Hypoperfusion vs Brain Spine Inflammation (Encephalomyelitis)

Hypoperfusion means reduced blood flow.

In the brain or spinal cord, this means tissues are not getting enough oxygen and nutrients at a given time. It's a circulation problem, not a primary tissue injury.

*Can be intermittent or positional (for example, worse upright)

*Often linked to autonomic dysfunction (e.g., blood pooling, low blood pressure regulation issues)

*Effects are usually reversible when blood flow improves

*Symptoms can include cognitive slowing, dizziness, fatigue, visual "fuzziness," and difficulty concentrating

Brain/spinal inflammation means immune activation and inflammatory signaling within nervous tissue. This is a biological activation/injury response, where immune cells and cytokines are active in or around the CNS.

*Can be persistent or fluctuating

*Involves immune mediators (cytokines, microglial activation, etc.)

*May lead to more sustained changes in neural function

*Symptoms can include cognitive impairment, sensory sensitivity, pain amplification, sleep disruption, and "sickness behavior" type fatigue

Key difference in simple terms:

*Hypoperfusion = not enough blood flow reaching the tissue

*Inflammation = the tissue is biologically "activated" or irritated by immune processes

How they can overlap

They are not mutually exclusive. In conditions like ME/CFS, hypotheses often include:

*Reduced cerebral blood flow (hypoperfusion) during orthostatic stress or exertion

*Neuroinflammatory or immune signaling changes affecting how the brain regulates blood flow and metabolism

So you can think of it as:

*Hypoperfusion = delivery problem

*Inflammation = control/immune signaling problem

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Hypoperfusion (reduced blood flow)

What scans/tests may show

Hypoperfusion is about circulation, so it's usually assessed with blood-flow or perfusion imaging:

*SPECT scans may show reduced regional cerebral blood flow

*PET scans can show reduced metabolic activity that often tracks with blood flow

*fMRI (task-based or resting) may show reduced activation in certain networks

*Transcranial Doppler (TCD) can show altered cerebral blood flow velocity

*Near-infrared spectroscopy (NIRS) sometimes shows reduced oxygenation, especially upright or post-exertion

Typical pattern clues

*Worsens with standing, exertion, heat, or dehydration

*Often improves with lying down

*Can fluctuate quickly (minutes to hours)

*May be strongly linked to autonomic dysfunction (e.g., orthostatic intolerance)

Common symptom pattern

*"Brain fog" that feels like slowed processing

*Lightheadedness or near-faintness

*Visual dimming, tunnel vision, or "not enough oxygen" feeling

*Difficulty thinking upright or after activity

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Brain / Spinal inflammation (neuroinflammatory activity). What scans/tests may show. Neuroinflammation is harder to detect directly in routine clinical practice, but research tools include:

*PET with TSPO ligands used to detect microglial activation (a marker of neuroinflammation)

*MRI (conventional) is often normal, but may show non-specific findings in some cases

*CSF analysis (spinal fluid) sometimes shows immune activation markers (not routine)

*Blood markers cytokines or immune dysregulation signals (indirect, not definitive)

*Advanced research imaging can show altered glial activity or neuroimmune signaling

Typical pattern clues

*More persistent baseline symptoms rather than position-dependent changes

*Often worsened by exertion but does not immediately improve lying down

*Slower recovery after triggers (hours to days, e.g., post-exertional worsening)

*Can include sensory hypersensitivity (light, sound, touch)

Common symptom pattern

*Cognitive dysfunction that feels more “inflamed” or overloaded rather than just slowed

Fatigue that feels systemic, like “flu-like exhaustion”

*Increased pain sensitivity or neurological sensitivity

*Sleep disturbance and unrefreshing sleep

Important nuance

In real conditions like MEICC or dysautonomia research, these often interact rather than exist separately:

*Poor autonomic regulation can reduce cerebral blood flow

*Immune signaling may affect vascular regulation and brain metabolism

*Reduced perfusion can secondarily stress immune/glial cells

So clinically, patients can show a mixed picture rather than a clean “either/or.”

How scans can reflect hypoperfusion?

These scans are looking at blood flow or metabolism, which often mirrors blood flow:

*SPECT (single-photon emission CT)

Can show regional hypoperfusion (reduced blood flow in specific brain areas). This has been reported in some ME/CFS research cohorts, often variably and not consistently across all patients.

*PET (FDG-PET)

Measures glucose metabolism. Lower activity can indirectly reflect reduced perfusion or reduced neuronal activity. ASL MRI (arterial spin labeling). A non-invasive MRI method that can directly estimate cerebral blood flow, sometimes showing reduced flow in certain regions or during orthostatic challenge studies.

These findings tend to align with functional “underactivity” rather than structural damage.

How scans can reflect neuroinflammation?

These are trying to detect immune/glial activation, which is more indirect and harder to capture:

*TSPO PET scans

The main research tool for neuroinflammation. It can show increased signal thought to reflect microglial activation (an immune response in the brain).

*MRI (indirect signs)

Routine MRI is often normal, but research sometimes looks at:

*subtle white matter changes

*altered connectivity patterns (functional MRI)

These are not specific for inflammation but may correlate with it in research settings.

*CSF (spinal fluid) studies (not imaging but related)

Can show immune activation markers supporting inflammation hypotheses.

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Why both can appear in the same patient group

This is where things overlap in conditions like ME/CFS and dysautonomia research:

Inflammation/immune signaling may affect:

*blood vessel regulation

*neurovascular coupling (how brain activity controls blood flow)

Hypoperfusion may lead to:

*secondary metabolic stress

*activation of glial cells as a response to low energy availability

So on imaging, you might see:

*reduced perfusion (blood flow issue)

*alongside signals suggesting immune activation (neuroinflammatory marker studies)

...but not always in the same scan type or at the same time.

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Key limitation

A critical point is that:

*Hypoperfusion = measured more directly and reproducibly

*Neuroinflammation = still largely inferred in humans via indirect markers

So when both are discussed in research, they're often complementary hypotheses rather than two clearly separable, routinely diagnosable scan findings.

References

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