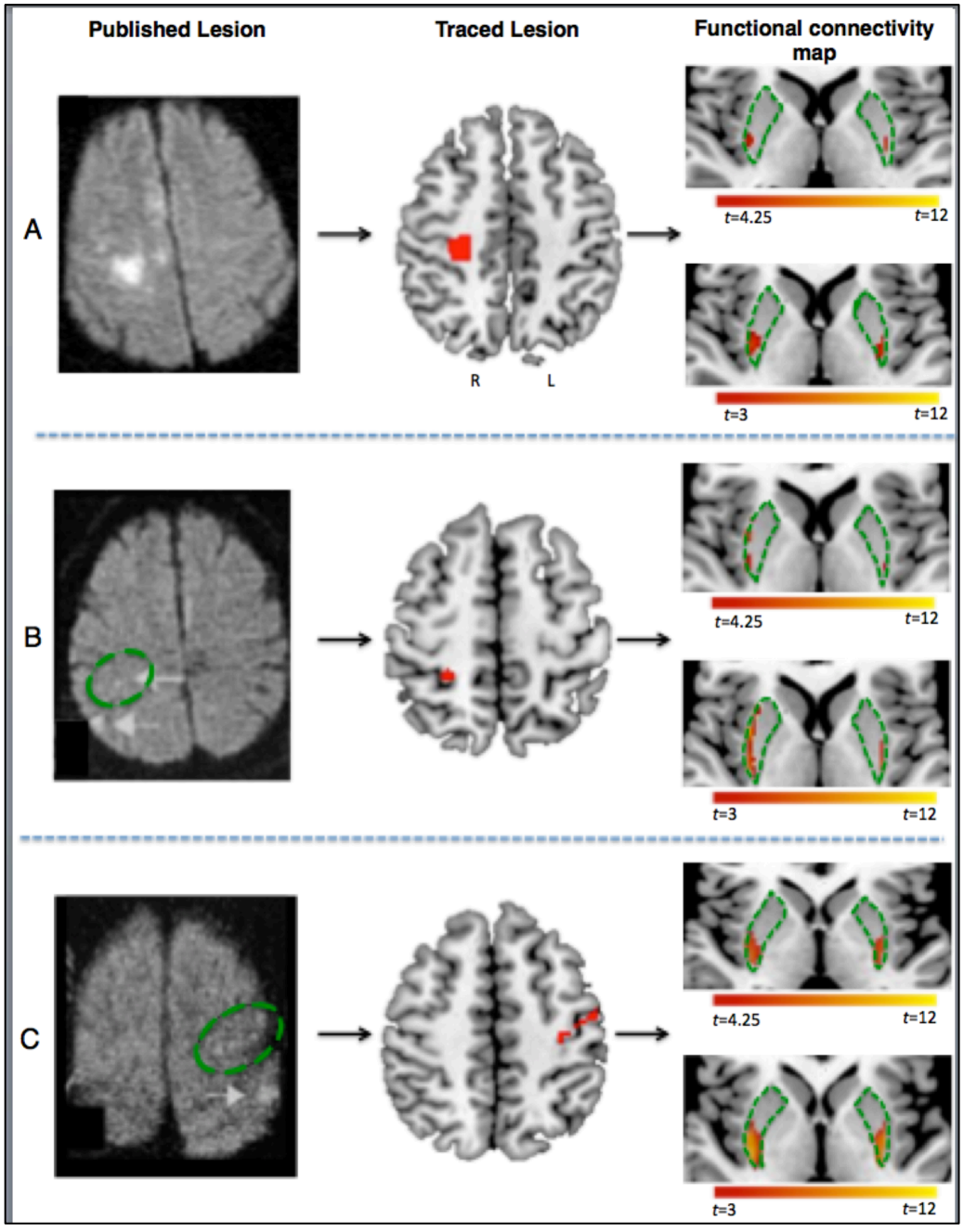


Figure e-2: Investigation into the three cases of hemichorea-hemiballismus without connectivity to our site of maximum overlap.

For each case, we show the original published image (left), a traced lesion (middle), and functional connectivity to the putamen at two thresholds, our original high threshold and a slightly lower threshold (right). Lesion #26 showed connectivity to the posterolateral putamen but to different voxels than the peak overlap of all lesions (A). Lesion # 13 was composed of two foci, the most anterior (circled in green) showed connectivity to the posterolateral putamen (B). Lesion #5, although reported as a solitary lesion, potentially had additional foci (circled in green) that were strongly connected to the posterolateral putamen (C). Images reproduced with permission from Springer Science+Business Media: Chung et al. (Hemichorea after stroke: clinical-radiological correlation. J Neurol 2004;251:725–729)


Published Lesion
Traced Lesion
Functional connectivity map
A
B
C
R
L
t=4.25
t=12
t=3
t=12
t=4.25
t=12
t=3
t=12
t=4.25
t=12
t=3
t=12

Supplementary Tables:

Table e-1: (A) Anatomical description of 29 cases of stroke-induced hemichorea-hemiballismus.

