AOPN
ACADEMY OF PEDIATRIC NEUROLOGY

NEUROBULLETS

Official Bulletin of AOPN

CLINICAL BULLETS FOR PRACTISING PEDIATRICIANS

SILVER JUBILEE EDITION
25 YEARS
October 2026

Every child's brain matters...





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Message from the National Chairperson

Academy of Pediatric Neurology, India



Prof. Jitendra Kumar Sahu
National Chairperson
Academy of Pediatric Neurology, India

It gives me great pleasure to introduce this special edition of the Neuro Bulletin, brought out to commemorate the 25th anniversary of the Academy of Pediatric Neurology, India.

Twenty five years is a meaningful milestone for any professional body. For our Academy, it marks the distance travelled from a small group of clinicians committed to the care of children with neurological disorders to a national community that today shapes training, research and standards of practice across the country. The growth of pediatric neurology in India over these years has been the collective work of many hands, and this anniversary belongs to every member who has contributed to it.

This commemorative edition features concise clinical reviews by leading pediatric neurologists and past presidents of AOPN from across the country. Each contribution distils practical, evidence informed guidance on conditions we encounter in everyday practice. The intention is deliberate: to offer readable, clinically useful summaries that a busy practitioner can turn to at the bedside, and that a trainee can rely on while building a foundation in the specialty. I am grateful to all the contributing authors, whose willingness to share their expertise has given this issue both its depth and its character.

I record my special thanks to Dr Amarjeet Wagh, Editor-in-Chief of the Journal of Academy of Pediatric Neurology, India, and to Dr Jasodhara Chaudhuri and Dr. Varsha Vaidya as Editors, for conceiving and executing this special edition. Bringing together contributions of this range and quality within the timelines of an anniversary issue calls for persistence, editorial judgement and an unusual degree of patience. They have brought all three to this task, and the result speaks for itself. My thanks also go to the wider editorial team, whose work behind the scenes has made this publication possible.

As we mark this silver jubilee, our responsibilities remain clear. Access to specialised neurological care for children is still uneven across our country, and our task in the years ahead is to widen that access, strengthen training in underserved regions, and build the research base that will improve outcomes for our patients. Publications such as the Neuro Bulletin serve that purpose directly, by carrying knowledge to every part of the country where a child with a neurological disorder is being cared for.

I congratulate the editorial team on this issue and commend it to our readers.

SECRETARY MESSAGE

NEUROBULLETIN - 25th NEUROPEDICON 2026, LUCKNOW



Dr. Vinit Wankhede
Secretary, Central AOPN
Academy of Pediatric Neurology

Dear Friends,
Colleagues and Members of the AOPN Family,
My heartfelt greetings to all of you!

It gives me immense joy, pride and gratitude to write this message as Secretary of Central AOPN at this historic milestone of our 25th Glorious Year, and to present this special Silver Jubilee Edition of NEUROBULLETIN during our 25th Annual National Conference, NEUROPEDICON 2025, Lucknow.

25 Glorious Years – A Journey to Cherish
The past 25 years have been a remarkable journey of growth, learning and service. From a small group of passionate child neurologists, AOPN has evolved into a strong national academic body with vibrant chapters across India.

Over these years, AOPN has:
- Helped establish Pediatric Neurology as a distinct academic discipline and brought it closer to pediatricians across the country, with membership growing to 1,100.
- Established its presence in 22 states, with nearly 290 office bearers working actively for the Academy.
- Conducted 160 CMEs, workshops, conferences, conclaves and summits, reaching professionals from metropolitan cities to smaller towns.
- Led public awareness initiatives on Epilepsy, Autism and Developmental Disorders through Neuropodcast, One Minute Neurology and other platforms.
- Strengthened academic publishing through Neurobulletin, books, family handbooks, research collaborations and the Indian Journal of Pediatric Neurology.
- Nurtured the next generation through our 10-year dedicated fellowship programme, with hundreds of pediatricians and child neurologists now serving children in India and abroad.

Being the Secretary of this esteemed Academy at this landmark moment is both a great honour and a humbling responsibility.
This Edition – "25 for 25"
To commemorate this special year, we have curated this edition around "25 Neurobullets – 25 Clinical Pearls in Pediatric Neurological Disorders", designed to provide practical and clinically relevant learning for practicing pediatricians.
I am deeply grateful to all our Past Presidents and Senior Child Neurologists who have contributed their clinical wisdom to make this edition academically rich and practically useful.
Special Appreciation
I place on record my sincere gratitude to Dr. Amarjeet Wagh, Editor-in-Chief, Neurobulletin, whose persistent efforts, editorial dedication and selfless commitment over the past 10 years have ensured that the academic updates and activities of AOPN reach every member. He has truly been the backbone of Neurobulletin.
My sincere thanks also to Dr. Varsha Vaidya and Dr. Jasodhara Chaudhary, the co-editors, and the entire editorial team for their valuable contributions. I also extend my heartfelt appreciation to the Editorial and Organizing Teams of NEUROPEDICON 2026, Lucknow.
Vision for the Next 25 Years

As we step into the next 25 years, our vision is to build an even stronger, more inclusive and globally respected AOPN by:
- Strengthening academically and socially active AOPN chapters across every state.
- Developing AOPN-accredited international fellowships and a uniform training curriculum in Pediatric Neurology.
- Expanding community outreach, early intervention and school health programmes.
- Promoting indigenous research, national registries and evidence-based guidelines.
- Leveraging technology, Artificial Intelligence and tele-neurology to reach every child in need.

Together, let us pledge to make AOPN a global leader in Child Neurology care, training and research.

Wishing you all a wonderful academic feast at Lucknow and happy reading!

Long Live AOPN!

From Editors' Desk

NEUROBULLETS: Clinical Pearls for Practising Pediatricians

A Silver Jubilee Edition of the Academy of Pediatric Neurology (AOPN)

It gives me immense pleasure and a deep sense of gratitude to present NEUROBULLETS – Clinical Pearls for Practising Pediatricians, the official Silver Jubilee edition of the Academy of Pediatric Neurology (AOPN), released during the Silver Jubilee year of academy at NEUROPEDICON 2026 , Lucknow

This edition is more than a commemorative publication; it is a tribute to the vision, dedication, and collective contributions of all those who have nurtured and strengthened AOPN over the past 25 years. In a special gesture of gratitude, this bulletin brings together articles contributed by the past Presidents and Secretaries of our Academy, whose leadership and commitment have shaped its journey. We are equally privileged to include contributions from eminent child neurologists, whose knowledge and clinical experience enrich this special edition.

