Cerebral Blood Flow: Regulation, Disorders, Impact

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Introduction

This article comprehensively reviews the intricate mechanisms governing cerebral blood flow (CBF) regulation, highlighting its critical role in maintaining brain function and health. It delves into metabolic, neurogenic, and myogenic factors influencing CBF, emphasizing how dysregulation contributes to various neurological disorders such as stroke, dementia, and traumatic brain injury [1].

This review explores the complex interplay between neuronal activity and cerebral blood flow, known as neurovascular coupling (NVC), which ensures adequate oxygen and nutrient supply to active brain regions. It outlines the cellular and molecular mechanisms underlying NVC, including signaling pathways involving astrocytes, pericytes, and endothelial cells [2].

This paper provides an overview of recent advancements in non-invasive techniques for measuring cerebral blood flow (CBF), emphasizing methods like arterial spin labeling (ASL) MRI, transcranial Doppler ultrasound (TCD), and near-infrared spectroscopy (NIRS). It discusses the principles, applications, advantages, and limitations of each technique [3].

This article reviews how cerebral blood flow (CBF) changes with healthy aging and its role in the progression of neurodegenerative diseases. It discusses the physiological decline in CBF observed in older adults and how this decline is exacerbated in conditions like Alzheimer's and Parkinson's disease [4].

This review examines the critical role of cerebral blood flow (CBF) in the acute phase and long-term recovery following ischemic stroke. It details how compromised CBF contributes to neuronal damage and how its restoration influences functional outcomes. The article discusses various therapeutic strategies aimed at optimizing CBF [5].

This review investigates how both acute bouts and long-term engagement in exercise influence cerebral blood flow (CBF). It elucidates the physiological mechanisms through which exercise enhances CBF, including improved endothelial function, increased cardiac output, and altered cerebrovascular reactivity. These changes improve cognitive function and reduce neurodegenerative disease risk [6].

This critical review examines the role of cerebral blood flow (CBF) dysregulation in the initiation and progression of various forms of dementia, including Alzheimer's disease and vascular dementia. It highlights how impaired CBF leads to chronic cerebral hypoperfusion, exacerbating amyloid-beta pathology, tauopathy, and neuroinflammation [7].

This comprehensive review focuses on cerebral autoregulation (CA), the brain's ability to maintain stable cerebral blood flow despite fluctuations in systemic arterial pressure. It details various non-invasive and invasive methods for assessing CA, including transcranial Doppler, near-infrared spectroscopy, and intracranial pressure monitoring. Impaired CA is clinically significant in conditions like TBI, stroke, and subarachnoid hemorrhage [8].

This article delves into complex changes in cerebral blood flow (CBF) and metabolism after traumatic brain injury (TBI). Primary and secondary brain injuries lead to widespread CBF dysregulation, including periods of hyperperfusion and hypoperfusion, contributing to secondary neuronal damage. Individualized CBF monitoring and management strategies optimize oxygen delivery [9].

This updated review synthesizes current understanding of the complex relationship between cerebral blood flow (CBF) and migraine pathophysiology. It discusses how abnormalities in CBF, including cortical spreading depression and altered cerebrovascular reactivity, contribute to the aura and headache phases of migraine. The article explores the role of various neurovascular mechanisms [10].

Description

Cerebral blood flow (CBF) regulation involves intricate mechanisms, crucial for maintaining brain function and health. Metabolic, neurogenic, and myogenic factors influence CBF, and their dysregulation contributes to neurological disorders like stroke, dementia, and traumatic brain injury. Neurovascular coupling (NVC) details the complex interplay between neuronal activity and CBF, ensuring adequate oxygen and nutrient supply to active brain regions. Cellular and molecular mechanisms, involving astrocytes and endothelial cells, underpin NVC, with impairments linked to conditions such as Alzheimer's disease and stroke [C001, C002].

