LINK-ISSUE 9

JULY 2026

LINK

THE OFFICIAL NEWSLETTER OF NEUROCRITICAL CARE SOCIETY OF INDIA

EDITOR'S NOTE

Dear Members,

It gives us great pleasure to present this issue of the Neurocritical Care Society of India Newsletter. Our specialty continues to expand in depth & scope — from trauma neurocritical care to advanced haemorrhage management, from postoperative neurosurgical care to complex neurological emergencies. Each of you contributes to this growth through your practice, your teaching, & your commitment to improving outcomes for critically ill neurological patients.

Neurocritical care today stands at a unique intersection of technology, physiology, and teamwork. The advances we see — in multimodal monitoring, new generation antibiotics & diagnostic tools, cerebral haemorrhage pathways, & structured ICU protocols — are reshaping how we care for patients and how we measure success.

We encourage you to browse through the editorial sections, clinical updates, & shared experiences. It is not only a scientific resource but also a social platform that keeps our community connected and inspired.

On a personal note, We wish each of you strength, growth, & fulfilment in your practice. Our society thrives because of your contributions. I warmly invite you to share your case reports, algorithms, protocols, and short articles for future issues — your voice strengthens our collective learning.

With warm regards,
Dr. Sindhu Priya. M & Dr. Mathangi. K

FROM THE PRESIDENT'S DESK

DR. BIBHUKALYANI DAS

Dear colleagues, friends and members of entire NCSI family,

Greetings from the office of the President !!

Another remarkable year in the journey of the Neurocritical Care Society of India (NCSI) reminds us how far we have come and how much more we can achieve together. What began as a shared vision has grown into a vibrant academic community, driven by innovation, collaboration, and an unwavering commitment to advancing neurocritical care across the country.

A major milestone this year has been the launch of post MBBS Neurocritical Care Certificate Course (PDCC) along with post doctoral Fellowship (PDF) programmes at Kauvery Hospital, Chennai, and Institute of Neurosciences Kolkata, strengthening structured training for future neurointensivists. NCSI has also expanded its academic footprint through active panel representations and collaborations with national critical care societies.

We are now 355 members and I encourage each and every member to remain actively involved to participate in our programs, contribute their expertise and support the growth of our shared mission. Our educational initiatives continue to flourish.

I must congratulate Dr. Ashish Bindra, lead E-Education NCSI for this. The Acute Neurocare Course remains a flagship programme, while our YouTube channel has grown to nearly 2,000 subscribers with 58 educational videos, extending quality learning to healthcare professionals nationwide.

This newsletter reflects the breadth of our Society's academic interests, bringing together thought-provoking articles on neurocritical care alongside perspectives on physician well-being through yoga and mindfulness. The enthusiastic contributions from both experienced consultants and residents exemplify the collaborative spirit and intellectual vitality of NCSI. Needless to emphasise, the sincere efforts of our talented editors Dr. Sindhu Priya.M and Dr. Mathangi Krishnakumar.

As we look forward to the 7th Annual Conference at Taj Taal Kutir, Kolkata, I warmly welcome colleagues from across the country to what promises to be an enriching scientific meeting. I am confident that the meeting will provide an outstanding opportunity for scientific exchange, professional networking, and collaborative learning. We also assure warm Kolkata hospitality with personal touch to each and every participant.

It is really a pleasure to serve the society as President, NCSI. I sincerely thank our members and office bearers for their dedication and cooperation. Together, let us continue to innovate, collaborate, and strive for excellence in neurocritical care.

Striving today for a stronger Neurocritical Care tomorrow!

Long live NCSI.

FROM THE SECRETARY'S DESK: A JOURNEY OF COLLECTIVE GROWTH & RESILIENCE

Dear Friends and Colleagues,

Serving as the Secretary of the Neurocritical Care Society of India (NCSI) for the past three years has been both a profound privilege and an incredibly rewarding journey. As my tenure draws to a close, I look back with immense gratitude for the trust you placed in me and the unwavering support this vibrant community has extended at every step.

Our shared vision has always been to elevate the standards of neurocritical care across the nation, bridging the gaps between neuro-anaesthesia, neurology, neurosurgery, and intensive care.

Over the last three years, thanks to the collaborative spirit of our executive team and the dedication of our members, we have turned challenges into milestones.

Here is a reflection on what we have achieved together:

1. Expanding Educational Horizons & Academic Rigor

-**Structured Training:** We successfully strengthened our fellowship programs and training modules, ensuring that our junior doctors and residents have access to robust, evidence-based training. We now have PDFC Programs in three hospitals, Kauvery Chrnnai INK KOLKATA, St John's , Bengaluru and post MBBS certificate program at INK Kolkata.

-**Webinars and CMEs:** Established regular high-impact webinars and interactive CMEs, increasing knowledge and popularity, especially on YouTube, thanks to the education committee's efforts.

2. Scientific Collaborations & Standardizing Care

This year for the first time we collaborated with ISCCM and SNCC in their national conferences and tried to bridge the gap between the societies for the greater good for our subspecialty.

3. Community Engagement & Membership Growth

-**Strengthening Our Base:** Over the last three years, our membership has grown by almost 50 percent.

-**Annual Conferences:** Our national conferences served as incredible melting pots of ideas, featuring stellar international faculties, high-quality free paper sessions, and hands-on simulation workshops that pushed the boundaries of our clinical practice.

"'No single milestone belongs to an individual; every success we celebrated over these three years is a testament to our collective dedication to the critically ill neurological patient."

With Deepest Gratitude

I owe a massive debt of thanks to our President, the past leadership, and my fellow Executive Committee members. Your mentorship, critique, and camaraderie kept the wheels turning seamlessly. To our secretarial staff and the editorial team of the newsletter—thank you for your tireless behind-the-scenes work.

Most importantly, thank you to the general body members. Your active participation in our initiatives and your dedication at the bedside are what truly define the soul of NCSI.

As I hand over the baton to the incoming secretariat in the upcoming confetence, I do so with great optimism. I am confident that the new leadership will steer the society to even greater heights.

While my official role ends, my commitment to NCSI and to the advancement of our specialty remains unchanged. I look forward to meeting you all at our upcoming conference in Kolkata from the other side of the podium.

Wishing the NCSI family continued success, good health, and academic excellence.

Long live NCSI.

Treasurer REPORT

Dear Colleagues and Friends,

As we enter the second half of the calendar year, July offers an appropriate opportunity to review our progress, assess emerging priorities, and refine our plans for the months ahead.

Financially, the Society continues to remain stable and responsibly managed. Prudent stewardship during the first half of the year has enabled us to sustain our core activities while gradually strengthening our capacity to support member-focused initiatives. Maintaining this financial discipline remains essential as we invest in areas of long-term value, particularly research support, educational programmes, and trainee engagement.

A continuing strength of NCSI has been the sustained performance of our Acute Neuro Care (ANC) courses, which remain academically relevant, well received, and financially viable. Their success reflects the commitment of our faculty, organisers, and participants, and reinforces the Society's important role in advancing structured education in neurocritical care.

As we move forward into the remainder of the year, I extend my sincere appreciation to all members for their continued trust, participation, and support. With sustained collaboration, thoughtful planning, and prudent financial management, NCSI remains well positioned to pursue its objectives with confidence and purpose.

With best wishes,

DR. KESHAV GOYAL

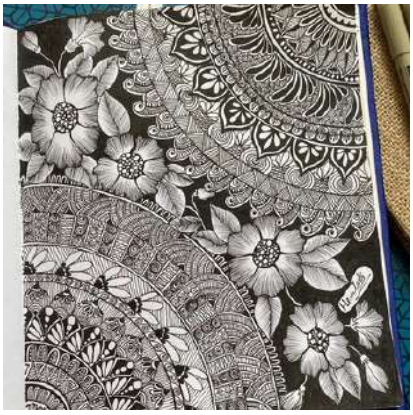
SECRET SUPERPOWER

Meet Dr. Hemalata, a creative whirlwind who doodles her way through downtime! What began as a casual pastime has blossomed into a full-blown passion, and she's even gearing up to showcase her masterpieces someday. She's got a knack for sketching portraits and intricate pencil art. Plus, she's completely enchanted by mandala and zentangle designs, diving into their mesmerizing patterns with enthusiasm.
Feast your eyes on her diverse art forms showcased here—get ready for a visual delight!

P.S. Her stunning artwork graces our cover page too!

Dr. Hemalata,
Professor,
King George Medical University, Lucknow










Evoked Potentials in Critical Care: A Contemporary Approach to Neurophysiological Monitoring

DR. VISHWANATH BHAIRE
LEAD CONSULTANT,
YASHODA HOSPIATAL,
SECUNDERABAD

The electrodes of evoked potentials "catch" your brain's and nerves' electrical signal reponses to the stimulus.

Evoked Potentials *in Critical Care*

A contemporary approach to neurophysiological monitoring — physiology, clinical applications, and the evolving role of EPs in multimodal neuroprognostication.

INTRODUCTION

Evoked potentials (EPs) are electrophysiological responses generated within the nervous system following sensory or motor stimulation. By assessing the functional integrity of specific neural pathways, they provide an objective bedside tool for neurological evaluation in critically ill patients. In the ICU — where clinical examination is frequently limited by sedation, mechanical ventilation, metabolic disturbance, or impaired consciousness — EPs remain valuable adjuncts for localization, monitoring, and prognostication.

Although neuroimaging and continuous EEG have transformed modern neurocritical care, EPs retain clinical relevance because they are **non-invasive, reproducible, and relatively resistant to sedative effects** compared with the routine neurological examination. Among available modalities, somatosensory evoked potentials (SSEPs) remain the most clinically validated in critical care, particularly for neuroprognostication following cardiac arrest.

PHYSIOLOGICAL BASIS

EPs are low-amplitude signals often obscured by spontaneous background EEG and environmental noise; repeated stimuli are therefore averaged to extract reproducible waveforms. Responses are characterized by **polarity** — negative [N] or positive [P] — and **latency**, the time in milliseconds from stimulus onset.

CLINICAL FRAMEWORK – THREE QUESTIONS

Is the neural pathway intact? Is neurological function stable, improving, or deteriorating? And can these findings inform prognosis? This framework is especially valuable when impaired consciousness makes EPs a source of objective data independent of patient cooperation.

EP MODALITIES AT A GLANCE

SSEP
Somatosensory Evoked Potentials

Median / posterior tibial nerve stimulation; assesses dorsal columns, medial lemniscus, thalamus, cortex. The cortical **N20** is the cornerstone waveform.

BAEP
Brainstem Auditory Evoked Potentials

Click-evoked; assess auditory nerve and brainstem auditory pathways. Useful for brainstem integrity.

VEP
Visual Evoked Potentials

Retina-to-occipital conduction. Limited ICU use — technical constraints, sedation, ocular pathology.

MEP
Motor Evoked Potentials

Corticospinal tract via transcranial stimulation. Chiefly intraoperative; selective ICU application.

Bottom line: SSEPs and BAEPs are the two modalities most relevant to everyday critical care practice.

Key Features of EPs

OBJECTIVE
Not dependent on patient cooperation

PATHWAY-SPECIFIC
Assesses functional integrity at multiple levels

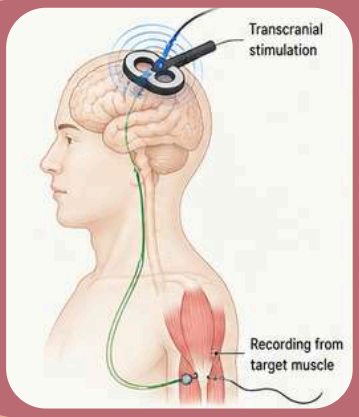
RESISTANT TO SEDATION
Less affected by sedatives than clinical exam or EEG

Neurological assessment in critically ill patients is often challenging.