With NEUROBULLETS, the clinical pearls presented in this edition aim to simplify complex neurological concepts, enhance clinical understanding, and empower practising pediatricians to recognize, evaluate, and manage neurological disorders in children with greater confidence. We believe that sharing knowledge in a concise, clinically meaningful format can help bridge the gap between specialized neurological expertise and the needs of children in everyday clinical settings.

The Silver Jubilee year offers us an opportunity to look back with pride, acknowledge the efforts of those who built this Academy, and look forward with renewed enthusiasm.

Adding another feather to our cap in this remarkable academic journey, we are proud to announce the recent achievement of JAOPN being an indexed journal now, marking a significant milestone in AOPN's continued commitment to academic excellence and the advancement of child neurology. This journal provides an opportunity for clinicians and researchers to contribute original work, generate evidence relevant to Indian children, document our unique clinical experiences, and participate more actively in the global pediatric neurology community.

This progression from learning, to sharing, to researching, and ultimately to creating new knowledge represents the academic maturation of pediatric neurology in India. I extend my heartfelt gratitude to all the past Presidents and Secretaries who have graciously contributed to this special edition, to the eminent child neurologists who have shared their expertise, and to everyone who has worked tirelessly to make this publication possible. We also want to thank all our editors in the editorial board and all reviewers who are have immensely contributed towards both of these publications as Neurobulletin and JAOPN. We are deeply grateful to all the current and past office bearers of AOPN for their continued trust and support over the years. This journey would not have been possible without their unwavering encouragement and faith in us.

As we celebrate 25 years of AOPN, may NEUROBULLETS continue to inspire learning, strengthen clinical practice, and foster a deeper commitment to the neurological health and developmental well-being of every child.

With warm regards and sincere gratitude,



Dr. Varsha Vaidya
Editor



Dr. Amarjeet Wagh
Editor-in-Chief



Dr. Jasodhara Chaudhuri
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NEUROBULLETS
Official Bulletin of AOPN

JAOPN
(Journal of Academy of Pediatric Neurology)

1. Neurocutaneous Syndromes: Practical Clinical Pearls

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1. Multisystem Nature & Examination Essentials

Neurocutaneous syndromes are multisystem disorders involving the brain, skin, eyes, and frequently the kidneys, heart, bones, or endocrine system. A complete skin examination—including the scalp, oral cavity, nails, and an inspection under a Wood's lamp—should be an integral part of every paediatric neurological assessment.

2. Genetics & Inheritance

Most neurocutaneous syndromes follow an autosomal dominant inheritance pattern with frequent de novo mutations. Some occur sporadically, and a few result from postzygotic somatic mosaicism.

3. Age-Dependent Manifestations & Evolution

Recognise age-dependent manifestations, as diagnostic signs often appear sequentially:

- Neurofibromatosis Type 1 (Nf1): Café-au-lait macules frequently precede neurofibromas.
- Tuberous Sclerosis Complex (TSC): Hypomelanotic macules are often the earliest clinical clue.

Children who do not yet meet formal diagnostic criteria require structured longitudinal follow-up rather than premature exclusion.

4. Phenotypic Approach to Differential Diagnosis

Use the presenting phenotype to direct clinical evaluation:

- Tumours: Consider NF1, NF2-related schwannomatosis, TSC, or von Hippel– Lindau (VHL) disease.
- Epilepsy: Consider TSC or Sturge–Weber syndrome (SWS
- Vascular Events: Consider SWS or hereditary haemorrhagic telangiectasia (HHT).
- Progressive Ataxia with Telangiectasia: Consider ataxia–telangiectasia

5. Suspecting NF1 vs. Legius Syndrome

- NF1: Suspect NF1 in a child presenting with multiple café-au-lait macules, axillary or inguinal freckling, neurofibromas, optic pathway gliomas, or characteristic osseous lesions.
- Legius Syndrome: Consider Legius syndrome when café-au-lait macules and intertriginous freckling occur in the absence of neurofibromas, Lisch nodules, or tumours.

6. NF1 Surveillance Protocol

Surveillance in NF1 is primarily clinical and requires annual assessments of blood pressure, neurological status, development, vision, and the skeletal system. Routine screening brain MRIs are not recommended for asymptomatic children; imaging should be reserved for cases where visual, neurological, endocrine, or rapidly progressive clinical abnormalities emerge.

7. Red Flags in Nf1

Persistent pain, rapid enlargement, hardening, or a new neurological deficit within a plexiform neurofibroma is a red flag for malignant peripheral nerve sheath tumour (MPNST). This warrants urgent whole-lesion MRI and immediate specialist referral. Selumetinib is an established targeted therapy for symptomatic, inoperable NF1-associated plexiform neurofibromas.

8. Evaluating Infants for TSC (Tuberous Sclerosis complex)

Actively screen for TSC in infants presenting with seizures or infantile spasms. Examine the skin for hypomelanotic macules and evaluate the brain, kidneys, heart, and eyes. Identifying a pathogenic TSC1 or TSC2 gene variant can confirm the diagnosis before full clinical criteria develop.

9. Managing Infantile Spasms in TSC

Infantile epileptic spasms in TSC require urgent intervention, with vigabatrin as the first-line disease-specific treatment. Serial EEG surveillance in young infants enables treatment initiation prior to or shortly after clinical seizure onset, which can significantly improve developmental outcomes.

10. Lifelong Surveillance in TSC

- TSC requires lifelong multisystem monitoring:
- Brain MRI: Every 1–3 years until approximately 25 years of age.
- Renal, cardiac, ophthal screening: Every 1–3 years.
- Routine Monitoring: Regular assessment of blood pressure, renal function, development, and behaviour.
- Psychiatric Screening: Systematic screening for TSC-associated neuropsychiatric disorders (TAND).

11. Sturge–Weber Syndrome (SWS) Presentation & Diagnosis

A facial port-wine birthmark involving the forehead or upper eyelid (V1 distribution) should prompt an evaluation for SWS. Arrange an urgent ophthalmological assessment to screen for glaucoma and order a specialist-directed, contrastenhanced brain MRI. Note that an early normal MRI does not completely rule out future cerebral involvement.

12. Acute Decompensations in SWS (Sturge–Weber Syndrome)

A new focal neurological deficit, prolonged seizure, severe headache, or developmental regression in SWS mandates urgent medical evaluation. Optimize seizure control, maintain adequate hydration, manage fevers aggressively, and evaluate for stroke-like episodes or raised intraocular pressure.

