



PHYSIOLOGY INSIGHT

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As the executive cortex refines thought and action, this eminent body guides Physiology Insight with intellect, intent, and enduring leadership

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EDITORIAL

ASSOPI – UP CHAPTER QUARTERLY NEWSLETTER (2026)



DR. DOLLY RASTOGI

PROFESSOR, DEPT OF PHYSIOLOGY, GSVM MEDICAL COLLEGE, KANPUR
VICE PRESIDENT, ASSOPI UP CHAPTER

It is a privilege to connect with the members of the Association of Physiologists of India (ASSOPI), UP Chapter, through this edition of our quarterly newsletter. As Vice President of the chapter, I am delighted to witness the continued enthusiasm and academic excellence demonstrated by our members in advancing the discipline of physiology through education, research, and professional collaboration. The past quarter has been marked by several meaningful academic activities, including CMEs/workshops, and our flagship *Physiology Distilled* webinar series. These platforms have provided valuable opportunities for faculty members, researchers, and students to engage in discussions on contemporary developments in physiology and their translational applications in healthcare and medical education. Such initiatives continue to strengthen our academic network while promoting lifelong learning and evidence-based practice. It is particularly encouraging to observe the growing participation of young faculty members and postgraduate students in research presentations and scholarly discussions.

Our collective efforts should continue to focus on promoting high-quality research, effective teaching methodologies, ethical professional conduct, and collaborative learning that ultimately benefits both learners and patients. This newsletter reflects the dedication and scholarly contributions of our members. I sincerely appreciate all authors, office bearers, reviewers, and organizers whose consistent efforts have made this publication a valuable academic resource. I also extend my gratitude to every member of the ASSOPI UP Chapter for their continued support, active participation, and commitment to strengthening our professional fraternity.

Let us continue working together with renewed enthusiasm to promote academic excellence, inspire meaningful research, and uphold the highest standards of physiology education and practice.

With warm regards,

DR. DOLLY RASTOGI

PROFESSOR, DEPT OF PHYSIOLOGY, GSVM MEDICAL COLLEGE, KANPUR
VICE PRESIDENT, ASSOPI UP CHAPTER

A RENEWED VOICE: EDITORIAL NOTE PIONEERING THE NEXT WAVE APRIL–JUNE 2026

Dear Esteemed Colleagues and Members of the Physiology Fraternity,

It is with great pleasure that we present the April – June 2026 edition of the ASSOPI – UP Chapter Quarterly Newsletter.

The past quarter has been an incredibly vibrant and dynamic period for our chapter. We have witnessed enthusiastic participation across various academic platforms throughout the state. From the spirited participation of our students/researchers in writing articles for our newsletter to forward-looking Continuing Medical Education (CME) programs focusing on the integration of artificial intelligence in contemporary medical research and education, these milestones reflect our collective commitment to advancing physiological sciences and keeping our teaching methodologies at the cutting edge.

As we navigate the evolving landscape of medical education and clinical research, it is truly heartening to see our community actively exploring diverse and impactful avenues. Whether our members are uncovering the intricate metabolic interplay of circadian rhythms, utilizing advanced diagnostic assessments in clinical settings, or championing physiological lifestyle interventions like yoga, the dedication to bridging foundational physiology with clinical application remains stronger than ever.

In this issue, we bring you a curated compilation of these recent academic endeavours, insightful scientific articles, and vital departmental updates from medical colleges across Uttar Pradesh. We hope these pages serve not only as a record of our recent achievements but also as a catalyst for future collaborative research and innovation within our fraternity.

We extend our deepest gratitude to all the contributors, senior faculty members, and the ASSOPI leadership for their unwavering support and guidance in bringing this edition to life. We also thank our readers for their continuous encouragement.

Happy reading, and we look forward to your valuable feedback.

- **EXECUTIVE EDITORS:** DR. AMIT KANT SINGH, DR. ANAMIKA SINGH
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Scientific Articles

Scientific Articles

The Pulse of
Scientific Progress



DIGITAL EYE STRAIN: UNDERSTANDING THE PHYSIOLOGY BEHIND AN EMERGING LIFESTYLE DISORDER

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MBBS 2025 BATCH, JNMC AMU

ABSTRACT

The digital revolution has transformed education, healthcare, and communication, making prolonged screen use an inevitable part of daily life. Medical students, in particular, spend several hours each day using smartphones, tablets, and laptops for lectures, e-books, research, and online assessments. While these technologies have improved access to knowledge, they have also led to a growing health concern known as Digital Eye Strain (DES), or Computer Vision Syndrome (CVS). Digital Eye Strain encompasses a group of ocular and extraocular symptoms such as eye fatigue, blurred vision, dry eyes, headaches, and neck pain associated with prolonged digital device use. The condition arises due to physiological changes involving reduced blink rate, tear film instability, sustained accommodation, and poor ergonomic posture.

INTRODUCTION:

Digital devices have become indispensable tools in modern education and healthcare. Medical students frequently use laptops, smartphones, and tablets for online classes, digital textbooks, question banks, and medical applications. Although these technologies enhance learning, prolonged screen exposure places significant stress on the visual system.

Digital Eye Strain (DES), commonly referred to as Computer Vision Syndrome, is a collection of eye and vision-related symptoms that occur during or after prolonged use of digital screens. According to the American Optometric Association, DES includes symptoms such as eye strain, dry eyes, blurred vision, headaches, and musculoskeletal discomfort. Recent studies have shown that more than two-thirds of medical students experience at least one symptom of DES, making it an important yet often neglected health issue.

Unlike reading printed material, digital screens require continuous focusing because images are formed by pixels rather than sharply defined characters. Combined with decreased blinking and poor posture, prolonged screen use creates physiological stress on the eyes and surrounding muscles, leading to visual discomfort and fatigue.

PHYSIOLOGY BEHIND DIGITAL EYE STRAIN:

The human eye maintains clear vision during near work through the coordinated action of accommodation, convergence, and pupillary constriction, collectively known as the near response. When viewing a nearby object, the ciliary muscle contracts, allowing the crystalline lens to become more

convex and increasing its refractive power. Simultaneously, both eyes converge to maintain single binocular vision.

During prolonged screen use, the accommodative system remains continuously active. Unlike printed text, digital images have less distinct edges, forcing the eyes to make constant micro-adjustments to maintain focus. Over time, this sustained accommodative effort results in accommodative fatigue, causing symptoms such as blurred vision, difficulty focusing, and eye strain.

Another important physiological mechanism involves the tear film, which consists of lipid, aqueous, and mucin layers. This thin film maintains a smooth optical surface and lubricates the cornea. Under normal conditions, humans blink approximately 15–20 times per minute. However, while concentrating on digital screens, blink frequency decreases significantly, often by nearly 50%. Incomplete blinking further prevents uniform distribution of tears over the ocular surface.

Reduced blinking increases tear evaporation, leading to tear film instability and ocular surface dryness. Consequently, individuals experience burning sensation, foreign body sensation, redness, watering, and irritation. Ironically, excessive watering often represents a reflex response to dryness rather than adequate lubrication.

Poor posture during prolonged device use also contributes to Digital Eye Strain. Continuous forward bending of the neck and sustained contraction of shoulder muscles produce neck pain, shoulder stiffness, and tension headaches, making DES a condition with both ocular and musculoskeletal manifestations.

EVIDENCE FROM SCIENTIFIC STUDIES:

Several studies have demonstrated the growing prevalence of Digital Eye Strain among students and professionals. Logaraj et al reported that 78.6% of medical students experienced symptoms of Computer Vision Syndrome, with prolonged screen exposure and poor ergonomic practices identified as major risk factors. Similarly, Ranasinghe et al. found a high prevalence of DES among office workers, highlighting prolonged computer use and inadequate workplace ergonomics as significant contributors. Rosenfield emphasized that accommodative stress, decreased blinking, and tear film instability are the primary physiological mechanisms responsible for Digital Eye Strain. Furthermore, Sheppard and Wolffsohn described DES as an emerging public health concern due to the increasing dependence on digital technology. The Computer Vision Syndrome Questionnaire (CVS-Q), developed by Seguí et al., has become a widely accepted tool for assessing DES symptoms in epidemiological studies. Together, these studies demonstrate that Digital Eye Strain is a multifactorial condition influenced by visual physiology, behavioral habits, and environmental factors.

PREVENTION: A PHYSIOLOGY-BASED APPROACH:

Fortunately, Digital Eye Strain is largely preventable. One of the simplest recommendations is the 20-20-20 rule, which advises looking at an object 20 feet away for 20 seconds after every 20 minutes of screen use. This allows the ciliary muscle to relax and reduces accommodative stress. Conscious blinking is equally important as it restores the tear film and prevents excessive evaporation. Maintaining an

appropriate viewing distance of approximately 50–70 cm, positioning the screen slightly below eye level, reducing glare, and ensuring adequate lighting also improve visual comfort. Regular breaks, proper posture, and correction of refractive errors further reduce the risk of DES. Educational institutions should encourage awareness regarding healthy digital habits, particularly among students who spend long hours using electronic devices.

CONCLUSION:

Digital Eye Strain has become an increasingly common consequence of prolonged screen use in today's technology-driven world. Although it does not usually cause permanent visual damage, its symptoms can significantly affect academic performance, productivity, and quality of life. Understanding the physiological mechanisms including accommodative fatigue, reduced blink rate, and tear film instability, provides the foundation for effective prevention. Simple behavioral changes such as regular breaks, conscious blinking, proper ergonomics, and healthy screen habits can substantially reduce the burden of Digital Eye Strain. As digital learning continues to expand, promoting awareness about ocular health should become an integral part of medical education, ensuring that technology remains a tool for learning rather than a source of visual discomfort.

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CORRELATION BETWEEN PHYSICAL ACTIVITY AND COGNITIVE PERFORMANCE AMONG MBBS STUDENTS

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ABSTRACT

BACKGROUND: Physical activity has long been recognized as a key determinant of overall health, yet its role in shaping cognitive function among medical students remains insufficiently explored. MBBS students, subjected to intense academic demands, sedentary study hours, and chronic psychological stress, constitute a uniquely vulnerable population. This article synthesizes existing physiological evidence to examine the relationship between physical activity levels and cognitive performance—encompassing memory, attention, executive function, and academic achievement—in undergraduate medical students.

METHODS AND SCOPE: A narrative review approach was adopted, drawing upon established principles of neurophysiology, exercise physiology, and cognitive neuroscience, with reference to standard textbooks and peer-reviewed evidence.

RESULTS: Regular aerobic and resistance exercise enhances hippocampal neurogenesis, augments cerebral blood flow, modulates neurotransmitter dynamics, and reduces hypothalamic-pituitary-adrenal (HPA) axis overactivation—all of which contribute to measurable improvements in cognitive performance. MBBS students who engage in structured physical activity demonstrate superior memory consolidation, faster processing speed, improved attention, and lower rates of academic burnout.

CONCLUSION: Physical activity should be integrated as a neurophysiological intervention within the medical curriculum. Educational institutions must facilitate structured exercise opportunities alongside academic schedules.

KEYWORDS: Physical activity, cognitive function, MBBS students, neuroplasticity, hippocampal neurogenesis, exercise physiology, academic performance.

1. INTRODUCTION:

The MBBS curriculum demands sustained intellectual rigour over an extended academic period. Students are expected to assimilate vast volumes of biomedical knowledge while simultaneously developing clinical reasoning and professional competencies. This academic intensity, compounded by irregular sleep schedules, nutritional compromise, and near-total physical inactivity, creates a physiological milieu that may paradoxically impair the very cognitive faculties essential for medical education. Cognitive

performance—broadly encompassing working memory, sustained attention, executive function, and information processing speed—is not merely a function of intellectual capacity. It is intimately governed by the neurobiological state of the brain, which is in turn profoundly influenced by physical activity levels. The neuroscience of exercise has evolved dramatically over the past two decades, transitioning physical activity from the domain of cardiovascular medicine into the frontier of cognitive neuroscience. Despite this growing evidence base, medical schools across India and internationally have been slow to incorporate physical activity as a structured component of the undergraduate curriculum. This is especially concerning given that MBBS students are the future bearers of population health—yet their own health behaviours during training may be undermining their cognitive potential. This article aims to explore, through a physiological lens, the mechanistic and empirical correlations between physical activity and cognitive performance in MBBS students, and to advocate for evidence-based integration of exercise into medical education.

2. PHYSIOLOGICAL MECHANISMS LINKING EXERCISE TO COGNITION:

2.1 HIPPOCAMPAL NEUROGENESIS AND BDNF-

The hippocampus, critical for memory encoding and spatial navigation, is one of the few brain regions capable of adult neurogenesis. Exercise—particularly sustained aerobic activity—has been shown to significantly upregulate brain-derived neurotrophic factor (BDNF), a neurotrophin that promotes neuronal survival, synaptic plasticity, and the proliferation of new neurons in the dentate gyrus of the hippocampus.¹ BDNF binds to tropomyosin receptor kinase B (TrkB) receptors, activating downstream signalling cascades including the MAPK/ERK and PI3K/Akt pathways, which in turn stimulate the expression of genes associated with long-term potentiation (LTP)—the cellular substrate of learning and memory. Guyton and Hall (2021) describe how hippocampal LTP underlies declarative memory consolidation, precisely the type of cognitive function demanded during MBBS examinations. Regular exercise, by potentiating BDNF-mediated LTP, thus creates a biological advantage for memory-intensive academic tasks.¹

2.2 CEREBRAL HEMODYNAMICS AND OXYGENATION-

Physical exercise induces a marked increase in cardiac output and regional cerebral blood flow (CBF), particularly to the prefrontal cortex and hippocampus. During aerobic exercise, cerebral autoregulation ensures that increased systemic perfusion pressure translates into proportional delivery of oxygen and glucose to metabolically active neural circuits. Chronic exercise training promotes cerebrovascular adaptations including angiogenesis, increased capillary density within cortical grey matter, and enhanced endothelial nitric oxide synthase (eNOS) activity. These changes collectively improve the efficiency of cerebral oxygen extraction and waste product clearance, sustaining neural metabolism during prolonged cognitive effort. For MBBS students, whose academic sessions often extend for several hours, this improved cerebrovascular reserve may substantially mitigate cognitive fatigue.

2.3 NEUROTRANSMITTER MODULATION-

Exercise exerts profound effects on the monoaminergic neurotransmitter systems. Acute aerobic exercise increases the synaptic availability of dopamine, serotonin, and norepinephrine within the mesocortical

and mesolimbic circuits. These neurotransmitters are central regulators of executive function, mood, motivation, and attentional control. Dopaminergic signalling within the prefrontal cortex (PFC) governs working memory and goal-directed behaviour, as outlined in the neurophysiology of cognition by Kandel et al. in *Principles of Neural Science* (2021). Serotonin, produced primarily in the dorsal raphe nuclei, modulates emotional regulation and impulse control. Norepinephrine, released from the locus coeruleus, optimises arousal and signal-to-noise ratios within cortical networks. Regular physical activity, by stabilising these neurotransmitter systems, produces a neurochemical environment conducive to sustained academic performance.²

2.4 HPA AXIS REGULATION AND STRESS ATTENUATION-

MBBS students are chronically exposed to academic stressors that activate the hypothalamic-pituitary-adrenal (HPA) axis, resulting in sustained elevations of cortisol. While transient cortisol release is adaptive, chronic hypercortisolemia is neurotoxic—particularly to the hippocampus, where glucocorticoid receptors are densely expressed. Prolonged cortisol exposure suppresses BDNF expression, inhibits neurogenesis, and causes dendritic atrophy in CA3 pyramidal neurons, all of which impair memory consolidation. Regular moderate-intensity exercise serves as a powerful neuroendocrine buffer. By habituating the HPA axis to repeated stressor exposure, exercise training reduces both basal cortisol levels and the magnitude of cortisol responses to psychological stressors. This attenuation of chronic stress neurobiology preserves hippocampal structural integrity and optimises conditions for memory formation—a critical benefit for MBBS students navigating year-round examination pressure.

2.5 SLEEP ARCHITECTURE AND MEMORY CONSOLIDATION-

Sleep disturbances are endemic among medical students, driven by examination anxiety, call duties, and disrupted circadian rhythms. Sleep is physiologically non-negotiable for memory consolidation: slow-wave sleep (SWS) facilitates hippocampo-neocortical transfer of declarative memories, while REM sleep consolidates procedural and emotional memories, as described in detail by Ganong's *Review of Medical Physiology* (2019). Physical exercise has a well-documented chronobiological effect. Moderate exercise, particularly in the morning or afternoon, advances sleep onset, deepens SWS, and stabilises circadian melatonin secretion from the pineal gland. For MBBS students, improved sleep architecture following regular physical activity represents an indirect but highly significant pathway to enhanced cognitive performance.³

3. PHYSICAL ACTIVITY AND SPECIFIC COGNITIVE DOMAINS IN MBBS STUDENTS:

3.1 MEMORY AND RECALL-

Medical education is fundamentally a memory-intensive enterprise. From gross anatomy to pharmacological mechanisms, students must encode and retrieve vast associative networks of factual knowledge. Aerobic exercise, through BDNF-mediated hippocampal neurogenesis and enhancement of synaptic plasticity, has been demonstrated to improve both the acquisition and long-term retention of declarative information. Studies comparing physically active and sedentary medical students have consistently reported superior performance in anatomy and biochemistry assessments among those

who engaged in regular aerobic activity—a finding aligned with the established neurophysiology of exercise and memory.^{4,6,8}

3.2 ATTENTION AND CONCENTRATION-

Attention—the ability to selectively focus cognitive resources—is governed by a distributed network involving the prefrontal cortex, anterior cingulate cortex, and parietal association areas. Exercise-mediated elevations in norepinephrine and dopamine enhance the gain of attentional networks, reducing distractibility and increasing processing efficiency.⁷ Medical students who exercise regularly demonstrate improved performance on sustained attention tasks and report fewer episodes of mind-wandering during lectures. This is particularly relevant during clinical skills sessions and simulation-based training, where lapses in attention carry direct patient safety implications.⁷

3.3 EXECUTIVE FUNCTION AND CLINICAL REASONING-

Executive functions—including working memory, cognitive flexibility, inhibitory control, and planning—are mediated primarily by the dorsolateral prefrontal cortex (DLPFC). These higher-order cognitive processes are indispensable for clinical decision-making, differential diagnosis, and procedural learning in medical training. Resistance training, in addition to aerobic exercise, has been shown to selectively enhance executive function through mechanisms distinct from aerobic pathways, including insulin-like growth factor-1 (IGF-1) signalling, which promotes synaptic density in the PFC. Integrating both modalities of exercise thus provides complementary cognitive benefits extending across memory and executive domains.^{11,12}

3.4 MOOD, MOTIVATION, AND ACADEMIC BURNOUT-

Burnout—characterised by emotional exhaustion, depersonalisation, and a sense of reduced personal accomplishment—is alarmingly prevalent among MBBS students globally, with reported rates ranging from 30% to over 60% in some cohorts. Burnout profoundly impairs cognitive engagement, academic motivation, and ultimately performance.⁹ Physical exercise is among the most robustly validated non-pharmacological interventions for depression and anxiety. Exercise-induced serotonin and endocannabinoid release, alongside reduced HPA axis reactivity, directly counters the neurobiological substrate of burnout. Medical students who maintain regular physical activity report significantly higher scores on validated measures of academic engagement and lower rates of clinical-level depressive symptoms.⁵

4. THE CURRENT LANDSCAPE: PHYSICAL INACTIVITY AMONG MBBS STUDENTS:

Despite the compelling evidence base, physical inactivity is paradoxically prevalent within medical student populations. Studies conducted in Indian medical colleges report that a significant proportion of MBBS students fail to meet WHO-recommended guidelines of at least 150 minutes of moderate-intensity aerobic activity per week. Barriers include excessive academic scheduling, inadequate recreational infrastructure within campus settings, social and cultural norms around prioritising study over physical activity, and the absence of institutional policy mandating exercise as part of student wellness programming.¹⁰ This irony—that the future physicians responsible for counselling patients about healthy

lifestyles are themselves among the least physically active graduate cohorts—demands urgent institutional attention. The cognitive costs of physical inactivity, as outlined throughout this article, are not trivial. They represent a systemic academic disadvantage with implications for both student wellbeing and the quality of future medical practice.

