



Gravitational ischemia in the brain—may explain why we sleep, and why we dream

J. Howard Jaster

Department of Medicine, London Corporation, Kuala Lumpur, Malaysia

Correspondence to: J. Howard Jaster, MD (ORCID: 0000-0001-5406-9554). Department of Medicine, London Corporation 159 Capital Square, Kuala Lumpur 50100, Malaysia. Email: harbert38104@yahoo.com.

Received: 01 May 2020; Accepted: 22 September 2020; Published: 25 March 2021.

doi: 10.21037/amj-20-116

View this article at: <http://dx.doi.org/10.21037/amj-20-116>

Is there a skin nurse in the house?

Healthcare students of all stripes are typically not very far into their hospital rotations when they first encounter the ‘skin nurse’, who makes daily visits to most hospital wards, pushing a large cart filled with all manner of salves, bandages, pillows, and props. The skin nurse administers treatment to bed sores, sacral decubitus ulcers, and other regional skin manifestations of immobility resulting from strokes, trauma, and paralysis. She often rolls her patients from one side to the other, and repositions their arms and legs to relieve pressure on areas of ischemia manifesting as focal incipient skin break-down. And she is not the only one whose skills are required to mitigate the effects of poor mobility. The respiratory therapist follows her to do chest percussions and postural drainage to mobilize pulmonary secretions which have become pooled in dependent parts of the lungs. Human physiology continuously battles an opponent so insidiously treacherous that few in medicine dare to speak its name—gravity.

‘Ischemia’ denotes the focal (and occasionally global) tissue changes that often occur while blood flow is transiently reduced, and the tissue has briefly incurred deprivation of glucose and other nutrients as well as gas exchange (1). The ‘tissue’ may be an area of skin, muscle, brain, or almost any part of the body. Ischemia is a type of injury, with a variety of etiologies, both internal (intra-luminal) and external (extra-luminal) to the blood vessels supplying the region (2-12). It may be caused by prolonged continuous regional external pressure as from sitting in a chair—as the chair ‘pushes’ against the skin and muscle of the buttocks. In comparison, most transient ischemic attacks (TIAs) are caused not by external pressure but by

intravascular disease (plaques and clots inside of blood vessels) in the brain or neck; and TIA’s cause focal (and occasionally global) neurological symptoms which typically resolve after a minute or so. Slurred speech is focal, neurologically, and unconsciousness is global. Ischemia is a dynamic (non-static) injury-process, most early stages of which are reversible—which correlates with a mild degree of injury, compared to injuries of lost blood flow that progress to more severe and permanent damage (infarction)—yet ischemia is nonetheless associated with significant intracellular changes.

The extra-vascular substrates of focal ischemia may be visually modelled by directly opposing the tip of the thumb to the tip of the index finger, which if pressed together typically reveal both nail beds blanching from pink toward white. It is not the application of pressure per se, but rather its resultant disruption in blood flow which leads to ischemia. Gravity affects the entire bodies of those of us on the earth’s surface. Except for a runner in mid-flight over a hurdle, gravity holds us in direct opposition to the surface of the earth, or maybe to some piece of furniture acting as its surrogate—and except for a ballerina doing a pirouette, those bodily areas of opposition to the earth’s surface are mostly broad and multiple at any given time. Potential focal gravitational ischemic lesions are always present somewhere in our skin and underlying muscles; and in normal physiology we move our bodies around to ‘chase’ them from one area of skin to another.

How the brain works in a nutshell

Encased in the skull, the brain is one of the least mobile and

least accessible organs in the body. The external surfaces of the brain lie still on the inside surface of the skull, just as the legs may sometimes lie still on the surface of a bed. The meninges and cerebrospinal fluid surrounding the brain may provide some cushioning, but do not mitigate the effects of gravity. Pancaking layers of progressively increasing weight from the over-lying brain tissue compress blood vessels and reduce blood flow in the bottom layers. In the arms, legs, and buttocks, potential focal gravitational ischemic lesions generate local discomfort over a period of time, and in normal physiology we relieve that discomfort by moving into a different position. Mental fatigue, and sleepiness may be the 'local discomfort' from regional gravitational ischemia in the brain, which we eventually relieve by changing body and head positions toward horizontal.