13. Diagnostic Workup

- Comprehensive evaluation includes:
- Wood's lamp skin examination
- Detailed ophthalmological assessment
- Electroencephalogram (EEG)
- Syndrome-directed MRI protocols
- Systemic imaging (renal, cardiac, skeletal)
- Genetic testing

14. Genetic Testing & Mosaicism

Genetic testing confirms the diagnosis, guides targeted tumour surveillance, and enables accurate genetic counselling. However, a negative result from blood DNA does not rule out somatic mosaicism. High-depth sequencing of affected skin, tumour, or surgical tissue may be required to detect low-level somatic variants.

15. Precision Medicine & Targeted Therapies

Syndrome / Condition	Target / Precision Pharmacotherapy
Neurofibromatosis Type 1 (NF1)	Selumetinib
NF2-Related Schwannomatosis	Bevacizumab
Tuberous Sclerosis Complex (TSC)	Everolimus / Sirolimus
von Hippel–Lindau (VHL) Disease	Belzutifan
PIK3CA-Related Overgrowth Spectrum (PROS)	Alpelisib
Proteus Syndrome	Miransertib
Gorlin Syndrome	Vismodegib / Sonidegib
Hereditary Haemorrhagic Telangiectasia (HHT)	Bevacizumab
Cerebrotendinous Xanthomatosis (CTX)	Chenodeoxycholic acid
Other Neurocutaneous Syndromes	None currently established

- Core Clinical Pearl: "When neurological symptoms seem unexplained, examine the skin; when a neurocutaneous diagnosis is made, surveil the whole child and not merely the brain".

2. Spinal Muscular Atrophy A Top Down View

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Introduction:

Spinal muscular atrophy(SMA) is an autosomal recessively inherited disorder which affects the anterior horn cells in spinal cord and brain stem causing progressive muscle weakness and wasting. Rarely it may inherited as AD or X-linked recessive form also. SMA is the second commonest cause for an inherited (AR) diseases (after cystic fibrosis) with an incidence of 1 in 6000-10,000.

Genetics:

Usually due to loss/mutation of the SMN1 gene which code for low SMN(Survival Motor Neuron) protein at 5q13, which inhibits the neuronal apoptosis. Without SMN protein, specialized nerve cells in the spinal cord (motor neurons) shrink and die.

Pathogenesis:

The pathological hall mark of SMA is the progressive denervation of muscle. This is compensated for in part by reinnervation from an adjacent motor unit, but gaint motor units are thus created with subsequent atrophy of muscle fibres,

Clinical features:

Symmetrical generalised floppiness, poor head control, and inability to sit, stand, or walk independently. The weakness affect lower limb more than upper limbs, proximal more than distal. Tongue fasciculations and selective involvement of axial and inter costal muscles are there. Often associated with absent/reduced reflexes

Due to involvement of intercostal respiratory muscles, there is typical paradoxical abdominal breathing pattern,, bell shaped chest, scoliosis and weak cough.

Children are alert with bright expression with preserved cognitive function. Sensory system, extra ocular muscles, diaphragm and bladder functions are usually unaffected.

Types of SMA:

5 main types; the severity depends on the age of onset and the highest physical milestones reached, which heavily dictates life expectancy

Type 0 (Congenital SMA)

Onset before birth or right at birth. This is the rarest and most severe form. Infants show extreme muscle weakness and breathlessness right from birth.

Type 1 (Infantile onset SMA-Werdnig-Hoffman disease):

Onset before 6 months of age. This is the most common form. Babies cannot sit without support and face serious feeding and breathing challenges.

Type 2 (Intermediate SMA-Dubowitz type):

Onset between 6 and 18 months of age. Children can sit up without help. However, they cannot stand or walk independently.

Type 3 (Juvenile onset SMA-Kugelberg-Welander disease):

Onset after 18 months of age. Children can walk on their own. Over time, some may develop walking difficulties or need a wheelchair later in life.

Type 4 (Adult-onset SMA)

Onset after 18 years or during adulthood. This form causes mild muscle weakness in the legs. People usually have a normal life expectancy

In addition to this there are rarer forms of SMA

- Distal Spinal Muscular Atrophy type V (DSMA-V)
- Spinal and Bulbar Muscular Atrophy (SBMA) – also known as Kennedy's Disease
- Spinal Muscular Atrophy with Lower Extremity Predominance (SMA-LED)
- Spinal Muscular Atrophy with Progressive Myoclonic Epilepsy (SMA-PME)
- Spinal Muscular Atrophy with Respiratory Distress 1 (SMARD1)
- X-linked Infantile Spinal Muscular Atrophy

Diagnosing SMA:

A thorough medical history and a physical exam followed by neurological exam will give an idea about the diagnosis.

- Chest Xray may show thin ribs and scoliosis.
- CPK level may be normal or minimally elevated.
- ECG may serve as a simple and practical tool showing a baseline tremor as an artifact representing muscle fibrillations, more prominent in lead II.
- Blood test to look for mutations or deletions of the SMN1 gene. This test identifies at least 95% of SMA Types I, II, and III, and may reveal if a person is a carrier for SMA. If the SMN1 gene is normal or the individual's history and examination are not typical of SMA, other diagnostic tests include:
- Electromyography to record the electrical activity of the muscles during contraction and at rest.
- Nerve conduction velocity studies to measure the nerve's ability to send an electrical signal .
- Muscle biopsy may be used to diagnose other conditions with symptoms similar to SMA(not always necessary)
- Carrier screening by dose analysis available and is based on semi quantitive real time PCR or MLPA.
- Newborn screening is aimed at identifying presymptomatic SMApatients.DNA extraction from newborn blood spots followed by either liquid microbead array or realtime PCR techniques, have been developed, which helps to identify SMN1 homozygous deletions

Treatment of SMA:

People with SMA can benefit from physical therapy, occupational therapy, rehabilitation to help improve posture, prevent joint immobility and slow muscle weakness and atrophy. Stretching and strengthening exercises may help reduce joint contractures, increase range of motion, and improve circulation. Assistive devices such as supports or braces, orthotics, speech synthesizers, and wheelchairs may be helpful to improve functional independence.

Children with SMA should get proper nutrition to help them maintain weight and promote muscle function. People who cannot chew or swallow may require a feeding tube. Non-invasive ventilation can improve breathing during sleep, and some individuals also may require assisted ventilation during the day due to muscle weakness of the respiratory muscles

Newer medical treatment:

The FDA-approved drugs for SMA include-

- Nusinersen (Spinraza™) increases production of the SMN protein and is approved to treat children and adults with SMA. This medication is administered via intrathecally.
- Onasemnogene abeparovec-xioi (Zolgensma™) is a gene therapy for children less than two years old who have infantile-onset SMA (Type I). This therapy replaces the SMN1 gene function by delivering a new working SMN gene to the person's motor neurons. This treatment has been shown to improve muscle movement, function, and survival.
- Risdiplam (Evrysdi™) is an orally administered drug to treat people age two months and older. Risdiplam works by increasing the concentration of SMN protein in the body.