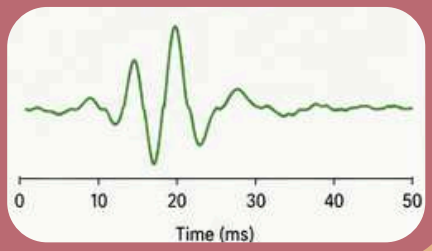
EPs provide an objective, non-invasive assessment of neural pathway integrity

Waveforms are characterized by their polarity (+ve/-ve) and latency (response time)

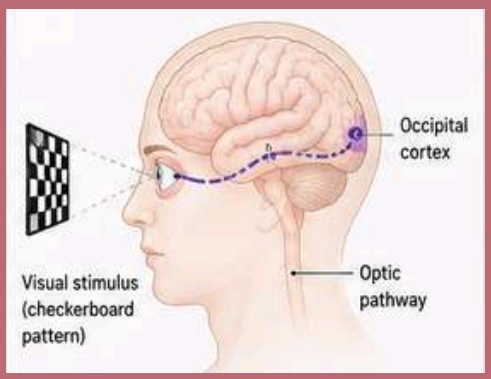
TYPES OF EPs

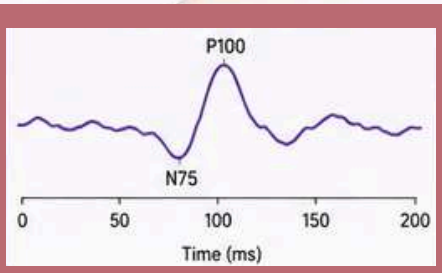


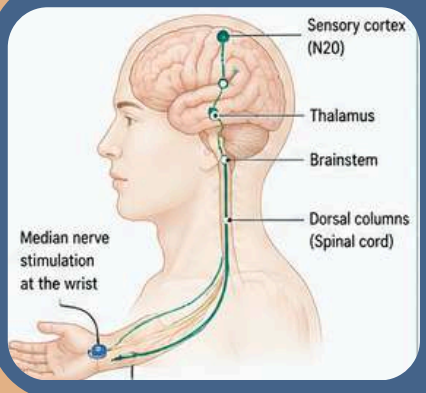
MOTOR EVOKED POTENTIALS (MEPs)
Assesses corticospinal tract



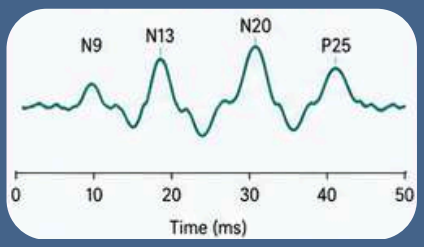
VISUAL EVOKED POTENTIALS (MEPs)
Evaluates visual pathway function

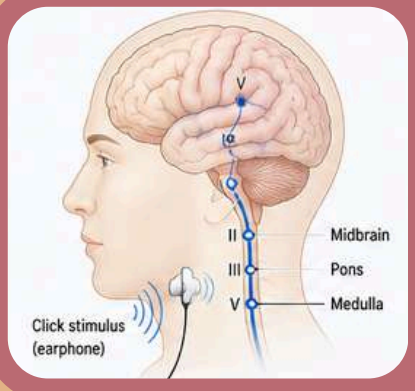




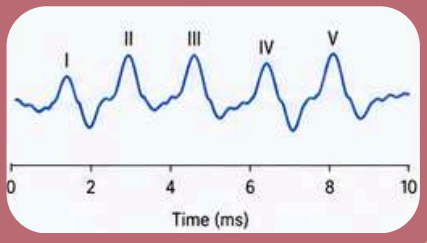


SENSORY EVOKED POTENTIALS (MEPs)
Measures sensory pathway conduction





BRAINSTEAM AUDITORY EVOKED POTENTIALS (MEPs)
Tests auditory nerve and brainstem



SSEPs — the critical-care workhorse

SSEPs are the most established EP modality in the ICU owing to their reproducibility, technical feasibility, and relative resistance to sedatives. Their greatest utility lies in prognostication after hypoxic–ischemic brain injury.

1 Post–cardiac arrest neuroprognostication

Bilateral absence of cortical N20 responses to median nerve stimulation, with preserved peripheral and cervical responses, is among the most robust predictors of poor neurological outcome after arrest, with extremely high specificity. The signal is **asymmetric**: absent N20 strongly suggests severe irreversible cortical injury, but preserved responses do not guarantee favorable recovery. Quantitative N20–P25 amplitude may add prognostic information beyond the present-versus-absent paradigm.

2 Traumatic brain injury

In severe TBI, SSEPs assess thalamocortical conduction and may flag secondary deterioration; serial worsening can track cerebral edema or rising ICP. Unlike post-arrest coma, the prognostic role here is less standardized and should not be read in isolation.

3 Subarachnoid haemorrhage

In poor-grade aSAH, SSEPs may aid prognostication and serial monitoring of secondary injury. Combined EP-based scores incorporating SSEPs and BAEPs show promise for prognostic stratification.

4 Spinal cord & brainstem pathways

Useful in selected patients with suspected spinal cord ischemia, dorsal column dysfunction, or postoperative deterioration after spine or vascular surgery.

KEY POINT

Interpret SSEPs within a **multimodal** prognostic framework — never as an isolated predictor of outcome or withdrawal decisions.

BAEPS — ASSESSING BRAINSTEM INTEGRITY

BAEPs offer a practical readout of brainstem conduction when examination is limited or confounded. Because they evaluate the auditory nerve and pontomesencephalic pathways, they are useful in suspected posterior-circulation stroke, pontine lesions, transtentorial herniation, brainstem encephalitis, and severe disorders of consciousness. Prognostic utility after arrest is less established than SSEPs, but BAEPs add complementary information — and, with middle-latency AEPs, may improve accuracy in indeterminate disorders of consciousness.

VEPS & MEPS — SELECTIVE, EMERGING ROLES

VEPs have limited routine adult ICU use given technical limits, sedation sensitivity, and dependence on intact ocular structures — largely reserved for optic-pathway injury, cortical visual dysfunction, or paediatric neurocritical care. MEPs remain predominantly intraoperative; future ICU roles may include corticospinal assessment in severe motor dysfunction, spinal cord injury, and prolonged disorders of consciousness.

COMMON PITFALLS WHILE ASSESSING EPs

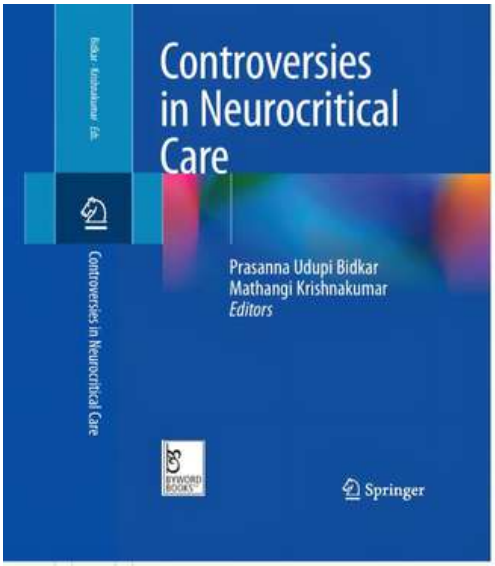
- Electrical Interference
- Peripheral Neuropathy
- Hearing Impairment (affecting BAEPs)
- Ocular Pathology (affecting VEPs)
- Neuromuscular Blockade (affecting MEPs)
- Sedative drugs
- Hypothermia
- Metabolic disturbances
- Hypotension

EP FINDINGS SHOULD BE INTEGRATED WITH:

- Neurological Examination
- Electroencephalography (EEG)
- Neuroimaging (CT and MRI)
- Biomarkers
- Clinical Trajectory

LEARNING TO HOLD THE WHOLE:

REFLECTIONS FROM MY FIRST EDITORIAL EXPERIENCE



"EVERY CHAPTER REFLECTS A RELATIONSHIP, NOT JUST EXPERTISE."

When I agreed to take on the role of a book editor in neurocritical care, I underestimated how profoundly the experience would reshape the way I think about academic work. Until then, my relationship with books and chapters had been linear: identify a question, write a manuscript, respond to reviewers, move on. Editing a book, however, required me to step outside my own intellectual lane and hold an entire ecosystem together—ideas, authors, timelines, disagreements, and expectations.

REACHING OUT: THE HUMAN SIDE OF ACADEMIA

Inviting contributors was unexpectedly daunting. Each email carried a quiet negotiation—of time, authority, and commitment. Senior authors brought depth and perspective; younger contributors brought energy and urgency. Balancing both while being respectful of competing clinical and academic demands taught me that editorial leadership is fundamentally relational. Deadlines, I learned quickly, are aspirational unless accompanied by empathy and clarity.

FROM IDEATION TO STRUCTURE

The process began not with chapters, but with questions. What did we truly want this book to achieve? Which controversies in neurocritical care were mature enough to be discussed, and which were still evolving? Ideation was less about originality and more about relevance—identifying themes that reflected real dilemmas faced at the bedside. Translating these ideas into a coherent table of contents was my first lesson in restraint: every attractive idea does not deserve a chapter.

Planning timelines on paper felt deceptively neat. In reality, life intervened—clinical surges, personal commitments, institutional responsibilities. Chapters arrived late, some required extensive restructuring, others needed gentle but firm redirection. I learned to build buffers, send reminders without guilt, and accept that momentum matters more than perfection. Progress, not polish, keeps a project alive.

LEARNING TO HOLD THE WHOLE:

REFLECTIONS FROM MY FIRST EDITORIAL EXPERIENCE

HOW IT CHANGED ME

This first editorial experience changed how I read, write, and teach. I now approach manuscripts with greater humility, aware of the invisible labour behind coherence. It has made me more patient with reviewers, more disciplined with my own writing, and more conscious of the responsibility that comes with shaping academic narratives.

"MENTORSHIP WAS NOT ABOUT FIXING PROBLEMS, BUT ABOUT KNOWING WHICH ONES MATTERED."

SETBACKS AND SELF-DOUBT

There were moments of genuine setback—chapters that did not align, revisions that went unanswered, and periods where the project felt stalled. At times, I questioned my readiness for the role. Yet, these moments were also instructive. They forced me to develop decisiveness, to resolve ambiguity, and to recognise when to move forward rather than wait for ideal conditions.

MENTORSHIP AND PERSPECTIVE

Working under the mentorship of Dr Prasanna was pivotal. The guidance was subtle but constant: how to frame feedback, when to push, when to pause, and how to protect the scientific integrity of the book without fracturing professional relationships. I learned that good editors do not dominate discussions—they anchor them.

Above all, it taught me that being an editor is not about control—it is about stewardship. Holding multiple perspectives together, navigating uncertainty, and delivering something meaningful despite imperfections is, in many ways, not unlike neurocritical care itself.

DR. MATHANGI KRISHNAKUMAR
ASSOCIATE PROFESSOR
SURGICAL AND NEURO ICU
DEPT OF ANAESTHESIA
ST JOHN'S MEDICAL COLLEGE HOSPITAL

MINUTE TO WIN IT ON READING CT BRAIN IN TRAUMATIC BRAIN INJURY

A 60-SECOND SYSTEMATIC APPROACH TO SAVE BRAINS

WHY IT MATTERS

- CT is the first and most crucial investigation in acute TBI.
- Early detection of life-threatening lesions → early intervention → better outcomes.
- **Don't miss the killers!**

1. FOLLOW A SYSTEMATIC APPROACH

B – BLEED
- Any extra-axial (EDH/SDH), intra-axial, or intraventricular bleed?

R – REGIONS
- Scan all areas: frontal, temporal, parietal, occipital, posterior fossa.

A – AIR & BONE
- Skull fractures? Pneumocephalus?

I – INFLAMMATION/ISCHAEMIA
- Early contusions? Hypodensity? Grey–white differentiation?

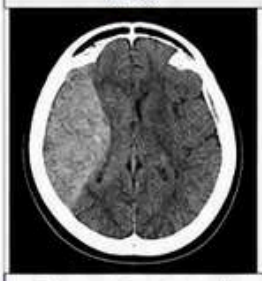
N – NEED TO ACT
- Mass effect? Midline shift? Basal cisterns?

S – SINUSES & ORBITS
- Associated facial injuries?

Tip: Always correlate with GCS, pupils & vitals.