5. RECOMMENDATIONS FOR MEDICAL EDUCATION:

Based on the neurophysiological evidence reviewed, the following recommendations are proposed: Firstly, structured physical activity should be formally incorporated into the MBBS timetable as a scheduled, non-negotiable component—at minimum three sessions of 45 minutes per week. Evidence supports aerobic modalities (brisk walking, jogging, swimming, cycling) for memory and stress regulation, while resistance training provides complementary executive function benefits. Secondly, medical institutions should invest in on-campus recreational facilities accessible to students throughout the academic day, eliminating logistical barriers to exercise participation. Thirdly, faculty and administrators should be sensitised to the neuroscientific rationale for exercise, enabling them to advocate credibly for physical activity within the culture of medical education rather than treating it as a distraction from academic priorities. Fourthly, peer wellness programmes led by senior MBBS students who model physically active lifestyles can leverage social influence to normalise exercise within medical school culture. Finally, longitudinal tracking of physical activity habits alongside academic performance metrics within individual institutions would generate institution-specific evidence to inform policy—a recommendation aligned with the broader movement toward evidence-based medical education.

6. CONCLUSION:

The correlation between physical activity and cognitive performance in MBBS students is not merely statistical—it is deeply rooted in the neurophysiology of the exercising brain. Through mechanisms encompassing hippocampal neurogenesis, cerebrovascular remodelling, neurotransmitter optimisation, HPA axis habituation, and sleep architecture enhancement, regular physical activity creates a neurobiological environment that is demonstrably more conducive to learning, memory, attention, and executive function. For MBBS students, physical activity is not a luxury or a peripheral wellness concern—it is a evidence-based cognitive intervention. The medical curriculum must evolve to reflect this understanding. In cultivating physically active medical students, institutions are not diverting time from education; they are investing in the neurological substrate upon which excellent medical education depends.

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TYPE 2 DIABETES MELLITUS: EPIDEMIOLOGY, RISK FACTORS, COMPLICATIONS, AND GLYCEMIC CONTROL

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ABSTRACT

Type 2 diabetes mellitus (T2DM) is one of the most common long-term metabolic conditions in today's world. It is marked by high blood sugar levels over time, caused by the body's reduced ability to use insulin effectively and a gradual weakening of the pancreatic cells that produce insulin. T2DM makes up about 90 to 95 percent of all diabetes cases globally and has a major impact on public health, especially in India, which has the second-highest number of people living with diabetes. The condition is strongly influenced by lifestyle choices like obesity, lack of physical activity, and poor eating habits, as well as unchangeable factors such as genetic background and aging. When not properly managed, T2DM can lead to serious health issues that affect daily living and raise the risk of death. Keeping blood sugar levels under control, mainly through checking HbA1c levels, is crucial in managing T2DM. This article provides an overview of the spread, risk factors, different health problems, and methods of controlling blood sugar in T2DM, with a focus on the Indian situation. [1,4,5,8] Keywords: Type 2 diabetes mellitus, hyperglycemia, insulin resistance, HbA1c, glycemic control, microvascular complications, macrovascular complications, India.

1. INTRODUCTION:

Type 2 diabetes mellitus (T2DM) is a long-term metabolic disease that is defined by consistently high levels of glucose in the blood. This happens due to the body's resistance to insulin and a slow loss of function in the pancreatic cells that make insulin. Unlike Type 1 diabetes, which is caused by the immune system attacking insulin-making cells, T2DM develops gradually over years and often goes unnoticed until it causes health problems. [4,8] T2DM has become a major global health issue, accounting for the majority — about 90 to 95 percent — of all diabetes cases worldwide. It is no longer just a problem for older people or those in wealthy countries; T2DM now affects people of all ages and backgrounds, driven by changes in food, physical activity, and urban living. [1,4,8]. In India, the situation is especially concerning. The country has the second-largest number of people with diabetes globally, and the number is expected to rise sharply in the coming years. Therefore, understanding T2DM — its causes, progression, and effects — is very important for doctors, public health workers, and decision-makers. [1,4].

2. EPIDEMIOLOGY:

The number of people with T2DM has increased greatly over the past few decades. In India alone, around 77 million people were living with diabetes as of 2019. This number is expected to go up to over 134

million by 2045, which is an increase of nearly 75% in just one generation. In addition, about 136 million Indians have prediabetes — a condition where blood sugar levels are higher than normal but not yet high enough to be diagnosed as diabetes — and these individuals are at a high risk of developing T2DM. [1,2,3,4] The diabetes problem in India is being driven mainly by fast urbanization, a shift to desk-based and sedentary jobs and hobbies, a change in diet towards foods that are high in calories and processed, increasing rates of obesity, and a genetic background that makes people from South Asia particularly vulnerable to insulin resistance even at lower weights compared to people in the West. [1,4,6,7]. There is also a big difference between regions within India. Northern states like Uttar Pradesh are seeing a rise in diabetes rates, closely connected to changes in lifestyles and access to healthcare. People living in rural and semi-urban areas — once thought to be at lower risk — are now also being affected as urbanization spreads beyond big cities. Alarming, many cases in these areas are not diagnosed or are only found after complications have already developed, showing a serious lack of screening and early diagnosis.[2,4].

3. RISK FACTORS:

The development of T2DM is influenced by many factors, both things that can be changed and things that cannot. Recognizing these factors is important for identifying people who are at risk and for creating effective prevention plans. [6,7,8]

3.1 NON-MODIFIABLE RISK FACTORS:

Age is one of the strongest risks for T2DM, with the risk increasing sharply after the age of 40. A family history of diabetes greatly raises the chance of developing the disease, indicating both shared genes and shared living conditions. People from South Asian backgrounds have a genetic tendency towards weight gain around the middle of the body, the accumulation of fat in internal organs, and early loss of function in insulin-producing cells, even with only slight increases in weight. [6,7,8]

3.2 MODIFIABLE RISK FACTORS:

Obesity, particularly the kind where fat is concentrated around the waist and the waist-to-hip ratio is high, is one of the strongest risk factors that can be changed. Excess body fat, especially internal fat, leads to insulin resistance through several ways, including inflammation, abnormal fat levels, and changes in signals from fat cells. Using the WHO Asia-Pacific criteria for body mass index (BMI) is a useful tool for identifying risk in healthcare settings. [6,7] Not being active is also linked to an increased risk of T2DM and poor blood sugar control in those who already have the condition. Regular physical activity helps the body use insulin better, manages weight, and has direct positive effects on blood sugar levels. On the other hand, a sedentary lifestyle — which is common in both urban and rural areas — speeds up the progression of the disease. [6,8] Diets high in refined carbohydrates, fats, and processed foods, along with low fibre intake, lead to obesity, insulin resistance, and abnormal fat levels. Smoking and excessive alcohol use are other lifestyle factors that are linked to poor blood sugar control and higher risk of heart and blood vessel problems in people with diabetes. [6,8] High blood pressure and abnormal fat levels are often found together with T2DM as part of a condition called metabolic syndrome, and each increases the risk of heart and blood vessel problems. Because of this, these issues need to be managed together, not just focus on controlling high blood sugar alone. [5,6,8]

4. COMPLICATIONS OF T2DM:

Over time, T2DM can cause serious health problems that fall into two main categories: microvascular and macrovascular. These complications are major causes of health issues, reduced quality of life, early death, and growing healthcare costs. [1,4,5,8]

4.1 MICROVASCULAR COMPLICATIONS:

Diabetic retinopathy is the main reason for preventable blindness among working-age adults around the world. Prolonged high blood sugar levels damage the small blood vessels in the retina, leading to gradual loss of vision that could result in total blindness if not treated. Regular eye exams are important for early detection. [1,4,8]. Diabetic nephropathy is the most common cause of kidney failure globally. High blood sugar causes damage to the small blood vessels in the kidneys, leading to increasing leakage of protein into the urine, a drop in kidney function, and eventually, the need for dialysis or a transplant. Early detection through testing the ratio of urine albumin to creatinine and maintaining tight control of blood sugar and blood pressure can slow the progression of the disease significantly. [1,4,5]. Diabetic peripheral neuropathy affects the nerves in the feet and legs, causing pain, tingling, or numbness. It can also lead to serious issues like foot ulcers and infections, which may result in amputation. Proper foot care, regular check-ups, and managing blood sugar levels are important for preventing these complications. [1,4,5]

4.2 MACROVASCULAR COMPLICATIONS:

Macrovascular complications include heart attacks, strokes, and peripheral artery disease. These are caused by damage to the larger blood vessels, often linked to high blood pressure, high cholesterol, and insulin resistance. They are serious and can be life-threatening, making it important to manage these risk factors alongside blood sugar control. [1,4,5].

5. CONCLUSION:

In conclusion, T2DM is a major health challenge that affects millions of people worldwide, with a particularly heavy toll in India. Addressing the risk factors, managing complications, and ensuring proper glycemic control are essential for reducing the burden of the disease. [1,4,5]

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CARDIOVASCULAR TRANSPLANTATION: PAST TO PRESENT

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ABSTRACT

BACKGROUND:

Imagine a period when it was purely speculative to replace a human heart. With rudimentary instruments, inadequate preservation techniques, and almost little prospect of long-term survival, heart transplantation was a last-ditch effort in the 1960s. A failing heart may now be removed, maintained beating outside the body by a machine, and replaced with one that is compatible with artificial intelligence.

OBJECTIVES:

This review captures the dramatic journey of cardiovascular transplantation — from early failures to groundbreaking successes — and asks: how did we get here, and where are we headed next? It aims to show how technology, science, and human determination turned a dream into a clinical reality.

METHODS:

We contrast today's extremely sophisticated systems with early transplant techniques, which depended on simple surgical instruments and no immunosuppressants. These include robotic-assisted surgery, AI-powered donor matching, ex vivo perfusion devices that maintain heart function outside the body, and customized immunosuppressive treatment.

RESULTS:

The median survival for patients has increased from a few days to more than 12 years. Success rates after a year currently surpass 85%. Patients resume their full lives, jobs, and sports. The once-failing technology now saves thousands of lives annually.

CONCLUSION:

Looking back makes it clear why we must keep moving forward. Despite incredible progress, donor shortages and chronic rejection remain. This review is a call to continue innovating — because every heartbeat counts. By understanding the path we've taken, we can better shape the future — one where no life is lost waiting for a new heart.

INTRODUCTION:

End-stage heart failure is the last common pathway of several cardiovascular illnesses, including coronary artery disease, cardiomyopathies, and valvular abnormalities. One of the leading causes of death globally is still heart disease. Even while medication and device-based treatments have greatly enhanced patient outcomes, a large number of people eventually reach a point when traditional treatments are no longer successful. Heart transplantation is the only realistic way to guarantee these people's long-term survival and improved quality of life. The rate of retransplants has remained relatively stable throughout time, accounting for 2–4% of all adult heart transplants. More than 40% of retransplants occur ten years or more after the first orthotopic heart transplant procedure, according to the median survival of transplant recipients. The concept of replacing a broken human heart with a donor organ was previously limited to science fiction. When Dr. Christiaan Barnard successfully conducted the first mortal-to-mortal heart transplant in Cape Town, South Africa, on December 3, 1967, this was drastically changed. Despite the recipient's brief 18-day survival, the treatment signaled the start of a groundbreaking medical era. Heart transplantation is a proven therapy option today, with one-year survival rates above 85% and continuous research to enhance long-term results. Despite these developments, the industry still faces difficult problems such as chronic rejection, donor organ shortages, and moral dilemmas relating to recipient selection and organ distribution equity.

HISTORICAL MILESTONES:

Experiments on dogs and primates marked the beginning of the field of heart transplantation. Among the many pivotal moments in the development of the area were the introduction of the biptome for endomyocardial biopsy, immunosuppressive therapies, and, more recently, the application of cell free DNA and molecular microscopy in the assessment of rejection. The availability of donors has significantly increased because to recent advancements in organ preservation and transport technologies like Paragonix and Transmedics OCS.

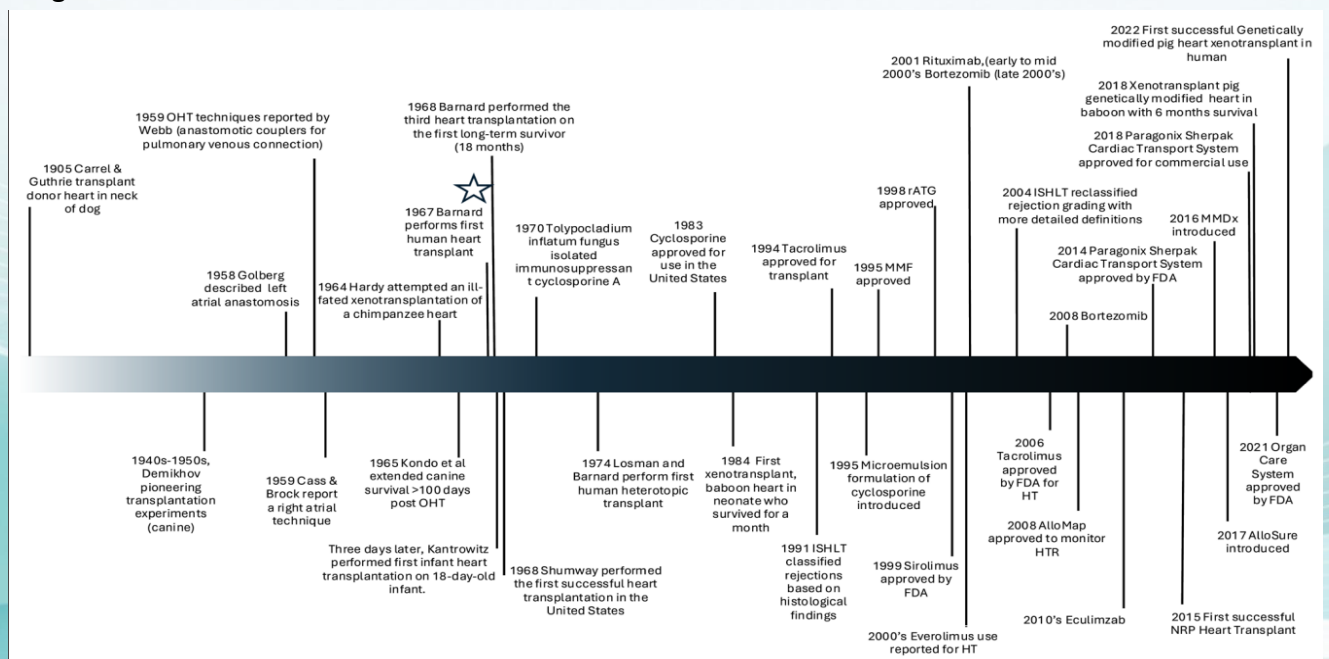


Figure-1

The remarkable survival rates observed today are the result of decades of advancements in surgical techniques, donor-recipient matching, organ preservation techniques, and immunosuppressive regimes. The history of heart transplantation shows both scientific tenacity and ethical development, progressing from a medical curiosity in the 1960s to a widely recognized standard of treatment.

ADVANCES IN SURGICAL AND MEDICAL PRACTICES:

The field of heart transplantation has seen tremendous advancements in surgical methods and medical care throughout the years, which have greatly boosted transplant recipients' success rates and longevity. To comprehend the transplant process, one must have a solid understanding of the heart and the main veins in the mediastinum. Two categories of transplant operations have been documented in the past:

(i) **ORTHOTOPIC HEART TRANSPLANTATION**, in which the recipient's heart is removed and a donor's heart is implanted in the recipient's chest.

(ii) **HETEROTROPIC HEART TRANSPLANTATION**, in which the donor's heart is placed into the recipient's chest without the recipient's heart being removed.

Improvements in anastomotic methods, cardiopulmonary bypass technology, and myocardial protection approaches have all contributed to a decrease in ischemia time and intraoperative complications. Additionally, although they are not currently common procedures in heart transplantation, the development of robotic-assisted and minimally invasive surgeries is being investigated to improve recovery time and surgical trauma. It's crucial to keep the donor heart intact between harvest and implantation. First, simple hypothermic storage in cold cardioplegic solutions was used to preserve the hearts. The development of ex vivo perfusion devices, such as the TransMedics Organ Care System, has improved organ assessment before implantation and prolonged viable ischemia time by enabling continuous perfusion and oxygenation. The field of immunosuppression has seen some of the most revolutionary developments in medicine. Early treatments included azathioprine and high-dose corticosteroids, which had serious adverse effects and little effectiveness. Cyclosporine, which was first introduced in the 1980s, became the mainstay of contemporary immunosuppressive procedures and dramatically decreased acute rejection. Current routines usually consist of: (i) Inhibitors of calcineurin: tacrolimus and cyclosporine (ii) Antimetabolites include azathioprine and mycophenolate mofetil (iii) Prednisone is a corticosteroid. (iv) Sirolimus and Everolimus are mTOR inhibitors that are used specifically to lower the risk of cancer and nephrotoxicity.

ISSUES AND CURRENT PRACTICES SURVIVAL STATISTICS:

When compared to the natural course of end-stage heart failure, survival rates after heart transplantation have improved significantly. Over the last few decades, steady improvements in surgical methods, care during and after surgery, and immunosuppressive therapy have greatly improved both short-term and long-term patient survival. Improvements in personalized immunosuppressive strategies have made it easier to control graft rejection while reducing the side effects of treatment. Also, new diagnostic and monitoring methods have made post-transplant surveillance and early detection of rejection even better. Endomyocardial biopsy and new molecular assessment methods have made it easier for doctors to make decisions and manage grafts over the long term. All of these improvements

have led to long-term improvements in transplant outcomes and have made heart transplantation a good treatment option for people with advanced heart failure.

ONGOING CHALLENGES AND PATRON DEARTHS :

One of the biggest problems with heart transplantation programs around the world is that there aren't enough donor hearts. In response to this challenge, transplant centers have gradually investigated the broadening of donor selection criteria, encompassing the inclusion of older donors under meticulously assessed clinical circumstances. Clinical evidence indicates that suitably chosen hearts from older donors may yield satisfactory early and long-term transplant outcomes, particularly when enhanced perioperative management and monitoring strategies are implemented. Improvements in donor evaluation, preservation methods, and postoperative monitoring have allowed doctors to use extended-criteria donors without greatly affecting the performance of the graft. While specific age-related risks, including vascular alterations or conduction irregularities, may necessitate more rigorous monitoring, the overall success of transplantation has progressively relied on thorough donor assessment rather than age alone. As a result, carefully raising the age limits for donors has become a useful way to partly solve the problem of not having enough organs. Long-term evaluation and improvement of recipient selection and postoperative care are still very important to make sure that the graft works well and the patient has the best possible outcome.

UNBORN PERSPECTIVE: XENOTRANSPLANTATION:

Xenotransplantation, or the transplanting of organs from nonhuman species, has been hailed as one of the most ambitious and progressive solutions in transplant biology given the increasing global burden of end-stage heart failure and shortage of organ donor hearts. Characteristics of an ideal xenogenic patron heart include similar physiology, scalability for a broad range of application and immunological compatibility. From a phylogenic perspective, NHP are the preferred donor species for xenotransplantation. yet this poses various ethical and practical dilemmas. NHP necessitates deep coffers and more extensive parentage builds. They mutate slowly, mature slowly, and keep producing tiny organs. Similarly, however innocent and immoral is to capture and breed NHP in order to harvest their organs. similarly, another major concern is the spread of diseases across related species. yet, unlike NHPs, breeding gormandizers is swifter, cheaper and poses fewer moral quandaries as their hearts are essentially identical to those of humans. These variables make gormandizers the most pragmatically utilitarian patron species for mortal xenotransplantation. Recent advances in gene- editing technologies similar as CRISPR/ Cas9 have allowed for the successful development of gormandizers that are genetically modified with hearts marrow unit more likely to be immune rejected by the mortal vulnerable system. Changes include elimination of hyperacute rejection through α -1,3-galactosyltransferase (GGTA1) knockout mortal complementary non-supervisory genes similar to as CD46 are introduced. To help interspecies infection, porcine endogenous retroviruses (PERVs) are rendered inactive. The successful transplantation of a genetically altered gormandizer heart into a mortal philanthropist at the University of Maryland in January 2022 was a major occasion. Despite the case's brief two- month survival, the fashion was clinically doable and represented a significant advancement in transplant remedy. Ongoing clinical studies and preclinical exploration are advancing xenotransplant practices. Xenotransplantation

could become a regular option for allograft transplantation by joining forces with bioprinting, stem cell engineering, and immune tolerance methods. If it works well, it might change how we treat heart issues and solve the problem of organ failure.