Genetic counselling depending on carrier screening tests or in the presence of a previously affected child with SMA.

Prenatal diagnosis should be offered to families with an index patient in the family. Antenatal screening by chorionic villus sampling between 10th and 12th gestational week of pregnancy may serve for SMN1 deletion or mutation analysis

Conclusion: SMA is a monogenic condition caused by by homozygous deletion of 5q13 gene(95% of all SMA) that is critical for viability of motor neuron. SMA is a leading cause of inherited death in infancy and is the second most common AR inherited diseases. The disease is characterised by progressive loss of lower motor neuron nerve. Progressive weakness and muscle atrophy is the end result. Severity of the disease depends on type of SMA.

Considered previously untreatable with high mortality, now beginning to have various interventions and antenatal diagnosis. Early diagnosis and early management would change the landscape of this disease in future.

3. Febrile Seizures: Revisited In Today's Era

Dr. Anoop Verma

Chairperson AOPN 2007 - 13

MD, FIAP

- A febrile seizure occurs, usually from 6 months to 5 years, with fever but without CNS infection, an acute metabolic cause, or a previous unprovoked seizure. Context and recovery matter more than fever height.
- It is simple when generalized, under 15 minutes, single in 24 hours, and the child is neurologically well. Focality, longer duration, or recurrence within 24 hours makes it complex.
- First stabilise airway, breathing, and circulation; check glucose if the seizure continues or recovery is delayed. Ask caregivers to time the event and, when safe, capture a smartphone video—it may clarify focality and exclude a mimic.
- What is new—earlier action: Once convulsive activity reaches 5 minutes, treat with an age-appropriate benzodiazepine by the fastest feasible route; do not wait for the historical 15-minute boundary.
- A well child who returns to baseline after a simple febrile seizure needs no routine CBC, electrolytes, calcium, EEG, CT, or MRI. Investigations should answer a clinical question, not reassure the team.
- Lumbar puncture is not routine. Perform it when CNS infection is suspected; individualise the decision in incompletely immunised infants, children pretreated with antibiotics, or those with persistent altered sensorium.
- What is new—selective testing: Even the label 'complex' does not automatically mandate CT, EEG, lumbar puncture, or admission. Persistent drowsiness or focal deficit, meningeal signs, seizures without recovery, or an unwell appearance should drive escalation.
- Paracetamol or ibuprofen improves comfort but does not reliably prevent recurrence. Tepid sponging, aggressive cooling, and alternating antipyretics should not be presented as seizure prevention.
- Daily antiseizure medication is seldom justified: toxicity outweighs benefit. Intermittent clobazam or diazepam may be considered selectively for frequent or prolonged recurrences after shared decision-making.
- What is new—care beyond hospital: After a prolonged event, provide a written home plan and intranasal/buccal midazolam or rectal diazepam where appropriate. Demonstrate positioning, timing, repeat dosing if advised, and when to seek help.
- Recurrence affects roughly one-third. Younger age at onset, a first-degree family history, brief fever before the seizure, and a relatively modest temperature increase risk.
- Later epilepsy remains uncommon after a simple event, but risk rises with focal or prolonged seizures, multiple complex features, abnormal development or examination, and a family history of epilepsy. Neither dismiss nor catastrophise.
- What is new—phenotype-led genetics: Onset before 6 months or after 5 years, repeatedly prolonged or hemiclonic events, afebrile seizures, developmental slowing, or a strong family history should prompt early epilepsy/genetic review for Dravet syndrome and related fever-sensitive epilepsies.
- What is new—better vaccine counselling: Vaccination may provide the fever that triggers a seizure, but does not cause epilepsy or justify abandoning immunisation. Explain the small, time-limited risk and continue the schedule unless a specific contraindication exists.
- Clinical pearl: The thermometer explains the trigger, not the whole diagnosis—treat at 5 minutes, investigate the child rather than the convulsion, and discharge after counselling and rescue planning where indicated.

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4. Muscular Dystrophies in Children

Dr. Arijit Chattopadhyay
Senior Child Neurologist and Chairperson elect AOPN 2025-26
Dr. Jasodhara Chaudhuri
Senior Child Neurologist, Kolkata

A Practical Clinical View

- Muscular dystrophies are inherited disorders causing progressive muscle degeneration. Duchenne muscular dystrophy (DMD) is the most common severe childhood form; but congenital, limb-girdle, Emery-Dreifuss, facioscapulohumeral and other dystrophies must also be considered.
- We should suspect DMD in a boy with delayed walking, frequent falls, difficulty running or climbing stairs, calf enlargement, toe-walking or a positive Gowers' sign—often before weakness is formally recognised.
- The pattern of weakness guides diagnosis: proximal pelvic-girdle weakness suggests DMD or limb-girdle dystrophy; facial-scapular weakness suggests facioscapulohumeral dystrophy; early contractures with cardiac involvement suggest Emery-Dreifuss muscular dystrophy.
- Persistent elevation of AST and ALT may originate from muscle rather than liver. Serum creatine kinase should therefore be checked before undertaking extensive hepatic investigations in a child with unexplained transaminase elevation.
- Genetic confirmation is preferred because it establishes the diagnosis, clarifies inheritance, enables family screening and determines eligibility for targeted therapy. Muscle biopsy is now mainly reserved for unresolved cases. Female dystrophin-variant carriers may develop weakness or cardiomyopathy.
- Management should begin before major disability develops and requires coordinated neuromuscular, rehabilitation, cardiac, respiratory, orthopaedic, nutritional, endocrine, bone-health, psychological and educational care.
- Corticosteroids remain central to DMD management because they preserve motor function and delay complications; growth, weight, behaviour, blood pressure, bone health, cataract risk and adrenal suppression require monitoring.
- Cardiac surveillance with ECG and echocardiography or cardiac MRI should begin early, even in an asymptomatic child, because cardiomyopathy may precede cardiac symptoms.
- Respiratory function, sleep-related breathing, swallowing and cough effectiveness require periodic assessment. Breathlessness, recurrent chest infections, morning headache, weak cough, dysphagia or nocturnal hypoventilation warrant urgent evaluation. The anaesthesia team must know the diagnosis, and succinylcholine should be avoided in dystrophinopathies.
- Exon-skipping therapies, gene-transfer therapy, vamorolone and givinostat represent important advances in DMD. Eligibility, regulatory approval, availability and expected benefits differ, and these treatments complement—but do not replace—multidisciplinary care.
- Mutation-specific and gene-based treatments require precise genetic selection and expert monitoring. Recent serious safety concerns, including acute liver injury with some gene therapies, make careful benefit-risk counselling and post-treatment surveillance essential.
- Families should be protected from expensive or experimental 'cures.' Discuss what is proven, what remains uncertain, the treatment burden and whether a meaningful clinical benefit—not merely a biomarker change—has been demonstrated from that therapy.
- Urgent specialist referrals are required for rapid functional decline, respiratory symptoms, dysphagia, recurrent aspiration, palpitations, syncope, chest pain, severe contractures or an acute reduction in mobility.
- Published guidelines are available for Standards of Care for DMD which emphasize multidisciplinary management.
- **Clinical pearl:** In a child with motor delay, frequent falls or unexplained elevation of transaminases, look for Gowers' sign and check the serum CK.
- **Take-home message:** Early recognition and genetic confirmation open the door to timely multidisciplinary care, family counselling and emerging therapies—but coordinated lifelong care remains the foundation of every successful outcome.

