Brain health is heart health

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We celebrated "World Brain Day" on 22nd July 2026 with the theme "Brain health: Access for all". This month, on 29th September, we celebrate World Heart Day under the theme "Don't Miss a Beat," to recognise hidden signs and symptoms of cardiovascular disease.

Both the brain and heart are vital organs, and their functions are closely interconnected through the heart–brain axis. One of the earliest clinical observations demonstrating this interaction was the presence of inverted wide T waves on the electrocardiogram following acute neurogenic dysfunction. This abnormality is termed "cerebral T waves or "Neurogenic T waves".[1] The cardiac–cerebral reflex represents an important component of this bidirectional relationship. The continuous linkage between cardiac sensory information and the central nervous system via the autonomic regulatory centre influences heart rate, cardiac conduction, and myocardial excitability.[2] The brain regulates cardiac function predominantly through the sympathetic and parasympathetic nervous system.[3] The central neurogenic pro-inflammatory signals from the hypothalamus and brainstem may influence vascular function and its circulation.[4] Emotional trauma, physical shock or sequelae of ischemic and haemorrhagic stroke led to sudden and excessive sympathetic discharge that may cause myocardial stunning and transient left ventricular dysfunction. This phenomenon is clinically recognised as "Takotsubo cardiomyopathy". The relationship between autonomic imbalance and Takotsubo syndrome has generated interest in therapies aimed at restoring autonomic equilibrium. Transcutaneous vagus nerve stimulation (VNS), which enhances parasympathetic activity, has been investigated in several clinical trials.[5]

The hypothalamic–pituitary–adrenal (HPA) axis represents an important component of the heart–brain axis, providing a neuroendocrine pathway through which chronic psychological or physiological stress can influence cardiovascular function.[3] During psychological stress, physiological stress, and alterations in circadian rhythm, neural signals activate the paraventricular nucleus of the hypothalamus, resulting in the release of corticotropin-releasing hormone (CRH) and arginine vasopressin. These hypothalamic hormones stimulate the anterior pituitary to secrete adrenocorticotropic hormone (ACTH), which subsequently acts on the adrenal cortex to promote the synthesis and release of glucocorticoids, predominantly cortisol. Under physiological conditions, the HPA axis is tightly regulated through a negative-feedback mechanism in which circulating glucocorticoids suppress CRH and ACTH secretion. Chronic stress, exogenous therapy, or endocrine disorders are associated with glucocorticoid excess. Prolonged glucocorticoid excess leads to obesity, metabolic syndrome, hypertension, and increases the risk of developing cardiomyopathies and subsequent heart failure.[6]

The brain and heart share many common vascular mechanisms, and systemic inflammation may contribute to vascular injury affecting both organs. Although cerebral small-vessel disease (SVD) and coronary atherosclerosis are distinct pathological entities, they share several important biological pathways, including endothelial dysfunction, chronic inflammation and oxidative stress. Furthermore, "microvascular angina" due to SVD of the heart was reported in patients with acute ischemic stroke. Berry et al., concluded in their paper that SVD is a multisystem disorder with a common pathophysiological basis that differentially affects the heart and brain in some patients.[7] Progressive changes in the intracranial vascular wall may contribute to arterial stenosis and, in selected conditions, aneurysm formation.[8] Diabetes mellitus further amplifies this vascular vulnerability through metabolic dysfunction, endothelial injury, oxidative stress, and impaired immune and inflammatory regulation. Modern neuroimaging, such as high-field magnetic resonance imaging (MRI), may detect subclinical cerebral vascular and microstructural abnormalities that reflect underlying small-vessel disease and may serve as a marker of systemic vascular risk. Microstructural changes intracranially can be detected by using high-tesla MRI, such as 3-Tesla or 7-Tesla, which demonstrates T2-weighted hyperintense white-matter lesions which may represent subclinical chronic small-vessel ischemic injury (silent micro-strokes).[9] Furthermore, high-resolution intracranial vessel-wall MRI High-resolution 3D T1-weighted black-blood vessel-wall MRI sequences with both pre- and post-contrast administration, combined with 3D T2-weighted sequences, provide detailed assessment of the arterial wall and can identify features of intracranial atherosclerotic disease that may not be apparent on conventional lumen-based angiographic imaging.[10] Avoidance of tobacco and nicotine exposure is important. Cigarette smoking and vaping cause endothelial injury affecting the vascular systems of both the heart and brain.[11-12]

The human brain receives approximately 20% of the body's total cardiac output. Changes in cardiac function and systemic haemodynamics can significantly affect cerebral perfusion and cognitive function.[13] Cardiovascular disorders such as atrial fibrillation (AF) and heart failure may

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adversely affect the brain through multiple mechanisms, including impaired cardiac output and abnormal cerebral perfusion. AF is particularly important because it substantially increases the risk of cardioembolic ischemic stroke. In this issue of the Medical Journal of Malaysia, Chee et al., discussed the benefits and challenges of anticoagulant therapy in elderly patients with AF for the prevention of recurrent stroke.[14] Although advanced age is associated with an increased risk of bleeding and other treatment-related complications, appropriate anticoagulation remains an important component of secondary stroke prevention in carefully selected elderly patients, with treatment decisions requiring an individualised assessment of both thromboembolic and bleeding risks.[14] For stroke-associated pneumonia, routine use of preventive antibiotic therapy is not recommended for all patients. Initiate antibiotic treatment promptly when pneumonia is clinically diagnosed, and follow local antimicrobial guidelines and microbiological considerations.[15] In this issue, Zulkfli et al. highlight the considerable burden of antimicrobial resistance among patients with pneumonia, emphasising the importance of targeted infection-control measures and effective antimicrobial stewardship.[16] Long-term brain health monitoring after stroke should incorporate comprehensive assessment and modification of vascular risk factors, lifestyle modification and psychosocial health assessment.[17-18]

