Supplemental Material (Boes et al)

Supplemental materials and methods

Peduncular hallucinosis case series

Case 1. A 17-year-old right-handed, healthy and well-adjusted athletic girl with a history of migraine headaches without aura presented in clinic as a follow-up from an emergency room visit for hallucinations that went undiagnosed. She reported a two-hour period in which she experienced brief hallucinations lasting a couple of minutes each. It was something that was entirely foreign to her. She was driving with her friend in the passenger seat when the symptoms began. She provided a detailed written description of the events stating that her eyes were acting like a zoom lens of a camera zooming in and out. She next noted a cartoon girl on the side of the road that looked like a crayon drawing of someone. The entire scene appeared animated as if drawn in crayon. She then drove onto the sidewalk without realizing it and pulled into a parking lot. While still in the car she recounts a second hallucination in which she was refereeing a soccer game (which she had actually done earlier that day). She experienced the physical sensation of running and raising a flag even though she was sitting still in the car. She could hear the sounds of a soccer game. Although she experienced the physical sensation that she was refereeing the game, it was as if she was watching herself from behind about a foot away, such that she saw the back of her head. The next hallucination occurred when she reached to grab her jacket. Individual chalk-drawn cartoon flowers sprouted up out of the jacked as if it was a flowerbed. All the imagined flowers then fell to the ground, the stems appearing like popsicle sticks as they fell. These hallucinations all occurred within a span of about 2 hours and she reported intermittent drowsiness during this two-hour period that extended a couple of hours

beyond the hallucinations. The relationship between the drowsiness and hallucinations could not be recalled.

Her neurological exam and EEG were normal. MRI revealed an ischemic stroke of the medial thalamus in the region of the centromedian\parafascicular nuclei, extending from the superior border of the red nucleus to the mediodorsal nucleus (Supplemental Fig. 1, Case U). The presumed stroke etiology was cardioembolic given risk factors that were discovered in the course of her workup. She remains free of any further hallucinations, neurological deficits, or any other sequelae. *Comment.* This case adds to a small number of pediatric peduncular hallucinosis cases (Dunn et al., 1983; Kumar and Kaur, 2000; Kumar et al., 1999). To our knowledge this is the first report of an autoscopic hallucination in peduncular hallucinosis, in this case involving a visual hallucination of seeing oneself from a higher vantage point despite intact bodily sensations corresponding to hallucinated movements (Anzellotti et al., 2011). We feel this case is particularly informative given the small, circumscribed lesion and the clarity with which she was able to articulate her experience.

Case 2. A 77-year-old woman had a pontine stroke and subsequently experienced visual hallucinations. Her hallucinations initially involved a matrix of lines on her right upper hemifield followed by a yellow circle and bright red dot. This happened on two occasions, lasting two and nine minutes. Four years later she had a stent placed for vertebrobasilar stenosis that was complicated by a small subarachnoid hemorrhage. During rehabilitation she experienced seeing a gold pattern in the shape of a flower pot that would replicate itself on any solid background. In addition she reported seeing jungle and domestic animals crawling on the wall. Years later she

experienced an exacerbation of her visual hallucinations while taking amitriptyline for neuropathic pain. She would see visual distortions prior to sleep such as a nightstand by her bed bending, the alarm clock numbers projecting forward in space, and the rug on her bedroom floor with moving ripples as if an air hose were underneath it with the top appearing like wheat swaying in the wind. Whenever she experienced these visual distortions she had a clear band extending down the midline of her visual field. She was never alarmed by any of these hallucinations and realized they were illusory. Imaging showed hyperintense signal change in the pontine tegmentum (Supplemental Fig. 1, Case W). *Comment.* The use of amitriptyline in this patient appeared to exacerbate hallucinations that were latent for years. Amitriptyline and other tricyclic antidepressants have been identified as a provoking factor in complex visual hallucinations previously, offering a potential clue about the underlying neurobiology (Cancelli et al., 2004). Cancelli et al propose that tricyclic antidepressants provoke hallucination through combined anticholinergic action and serotonin augmentation. This imbalance favors hallucinatory states, though the mechanisms by which this neurotransmitter imbalance affects large-scale network activity is not clear.

