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Reimagining bell's palsy care: integrating evidence-based medicine, surgical innovation, and Neurorehabilitative advances. A narrative review.

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Abstract

Bell's palsy is the most common cause of acute unilateral lower motor neuron facial paralysis and is widely attributed to herpes simplex virus type 1 reactivation, leading to intraneural inflammation and edema within the narrow labyrinthine segment of the Fallopian canal. Although most patients recover spontaneously, a substantial proportion experience incomplete recovery, chronic weakness, or synkinesis, underscoring the need for optimized, evidence-based management. Current evidence consistently supports early corticosteroid therapy within 72 hours of symptom onset as the cornerstone of treatment, significantly improving rates of complete recovery. Adjunctive antiviral therapy appears to confer additional benefit only in selected patients with severe paralysis or suspected viral reactivation. Electrodiagnostic studies, particularly electroneuronography and electromyography, remain critical for prognostication and surgical decision-making, with ≥90% degeneration predicting poor spontaneous recovery and identifying the small subset of patients who may benefit from early facial nerve decompression via transmastoid or middle cranial fossa approaches. For patients with irreversible paralysis, late-stage facial reanimation techniques, including nerve transfers, cross-facial nerve grafting, and free functional muscle transfer, offer meaningful improvements in dynamic facial movement and symmetry. Rehabilitation plays a central role across all stages of care. Neuromuscular retraining, biofeedback, photobiomodulation, and targeted botulinum toxin therapy have demonstrated benefit in improving functional outcomes and reducing synkinesis. Emerging technologies, such as three-dimensional photogrammetry, deep-learning-based facial movement analysis, and multi-omics profiling, promise greater diagnostic precision and individualized treatment planning. Overall, contemporary management of Bell's palsy requires a multidisciplinary, evidence-based approach integrating early medical therapy, selective surgical intervention, structured rehabilitation, and evolving AI-driven assessment tools to optimize patient outcomes.

Keywords**:** Bell's palsy; facial nerve paralysis; corticosteroids; antiviral therapy; electrodiagnostic studies; facial nerve decompression; facial reanimation surgery; neuromuscular retraining; botulinum toxin; photobiomodulation; synkinesis; herpes simplex virus type 1 (HSV-1) reactivation; artificial intelligence; 3D photogrammetry
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Introduction

Bell palsy is an idiopathic, rapid-onset, acute peripheral facial (cranial nerve VII) palsy that presents with hemifacial weakness and develops within hours to days. It is the most frequent cause of isolated peripheral facial paralysis (1). Characteristic symptoms include the inability to close the eye, smile asymmetrically, experience drooling, and suffer from consequent otalgia, hyperacusia, and disturbed taste (1). Diagnosis is based on clinical signs and exclusion of other causes that include central lesions, Lyme disease, Ramsay-Hunt syndrome, neoplasm, or inflammatory neuropathies (1). The epidemiologic literature indicates a yearly incidence of about 11 to 40 per 100 000 population, with the highest incidences in young and middle adulthood and a second high increase in older ages (2). Reproducible risk factors related to increased incidence and poor initial severity are diabetes mellitus and pregnancy, especially in the third trimester and postpartum period (2). Modern pathophysiologic hypotheses focus on inflammation by the virus and intraneural edema in the limited-diameter Fallopian canal as the key pathogenesis. Molecular and PCR research indicate viral herpes simplex virus type 1 (HSV-1) reactivation in the geniculate ganglion in most cases. Other infectious or immune triggers, such as varicella-zoster virus, are less prevalent. A variable extent of axonal degeneration may be induced by inflammatory swelling of the canal, which can trigger ischemia and a fluctuating level of axonal degeneration related to the duration and severity of compression (3). Therapeutically, early corticosteroid therapy, administered within the first 72 hours of symptom onset, offers the highest chances of full functional recovery(4). The use of antivirals as adjunctive therapy is controversial: a statistical fragility analysis established that combination steroid-antiviral trials were not necessarily robust (5), and a 2025 network meta-analysis by Dehghani et al. demonstrated that a possible extra benefit of combination therapy might exist in severe cases (6). Surgical