5. Guidelines on Management of Status Epilepticus

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Status epilepticus (SE) is a potentially life-threatening neurological emergency. Prompt recognition, appropriate treatment and effective supportive care can prevent adverse outcomes. Although recognised since antiquity, varying definitions have complicated comparisons between studies. Traditionally, SE describes continuous seizure activity lasting more than 30 minutes, or recurrent seizures without complete recovery of consciousness. In practice, prolonged convulsive seizures require treatment much earlier.

Classification of Status Epilepticus:

SE may be generalised or focal, convulsive or non-convulsive. Generalised forms include tonic-clonic, tonic, myoclonic, typical or atypical absence, and atonic status. Focal forms include focal motor status, epilepsia partialis continua, and focal status with impaired awareness. Recognising non-convulsive forms is important when altered consciousness persists.

An underlying cause may not always be identified. Acute causes include febrile seizures, epilepsy, poor adherence to antiseizure medication, meningoencephalitis, intracranial space-occupying lesions, trauma, hypoglycaemia, hypocalcaemia, drugs or toxins, hypoxia, hyperpyrexia and dehydration. Chronic causes include congenital brain malformations, degenerative neurological disorders, endocrine disease and chronic encephalopathies.

Pathophysiology

Prolonged seizures produce venous congestion, cerebral oedema and petechial haemorrhages. Vulnerable regions include the hippocampus, amygdala, cerebellum, cerebral cortex and thalamus. Continuing seizure activity may cause lactic acidosis, raised intracranial pressure and hyperthermia, increasing the risk of neurological injury.

Principles of Management

Management proceeds simultaneously along three priorities: supporting vital functions, terminating seizures and treating the underlying cause. The likelihood of complications increases with seizure duration; assessment and treatment must therefore proceed without unnecessary delay.

Supportive Care

Position the head and neck to maintain a patent airway; protect the cervical spine when trauma is suspected. Clear secretions, provide oxygen and assist ventilation when required. Airway interventions should be undertaken appropriately, without forcibly opening the mouth. Intubation may be necessary for impaired ventilation or refractory seizures.

Establish vascular access, assess circulation and correct shock with isotonic fluids. Check blood glucose immediately and treat hypoglycaemia promptly. Correct electrolyte and other metabolic abnormalities. Consider gastric decompression when clinically indicated.

Monitor consciousness, oxygen saturation, respiration, pulse, blood pressure, temperature and cardiac rhythm. Respiratory compromise may result from seizures, antiseizure medication or both.

Seizure Control

Benzodiazepines are the initial treatment. Lorazepam has a longer anticonvulsant action than diazepam. Midazolam provides additional administration options, particularly when intravenous access is unavailable. Diazepam acts rapidly but its shorter anticonvulsant effect makes recurrence possible, requiring subsequent longer-acting treatment.

The article discusses phenytoin or fosphenytoin, phenobarbitone and valproate as further treatment options. Drug selection depends on seizure type, previous medication, underlying disease and available monitoring.

Intravenous phenytoin requires appropriate dilution and controlled administration, with cardiac and blood-pressure monitoring because arrhythmias and hypotension can occur. Fosphenytoin is converted to phenytoin and offers alternative administration options. Phenobarbitone is effective in seizure control but may cause sedation, respiratory depression and hypotension. Valproate provides another treatment option.

Anticonvulsant drugs can also be administered by the intraosseous route. Midazolam may be administered intramuscularly or intranasally, while suitable rectal preparations provide alternatives when intravenous access is unavailable.

Refractory Status Epilepticus

Persistent seizures despite appropriate treatment require intensive care, ventilatory support and EEG monitoring. Options discussed include midazolam infusion, thiopentone and propofol. Anaesthetic treatment requires specialist supervision, and prolonged propofol use in children warrants particular caution.

Once seizures stop, maintain appropriate antiseizure therapy, address the precipitating cause and arrange regular follow-up.

Evaluation

Obtain a detailed history and examination while stabilisation continues. Investigations may include blood counts, glucose, urea, electrolytes, calcium, magnesium, lactate, blood gases, antiseizure drug levels and metabolic screening. Cerebrospinal fluid examination and urine toxicology are undertaken when indicated.

Complications and Outcome

Complications include cardiac failure or arrest, hypotension, shock, arrhythmias, respiratory acidosis, aspiration pneumonia, acute renal failure, myoglobinuria, glucose disturbances, metabolic and lactic acidosis, hyperkalaemia and hyperthermia.

Modern paediatric intensive care, early seizure control, effective supportive therapy and treatment of the cause have improved neurological outcomes. Rapid, coordinated action remains the cornerstone of management.

Editorial note: Conflicting dosing details in the original have been omitted.
The treatment section requires clinical updating before publication.