Case 3. A 66-year-old man with obesity was hospitalized for a gastric bypass operation. Upon waking up from surgery he describes that he saw a scene as if he were in the fiberglass hull of a boat that was flooded. He recalls vividly seeing young female synchronized swimmers in red, white and blue uniforms. He could hear a buzzing sound in the background and could hear water splashing. He also saw clocks and pictures on the wall as if they were growing in size and warped, in addition to red patterns on the white walls as if they were dripping and melting. He saw two large snakes at the foot of his bed. When the compression stockings around his legs

inflated he thought there were snakes wrapping around his feet. Where the floor meets the wall he thought the floor was moving underneath the wall continuously, though illusory motion of his own body was not experienced. He saw clock hands moving backwards and mistook a box of gloves in the room for an aquarium filled with fish. One of the last hallucinations he had was the experience of seeing smoke rising from the nursing station. Although the smoke looked completely real he had enough insight at that point to identify that the smoke was likely a hallucination. Throughout this time of vivid hallucinations he was completely oriented and lucid, responding to all questions normally. His neurological exam was normal. An MRI was performed that showed a punctate area of diffusion restriction located at the left inferior colliculus (Supplemental Fig. 1, Case V). There had been a marked reduction in hallucinations by hospital day nine and he was discharged. He continued to occasionally see illusory shadows in his periphery despite normal visual fields. After this event his wife reported that he began to act out his dreams at night, consistent with REM behavior disorder. *Comment.* REM behavior disorder has been reported previously in the setting of peduncular hallucinosis (Vetrugno et al., 2009), and in this case it persisted long after the hallucinations had resolved. The lesion location in this case is unexpected in that the midbrain tectum has not been implicated in peduncular hallucinosis or REM behavior disorder previously. It is possible that a second puncate infarct of the brainstem or diencephalon went undetected on MRI.

Specificity analysis: Alternate statistical approaches

In addition to comparing network overlap from actual lesions and randomized lesions using a voxelwise Liebermeister test we also analyzed the results using a simple two-tailed t-test. In this analysis the voxel intensity resulted from the total number of subjects with overlapping networks at that site. For example if 18 of 23 peduncular hallucinosis patients had a positive correlation at a given voxel that exceeded the 4.25 t-value threshold then the voxel intensity is 18. The average voxel intensity across the cortical region of interest was then calculated and compared between networks derived from actual and randomized lesions. In addition, a t-test was also used to compare average voxel intensity within a priori region of interest to that of all other cortical regions, without reference to randomized lesion networks. The purpose of this was to test whether network overlap was higher in the a priori region of interest relative to other cortical areas.

Finally, in addition to the voxelwise Liebermeister test and the t-test, a subtraction analysis was performed. Randomized lesion networks were subtracted from actual lesion networks (Karnath *et al.*, 2004) and expressed as a percentage (e.g. overlap in at given voxel in 100% of actual lesions and 50% of randomized lesions would be represented as 50%).

Addressing possible confounds: An alternate method to global signal regression

Global signal regression remains the most common approach to preprocessing for fcMRI data because it improves neuroanatomical specificity (Fox *et al.*, 2009), correspondence to anatomical connectivity (Fox *et al.*, 2009), concordance with electrophysiology (Keller *et al.*, 2013), and its effect on resting state data has been extensively investigated with both positively and negatively correlated findings (Carbonell *et al.*, 2011; Chai *et al.*, 2012; Wong *et al.*, 2012).

To ensure the network results of peduncular hallucinosis were not dependent on global signal regression we pursued an alternative method, anatomical CompCor (Behzadi et al., 2007) implemented in the Conn toolbox (Whitfield-Gabrieli and Nieto-Castanon, 2012). This method applies principle component analysis (PCA) to the blood oxygen level dependent (BOLD) signal from individualized white matter and cerebrospinal fluid regions of interest. These principle components are then removed from the BOLD data through linear regression in place of the global signal, white matter, and cerebrospinal fluid regressors in the primary analysis. All other aspects of rs-fcMRI processing were identical to the primary analysis, including thresholding of the network maps at a t value of positive and negative 4.25 ($P<0.005$, uncorrected). A voxelwise Liebermeister test compared network results from the actual and randomized lesions, as performed in the main analysis.

