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Beyond Descriptive Atrophy: A Quantitative Neuroimaging Conceptual Framework for Alcohol Use Disorder

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Abstract

Background: Alcohol Use Disorder (AUD) is associated with highly region specific selective structural brain injury, yet radiology reports frequently rely on qualitative descriptors such as "generalized atrophy." This "semantic gap" overlooks measurable neurobiological changes and the potential for tracking neuroplastic recovery.

Objective: This paper proposes a quantitative neuroimaging conceptual framework aimed at transitioning AUD assessment from descriptive interpretation to precision-based metrics.

Methods: We follow through the evolution of morphometric assessment, ranging from classical two-dimensional linear indices-such as the Evans Index (EI), Bicaudate Distance Index (BCDI), and Occipital-Frontal Horn Ratio (OFHR)-to modern three-dimensional volumetry, including the Ventricle-to-Brain Ratio (VBR) and Z-score normalization.

Results: The discussion in this article emphasizes on integration of advanced modalities such as Voxel-Based Morphometry (VBM) for long term recovery tracking and applications of radiomics for detecting microstructural alterations. We further discuss "Brain Age" prediction models, including the cutting-edge research on Graph Neural Networks (SAGE Fusion Net), as biomarkers for accelerated ageing.

Conclusion: By integrating quantitative MRI techniques into routine clinical workflows, radiology can provide objective, reproducible, and actionable data. This transition enables earlier detection of injury, personalized monitoring of abstinence-driven recovery, and improved prognostic accuracy in the management of AUD.

Keywords: Alcohol Use Disorder (Aud); Quantitative Neuroimaging; Brain Volumetry; Evans Index; Ventricle-To-Brain Ratio (Vbr); Radiomics; Brain Age Gap; Neuroplasticity; Voxel-Based Morphometry (Vbm)

Abbreviation: AUD: Alcohol Use Disorder; BCDI: Bicaudate Distance Index; CSF: Cerebrospinal Fluid; EI: Evans Index; GABA: Gamma-Aminobutyric Aci; MPRAGE: Magnetization-Prepared Rapid Gradient-Echo; MRI: Magnetic Resonance Imagin; OFHR: Occipital-Frontal Horn Ratio; SBM: Surface-Based Morphometry; VBM: Voxel-Based Morphometry; VBR: Ventricle-to-Brain Ratio

Introduction: Alcohol and Brain Injury

Alcohol Use Disorder (AUD) stands as a one of the primary causes of neurological decline worldwide, marked by brain injury that is as progressive as it is regionally specific. Yet, a frustrating disconnect persists in modern medicine. Despite a mountain of evidence showing that this damage is both measurable and-crucially-reversible, standard radiology reports still lean on vague "eyeball" assessments like "generalized atrophy" or "ventricular enlargement." This isn't just a matter of terminology; it's a profound "semantic gap" that hides the true neurobiological chaos of chronic ethanol exposure and misses the opportunity to track a patient's actual recovery. Ethanol disrupts glutamate and GABA neurotransmission, induces oxidative stress, and activates microglial cells, leading to neuroinflammation, dendritic retraction, synaptic loss, and ultimately neuronal death in vulnerable regions Crews & Nixon [1] & Zahr et al. [2]. These neuroinflammatory mechanisms disproportionately affect the prefrontal cortex,

hippocampus, cerebellum, and limbic systems -regions essential for executive control, memory, motor coordination, and emotion regulation Goldstein & Volkow [3] & Zahr & Sullivan [4]. Evidence from postmortem specimens and animal studies establishes a biological basis for volumetric loss in AUD. Rodent models of chronic alcohol exposure show dendritic spine reduction in frontal cortex and hippocampus, suppressed neurogenesis in the dentate gyrus, and white matter microstructural alteration-demonstrating that alcohol's neurotoxic effects are both region-specific (affecting grey matter) and equally affecting white matter tracts He & Crews [5] & Vetreno & Crews [6]. In humans, disrupted integrity of white matter tracts and reduced hippocampal subfields on diffusion imaging support a model in which microstructural injury precedes gross volume loss Pfefferbaum et al. [7].

Historical Evolution of Volumetric Assessment

The quest to quantify alcohol-related structural change long predates the advent of modern morphometric techniques. Early MRI studies in the 1990s used semi-automated segmentation to compare grey and white matter volumes between alcohol-dependent individuals and controls subjects, demonstrating that individuals with chronic alcohol use disorder exhibited reduced parenchymal volumes and increased cerebrospinal fluid (CSF) spaces beyond the effects of normal ageing Pfefferbaum et al. [8] & Hommer et al. [9]. Longitudinal studies revealed that sustained abstinence is associated with partial structural recovery, while relapse correlates with continued decline-establishing volumetry as a measure of both injury and neuroplastic resilience Pfefferbaum et al. [10] & Shear et al. [11]. The development of voxel-based morphometry (VBM) and surface-based morphometry (SBM) allowed unbiased, whole-brain assessment. Voxel-based morphometry (VBM) studies demonstrate reduction in volume of fronto-limbic, insular, and cerebellar regions compared with controls Cardenas et al. [12]; Roberts & Schweinsburg [13]. Surface-based morphometry (SBM) enables precise cortical thickness measurement, revealing regional specific thinning in the dorsolateral and orbitofrontal cortices-the regions essential for cognitive control and decision-making Makris et al. [14]. Longitudinal studies document time-dependent recovery trajectories in grey matter after sustained abstinence, demonstrating plasticity even in individuals with chronic AUD Bühler et al. [15].