In any given body position, from upright to horizontal, the 'top' half of the brain (farthest from the center of the earth) is sitting on the 'bottom' half as a weight-burden. Our head and body positions are roughly vertical for 16 hours a day, and then roughly horizontal for 8 hours at night during sleep. During our vertical (upright) and waking hours of the day, our brain stem and hypothalamus are located toward the 'bottom' of the brain where they incur the most gravitational ischemia. They contain significant components of the reticular activating system, which mediates arousal, alertness, and wakefulness. Gradually increasing levels of regional ischemia occurring throughout the day are associated with increasing fatigue, and finally drowsiness. How much does gravitational ischemia influence brain physiology? Maybe a lot.

To sleep, perchance to dream

It is plausible that a primordial physiological adaptation in the brain was the development of diurnal sleep cycles to intermittently encourage a horizontal position relative to gravity so as to induce a restoration of blood flow in weight-bearing regions of the brain which would otherwise be continuously ischemic—ultimately incurring the more severe and permanent changes to infarction. The horizontal positioning associated with sleep (often with nose up, and back of the head down) helps to redistribute both gravitational ischemia and blood flow.

The reversibility of ischemia is of critical importance for it possibly to function as a component of 'normal' physiology. The occurrence of incipient structural and chemical changes at the neuronal cellular level in brain ischemia, although studied mostly in occlusive cerebrovascular disease

in animals (13), opens the door to possible transient re-routing of neuronal pathways by blocking some pathways and opening others (14)—ultimately allowing for sleep, which is an electrical phenomenon, not purely gravitational. We may get clues about an 'ischemic re-routing' of neural circuitry from phenomena like seizures (abnormal electrical discharges) that can be provoked or exacerbated in susceptible individuals by fatigue or by prolonged externally enforced wakefulness—typically associated with vertical body position and increased gravitational ischemia in brain regions near the skull base, such as the temporal lobes, a frequent etiological focus for seizure activity.

Yet sometimes the transformation of ischemic cellular changes to electrical sleep phenomena does not happen efficiently. Everyone on the earth's surface presumably has gravitational ischemia in the brain, but not everyone sleeps well. Insomnia is an annoying and potentially dangerous inability to sleep—often associated with an inability to feel rested and alert following attempted sleep. Although insomnia may potentially be related to many environmental and life-style factors (noise, caffeine), it may also be related to a mis-match of gravitational ischemia to the reticular activating system geographically (in spatial distribution). The reticular activating system is a loose network of neurons, typically located in certain general areas, but there may be individual variations, anatomically—and especially in relationship to the specific geography of gravitational ischemia.

Clinical relevance may be found in the relationship between gravity and benign positional vertigo (spinning sensation with imbalance). This potentially disabling condition may be related to the disruption of fluid flow in the semicircular canals of the inner ear. And therapy often consists of head tilting exercises which 'train the brain' to respond normally to positional changes. Benign positional vertigo itself provides empirical evidence of how the brain uses gravity-based mechanisms to serve crucial functions like balance, and how such mechanisms can sometimes become disordered—and how therapy can sometimes be relatively simple and non-invasive. Head-tilting manoeuvres may as well be therapeutic in some cases of insomnia. Further research is needed.

An injury or metabolic insult to the nervous system may simultaneously or alternately produce paired signs or symptoms—one 'positive' (displaying aberrant hyperactivity) and the other 'negative' (displaying partial inactivity) in terms of the specific function of the involved neural tissue. Common pairs include tingling and numbness, tinnitus

(ringing) and hearing loss, manic and depressive thoughts and behavior (15-17), scintillating scotomas and blind spots. Dream-images during sleep may be a ‘positive’ symptom of gravitational ischemia in the occipital (visual) cortex, which is typically on the ‘bottom’ of the brain during sleep—while vision during sleep is functionally depressed, a ‘negative’ symptom.

This model of gravitational ischemia is not intended to undermine current concepts of psychological and environmental influences upon dreaming—but rather to potentially provide biological support. However, the intensity of a neurological response is often proportional to the severity of the injury. A small degree of occipital ischemia may be associated with dreaming, while a greater degree is associated with nightmares. This could again relate to individual anatomical variations.

Previously considered to be strictly a pathological process—and in the brain associated mostly with TIAs and ischemic penumbras (shadows) surrounding new strokes, both resulting from occlusive cerebrovascular disease— ischemia may function as an important and pervasive component of normal neurophysiology caused by gravity.