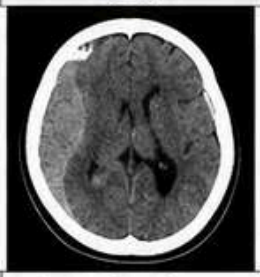
2. KNOW YOUR KILLERS – SPOT THEM FAST

EPIDURAL HEMATOMA (EDH)



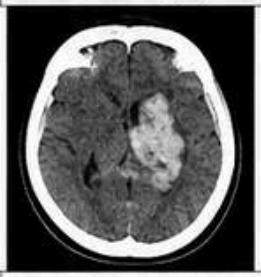
- Biconvex (lens-shaped)
- Does not cross sutures
- Usually arterial (MMA)
- May have lucid interval

SUBDURAL HEMATOMA (SDH)



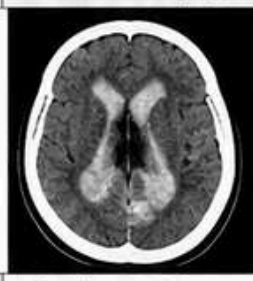
- Crescent shaped
- Crosses sutures
- Venous bleed (bridging veins)
- Acute / subacute / chronic

INTRAPARENCHYMAL HEMATOMA (IPH)



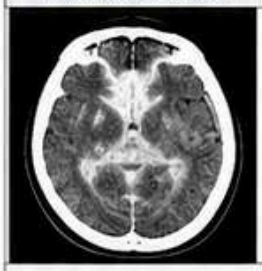
- Within brain parenchyma
- May be focal or multiple
- Look for surrounding edema

INTRAVENTRICULAR HEMORRHAGE (IVH)



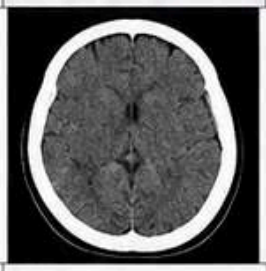
- Blood in ventricles
- Look for hydrocephalus

SUBARACHNOID HEMORRHAGE (SAH)



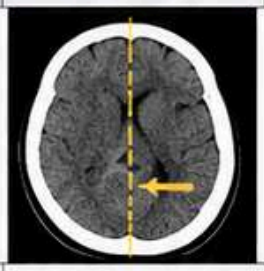
- Hyperdensity in sulci, cisterns
- Traumatic SAH common

DIFFUSE CEREBRAL EDEMA



- Effaced sulci
- Compressed ventricles
- Loss of grey-white differentiation

MIDLINE SHIFT



- Shift of septum pellucidum
- >5 mm is significant
- Look for uncal/basal herniation

BASAL CISTERN EFFACEMENT / HERNIATION

- Effaced cisterns
- May see uncal, central or tonsillar herniation

3. QUICK CT CHECKLIST (60 SECONDS)

- 0–10 sec Scan the whole head – look for major bleed, mass effect, midline shift.
- 10–20 sec Check extra-axial spaces (EDH/SDH), then intra-axial bleeds.
- 20–30 sec Look at bone windows – fractures? Pneumocephalus?
- 30–40 sec Assess ventricles – size, symmetry, IVH?
- 40–50 sec Look for edema – sulci, grey-white differentiation.
- 50–60 sec Check midline, basilar cisterns – any herniation?

Practice makes it sub-60 seconds!

4. RED FLAGS – ACT IMMEDIATELY

- ⚠ EDH > 30 mL or thickness > 15 mm
- ⚠ Acute SDH > 10 mm or shift > 5 mm
- ⚠ **Any mass lesion with** neurological deterioration
- ⚠ **Midline shift > 5 mm**
- ⚠ **Effaced basal cisterns**
- ⚠ **Traumatic IVH with hydrocephalus**
- ⚠ **Depressed skull fracture with** neurological deficit

5. CORRELATE & COMMUNICATE

- ✓ Correlate CT with clinical status (GCS, pupils, vitals).
- ✓ Communicate key findings clearly and early to the treating team.
- ✓ Document size, volume, shift, and associated injuries.

6. PEARLS TO REMEMBER

- ★ A normal CT does not exclude DAI – clinical correlation is key.
- ★ Repeat CT if neuro status worsens.
- ★ Use bone windows for fractures.
- ★ Look posterior fossa carefully – small bleeds can be missed.
- ★ When in doubt, show it to your senior – early decision saves lives.

REFERENCES

1. Brain Trauma Foundation. Guidelines for the Management of Severe TBI. J Neurotrauma. 2016.
2. Stiell IG, et al. Canadian CT Head Rule for Patients with Minor Head Injury. Lancet. 2001.
3. Robba C, et al. Computed Tomography in Traumatic Brain Injury: An Update. Curr Opin Crit Care. 2020.

INSTACLIK

Neuro Critical Care Team, Neurofoundation hospital

Patient Care Staff,
Neurofoundation Hospital.

Nutritionists,
Neurofoundation Hospital.

Physiotherapy Team, NeuroFoundation Hospital

Clinical Pharmacist, NeuroFoundation Hospital

Auxillary Nursing Team,
Neurofoundation Hospital.

2026

"The strength of the team is each individual member. The strength of each member is the team."

BioFire *in Neurocritical Care*

1	14	~1 hr	0.2 mL	2 min
TEST	TARGETS	SAMPLE TO REPORT	CSF REQUIRED	HANDS-ON TIME

THE CLINICAL PROBLEM

Meningitis and encephalitis carry high mortality and morbidity, including serious long-term sequelae. Conventional diagnosis relies on CSF culture, Gram stain, and individual viral PCRs — a workup that can take **24 to 72 hours**. Because delayed treatment causes harm, most guidelines recommend empiric therapy for bacterial meningitis and HSV encephalitis until microbiological reports return.

Conventional methods carry three significant limitations: long turnaround time, the need for a specialised laboratory, and **reduced culture sensitivity in patients pre-treated with antibiotics**.

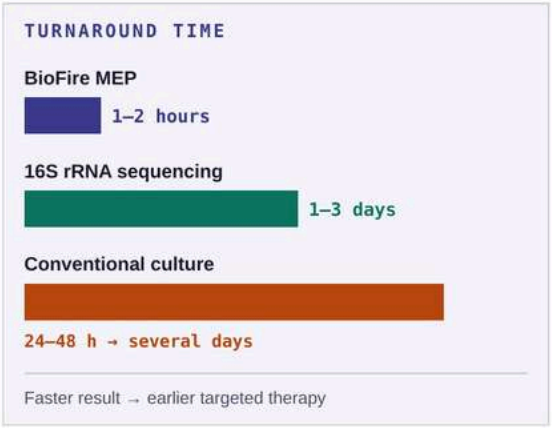


Fig 1. Relative sample-to-report times. Bars are schematic, not to exact scale.

THE INTERVENTION

The BioFire FilmArray Meningitis/Encephalitis Panel (MEP) was the first commercially approved multiplex PCR panel for infectious meningoencephalitis, in **2015**. It delivers rapid results and has the potential to reduce empiric antimicrobial use and length of hospitalisation compared with conventional testing.

WHY IT FITS THE ICU

- ✓ Only **0.2 mL** CSF required
- ✓ **2 minutes** hands-on time
- ✓ No precise measuring or pipetting
- ✓ Sample to result in **1 hour**
- ✓ No specialised laboratory needed

STANDARD PCR VS. MULTIPLEX PCR

Standard PCR amplifies a **single** DNA/RNA target per reaction. Multiplex PCR uses multiple primer sets to amplify **several** targets simultaneously in the same tube — faster and more efficient reporting from one sample.

SO HOW IS BIOFIRE DIFFERENT?

BioFire is a proprietary brand that *uses* multiplex PCR. Traditional multiplex PCR requires manual sample preparation and batch testing; the FilmArray system **automates the entire process in a closed pouch**, delivering results in about an hour.

DR MAHENDRANATH M
CONSULTANT
AIG HOSPITAL
HYDERABAD

01 — How the FilmArray Works

CLOSED-POUCH NESTED MULTIPLEX PCR
14 TARGETS · ONE REPORT

CLOSED-POUCH WORKFLOW

Reagents for sample prep, reverse transcription, PCR and detection are stored freeze-dried in the pouch.
The user injects hydration solution, the CSF sample and buffer — the instrument does the rest.

Fig 2. The FilmArray performs nested multiplex PCR: one large multiplexed first-stage reaction, then individual singleplex second-stage reactions, resolved by endpoint melting curve analysis.

The 14 targets

The BIOFIRE ME Panel simultaneously detects 14 common pathogens in CSF — **6 bacteria, 7 viruses, and 1 yeast**.

BACTERIA 6

- *Escherichia coli*
- *Haemophilus influenzae*
- *Listeria monocytogenes*
- *Neisseria meningitidis*
- *Streptococcus agalactiae*
- *Streptococcus pneumoniae*

VIRUSES 7

- *Cytomegalovirus (CMV)*
- *Enterovirus*
- *Herpes simplex virus 1*
- *Herpes simplex virus 2*
- *Human herpesvirus 6*
- *Human parechovirus*
- *Varicella zoster virus*

FUNGUS 1

- *Cryptococcus neoformans/gattii*

INDICATIONS

1 Suspected meningitis / encephalitis
Sudden fever, stiff neck, severe headache, altered mental status, or focal neurological deficits.

2 When rapid diagnosis is needed
Critical cases where timely initiation — or de-escalation — of targeted therapy is essential.

3 When CSF volume is limited
Requires only ~0.2 mL, where multiple singleplex cultures or PCRs cannot be performed.

4 In the immunocompromised
Rapid identification of opportunistic infections such as *Cryptococcus neoformans* or viral pathogens.

02 — BioFire vs. Conventional

	BioFire MEP	Conventional testing
Technology	Multiplex PCR simultaneously amplifies and identifies the DNA/RNA of **6 bacteria, 7 viruses and 1 yeast** from a single CSF sample.	A combination of CSF Gram stain, bacterial and fungal cultures, and specific viral tests (individual viral PCRs).
Sample to report	**Approximately 1 to 2 hours.**	Cultures take **24–48 hours** for bacteria and up to several days for slower-growing organisms.
Scope	Highly sensitive — **especially if the patient has already received antibiotics**, which can kill bacteria and cause false-negative cultures. Cannot detect pathogens outside its designated list.	Cultures can identify virtually any bacteria, fungi or mycobacteria growing in the sample. Standalone viral PCRs can be ordered for a wider variety of specific viral agents.
Limitations	• Misses any pathogen not on the target list • Higher cost; equipment availability	• Slow turnaround time • Culture sensitivity drops significantly if antibiotics are given before sample collection

CLINICAL USE

Theoretically, rapid results allow faster institution of **targeted therapy** and reductions in empiric antimicrobial use and length of hospital stay.

Current guidelines recommend BioFire MEP as a **rapid add-on test** to conventional workup — *not* a full replacement. It is highly useful for ruling in or out common viral and bacterial etiologies in the emergency room, while cultures remain necessary to guide specific, long-term antibiotic therapy.

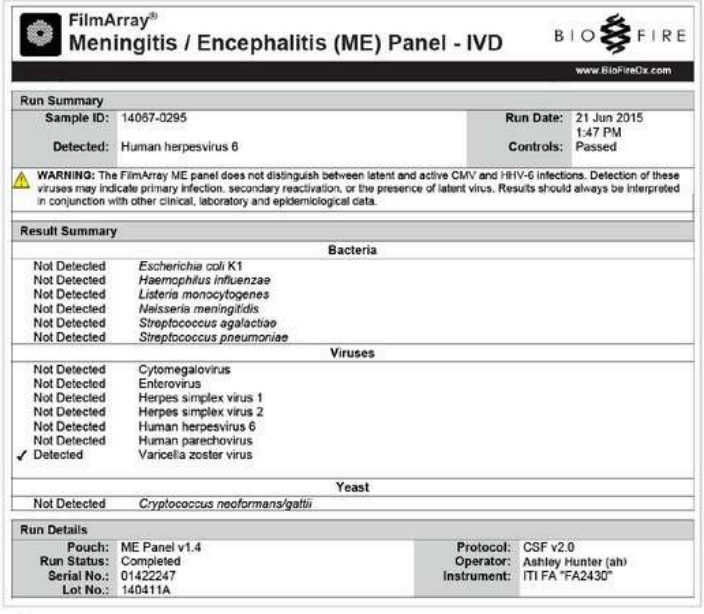
Studies show implementation of the FilmArray for ME diagnosis is associated with a **reduction in duration of acyclovir treatment**, and in some, reduced antimicrobial use and shorter hospital stay.

BOTTOM LINE

BioFire works as **point-of-care testing where time-critical diagnosis is needed**, giving rapid, actionable results to guide antimicrobial therapy while cultures are awaited.

DRAWBACKS TO REMEMBER

EXPENSIVE · LOW DETECTION RATE · NO MYCOBACTERIA · NO ARBOVIRUSES · FIXED TARGET LIST



FilmArray®
Meningitis / Encephalitis (ME) Panel - IVD
BIOFIRE
www.BioFireDx.com

Run Summary

Sample ID:	14067-0295	Run Date:	21 Jun 2015 1:47 PM
Detected:	Human herpesvirus 6	Controls:	Passed

⚠ **WARNING:** The FilmArray ME panel does not distinguish between latent and active CMV and HHV-6 infections. Detection of these viruses may indicate primary infection, secondary reactivation, or the presence of latent virus. Results should always be interpreted in conjunction with other clinical, laboratory and epidemiological data.

Result Summary

Bacteria	
Not Detected	*Escherichia coli* K1
Not Detected	*Haemophilus influenzae*
Not Detected	*Listeria monocytogenes*
Not Detected	*Neisseria meningitidis*
Not Detected	*Streptococcus agalactiae*
Not Detected	*Streptococcus pneumoniae*
Viruses	
Not Detected	Cytomegalovirus
Not Detected	Enterovirus
Not Detected	Herpes simplex virus 1
Not Detected	Herpes simplex virus 2
Not Detected	Human herpesvirus 6
Not Detected	Human parechovirus
✓ Detected	Varicella zoster virus
Yeast	
Not Detected	*Cryptococcus neoformans/gattii*

Run Details

Pouch:	ME Panel v1.4	Protocol:	CSF v2.0
Run Status:	Completed	Operator:	Ashley Hunter (ah)
Serial No.:	01422247	Instrument:	ITI FA "FA2430"
Lot No.:	140411A		

Fig 3. A representative FilmArray ME Panel run report, as supplied in the source material — every target is reported as Detected or Not Detected on a single page.