CONCLUSION:

From a daring experimental operation, heart transplantation has developed into a proven and life-saving treatment for individuals suffering from end-stage heart failure. The first successful transplant in 1967, the advent of potent immunosuppressants like cyclosporine, and the advancement of organ preservation technologies are just a few of the major turning points that have changed the course of history and broadened the range of what is clinically feasible. The industry now boasts impressive short- and mid-term survival rates, with many patients reporting notable improvements in their quality of life. However, there is still more work to be done. Long-standing problems like donor organ shortages, chronic rejection, long-term effects, and socioeconomic inequalities continue to limit the full potential of transplantation as a globally accessible choice. The issues of immunological rejection and donor dependency may be resolved in the future through innovative methods including stem cell therapy, bioartificial hearts, xenotransplantation, and customized immunomodulation. These innovative technologies, however still in their early stages, have the potential to revolutionize the treatment of heart failure and push the boundaries of cardiovascular medicine. Heart transplantation is still a clinical success story and a representation of interdisciplinary advancement as science, ethics, and policy continue to advance together. To guarantee that every patient in need can benefit from this amazing therapy, more funding must be allocated to research, innovation, and a fair healthcare system.

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RELIGIOUS-INSTITUTION-CENTRED APPROACHES TO DENTAL CARIES PREVENTION AND ORAL HYGIENE PROMOTION IN INDIA: A NARRATIVE REVIEW OF EFFECTIVENESS AND ECONOMIC POTENTIAL

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ABSTRACT

Dental caries is among India's most prevalent yet most neglected diseases, sustained by low public investment, fragmented prevention, and socioeconomic inequity. Places of worship — masjids, mandirs and gurudwaras — reach almost the whole population repeatedly, carry high trust, and already host government health campaigns, making them a plausible but under-tested platform for caries prevention. This narrative review synthesises the limited direct evidence — a US Gurdwara-based oral-health program, a Bangladeshi mosque-based behavioural trial, and Indian data on miswak use — alongside analogues from faith-based promotion of other conditions and from school-based oral health. Faith-centred delivery appears feasible and culturally acceptable and can plausibly improve oral-hygiene knowledge and behaviour, but caries-outcome data specific to these settings are almost absent. Against an oral-disease burden that is large in aggregate and increasingly catastrophic for Indian households, low-cost preventive gains delivered through religious institutions could be cost-effective — though the magnitude of any economic “resolution” remains an extrapolation, not a measured quantity.

1. INTRODUCTION:

Dental caries is among the most widespread chronic diseases in India, and it is largely preventable, yet it persists as a quiet public-health failure. Community surveys illustrate the scale: in a population-based survey in Ballabgarh, Haryana, caries prevalence was 33.2% in 5–7-year-olds, 31.3% in 12–15-year-olds, 64.9% in 35–44-year-olds and 50.1% in 65–74-year-olds.¹ India's preventive and treatment infrastructure has not kept pace with this burden. Per-capita expenditure on dental disease is among the lowest in the world — an average Indian spent only about US\$0.14 on dental disease in 2015 — meaning most disease goes untreated rather than treated cheaply.² The structural problem is well recognised. India trains a large dental workforce but concentrates it in private, urban, treatment-oriented practice, while population-level prevention is thin and there is no organised national oral-health programme delivering preventive care at scale.^{2,3} Reviews of the Indian situation repeatedly conclude that the country must shift emphasis from chairside restoration toward population-based prevention, because the cost of restoring even children's caries would exceed available public budgets.^{2,3} This sets up the central question of this review: if conventional health-system channels are insufficient, can existing community institutions — specifically religious congregations — be harnessed as preventive

platforms? India is among the most religiously observant societies in the world, and masjids, mandirs and gurudwaras function as recurring, trusted, high-footfall community hubs; this review asks whether oral-health promotion delivered through them can meaningfully improve oral hygiene and reduce caries, and what share of the economic burden such an approach might realistically address.

2. THE RATIONALE FOR RELIGIOUS-INSTITUTION-CENTRED HEALTH PROMOTION:

Faith-based organisations (FBOs) are not marginal to health delivery in low- and middle-income countries; they are part of the infrastructure. A systematic review and meta-analysis of FBO participation in health-service delivery in developing countries found measurable contributions to outpatient care, highest in Sub-Saharan Africa (around 6.8% of outpatient care sought at religiously affiliated facilities) and lower but non-trivial in South Asia and South-East Asia (around 1%).⁴ Beyond service provision, religious leaders function as trusted messengers whose endorsement can shift behaviour on a communal scale — a mechanism repeatedly invoked in the health-promotion literature on imams and mosques.⁵ The behaviour-change evidence for faith-based promotion is most mature for non-communicable diseases. A synthesis cited in Caribbean work on church-based programmes reported that such programmes were effective in roughly two-thirds of weight-reduction and blood-pressure-control studies and about half of physical-activity and diet studies⁶ — a signal that congregational settings can move risk-factor behaviours, the same terrain as toothbrushing, dietary sugar and dental visits. Qualitative work in Muslim communities similarly finds broad community support for integrating faith-based elements into health interventions, while flagging gendered access and organisational dynamics as design constraints.⁷ Critically, India itself already uses places of worship as a public-health platform. Under the *Jan Bhagidari* approach to the TB-free India campaign, the Ministry of Health and Family Welfare convened religious organisations and faith-based organisations to run awareness activities in temples, churches, gurudwaras and mosques, coupling them with anti-spitting and cleanliness messaging.⁸ This establishes both precedent and operational machinery: the question for oral health is not whether faith institutions *can* be mobilised, but whether oral-hygiene content delivered through them changes outcomes.

3. DIRECT EVIDENCE: MASJID-CENTRED APPROACHES:

The most directly relevant interventional evidence comes from a mosque-based oral-health study in Bangladesh using a Solomon four-group design.⁹ The intervention embedded oral-health education within the Friday *khutba* (sermon): a 15-minute sermon during the first Friday congregation, a reinforcement sermon and educational leaflets the following week, distribution of a miswak after clinical assessment and a toothbrush at reinforcement, with clinical and questionnaire follow-up across three consecutive Fridays.⁹ The design is notable because it deliberately leverages the existing rhythm and authority structure of the mosque — the sermon as the delivery vehicle, the imam as the messenger, and the weekly congregation as the cohort — rather than importing an external clinic model. It also illustrates a recurring feature of masjid-based oral health: the natural fit with the Islamic tradition of the miswak, which functions simultaneously as a culturally endorsed and a clinically active oral-hygiene tool (Section 5). This single study cannot establish caries-outcome effectiveness; its follow-up is short and its outcomes are plaque and behaviour rather than longitudinal caries increment. But it demonstrates the

feasibility of a fully mosque-embedded protocol and offers a replicable template for the Indian context, where Friday congregational attendance is high and predictable.

4. DIRECT EVIDENCE: GURUDWARA- AND MANDIR-CENTRED APPROACHES:

For gurdwaras, the clearest evidence is the *Sikh American Families Oral Health Promotion Program*, implemented across five to six gurdwaras in New York and New Jersey.¹⁰ Although set in the diaspora, it is in effect a worked example of gurdwara-centred oral-health delivery for a Sikh population. Using a community-based participatory research approach and the Consolidated Framework for Implementation Research, the program trained Sikh community members from each gurdwara as community educators who delivered oral-health workshops on site, with hands-on brushing and flossing instruction and culturally tailored dietary guidance (including the *langar*/healthy-meal context).¹⁰ The gurdwara was treated as the “inner setting” for implementation, and the program aimed to build links between congregations and local oral-health providers to improve access.¹⁰ The model translates naturally to Indian gurdwaras, which combine worship, community kitchen (*langar*) and *seva* (service) traditions that align well with health-promotion delivery. For mandirs, the picture is the most sparse: there is, to this author’s knowledge from the available literature, no published controlled evaluation of a temple-centred dental-caries prevention or oral-hygiene programme. Temples are demonstrably usable as health-awareness venues — they featured in the national TB faith-based campaign⁸ — and Hindu institutions host a wide range of community welfare and camp activities, but the specific evidence base for mandir-delivered oral-health outcomes is effectively empty. This is the single largest gap in the field and is discussed further in Section 8. The honest position is that mandir-centred oral health is a plausible, untested extension of a model that has only preliminary support in masjid and gurdwara settings.

5. CULTURAL AND BEHAVIOURAL ASSETS THAT STRENGTHEN FAITH-BASED DELIVERY:

A distinctive advantage of faith-centred oral health in the South Asian context is that several culturally embedded practices are themselves evidence-supported oral-hygiene behaviours, narrowing the gap between religious message and clinical recommendation. The miswak (*Salvadora persica* chewing stick), associated with Islamic practice for over a millennium, is the clearest example. A PRISMA-compliant systematic review and meta-analysis concluded that the miswak controls plaque comparably to a standard toothbrush and has a better anti-gingivitis effect, supporting its use as an effective alternative or adjunct.¹¹ Mechanistic work indicates that much of its benefit derives from mechanical cleansing action, with additional contributions from active constituents.¹¹ Importantly for India, real-world use has been documented domestically: a cross-sectional study among Muslim schoolchildren in Lucknow examined the miswak as a traditional oral-hygiene aid, situating this practice within the Indian population rather than only the Gulf or African contexts.¹² A masjid-based programme can therefore reinforce an already-present, low-cost, clinically valid behaviour rather than introducing an unfamiliar product — and adjunctive use of miswak alongside toothbrushing appears to improve oral-hygiene and gingival outcomes.¹¹ Comparable anchors exist across traditions: gurdwara *seva* and *langar* offer a structured route to dietary and hygiene messaging, as the Sikh program demonstrated,¹⁰ and Hindu temple welfare activity provides an analogous, if untested, channel. Faith-based oral health is most

plausible where the tradition already carries a hygiene or dietary practice that the programme can validate and amplify.

6. EXTRAPOLATING FROM SCHOOL-BASED AND COMMUNITY ORAL-HEALTH PROGRAMMES:

Because direct faith-based caries-outcome data are limited, the strongest quantitative evidence for what community-delivered oral health can achieve in India comes from the school setting, which shares the key features of a captive, trusted, repeatedly accessible population. School-based oral-health education in India produces measurable improvement. A quasi-experimental study in Lucknow reported a significant reduction in mean DMFT in the intervention group from 3.20 to 2.10 and in gingival index from 1.80 to 1.20 over 12 months.¹³ A systematic review of Indian school-based programmes found that oral-health education delivered by trained schoolteachers was as effective as that delivered by qualified dentists for improving plaque, gingival and caries-related outcomes, although results were not uniform across all studies.¹⁴ Curriculum-based interventions tailored to age have likewise shown significant reductions in decayed primary teeth among Indian schoolchildren,¹⁵ and cluster-randomised work in Kerala has been designed specifically to test caries-increment reduction from an embedded oral-health curriculum.¹⁶ Historically, the foundational developing-country oral-health models — such as Sri Lanka's self-reliance programme — explicitly incorporated teachers, family health workers, traditional practitioners and religious leaders as front-line educators,¹⁷ which is precisely the lay-deliverer logic that faith-based programmes depend on. Two inferences follow. First, the active ingredient in these successes is not the clinician but trusted lay deliverers, repetition, and a captive audience — all of which religious congregations also supply. Second, schools reach children but not the caregivers of the under-six group, where early childhood caries concentrates and behaviour is set; faith institutions reach whole families, making them complementary to schools rather than redundant. This is the most defensible argument for the approach: not that faith-based delivery is proven superior, but that it plausibly extends a demonstrably effective community-education model to populations and age groups schools miss.

7. ECONOMIC BURDEN AND THE POTENTIAL FOR FAITH-BASED PREVENTION TO REDUCE IT:

The aggregate economic burden of oral disease is large. Globally, the economic impact of oral conditions in 2019 was estimated at US\$710 billion, comprising roughly US\$387 billion in direct treatment costs and US\$323 billion in productivity losses across the five main oral conditions.¹⁸ Dental caries alone accounts for a large share of global oral-disease cost.¹⁹ The same global analysis exposes the core inequity: low-income countries spent on average about US\$0.52 per capita on dental care versus about US\$260 in high-income countries — a roughly 500-fold difference.¹⁸ India sits at the low end, with per-capita dental expenditure around US\$0.14 and an estimated per-capita productivity loss of about US\$1.8 (≈₹117) in 2015.² The burden in India is not only large in aggregate but increasingly catastrophic at the household level. A 2026 systematic review and meta-analysis of the economic burden of oral diseases in India found high out-of-pocket expenditure, a pooled prevalence of catastrophic dental-health expenditure (≥20% of household income) of about 18.8%, insurance coverage below 15%, and up to one-third of patients resorting to distress financing such as borrowing or selling assets to pay for dental care.²⁰ In other words, untreated caries does not stay cheap: it converts into expensive, sometimes impoverishing, treatment episodes downstream, especially for the poor. This cost structure is exactly

the kind that prevention is best placed to attack, because the marginal cost of education-based prevention is low and the averted cost of advanced treatment is high. Faith-based delivery is attractive economically for three reasons: it uses existing physical infrastructure (no clinic to build), it relies on trained lay educators rather than scarce dentists,^{10,14,17} and it can reinforce zero- or low-cost behaviours such as miswak use and reduced sugar intake.¹¹ Where it can shift caregivers of young children toward better hygiene and dietary behaviour, it acts at the point in the disease pathway before catastrophic costs accrue. How much of the burden could it resolve? Intellectual honesty is essential here: no study quantifies the economic effect of *faith-based* oral-health programmes specifically, so any figure is an extrapolation, not a finding. An illustrative argument can be made — school-based education reduces DMFT by roughly a third over a year in Indian settings,¹³ caries is a large fraction of oral-disease cost,¹⁹ and the costliest, most impoverishing care is the untreated-caries-to-extraction pathway²⁰ — so a preventive platform that even partially shifts behaviour at scale could avert a meaningful share of avoidable treatment cost and productivity loss. But the responsible claim is bounded: the value of faith-based oral health lies in its low cost, wide reach and access to populations the formal system misses; its likely return is best framed as high cost-effectiveness on avoidable disease, not a precise percentage of the national burden “resolved.” An actual rupee figure would require costed, outcome-measured trials that do not yet exist.

8. LIMITATIONS AND THE EVIDENCE GAP:

The principal limitation of this field is the near-absence of direct, caries-outcome evidence from religious settings, particularly in India. The masjid evidence rests largely on a single short-follow-up behavioural trial in Bangladesh;⁹ the gurudwara evidence rests on one diaspora implementation program with process and acceptability outcomes rather than caries-increment data;¹⁰ and the mandir evidence is effectively non-existent. Most supporting evidence is therefore analogical — drawn from faith-based promotion of other conditions^{4–8} and from school-based oral health^{13–17} — which establishes plausibility but not proof for caries endpoints. Several design challenges are specific to faith-based delivery and must be acknowledged. Access can be gendered, particularly in mosque settings where congregational attendance skews male, requiring deliberate strategies to reach women and young children.⁷ Dependence on religious leaders means programme quality and reach vary with individual leaders' engagement.⁵ Sustainability beyond grant funding, fidelity of lay-delivered content, and the difficulty of measuring caries increment (which requires long follow-up) all constrain the evidence, and a model that works in one tradition or diaspora context may not transfer unchanged. Finally, faith-based programmes should be positioned as complements to — not substitutes for — fluoride exposure, school programmes, and access to affordable restorative care, since education alone does not address the structural under-supply of dental services.^{2,3}

9. CONCLUSION:

Religious institutions in India — masjids, mandirs and gurudwaras — offer a trusted, recurring, low-cost and near-universal platform for oral-health promotion, and India has already mobilised them for national health campaigns.⁸ Yet the direct evidence that such platforms reduce dental caries is genuinely thin: feasibility is reasonably supported in mosque⁹ and gurudwara¹⁰ settings, several embedded

practices such as miswak use are clinically valid,^{11,12} and the broader logic rests on the proven effectiveness of community- and school-based education by trusted lay educators.¹³⁻¹⁷ Against a substantial and increasingly impoverishing oral-disease burden,¹⁸⁻²⁰ a faith-centred model is best understood as a high-cost-effectiveness, population-reaching complement to existing strategies, not a proven or quantifiable solution. The decisive next step is not advocacy but costed, outcome-measured trials — ideally cluster-randomised, with caries-increment and economic endpoints — embedded in masjids, mandirs and gurudwaras in India, so the model's effectiveness and true economic return can be established rather than inferred.

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IMPLEMENTATION OF FLIPPED CLASSROOM IN BASIC SCIENCES: ATTITUDES AND ITS EFFECTS ON STUDENTS' PERFORMANCE AND LEARNING.

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ABSTRACT

BACKGROUND: The Flipped Classroom is a teaching - learning method where classroom activities and homework switch places. Students dive into foundational concepts outside the classroom, often through digital resources like videos, podcast, interactive modules etc. Subsequently the 'in-class' time is used for applying that knowledge, tackling complex problems, and engaging in collaborative activities. The aim of our study was to implement the flipped classroom method of teaching for 1st year students of Batch 2021-22 batch.

METHODS: The study was conducted on 125 first year MBBS students in the Department of Anatomy. After didactic lectures - understanding and retention of knowledge was assessed by 10 clinical scenario based (DL1) MCQs. Recall of knowledge was assessed by clinical scenario based MCQ (10) via Google forms, 2 weeks after first assessment (DL2). Understanding and retention of knowledge was assessed by 10 clinical scenario based MCQ after Didactic Lecture (FC1). Recall of knowledge was assessed, 2 weeks after first assessment by 10 scenario based MCQ (FC2). In addition, student and faculty perception was taken about the flipped classroom approach. Total 6 such sessions were conducted.

RESULTS: The performance scores of the students were much better after the 'flipped classroom' sessions in comparison to didactic lectures. Knowledge retention and recall after 2 weeks was also better than the conventional didactic lecture method. Most of the faculty members had a positive opinion about the implementation of the 'Flipped Classroom' approach.

CONCLUSION: The students found the shared material before the class to be adequate, relevant and engaging. Their involvement in pre-class preparation was quite deep. The collaborative work in problem solving and opportunity to interact with teacher on one-to-one basis proved to be the winner. After attending 6 such sessions over a period of 6 months, majority of students expressed a change in attitude towards their own learning. They felt the need to go prepared for every class and to be actively engaged in learning activities.

KEYWORDS: Flipped classroom, Anatomy, Teaching-learning method, Active learning, Knowledge Retention, Medical education

INTRODUCTION:

The term “inverted classroom” or “classroom flip” was coined by Lage^[1] and Baker^[2], and this concept has caught imagination of the whole academic world and has been the method of choice for learning across the institutes globally. Here the traditional classrooms are being flipped! The way we learn is undergoing a revolution, moving beyond passive lectures and rote memorization. In this teaching - learning method, classroom activities and homework switch places. Here students dive into foundational concepts outside the classroom, often through digital resources like videos, podcasts, or interactive modules.^[3, 4] Then, the precious ‘in-class’ time transforms into a dynamic space for applying that knowledge, tackling complex problems, and engaging in collaborative activities.^[5] Why the flip? This student-centred approach caters to the needs of our modern learners, born and raised in the digital age. It empowers them to learn at their own pace, rewind and revisit challenging concepts, and leverage their digital prowess. Gone are the days of cell phones banished in the back pocket; here technology becomes a tool for deeper understanding and collaboration. Research supports the flipped classroom’s effectiveness in terms of better learning, conceptual understanding, and analytical skills compared to traditional methods. While the concept is gaining traction globally, it’s still in its early stages in India. This presents an opportunity to explore its potential within the context of Indian medical education and customize it to meet the needs of future healthcare professionals. The western literature is replete with substantial evidence supporting the efficacy of the Flipped Classroom model; however its integration into medical education in India is still in its infancy.^[6] With the recent adoption of Competency Based Medical Education (CBME) curriculum by NMC in the year 2019 the interest in implementing Flipped Classroom approach has gained momentum.^[7] However, the unique characteristics of the Indian medical education system, such as early entry into medical schools and predominantly traditional teaching approach in pre-medical subjects of first year MBBS, warrant a region-specific exploration of the model’s impact.^[8] Keeping this back ground in minds, our study was planned with the following objectives:

1. To assess the effectiveness of flipped classroom in improving the performance of students in comparison to didactic lectures
2. To assess the effectiveness of flipped classroom in knowledge retention.
3. To assess the perception of both faculty and students towards the flipped classroom approach
4. To take an opinion from students about how well the flipped classroom sessions were implemented.

METHODS:

This was an educational interventional study that was conducted in the Department of Anatomy of a Govt. Medical College located in Haryana over a period of 6 months. All 125 students of first year MBBS were a part of this project. Before starting the study written informed consent was taken from all the participants and clearance was taken from the Institutional Ethical Committee.

Sensitization and orientation sessions about Flipped Classroom were conducted for both the students and faculty members of the Department. A core committee was formed, and 6 modules were prepared. The core committee member's peer reviewed the MCQs that were used at the end of each session as well as the questionnaires that were used to take feedback from both the students and faculty members. The sessions were conducted in the last week of teach month over a period of 6 months.