Additional lesion syndromes

Cases of auditory hallucinosis were derived from a search of pubmed.org for 'brainstem auditory hallucinosis' and 'auditory hallucinations AND lesion' and citations from each selected article were cross-referenced. The search was performed in August of 2013 and limited to articles in English. Inclusion criteria included patients with auditory hallucinations presumed to have been caused by a focal intra-parenchymal lesion restricted to the brainstem or diencephalon as demonstrated by imaging or anatomic examination. Exclusion criteria included: 1) co-occurring cortical lesions, 2) extrinsic compression injuries without a clearly delineated intra-parenchymal lesion, 3) the presence of obvious competing etiologies for the hallucinations (e.g. a patient with comorbid psychosis or prior hallucinations from psychiatric disease, alcoholism, drug abuse or a pharmacologic or metabolic cause), or 4) poor image resolution such that lesion

boundaries could not be delineated. Notably, we did not exclude cases with co-occurring visual hallucinations and many of the cases were derived from patients with peduncular hallucinosis where auditory hallucinations were also explicitly mentioned. Each case is presented with additional details in Supplemental Table 2.

Cases of central post-stroke pain were derived from the literature through a systematic search of pubmed.org with search terms of 'central pain' or 'central post-stroke pain.' Inclusion criteria included patients who developed a pain syndrome in response to a focal intra-parenchymal lesion of the brainstem or diencephalon as demonstrated by imaging. Cases with cortical lesions or subcortical lesions outside of the brainstem and thalamus were excluded. Each case is presented in Supplemental Table 3 with additional details.

Cases of subcortical expressive aphasia were derived from a prior publication of the largest case series to date with accompanying figures showing individual lesions (N = 16) (Kim et al., 2012). Our target symptom was expressive aphasia. Patients with expressive aphasia and global aphasia were included, while receptive aphasia cases were excluded (cases 4, 6, 12, 13). The lesion location was traced onto a template brain, identical to the technique described for peduncular hallucinosis cases derived from the literature. Additional details for each case is presented in Kim et al, 2012.

A Priori Regions of Interest

The Harvard Oxford Atlas from FSL was used to derive a priori regions of interest for each syndrome. For auditory hallucinosis the posterior superior temporal gyrus was selected, as this region has been implicated in auditory hallucinations in prior publications (Allen et al., 2008; Griffiths, 2000; Kumar et al., 2014). For central post-stroke pain we chose the posterior insula.

Per Garcia-Larrea's recent review of the neuropathic pain matrices (Garcia-Larrea and Peyron, 2013) the posterior insula is the cortical site most heavily implicated in central post-stroke pain. To create this region of interest we modified the Harvard Oxford Atlas insular region to include only the insular cortex posterior to the central insular sulcus, per the standard anatomical delineation of the posterior insula (Afif and Mertens, 2010). Finally, for subcortical expressive aphasia the pars opercularis, the posterior inferior frontal gyrus, was selected as the region most closely approximating Broca's area (Petrides, 2006).

Specificity Analyses

For the three additional syndromes studied we assessed whether the localization of the lesions was critical to the results by randomizing the lesion location, as described for peduncular hallucinosis. The lesions were randomized on 20 iterations. After performing the initial statistical tests of network overlap for peduncular hallucinosis the results suggested that 20 randomization iterations would be sufficient, and this was performed for all subsequent analyses. For each condition the randomization of lesions occurred within a mask that contained all the lesions. A brainstem and thalamus mask was used for all conditions except subcortical aphasia, where a left hemisphere subcortical mask was used. The actual lesion networks were compared to the randomized lesion networks using the same statistical approach and parameters as in peduncular hallucinosis, namely a voxelwise Liebermeister test. The average network overlap was also compared between the a priori region of interest and all other cortical regions defined by the Harvard Oxford

Atlas. The same two alternate statistical approaches described for peduncular hallucinosis, a t-test and subtraction analysis, were also performed.

Between-Syndrome Lesion Network-Symptom Mapping

A between-syndrome analysis was performed to assess whether lesions or lesion networks are better able to segregate between syndromes. For example, all peduncular hallucinosis lesion masks were compared to all lesion masks associated with other lesion syndromes (auditory hallucinosis, central post-stroke pain, and subcortical aphasia). A voxelwise Liebermeister test was used and the resulting Z-maps were thresholded at a False Discovery Rate (FDR)-corrected $P<0.05$ (Rorden *et al.*, 2007). Next, the same analysis was repeated but the lesion networks were used in place of the lesion masks. Other parameters of the analysis were identical.