DECISION FATIGUE IN THE ICU

Protecting clinical judgment when the **hardest thing to find** is the willingness to think once more.

DR. MATHANGI D.C
HOD,
MIND, BODY MEDICINE
SRMC, PORUR, CHENNAI

By the end of a long shift you are not yawning. You could recite the doses without hesitation. But a question arrives — **should this patient be extubated today?** — and instead of thinking it through, you reach for whatever answer asks the least of you.

01

DENSITY

Dozens of consequential choices per round, with no recovery between them.

02

IRREVERSIBILITY

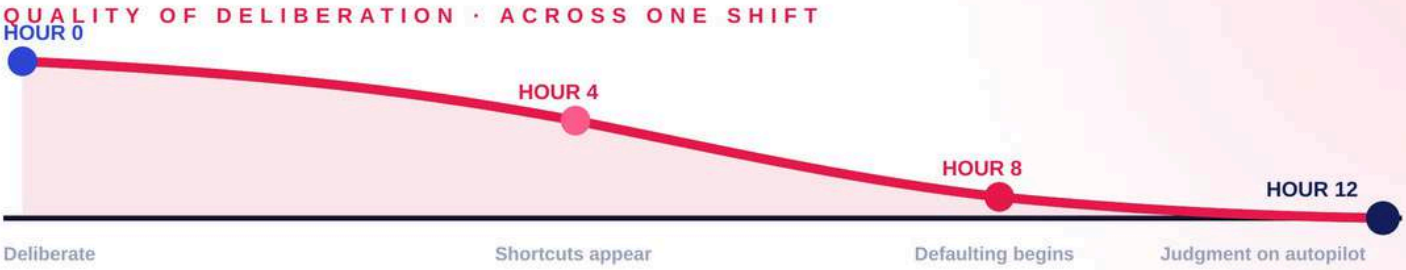
Decisions that cannot be walked back resist delegation to habit.

03

AMBIGUITY

Data rarely resolve cleanly, so uncertainty must be tolerated again and again.

01 THE PROBLEM



The shape of the problem. Decision quality rarely collapses — it erodes, most steeply when the sickest patients declare themselves.

There is a kind of tiredness that **does not feel like tiredness.**

Decision fatigue is the deterioration in decision quality after a long run of deciding — not a failure of character, but the predictable cost of hundreds of consequential choices in sequence.

WHY THE ICU IS A WORST CASE

Every environment involves decisions; the ICU concentrates them. One round may hold dozens of judgments — narrow margins, thin data, consequences in hours.

> **"FATIGUE DOES NOT ANNOUNCE ITSELF AS FATIGUE. IT ARRIVES DISGUISED AS CERTAINTY."**
>
> ON RECOGNISING DEPLETION

DEPLETION LOOKS LIKE DRIFT

Depletion does not look like error. It looks like *drift* — the mind, seeking economy, moving toward one of two poles.

THE DEFAULT

Choosing what needs no justification — continue the plan, order one more test, defer to the next shift. It feels safe because nothing changes. Often, nothing changing *is* the decision.

THE SHORTCUT

Taking the first plausible answer and stopping. Anchoring on the admission diagnosis. Accepting the handover framing unexamined. Mistaking speed for clarity.

Both are efficient, and mostly harmless. Neither announces the exception — and in the ICU, the exception is where harm lives.

THE SIGNS, IN YOURSELF

- Irritation at reconsidering
- Reluctance to reopen the chart
- Preferring the plan that ends the conversation
- Can't hold two possibilities at once
- Relief when someone else decides
- Everything feels roughly fine
- Skipping your usual step

None is diagnostic alone. Together — noticed *at the time* — they signal the next decision needs more scaffolding.

02 THE PRACTICE

The remedy is not to decide less. It is to build **small, repeatable structures** that carry what the mind cannot carry unaided.

1
NAME THE STATE

The most useful intervention takes four seconds. Say silently: *this is the tired version of my judgment.* Naming a state turns it from a lens you look through into an object you can look at.

2
FRONT-LOAD WHAT MATTERS

Where you control sequence, place the hardest decisions early. Goals-of-care talks are best made when the resource is full. Protocolised work tolerates depletion better.

3
USE THE PAUSE

Before any irreversible decision, breathe once and ask: *What would I want to know if this went badly?* It costs nothing, and surfaces what hurried thinking skips.

4
EXTERNALISE THE CHECKLIST

A depleted mind forgets to be thorough but can follow a written prompt. Checklists are not overhead — they are cognitive prosthetics, most valuable when you feel you don't need them.

5
NORMALISE SECOND OPINIONS

Asking a colleague to look again should imply no weakness. Teams that normalise *"sanity-check this?"* spread the load across people whose curves aren't synchronised with yours.

6
PROTECT SMALL RECOVERIES

Food, water, two minutes without an alarm are not indulgences. They are maintenance for the instrument you decide with. A shift with no recovery borrows against its last patient.

A NOTE ON THE CULTURE

Individual practice matters, but decision fatigue is substantially a **design problem**. Rota structure, handover quality, staffing and interruption load shape the curve far more than willpower.

Framing this as purely personal resilience quietly moves a systems failure onto the individual clinician.

BEFORE YOU HAND OVER

- ☐ Which decision did I make **fastest** — was speed warranted?
- ☐ What did I **defer**, and to whom?
- ☐ Is there a patient I have **stopped re-examining**?
- ☐ What does the next clinician need that **isn't in the notes**?

THE CLOSING THOUGHT

Clinical judgment is often called a fixed property — formed in training, thereafter stable. It is better understood as a **state**: variable within a day, sensitive to sleep and interruption.

Mindfulness here is not stillness. It is noticing which version of yourself is making the decision — and adjusting accordingly.

The Invisible IntensiveCare Unit: Family Caregivingas a Determinant of Post-Neurointervention and Neurosurgical Outcomes

DR SHIVANI FOTEDAR
DRNB TRAINEE,
ARTEMIS HEALTH INSTITUTE, GURUGRAM

" It is the caregiver vigilance, exhaustion, and resilience that often decide whether that "good outcome" on paper becomes a truly independent life."

The moment a neurosurgical patient leaves our ICU, a second, largely
invisible ICU switches on at home —and the intensivist in charge is usually a worried spouse, daughter, or parent.

We obsess over
door-to-needle times and TICI scores, but three months later, it is caregiver vigilance, exhaustion, and resilience that often decide whether that “good outcome” on paper becomes a truly independent life.

From monitors to living rooms

In the unit, we have real-time neuromonitoring, instant CT access,
emergency craniotomies, and slick thrombectomy pathways. Mortality
has dropped dramatically, but many patients go home with “soft”
deficits: slowed thinking, poor initiation, word-finding difficulty, subtle hemiparesis, judgment lapses that only a family
member will notice.

On the ward their Barthel scores often sit in the severely dependent range, but our discharge summary may still read “stable, for home with family“ .

The reality is that the post-acute 2–6 month window is not a passive recovery phase—it is an active neuroplasticity lab, and the family is running the experiment day & night. In this period, caregiver behaviour, understanding, and burnout often matter more for mRS at 3 months than the elegance of the clipping or the speed of recanalization.

Families as the “external cortex”

At home, our patients suddenly have to have their own nurse, pharmacist, physio, and psychologist—except many cannot. Cognitive impairment after stroke or neurosurgery is common and directly undermines medication adherence, especially for high-stakes regimens like DAPT after stenting or flow diversion.

A single missed dose of clopidogrel or aspirin can undo a perfect angiographic result. Here, the caregiver becomes an “external executive frontal lobe”: organizing pill boxes, setting alarms, insisting that tablets are actually swallowed, and calling when something feels off.

Studies show that when caregivers understand the purpose of each drug and are not overwhelmed, adherence can exceed 90%; when burden is high, it drops into the 60–70% range, with a very real increase in thrombotic risk.

The same dynamic plays out with rehab—motivated, informed families quietly convert thrice-weekly formal therapy into daily, meaningful practice at home.

Families as the “early warning systems”

Complications rarely present like the textbook. Retroperitoneal bleed after femoral access may show up as “back pain and restlessness, vasospasm after SAH may first appear as “he seems a bit off today.” A nurse who reviews the patient once a week can miss these nuances; a spouse who sees them 24/7 cannot unsee the change in facial expression, gait, or conversation.

The hidden cost: caregiver burden as a safety issue

Caregiver burden is a significant issue for relatives of brain surgery patients, as confirmed by literature and clinic observations.

Factors such as age, gender, marital status, income, and employment influence the burden level, with more dependent patients leading to heavier loads for caregivers.

This burden has practical implications, correlating with missed medication doses and skipped rehabilitation. Emotional dynamics also play a role, as depressed caregivers result in decreased vigilance and poorer patient outcomes. Thus, caregiver emotional well-being is crucial for patient safety.

From “patient education” to “dyad management”

Families play a crucial role in the care of neurocritical patients, necessitating a shift in discharge planning. A single-page summary is insufficient for transferring complex care responsibilities to families. Key recommendations include:

- **Start Early** : Involve families in rounds to familiarize them with medical terminology and expectations.
- **Teach, Don’t Just Inform** : Provide hands-on training for medication administration, patient positioning, and recognizing deterioration, verifying understanding through “teach back.”
- **Normalize Symptoms** : Use simple explanations to reduce anxiety about common post-injury symptoms.
- **Monitor the Dyad** : Assess both the patient's condition & the family's coping mechanisms during follow-ups using tools like the Zarit Burden Interview.

Family-based programs combining education and support over 8–12 weeks have shown to improve patient outcomes and reduce caregiver burden.

Rethinking phases of recovery

Stroke is typically categorized into acute, subacute, and chronic phases, but the critical "reintegration phase" from discharge to six months is often overlooked. During this time, families play a key role, and metrics should expand beyond mRS and NIHSS to include:

- Caregiver burden scores
- Caregiver depression/anxiety measures
- Medication adherence
- Time to first complication/ readmission
- Rehab attendance and participation

The “invisible ICU” we all rely on- For those of us in neuroanesthesia & neurocritical care, there is a humbling takeaway. Our work in the operating room & ICU is essential, but it is only the opening act. The real, prolonged intensive care happens off the monitor, in small apartments & crowded homes, monitored not by nurse call bells but by a caregiver’s light sleeper’s ear.

If we start treating family members as true colleagues in care—training them, listening to them, measuring their burden, & designing services around their needs —we are likely to see the same kind of step-change in long-term outcomes that thrombectomy brought to hyperacute stroke. The “invisible intensive care unit” is already there; our next task as a specialty is to acknowledge it, resource it, & partner with it intentionally.

Recent Rehabilitation Techniques after Stroke: Harnessing Neuroplasticity

DR. AMIYA KUMAR BARIK,
ASSOCIATE PROFESSOR,
AIIMS BHUBANESHWAR

> **Stroke continues to be the second leading cause of mortality and the third leading cause of mortality and disability combined.[1]**

Introduction

Stroke remains a significant global health burden (approximately 13.7 million cases) across continents and demographics.

The aftermath of a stroke extends beyond the individual affected, impacting families, caregivers, and society at large. It imposes emotional, financial, and practical burdens on families.

Traditionally, stroke rehabilitation focused mainly on physiotherapy and occupational therapy to help restore basic motor function.