The performance of students was assessed immediately at the end of didactic lecture, which is considered the 'default' method of teaching, by 10 scenario based MCQs. The MCQs were shared with the students with the help of Google forms and were labelled as DL1. Further the retention of knowledge was assessed by students after 2 weeks of the didactic lectures by again scenario based MCQs that were 10 in number and were shared with the students with the help of Google forms. They have been labelled as DL2. Before conducting each session of flipped classroom, relevant study material like pdf of chapters, textbook references, videos, recorded audios, podcasts etc. Were shared with help of social media platforms at-least 2 days prior to the day of flipped classroom session. During the pre-class period the students were interviewed in-person/telephonically to check if they have accessed and read/seen the shared reading material/video link, prior to attending class on flipped classroom. During flipped classroom session that was conducted in small groups, their understanding of the topic was assessed by interactive session with the help of 10 scenario based MCQs that was labelled as (FC1). The retention of knowledge was tested after 2 weeks with the help of case-based MCQs and was labelled as (FC2). The results of DL1, DL2, FC1, and FC2 were shared with the students to give them a feedback on their understanding of the topics covered and to deepen their involvement in learning process. The perception of the 6 faculty members as well as all 125 students was assessed by using two separate questionnaires framed on a Likert scale from 1-5. Feedback was also taken from the students about how well the modules were implemented at the end of 6 sessions of Flipped Classroom.

STASTICAL ANALYSIS:

The collected data was tabulated and analysed by SPSS software (version 21) was used for the analysis of (IBM, SPSS. INC, Chicago, IL). The values of DL1, DL2, FC1 and FC2 has been expressed as mean $\pm$ SD. Comparison of scores was done using unpaired 't-test' where p value < 0.05 was considered to be statistically significant. The feedback from students and faculty has been taken on a Likert scale and has been expressed as percentages.

Results

The study was conducted to assess the effectiveness of implementation of the 'Flipped classroom' approach in comparison to didactic lectures in the Department of Anatomy in a Govt. Medical College of Haryana. The mean scores achieved after DL1, FC1, DL2 and FC2 can be seen in **Table 1**.

Table 1: Mean and SD of average scores after DL1, FC1, DL2 and FC2

Sessions	DL1	FC1	DL2	FC2
A	7.02 ± 2.84	8.57 ± 3.0 3	5.36 ± 2.4 6	7.96 ± 2.2 6
B	7.31 ± 2.9 5	8.64 ± 3.0 5	5.17 ± 2.3 7	7.64 ± 2.1 6

C	6.87±2.7 7	8.03±2.7 8	5.63±2.5 8	6.93±1.9 6
D	7.53±3.0 4	7.97±3.2 1	4.97±2.2 8	7.09±2.0 1
E	6.91±2.7 9	8.39±3.0 1	5.48±2.5 1	7.74±2.1 9
F	6.43±2.6 0	8.01±2.9 7	5.93±2.7 2	6.92±1.9 8
Average	7.01±2.8 3	8.26±3.0 0	5.42±2.4 8	7.38±2.0 9

DL1 – Didactic Lecture 1, FC1 – Flipped Class, DL2 – Didactic Lecture (after 2weeks), FC2 – Flipped Class (after 2 weeks).

Table 2: Comparison of scores after DL1 & FC1

Interventi on	Mean	SD	SEM	P value
DL1	7.01	2.83	0.2531	0.0008
FC1	8.26	3,00	0.2683	

On comparing the mean scores of students taught through these two different approaches, the unpaired 't-test' yielded a 'p value' of 0.0008 implying the difference being statistically highly significant showing that the test scores after Flipped classroom approach are significantly higher in comparison to scores after didactic lectures.(Table 2)

Table 3: Comparison of scores after DL2 and FC2

Interventi on	Mean	SD	SEM	P value
DL2	5.42	2.48	0.2218	0.0001
FC2	7.38	2.09	0.1869	

Comparison of scores obtained after DL2 and FC2 can be seen in Table 3. Unpaired 't-test' yielded a 'p value' of 0.0001 implying the difference being statistically highly significant. This implies that the test scores for knowledge retention after 2 weeks are better and statistically highly significant after implementation of Flipped Classroom in comparison to Didactic Lecture.

QUALITATIVE PARAMETERS:

Students' perception about the Flipped classroom can be seen in Table 4. Majority of the students gave a positive feedback about benefits and efficacy of Flipped Classroom sessions, Some of the students felt that this technique was time consuming and put too much pressure on them, adding to their existing burden of an already extensive curriculum.

Table 4: Students' perception and feedback on 'FC' on a Likert scale (in %)

Description of question	1	2	3	4	5
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Provided study material was useful	0	0	6 . 1	4 5 . 5	4 8 . 5
FC was engaging and improved my motivation to learn	0	0	0	5 7 . 6	4 2 . 4
FC gave me opportunity to communicate with the teacher	0	0	3	4 5 . 5	5 1 . 5
I was able to keep pace with the class	0	0	1 2 . 1	5 4 . 5	3 3 . 3
FC improved my understanding of the concept	0	0	3	5 1 . 5	4 5 . 5
I would like to have more FC sessions in the future	0	9 . 1	9 . 1	3 3 . 3	4 8 . 5
I am satisfied with the course content covered in FC	0	3	6 . 1	4 8 . 5	4 2 . 4
FC was time consuming	1 5 . 2	2 7 . 3	3 3 . 3	1 8 . 2	6 . 1
FC gave me too much burden and pressure	3 6 . 4	3 0 . 3	1 5 . 2	1 2 . 1	6 . 1
I learnt more and better in flipped class	0	0	3	5 4 . 5	4 2 . 4
I think FC guided me to be more responsible and motivated for learning	0	0	0	4 5 . 5	5 4 . 5

LIKERT SCALE - 1 strongly disagree, 2 Disagree, 3 Neutral, 4 Agree, 5 strongly agree.

Perception of the faculty about the flipped classroom technique can be seen in Table 5. The faculty felt that the practice of flipped classroom was richly rewarding as the students' attention, engagement, enthusiasm, and knowledge retention improved significantly. They also opined that 'FC' sessions should be done intermittently as these are not suitable for all the topics to be covered in Anatomy.

Table 5: Faculty perception and feedback on FC on Likert scale (in %)

Description of questions	1	2	3	4	5
FC has resulted in better understanding of subject in students	0	0	0	50	50
FC encouraged active participation of students in learning the subject of anatomy	0	0	0	50	50
The response of the students to the FC was enthusiastic	0	0	0	0	100
FC Improved student teacher interaction	0	0	0	60	40
FC improved students' knowledge retention	0	0	0	60	40
FC can be incorporated in routine teaching	0	0	20	40	40
FC needs more manpower	0	0	0	50	50
FC is burden to the faculty	40	40	20	0	0
FC is time consuming	0	20	40	20	0
FC is not feasible for all topics	0	0	10	50	40
FC sessions encourage self-directed learning in students	0	0	0	20	80

LIKERT SCALE - 1 strongly disagree, 2 Disagree, 3 Neutral, 4 Agree, 5 strongly agree.

Students' feedback for Learning Practice

Table 6 represents the student feedback on a Likert scale (expressed in percentages) regarding the implementation of Flipped Classroom (FC) practices, that was taken after implementation of all 6 sessions of 'Flipped Classroom'. Majority of the students (>85%) experienced the change in their awareness, preparedness, and scores with the practice of FC. They also felt more responsible for their learning – a step towards becoming self-directed learners.

Table 6: Feedback of students regarding implementation of Flipped Classroom (in %)

Statement	5	4	3	2	1
I think other subjects should also include flipped classroom sessions	51	39	10	0	0

My scores have improved due to better preparation	41	4 4	1 5	0	0
I feel motivated to be prepared for every class	21	6 1	1 8	0	0
I feel more responsible for my learning	21	6 1	1 8	0	0
I read the topic before going to the class	31	4 1	2 8	0	0
I am more aware of my role as student	51	3 9	1 0	0	0
I feel the need to prepare for every class	30	4 5	2 5	0	0

LIKERT SCALE - 5 - strongly agrees, 4 -Agree, 3- Neutral, 2-Disagree, 1-Strongly agree

DISCUSSION:

Our study shows that the Flipped Classroom approach had a positive impact on the performance of students in comparison to didactic lectures in Anatomy. Students and faculty members who participated in this study had a positive attitude towards the implementation of this teaching - learning method.

A lot has been said about the practice of blended learning and many new interventional strategies are being planned and executed. The positives of Flipped Classroom clearly are better engagement of the students with learning at their own pace, access to the study materials all the time and deep learning to name a few. ^[9] In implementation of FC many of the previous researchers ^[10-13] have found it to be an effective educational intervention. They divided the participants into experimental and control groups and conducted pre and post-tests as a tool to collect and analyse data. In the present study we have included all the participants in the interventional fold, giving every student equal opportunity and exposure to this interventional method. The perceptions recorded by all of them stated increased learning engagement of students. Some studies ^[14], did not find any statistically significant difference in the performance of the students with and without flipped classroom, probably due to differences in interest level, preparedness, and motivation of students. The student's feedback and perceptions for FC practice demonstrated increased engagement, active consumption of shared resource materials, pre-class preparation, better topic understanding, and deep learning resonating with many of the previous studies. ^[10-12, 15-19] Faculty perceptions of FC confirmed better student teacher interaction, knowledge retention and active participation. The preferences of the study material shared with the students and their effectiveness vary, along with the time required for preparation of classes in this flipped classroom approach. ^[20] As per Ming Ji et al ^[13], the short-term and long-term learning outcomes for the students exposed to flipped classroom practice fare much better than the traditional didactic method, which is in total conformity with the results of our study. A change in attitude is a pre-requisite for making the students into self-directed learners. The first step for it would be awareness of the role of the learner in the teaching-learning environment. An aware and eager to learn student can be easily motivated to participate in learning actively and responsibly. Such a learner shall prepare before-hand to maximize the gains from a class and take learning to a whole new level. Learners when self-aware and motivated can be more easily helped to become self-directed. The feedback of the students in the present study

shows that the students are aware, prepared and motivated for their study after the periodic exposure of flipped classroom.

LIMITATIONS:

All the students could not reap the same benefits as others owing to differences in academic level, learning preferences and speed.

We have seen the effectiveness of this approach only in a single subject and only in a single batch of MBBS students. To consolidate full potential gains of this interactive educational intervention (FC), it's necessary to include FC in the curriculum as one of the interventional methods of choice across different professional years.

CHALLENGES TO CONSIDER:

1. Technology access and infrastructure: Unequal access to technology and internet connectivity across different regions may hinder student's participation in FC.
2. Faculty training and development: Adequate training and support for educators is essential for transitioning to FC strategy for successful implementation. It also mandates maintenance of adequate student: teacher ratio.
3. Student preparedness and learning styles: Adapting to independent learning and active participation is difficult initially as the students are not exposed to this way of learning during their school days which requires preparation and guidance from faculty.
4. Assessment and evaluation: Traditional exams focuss on factual recall may not effectively measure the deeper learning and critical thinking skills fostered by flipped classrooms. Developing new assessment methods aligned with the learning objectives of the flipped model is crucial.

POTENTIAL BENEFITS:

- Improved engagement and learning: Flipped classrooms entail active learning. This can lead to deeper understanding, improved critical thinking skills, and better application of knowledge to real-world scenarios.
- Individualized learning: FC caters to individual needs and promotes self-directed learning by providing choice of study material, style and pace.
- Enhanced student-teacher interaction: Closer student-teacher interactions enable faculty to identify and address individual learning gaps and provide timely feedback.
- Alignment with Competency-Based Medical Education (CBME): Flipped classrooms align with this approach by fostering application of knowledge, problem-solving, and teamwork through active learning and collaborative activities.

CONCLUSION:

Our study clearly shows that the flipped classroom technique is more effective in improving the performance of students in comparison to the tradition method of teaching through didactic lectures. The faculty members as well as the students held a positive view about this teaching learning technique and students were satisfied with the way that these sessions were conducted.

For earning wholesome benefits of FC, it should be implemented by every department periodically and incorporated in the curriculum. Rather than remaining as a one-off intervention, FC should be an important teaching learning strategy for key topics which demand clarity of concepts, interactive discussions and application for problem solving. If done in a coordinated manner at the institutional level, FC can foster skills that encourage responsible and self-directed learning traits.

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PHYSIOLOGICAL EFFECTS OF INTERMITTENT FASTING ON HUMAN HEALTH

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ABSTRACT

Intermittent fasting (IF) is a dietary approach that alternates periods of eating and fasting and has gained attention for its potential health benefits. Common methods include time-restricted eating, alternate-day fasting, the 5:2 diet, and periodic fasting. IF induces metabolic changes in which the body shifts from glucose utilization to fat oxidation, resulting in ketone production and improved energy metabolism. Research suggests that intermittent fasting enhances insulin sensitivity, promotes weight loss, and improves glucose regulation, thereby reducing the risk of metabolic disorders such as obesity and Type 2 diabetes. Fasting also influences hormonal balance by reducing insulin levels and increasing glucagon and growth hormone secretion, which support fat metabolism and preservation of lean body mass. Cardiovascular benefits include reductions in blood pressure, cholesterol, triglycerides, and inflammatory markers. In the nervous system, ketone bodies and increased brain-derived neurotrophic factor (BDNF) may improve cognitive function and protect against neurodegenerative diseases. In addition, intermittent fasting stimulates autophagy and cellular repair mechanisms, which may contribute to healthy aging and longevity. It also modulates immune responses and reduces oxidative stress and chronic inflammation. However, intermittent fasting may cause side effects such as hunger, fatigue, dizziness, and nutritional deficiencies if practiced improperly. Certain populations, including pregnant women, individuals with chronic diseases, and those with eating disorders, require medical supervision. Overall, intermittent fasting appears to be a promising nutritional strategy for improving physiological health, although further long-term studies are needed to establish its safety and effectiveness.

INTRODUCTION:

Intermittent fasting (IF) is a dietary approach that alternates periods of eating and fasting. Unlike traditional diets that mainly focus on calorie restriction, intermittent fasting emphasizes the timing of food intake. Common methods include alternate-day fasting, the 5:2 diet, and time-restricted eating such as the 16:8 pattern. In recent years, intermittent fasting has gained global popularity because of its potential benefits on body weight, metabolism, cardiovascular health, and overall physiological functioning. Scientific research suggests that fasting induces several metabolic and hormonal adaptations that may improve health outcomes and reduce the risk of chronic diseases.[1] When you fast, your body switches from using sugar for energy to burning fat. As the stored sugar (glycogen) gets used up, your body starts breaking down fat and making ketones for fuel. These shifts affect many systems in the body, such as the hormone, heart, nerve, and immune systems.[2] Research also links

intermittent fasting to better insulin response, less inflammation, improved cell cleanup (autophagy), and stronger repair processes in cells.[3] Despite its increasing popularity, the physiological effects of intermittent fasting remain an active area of research. Some studies demonstrate significant benefits, while others suggest that the effects may vary according to age, gender, duration of fasting, and individual health conditions.[4] Therefore, understanding the physiological mechanisms and health implications of intermittent fasting is important for healthcare professionals and researchers.

TYPES OF INTERMITTENT FASTING:

Several forms of intermittent fasting are practiced worldwide. The most common methods include:

1. Time-Restricted Eating (TRE): Food intake is limited to a specific number of hours each day, usually 8–10 hours, while fasting continues for the remaining hours.
2. Alternate-Day Fasting (ADF): Individuals alternate between normal eating days and fasting days.
3. 5:2 Diet: Individuals consume normal meals for five days per week and restrict calorie intake on two non-consecutive days.
4. Periodic Fasting: Fasting is practiced for extended periods such as 24–48 hours once or twice monthly.[5]

Each method produces physiological changes depending on fasting duration and nutritional intake.

METABOLIC EFFECTS OF INTERMITTENT FASTING:

One of the most important physiological effects of intermittent fasting is improvement in metabolic health. During fasting, insulin secretion decreases and glucagon levels rise. Reduced insulin promotes breakdown of glycogen and stimulates fat oxidation. As fasting continues, ketone bodies become an alternative energy source for tissues including the brain.[6]

Intermittent fasting enhances insulin sensitivity and improves glucose regulation. This effect may help reduce the risk of Type 2 Diabetes. Several studies have shown reductions in fasting blood glucose and glycated hemoglobin levels among individuals practicing intermittent fasting.[7]

The metabolic switch occurring during fasting also promotes weight loss. Caloric intake generally decreases, leading to reduced adipose tissue mass. Decreased visceral fat improves metabolic efficiency and lowers the risk of obesity-related disorders.[8]

Fasting activates AMP-activated protein kinase (AMPK) and suppresses the mammalian target of rapamycin (mTOR) pathway, thereby stimulating autophagy. Autophagy is a cellular process that removes damaged proteins and organelles, contributing to cellular repair and longevity.[9]

HORMONAL CHANGES DURING FASTING:

Intermittent fasting significantly affects endocrine function. Insulin levels decrease during fasting, whereas glucagon secretion increases to maintain blood glucose levels. Growth hormone secretion also rises during fasting, helping preserve lean muscle mass and stimulate fat metabolism.[10]

Cortisol levels may transiently increase during fasting as part of the body's adaptive response to stress. Short-term increases in cortisol help maintain energy availability by promoting gluconeogenesis and lipolysis. However, prolonged or excessive fasting may lead to elevated stress hormone levels and adverse physiological consequences.[11]

Leptin and ghrelin, hormones involved in appetite regulation, are also affected by fasting. Ghrelin levels increase before meals and decrease after eating, while leptin levels are associated with body fat stores. Intermittent fasting may improve leptin sensitivity and appetite control over time.[12]

Thyroid hormones may show slight reductions during prolonged fasting due to decreased metabolic rate. These hormonal adaptations help conserve energy during periods of nutrient deprivation.[13]

CARDIOVASCULAR EFFECTS:

Intermittent fasting has shown promising effects on cardiovascular health. Studies report reductions in blood pressure, serum triglycerides, total cholesterol, and low-density lipoprotein (LDL) cholesterol levels among fasting individuals.[14]

Fasting improves endothelial function and reduces oxidative stress, thereby decreasing the risk of atherosclerosis and cardiovascular disease. Improved insulin sensitivity and reduced body weight further contribute to cardiovascular protection.[15]

Inflammatory markers such as C-reactive protein (CRP), tumor necrosis factor-alpha (TNF- α), and interleukin-6 may decrease during intermittent fasting. Chronic inflammation is strongly associated with cardiovascular disease; therefore, reduction of inflammatory mediators may provide protective effects.[16]

However, some recent studies suggest that excessively restrictive eating windows may be associated with increased cardiovascular risk in certain populations. More long-term clinical trials are necessary to clarify these findings.[17]

NEUROLOGICAL AND COGNITIVE EFFECTS:

The nervous system is highly influenced by fasting-induced metabolic changes. During fasting, ketone bodies serve as an efficient energy source for neurons and may improve cognitive performance. Ketones also reduce oxidative stress and support mitochondrial function.[18]

Intermittent fasting stimulates the production of brain-derived neurotrophic factor (BDNF), a protein involved in neuronal growth, learning, and memory. Increased BDNF levels may protect against neurodegenerative diseases such as Alzheimer Disease and Parkinson Disease.[19]

Animal studies indicate that fasting may enhance synaptic plasticity and neurogenesis. Human studies also suggest improvements in concentration, alertness, and mental clarity during controlled fasting periods.[20]

Furthermore, intermittent fasting may reduce neuroinflammation and support healthy aging of the brain. Nevertheless, prolonged fasting may cause headaches, irritability, fatigue, and impaired concentration in some individuals.[21]

EFFECTS ON IMMUNE FUNCTION AND INFLAMMATION:

Intermittent fasting may positively influence immune function through modulation of inflammatory pathways. Fasting reduces oxidative stress by decreasing production of reactive oxygen species. Reduced oxidative stress protects cellular structures from damage.[22]

Autophagy induced during fasting helps remove dysfunctional immune cells and promotes regeneration of healthy cells. This process contributes to immune system maintenance and may reduce susceptibility to chronic inflammatory disorders.[23]

Studies have demonstrated decreased inflammatory cytokine production during fasting. Such effects may benefit conditions associated with chronic inflammation including obesity, metabolic syndrome, and autoimmune disorders.[24]

In addition, fasting may alter gut microbiota composition, promoting beneficial bacterial growth and improving intestinal health. Gut microbiota plays an important role in immunity and metabolic regulation.[25]

EFFECTS ON BODY WEIGHT AND OBESITY:

Obesity is a major global health problem associated with diabetes, hypertension, and cardiovascular disease. Intermittent fasting has become a popular strategy for weight management due to its simplicity and effectiveness.[26]

Fasting reduces overall caloric intake and promotes fat utilization. Increased lipolysis during fasting decreases adipose tissue accumulation. Studies report reductions in body weight, waist circumference, and body fat percentage among individuals practicing intermittent fasting.[27]

Unlike continuous calorie restriction, intermittent fasting may preserve lean muscle mass because of increased growth hormone secretion and enhanced fat metabolism.[28]

However, weight loss outcomes vary among individuals. Some recent meta-analyses suggest that intermittent fasting may not produce significantly greater weight loss compared to conventional calorie-restricted diets.[29]

CELLULAR REPAIR AND LONGEVITY:

One of the most interesting physiological effects of intermittent fasting is its role in cellular repair and longevity. Fasting activates stress-response pathways that enhance cellular resilience. Increased autophagy helps eliminate damaged proteins and dysfunctional mitochondria.[30]

Intermittent fasting may reduce oxidative damage and improve mitochondrial efficiency, thereby slowing cellular aging processes. Experimental studies suggest that fasting may increase lifespan in certain animal models.[31]

Activation of sirtuins and suppression of inflammatory pathways may further contribute to healthy aging. These molecular mechanisms are being extensively investigated in current biomedical research.[32]

RISKS AND LIMITATIONS OF INTERMITTENT FASTING:

Although intermittent fasting offers potential benefits, it may not be suitable for everyone. Common side effects include hunger, headache, dizziness, irritability, weakness, and fatigue during the initial adaptation period.[33]

Excessive fasting may lead to nutritional deficiencies if dietary intake is inadequate. Individuals with diabetes, pregnancy, eating disorders, chronic illnesses, or underweight conditions should practice fasting only under medical supervision.[34]

Women may experience menstrual irregularities and hormonal disturbances with prolonged restrictive fasting. Additionally, strict fasting schedules may negatively affect social eating behaviors and psychological well-being.[35]

Current evidence regarding long-term safety remains limited. Therefore, individualized approaches and balanced nutrition are important while practicing intermittent fasting.[36]

CONCLUSION:

Intermittent fasting is an increasingly popular dietary strategy with significant physiological effects on human health. It influences metabolism, endocrine function, cardiovascular health, nervous system activity, immune responses, and cellular repair mechanisms. Scientific evidence suggests that intermittent fasting may improve insulin sensitivity, promote weight loss, reduce inflammation, and enhance metabolic health.