Following neurological injury, neurorehabilitation provides an important mechanism for restoring functional connectivity. Passive limb movement can provide sensory input to the central nervous system and engage sensorimotor feedback pathways. Repeated, task-oriented rehabilitation may facilitate adaptive neuroplasticity and support the reorganisation of neural networks after stroke or other neurological injury.[19] Neuromodulation represents another emerging component of neurorehabilitation. VNS, with its ability to modulate autonomic and central neural networks, may have therapeutic potential in cognitive and functional recovery following ischaemic stroke.[20-21] The close relationship between cognitive decline and cerebrovascular health has also been highlighted by the Lancet Commission on dementia prevention, intervention and care. The 2024 Commission emphasised that a substantial proportion of dementia risk may be associated with potentially modifiable factors across the life course, including hypertension, smoking, physical inactivity, diabetes, excessive alcohol consumption, obesity, depression, social isolation, hearing impairment, traumatic brain injury and air pollution.[22]

Chronic mental exhaustion and prolonged psychological or physical stress can lead to persistent activation of the body's stress-response systems. Sustained activation of the sympathetic nervous system and HPA axis may contribute to hypertension, endothelial dysfunction, metabolic disturbances and chronic low-grade inflammation, thereby increasing cardiovascular and cerebrovascular vulnerability. These processes may be particularly relevant in ageing-related chronic inflammatory conditions.[23] Repetitive stress and prolonged overwork may also alter central nervous system processing. These factors can produce maladaptive neural plasticity, including central sensitisation and alterations in facilitatory and inhibitory neural systems.[24] Depression, chronic psychological stress and autonomic imbalance have been associated with adverse effects on cardiovascular function, including altered heart-rate variability and increased sympathetic activity.[25] Recognition and management of psychological stress should therefore form part of comprehensive cardiovascular and cerebrovascular risk reduction.

Brain health can be broadly defined as preserving neurological function by preventing or reducing factors that may cause brain injury, promoting recovery after neurological insult, and enhancing compensatory neural mechanisms through neuroplasticity and neuromodulation.[26-27] Nutrition represents an important modifiable determinant of both brain and cardiovascular health. A balanced, nutrient-rich diet can help reduce metabolic dysfunction, systemic inflammation, excess weight gain and vascular injury. A brain- and heart-healthy diet includes intake of leafy greens, berries, whole wheat, nuts and fish rich in omega-3 fatty acids. Conversely, excessive consumption of highly processed foods, added sugars, saturated fats and alcohol should be minimised.[28] Regular physical activity provides another important link between cardiovascular fitness and brain health. Activities such as walking, yoga, strength training, and balance exercises can improve cardiovascular fitness, peripheral and cerebral circulation and vascular function.[29] They can also promote neuroplasticity and support cognitive and functional health.[30] Sleep is another essential component of the heart–brain axis. Adequate restorative sleep, particularly sufficient deep sleep, supports neuronal recovery, memory consolidation, metabolic homeostasis and autonomic regulation. Conversely, persistent sleep disruption may adversely affect cardiovascular and neurological health.[31-32]

Maintaining brain health requires a proactive approach to the early identification and management of neurological and vascular risk factors. Brain health monitoring includes assessment of cognitive function, brain structure and volume, neurovascular status and neurofunctional assessment.[33] Key strategies for maintaining brain health and stroke prevention include controlling modifiable risk factors such as hypertension, diabetes, atrial fibrillation and smoking.[34] Metabolic health should also be addressed through appropriate control of blood glucose, lipid levels and body weight, as diabetes, dyslipidaemia and obesity contribute substantially to both cerebrovascular and cardiovascular disease.

Effective pharmacological management, however, depends not only on prescribing appropriate treatment but also on treatment adherence and implementation. In this issue, Devaraj et al., reported that the use of single-pill combination (SPC) therapy among hypertensive patients in a cross-sectional study was only 27.9%. Although single-pill combinations may simplify treatment regimens and potentially improve adherence and blood-pressure control, their utilisation remained limited and was influenced by factors including the presence of chronic kidney disease, physician preference and the absence of diabetes mellitus.[35] In this issue too, Ang et al., reported that medications acting on the cardiovascular system accounted for 39.6% of returned medications and highlighted medication adherence as an important factor associated with this problem.[36]

Despite increasing recognition of the heart–brain axis, important knowledge gaps remain. Current evidence does not yet fully explain how subtle cardiovascular abnormalities translate into structural and functional alterations within the brain, particularly before the development of clinically overt cardiovascular or neurological disease.[37] Further research integrating cardiovascular imaging, neuroimaging, autonomic assessment, biomarkers and longitudinal cognitive and functional outcomes will be necessary to better define these bidirectional pathways.

In conclusion, the heart and brain should not be considered as independent organs. Neurological injury can profoundly affect the heart, while cardiovascular dysfunction can, in turn, compromise cerebral perfusion and neurological function. Understanding this bidirectional heart–brain axis is therefore increasingly important in the prevention, diagnosis, and management of both cardiovascular and neurological disease.

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