Lesion #	Stroke type	Primary Location	Secondary Location	Side of symptom	Reference
1	ischemic	Cortical (R insula)		Contralateral	e1
2	ischemic	GPI (Right)		Contralateral	e2
3	ischemic	Cortical (R opercular)		Contralateral	e1
4	hemorrhagic	Putamen (Right)	GPI (Right)	Contralateral	e3
5	ischemic	Cortical (L parietal)		Contralateral	e1
6	ischemic	STN (Right)	R thalamic-STN	Contralateral	e1
7	ischemic	Caudate (Left)		Contralateral	e1
8	ischemic	Putamen (R posterior)		Contralateral	e1
9	hemorrhagic	STN (Right)		Contralateral	e1
10	hemorrhagic	STN (Right)		Contralateral	e1
11	hemorrhagic	Caudate (Left)		Contralateral	e1
12	ischemic	STN (Right)		Contralateral	e1
13	ischemic	Cortical (R parieto-temporal)		Contralateral	e1
14	ischemic	Cortical (L tempora-insular)		Contralateral	e1
15	hemorrhagic	STN (Left)		Contralateral	e1
16	ischemic	Putamen (Right)		Contralateral	e4
17	ischemic	Subcortical (Right parietal)		Contralateral	e1
18	ischemic	Cortical (Right parietal)		Contralateral	e5

19	ischemic	STN (Left)	L thalamic- STN	Contralateral	[e1]
20	ischemic	Thalamic (Left)		Contralateral	[e6]
21	ischemic	Caudate (Right)		Contralateral	[e7]
22	ischemic	Caudate (Right)		Contralateral	[e8]
23	ischemic	Putamen (Left)		Contralateral	[e9]
24	ischemic	Cortical (Right)	R putamen	Contralateral	[e10]
25	ischemic	Putamen (Right)	Caudate (Right)	Contralateral	[e11]
26	ischemic	Cortical (Right parietal)		Contralateral	[e12]
27	ischemic	STN (Right)		Contralateral	[e13]
28	ischemic	STN/Midbrain (Right)	Midbrain	Contralateral	[e14]
29	ischemic	Cortical (R fronto-parietal)		Contralateral	[e1]

(B) Anatomical description of 10 replication cohort cases of stroke-induced hemichorea-hemiballismus.

Lesion #	Stroke type	Primary Location	Secondary Location	Side of symptom	Reference
1	ischemic	Thalamus (right)	GPI	Contralateral	*
2	ischemic	STN (left)	Thalamus	Contralateral	**
3	ischemic	STN, Thalamus (left)	Centrum semiovale (left)	Contralateral	Eternadifar et al. [e15]
4	ischemic	Caudate (right)	GPe (right), putamen (right)	Contralateral	Pantano et al. [e16]
5	ischemic	STN (left)		Ipsilateral	Crozier et al. [e17]
6	ischemic	Centrum semiovale (right)		Contralateral	Fukui et al. [e18]
7	ischemic	Precentral cortex (left)		Contralateral	Fukui et al. [e18]
8	ischemic	Centrum semiovale (right)		Contralateral	Fukui et al. [e18]
9	ischemic	Caudate head (right)		Contralateral	Kirk et al. [e19]

10	hemorrhagic	Corona radiate (right)		Contralateral	Barinagarreementaria et al. [e20]

* This patient was an 80 year old right handed gentleman with prior history of strokes/TIAs and cognitive impairment, nursing home resident who was sent to the ED for evaluation after developing new onset involuntary movements of his right hand. He was a poor historian and therefore the exact detailed information regarding onset was lacking on presentation. On exam by the attending neurologist, he was reported to have very prominent, athetoid-dystonic movements of his right hand that occurred involuntarily and seemed difficult to suppress. They interfered with his ability to use his hand to grasp and use objects. No hemiballismus or hemiasterixis noted and the strength was otherwise normal in the upper extremity except for some right proximal weakness in the range of 4/5. Despite treatment with olanzapine his movements persisted for several months.

** This patient was a 66yo right handed woman with a history of hypertension, hyperlipidemia, DM and graves disease, together with longstanding tobacco abuse, presented to the ED following two weeks of abnormal left sided arm and leg movements. She explained that she has been in her usual state of health, and these movements started somewhat abruptly two weeks prior. Since the onset, they did not really get worse or better. On exam, the observed movements appeared to be of a somewhat writhing variety in the left leg, together with some twisting movements. They started and stopped abruptly. The left leg was affected much more than the left arm. There was no perceptible weakness. Treatment with haloperidol led to significant improvement in the amplitude of all movements. The movements persisted for more than 12 months on follow up. Although bothersome, the small amplitude of the movement did not interfere with normal daily function.

E-References

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e-Methods:

Methodological and processing details for the normative resting state functional connectivity MRI (rs-fcMRI) dataset

Participants completed one or more rs-fcMRI scans during which they were asked to rest in the scanner with their eyes open. Rs-fcMRI data were processed in accordance with the strategy of Fox et al. [1] as implemented in Van Dijk et al. [2]. Functional data were preprocessed to decrease image artifacts and between-slice timing differences. Data were then spatially smoothed using a Gaussian kernel of 6 mm full-width at half-maximum and temporally filtered (0.009 Hz < f < 0.08 Hz). Next, several spurious or nonspecific sources of variance were removed by regression of the following variables: 1) six movement parameters computed by rigid body translation and rotation during preprocessing, 2) mean whole brain signal [3 4], 3) mean brain signal within the lateral ventricles, and 4) the mean signal within a deep white matter ROI. Inclusion of the first temporal derivatives of these regressors within the linear model accounted for the time-shifted versions of spurious variance.

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