The metabolic switch from glucose utilization to fat oxidation represents a major physiological adaptation during fasting. Hormonal changes including reduced insulin and increased growth hormone contribute to improved energy balance and fat metabolism. Fasting-induced autophagy and cellular repair mechanisms may support healthy aging and disease prevention.

Despite these potential benefits, intermittent fasting is not universally appropriate and may carry risks when practiced excessively or without proper nutritional guidance. Long-term studies are still required to establish its safety, efficacy, and optimal fasting protocols in different populations.

Overall, intermittent fasting represents a promising nutritional intervention that may contribute to improved physiological health when practiced responsibly and under appropriate supervision.

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AUTONOMIC DYSREGULATION AND COCHLEAR MICROCIRCULATION IN IDIOPATHIC SUDDEN SENSORINEURAL HEARING LOSS: A NARRATIVE REVIEW

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ABSTRACT

Idiopathic sudden sensorineural hearing loss (SSNHL) is an otologic emergency defined as a hearing reduction of at least 30 dB across three contiguous frequencies within 72 hours, occurring without an identifiable cause. Its pathogenesis remains unresolved, with viral, vascular, and autoimmune mechanisms forming the principal hypotheses. This narrative review focuses on the vascular hypothesis and its physiological extension toward autonomic regulation. The cochlea is an end-organ supplied by a single labyrinthine artery and sustained by an extremely low and finely regulated blood flow; it is therefore highly susceptible to ischaemic injury. Because cochlear and systemic vascular tone are modulated by the autonomic nervous system, autonomic dysfunction has been proposed as a contributor to the microvascular compromise implicated in SSNHL. Heart rate variability (HRV) and blood pressure variability (BPV), established non-invasive markers of autonomic activity, have been examined in several clinical studies, which report altered autonomic regulation, reduced HRV, and elevated night-time blood pressure in affected patients, with associations to severity and prognosis. The evidence remains limited by small, cross-sectional, single-centre designs and methodological heterogeneity, and the direction of causation is unresolved. This review summarises the relevant cochlear vascular physiology, the autonomic evidence base, and the principal gaps that future research must address.

KEYWORDS: sudden sensorineural hearing loss; cochlear blood flow; autonomic nervous system; heart rate variability; vascular etiology.

INTRODUCTION:

Sudden sensorineural hearing loss (SSNHL), or sudden deafness, is conventionally defined as a sensorineural hearing reduction of at least 30 dB affecting three contiguous audiometric frequencies and developing within 72 hours¹. It constitutes an otologic emergency, since the probability of recovery is closely linked to the promptness of treatment. The condition is broadly classified as primary or secondary according to whether a cause can be established². Primary SSNHL is attributed to mechanisms such as viral infection, impaired inner-ear perfusion, or immune-mediated injury, and some evidence implicates stress-related changes in cochlear cells, including pathways involving nuclear factor kappa B³. Secondary SSNHL arises from an identifiable cause—neoplasm, stroke, or radiation, among others—and

is diagnosed after exclusion of trauma, Ménière's disease, noise injury, and ototoxic exposure. Reported recovery varies considerably with severity and with differing definitions of improvement. Spontaneous recovery has historically been estimated at approximately 65%⁴, yet among patients with severe-to-profound loss only a minority recover meaningfully: complete recovery has been reported in the majority of mild cases but in a small fraction of profound cases⁴, and large cohorts of profound SSNHL describe partial recovery in roughly one third and full restoration in only a few per cent⁵. A poorer initial pure-tone average, together with comorbidities such as diabetes mellitus and hypercholesterolaemia, is consistently associated with worse prognosis⁶. Despite extensive investigation, the pathogenesis of idiopathic SSNHL remains unclear. The dominant hypotheses—viral infection, vascular compromise, and autoimmune injury—have not been conclusively validated in human subjects by non-invasive imaging or by laser-Doppler measurement of cochlear blood flow. Nonetheless, several lines of evidence support a vascular component in at least a subset of cases, in whom hearing loss appears attributable to cardiovascular factors⁷. The aim of this review is to examine the vascular hypothesis through the lens of physiology, and in particular to consider whether autonomic dysregulation, acting upon a vulnerable cochlear microcirculation, represents a plausible contributor to SSNHL.

LITERATURE SEARCH:

PubMed and Scopus were searched for English-language articles combining the terms sudden sensorineural hearing loss, idiopathic, cochlear blood flow, vascular, autonomic nervous system, heart rate variability, and blood pressure variability. Priority was given to studies of autonomic function in SSNHL and to physiological accounts of cochlear microcirculation. As a narrative review, this work does not claim systematic completeness; sources were selected for mechanistic relevance and methodological clarity.

ETIOLOGICAL HYPOTHESES:

Three mechanisms dominate discussion of idiopathic SSNHL. The viral hypothesis is supported by seasonal patterns, antecedent upper-respiratory illness, and histopathological findings consistent with labyrinthitis. The autoimmune hypothesis draws on the corticosteroid responsiveness of some patients and on inflammatory mechanisms of cochlear injury, including stress-activated intracellular pathways³. The vascular hypothesis rests on the anatomy of the inner-ear blood supply and on the cochlea's sensitivity to ischaemia, and it is reinforced by the association of SSNHL with systemic cardiovascular disease⁷. These hypotheses are not mutually exclusive; inflammation, infection, and vascular compromise may interact. The present review concentrates on the vascular mechanism because it connects most directly to a measurable physiological system—autonomic cardiovascular control.

COCHLEAR MICROCIRCULATION AND ISCHAEMIC VULNERABILITY:

The susceptibility of the cochlea to circulatory disturbance is rooted in its vascular anatomy (Figure 1). The inner ear functions as a terminal, or "end," organ, typically supplied by a single labyrinthine artery—occasionally two—most often arising from the anterior inferior cerebellar artery (AICA)⁸. The principal cochlear vessel, the spiral modiolar artery, is a terminal branch of this supply; it courses over the scala vestibuli and across the spiral lamina, sending radiating arterioles to the lateral wall, where they form

two parallel capillary networks, one within the spiral ligament and one within the stria vascularis⁹. These networks run alongside one another without cross-connections, a configuration that limits collateral compensation⁹. Cochlear blood flow is remarkably low—on the order of one ten-thousandth of cardiac output in rodents, and lower still in humans—and is non-pulsatile, an arrangement that minimises mechanical interference with the sensory epithelium¹¹. The stria capillaries are larger and more densely packed with erythrocytes than those of the spiral ligament, and flow velocities in the lateral wall are correspondingly slow¹¹. The pre- and post-capillary vessels of the spiral ligament contain smooth-muscle cells and are considered important regulators of lateral-wall perfusion, while the stria vessels, though contributing little to flow regulation, are essential for maintaining the endocochlear potential, ion exchange, and fluid balance. A notable feature of this microvasculature is its high density of pericytes—contractile, smooth-muscle-like cells that, in the spiral ligament, express α -smooth-muscle actin and possess vasocontractile properties¹². In other organs, pericytes regulate capillary flow, angiogenesis, and endothelial signalling through a range of vasoactive mediators; their role in cochlear microcirculation, though historically underexplored, is increasingly recognised¹². Taken together, the single end-artery supply, the absence of meaningful collateral pathways, the very low baseline flow, and the active contractile regulation of the lateral-wall vessels render the cochlea exquisitely vulnerable: even a transient reduction in perfusion can rapidly impair the organ of Corti, whose hair cells are highly sensitive to hypoxia and ischaemic damage.

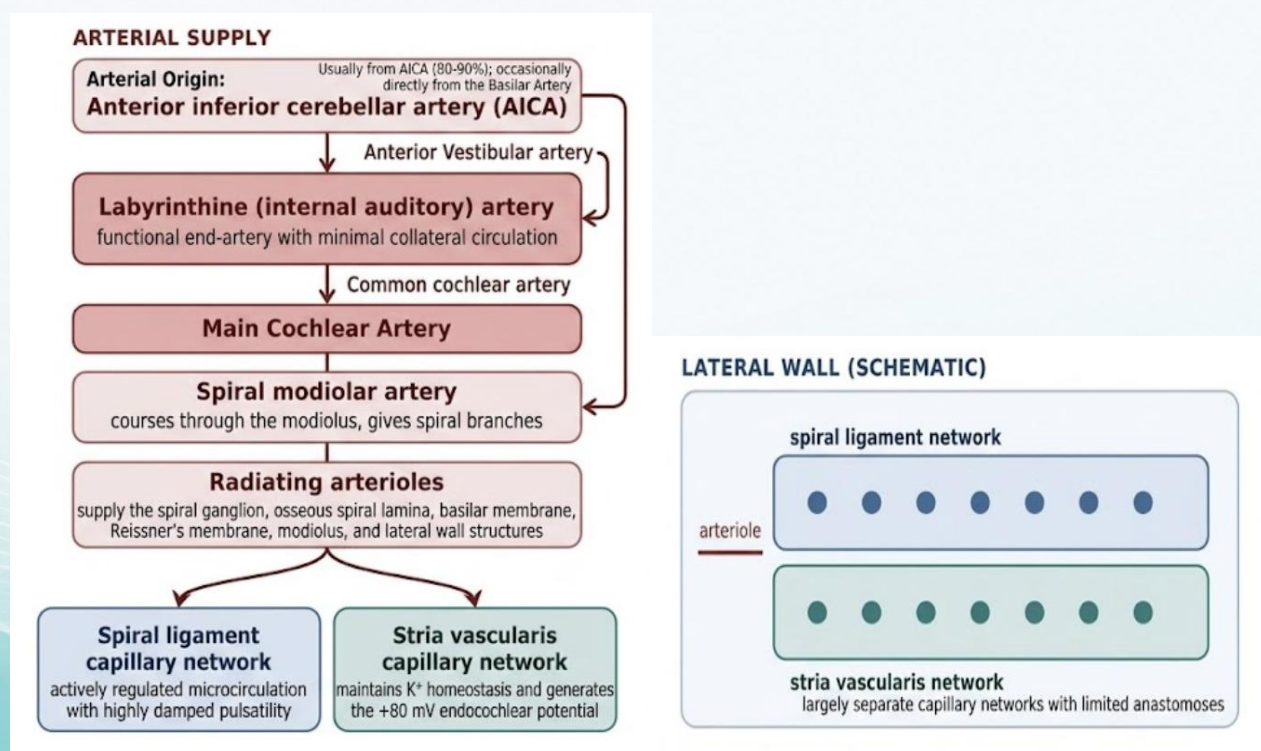


Figure 1. Schematic representation of the cochlear blood supply. The anterior inferior cerebellar artery gives rise to the labyrinthine (internal auditory) artery, a functional end-artery without collateral supply; its continuation, the spiral modiolar artery, sends radiating arterioles to the cochlear lateral wall, forming two parallel capillary networks—within the spiral ligament and the stria vascularis—that run alongside one another without anastomoses. Cochlear blood flow is exceptionally low and non-pulsatile, and lateral-wall vessels are actively regulated by smooth-muscle cells and pericytes. Original illustration

prepared by the author; anatomical relationships based on Axelsson (1968)⁹ and Mudry & Tange (2009)¹⁰.

EVIDENCE FOR VASCULAR INVOLVEMENT IN SSNHL:

Several independent observations support a vascular contribution to SSNHL. In patients over 50 years of age, reduced vertebrobasilar blood flow has been demonstrated on magnetic resonance imaging substantially more often than in controls imaged for headache, and transcranial Doppler ultrasonography has likewise identified abnormal vertebrobasilar circulation more frequently in SSNHL patients than in comparison groups¹³. Vertebrobasilar insufficiency or occlusion is a recognised cause of sudden deafness, which may be the presenting feature even in the absence of other neurological signs¹⁴.

HAEMORHEOLOGICAL ABNORMALITIES PROVIDE FURTHER SUPPORT: increased whole-blood and plasma viscosity and elevated plasma fibrinogen have been described in SSNHL patients, each capable of reducing cochlear blood flow and promoting thromboembolic events¹⁵. The occurrence of SSNHL in association with systemic conditions characterised by vascular compromise—and the reported relationships between SSNHL and elevated cholesterol, fibrinogen, and homocysteine—reinforce the hypothesis that, in a subset of patients, the disorder reflects an underlying vascular dysfunction¹⁶. A recurring theme across these studies is a higher prevalence of elevated systolic blood pressure and of a personal or family history of arterial occlusive events among affected individuals, observations that point beyond the cochlea toward systemic regulation of the circulation.

AUTONOMIC REGULATION OF THE CARDIOVASCULAR SYSTEM AND HEART RATE VARIABILITY:

If systemic vascular control is relevant to SSNHL, the autonomic nervous system (ANS) becomes a natural focus, since it governs heart rate, blood pressure, and vascular tone. Autonomic dysfunction is implicated in hypertension, heart failure, stroke, sleep apnoea, hypoxia, and diabetes mellitus—conditions that themselves overlap with the cardiovascular risk profile of SSNHL. Heart rate variability (HRV), the beat-to-beat variation in the interval between successive heartbeats, is a widely used, non-invasive measure of autonomic modulation of the heart¹⁷. It reflects the balance between parasympathetic (vagal) and sympathetic influences on the sinoatrial node: vagal activity, mediated by acetylcholine, slows the heart and increases variability, whereas sympathetic activity, mediated by catecholamines, accelerates it and reduces variability. Analysis is commonly performed in the time domain (for example, the standard deviation of normal-to-normal intervals), the frequency domain, and by non-linear methods. In the frequency domain, the high-frequency (HF) component reflects parasympathetic modulation, the low-frequency (LF) component reflects combined sympathetic and parasympathetic influence, and the LF/HF ratio is used as an index of sympatho-vagal balance¹⁷. A predominance of sympathetic activity is associated with reduced overall variability, and low HRV has been linked to hypertension and to adverse cardiovascular outcomes. Importantly, HRV has been proposed as a marker of vascular tone, the same physiological variable that governs cochlear perfusion.

EVIDENCE FOR AUTONOMIC DYSFUNCTION IN SSNHL:

A small but consistent body of work has examined autonomic function directly in SSNHL. In an early study of 23 patients and matched controls, conventional HRV indices did not differ significantly between

groups, yet patients showed altered cardiovascular coupling and reduced short-term blood pressure variability, indicating disrupted cardiovascular regulation; the authors suggested these markers might help identify vascular-related SSNHL in particular subgroups¹⁸. A subsequent bivariate analysis by the same group confirmed altered autonomic regulation, with significant differences in non-linear heart-rate and blood-pressure dynamics, cardiovascular coupling, and vascular indices that were independent of age, sex, hypertension, and medication¹⁹. Later studies extended these findings. Using Holter and ambulatory blood-pressure monitoring in 58 participants, one investigation found significantly higher night-time systolic and diastolic blood pressure and reduced HRV in SSNHL patients, consistent with autonomic dysfunction contributing to disease onset²⁰. In a larger study of 55 patients and 40 controls, HRV—total power, LF, and HF—was significantly reduced in the patient group; although HRV did not distinguish mild from severe cases, LF and normalised HF were associated with hearing recovery, and age and LF HRV emerged as predictors of clinical improvement, implicating autonomic function in prognosis as well as pathogenesis²¹. Most recently, a study of 63 patients using the COMPASS-31 autonomic symptom questionnaire alongside HRV reported reduced HRV parameters and elevated night-time blood pressure, interpreted as a shift toward sympathetic dominance and as evidence that SSNHL may involve systemic autonomic dysregulation rather than a purely cochlear disorder²². Across these studies a pattern emerges of reduced variability, altered blood-pressure regulation, and relative sympathetic predominance in SSNHL patients. It is notable, however, that the signal is not uniform across measures: blood pressure variability and cardiovascular coupling have at times discriminated patients from controls where standard HRV did not¹⁸, suggesting that the choice of autonomic measure materially affects sensitivity.

A PROPOSED MECHANISTIC LINK:

These threads converge on a coherent, if still unproven, mechanism (Figure 2). Predisposing influences—chronic stress, cardiovascular and metabolic disease, and ageing—shift autonomic balance toward sympathetic predominance. Heightened sympathetic tone, acting through the contractile regulation of the cochlear lateral-wall vessels and upon a circulation that lacks collateral reserve, may precipitate microvascular vasoconstriction and hypoperfusion. The resulting transient ischaemia would injure hypoxia-sensitive hair cells, producing sudden hearing loss. Within this framework, reduced HRV and altered BPV are not themselves causal but serve as measurable correlates of the underlying autonomic state. Framed in this way, autonomic dysregulation does not compete with the vascular hypothesis but offers a physiological mechanism upstream of it, while also accommodating the documented associations between SSNHL and systemic cardiovascular risk.