Decompression is only used in those patients who have complete or rapidly progressive palsy with greater than 90% degeneration on electrodiagnostic measures. In a 2025 systematic review, Wang et al. discovered that the evidence was not sufficient to recommend the routine use of decompression, but they suggested limiting surgery to those cases that were carefully selected after multidisciplinary assessment (7). Rehabilitative innovations have gained a place in

long-term management. According to Kim et al. (2023), symmetry and facial muscle control were more positively affected by the early neuromuscular retraining therapy in a severe case (8). Marco et al. (2025) found that Neuromuscular retraining combined with botulinum toxin A resulted in much better outcomes and reduction of synkinesis (9). Dagenais et al. (2023) suggested a telerehabilitation protocol that uses mirror therapy and facial retraining, which demonstrated the potential for functional improvement (10). With the frequency of the condition and the possibility of disabling sequelae, and no consistent consensus on the best use of adjunctive or targeted therapy, a strategic review of medical, surgical, and rehabilitative therapeutic innovations is timely. The purpose of the review is to establish the current best practices, areas of knowledge gaps, and future research priorities.

Methods:

This study was conducted as a narrative review aimed at synthesizing and critically appraising contemporary evidence on the medical, surgical, and neurorehabilitative management of Bell's palsy. A comprehensive literature search was performed to identify relevant studies published between January 1990 and December 2025. The following electronic databases were systematically searched: PubMed/MEDLINE and Google Scholar. Search terms included combinations of controlled vocabulary and free-text keywords such as *Bell's palsy*, *facial nerve paralysis*, *herpes simplex virus type 1*, *corticosteroids*, *antiviral therapy*, *electroneuronography*, *electromyography*, *facial nerve decompression*, *facial reanimation*, *neuromuscular retraining*, *botulinum toxin*, *photobiomodulation*, *neurorehabilitation*, and *artificial intelligence*, using Boolean operators as appropriate.

Eligible study designs included systematic and narrative reviews, clinical guidelines, consensus statements, randomized and non-randomized clinical studies, cohort studies, and selected case series that provided meaningful insights into disease mechanisms or management. Case reports were included selectively when they offered novel perspectives. Data were synthesized qualitatively across predefined thematic domains.

Discussion:

Pathophysiology of Bell's palsy

Viral reactivation-induced neuritis of the facial nerve is the most popular theory that explains the palsy in Bell's palsy. The molecular and virological findings have provided evidence of HSV-1 DNA in endoneurial fluid and skeletal muscle, which is in support of the notion that latent reactivation of HSV-1 in the ganglion of the geniculate causes inflammation and acute LMN facial paralysis.

(11). HSV-1 and sometimes VZV reactivation have been established serologically, and stronger viral activity correlates with more adverse clinical features (12). After reactivation, the facial nerve, particularly the labyrinthine part of the Fallopian canal, is inflamed and edematous. This is the most vulnerable part that is prone to compression, as it has low inherent vascular supply (13,

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14). Breakdown of the blood-nerve barrier and active inflammation are manifested in the 3T MRI acute phase, which is characterized by contrast enhancement and swelling of the labyrinthine and geniculate segments (13). Recurring edema can result in ischemia, demyelination, and, in the worst-case scenario, destruction of the axon.

Axonal loss strongly correlates with long-term functional outcomes, and neural injury lies between neurapraxia and Wallerian degeneration. It has been demonstrated through Electrodiagnostic studies that show more than a 90 percent decrease in the amplitude of muscle action potentials indicates an incomplete recovery and increased chances of synkinesis (15). Advanced age, diabetes mellitus, the initial House-Brackmann grade, and delayed corticosteroid initiation have also been consistently associated with poorer outcomes, which are probably caused by poor microvascular reserve and neural regenerative capacity (16-18).

Clinical Presentation and Diagnosis

The presentation of Bell palsy is usually that of acute unilateral lower motor neuron facial paralysis, which reaches its peak weakness in 72 hours. The patients tend to complain of the inability to close the eye, reduced brow rise, loss of nasolabial fold, loss of muscle to smile, drooling, and loss of ability to retain liquids (16, 17). Other symptoms can also be retroauricular pains, hyperacusis, and loss of taste on the anterior two-thirds of the tongue (16, 17). The involvement of the forehead assists in distinguishing between LMN Bell and central lesions, where the forehead remains unaffected (16).

Since palsy is a diagnosis of exclusion in Bell, clinicians need to examine other causes in typical, bilateral, progressive, recurrent, or abnormally painful cases. The indicators of a red flag include vesicles that are suggestive of Ramsay-Hunt syndrome, Lyme, Guillain-Barré, recurring ipsilateral weakness (possible tumor), chronic otologic disease, or central neurologic symptoms (16-18). Imaging is not necessary in common situations, but MRI is suggested when the symptoms are non-resolving, long-term, and atypical (18).