From Classical Indices to Volumetric Ratios

Quantitative assessment of brain atrophy in AUD traditionally relied on linear indices, which, despite their two-dimensional nature, retain considerable clinical utility today. These classical indices laid the groundwork for objective brain measurement.

a) The Evans Index (EI) is calculated as the maximum width of the frontal horns of the lateral ventricles divided by the internal diameter of the skull at the same axial level. This index offers a rapid estimate of ventricular enlargement, which may reflect global parenchymal loss. In the context of AUD, enlargement of the frontal horns often corresponds to thinning of the prefrontal cortex and the executive deficits commonly observed in affected individuals. While the EI is simple and reproducible, its principal limitation lies in capturing only frontal ventricular width, exclusion of posterior ventricular changes or overall volumetric relationships of the brain.

b) The Bicaudate Distance Index (BCDI) provides a specific assessment of striatal atrophy by calculating the ratio between the inter-caudate distance and the total brain width at the same axial level. In the context of AUD, an increased BCDI serves as a proxy for the shrinkage of the caudate nuclei and surrounding white matter-areas vital for reward processing, habit formation, and motor coordination. Despite its utility in identifying regional neurotoxicity, the BCDI is inherently limited as a two-dimensional metric, failing to account for volumetric changes along the anterior-posterior or superior-inferior axis.

c) The Occipital-Frontal Horn Ratio (OFHR) offers a more comprehensive look at lateral ventricular expansion than the Evans Index alone. By calculating the sum of the frontal and occipital horn widths against the internal skull diameter at the same axial level, this ratio indicates posterior horn enlargement-a key radiological marker that often mirrors the hippocampal and limbic atrophy seen in patients with memory deficits. However, despite providing a broader anatomical perspective, the OFHR remains a two-dimensional proxy; it cannot quantify the actual volume of the ventricles or account for the overall proportion of brain parenchyma.

While these 2D metrics are objective and easy to use, they simply aren't enough to map the volumetric complexity of the alcohol-damaged brain. To move beyond these constraints of linear 2D indices, we must shift into three dimensions. The Ventricle-to-Brain Ratio (VBR) does exactly this by expressing the relationship between total ventricular CSF (ml) and total brain parenchymal volume (ml). Unlike linear measures, the VBR is sensitive enough to detect subtle asymmetries and longitudinal shifts in tissue density. The real clinical power, however, lies in further refinement: normalizing regional volumes-such as the prefrontal cortex or hippocampus-to total intracranial volume. When we express these findings as Z-scores against standard population databases, we move from a subjective "guess" to a standardized, age-adjusted assessment. This provides a clear, statistically grounded picture of a patient's unique neuroanatomical profile.

Implementation in Clinical Workflow

Modern MRI workflows have evolved from purely subjective visual inspection of films to precise digital measurement. Using high-resolution 3D T1-weighted sequences (such as MPRAGE), clinicians are now able to generate a comprehensive volumetric

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map of the brain. Automated software platforms, including FreeSurfer, FSL, and 3D Slicer, are used to segment the brain into its individual components-measuring the thickness of the cortex and the volume of deep brain structures. Now these measurements are compared to large known databases of healthy individuals. This permits the findings to be expressed as Z-scores, in other words, how much on average a given value deviates from the population mean. Although this technique is largely automated, rigorous quality control remains indispensable; radiologists must visually re-inspect the scans to correct artefacts arising from motion or severe atrophy. Additionally, tools such as ComBat are used to "harmonise" data results, ensuring that results are consistent even if the patient is scanned on different MRI machines across different centres.

Neurorecovery, Radiomics, and Brain Age

Quantitative MRI volumetry transcends the boundaries of provision of a static snapshot of brain injury; it enables follow-up of structural recovery. Longitudinal studies have shown that within weeks to months of sustained abstinence, partial restoration of grey and white matter volumes can be observed, particularly in the prefrontal cortex, hippocampus, and cerebellum Bühler et al. [15] & Monnig et al. [16]. Voxel-Based Morphometry (VBM) addresses the limitations of visual inspection by analyzing brain scans at the voxel level Ashburner & Friston [17], enabling the detection of subtle structural changes that correlate with clinical recovery. Radiomics extends this analytical scope by extracting quantitative features such as texture, intensity patterns, and spatial heterogeneity Gillies et al. [18]. This radiomic information can reveal microstructural alterations caused by neuroinflammation or demyelination even before significant atrophy is visible. Beyond volumetry and radiomics, brain age prediction models use machine learning to estimate an individual's neurobiological age. In individuals with AUD, this creates a brain-age gap that reflects accelerated neurobiological ageing Gaser et al. [19] & Franke et al. [20]. Sagefusionnet is a new artificial intelligence tool that looks at how different parts of the brain are connected, helping to predict a person's brain age with greater accuracy. Right now, it's still being tested in research studies, so it isn't used by doctors in everyday care yet. But in the future, tools like Sagefusionnet could help doctors give patients clearer, more personal information about their brain health and what to expect as they age Kumar S &Hazarika S [21].

Conclusion

Towards Quantitative Clinical Translation. Merging traditional linear indices with modern volumetric ratios and radiomics does more than just update our toolkit-it builds a truly rigorous framework for diagnosing Alcohol Use Disorder. By moving toward quantitative markers like the Ventricle-to-Brain Ratio (VBR) and region-specific volumes, we can finally stop guessing and start measuring. These metrics provide what the field has lacked: objective, reproducible biomarkers that not only map the extent of an injury but also track the precise path of a patient's recovery. This synthesis of decades of imaging research isn't just a theoretical exercise; it offers a practical, ready-to-use roadmap for bringing volumetric MRI out of the lab and into the daily routine care of those struggling with AUD.

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