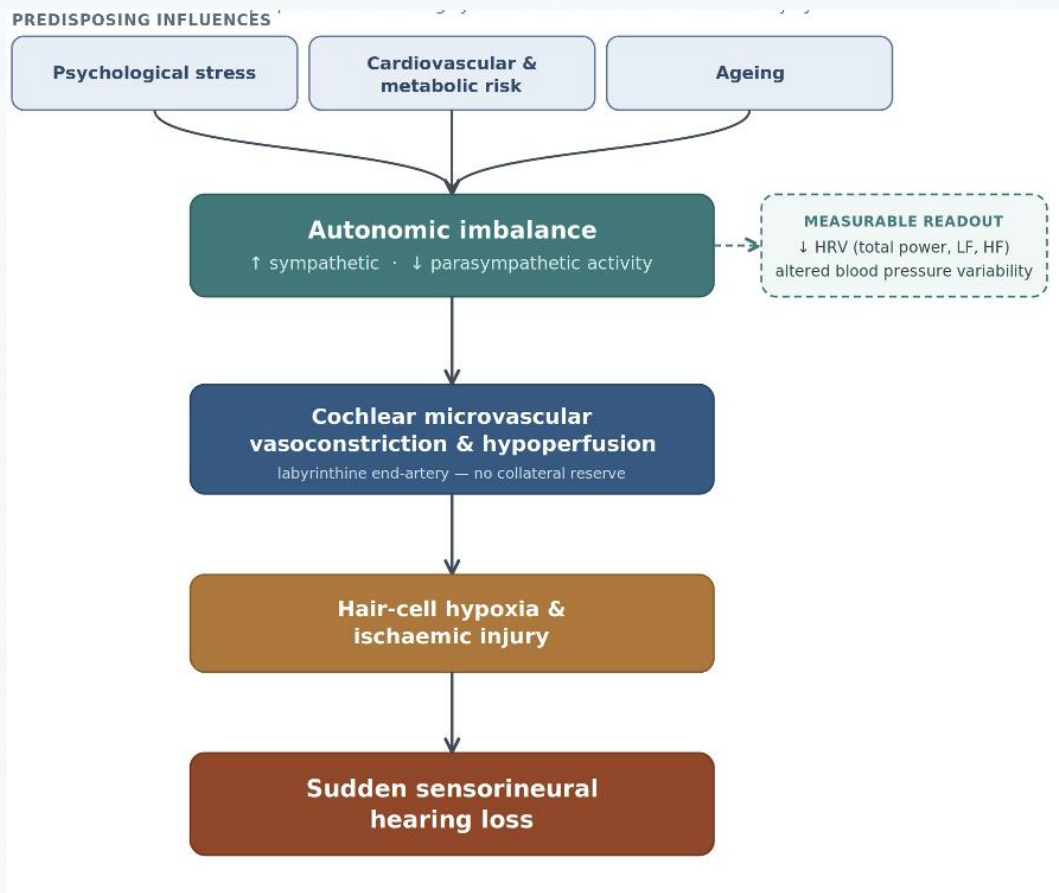


Figure 2. Proposed mechanistic model. Predisposing influences (psychological stress, cardiovascular and metabolic risk, ageing) shift autonomic balance toward sympathetic predominance; acting on a labyrinthine end-artery without collateral reserve, this may promote cochlear microvascular vasoconstriction and hypoperfusion, hair-cell hypoxia, and sudden sensorineural hearing loss. Reduced heart rate variability (HRV) and altered blood pressure variability (BPV) serve as measurable, non-causal correlates of the autonomic state. The downstream chain rests on established cochlear physiology; its operation as the trigger in idiopathic SSNHL is proposed, and the direction of causation remains unestablished. Original figure prepared by the author, synthesised from references 7 and 18–22.

GAPS IN THE LITERATURE:

Although vascular abnormality is widely regarded as a predisposing factor in SSNHL, the mechanism by which autonomic dysfunction might compromise cochlear microcirculation has not been clearly established. HRV is a validated measure of cardiac autonomic function, but the pathway linking autonomic dysregulation to cochlear hypoperfusion remains inferential rather than demonstrated. Several specific limitations recur across the literature. First, the available studies are predominantly cross-sectional, leaving the temporal relationship—and thus causation—unresolved; reduced HRV may predispose to hypoperfusion, may arise as a stress response to acute hearing loss, or may mark a shared cardiovascular vulnerability. Second, most studies are small and single-centre, with methodological heterogeneity and no standardised protocol for autonomic assessment, limiting comparison and generalisability. Third, longitudinal data on how autonomic markers evolve over the course of the disease or in response to treatment are largely absent. Finally, interventional research evaluating whether autonomically targeted strategies improve outcomes is lacking. These gaps indicate a need for

larger, multicentre, prospective studies employing standardised autonomic measures and, ideally, assessment in the earliest phase of the disease.

CONCLUSION:

Idiopathic SSNHL remains etiologically uncertain, but its most physiologically tractable mechanism—cochlear hypoperfusion in a vulnerable end-organ—has a plausible upstream regulator in the autonomic nervous system. Clinical studies using HRV and BPV consistently report altered autonomic regulation and relative sympathetic predominance in affected patients, with associations to severity and prognosis. The evidence is, however, preliminary: it is drawn from small cross-sectional cohorts, the autonomic signal varies with the measure employed, and causation has not been established. Autonomic dysregulation is therefore best regarded at present as a coherent and testable contributing mechanism rather than a confirmed cause. Larger prospective studies combining standardised autonomic assessment with measures of cochlear perfusion are required to determine its true role and its potential value for diagnosis and prognosis.

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STUDY OF DYNAMIC PROPERTIES OF ARTERIAL PULSE: CLINICAL SIGNIFICANCE OF PULSE WAVE VELOCITY AND ARTERIAL STIFFNESS

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The arterial pulse is the abrupt expansion of an artery resulting from the sudden ejection of blood into the aorta and its transmission throughout the arterial system.

PROPERTIES OF PULSE:

General Properties	Dynamic Properties
Rate	Pulse Wave Velocity
Rhythm	Pulse Wave Reflection
Volume	Pulse Transit Time
Character	Pulse Pressure Amplification
Tension	Arterial compliance
Vessel wall condition	

PULSE WAVE VELOCITY:

Pulse Wave Velocity is the speed at which the arterial pressure wave propagates through the arterial tree. Pulse Wave Velocity (PWV) is one of the most widely accepted indicators of arterial stiffness and vascular health. PWV is directly proportional to arterial stiffness, such that an increase in arterial rigidity results in faster propagation of the pulse wave through the arterial tree. Conversely, highly compliant arteries slow pulse wave transmission by absorbing and storing a portion of the systolic energy generated by ventricular contraction. As arterial compliance decreases, Pulse Wave Velocity increases and the normal Windkessel effect of the aorta is progressively lost. Consequently, PWV is considered the gold-standard surrogate marker for assessing arterial stiffness and vascular aging. Beyond its role as a marker of vascular mechanics, elevated PWV has been strongly associated with end-organ damage. Increased arterial stiffness allows excessive pulsatile energy to reach the microcirculation, resulting in endothelial dysfunction, impaired tissue perfusion, chronic ischemic injury, and structural remodeling of target organs. High PWV has been linked with cerebral microvascular disease, retinal vascular injury, renal dysfunction, and cardiac remodeling, making it an important predictor of future cardiovascular and systemic complications.

ARTERIAL STIFFNESS:

Under normal physiological conditions, the aortic wall is composed primarily of elastin fibers, collagen fibers, and vascular smooth muscle cells. Elastin provides elasticity and enables the aorta to expand during systole and recoil during diastole, thereby storing part of the systolic energy as elastic potential energy and maintaining the Windkessel function. Collagen provides tensile strength and prevents excessive distension of the vessel wall under high pressures, while vascular smooth muscle regulates vascular tone and contributes to the structural integrity of the artery. Arterial stiffness develops as a

consequence of age-related and pathological alterations in these structural components. Fragmentation and degeneration of elastin fibers reduce arterial compliance, while increased collagen deposition makes the vessel wall more rigid. Additional factors such as smooth muscle hypertrophy, vascular calcification, and endothelial dysfunction further contribute to loss of arterial elasticity. These structural changes impair the ability of arteries to buffer pulsatile blood flow, resulting in increased Pulse Wave Velocity and heightened cardiovascular risk.

DETERMINANTS OF ARTERIAL STIFFNESS:

Arterial stiffness is influenced by a combination of physiological, structural, hormonal, and lifestyle-related factors. Aging is the most important determinant and is associated with progressive degeneration and fragmentation of elastin fibers, increased collagen deposition, vascular calcification, and endothelial dysfunction, all of which reduce arterial compliance and increase Pulse Wave Velocity (PWV). Gender also plays a significant role in vascular stiffness. Premenopausal women generally exhibit lower arterial stiffness due to the vasoprotective effects of estrogen, which enhances endothelial function, promotes vasodilation, and preserves arterial elasticity. Following menopause, declining estrogen levels contribute to increased arterial stiffness, elevated PWV, and a higher risk of cardiovascular disease. Structural characteristics of the arterial wall, including elastin content, collagen content, and vascular smooth muscle tone, are major determinants of arterial mechanical properties. Arteries rich in elastin demonstrate greater compliance, whereas increased collagen content and smooth muscle hypertrophy contribute to vascular rigidity. In addition, several modifiable lifestyle factors influence arterial stiffness. Obesity, smoking, sedentary behavior, hypertension, and diabetes mellitus accelerate vascular aging through mechanisms involving chronic inflammation, oxidative stress, endothelial dysfunction, and adverse structural remodeling of the arterial wall. Together, these factors contribute to increased arterial stiffness and heightened cardiovascular risk.

METHODS OF PULSE WAVE VELOCITY MEASUREMENT:

Pulse Wave Velocity is calculated by dividing the distance traveled by the pulse wave by the time required for its propagation between two arterial sites. Among the available techniques, carotid–femoral Pulse Wave Velocity (cfPWV) is considered the gold-standard method for assessing aortic and central arterial stiffness. This technique measures the transit time of the pulse wave between the carotid and femoral arteries and provides a reliable estimate of large artery stiffness. Current recommendations from the European Society of Cardiology recognize cfPWV as the reference standard for clinical and research applications. Several alternative methods have also been developed for PWV assessment. Brachial–ankle PWV provides a simpler and more convenient measure of systemic arterial stiffness, while oscillometric devices estimate PWV using automated blood pressure cuffs etc.; they all aim to assess vascular stiffness and cardiovascular risk through measurement of pulse wave transmission characteristics.

Age category (years)	Mean (± 2 SD)	Median (10–90 pc)
<30	6.2 (4.7–7.6)	6.1 (5.3–7.1)
30–39	6.5 (3.8–9.2)	6.4 (5.2–8.0)
40–49	7.2 (4.6–9.8)	6.9 (5.9–8.6)
50–59	8.3 (4.5–12.1)	8.1 (6.3–10.0)
60–69	10.3 (5.5–15.0)	9.7 (7.9–13.1)
≥ 70	10.9 (5.5–16.3)	10.6 (8.0–14.6)

SD, standard deviation; 10 pc, the upper limit of the 10th percentile; 90 pc, the lower limit of the 90th percentile.

[1] PWV of individual of different age groups

DISEASE ASSOCIATIONS:

Arterial stiffness has been implicated in the pathogenesis of numerous cardiovascular and systemic diseases. In the cardiovascular system, increased arterial stiffness contributes to endothelial dysfunction, accelerated atherosclerosis, elevated systolic blood pressure, and increased left ventricular workload, thereby promoting the development of coronary artery disease and other cardiovascular disorders.

Within the central nervous system, elevated PWV and arterial stiffness are associated with cerebral small vessel disease, cognitive decline, vascular dementia, and brain atrophy. Excess pulsatile stress transmitted to delicate cerebral microvessels leads to chronic hypoperfusion, white matter injury, endothelial damage, and progressive neuronal loss. Renal function is also adversely affected by increased arterial stiffness. The kidneys contain highly specialized microvascular networks that are particularly vulnerable to excessive pulsatile pressure. Chronic exposure to such stress can result in glomerular injury, proteinuria, progressive nephron loss, and ultimately chronic kidney disease and nephropathy. Similarly, retinal circulation is susceptible to vascular injury caused by increased pulsatile transmission. Arterial stiffness has been associated with hypertensive retinopathy and diabetic retinopathy through mechanisms involving retinal microvascular damage and impaired vascular autoregulation. Although less extensively studied, arterial stiffness has also been linked with pulmonary vascular disorders, including pulmonary hypertension and reduced pulmonary vascular compliance. The relationship appears to involve alterations in vascular mechanics and hemodynamic stress. Emerging evidence suggests that arterial stiffness may influence reproductive health as well. Reduced microvascular perfusion resulting from vascular dysfunction may impair testicular blood flow and spermatogenesis in males, while compromised ovarian perfusion may contribute to ovarian dysfunction and infertility in females.

CONCLUSION:

Pulse Wave Velocity is a dynamic property of the arterial pulse and a sensitive marker of arterial stiffness. Structural alterations in elastin, collagen, and vascular smooth muscle increase arterial rigidity and accelerate pulse wave propagation. Elevated PWV is associated with cardiovascular, cerebral, renal, retinal, and other forms of end-organ damage. PWV may be used for Early vascular aging assessment, Cardiovascular risk stratification, Monitoring hypertension, Monitoring diabetes-related vascular damage, Predicting stroke risk, Predicting chronic kidney disease progression, Monitoring treatment response etc. As a non-invasive measure of vascular health, PWV offers valuable insight into early vascular aging and may aid in the detection and monitoring of disease before overt clinical manifestations become apparent.

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THE GUT MICROBIOME, ESTROGEN, AND WOMEN'S HEALTH

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Over the past decade, our understanding of health has shifted in remarkable ways. At the center of this transformation is the gut microbiome, a vast and dynamic ecosystem that lives within us. Once viewed merely as a digestive helper, it is now known to be an active organ that regulates metabolism, immunity, and hormonal balance. For women, this connection between gut bacteria and hormones, particularly estrogen, opens new doors to understanding fertility, mood, and long-term well-being.

THE GUT MICROBIOME: A HIDDEN REGULATOR:

The gut microbiome is made up of trillions of microorganisms, including bacteria, fungi, and viruses. These microbes interact with our body in countless ways, influencing everything from digestion to immunity. When this community is balanced, it supports a state of health known as *eubiosis*. When it is disrupted, a condition called *dysbiosis* arises, leading to inflammation and metabolic imbalance.

In women's health, this balance plays a particularly significant role because the gut microbiota and female sex hormones continuously influence one another. Hormones shape which microbes thrive in the gut, while the microbiome, in turn, affects how hormones are metabolized, activated, or cleared from the body. This intricate partnership lies at the heart of the emerging field known as the microgenderome.

THE ESTROBOLOME AND HORMONAL BALANCE:

Within the microbiome exists a specialized group of bacterial genes known as the estrobolome. These genes regulate the metabolism and circulation of estrogen, one of the key hormones that governs reproductive and metabolic health. The estrobolome produces enzymes, such as beta-glucuronidase, that control whether estrogen is excreted or reabsorbed into the bloodstream.

When the microbiome is balanced, this process maintains healthy estrogen levels. When it is disrupted, estrogen can accumulate, leading to symptoms such as heavy bleeding, bloating, fibrocystic breasts, mood swings, and weight gain. Over time, chronic hormonal imbalance contributes to more serious conditions, including infertility, endometriosis, and estrogen-dependent cancers of the breast and endometrium.

WHAT CAUSES DYSBIOSIS:

Many factors can disturb the gut ecosystem. Repeated antibiotic use, poor diet, chronic stress, lack of exercise, and insufficient sleep all reduce microbial diversity. Diets low in fiber and high in processed foods are particularly harmful because they starve beneficial bacteria of the nutrients they need to survive. When this balance is lost, harmful bacteria multiply, leading to increased intestinal permeability, also known as "leaky gut." This allows toxins and bacterial fragments to enter the bloodstream, triggering inflammation and oxidative stress throughout the body. The result is a cascade that affects metabolism, immunity, and reproductive health.

THE GUT-THYROID CONNECTION:

The microbiome also communicates closely with the thyroid gland. Several studies have shown that people with autoimmune thyroid diseases, such as Hashimoto's thyroiditis and Graves' disease, often have distinct microbial patterns compared to healthy individuals. Certain bacteria, including *Neisseria* and *Streptococcus*, tend to increase in thyroid disorders, while beneficial bacteria like *Lactobacillus* and *Butyricimonas* are often reduced. These microbial shifts influence bile acid metabolism and the immune system, creating conditions that alter thyroid hormone function and feedback.

THE GUT, METABOLISM, AND PCOS:

The gut microbiome influences how efficiently we process nutrients and store energy. It helps produce short-chain fatty acids (SCFAs) such as butyrate and propionate, which lower inflammation and improve insulin sensitivity. When SCFA-producing bacteria decline, insulin resistance and fat accumulation increase, which in turn affect reproductive hormones.

In women with polycystic ovary syndrome (PCOS), the gut flora tends to show reduced levels of *Bifidobacterium* and *Faecalibacterium*, alongside higher levels of *Clostridium* and *Parabacteroides*. This imbalance amplifies inflammation and disrupts normal hormone signaling, contributing to hyperandrogenism, irregular ovulation, and subfertility. Research even shows that transferring gut microbes from lean individuals to those with metabolic syndrome can improve insulin sensitivity, highlighting the microbiome's role as a therapeutic target.

PREGNANCY AND THE MICROBIOME:

Pregnancy brings its own unique microbial shifts. Early in pregnancy, the gut supports nutrient absorption and immune tolerance, while in later stages, it helps store energy to sustain both mother and baby. During the third trimester, the population of *Proteobacteria* and *Actinobacteria* naturally increases to meet rising metabolic demands.

However, excessive imbalance during this time can lead to complications such as gestational diabetes, obesity, or preterm birth. The maternal microbiome also helps shape the baby's early gut flora, which begins colonizing during vaginal birth and breastfeeding. A healthy maternal microbiome is now considered one of the strongest predictors of a child's long-term immune and metabolic health.

THE MENOPAUSE TRANSITION:

After menopause, declining estrogen levels bring new changes to the gut. The reduction in hormonal protection increases gut permeability and systemic inflammation. These changes can contribute to weight gain, insulin resistance, and cardiovascular risk. Animal studies have shown that ovariectomy, which mimics menopause, alters gut microbial composition and increases intestinal inflammation.

Supporting gut health through diet and lifestyle during this stage can significantly improve quality of life. A fiber-rich diet, physical activity, and adequate sleep all help maintain a healthier microbiome, which in turn stabilizes metabolism and reduces inflammation.

MICROBIOME AND CANCER RISK:

The relationship between gut bacteria and cancer development is increasingly recognized. Certain bacteria can convert inactive estrogen into its active form, raising circulating estrogen levels and potentially promoting hormone-sensitive tumors. Others can create chronic inflammation that damages DNA and accelerates cell growth. In contrast, a healthy, balanced microbiome supports the immune system and helps detoxify carcinogenic compounds. Diets high in fiber, omega-3 fatty acids, and phytoestrogens have been found to protect against estrogen-driven cancers by supporting beneficial gut bacteria and improving estrogen metabolism.

RESTORING AND SUPPORTING GUT HEALTH:

Fortunately, the microbiome is remarkably adaptable. Even small changes in diet and routine can restore microbial balance. Eating a variety of whole plant foods, fermented products like yogurt and kefir, and high-fiber options such as legumes, fruits, and vegetables helps beneficial bacteria thrive. Each color of fruit or vegetable provides different phytonutrients that nourish unique species of microbes.

Regular exercise, mindful stress management, and quality sleep are equally important. These habits not only reduce inflammation but also encourage microbial diversity. Avoiding unnecessary antibiotics, minimizing alcohol intake, and staying hydrated also support the gut's delicate balance.

THE BIGGER PICTURE:

The gut microbiome is not a passive collection of organisms. It is an intelligent, responsive ecosystem that interacts with every organ system, including those that regulate hormones, immunity, and metabolism. For women, this relationship is particularly profound because hormonal balance, fertility, and emotional well-being are all influenced by gut health. By caring for the microbiome, women can strengthen the foundation of their physical and emotional vitality. The gut truly acts as a silent partner, shaping how we think, feel, and function. A healthy gut fosters a balanced body, and in turn, a balanced life.

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THYROID DISORDERS & ROLE OF LIFE STYLE MODIFICATION

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Thyroid disease is an increasingly common disease across the world. According to health studies, thyroid disorders affect around 4–5% of people in developed countries, while in India the prevalence is estimated to be as high as 10–11% (1, 2). The thyroid is a small butterfly-shaped gland located in the neck that produces hormones responsible for controlling the body's metabolism, energy levels, heart rate, and temperature regulation. There are two major types of thyroid disorders — hypothyroidism and hyperthyroidism. Both conditions affect hormone balance in different ways and can significantly impact daily life if left untreated.

HYPOTHYROIDISM: Hypothyroidism occurs when the thyroid gland does not produce enough thyroid hormone. This condition is relatively common and affects nearly 1 in 10 people in India. People with hypothyroidism often experience a sudden increase in weight, low energy and fatigue, slower heartbeat, feeling unusually cold, depression or low mood, dry and rough skin and reduced or normal appetite. A sluggish thyroid can slow down the body's metabolism, making even routine activities feel exhausting (1-3).

HYPERTHYROIDISM: Hyperthyroidism is the opposite condition, where the thyroid gland produces excessive hormones, causing the body to work faster than normal. Common symptoms include sudden weight loss, restlessness and nervousness, rapid heartbeat, feeling excessively hot, anxiety and irritability, sweaty or warm skin and increased appetite. If not properly managed, hyperthyroidism may affect heart health, sleep, and emotional wellbeing (1-3).