Electrodiagnostic tests are unnecessary in diagnosis but significant in prognostication. The ENoG, which should ideally be performed 3-14 days after the onset of symptoms, evaluates the extent of nerve degeneration by comparing the amplitude ratios of both sides; a degeneration rate greater than 90% is a reliable predictor of poor recovery and serves as a critical threshold for surgical decompression (15, 19). The EMG of the needle assesses voluntary muscle recruitment and denervation. The fibrillation potentials, which indicate absent recruitment of preserved motor, a high number of units portends a bad prognosis (19, 20). In combination with clinical grading scales, ENoG and EMG can be used to a large extent to predict recovery and inform decisions on early rehabilitation or surgical referral

Medical Management of Bell's Palsy

The gold-standard and first-line treatment for Bell's palsy is corticosteroid therapy, which has the strongest evidence for enhancing both the frequency and total recovery of the facial nerve. The effect of therapy is based on the decreased edema of nerves, inflammation, and compression in the small Fallopian canal. Giant randomized controlled studies have confirmed that oral prednisone started within 72 hours is associated with higher chances of complete recovery over placebo (21, 22). The most common treatment is prednisone 50-60 mg/day for 5 days with a tapering progression in another 5 days (21). Systemic corticosteroids are highly recommended as the main treatment of any acute cases unless it is contraindicated by national and international guidelines, and even based on the Japan Society of Facial Nerve Research (23). Initial delay later than 72 hours is always linked with decreased effectiveness (21, 22).

Potential reactivation of the HSV-1, which is an etiologic agent widely implicated in Bell palsy, is targeted by antiviral agents, including acyclovir or valacyclovir. Nevertheless, there is still some contradicting evidence about their advantage. Although anecdotal and subgroup evidence indicate that the addition of antivirals to corticosteroids might have some added effect in severe (House-Brackmann V-VI) disease or that of a suspected viral cause (e.g., high levels of prodromal pain) (21,24), large randomized trials and Cochrane reviews have found no significant effect of the addition of antivirals to steroid treatment (22,25). As a result, the majority of the guidelines advise against the use of antivirals in general but select high-risk patients (those with complete paralysis, severe axonal degeneration, or those who are suspected of having herpes zoster or HSV-1 reactivation) instead.

Positive management is critical and can be the determinant of functional performance in the long run. Eyeglasses are essential where the cornea is exposed to the danger of being dry, abraded, or even ulcerated due to incomplete eyelid closure. It has been suggested to have regular lubricating drops, night ophthalmic ointments, and eyelid taping to ensure the corneal moisture and avoid exposure keratopathy (21, 26). Augmentin is usually sufficient; pain in the retroauricular region is common, and analgesics like NSAIDs or acetaminophen are usually sufficient. Facial massage and early gentle movement can help lessen the pain, but are not a substitute for the structured adjunctive medical therapies.

A number of non-pharmacological and adjunctive interventions have demonstrated potential in changing recovery or lowering the long-term synkinesis, but the data are still preliminary.

Randomized controlled trials have shown that the inclusion

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of facial nerve neural mobilization exercises in the usual care enhances the facial symmetry and muscle patterns as well as patient reports of functional recovery in comparison to the usual care only (27). Such effects are explained by increased neural gliding, decreased intraneural adhesions, and better recruitment of motor units. Acupuncture is also becoming a field of research as an additional treatment.

Systematic reviews indicate that acupuncture has the potential to improve functional scores and facial nerve regeneration, especially when used in combination with corticosteroid therapy.

(28). Nonetheless, issues with methodology and inter-trial heterogeneity lead to the need for interpretation.

It seems that low-level laser therapy, or photobiomodulation, facilitates the activation of mitochondria and the regeneration of axons. The small clinical studies and case series show quicker enhancement in facial muscle functioning and less neuropathic pain (29). Although encouraging, high-quality RCTs should be adopted prior to routine use.

New data indicate that repetitive TMS can also induce cortical and subcortical facial motor circuits, which enhance the synaptic plasticity and the functional recovery. The early studies reveal the enhancement of symmetry and oral motor control with the help of TMS in combination with traditional therapy (30). Currently, TMS is experimental, yet it is one of the most promising neuromodulatory adjuncts.