Supplemental Results

Insert Supplemental Fig. 1.

Insert Supplemental Fig. 2.

Insert Supplemental Fig 3.

Insert Supplemental Figure 4

Supplemental Table 1. Peduncular hallucinosis cases, listed in chronological order.

Citation	Lesion Location (age, handedness, sex)	Method of localization
1 (A). (Van Bogaert, 1927)	Midbrain, bilateral but only left side studied (59, F)	Autopsy
2 (B). (Geller and Bellur, 1987)	Midbrain tegmentum, R > L (61, M)	MRI
3 (C). (Hattori et al., 1988)	Pons, midline (73, M)	MRI
4 (D). (Feinberg and Rapcsak, 1989)	Right paramedian thalamus (83, RH, M)	MRI
5 (E). (McKee et al., 1990)	Bilateral substantia nigra pars reticulate (83, LH, M)	Autopsy
6 (F). (Kolmel, 1991)	Midbrain and paramedian thalamus, bilateral (56, M)	MRI
7 (G). (Serra Catafau et al., 1992)	Right posterolateral thalamus (68, RH, M)	MRI
8 (H). (Noda et al., 1993)	Right intralaminar thalamus (72, RH, M)	Autopsy
9 (I). (Noda et al., 1993)	Left thalamus (46, RH, F)	MRI
10 (J). (Howlett et al., 1994)	Junction of left midbrain \ inferior thalamus (63, M)	MRI
11 (K). (Yalcin et al., 1996)	Right thalamus, extending to midbrain tegmentum (48, RH, F)	CT
12 (L). (Yalcin et al., 1996)	Bilateral paramedian thalamus (51, RH, M)	MRI
13 (M). (Kamalakannan et al., 2004)	Midbrain, Near midline, L > R (84, M)	CT
14 (N).(Mocellin et al., 2006), Case A	Bilateral pons (85, F)	MRI
15 (O).(Mocellin et al., 2006), Case B	Right paramedian thalamus (68, F)	MRI
16 (P).(Benke, 2006), Case 2	Bilateral pons - mesencephalon junction (46, M)	MRI
17 (Q).(Benke, 2006), Case 3	Midline pons (41, M)	MRI
18 (R).(Benke, 2006), Case 4	Right paramedian thalamus and right midbrain tegmentum extending ventrally to cerebral peduncle (73, F)	MRI
19 (S). (Chrispal et al., 2009)	Right paramedian midbrain, mostly in tegmentum (72 F)	CT
20 (T). (Lee et al., 2011)	Left anterior paramedian thalamus (20, RH, M)	MRI
21 (U). Boes (current)	Left paramedian thalamus (17, RH, F)	MRI
22 (V). Boes (current)	Left inferior colliculus (66, M)	MRI
23 (W). Boes (current)	Bilateral pons (77, F)	MRI

Abbreviations: F Female, L Left, LH Left-handed, M Male, R Right, RH Right-handed

Supplemental Table 2. Auditory hallucinosis cases

Citation	Lesion Location (age, handedness, sex)
1. (Cascino and Adams, 1986)	L pontine tegmentum (32, RH F)
2. (Cascino and Adams, 1986)	R pontine tegmentum (42, F)
3. (Cascino and Adams, 1986)	Lower midbrain, L inferior colliculus (53, RH F)
4. (Lo et al., 2011)	L midbrain (22, M)
5. (Murata et al., 1994)	R pons (55, RH M)
6. (McKee et al., 1990)[a]	Bilateral substantia nigra pars reticulata (83, LH, M)
7. Kolmel (Kolmel, 1991) [a]	Midbrain and paramedian thalamus, bilateral (56, M)
8. (Noda et al., 1993) [a]	Right intralaminar thalamus (72, RH, M)
9. (Noda et al., 1993) [a]	Left thalamus (46, RH, F)
10. (Mocellin et al., 2006) [a]	Right paramedian thalamus (68, F)
11. (Benke, 2006) [a]	Bilateral pons - mesencephalon junction (46, M)
12. (Benke, 2006) [a]	Midline pons (41, M)
13. (Benke, 2006) [a]	Right paramedian thalamus and right midbrain tegmentum extending ventrally to cerebral peduncle (73, F)
14. Case 1, Current report [a]	Left paramedian thalamus (17, RH, F)
15. Case 2, Current report [a]	Left inferior colliculus (66, M)