HEALTHY LIFESTYLE CAN MAKE A DIFFERENCE ALONG WITH THE STANDARD CARE:

Research have shown association between poor life style and thyroid disorders (4). Experts believe that a healthy lifestyle plays an important role in managing both hypothyroidism and hyperthyroidism. Proper nutrition, regular exercise, stress control, and good sleep habits can help maintain hormonal balance and improve overall health. Simple lifestyle measures can support thyroid function naturally by consuming iodine-rich foods to help hormone production. Include selenium-rich foods to support thyroid health. Exercise regularly to improve metabolism and energy. Eat a balanced diet rich in fruits, vegetables, and whole grains and avoid excessive junk and processed foods. Sleep for at least 7–8 hours daily and practice yoga, meditation, and deep breathing to reduce stress. Similarly, managing an overactive thyroid also requires dietary and lifestyle care. Eat foods low in iodine to reduce excess hormone production and include cruciferous vegetables such as cabbage, cauliflower, and broccoli in diet. Ensure adequate calcium and vitamin D intake and consume nuts and whole grains for sustained

energy. Practice stress-management techniques like yoga and meditation. Herbal teas may help calm anxiety and rapid heartbeat.

YOGA: A NATURAL SUPPORT FOR THYROID HEALTH:

Yoga is increasingly being recognized as an effective supportive therapy for thyroid disorders. It helps improve blood circulation, reduce stress, regulate hormones, and support mental wellbeing.

YOGA FOR HYPOTHYROIDISM:

Yoga practices for hypothyroidism aim to stimulate thyroid activity and improve metabolism. Recommended Asanas include Sarvangasana, Halasana, Matsyasana, Bhujangasana, Ushtrasana, Setubandhasana, Simhasana and Grivasanchalana Kriyas. Recommended Pranayama include Anulom-Vilom and Ujjayi Pranayama. Experts recommend practicing yoga for 20–30 minutes daily in the morning on an empty stomach. However, people with neck pain or high blood pressure should avoid inverted postures like Sarvangasana and Halasana.

YOGA FOR HYPERTHYROIDISM:

For hyperthyroidism, yoga focuses on calming the nervous system and reducing stress. Recommended Asanas include Shavasana, Sukhasana, Balasana, Shashankasana, Viparitakarni, Setubandhasana and Marjariyasana. Recommended Pranayama include Anulom-Vilom, Bhramari Pranayama and Shitali Pranayama. Gentle walking, meditation, proper sleep, and avoiding excess caffeine can also help manage symptoms effectively.

Thyroid Disorders & Role of Yoga / Life Style Modification

→ Balance Your Thyroid, Balance Your Life ←

ABOUT THYROID

The thyroid is a butterfly-shaped gland in the neck that produces hormones (T3, T4) which regulate metabolism, energy, temperature and many vital functions.

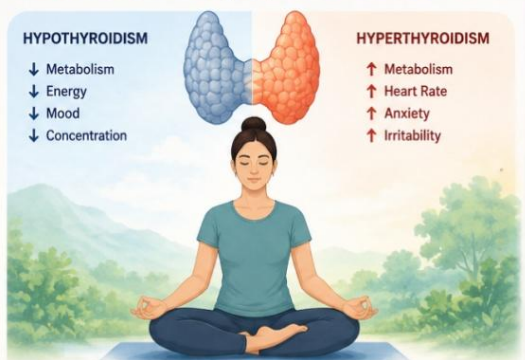


COMMON THYROID DISORDERS

HYPOTHYROIDISM
Underactive thyroid
Symptoms: fatigue, weight gain, cold intolerance, dry skin, constipation, depression.

HYPERTHYROIDISM
Overactive thyroid
Symptoms: weight loss, anxiety, palpitations, heat intolerance, irritability, sweating.

YOGA POSES BENEFICIAL FOR THYROID



Sarvangasana
(Shoulder Stand)

Halasana
(Plough Pose)

Matsyasana
(Fish Pose)

Bhujangasana
(Cobra Pose)

Dhanurasana
(Bow Pose)

Ushtrasana
(Camel Pose)

LIFESTYLE MODIFICATIONS

EAT BALANCED DIET
Include iodine-rich foods (iodized salt, dairy, seaweed), selenium (nuts, seeds), zinc, iron and plenty of fruits, vegetables and whole grains.

MAINTAIN HEALTHY WEIGHT
Helps regulate hormones and decreases strain on thyroid.

YOGA & LIFESTYLE MODIFICATION

can support healthy thyroid function, reduce symptoms and improve quality of life alongside medical treatment.



PRANAYAMA

- Anulom Vilom
- Bhramari
- Ujjayi

RELAXATION

- Yoga Nidra
- Meditation
- Deep Breathing

CONSISTENCY IS KEY
Yoga and healthy lifestyle choices empower your body to heal, balance hormones and enhance well-being naturally.



Yoga does not replace medical treatment.

Practice under guidance if you have severe symptoms or complications.

A balanced lifestyle is the foundation of a healthy thyroid and a healthy you.

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WHY THE MYOCARDIUM EXCEEDINGLY RARELY DEVELOPS MALIGNANCY

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ABSTRACT

Primary and metastatic cardiac malignancies are exceptionally rare, presenting in fewer than 0.3% of autopsies globally. Despite possessing an environment rich in oxygen, blood flow, and vascular nutrients, the myocardium has displayed a profound resistance to tumorigenesis. Until recently, this immunity was attributed solely to the non-proliferative state of mature cardiomyocytes. However, recent landmark research demonstrates that persistent, cyclical mechanical strain plays an active role in suppressing tumor growth. This review highlights how hemodynamic load alters chromatin accessibility via certain specific nuclear mechanosensors, called Nesprin-2 that inhibits the proliferation of invading malignant cells.

THE POST-MITOTIC STATE OF CARDIOMYOCYTES:

Following birth, cardiomyocytes permanently exit the cell cycle, with lesser than 1% of myocardial cells undergoing renewal annually. While this permanent mitotic arrest protects the organ from abnormal, uncontrolled proliferation, over time, the myocardium experiences a progressive accumulation of DNA damage and a concurrent decline in endogenous DNA repair mechanisms. Notably, aged cardiomyocytes accumulate DNA injury at a rate approximately three times faster than central nervous system neurons, despite the intricate and delicate structure of neural cells. Rather than leading to oncogenesis, this genetic instability more frequently manifests as somatic mutations linked to degenerative pathologies, such as coronary artery disease, or leads directly to cell death.

INTRODUCTION:

THE PROLIFERATIVE PARADOX AND TISSUE SUSCEPTIBILITY-

In broader physiological systems, cellular regeneration and oncogenic risk are inherently coupled. Highly proliferative tissues, such as the epithelium or hepatic parenchyma, frequently undergo mitotic renewal to repair damage inflicted by environmental toxins, radiation, or mechanical trauma. For instance, dermal cells turn over every 40 to 56 days. This continuous replication inherently exposes the genome to replication errors and mutational accumulation, serving as the primary driver for tumorigenesis. Conversely, tissues comprised of non-dividing or enucleated cells largely evade malignant transformation. Mature neurons rarely form primary carcinomas, and erythrocytes, lacking nuclei and DNA, are entirely incapable of oncogenic mutation. The heart, despite being subjected to lifelong

metabolic stress and contractile wear, its lack of cellular turnover acts as a primary baseline defense against localized carcinogenesis.

METHODOLOGY:

Experimental that have been conducted to study Mechanical Suppression of Oncology

1. IN VIVO GENOMIC MODELS OF MYOCARDIAL RESISTANCE-

To confirm that the heart possesses an intrinsic resistance to oncogenic transformation independent of environmental factor, researchers utilized a Cre-mediated recombination mouse model. This model induced the simultaneous overexpression of mutated K-Ras and the deletion of the tumor suppressor p53. Comparing the liver, skeletal muscle, and myocardium, multi-organ malignancies developed extensively throughout various anatomical sites, except the heart, which remained entirely free of tumor formation.

2. THE HETEROTOPIC HEART TRANSPLANT MODEL-

To isolate the specific impact of mechanical strain from purely biological or metabolic factors, investigators established a mouse model utilizing heterotopic heart transplantation.

In this surgical design, a donor heart is engrafted into the cervical (neck) region of a recipient mouse. The donor aorta and pulmonary artery are anastomosed to the host's carotid artery and external jugular vein, respectively. This configuration restores coronary perfusion and maintains myocardial viability, but completely unloads the left ventricle, thus rendering the transplanted heart non-functional and non-pumping. Meanwhile, the host's native heart continues to pump normally against physiological pressure. Following the injection of lung adenocarcinoma, colon carcinoma, or melanoma cells into the myocardium, distinct pathological outcomes were observed:

- **The Mechanically Unloaded (Transplanted) Heart:** Lacking mechanical strain, the tissue was rapidly infiltrated, with malignant cells extensively proliferating and overtaking nearly the entirety of the healthy myocardium within two weeks.
- **The Mechanically Loaded (Native) Heart:** The native, continuously beating heart vigorously resisted tumor colonization; malignant masses were significantly suppressed, rarely occupying more than 20% of any single cardiac chamber.

3. EX VIVO ENGINEERED HEART TISSUE (EHT)-

To further study the confounding systemic factors, researchers utilized *ex vivo* engineered heart tissues (EHTs) where mechanical pacing could be precisely modulated. In these models, mechanical load consistently inhibited the proliferation of invading cancer cells, whereas stopping the contraction directly promoted tumor expansion.

DISCUSSION:

THE ROLE OF NESPRIN-2 AS A KEY MECHANOSENSOR-

The molecular pathway translating macro-mechanical forces into microscopic nuclear changes relies on the linkers of nucleoskeleton and cytoskeleton (LINC) complex. Spatial transcriptomics of human cardiac metastases alongside EHT profiles revealed that Nesprin-2, a giant outer nuclear membrane protein, functions as the vital mechanosensor mediating this anti-proliferative response.

When cancer cells are engrafted into a beating myocardium, they are subjected to continuous mechanical strain from cardiomyocyte contraction and pressure-volume changes. Nesprin-2 binds to the cytoplasmic actin cytoskeleton and bridges it to the nuclear interior. Under mechanical load, Nesprin-2 transmits physical tension directly to the nucleus, causing physical deformation of the nuclear envelope. This nuclear strain shifts chromatin architecture by decreasing repressive histone modifications (such as H3K27me3 marks) and altering epigenetic packaging, resulting in localized chromatin decompaction and accessibility adjustments at critical proliferation-related loci. This mechanical remodeling effectively silences core oncogenic driver genes, like *MYC* and *CCND1* (Cyclin D1). Crucially, when Nesprin-2 is knocked out or silenced via CRISPR techniques, invading cancer cells become "blind" to the heartbeat, losing their mechanosensitive protection and proliferating freely within active cardiac tissue.

CLINICAL IMPLICATIONS AND FUTURE THERAPEUTIC FRAMEWORKS:

The profound oncogenic resistance of the heart is not merely a consequence of its post-mitotic state, but an active manifestation of continuous, cyclical mechanical compression. These mechanobiological insights into clinical oncology presents a novel therapeutic paradigm. Current collaborative research between oncologists and biomedical engineers aims to mimic these cardioprotective physical forces exogenously. For example, wearable, transdermal devices capable of delivering high-frequency, localized micro-compressions are currently being conceptualized to limit the proliferation and localized progression of cutaneous malignancies like melanoma, essentially "massaging" the tumor to recreate the mechanical microenvironment of a beating heart.

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ZEITGEBER DYSREGULATION AND GENERALIZED ANXIETY DISORDER: A CIRCADIAN PERSPECTIVE

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ABSTRACT

BACKGROUND: Generalized Anxiety Disorder (GAD) is among the most prevalent anxiety disorders worldwide, yet its neurobiological underpinnings remain incompletely understood. Emerging evidence implicates circadian rhythm disruption — mediated through dysregulated zeitgebers (external time-cues) — as a significant contributor to GAD pathophysiology. **Objective:** This review synthesizes current evidence on the interplay between zeitgeber dysregulation and GAD from a circadian neuroscience perspective. **Methods:** A narrative review of peer-reviewed literature (2000–2025) was conducted using PubMed, Scopus, and Google Scholar databases. **Results:** Disrupted light exposure, irregular feeding schedules, social isolation, and sleep-wake cycle disturbances impair the suprachiasmatic nucleus (SCN)-mediated master clock, precipitating HPA-axis hyperactivation, serotonin-melatonin imbalance, and amygdala hyperreactivity — all implicated in GAD. **Conclusion:** Chronotherapeutic interventions targeting zeitgeber normalisation represent a promising adjunct in GAD management.

KEYWORDS: *Zeitgeber; Circadian rhythm; Generalized Anxiety Disorder; Suprachiasmatic nucleus; Chronotherapy; HPA axis*

INTRODUCTION:

Anxiety disorders represent the most common category of psychiatric illness globally, affecting approximately 284 million individuals. Among these, Generalized Anxiety Disorder (GAD) — characterised by persistent, uncontrollable worry accompanied by somatic symptoms including muscle tension, fatigue, and sleep disturbances — has a lifetime prevalence of 5–9% in the general population.¹ While pharmacological and cognitive-behavioural interventions remain first-line treatments, a substantial proportion of patients exhibit treatment resistance, warranting exploration of novel pathophysiological mechanisms. The human body operates on an approximately 24-hour endogenous circadian cycle, orchestrated by the master pacemaker located in the suprachiasmatic nucleus (SCN) of the anterior hypothalamus. This intrinsic rhythm is continuously entrained by external environmental signals known as zeitgebers — a German term meaning "time-givers." The primary zeitgeber is photic (light) input via the retinohypothalamic tract, while secondary zeitgebers include meal timing, physical activity, social interaction, and ambient temperature.² Modern lifestyle patterns — characterised by shift work, artificial light exposure at night (ALAN), irregular meal timing, reduced outdoor activity, and social fragmentation — increasingly disrupt zeitgeber signalling. The resultant circadian misalignment has been linked to a spectrum of metabolic, cardiovascular, and psychiatric disorders. However, the specific mechanistic links between zeitgeber dysregulation and GAD pathophysiology have received

comparatively little systematic attention. This review aims to address that gap by examining the circadian biology underlying GAD, with implications for novel therapeutic strategies.

THE CIRCADIAN SYSTEM: AN OVERVIEW:

The SCN contains approximately 20,000 neurons expressing core clock genes including CLOCK, BMAL1, Period (PER1-3), and Cryptochrome (CRY1-2). These genes operate through interlocking transcription-translation feedback loops with a periodicity of ~24.2 hours in humans, requiring daily resetting via zeitgeber cues.³ The SCN relays timing signals to peripheral clocks throughout the brain and body — including the amygdala, prefrontal cortex, hippocampus, and adrenal glands — via hormonal messengers (primarily melatonin and cortisol) and autonomic neural pathways. Melatonin, synthesised by the pineal gland under SCN direction, serves as the principal chemical signal of darkness, promoting sleep onset and modulating immune and neuroendocrine functions. Cortisol follows a diurnal pattern with a peak at awakening (the cortisol awakening response) and a nadir at midnight, critically regulating arousal, metabolic function, and stress reactivity.⁴ Disruption of these rhythms impairs the temporal coordination of physiological processes — a state termed chronodisruption — with wide-ranging consequences for mental health.

ZEITGEBERS AND THEIR DYSREGULATION IN MODERN LIFE:

1. PHOTIC ZEITGEBERS-

Light is the most powerful zeitgeber. Intrinsically photosensitive retinal ganglion cells (ipRGCs) containing the photopigment melanopsin (peak sensitivity ~480 nm, blue spectrum) transmit luminance information directly to the SCN via the retinohypothalamic tract. Exposure to artificial blue-spectrum light in the evening delays the dim-light melatonin onset (DLMO) and advances the sleep phase, producing a state of chronic social jetlag. Population studies indicate that over 60% of individuals in developed nations are exposed to clinically significant ALAN, with strongest effects in urban populations.⁵

2. NON-PHOTIC ZEITGEBERS-

Meal timing represents a potent non-photic zeitgeber that primarily entrains peripheral clocks in the liver, gastrointestinal tract, and adipose tissue. Time-restricted eating (TRE) studies demonstrate that caloric intake misaligned with the light-dark cycle — as in shift workers and night-eaters — disrupts peripheral clock gene expression and dysregulates ghrelin-leptin signalling, with downstream effects on the gut-brain axis.⁶ Social zeitgebers, including scheduled social interactions and work routines, stabilise circadian rhythms through a process termed social synchrony. Social isolation, as witnessed during the COVID-19 pandemic lockdowns, precipitated widespread circadian disruption and a parallel surge in anxiety disorders, providing a natural experiment illustrating the psychiatric consequences of social zeitgeber deprivation.⁷

CIRCADIAN DYSREGULATION IN GAD: NEUROBIOLOGICAL MECHANISMS:

1. HPA AXIS HYPERACTIVATION-

Circadian control of the hypothalamic-pituitary-adrenal (HPA) axis is critical for appropriate stress responsivity. Clock gene mutations in murine models produce HPA axis hyperactivity, elevated basal corticosterone, and anxiety-like behaviours in open-field and elevated plus-maze tests.⁸ In clinical studies, patients with GAD demonstrate a blunted cortisol awakening response (CAR) alongside elevated evening cortisol — a pattern consistent with HPA axis dysregulation that parallels chronodisruption. Chronic zeitgeber disruption may therefore perpetuate a state of neuroendocrine allostatic overload, sustaining the worry-arousal cycle central to GAD.

2. AMYGDALA HYPERREACTIVITY AND PREFRONTAL HYPOACTIVATION-

The amygdala — the brain's primary threat-detection centre — expresses robust circadian rhythms in neuronal excitability and synaptic plasticity. Studies using functional MRI demonstrate that amygdala reactivity to emotional stimuli is significantly greater in the evening (when melatonin is rising) than in the morning, and this pattern is amplified by sleep deprivation and circadian misalignment.⁹ GAD is characterised by sustained amygdala hyperactivation and attenuated prefrontal cortex (PFC) top-down inhibition. Circadian disruption may exacerbate this imbalance by differentially degrading PFC clock-dependent functions — including emotion regulation, cognitive flexibility, and extinction learning — while sensitising amygdala threat circuitry.

3. SEROTONIN-MELATONIN AXIS DYSREGULATION-

Serotonin (5-HT), the precursor to melatonin, demonstrates a distinct diurnal rhythm with daytime peaks in the dorsal raphe nucleus. Zeitgeber disruption impairs serotonergic transmission, reducing 5-HT availability for both daytime mood regulation and nocturnal melatonin synthesis. The resulting melatonin deficiency — documented in numerous GAD studies — impairs sleep architecture, diminishes anxiolytic GABAergic tone (melatonin enhances GABA-A receptor function), and exacerbates anticipatory anxiety.¹⁰ Notably, the melatonin receptor agonist agomelatine has demonstrated efficacy in GAD, directly implicating the melatonin-serotonin axis as a therapeutic target.

4. GUT MICROBIOME-CIRCADIAN-ANXIETY INTERFACE-

The gut microbiome demonstrates diurnal compositional oscillations that are entrained by feeding zeitgebers and in turn modulate host circadian gene expression through microbial metabolites including short-chain fatty acids (SCFAs) and tryptophan metabolites. Circadian disruption via irregular feeding or light exposure reduces microbial diversity, diminishes SCFA production, impairs intestinal barrier integrity, and elevates systemic lipopolysaccharide (LPS) — triggering neuroinflammation via the vagal-gut-brain axis. Emerging evidence links this dysbiotic state to heightened anxiety behaviour, suggesting a tripartite zeitgeber-microbiome-anxiety circuit that merits further clinical investigation.¹¹

CLINICAL EVIDENCE: CIRCADIAN MARKERS IN GAD:

Several clinical studies have documented objective circadian dysregulation in GAD patients. Harvey et al. (2011) reported significantly delayed DLMO in anxiety disorder patients compared to healthy controls, correlating with greater sleep onset latency and higher worry scores.¹² A large cohort study

(n=32,470) by Lyall et al. (2018) found that individuals with circadian preference misalignment (evening chronotype with daytime obligations) had 94% higher odds of mood disorder diagnosis, including GAD, independent of sleep duration.¹³

Actigraphy studies in GAD patients consistently reveal increased intraday variability (IV) and reduced interdaily stability (IS) in rest-activity rhythms — quantitative indices of circadian fragmentation.¹⁴ These objective markers correlate with self-reported worry severity and poor emotion regulation, suggesting that circadian fragmentation may be both a biomarker and mechanistic contributor to GAD symptomatology. Polysomnographic studies further demonstrate reduced slow-wave sleep (SWS) and increased REM density in GAD, consistent with disrupted sleep-stage architecture driven by circadian-sleep process misalignment.