Surgical intervention is considered only in complete Bell's palsy, accompanied by a poor prognosis demonstrated on electrophysiologic testing[32]. The House–Brackmann scale remains the standard measure of clinical severity, while prognostication heavily depends on ENoG and EMG. [33] ENoG performed between days 4 and 14 after onset is critical because it captures the extent of Wallerian degeneration; ≤25% degeneration predicts nearly 98% satisfactory recovery without surgery, whereas ≥90% degeneration within 14 days suggests a poor prognosis and is the primary surgical indication. [33, 3 2] EMG is used adjunctively when degeneration reaches ≥90%, as the presence of active motor units indicates retained axonal integrity and therefore a more favorable natural recovery. [34] Age also influences decision-making: outcomes are consistently poorer in patients ≥60 years old. [35] Facial nerve decompression is most effective when performed in the first 3 months after onset; outcomes decline significantly beyond this window, emphasizing the importance of early identification of non-recovering patients. [36]

Surgery in the Early Stage: Decomposition of the Facial Nerve:

Transmastoid Approach

The transmastoid method involves the separation of the nerve between the ganglion of the geniculate and the stylomastoid foramen and is therefore appropriate to lesions that involve only the tympanic and mastoid parts [37]. The clinical evidence available is inconclusive. The future comparison showed that there was no significant enhancement in complete recovery when decompression was carried out between 21 and 70 days against medical therapy alone (75% vs. 70%, p=0.746).[38]. Anatomical considerations have a strong impact: poorer prognosis after decompression is linked to bony dehiscence of the facial canal. [39]. In patients with distal pathological involvement (≈6%), the extension through posterior tympanotomy is suggested.

Middle Cranial Fossa (MCF) Approach.

The most effective approach to reach the labyrinthine segment, which is the smallest part of the facial canal and the most susceptible during viral reactivation-induced edema, is the MCF approach [41]. This technique enables simultaneous visualization of both the internal auditory canal and geniculate ganglion and has the benefit of preservation of hearing [34]. Although the process is better exposed to, it has increased risk, such as hemorrhage, seizures, and CSF leakage, because of temporal lobe manipulation and is thus restricted to select centers only [42]. It has been demonstrated through meta-analyses that MCF decompression has the potential to provide functional benefit when performed within 14 days following onset in individuals with

>90% degeneration,[43] but again, the evidence is inconsistent and relies heavily on small cohort studies. It has been suggested to use intraoperative EMG to localize pathological segments and the extent of decompression directions [40].

In the contemporary literature, there is no consensus that a high dose of steroid therapy performs better than decompression, either transmastoid or MCF, which is to be used on most patients. A Cochrane review of 2021 identified a lack of high-quality evidence to make a recommendation to use decompression as a routine intervention because of inconsistent indications, timing, and reporting of complications. [31]. However, it can work in a small, stringently selected group of those with complete paralysis, ≥90% degeneration, and no voluntary recruitment of motor units on EMG.

Late-Phase Surgery: Facial Reanimation Surgeries:

The patients who come with irreversible paralysis of above 12-24 months (irreversible) should be reanimated, not decomposed. These procedures have the purpose of either recovering symmetry at rest (static techniques) or reestablishing active movement (dynamic techniques). Static Procedures

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There is an enhancement of resting facial symmetry and ocular protection in static reanimation, but it is unable to restore motion [44].

Gold Weight Implantation: The most common way of treating lagophthalmos, this method can offer a theory of gravity-assisted eyelid closure, which has high success rates and a low complication profile [45, 46].

Fascial Slings: Autologous fascia lata or biomaterials lend support to the oral commissure and midface, enhancing oral competence and symmetry. [47, 48].

Rejuvenation Techniques: Endoscopic brow lifts and midface lifts treat asymmetry and ptosis, which may need to be combined to make the best of the periorbital aesthetics
[49-51].

Dynamic Procedures

Dynamic reanimation involves the restoration of voluntary movement through mobilization of operating muscle or nerve sources [52].

Nerve Grafts: Motor nerve grafts have been shown to recover better when used to fill gaps, in particular in longer gaps, or where the area has been irradiated in the past [53, 54].

Nerve Transfers: Hypoglossal-facial or masseter transfer: These provide powerful, consistent reinnervation in the event of irreversible damage to the upper facial nerve. Dual nerve transfers enhance tone and spontaneous movement.[55, 56].