Recent advancements offer non-invasive techniques for measuring CBF, including arterial spin labeling (ASL) MRI, transcranial Doppler ultrasound (TCD), and near-infrared spectroscopy (NIRS). These methods have

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distinct principles, applications, and limitations, proving useful in research and clinical settings. Furthermore, cerebral autoregulation (CA) represents the brain's ability to maintain stable CBF despite systemic arterial pressure fluctuations. Various non-invasive methods assess CA, and its impairment is clinically significant in conditions like traumatic brain injury (TBI), stroke, and subarachnoid hemorrhage, guiding patient management [C003, C008].

CBF changes are observed with healthy aging, showing a physiological decline in older adults that is exacerbated in neurodegenerative diseases like Alzheimer's and Parkinson's. The mechanisms behind age-related CBF changes include vascular stiffening and endothelial dysfunction, highlighting CBF as a biomarker for early detection. Moreover, CBF dysregulation plays a central role in the initiation and progression of various forms of dementia, including Alzheimer's disease and vascular dementia, by causing chronic cerebral hypoperfusion and exacerbating pathologies [C004, C007].

In the context of ischemic stroke, CBF is critical for both acute phase management and long-term recovery. Compromised CBF directly contributes to neuronal damage, while its restoration significantly influences functional outcomes. Therapeutic strategies focus on optimizing CBF through approaches like reperfusion therapies and neuroprotection. Similarly, traumatic brain injury (TBI) induces complex changes in CBF and metabolism, with widespread dysregulation, including periods of hyperperfusion and hypoperfusion. Individualized CBF monitoring and management strategies are vital to optimize oxygen delivery and metabolic support, mitigating TBI-related morbidity and mortality [C005, C009].

Exercise, both acute and chronic, positively influences CBF through physiological mechanisms such as improved endothelial function, increased cardiac output, and altered cerebrovascular reactivity. These CBF changes have clinical implications for enhancing cognitive function and reducing the risk of neurodegenerative diseases. Finally, the relationship between CBF and migraine pathophysiology reveals abnormalities like cortical spreading depression and altered cerebrovascular reactivity, contributing to migraine's aura and headache phases. Targeting CBF modulation thus offers novel therapeutic avenues for migraine management [C006, C010].

Conclusion

Cerebral blood flow (CBF) regulation is crucial for brain function, influenced by metabolic, neurogenic, and myogenic factors. Dysregulation leads to various neurological disorders such as stroke, dementia, and traumatic brain injury. Neurovascular coupling (NVC), the interplay between neuronal activity and CBF, ensures adequate oxygen and nutrient supply to active brain regions, and its impairment contributes to conditions like Alzheimer's disease. Non-invasive techniques such as ASL MRI, TCD, and NIRS are advancing CBF measurement in both research and clinical settings for diagnosis and monitoring conditions like stroke, dementia, and psychiatric disorders.

CBF changes with healthy aging, showing a physiological decline that worsens in neurodegenerative diseases such as Alzheimer's and Parkinson's, making CBF a potential biomarker. In ischemic stroke, CBF is critical for acute phase management and long-term recovery, where its optimization through reperfusion therapies and neuroprotection impacts functional outcomes. Exercise, both acute and chronic, significantly enhances CBF through improved endothelial function and cardiac output, positively impacting cognitive function and reducing neurodegenerative disease risk.

CBF dysregulation is a key factor in the progression of dementias, including Alzheimer's and vascular dementia, by causing chronic cerebral hypoperfusion and exacerbating pathologies. Cerebral autoregulation (CA) maintains stable CBF despite blood pressure fluctuations, and its impairment is clinically significant in conditions like traumatic brain injury (TBI) and subarachnoid hemorrhage. TBI itself involves complex CBF and metabolism changes, necessitating individualized monitoring to optimize oxygen delivery. Finally, abnormalities in CBF, like cortical spreading depression, are implicated in migraine pathophysiology, suggesting CBF modulation as a therapeutic target.

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