Acknowledgments

Funding: None.

Footnote

Provenance and Peer review: This article was commissioned by the editorial office, *AME Medical Journal*. The article has undergone external peer review.

Conflicts of Interest: The author has completed the ICMJE uniform disclosure form (available at <http://dx.doi.org/10.21037/amj-20-116>). The author has no conflicts of interest to declare.

Ethical Statement: The author is accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved.

Open Access Statement: This is an Open Access article distributed in accordance with the Creative Commons Attribution-NonCommercial-NoDerivs 4.0 International License (CC BY-NC-ND 4.0), which permits the non-commercial replication and distribution of the article with

the strict proviso that no changes or edits are made and the original work is properly cited (including links to both the formal publication through the relevant DOI and the license). See: <https://creativecommons.org/licenses/by-nc-nd/4.0/>.

References

1. Wang S, Du H, Lin B, et al. Spatial and temporal identification of cerebral infarctions based on multiphotonmicroscopic imaging. *Biomed Opt Express* 2018;9:2312-25.
2. Jaster JH. Reperfusion injury to ischemic medullary brain nuclei after stopping continuous positive airway pressure-induced CO₂-reduced vasoconstriction in sleep apnea. *J Thorac Dis* 2018;10:S2029-31.
3. Jaster JH. Novel mechanisms and prevention for sudden unexpected death related to medullary brain lesions. *Arch Neurol* 2010;67:1288.
4. Jaster JH. Acute Solitary Tract Nucleus Insufficiency in Chronic Heart Failure. *Neurol Curr Res* 2018;1:1001.
5. Jaster JH. Ischemic lesion formation in solitary tract nuclei during central sleep apnea with heart failure. *Circ J* 2016;80:1047.
6. Jaster JH. Medullary neuropathology in sleep apnoea. *Respirology* 2017;22:829.
7. Jaster JH. Medicine in the future—with subspecialists in medullary neurology and brain dentistry. *World J Neuro* 2015;4:107-12.
8. Jaster JH. Interfacing pathophysiology of respiratory sleep disorders, cardiac dysfunction, and focal autonomic medullary brain ischemia. *J Thorac Dis* 2015;7:E646-7.
9. Jaster JH. CO₂-Related Vasoconstriction Imposed on Medullary Brain Ischemia During Sleep Apnea in Heart Failure by Adaptive ServoVentilation May Trigger Sudden Death. *J Forensic Med Forecast* 2018;1:1010.
10. Jaster JH. Laryngospasm and “sudden unexpected death related to medullary brain lesions”. *Arch Neurol* 2011;68:399.
11. Jaster JH, Zamecnik J, Gianni AB, et al. CO₂-related vasoconstriction superimposed on ischemic medullary brain autonomic nuclei may contribute to sudden death. *Cardiovasc Pathol* 2019;38:42-5.
12. Jaster JH. Phrenic nerve stimulation—and chorda tympani nerve stimulation—may prevent lethal cardiac arrhythmias during sleep apnea with heart failure. *Int J Neurol Res* 2017;3:323-6.
13. Barth AM, Mody I. Changes in hippocampal neuronal activity during and after unilateral selective hippocampal

- ischemia in vivo. *J Neurosci* 2011;31:851-60.
14. Taxin ZH, Neymotin SA, Mohan A, et al. Modeling molecular pathways of neuronal ischemia. *Prog Mol Biol Transl Sci* 2014;123:249-75.
 15. Zhu J, Zhuo C, Liu F, et al. Neural substrates underlying delusions in schizophrenia. *Sci Rep* 2016;6:33857.
 16. Jaster JH. COPD-and-smoking-induced “down regulation” of CO₂-related vasoconstriction in the brain during CPAP for sleep apnea may paradoxically reduce risk of cardiovascular events. *Sleep Breath* 2020;24:1181-2.
 17. Jaster JH, Zamecnik J, Bartos A, et al. Unexpected sudden death caused by medullary brain lesions involves all age groups and may include ‘sudden infant death syndrome’ as a subset. *Acta Neuropathol* 2005;109:552-3.

doi: 10.21037/amj-20-116

Cite this article as: Jaster JH. Gravitational ischemia in the brain—may explain why we sleep, and why we dream. *AME Med J* 2021;6:11.