Cross-Facial Nerve Grafting (CFNG): Ideally should be done in the first 6 months of injury; results are better when it is combined with masseteric co-innervation. [57].

Free Functional Muscle Transfer (FFMT): The gracilis free flap is the standard when it comes to long-term paralysis. CFNG + masseteric nerve has been reported to increase excursion and spontaneity [58, 59].

Rehabilitation is necessary after surgery and among medically ill patients. The most common are neuromuscular re-education, biofeedback, mime therapy, and electrotherapies.

Neuromuscular retraining enhances symmetry through training proper motor patterns backed up by RCT evidence of superiority over conventional therapy [60]. There are no notable differences between modalities in enhancing voluntary control in biofeedback (EMG-based or mirror). [61] Mime therapy enhances coordination in the long term and diminishes the synkinesis [62].

Variably but occasionally beneficial effects of electrical stimulation, laser therapy, or diathermy as modalities of electrotherapy have been reported in chronic arthralgia [63-65]. Botulinum toxin and fillers are used to treat synkinesis and asymmetry, especially in chronic or post-surgical patients.[66]

The future of facial palsy care is being transformed by the development of imaging, the discovery of biomarkers, and evaluation with the help of AI.

The multi-omics analysis has revealed that SLIT2-ROBO signaling was a potential therapeutic option in the treatment of peripheral facial paralysis, and it has highlighted novel nerve repair and immune regulation pathways[67]. New techniques in assessment have dynamic 3D photogrammetry with deep-learning that has an accuracy on facial movement quantification of over 95 percent, providing objective pre- and post-surgery assessment.[68]. Video-based video tools using machine learning have the potential to categorize palsy patterns and follow recovery patterns to assist in making individual-specific decisions during surgical planning [69]. Also, the photobiomodulation therapy still remains promising, but more extensive controlled trials are needed [70].

The majority (70-90% of patients) of the patients respond to medical treatment, but the poorer prognosis is always linked to advanced age, diabetes, high initial House-Brackmann grade, and late initiation of the steroid therapy [35].

Extensive axonal injury (>90% loss) significantly raises the risk of impaired recovery and synkinesis, which supports the need to have electrophysiologic assessment in an early stage of injury treatment. [34]. Prior surgery (less than 3 months in the case of decompression, less than 6 months in the case of dynamic transfer) has better historical outcomes, but at late-stage paralysis, there is a need to do reanimations.

Limitations

Despite advances in our knowledge and treatment of Bell's palsy, there are still a number of inherent limitations with the current body of evidence. Heterogeneity in study designs, outcome measures, and follow-up periods, along with inconsistent definitions of recovery and varying facial grading systems, poses a significant challenge to cross-study comparability and undermines pooled analyses. Although there is strong randomized evidence to support corticosteroid therapy, information about neuromodulatory therapies, facial nerve decompression, adjunctive antivirals, and a number of rehabilitative interventions is mostly derived from small trials, retrospective cohorts, or single-center case series, which introduces selection bias and limited statistical power. It is particularly challenging to draw firm conclusions about efficacy in the surgical literature due to non-randomized designs, inconsistent patient selection criteria, and variations in timing, surgical technique, and electrophysiologic thresholds. Prognostic tools such as electroneuronography and electromyography, although widely used, lack standardized protocols across institutions, resulting in variability in interpretation and

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predictive accuracy. Rehabilitation studies frequently demonstrate methodological limitations, including heterogeneous treatment protocols, limited blinding, and insufficient long-term follow-up. Finally, emerging diagnostic and prognostic technologies, including artificial intelligence–based facial analysis and molecular profiling, remain largely exploratory, with limited external validation and uncertain clinical applicability.

Future Directions

Future research in Bell's palsy is increasingly focused on improving prognostication, standardizing outcomes, and individualizing treatment strategies. Large, multicenter randomized controlled trials are needed to better define the role, timing, and patient selection criteria for facial nerve decompression and advanced rehabilitative interventions, using uniform electrophysiologic thresholds and validated outcome measures (7,31). The integration of objective facial assessment tools, including three-dimensional photogrammetry and deep-learning–based motion analysis, has demonstrated high accuracy in quantifying facial movement and holds promise for standardized longitudinal assessment and trial endpoints (68,69). Advances in molecular research suggest that multi-omics profiling may identify novel biomarkers and therapeutic targets involved in neural repair and immune regulation, potentially enabling earlier risk stratification and targeted interventions (67). Rehabilitation research is moving toward multimodal and staged approaches, combining neuromuscular retraining with botulinum toxin or neuromodulation, supported by longer follow-up and protocol standardization (9). Additionally, tele-rehabilitation platforms and remote monitoring strategies have emerged as promising tools to enhance accessibility, adherence, and continuity of care, particularly in resource-limited settings (10). Collectively, these directions reflect a shift toward precision-based, technology-assisted, and multidisciplinary management of Bell's palsy.