CHRONOTHERAPEUTIC IMPLICATIONS FOR GAD MANAGEMENT:

Recognition of zeitgeber dysregulation as a pathogenic factor in GAD opens several evidence-based therapeutic avenues:

LIGHT THERAPY: Morning bright-light exposure (2500–10,000 lux, 30 minutes) advances the circadian phase, suppresses ALAN-induced melatonin disruption, and enhances serotonergic tone. Controlled trials in mood and anxiety disorders report significant anxiolytic effects with excellent tolerability.¹⁵

SLEEP-WAKE SCHEDULING: Consistent sleep and wake times, even on weekends (reduced social jetlag), stabilise SCN entrainment. Cognitive Behavioural Therapy for Insomnia (CBT-I), which incorporates stimulus control and sleep restriction, has demonstrated efficacy in comorbid GAD-insomnia presentations.

TIME-RESTRICTED EATING (TRE): Aligning caloric intake with the active phase (typically a 8–10 hour daytime eating window) optimises peripheral clock synchrony and has shown benefits for anxiety-related biomarkers in early human trials.¹⁶ Melatonin and Melatonin Receptor Agonists: Low-dose melatonin (0.5–3 mg) timed to DLMO can phase-advance delayed rhythms. Agomelatine (MT1/MT2 agonist and 5-HT_{2C} antagonist) has demonstrated superiority over placebo in GAD in randomised controlled trials, with added advantages of improved sleep architecture and minimal metabolic side effects.¹⁰

PHYSICAL ACTIVITY AS ZEITGEBER: Morning aerobic exercise serves as a non-photic zeitgeber, advancing circadian phase and increasing BDNF and serotonin synthesis. Meta-analyses confirm moderate aerobic exercise reduces GAD symptom severity with effect sizes comparable to pharmacotherapy.¹⁷ Digital Zeitgeber Hygiene: "Blue light hygiene" practices — including use of blue-light-blocking spectacles after sunset, activating night-mode displays, and reducing screen exposure 2 hours before bedtime — represent accessible, low-cost public health interventions to preserve melatonin rhythmicity.

LIMITATIONS AND FUTURE DIRECTIONS:

The current evidence base, while compelling, is predominantly from animal models and observational human studies. Causality between zeitgeber dysregulation and GAD incidence requires confirmation through prospective longitudinal cohorts and interventional chronotherapy trials with active comparators. Standardised circadian phenotyping using DLMO, actigraphy, and circadian transcriptomic profiling (via time-of-day gene expression in blood) should be incorporated into GAD research protocols to advance precision chronotherapeutics. Additionally, the role of genetic chronotype variants (e.g., PER3 VNTR polymorphisms) in modulating vulnerability to zeitgeber-induced GAD deserves systematic investigation. The emerging field of chrono-pharmacology — timing drug administration according to circadian biology — may optimise the efficacy of existing anxiolytics and warrants dedicated clinical investigation.

CONCLUSION:

Zeitgeber dysregulation emerges as a significant, yet under-recognised, dimension of GAD pathophysiology. Through disruption of SCN entrainment, HPA axis rhythmicity, amygdala-PFC balance, serotonin-melatonin signalling, and the gut-brain axis, impaired time-giver signals create neurobiological conditions conducive to chronic anxiety. The convergence of circadian neuroscience and clinical psychiatry opens a promising new frontier for GAD management — one that complements existing pharmacological and psychological approaches with targeted chronotherapeutic strategies. Integrating zeitgeber assessment and circadian restoration into routine psychiatric practice may represent an important step toward more holistic, mechanism-based care for GAD patients.

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Creative Corner

Creative Corner



THE SHIFT FROM PCOS TO PMOS: A NEW ERA IN UNDERSTANDING WOMEN'S ENDOCRINE HEALTH

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There are moments in medicine when a single reframing changes everything — not because the disease changed, but because our understanding finally caught up with the patient.

For decades, Polycystic Ovarian Syndrome has carried its name like a verdict: *polycystic*, evoking images of ovaries studded with cysts, of something structurally wrong, something visibly broken. Millions of women were handed this label along with a prescription, a diet chart, and — often — a quiet resignation that their hormones were simply “out of order.”

But science, when it moves, moves beautifully.

The emerging concept of Polycystic Morphology of Ovarian Syndrome (PMOS) — or more broadly, the growing push to rename and reconceptualize PCOS — is one such movement. The Rotterdam criteria already acknowledged that polycystic *ovarian morphology* (PCOM) is just one feature among many, not the defining lesion. The real story, researchers now argue, is far more upstream: dysregulated GnRH pulsatility, exaggerated androgen biosynthesis, and insulin-driven disruption of the hypothalamic-pituitary-ovarian axis. The ovaries, it turns out, are less the cause and more the canvas.

This is not merely semantic housekeeping. When we call it a *syndrome of cysts*, we inadvertently anchor clinical thinking — and patient identity — to morphology. Women without visible cysts are dismissed; those with cysts but no metabolic features are over-treated. The name shapes the narrative, and the narrative shapes care.

The proposed shift acknowledges what physiology has long whispered: that this condition is fundamentally one of neuroendocrine dysregulation, with the ovary as a downstream responder. It invites us to look at the hypothalamus, the adrenal axis, insulin signaling, and even the gut microbiome as co-conspirators rather than bystanders.

For those of us who teach physiology, this moment carries a particular weight. Our students are learning today what they will practice for the next forty years. If we hand them only the old map — cysts, androgens, anovulation — we limit the territory they can explore. But if we teach them to ask *why the axis misfires, why insulin amplifies androgen synthesis at the theca cell, why stress and sleep deprivation can phenocopy PCOS* — we give them something far more powerful: a physiological imagination.

The women sitting across from future clinicians deserve more than a syndrome named for what their ultrasound showed on one Tuesday morning. They deserve a diagnosis that speaks to the full orchestration of their endocrine system — and a medical community willing to keep revising the score.

ONDINE'S CURSE: DON'T FORGET TO BREATHE!

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Isn't it quite interesting that we humans are totally unaware about breathing, the very act on which our survival actually depends? Well, this is because our brain's respiratory centers are "hard-wired" in such a way that normal breathing is mainly under subconscious, involuntary control, unless of course we decide to "consciously focus" our attention towards it, for example, during breathing exercises, playing wind instruments or when taking deep breaths.....But what if we actually had to "always remember" to breathe, or so as to say "make conscious efforts" to maintain normal breathing? This is what precisely happens in patients of this unique medical condition called "Ondine's Curse" or "Central Hypoventilation Syndrome" in which the automatic breathing control system breaks down. This unique name owes its origins to a famous Greek legend about a water nymph and her curse.....

Once upon a time, there was a King of water nymphs, who ruled over the underwater realm of a deep, dark lake situated near a dense forest. He had a beautiful little daughter called Ondine. The king was under constant worry for he and his kinsmen did not possess a soul. It was thus his most fateful wish to get a "soul" for his lovely daughter by any means necessary. He had a brother named Kulheborn who was a sorcerer and practiced the forbidden, dark arts.

And so the King, driven by his intense longing to beget a soul for his lovely daughter, sought the advice of his sorcerer brother who told him, *"the princess shall beget a soul only upon marital conjugation with a mortal human"*. Hearing thus, the King decided to send his little girl into the realm of the mortals above. There she was adopted by a fisherman who had found her stranded by the lake one day and had taken pity on her. It so happened that the fisherman and his wife had lost their daughter in this very lake mysteriously many years back. The overjoyed couple thanked the lord for blessing them with another daughter.

Gradually the little princess grew up into a beautiful young damsel, under the love and care of her foster parents. One day a handsome prince, Count Huldbrand of Ringstetten was out hunting in the forest and ventured deep while chasing a ferocious beast. As he stopped by that very lake, thinking about his predicament, he saw a lovely lady sitting there. It was love at first sight for both of them. But little did they both know that this was not some favorable twist of fate, but was the design of Ondine's uncle Kulheborn, who was driven by his brother's desire.

And so it happened that both Ondine & Count Huldbrand got married amidst much fanfare, but alas this romantic tale was not meant to end with *"they lived happily ever after"*. Ondine soon discovered that her husband was having a secret affair with a beautiful maiden named Bertalda. Ondine was deeply hurt with Count Huldbrand upon discovering his infidelity and lamented her misery to her father. The king was furious and summoned his brother Kulheborn to punish the Count. Kulheborn was about to put a death spell but Ondine begged her uncle to mitigate the curse as she was still in love with the Count. So Kulheborn modified the curse and pronounced to the Count, *"As you had undertaken the sacred vow upon the marital altar to love Ondine till your last breath and to die, if you should ever forget her. So let this be known to all angels, demons, mortals and spirits that from this very moment, you shall live as long as you remember to breathe."* The terrified Count begged for forgiveness but Kulheborn refused. And

from that day started a daily ordeal where the Count had to remain continuously awake and remind himself to breathe. He eventually fell asleep one day from sheer exhaustion and his respiration stopped. The story of Ondine's curse ends here but the legend remains alive to this day in medical literature as Ondine's curse (or Central Hypoventilation Syndrome).

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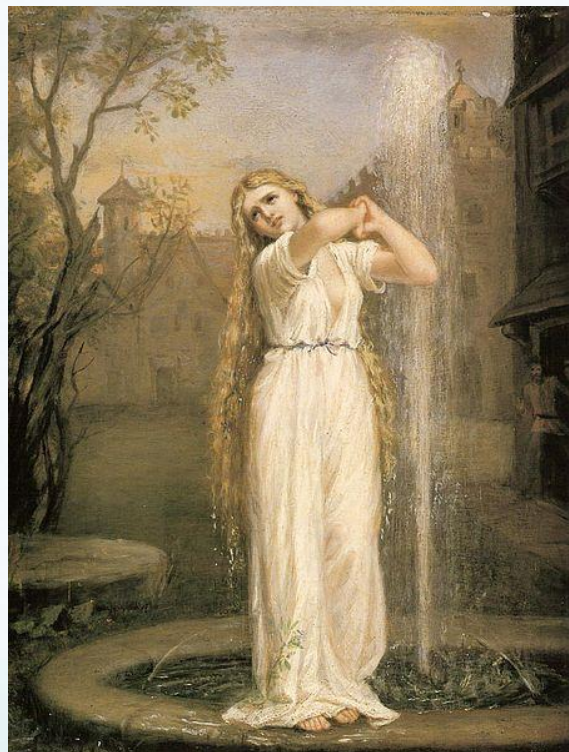


Image: Undine...oil on canvas by John William Waterhouse

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THE WHITE COAT

VAIBHAV TIWARI, JNMC, 1ST YEAR

They called him God in days gone by,
With folded hands and hopeful eyes.
A healer standing by the bed,
Giving strength where hope had fled.

Through sleepless nights and endless pain,
He fought for lives again and again.
Missing moments, missing rest,
Giving his all, giving his best.

But times have changed, the world moved on,
The old respect is nearly gone.
A life is lost, a finger points,
And trust begins to break at joints.

People forget the tears he hides,
The weight of grief he keeps inside.
For every patient he could not save,
Another sorrow he must brave.

He is not God, he never was,
Just a human, just like us.
A beating heart beneath the coat,
A silent prayer in every note.

So judge him kindly when things go wrong,
His road is hard, his days are long.
For even now, through joy and strife,
He spends his own to save a life.

WHY HOBBIES MATTER: THE SCIENCE OF STAYING HAPPY, HEALTHY AND SHARP

DR. SHALINI JAIN AGRAWAL (M.S. OBGY, FICOG, FMAS, CIMP)

In the rush of modern life, we often treat hobbies as optional extras, something to do once the “real work” is done. But growing evidence tells a different story. What we choose to do in our leisure time may be one of the most powerful investments we make in our long-term health and happiness.

The Evidence

A landmark Harvard study involving more than 93,000 adults across 16 countries found that people who regularly engaged in hobbies consistently reported better health, higher life satisfaction and lower rates of depression. The benefits held true across age, culture and income. In short, hobbies are a global prescription for well-being.

How Hobbies Shape the Brain

When we learn a new skill, paint, play an instrument or even tend to a garden, the brain forms fresh connections. This ability, known as neuroplasticity, helps the brain stay flexible and strong. Such activities stimulate the hippocampus, the center of memory and learning, and over time help build cognitive reserve which is the brain’s buffer against aging and decline.

People who keep their minds active through creative or mentally stimulating hobbies have a significantly lower risk of dementia and cognitive impairment. In simple terms, hobbies keep the brain youthful and resilient.

How the Body Responds

Hobbies also work through the body’s built-in relaxation system, led by the vagus nerve. When we immerse ourselves in enjoyable or calming activities like music, painting, gardening or yoga, the vagus nerve signals the body to slow down and restore balance. This reduces heart rate and blood pressure, lowers inflammation, and supports healthy hormone levels. Regular activation of this pathway strengthens emotional stability and physical resilience.

The Chemistry of Joy

Pleasurable activities release dopamine and serotonin, the brain’s natural mood boosters. These chemicals lift motivation, sharpen focus and counteract cortisol, the stress hormone. Over time, this biochemical balance improves sleep, stabilizes mood and protects mental health. Many people describe reaching a “flow state” during hobbies. It’s that effortless concentration where stress fades and creativity thrives. It is both soothing and deeply restorative.

The Power of Connection

Hobbies that involve others add another layer of benefit. Joining a group, class or team nurtures connection and belonging, both of which are critical for emotional and physical health. Social engagement has been shown to protect against loneliness, which is now recognized as a serious health risk comparable to smoking or obesity. Shared activities build confidence, empathy and a sense of purpose- all of which are things every mind needs to stay well.

A Habit Worth Keeping

The most powerful hobby is the one that brings you joy. Choose something that challenges you just enough to stay interesting, and make it a regular part of your life. It is never too late to begin. The human brain remains capable of growth and adaptation at every age.

In Summary

Hobbies are not distractions from life; they are the heartbeat of a healthy one. They sharpen the mind, calm the body and open doors to connection and creativity. Whether through music, movement or mindful creation, a hobby is one of the simplest and most effective ways to invest in your future well-being.

AWAKENED

BY AKALAVYA KUMAR, MBBS 2025 BATCH
JNMC AMU



BY DR. SHIVANI VERMA
SENIOR RESIDENT, DEPARTMENT OF PHYSIOLOGY
ASMC AMETHI UP



Moments in Motion

Moments in Motion



PHYSIOLOGY DISTILLED: ASSOPI UP CHAPTER WEBINAR SERIES (APRIL – JUNE HIGHLIGHTS)

21 APRIL 2026

SPEAKER: DR. CHARUSHILA

TOPIC: Reversing PCOS Through Lifestyle Medicine: A Physiology-Based Approach.

The webinar featured Dr. Charushila, Assistant Professor, Department of Physiology, AIIMS Gorakhpur, who delivered an informative lecture on "Reversing PCOS Through Lifestyle Medicine: A Physiology-Based Approach."

The session highlighted the physiological basis of polycystic ovary syndrome, emphasizing the role of lifestyle modifications, including nutrition, exercise, stress management, and sleep, in improving metabolic and reproductive health. The webinar enhanced the understanding of evidence-based lifestyle interventions in PCOS.

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Webinar series of ASSOPI- UP Chapter

**TOPIC : Reversing PCOS Through Lifestyle Medicine: A
Physiology-Based Approach**

21st April 2026 Tuesday | 03:00 PM Onwards

Speaker



Dr. Charushila
Assistant Prof., Dept. of Physiology,
AIIMS GORAKHPUR.

President



Dr. Shraddha Singh
Prof. & Head, Dept. of
Physiology, KGMU, Lko.
President, ASSOPI- U P
Chapter

Secretary



Dr. Amit Kant Singh
Prof. & Head, Dept. of
Physiology, UPUMS, Saifai
Secretary, ASSOPI- U P
Chapter

Convenor



Dr. Shivam Verma
Assistant Professor,
Dr. RMLIMS, Lko
Assistant Secretary, ASSOPI
UP Chapter

Moderator



Dr. Khushboo Singhal
Assistant Professor,
Subharti Medical College,
Meerut

19 MAY 2026

SPEAKER: DR. AYESHA JUHI

TOPIC: The Default Mode Network: A Physiological Bridge Between Internal Awareness and Cognitive Function

The webinar was delivered by Dr. Ayesha Juhi, Additional Professor and Head, Department of Physiology, AIIMS Deoghar, on the topic "The Default Mode Network: A Physiological Bridge Between Internal Awareness and Cognitive Function."

The session provided an insightful overview of the Default Mode Network, its neurophysiological basis, and its significance in cognition, self-awareness, memory, and mental health. The session also explored recent advances in neuroscience and their implications for clinical practice and research. Participation in this webinar broadened the understanding of higher brain functions

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**TOPIC : The Default Mode Network: A Physiological Bridge
Between Internal Awareness and Cognitive Function**

19th May 2026 Tuesday | 03:00 PM Onwards

Speaker



Dr. Ayesha Juhi
Additional Professor and HoD, Dept. of
Physiology, AIIMS Deoghar, Jharkhand

President



Dr. Shraddha Singh
Prof. & Head, Dept. of
Physiology, KGMU, Lko.
President, ASSOPI- U P
Chapter

Secretary



Dr. Amit Kant Singh
Prof. & Head, Dept. of
Physiology, UPUMS, Saifai
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Chapter

Convenor



Dr. Shivam Verma
Assistant Professor,
Dr. RMLIMS, Lko
Assistant Secretary, ASSOPI
UP Chapter

Moderator



Dr. Khushboo Singhal
Assistant Professor, Subharti
Medical College, Meerut.
Joint Secretary, ASSOPI
UP Chapter

23 JUNE 2026

SPEAKER: DR. ARVIND KUMAR PAL

TOPIC: Pathophysiological Mechanism Underlying Secondary Hypertension

The scientific session was delivered by Dr. Arvind Kumar Pal, Associate Professor, Department of Physiology, Dr. RMLIMS, Lucknow, on "Pathophysiological Mechanism Underlying Secondary Hypertension."

The lecture comprehensively covered the physiological and pathophysiological mechanisms responsible for secondary hypertension, including endocrine, renal, vascular, and neurological causes, along with their clinical implications. The webinar provided valuable insights into recent developments in hypertension research and reinforced the importance of understanding disease mechanisms for effective diagnosis and management.

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Webinar series of ASSOPI- UP Chapter

**TOPIC : Pathophysiological mechanism underlying
Secondary Hypertension**

23rd June 2026 Tuesday | 03:00 PM Onwards

Speaker
Dr. Arvind Kumar Pal
Associate Professor, Dept. of Physiology,
Dr. RMLIMS, Lko

President
Dr. Shraddha Singh
Prof. & Head, Dept. of
Physiology, KGMU, Lko.
President, ASSOPI- U P
Chapter

Secretary
Dr. Amit Kant Singh
Prof. & Head, Dept. of
Physiology, UPUMS, Saifai
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Dr. RMLIMS, Lko
Assistant Secretary, ASSOPI
UP Chapter

Moderator
Dr. Khushboo Singhal
Assistant Professor, Subharti
Medical College, Meerut.
Joint Secretary, ASSOPI
UP Chapter

ANNUAL CME & ALUMNI MEET: "INTEGRATION OF ARTIFICIAL INTELLIGENCE IN CONTEMPORARY MEDICAL RESEARCH & EDUCATION"

An Annual CME & Alumni Meet was organized under the aegis of the ASSOPI UP Chapter by the Department of Physiology, Dr. Ram Manohar Lohia Institute of Medical Sciences (Dr. RMLIMS), Lucknow, on 13th May 2026. The theme of the CME was "Integration of Artificial Intelligence in Contemporary Medical Research & Education."

The scientific program comprised expert lectures on the role of Artificial Intelligence in medical education and research. Prof. (Dr.) R. K. Garg delivered an insightful session on the importance of AI, highlighting its advantages, limitations, and future applications in healthcare. Prof. (Dr.) Sandeep Bhattacharya discussed how AI is transforming learning methodologies and enhancing the educational experience for medical students and faculty.

The CME provided valuable insights into the evolving role of artificial intelligence in academics, research, and clinical practice. Participation in this academic event enhanced my understanding of emerging technologies in medical education and encouraged the integration of innovative digital tools into teaching, research, and professional development.



Call for Articles

Physiology Insight

(Vol 2, Issue 3 | July – Sept 2026)

ASSOPI–UP Chapter invites submissions for the upcoming newsletter.

Sections:

- Tribute of Gratitude & Grace
- From the Editor's Lens
- Scientific Articles (≤ 3000 words)
- Creative Corner
(poems/stories/photos/art)
- Moments in Motion
- Achievements
- Student Contributions

Guidelines:

- Word file, TNR 12, spacing 1.5
- Max 2 images
- Vancouver references
- Plagiarism $< 20\%$
(iThenticate/Turnitin)

Submit to: newsletterassopiup@gmail.com

Deadline: 31st August 2026

Contact:



• Dr. Tanuj Mathur – 9411654777



• Dr. Khushboo Singhal – 9457290310 •

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