Brain imaging in ME has demonstrated:

* Chronic neuroinflammation

* Autonomic and vascular dysfunction

* Reduced cerebral blood flow and metabolism

* Structural and functional injury to brainstem and cortical areas

Brain imaging studies have consistently shown measurable abnormalities in Myalgic Encephalomyelitis (ME), distinguishing it from psychiatric or purely functional conditions. Here's a clear overview of what has been found through different neuroimaging methods (MRI, fMRI, PET, SPECT, etc.) and what these findings reveal about encephalomyelitis (inflammation of the brain and spinal cord).

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Structural MRI (Anatomical Imaging)

Findings:

* Reduced grey matter volume, especially in:

* The brainstem

* Anterior cingulate cortex

* Prefrontal cortex

* Temporal lobes

Significance:

* Suggests neurodegeneration or chronic inflammation leading to tissue loss.

* Brainstem changes are especially important because the brainstem controls autonomic, cardiovascular, and respiratory functions — all commonly disturbed in ME.

Key Studies:

* Barnden et al., 2011–2019 (Australia): Repeated MRI studies showed structural and signal abnormalities in the brainstem, consistent with neuroinflammation and autonomic dysfunction.

* de Lange et al., 2005: Found grey matter volume reduction that correlated with the severity of fatigue and cognitive dysfunction.

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Functional MRI (fMRI)

Findings:

* Abnormal activation patterns during cognitive tasks (such as working memory or attention tests).

* Reduced connectivity between regions involved in energy regulation, motor control, and cognition.

* Compensatory overactivation in some regions — meaning patients' brains work harder for the same task.

Significance:

* Confirms impaired neural efficiency and possible energy metabolism issues in the brain.

* Supports the concept of post-exertional neuroimmune exhaustion (PENE) in the ICC 2011 definition.

Key Studies:

* Cook et al., 2017: After exertion, fMRI showed widespread reduction in connectivity, corresponding to the worsening of symptoms (post-exertional malaise).

* Boissoneault et al., 2016: fMRI revealed disrupted default mode network (DMN) activity — consistent with impaired information processing.

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PET Scans (Neuroinflammation)

Findings:

* Microglial activation and neuroinflammation detected in:

* Cingulate cortex

* Hippocampus

* Amygdala

* Thalamus

* Midbrain and pons (brainstem)

Significance:

* PET imaging provides direct evidence of neuroinflammation — the "encephalomyelitis" component of ME.

* The intensity of inflammation correlates with symptom severity.

Key Studies:

* Nakatomi et al., 2014 (Japan): Landmark study using [^{11}C]PK11195 PET tracer found widespread microglial activation in ME/CFS patients.

* Yamato et al., 2021: Confirmed earlier findings, especially in patients with severe neurological symptoms.

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SPECT Scans (Cerebral Perfusion)

Findings:

* Hypoperfusion (reduced blood flow) in:

* Frontal lobes

* Temporal lobes

* Brainstem

* Parietal regions

Significance:

* Indicates vascular dysregulation and metabolic underactivity in the brain.

* Correlates with orthostatic intolerance, cognitive impairment, and fatigability.

Key Studies:

* Costa et al., 1995; Ichise et al., 1992: Early SPECT studies showed reproducible patterns of cerebral hypoperfusion in ME/CFS patients compared with healthy controls.

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Diffusion Tensor Imaging (DTI)

Findings:

* Altered white matter integrity, especially in the midbrain and limbic system tracts.

* Reduced axonal density and myelin integrity.

Significance:

* Suggests neuroinflammatory damage or immune-mediated injury to neural pathways — aligning with the “encephalomyelitis” aspect.

Key Studies:

* Zeineh et al., 2015 (Stanford): Detected distinct brain anomalies in ME/CFS patients, particularly involving right arcuate fasciculus and white matter integrity changes.

References (see comments for links)

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ME-ICC Criteria: https://onlinelibrary.wiley.com/.../j.1365-2796.2011.02428.x

#GAMEICC

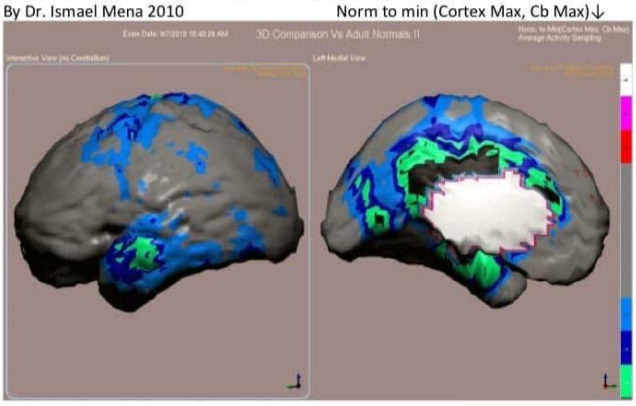
#NightingaleContinuum

Neurological Abnormalities

Neurocognitive, sleep, autonomic and sensory disturbances, pain, headaches, and paresthesias are prominent neurological signs and symptoms. Cognitive impairments including slow processing of information, poor attention, word finding, and working memory are some of the most functionally disabling symptoms.[1, 73, 74]

Structural and functional abnormalities within the brain and spinal cord are consistent with pathological dysfunction of the regulatory centers and communication networks of the brain, CNS and ANS, and are essential for effective ongoing self-organization.[1, 75] Reduced brainstem gray matter volume is consistent with insult to the midbrain at fatigue onset. Feedback control loops may suppress cerebral motor and cognitive activity, disrupt CNS homeostasis, and reset elements of the ANS.[76] These abnormalities play crucial roles in neurological and neurocognitive symptoms.[1, 5, 11, 57, 65] Greater source activity and more parts of the brain are utilized in cognitive processing, which supports patients' perception of greater effort.[73, 77, 78] Reduced duration of uninterrupted sleep may explain reported unrefreshed sleep, pain and overwhelming fatigue.[79] These observed pathological changes are consistent with neurological disorders but not psychiatric conditions.

3D Comparison VS Adult Norms II – Avg. activity sampling
By Dr. Ismael Mena 2010 Norm to min (Cortex Max, Cb Max)↓



↑Interactive view – no cerebellum ↑Left medial view

Extensive areas of hypoperfusion are characteristic of ME: HMPAO c99m radiopharmaceutical for brain blood flow assessment. Images of the patient are reconstructed and compared against normal age matched data-base by means of Oasis Segami USA Software. In color gray normal perfusion equal to mean ± 2 St Dev, colors blue, green and black, 2-5 St dev. below the normal mean denoting hypoperfusion. Left lateral view shows marked hypoperfusion in the lateral aspects of the temporal lobe, extending to the frontal and parietal lobes. Left medial view shows extensive hypoperfusion in the limbic system involving anterior, medial and posterior cingulates. There is left temporal medial hypoperfusion that denotes hypofunction in the projection of the hippocampus. Both posterior cingulate and hippocampal hypofunction denote cognitive impairment. (Ventricular system is in color white.) Finally, there is hypoperfusion in the occipital lobe. *Ismael Mena, MD, nuclear medicine*

Neurological Structural & Functional Abnormalities

Hypoperfusion[80 - 84] (Neuro-SPECT, arterial spinning labeling)
↓ **regional blood flow** (rCBF), ↓ **absolute cortical blood flow**[46, 85]
↓ **hypoperfusion in brainstem** distinguishes ME from depression[83]
↓ **further reduction in cerebral blood flow after exercise**
Greater involvement of the brain correlates with greater severity[46]