Conclusion

Bell palsy is managed surgically through a small group of patients who are completely paralyzed and whose prognosis is poor in an objective manner, according to electrophysiologic measures.

Decompression at an early stage can only be of benefit under highly selective conditions, whereas in irreversible cases of paralysis, functional and aesthetic restoration is offered by
late-phase reanimation. Rehabilitation and newly developed AI-based technologies are crucial to maximizing results. Finally, the treatment of Bell's palsy needs a multidisciplinary strategy that combines early medical treatment, proper choice of surgery, organized rehabilitation, and new diagnostic methods.

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List of Abbreviations

AI – Artificial Intelligence

CFNG – Cross-Facial Nerve Grafting

EMG – Electromyography

ENoG – Electroneuronography

FFMT – Free Functional Muscle Transfer

HB – House–Brackmann

HSV-1 – Herpes Simplex Virus Type 1

LMN – Lower Motor Neuron

MCF – Middle Cranial Fossa

MRI – Magnetic Resonance Imaging

RCT – Randomized Controlled Trial

TMS – Transcranial Magnetic Stimulation

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Conflict of Interest

The author declares no conflict of interest.

Author Contributions

The author was solely responsible for the conception and design of the study, literature search and selection, data synthesis and interpretation, manuscript drafting, and final approval of the version to be published.

Data Availability

No new data were generated or analyzed in this study. All data supporting the findings of this narrative review are derived from previously published literature and are available in the public domain.

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Author biography:

Sana Shaik is a medical graduate of Tbilisi State Medical University, having completed undergraduate training with a strong foundation in clinical medicine and patient care. During medical school, Sana developed robust clinical skills and undertook the CBME surgical training course, which further strengthened exposure to surgical principles and operative care. This early hands-on experience played a key role in shaping a strong interest in cardiology, cardiothoracic surgery, and internal medicine. Alongside clinical training, Sana has been actively involved in research across multiple study designs, including literature reviews, systematic reviews, meta-analyses, case reports, and cross-sectional studies. This combination of clinical competence, structured surgical training, and diverse research experience highlights Sana's potential to contribute meaningfully to academic medicine and evidence-based clinical practice.

Bhavana Gouda is a third-year MBBS student at Shimoga Institute of Medical Sciences, Karnataka. Her main interests are clinical medicine, genomics, and computational neuroscience. She actively volunteers for health campaigns and contributes to public health.

Aditi is a third-year MBBS student at ACS Medical College and Hospital with a strong interest in neurosurgery and neurological research. She is driven by curiosity about the brain and its complexities and is motivated to integrate patient care with meaningful research. She approaches her medical training with discipline, consistency, and a genuine eagerness to learn, and is committed to steadily developing both her clinical understanding and research skills.

Eisha is a dedicated medical student at Liaquat University of Medical and Health Sciences, Pakistan, with a strong academic focus and a growing interest in research. She is known for her discipline, perseverance, and ability to engage deeply with complex medical concepts.

Snehakshi Roy is a 4th-year medical student at Gauhati Medical College & Hospital, Guwahati, with a keen interest in clinical medicine and medical research. During her undergraduate education, she has actively participated in community health initiatives and has organized multiple health camps in collaboration with non-governmental organizations, contributing to improved healthcare access and awareness at the community level.

Dr. Nidhi Vadhavekar is a medical professional who earned her MBBS degree from Padmashree Dr. D.Y. Patil School of Medicine in India. She has demonstrated strong leadership skills through her extensive involvement in research, having published over ten articles. As a mentor, Dr. Vadhavekar was instrumental in the research process. Her leadership was evident from the early stages of conceptualization, where she helped shape the study's direction. She continued to lead the effort through the critical phases of revising and finalizing the drafts, ensuring the project's successful completion. This experience highlights her capacity to take initiative and drive a complex scientific undertaking.

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