6. Movement disorders in children: Clinical Overview

Dr. Gouri Rao Passi
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1. The movements we make are finely orchestrated and controlled by messaging within key brain areas such as the motor cortex, basal ganglia and cerebellum.
2. Ineffective messaging between these centres can result in abnormalities of speed, accuracy, grace and timing of movements. These sets of disorders are broadly called movement disorders.
3. They may be excessive movements (hyperkinetic), decreased movements (hypo kinetic).
4. The **hyperkinetic movements** are most common in pediatrics and include tics, chorea, athetosis, dystonic, stereotypies, tremors and myoclonus. **NEURO BULLETIN** include features like Parkinsonism including tremors and rigidity. Ataxia is characterised by uncoordinated and imprecise movements.
5. **Tics** are rapid, jerky movements or phonations and often have a premonitory urge, are worsened by stress or excitement and are partially suppressible. Vocal tics may be sudden involuntary vocalisations including sounds or may be more complex like words.
6. **Chorea** includes voluntary, irregular, arrhythmic movements which are non-suppressible and are more proximal compared to athetosis which is more distal and more writhing in character.
7. **Dystonia** consists of sustained twisting movements with variable tone often precipitated by action or touch or stress. Clinical findings include the stratal toe (extension of the great toe) and the splaying approach (fingers spread wide apart while approaching objects). They may be characterised based on age of onset, distribution and associated features as well as inheritance patterns and etiology.
8. When evaluating **etiology** first make sure that it is not part of normal developmental movement patterns which are sometimes seen in infancy and young childhood. Next confirm they are not functional neurological disorders which are characterised by entrainment, inducability and suppresibility. Then one must check for drugs adverse effects, which may be the underlying cause for movement disorders. The next step is to undertake neuroimaging to identify structural causes. Neuroimaging may sometimes give clues for metabolic disorders as well. A clinical history of consanguinity, episodic encephalopathy, vomits etc may steer us towards work up of inborn error sof metabolism. Finally genetic testing such as whole exome sequencing often uncovers the underlying genetic etiology.
9. **Treatment** for dystonia include levo dopa, trihexphenidyl, baclofen, gabapentin, clonazepam, tetrabenazine, botulinum toxin, and deep brain stimulation.
10. Drugs for tremors include propranolol, primidone and benzodiazepine.
11. Medications which are tried for chorea include risperidone, valproate, carbamazepine and tetrabenazine. Immune mediated chorea like sydenhams may respond to corticosteroids or IVIG.
12. Tics are initially managed with behaviour therapy interventions like habit reversal therapy and relaxation training and may need medications like risperidone, haloperidol, clonidine and topiramate.
13. Myoclonus often responds to clonazepam.

7. Infantile Spasms: Essential for Pediatricians

Prof. Jitendra Kumar Sahu
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PGIMER, Chandigarh
National Chairperson AOPN 2026-27

Infantile epileptic spasms syndrome (IESS) is the most common cause of difficult-to-treat epilepsy in infancy. It is characterised by onset in infancy, mostly 2-18 months of age and presence of epileptic spasms, mostly occur in clusters and in relation to sleep-wakefulness transition. It is associated with developmental arrest and EEG reveals hypsarrhythmia on electroencephalogram (EEG), which highlights underlying developmental and epileptic encephalopathy. The most common underlying etiologies are acquired structural pathologies: perinatal hypoxic ischemic brain injuries and neonatal hypoglycaemic brain injuries. Once paediatricians make the diagnosis of IESS, it signifies poor prognosis. However, in a small proportion, the outcome can be improved with two things: 1: accurate and prompt diagnosis and, 2) right therapeutic decisions.

1. Mitigating Treatment lag: Mitigating treatment lag is the most crucial. The median duration of treatment lag is approximate 3 months. It is inversely associated with adverse neurological outcome. The reasons are missed diagnosis by pediatricians, delayed reporting by parents and lack of prompt access to pediatric neurological services. These can be improved by increased awareness of parents of high-risk neonates, surveillance during routine immunization, empowerment and training of pediatricians, telehealth access, and availability of first-line medications (hormonal therapy and vigabatrin).
2. Choosing best therapy: The high-quality evidence for first-choice treatment is for IESS are: hormonal therapy and vigabatrin. It is important that valproate, phenobarbitone, levetiracetam etc should not be used as first-choice options.

Key points:

1. IESS is the most common cause of infantile-onset, difficult-to-treat epilepsy with poor prognosis.
2. Prompt recognition and treatment is essential to improve outcomes.
3. High-dose hormonal therapy is the preferred therapeutic choice.



8. HEADACHE

Prof. (Dr.) Kalpana Datta
Chairperson AOPN 2023 - 24
Prof. & Head, Pediatrics, Medical College Kolkata

"In pediatric medicine, the smallest patients often carry the biggest puzzles."

Headache is one of the common complains in childhood and adolescence. Prevalence ranges from 57%-82% in different studies. 70% of school children have headaches at least once a year & 20% of adults have the onset before age of 10 years.
Most headaches in children are benign. Incidence is more in male than female in school age; but after puberty female are having more than male.

Pain carrying structures in head are –
Extra cranial pain sensitive structures (like sinuses blood vessels, orbits, ears, nose, teeth & temporomandibular junction) and Intra cranial pain sensitive structures (arteries of circle of Willis and dural arteries, dural veins and venous sinuses and meninges)
According to the International classification of headache disorders (ICHD –3), it is divided into primary headache, secondary headache and painful cranial neuropathies.

Primary headache	Secondary headache	Painful cranial neuropathies and facial pain
☑ Migraine ☑ Tension type ☑ Trigeminal autonomic cephalalgias ☑ Other primary headache	☑ Attributed to trauma to head and neck ☑ Tumors ☑ Vascular ☑ Psychiatric ☑ Infections	☑ Painful cranial neuropathies and other facial pains. ☑ III, IV,V,VI, IX Cr. nerve involvement

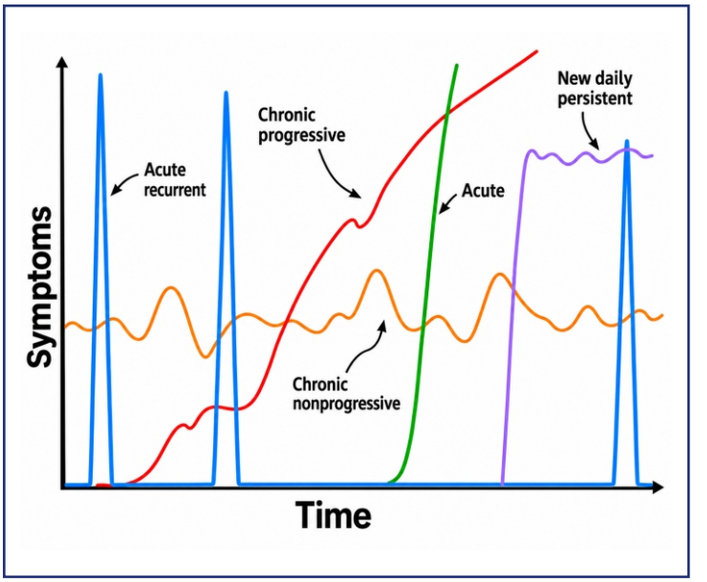
Classification according to temporal profile are