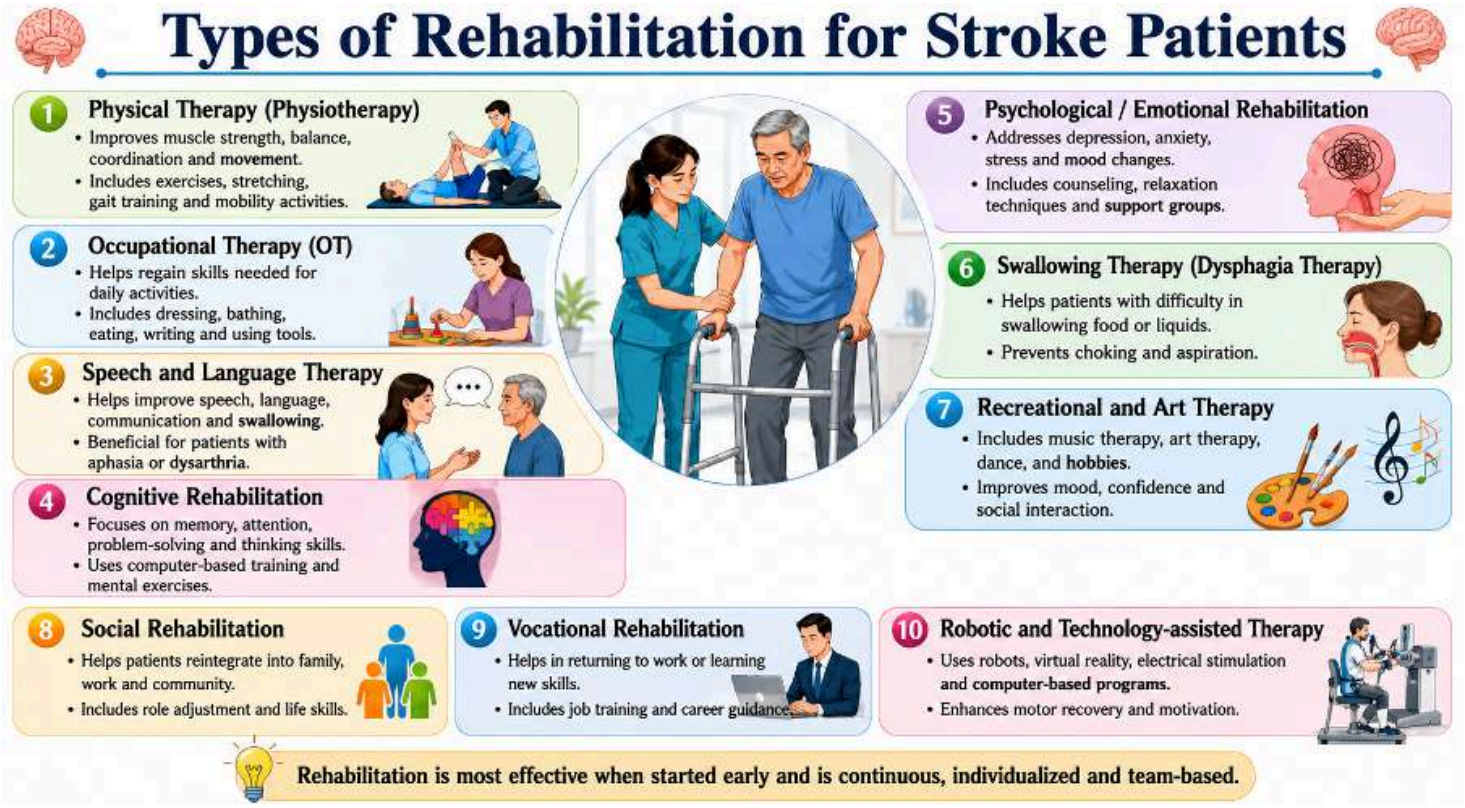
However, recent developments in neuroscience particularly neuroplasticity, technology, and collaborative care have greatly changed post-stroke rehabilitation.

Principles of Modern Stroke Rehabilitation:

Current stroke rehabilitation is based on the concept of neuroplasticity, which is the brain's ability to reorganize neural pathways after injury.

Repetitive, task-focused activities help stimulate cortical changes and functional recovery.

Starting rehabilitation early, typically within 24 to 48 hours after stabilization, has shown better results.



Key goals include:

· Restoration of motor and sensory function.

· Prevention of complications like contractures and pressure sores.

· Improvement of communication and swallowing.

· Enhancement of cognitive and psychological health.

· Reintegration into family and community life.

Constraint-Induced Movement Therapy:

CIMT is an effective method for upper limb rehabilitation. It involves restraining the unaffected limb while intensively training the affected limb, helping to overcome "learned non-use."

Studies show that CIMT leads to significant improvements in motor performance, dexterity, & functional independence.

Virtual Reality (VR) and Gamification:

This technology employs computer-generated interactive environments to engage patients in therapy, allowing them to perform simulated tasks like reaching and walking.

Benefits include enhanced motivation, real-time feedback, safe simulation of complex activities, and improved motor learning.

Robotic-assisted rehabilitation:

It has emerged as a key advancement in stroke recovery. It utilizes robotic devices for accurate, repetitive movements of limbs, enhancing therapy intensity while reducing therapist fatigue and enabling objective progress tracking.

Types of robotic therapy include:

- Exoskeleton robots for gait training
- End-effector devices for arm rehabilitation
- Wearable robotic gloves for hand function

Non-Invasive Brain Stimulation:

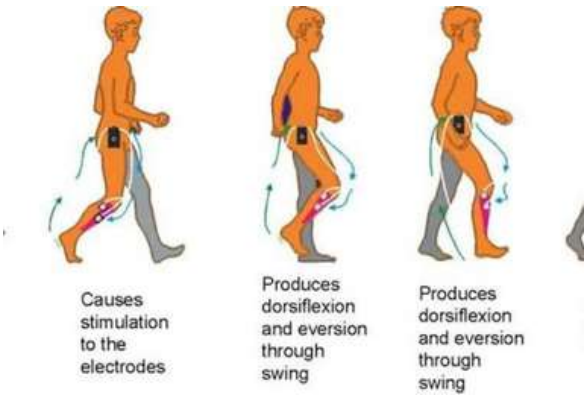
There is increasing interest in neuromodulation techniques to boost neuroplasticity after a stroke.

Although still under research, these techniques show promise in enhancing motor function, aphasia, and cognitive deficits.

Functional Electrical Stimulation (FES):

FES applies low-level electrical currents to stimulate weak or paralyzed muscles, enhancing strength, reducing spasticity, and aiding movements like grasping or walking.

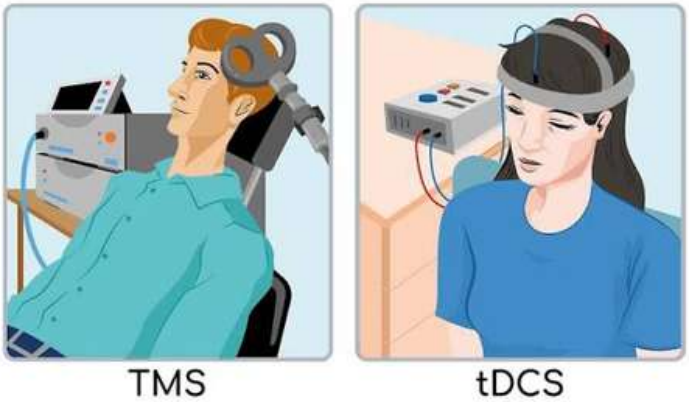
Combined with physiotherapy, it fosters motor relearning and improves outcomes. Common uses include correcting foot drop, aiding upper limb recovery, and preventing shoulder subluxation.



Transcranial Direct Current Stimulation:

tDCS delivers low-intensity electrical currents via scalp electrodes to modulate cortical excitability.

It is low-cost, portable, and increasingly studied as a supplement to traditional rehabilitation.



Body Weight-Supported Treadmill Training:

Patients walk on a treadmill while part of their body weight is supported by a harness system. This allows early gait training even for those with severe impairments.

Transcranial Magnetic Stimulation

TMS employs magnetic pulses to target specific brain areas.

Repetitive TMS can enhance activity in the affected hemisphere or reduce overactivity in the unaffected hemisphere, aiding motor recovery and language function.

Mirror Therapy:

Mirror therapy is an effective method for motor recovery, involving a mirror placed between limbs to create the illusion of movement in the affected limb when the unaffected limb moves.

This visual feedback activates mirror neurons and promotes cortical reorganization. It's particularly useful for upper limb rehabilitation and managing post-stroke pain syndromes.

Telerehabilitation

Telerehabilitation, essential during the COVID-19 pandemic, uses video conferencing, wearable sensors, and mobile apps for remote rehabilitation services.

Benefits include improved accessibility for rural patients, reduced travel, consistent care, and cost-effectiveness. Home-based programs allow patients to perform supervised exercises while maintaining contact with healthcare providers.

Computer-Based Cognitive Training

Digital platforms offer exercises targeting attention, memory, executive function, and problem-solving skills.

Cognitive & Modern Aphasia Therapy

Speech rehabilitation increasingly incorporates:

·Computer-assisted language therapy

·Melodic intonation therapy

·AI-assisted communication tools

Early intensive speech therapy improves language recovery and social interaction.

Stem Cell Therapy & Regenerative Medicine:

Stem cell therapy is an emerging area in stroke rehabilitation. Research suggests that stem cells may encourage neurogenesis, angiogenesis, and repair of damaged neural tissue.

Psychological Rehabilitation:

Depression, anxiety, and emotional issues are common after stroke.

Modern rehabilitation includes psychological counselling, cognitive behavioural therapy, mindfulness techniques, and caregiver support programs.

Conclusion

Stroke rehabilitation has evolved significantly, now emphasizing individual needs and leveraging technology and teamwork.

While traditional physiotherapy remains vital, integrating new techniques can enhance neuroplasticity, independence, and quality of life for stroke survivors.

Future advancements in artificial intelligence, brain-computer interfaces, and regenerative therapies may further revolutionize rehabilitation, providing new hope for patients globally.

KETOGENIC DIET IN NEUROCRITICAL CARE: METABOLIC THERAPY AT THE BEDSIDE

DR. PRIYA THAPPA
Assistant Professor
AIIMS, Nagpur

Introduction

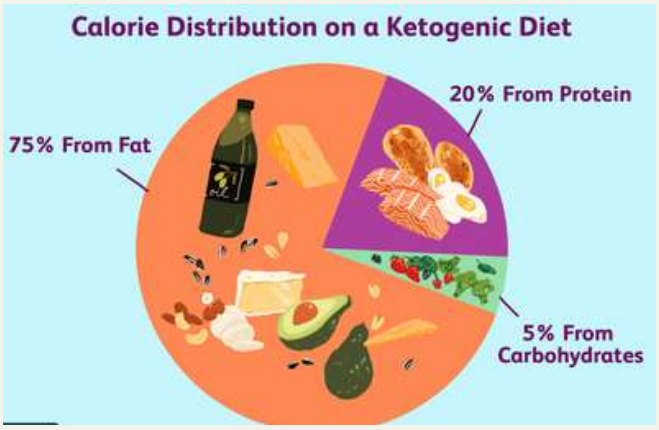
The ketogenic diet (KD), once considered a niche therapy primarily reserved for refractory pediatric epilepsy, is increasingly gaining recognition in neurocritical care as a promising metabolic intervention.

In conditions such as TBI, status epilepticus, ischemic stroke, subarachnoid hemorrhage, and hypoxic brain injury, impaired glucose utilization and mitochondrial dysfunction contribute significantly to secondary neuronal injury.

Against this backdrop, ketogenic therapy has emerged as a compelling strategy aimed at providing alternative cerebral fuel, reducing neuroinflammation, and stabilizing neuronal excitability.

Composition

The ketogenic diet is a high-fat, low-carbohydrate, moderate-protein nutritional regimen.



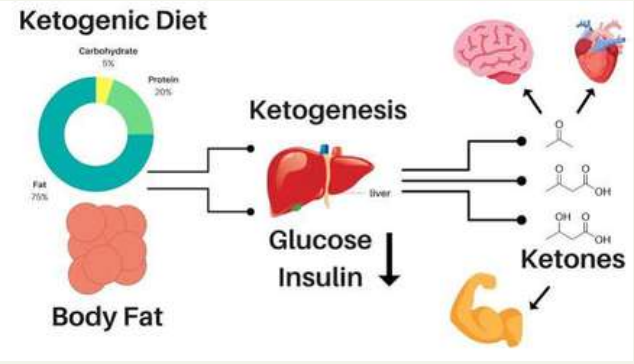
The Metabolic Basis of Ketosis

The ketogenic diet is a high-fat, low-carbohydrate, moderate-protein nutritional regimen (Typical macronutrient distribution : 55% to 60% fat, 30% to 35% protein, and 5% to 10% carbohydrates) that induces production of ketone bodies.

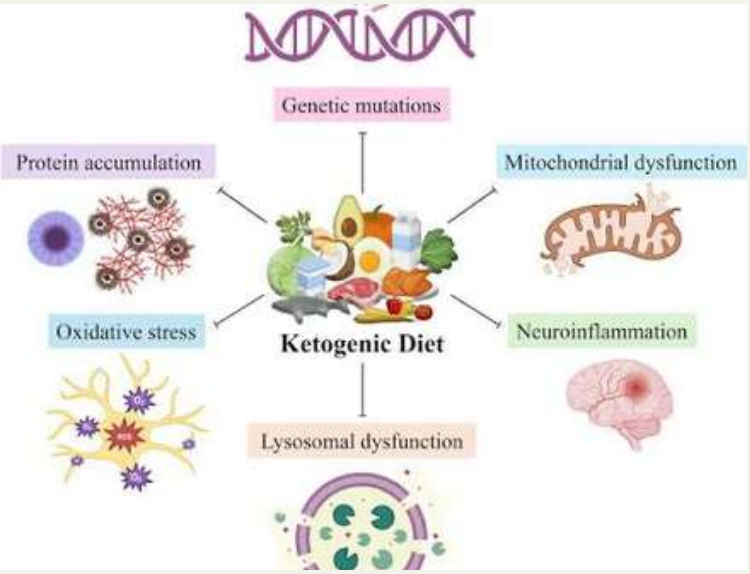
Under carbohydrate restriction, ketones become the main energy source for the brain, which typically relies on glucose.