[a] Cases with visual and auditory hallucinations, also included in peduncular hallucinosis analysis

Abbreviations: F Female, L Left, LH Left-handed, M Male, R Right, RH Right-handed

Supplemental Table 3. Central post-stroke pain cases

Citation	Lesion Location
1. (Lorenz et al., 1998)	L medulla
2. (Kamano, 2003)	R medulla
3. (Bowsher, 2005)	L Posteroventral thalamus
4. (Bowsher, 2005)	R Pontine infarct
5. (Bowsher, 2005)	Pons
6. (Bowsher, 2005)	Medulla
7. (Bowsher, 2005)	Medulla
8. (Bowsher, 2005)	L posteroventral thalamus
9. (Bowsher, 2005)	R pontine lesion
10. (Montes et al., 2005)	R thalamus
11. (Yoshita and Yamada, 2006)	L thalamus
12. (Yoshita and Yamada, 2006)	R thalamus
13. (Kim et al., 2007)	L lateral thalamus
14. (Kim et al., 2007)	L lateral thalamus
15. (Kim et al., 2007)	L lateral thalamus
16. (Kim et al., 2007)	R lateral thalamus
17. (McGeoch et al., 2008)	Rt thalamic infarct
18. (McGeoch et al., 2008)	Rt posterolateral thalamus
19. (Kumar et al., 2009)	L lateral thalamus
20. (Alstadhaug and Prytz, 2012)	Bilateral ventral posterior thalamus
21. (Sprenger et al., 2012)	R thalamus
22. (Brigo et al., 2013)	R thalamus
23. (Sposato et al., 2014)	L thalamus

Abbreviations. L left, R right

Supplemental Table 4. Coordinates of peduncular hallucinosis lesion overlap results

Overlap	Coordinates (xyz)	Region
N=6	6, -16, 0	R thalamus
N=6	-4, -20, 0	L thalamus
N=3	14, -14, 0	R lateral thalamus
N=3	0, -28, -12	Median mesencephalon
N=3	2, -24, -32	Mid-pons
N=3	-6, -12, -12	L substantia nigra pars reticulata
N=3	10, -12, -12	R substantia nigra pars reticulata
N=3	2, -24, -22	Ponto-mesencephalic junction

Abbreviations: L Left, R Right

Supplemental Table 5. Coordinates of peduncular hallucinosis lesion network mapping results, comparing actual and randomized lesion networks

Intensity (Z-max)	Coordinates (xyz)	Region
Anticorrelated functional connectivity results		
9.56	-48 -64 -14	L inferior lateral occipital cortex
8.67	50 -66 -16	R inferior lateral occipital cortex
8.48	-24 -40 68	L post-central gyrus
Positive functional connectivity results		
10.1	-12 32 14	L anterior cingulate cortex
7.1	-44 -48 -34	L cerebellar cortex
7.51	14 -62 40	R posteromedial cortex
7.84	-18 -26 -6	L lateral geniculate nucleus
8.48	-38 18 -8	L insula
8.95	8 -34 -6	R superior colliculus

Abbreviations. L left, R right

Supplemental Table 6. Coordinates of lesion overlap results: auditory hallucinosis, central post-stroke pain, subcortical expressive aphasia.

Overlap	Coordinates (xyz)	Region
Auditory Hallucinosis		
N=3	5, -14, 0	R thalamus
N=3	3, -28, -14	R dorsal inferior midbrain
N=3	9, -24, -14	R lateral midbrain
N=3	9, -23, -20	R lateral midbrain-pons junction
N=3	1, -25, -21	Mid pons-mesenecphalon junction
Central Post-Stroke Pain		
N=6	14, -21, 4	R ventrolateral thalamus
Subcortical Expressive Aphasia		
N=4	-27, -11, -4	L putamen – globus pallidus junction

Abbreviations. L left, R right

Supplemental Table 7. Coordinates of lesion network mapping results: auditory hallucinosis, central post-stroke pain, subcortical expressive aphasia.