Acute:
URI, systemic infections, trauma, vascular

Acute recurrent:
Migraine, tension type headache, cluster headache

Chronic progressive:
Tumor, brain abscess, chronic SDH, IIHT, hydrocephalus

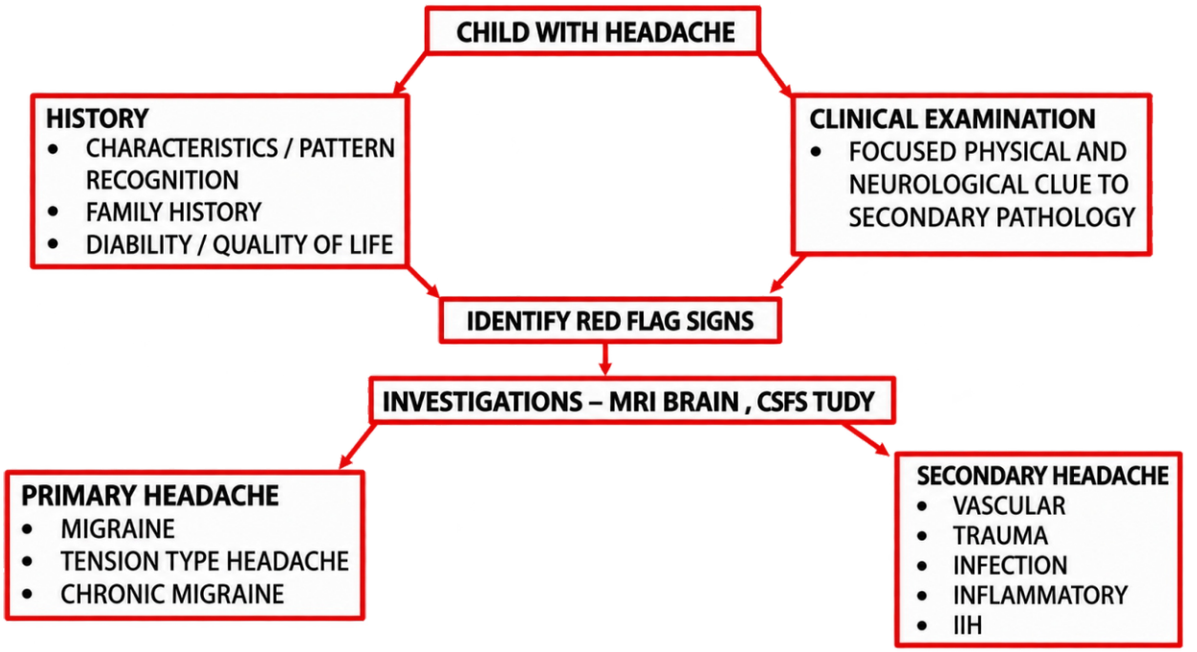
Chronic non progressive:
Chronic migraine, chronic tension type, medication overuse



It is always prudent to differentiate **Primary from secondary headache**

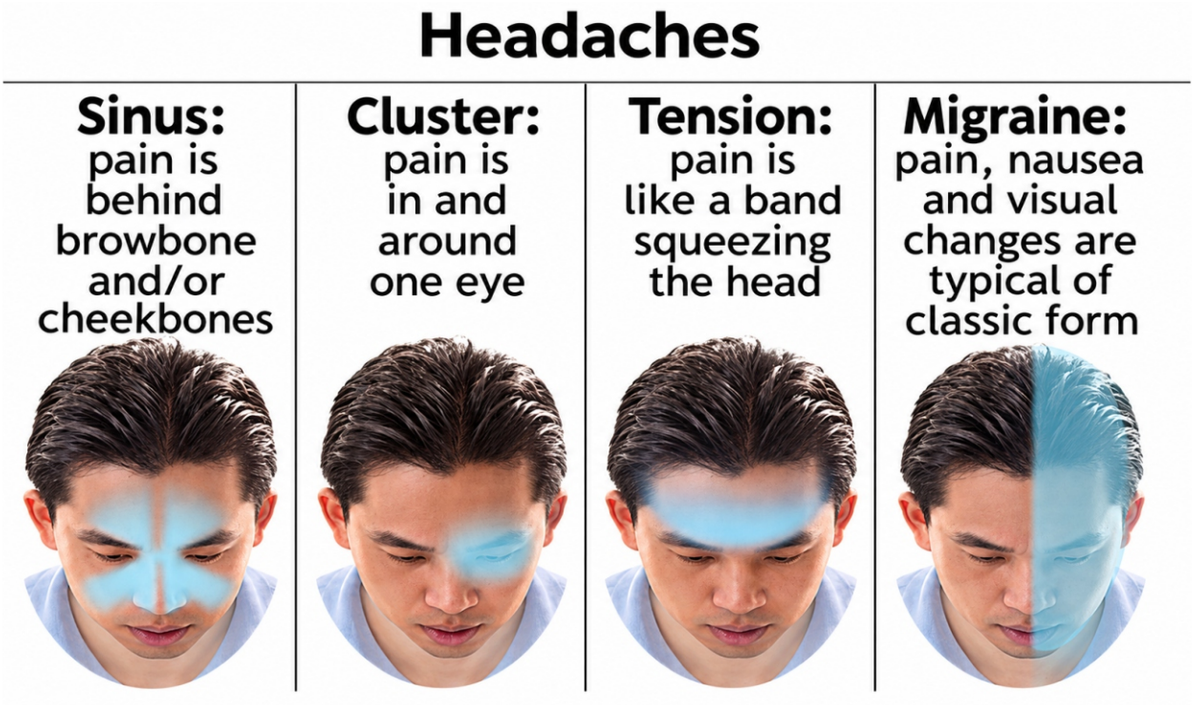
Features	Primary headache	Secondary headache
Length of stay	Chronic > 6 months	Acute / subacute
Temporal pattern	Recurrent or daily	Progressive
location	Frontal, temporal	Posterior
Quality	Throbbing, squeezing	Pressure
Time of the day	Any time	Earlymorning / awakening
Frequency / duration	Varied / hours to day	Constant
Nausea / vomiting	Nausea > vomiting	Vomiting >nausea
Visual aura / diplopia	Aura	Diplopia
Predectable triggers	++	No
Neurol exam	Normal	Usually abnormal

Approach of headache



IMPORTANT POINTS IN HISTORY

- Duration and frequency (daily, weekly, and monthly),
- Location (frontal, temporal, and occipital),
- Severity (mild, moderate, and severe),
- Types (one type or more than one type),
- Static or progressive based on the frequency/ severity
- Associated nausea and vomiting
- Photophobia and phonophobia
- Relation to specific circumstances, food, and medication
- Able to perform own activities?
- Past history of head trauma, viral infection, surgery
- History of drug intake (anticonvulsants, anticoagulants)
- History of school experiences, dietary habits, and family relationships
- History of dental or facial pain, nasal discharge seizure, altered sensorium, vertigo, gait abnormality, weakness, vision, and hearing



It is very important to identify the triggers

- Stress
- Weather change
- Fatigue
- Sleeplessness
- Hunger
- Menstruation
- Analgesics overuse
- Monosodium glutamate present in Chinese food
- Caffeine withdrawal

Look for comorbid conditions which may be associated with headache or may aggravate.