During metabolic stress, ketone bodies can cross the blood-brain barrier and are converted to acetyl-CoA for ATP production in the TCA cycle.

Neurons and astrocytes can utilize ketones, which generate ATP more efficiently than glucose, potentially enhancing bioenergetic efficiency in injured brain tissue.



Ketogenic Diet in Refractory Status Epilepticus



The strongest clinical evidence supporting ketogenic therapy in neurocritical care currently exists in refractory status epilepticus and super-refractory status epilepticus.

Seizure cessation rates ranging from 50-90% have been reported after induction of ketosis.

The precise anti-seizure mechanism remains incompletely understood but is thought to involve enhanced GABA activity, reduced glutamatergic transmission, modulation of ATP-sensitive potassium channels, and improved mitochondrial stability.

Importantly, ketosis can often be achieved relatively rapidly in intensive care settings through enteral ketogenic formulations or parenteral ketogenic protocols.

Many centers now employ ketogenic therapy as an adjunctive option when conventional anti-seizure therapies fail.

Despite promising outcomes, evidence remains limited largely to retrospective analyses and small prospective cohorts.

Large randomized controlled trials are still lacking, and optimal protocols for initiation, maintenance, and discontinuation continue to evolve.

Challenges

Nutritional Formulation:

- Standard ICU feeds are carbohydrate-rich; ketogenic protocols require specific fat-to-carbohydrate ratios (2:1 to 4:1).

Monitoring Requirements:

- Frequent checks of serum beta-hydroxybutyrate, glucose, electrolytes, liver function, lipid profile, and acid-base status are crucial.

Metabolic Complications:

- Potential adverse effects include hypoglycemia, hypertriglyceridemia, metabolic acidosis, hepatic dysfunction, nephrolithiasis, and gastrointestinal intolerance.

Drug Compatibility:

- Propofol, being lipid-based, may heighten metabolic risks when used with ketogenic therapy.

Traumatic Brain Injury and Cerebral Energy Crisis

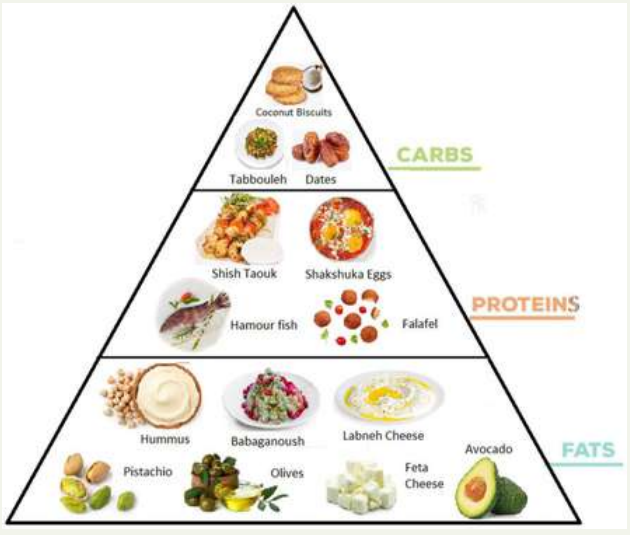
Following traumatic brain injury (TBI), cerebral glucose metabolism is disrupted due to mitochondrial dysfunction and impaired perfusion, leading to ongoing neuronal damage.

While preclinical studies suggest ketogenic therapy may improve cerebral bioenergetics and reduce metabolic dysfunction, human data on outcomes like cerebral microdialysis and brain oxygenation are limited.

The ketogenic diet's neuroprotective effects may stem from its influence on mitochondrial permeability transition pore (mPTP) activity and cellular bioenergetics.

However, challenges in clinical implementation arise due to severe TBI patients facing hypercatabolism, insulin resistance, and fluctuating nutritional tolerance, making sustained ketosis difficult.

Additionally, there is insufficient evidence for improved long-term neurological outcomes.



Ketosis in Ischemic Stroke and Hypoxic Brain Injury

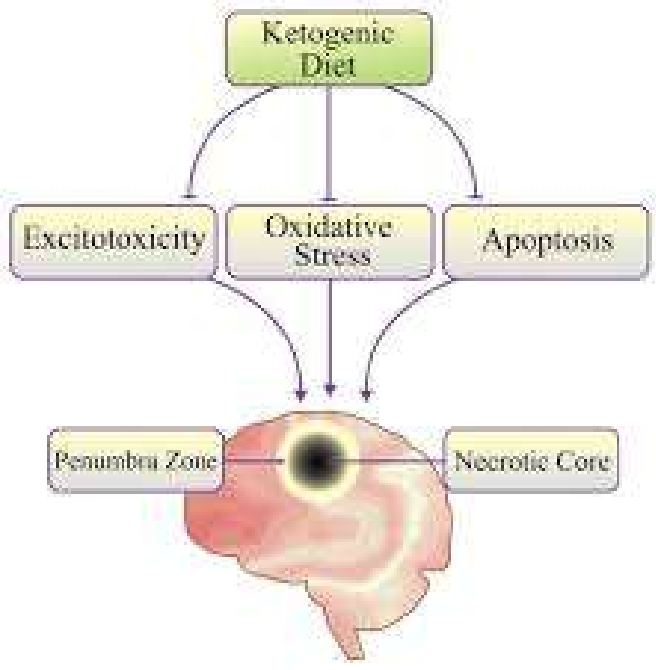
Cerebral ischemia leads to oxidative phosphorylation failure and rapid energy depletion in neurons.

Experimental models indicate that ketone bodies might help preserve mitochondrial integrity, reduce infarct size, and lessen post-ischemic inflammation.

In cases of hypoxic-ischemic brain injury, ketosis may enhance neuronal survival by mitigating oxidative damage and excitotoxicity.

However, clinical evidence in humans is limited, primarily consisting of small feasibility or mechanistic studies rather than conclusive outcome-based trials.

Therefore, ketogenic therapy for stroke and hypoxic brain injury is still considered investigational.



Enteral vs Parentral:

Enteral ketogenic therapy is preferred when gastrointestinal function is intact due to its physiological benefits, lower infection risk, and ease of maintenance.

Common formulations like KetoCal®, KetoVie®, and Ketonia® provide high-fat, low-carbohydrate ratios (2:1 to 4:1) and are administered via feeding tubes in critically ill patients. Modular supplements may help achieve target ratios.

When enteral nutrition is contraindicated (e.g., severe ileus, bowel ischemia), parenteral ketogenic nutrition using lipid emulsions and amino acids may be considered but is harder to maintain due to risks like hypertriglyceridemia and metabolic acidosis.

Thus, enteral ketogenic nutrition is generally favored in neurocritical care.

Current Evidence & Future Directions:

While enthusiasm surrounding ketogenic therapy in neurocritical care is growing, the field remains in an early translational phase.

Much of the current evidence derives from animal models, retrospective cohorts, and small prospective studies.

Heterogeneity in ketogenic protocols, patient selection, ketosis targets, and outcome measures further complicates interpretation.

Future research must focus on adequately powered randomized controlled trials evaluating meaningful clinical outcomes such as mortality, functional recovery, seizure control, and cognitive performance. Precision approaches may also help identify patient populations most likely to benefit from metabolic therapy.

Conclusion

The ketogenic diet represents a fascinating convergence of metabolism and neurocritical care. By targeting cerebral bioenergetics, mitochondrial dysfunction, and neuroinflammation, ketogenic therapy offers a novel adjunctive approach for managing acute neurological injury.

Although definitive evidence remains limited, early clinical experience, particularly in super-refractory status epilepticus is encouraging. As understanding of cerebral metabolism deepens, ketogenic therapy may evolve from an experimental intervention into a standard component of neurocritical care practice.

LINK–JULY 2026

Update This Month

WHATS UP!

The NEW ENGLAND JOURNAL of MEDICINE

Three Antihypertensives in a Single Pill after Intracerebral Hemorrhage

A Research Summary based on the Trident Research Group | 10.1056/NEJMoa2515043 | Published on April 23, 2026

WHY WAS THE TRIAL DONE?

In patients with spontaneous intracerebral hemorrhage, effective blood-pressure reduction is the only treatment proven to prevent recurrence. However, long-term blood-pressure control is often inadequate. Whether a single pill combining three antihypertensive drugs at low doses can lower blood pressure more than standard antihypertensive treatment alone and reduce the risk of recurrent stroke is uncertain.

HOW WAS THE TRIAL CONDUCTED?

Adults with intracerebral hemorrhage who were in clinically stable condition were eligible if they had a systolic blood pressure of 130 to 160 mm Hg. After a 2-week run-in phase during which all the patients received a once-daily pill containing low doses of three antihypertensive agents (20 mg of telmisartan, 2.5 mg of amlodipine, and 1.25 mg of indapamide; the triple pill), patients were randomly assigned to continue receiving the triple pill or to receive matching placebo, in addition to standard care. The primary outcome was the first recurrent stroke. Safety was also assessed.

TRIAL DESIGN

- Double-blind
- Randomized
- Placebo-controlled
- Location: 61 sites in 12 countries

RESULTS

At a median follow-up of 2.5 years, recurrent stroke had occurred in 4.6% of the patients in the triple-pill group and in 7.4% of those in the placebo group. The percentage of patients who discontinued the trial regimen early because of an adverse event was approximately twice as high in the triple-pill group as in the placebo group, most commonly owing to an increase of 20% or more in the serum creatinine level.

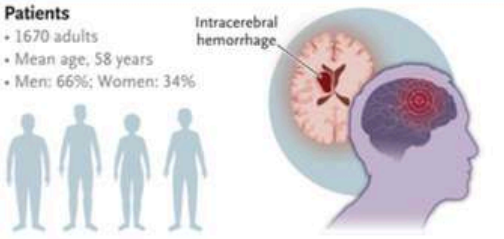
LIMITATIONS AND REMAINING QUESTIONS

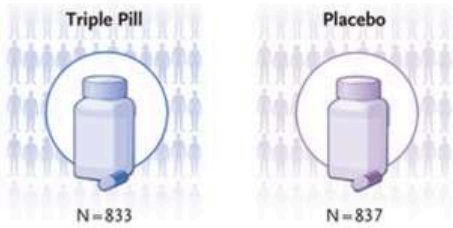
- The majority of the patients were from Sri Lanka, which may limit the generalizability of the results.
- Because the trial included an active run-in phase, the findings apply only to patients who were able to adhere to an initial 2 weeks of therapy without adverse effects.
- The trial protocol excluded patients with an increase in serum creatinine levels of 20% or more, but evidence now suggests that an increase of at least 30% is a more reasonable threshold to trigger clinical evaluation and consideration of dose reduction or cessation.

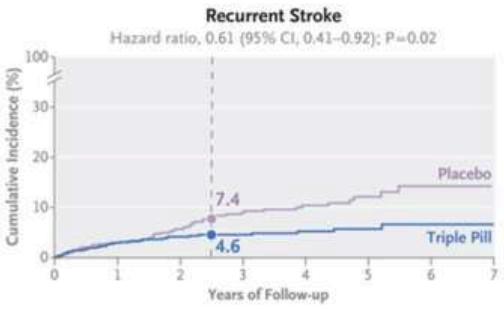
CONCLUSIONS

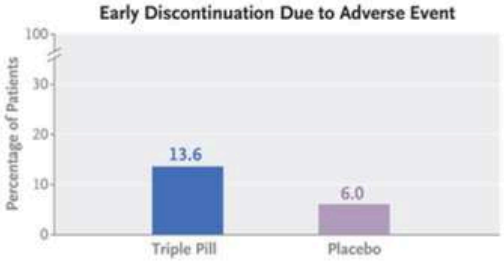
Among patients with intracerebral hemorrhage, treatment with a combination of three low-dose antihypertensive agents in a single pill, in addition to standard care, was associated with a lower incidence of recurrent stroke than placebo.