Intensity (Z max)	Coordinates (xyz)	Region
Auditory Hallucinosis – Anticorrelation		
4.39	-52 -44 12	L superior temporal gyrus
4.4	64 -20 2	R superior temporal gyrus
4.6	20 -46 60	R parietal
4.66	-20 -42 58	L parietal
5.27	-50 -78 -12	L inferior occipital cortex
5.21	56 -68 -12	R inferior occipital cortex
4.85	-56 -68 6	L occipito-temporal cortex
4.57	-46 16 -36	L temporal pole
Auditory Hallucinosis – Positive Correlations		
5.07	-30 16 -18	L fronto-insular cortex
4.95	26 12 -20	R fronto-insular cortex
4.9	14 34 22	R anterior cingulate cortex
Central Post-Stroke Pain – Anticorrelation		
7.19	56 -20 -18	R middle temporal gyrus

Intensity (Z max)	Coordinates (xyz)	Region
7.43	48 -60 32	R parietal cortex
7.63	-4 44 -12	L ventromedial prefrontal cortex
7.80	0 -44 30	Retrosplenial cingulate cortex
Central Post-Stroke Pain – Positive Correlation		
7.52	-40 -6 -6	L insula
7.42	34 -18 18	R insula
6.97	62 -28 32	R parietal
7.48	-14 -38 50	L posteromedial cortex
6.69	8 6 42	R anterior cingulate cortex
Subcortical Aphasia – Anticorrelations		
5.74	-18 -46 30	L precuneus \ white matter
4.99	40 -34 -12	R medial temporal fusiform gyrus
4.64	26 18 38	R middle frontal gyrus
Subcortical Aphasia – Positive Correlations		
6.45	-58 4 2	L inferior frontal gyrus
8.13	60 12 2	R inferior frontal gyrus
8.26	60 -34 40	R parietal
4.72	28 -68 -24	R lateral cerebellum

Intensity (Z max)	Coordinates (xyz)	Region
7.4	-54 8 -8	L anterior temporal lobe
7.85	6 10 48	R medial prefrontal cortex

Abbreviations. L left, R right

Supplemental Tables 8 – 10 are presented separately, in landscape view

Supplemental Figure Legends

Supplemental Figure 1. 23 Peduncular hallucinosis lesions

The lesions of 23 cases of peduncular hallucinosis are displayed. The letter corresponding to each lesion is cross-referenced with Supplemental Table 1, column 1.

Supplemental Figure 2. Traditional lesion mapping results of peduncular hallucinosis, expanded from Figure 2.

Results of the peduncular hallucinosis lesion mapping are displayed. The degree of overlap is color-coded, as indicated on the color bar, with maximum overlap of 6 cases. The MNI coordinate of each axial slice is shown above the image. Coordinates are reported in Supplemental Table 4.

Supplemental Figure 3. Lesion network mapping results of peduncular hallucinosis.

Lesion network mapping results are displayed. Areas of positive correlation are shown with warmer colors (red, yellow) and anticorrelation is shown in blue. The results are thresholded to

display areas of common overlap in at least 18 of 23 cases. The MNI coordinate of each axial slice is displayed above the image.

Supplemental Figure 4. Addressing possible confounds.

A shows the a priori cortical region of interest for peduncular hallucinosis. B shows the results using an alternate technique that avoids global signal regression (aCompCor with 3 regressors). C shows the network results derived from a separate cohort of older adults with a mean age of 70. Column B and C represent Z-score maps comparing actual network output to randomized networks. All voxels displayed are significant at a false discovery rate of 5%.

Supplemental Figure 5. 2D Versus 3D lesions.

Networks associated with peduncular hallucinosis lesions from local case 1 and 2 are shown. Networks derived from a single 2 dimensional axial slice representing the lesion (A & C) are compared to the full 3 dimensional lesions (B & D). Axial slices are shown, from left to right for A-D are -8, 18, 40.

Supplemental Figure 6. Lesion network mapping using subtraction analysis.

Network overlap was also compared between actual and randomized lesions using subtraction analysis instead of the voxelwise Liebermeister approach used in Fig. 5 of the main article. The images on the far left show the a priori cortical region of interest for each condition with subtraction results in the middle column. The average voxel intensity within the a priori cortical region of interest is shown in the white bars and the gray bars show average voxel intensity in

cortical areas outside the a priori region of interest, compared using a t-test. * $P < 0.05$, ** $P < 0.01$

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