NEJM QUICK TAKE | EDITORIAL









Update This Month

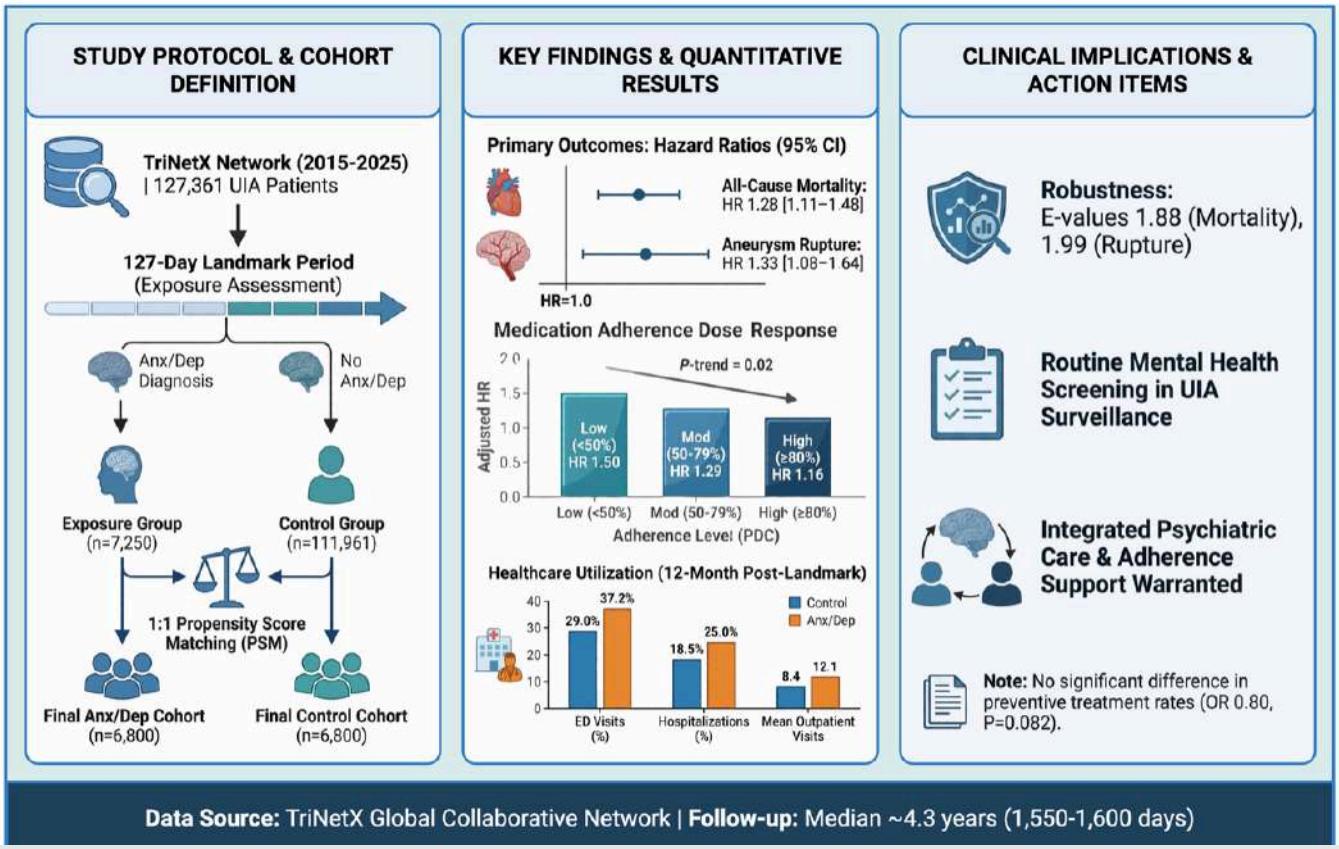
LINK –JULY 2026

WHATS UP!

Postdiagnostic Anxiety and Depression Increase Rupture Risk and Mortality in Unruptured Intracranial Aneurysms

Muhammed Amir Essibayi, MD, MSc ; Ahmed Y. Azzam, MD, MEng ; Hamza Adel Salim, MD ; Alireza Karandish, S. Alhasan, MBBS, DABR ; Dhairya A. Lakhani, MD ; Huanwen Chen, MD ; Dheeraj Gandhi, MD ; Adam A. Dmytriw MSc ; Deepak Khatri, MD ; Neil Haranhalli, MD, MSc; and David J. Altschul, MD ; AUTHOR INFO & AFFILIATIONS

Stroke • Volume 57, Number 5 • https://doi.org/10.1161/STROKEAHA.126.054546



CONCLUSIONS:

Postdiagnostic anxiety and depression are associated with increased rupture risk and mortality in patients with unruptured intracranial aneurysms. Residual confounding cannot be excluded. These findings require further investigation to establish causality and suggest that an integrated psychiatric assessment may be considered in unruptured intracranial aneurysm management.

Update This Month

LINK –JULY 2026

WHATS UP!

ORIGINALLY PUBLISHED JUNE 15, 2026

Left Atrial Appendage Electrical Isolation Reduces Residual Patency After Device Closure in Patients With Persistent Atrial Fibrillation

Peng-Cheng Yao, Mu Chen, Qun-Shan Wang, [...], Yi-Gang Li

Abstract

Background

Left atrial appendage (LAA) patency after catheter ablation and LAA closure (LAAC) is a key determinant of postprocedure stroke. The effect of LAA electrical isolation (LAAEI) on LAA patency after LAAC in patients with atrial fibrillation (AF) remains unknown. Our objective was to explore the effect of LAAEI on LAA patency after catheter ablation and LAAC in patients with AF.

Methods

Patients with persistent AF who underwent combined catheter ablation and LAAC were enrolled. LAA patency was detected by cardiac computed tomographic angiography at 3 months after the procedure.

Results

A total of 686 patients with persistent AF with 3-month device surveillance by cardiac computed tomographic angiography were included initially. Propensity score matching analysis was conducted on the basis of clinical features, echocardiographic parameters and procedural characteristics, and 469 patients were matched, including 363 with non-LAAEI and 106 with LAAEI. Patients in matched groups showed 53.7% complete closure (no patency). Of the 46.3% of cases with LAA patency, visible peridevice leak was seen in 72.8%, and 27.2% had patency without visible peridevice leak. Compared with the non-LAAEI group, the LAAEI group had a lower rate of LAA patency (49.6% versus 34.9%; adjusted odds ratio, 0.69 [95% CI, 0.48–0.98]; *P*=0.040). LAAEI was protective against LAA patency with an odds ratio of 0.68, and the other predictor of contrast leak into the LAA was maximum diameter of LAA orifice (odds ratio, 1.04 [95% CI, 1.00–1.08]; *P*=0.046).

Conclusions

LAAEI is an effective strategy for reducing LAA patency in patients with persistent AF undergoing combined catheter ablation and LAAC.

LINK –JULY 2026

Update This Month

WHATS UP!

ORIGINAL ARTICLE

Medical Management and Revascularization for Asymptomatic Carotid Stenosis

BACKGROUND

Improvements in medical therapy, carotid-artery stenting, and carotid endarterectomy call into question the preferred management of asymptomatic carotid stenosis. Whether adding revascularization to intensive medical management would provide greater benefit than intensive medical management alone is unclear.

METHODS

We conducted two parallel, observer-blinded clinical trials that enrolled patients with high-grade (≥70%) asymptomatic carotid stenosis across 155 centers in five countries. The stenting trial compared intensive medical management alone (medical-therapy group) with carotid-artery stenting plus intensive medical management (stenting group); the endarterectomy trial compared intensive medical management alone (medical-therapy group) with carotid endarterectomy plus intensive medical management (endarterectomy group). The primary outcome was a composite of any stroke or death, assessed from randomization to 44 days, or ipsilateral ischemic stroke, assessed during the remaining follow-up period up to 4 years.

RESULTS

A total of 1245 patients underwent randomization in the stenting trial and 1240 in the endarterectomy trial. In the stenting trial, the 4-year incidence of primary-outcome events was 6.0% (95% confidence interval [CI], 3.8 to 8.3) in the medical-therapy group and 2.8% (95% CI, 1.5 to 4.3) in the stenting group (P=0.02 for the absolute difference). In the endarterectomy trial, the 4-year incidence of primary-outcome events was 5.3% (95% CI, 3.3 to 7.4) in the medical-therapy group and 3.7% (95% CI, 2.1 to 5.5) in the endarterectomy group (P=0.24 for the absolute difference). From day 0 to 44, in the stenting trial, no strokes or deaths occurred in the medical-therapy group and seven strokes and one death occurred in the stenting group; in the endarterectomy trial, three strokes occurred in the medical-therapy group and nine strokes occurred in the endarterectomy group.

CONCLUSIONS

Among patients with high-grade stenosis without recent symptoms, the addition of stenting led to a lower risk of a composite of perioperative stroke or death or ipsilateral stroke within 4 years than intensive medical management alone. Carotid endarterectomy did not lead to a significant benefit. (Funded by the National Institute of Neurological Disorders and Stroke and others; CREST-2 ClinicalTrials.gov number, NCT02089217.)

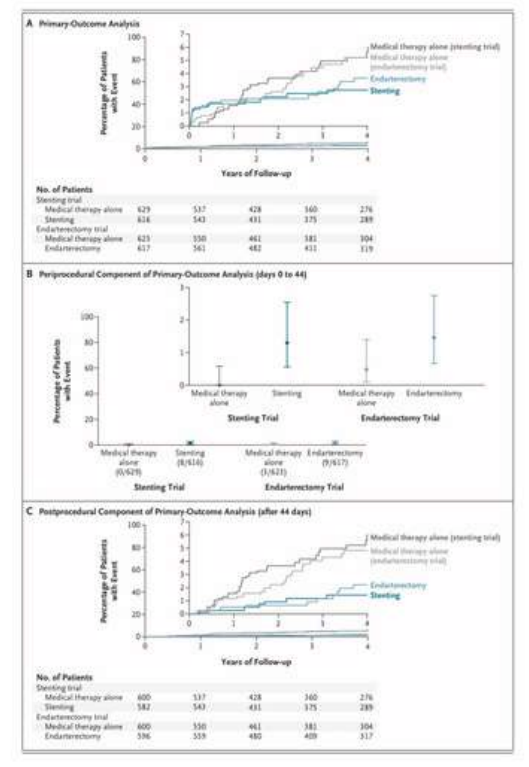
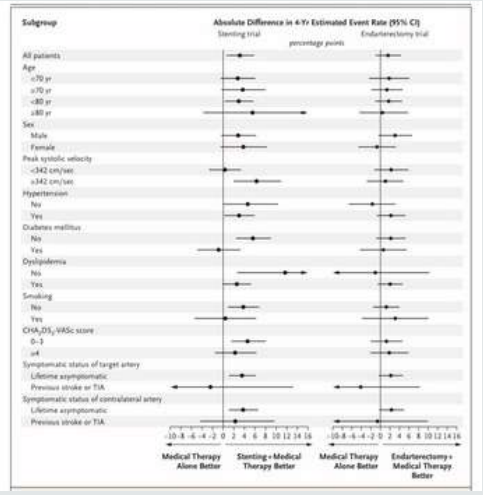


Table 2. Analysis of Primary Outcome and Components.

Variable	Stenting Trial		Endarterectomy Trial	
	Medical Therapy Alone	Stenting	Medical Therapy Alone	Endarterectomy
Primary 4-yr composite outcome*				
Event rate (95% CI) — %	6.0 (3.8 to 8.3)	2.8 (1.5 to 4.3)	5.3 (3.3 to 7.4)	3.7 (2.1 to 5.5)
Absolute difference (95% CI) — percentage points†	3.2 (0.6 to 5.9)		1.6 (−1.1 to 4.3)	
P value for difference	0.02		0.24	
Relative risk (95% CI)†	2.13 (1.15 to 4.39)		1.43 (0.78 to 2.72)	
Components of primary outcome				
Periprocedural period: stroke or death				
No. of events/no. of patients	0/629	8/616	3/623	9/617
Percent of patients with event (95% CI)	0.0 (0.0 to 0.6)	1.3 (0.6 to 2.5)	0.5 (0.1 to 1.4)	1.5 (0.7 to 2.8)
Difference (95% CI) — percentage points	−1.3 (−2.2 to 0.4)		−1.0 (−2.1 to 0.1)	
Postprocedural period: ipsilateral ischemic stroke				
No. of person-yr	1686	1714	1761	1823
No. of events/no. of patients	28/600	7/582	23/600	10/596
Annual event rate per person-yr (95% CI) — %	1.7 (1.1 to 2.4)	0.4 (0.2 to 0.9)	1.3 (0.9 to 2.0)	0.5 (0.3 to 1.0)
Relative risk (95% CI)	4.07 (1.78 to 9.31)		2.38 (1.13 to 5.00)	



LINK –JULY 2026

Update This Month

WHATS UP!

Selective Decontamination of the Digestive Tract during Ventilation in the ICU

Author: The SuDDICU Investigators for the Australia and New Zealand Intensive Care Society Clinical Trials Group and the Canadian Critical Care Trials Group Author Info & Affiliations

ABSTRACT

BACKGROUND

Whether selective decontamination of the digestive tract (SDD) reduces mortality among patients undergoing mechanical ventilation and whether it adversely affects microbial ecology in the intensive care unit (ICU) remain unclear. In an earlier analysis of data from Australia, SDD did not result in a lower incidence of in-hospital death than standard care, but data from the full international trial are needed.

METHODS

We randomly assigned ICUs in Australia and Canada to use SDD or to continue standard care for two 12-month periods in patients undergoing mechanical ventilation. Patients in the SDD group received specific oral and gastric antimicrobial interventions for the duration of ventilation and an intravenous antibiotic agent for the first 4 days after enrollment. All other patients in the ICU were included in an observational ecologic assessment. Previously reported data from Australia are now combined with data from Canada. The primary outcome was in-hospital death from any cause at 90 days. The secondary clinical outcomes, assessed at 90 days, were death in the ICU and the number of days alive and free of mechanical ventilation, ICU admission, and hospitalization. Microbiologic secondary outcomes included new positive cultures for bloodstream infections and antibiotic-resistant organisms. For the ecologic assessment, the microbiologic outcomes were tested for noninferiority (noninferiority margin, 2 percentage points).

RESULTS

In this trial involving 20,000 patients in 26 ICUs, 9289 patients were enrolled in the randomized trial and 10,711 were included in the ecologic assessment. At 90 days, 1175 of 4215 patients (27.9%) in the SDD group and 1494 of 5065 (29.5%) in the standard-care group had died before hospital discharge (odds ratio, 0.94; 95% confidence interval [CI], 0.84 to 1.05; P=0.27). New bloodstream infections occurred in 4.9% of the patients in the SDD group and in 6.8% of those in the standard-care group (adjusted mean difference, –1.30 percentage points; 95% CI, –2.55 to –0.05); antibiotic-resistant organisms were cultured in 16.8% and 26.8%, respectively (adjusted mean difference, –9.60 percentage points; 95% CI, –12.40 to –6.80). In the ecologic assessment, noninferiority of SDD was not confirmed for the development of new antibiotic-resistant organisms. Adverse events considered to be related to SDD or standard care were reported in 12 patients (0.3%) in the SDD group and in no patients in the standard-care group. Serious adverse events occurred in 47 patients (1.1%) and 59 patients (1.2%), respectively.

CONCLUSIONS

Among critically ill patients undergoing mechanical ventilation, SDD did not result in a lower incidence of in-hospital death than standard care. (Funded by the National Health and Medical Research Council of Australia and the Canadian Institutes of Health Research; ClinicalTrials.gov number, NCT02389036.)

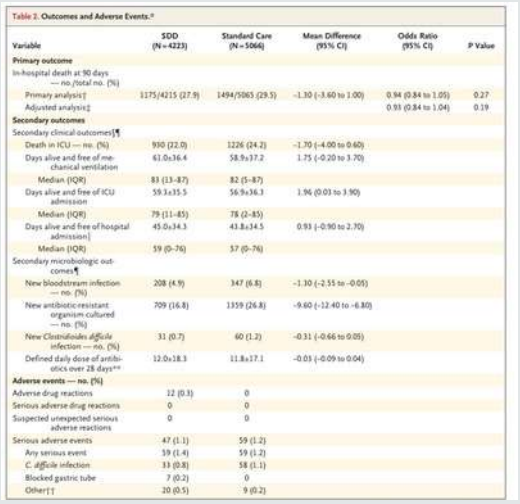


Table 2. Outcomes and Adverse Events.*

Variable	SDD (N=4223)	Standard Care (N=5066)	Mean Difference (95% CI)	Odds Ratio (95% CI)	P Value
Primary outcome					
In-hospital death at 90 days — no./total no. (%)					
Primary analysis†	1175/4215 (27.9)	1494/5065 (29.5)	−1.30 (−3.60 to 1.00)	0.94 (0.84 to 1.05)	0.27
Adjusted analysis‡				0.93 (0.84 to 1.04)	0.19
Secondary outcomes					
Secondary clinical outcomes§¶					
Death in ICU — no. (%)	930 (22.0)	1226 (24.2)	−1.70 (−4.00 to 0.60)		
Days alive and free of mechanical ventilation	61.0±36.4	58.9±37.2	1.75 (−0.20 to 3.70)		
Median (IQR)	83 (13–87)	82 (5–87)			
Days alive and free of ICU admission	59.3±35.5	56.9±36.3	1.96 (0.03 to 3.90)		
Median (IQR)	79 (11–85)	78 (2–85)			
Days alive and free of hospital admission‖	45.0±34.3	43.8±34.5	0.93 (−0.90 to 2.70)		
Median (IQR)	59 (0–76)	57 (0–76)			
Secondary microbiologic outcomes¶					
New bloodstream infection — no. (%)	208 (4.9)	347 (6.8)	−1.30 (−2.55 to −0.05)		
New antibiotic-resistant organism cultured — no. (%)	709 (16.8)	1359 (26.8)	−9.60 (−12.40 to −6.80)		
New *Clostridioides difficile* infection — no. (%)	31 (0.7)	60 (1.2)	−0.31 (−0.66 to 0.05)		
Defined daily dose of antibiotics over 28 days**	12.0±18.3	11.8±17.1	−0.03 (−0.09 to 0.04)		
Adverse events — no. (%)					
Adverse drug reactions	12 (0.3)	0			
Serious adverse drug reactions	0	0			
Suspected unexpected serious adverse reactions	0	0			
Serious adverse events	47 (1.1)	59 (1.2)			
Any serious event	59 (1.4)	59 (1.2)			
C. difficile infection	33 (0.8)	58 (1.1)			
Blocked gastric tube	7 (0.2)	0			
Other††	20 (0.5)	9 (0.2)			

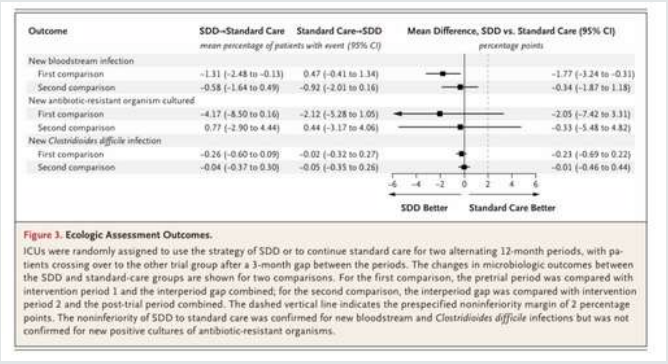


Figure 3. Ecologic Assessment Outcomes.

ICUs were randomly assigned to use the strategy of SDD or to continue standard care for two alternating 12-month periods, with patients crossing over to the other trial group after a 3-month gap between the periods. The changes in microbiologic outcomes between the SDD and standard-care groups are shown for two comparisons. For the first comparison, the pretrial period was compared with intervention period 1 and the interperiod gap combined; for the second comparison, the interperiod gap was compared with intervention period 2 and the post-trial period combined. The dashed vertical line indicates the prespecified noninferiority margin of 2 percentage points. The noninferiority of SDD to standard care was confirmed for new bloodstream and *Clostridioides difficile* infections but was not confirmed for new positive cultures of antibiotic-resistant organisms.

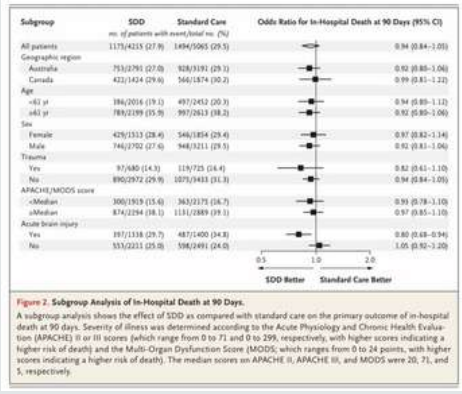
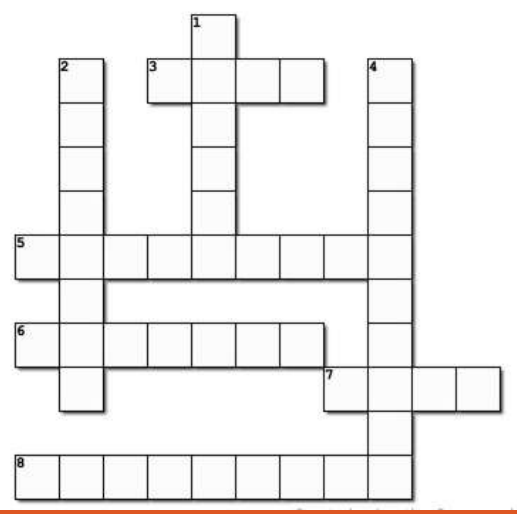


Figure 2. Subgroup Analysis of In-Hospital Death at 90 Days.

A subgroup analysis shows the effect of SDD as compared with standard care on the primary outcome of in-hospital death at 90 days. Severity of illness was determined according to the Acute Physiology and Chronic Health Evaluation (APACHE) II or III scores (which range from 0 to 71 and 0 to 299, respectively, with higher scores indicating a higher risk of death) and the Multi-Organ Dysfunction Score (MODS; which ranges from 0 to 24 points, with higher scores indicating a higher risk of death). The median scores on APACHE II, APACHE III, and MODS were 20, 71, and 5, respectively.

JUMBLE

LINK–NINTH EDITION– JULY, 2026



Across

4. A 10-point quantitative topographic score used on a baseline non-contrast head CT to assess early ischemic changes.
5. A European trial evaluating thrombolysis in stroke patients with an unknown time of onset using MRI mismatches.
6. An etiology where a blood clot travels from another part of the body to occlude a cerebral artery.
7. A structural stroke etiology involving a tear in the tunica intima of a carotid artery neck

Down

1. Signals detected on transcranial Doppler, indicating microemboli passing through the cerebral circul
2. A type of advanced CT or MRI imaging modality that maps blood flow delivery to help identify salvageable tissue penumbra.
3. The standard intravenous thrombolytic drug administered to eligible patients within 4.5 hours of ischemic stroke onset.
7. Landmark 2018 trial that expanded the mechanical thrombectomy window up to 24 hours for patients with clinical-core mismat

OUR GO-GETTERS

Dr. Shivani Fotedar,
DRNM Trainee, Artemis Health Institute, Gurugram

-Recived Patent grant - May 2026
Granted Patent on therapeutic nipple eversion system which overcomes lactational failiure.
-Best Reviewer Award – Indian Journal Of Clinical AnesthesiaMarch 2026

Dr. Seham Syeda
Consultant, Ramaiah Medical College, Bengaluru

Secured First place in poster presentation at the World Critical Care and Anaesthesiology Conference held in Singapore. Title: TCD on Prognostication of Traumatic Brain Injury.

Dr. Gudela Mohan Sai,
Consultant,
KIMS Hospital,
Visakhapatnam

Won the Young Talent Search in ISCCM conference held in Chennai on Feb 2026

BREWING UP


KOLKATA
NCSI 2026
DATE
11-12 September - Conference
13th September - Workshop
VENUE
Taj Taalkutir, Newtown, Kolkata
FROM BASICS TO BREAKTHROUGHS:
ADVANCING NEUROCRITICAL CARE
To serve to worship

BREWING UP


ISCCM
INDIAN SOCIETY OF CRITICAL CARE MEDICINE
Standardizing Critical Care
CRITICARE 2027
Bridging the Gap from Standards to the Bedside
Welcome to CRITICARE 2027, Agra.
ISCCM
INDIAN SOCIETY OF CRITICAL CARE MEDICINE
CRITICARE 2027
33RD ANNUAL CONFERENCE OF INDIAN SOCIETY OF CRITICAL CARE MEDICINE
4-7 February 2027
Jaypee Palace Hotel & Convention Centre, Agra
Standardizing Critical Care: Bridging the Gap from Standards to the Bedside


World Stroke Organization
18TH WORLD STROKE CONGRESS
2026 OCTOBER 21 — 23
SEOUL, KOREA
COMBINED WITH INTERNATIONAL CONFERENCE STROKE UPDATE
ONE WORLD VOICE FOR STROKE
one voice

BREWING UP


28th Annual Conference of the Indian Society of
Neuroanesthesiology and Critical Care
ISNACC
2027
Bhubaneswar
Bhubaneswar,
Odisha
Conference 21 22 23 January 2027
Workshop 24 January 2027

BREWING UP

NCS ANNUAL MEETING

24th Annual Meeting

October 20-23, 2026
Seattle Convention Center
Seattle

Save the date to "Reach New Heights" with the NCS community at the NCS 24th Annual Meeting — held October 20-23, 2026, in Seattle.

BREWING UP


9TH CONGRESS OF
ASNACC
Kuching, Malaysia • 2027
www.asnacc2027.org
9th Congress of the Asian Society for Neuroanesthesia & Critical Care
Neuro Synergy:
Together Towards Excellence in Care
6 – 9 May 2027
Borneo Convention Centre Kuching, Sarawak, Malaysia
ABSTRACT ENTRIES ARE OPEN
ABSTRACT THEMES
Basic Science and Clinical Research related to perioperative neuroscience (neuroanaesthesiology and neurocritical care)
SUBMIT ABSTRACT

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