- Obesity, epilepsy, allergies, sinus disease, psychological and emotional disorder are to be treated along with headache treatment.

All cases of headache red flags are to be ruled out.

- Daily/near-daily vomiting esp. early morning
- Headache waking the child up
- A new/worse/abrupt onset headache
- Increasing severity and frequency
- Occipital Location
- Signs of raised ICT
- Symptoms/signs of systemic/CNS infections

Conclusion:

A complete history, detailed exanimation helps to distinguish between primary and secondary headaches.
One must always search for red flags - prompt neuro imaging to be done Life style changes, diary of headaches are useful in the management Preventive therapy - if frequent disabling headaches >3 episodes/month
New symptoms or persistent symptoms should prompt review

9. Sleep Disorders In Children

Dr. Kiran Makhija
Secretary AOPN 2023-2025

Sleep is fundamental to brain development and well-being. A disturbance in sleep can happen in growing and developing children which causes a parental concern and is significantly more often seen in cases of neurodevelopmental disorders, medical illnesses or psychiatric conditions. This may causes Emotional dysregulation, Behavioral problems and Cognitive difficulties in children.

Sleep is of 2 types – The Non-rapid eye-movement (NREM) sleep with stages 1, 2 & 3 (slow wave) and Rapid eye-movement (REM) sleep, is also called as dream sleep. The ratio of NREM:REM sleep time is approximately 50:50 at birth and 75:25 in adults

Sleep disorders are divided broadly into 3 categories (DSM-5).

A) Dyssomnia – When the quality and quantity is affected. Examples are insomnia, hypersomnia, breathing-related or circadian rhythm problem, narcolepsy.

B) Parasomnia – When there is abnormal events during sleep. Examples are sleep talking/sleepwalking, nightmares/night terrors, sleep bruxism, nocturnal enuresis, restless legs syndrome/sleep-related rhythmic movements.

C) Medical/psychiatric/ substance/ drug related sleep disorders

BEARS is pneumonic of a screening tool which gives the assessment of red flags of sleep problems like Bedtime problems – difficulty falling asleep, Excessive daytime sleepiness, unexplained tiredness, Awakenings (1 or more on most nights), Regularity and duration of sleep and Sleep disordered breathing (loud snoring, unusual position, restlessness, gasping, apnea).

Assessment is done by History and examination - sleep duration, sleep–wake schedule, sleep habits, nocturnal symptoms. Ask for videos from parents and sleep diary for a minimum of two weeks period. Assess daytime sleepiness at school and add any daytime naps. Physical examination is required to rule out treatable conditions.

Investigations are of 2 types. outpatient department-based and under supervision of specialist centers

Sleep disordered breathing monitored by oxygen saturation, HR and breathing. It is useful in identifying obstructive sleep apnea, hypoventilation and need for nocturnal oxygen.

This can be done by:

Actigraphy - It is cheaper, simpler , a wrist watch used in non-dominant hand & as an adjunct to other clinical information because it has got limited validation .

Polysomnography – It is a gold standard investigation but costlier & cumbersome. The electroencephalogram (EEG) (sleep stages/wakefulness), electromyogram (EMG) (muscle tone), electrooculogram (EOG) (eye movements), electrocardiogram (ECG), vital signs and video recording of respiratory efforts and body positioning are simultaneous measured.

Differential Diagnosis, Sleep is fundamental to brain development and well-being. A disturbance in sleep can happen in growing and developing children which causes a parental concern and is significantly more often seen in cases of neurodevelopmental disorders, medical illnesses or psychiatric conditions. This may causes Emotional dysregulation, Behavioral problems and Cognitive difficulties in children.

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Differential Diagnosis,

Bedtime struggles :

Often associated with behavioral problems like watching television

Restless leg syndrome : Moving legs when sleep because of uncomfortable sensations during rest or night time and relieved by movement. There is positive family history and also check for Fe deficiency

Sleep apnea : Periodic and sleep related airway obstruction seen in large adenoids, short neck or obesity and is suggested by pneumonic GASP (Gasps , Apnea , Snores or Perspire when asleep at night)

Narcolepsy :

Fall asleep anywhere and anytime.

Nocturnal seizures : Recurrent and similar seizures during sleep, common in initial phase of sleep or in early morning.

Neurodevelopmental disorders :

There is up to 80% of prevalence of sleep disorders in this group of children .Early identification is essential and therefore sleep problems should be screened during developmental and behavioral assessment. Timely behavioral and environmental sleep interventions can significantly improve the sleep in these children.

Management:

Non-Pharmacological Management - always be the 1st-line treatment

Most pediatric sleep problems are learnt behavior, and respond to consistent sleep

routines and sleep hygiene.

The behavioral management is with implementing sleep routine and hygiene by working on child's behavior and environment are essentials.

Have a set bedtime and wake-up time, regular daily exercise, daytime naps (only in very young children) should be short (not >1 hour). One-hour quiet time' before bed, avoid all high energy and screen activities. Keep TV out of child's bedroom.

Avoid caffeinated products & heavy meals, Relaxing (wash/change clothes), calming activities (bedtime story, soft music) before bed will help. The bed should be used for sleeping only and Keep bedroom quiet and dark.

Pharmacological Management - Commonly used medication in children is melatonin, helps with regulation of circadian rhythm of sleep. It is an endogenous hormone secreted by the hypothalamus functioning and begins at night with peaks around 3 a.m. The Secretion is suppressed by light by inhibiting hypothalamus. Antipsychotics or antidepressants, should be used only by specialist .

Refer when suspected obstructive sleep apnea or seizures or a child not responding to 1st- or 2nd-line management.

Early identification and timely intervention improve outcomes, reducing long-term behavioural and psychosocial consequences.

SUGGESTED READING

The 360° Pediatrics- Topic: Sleep Disorders in Children

Link https://iapindia.org/pdf/action-plan-2026/Growth-Development-and-Behaviour/012-Sleep-Disorders-in-Children.pdf

Choudhury J. Sleep problem. In:

Choudhury J, ed. Behavioral Problems in Children and Adolescents.

2nd ed. New Delhi, India: Jaypee Brothers Medical Publishers; 2023:160-165.

Owens JA, Dalzell V. Use of the 'BEARS' sleep screening tool in a pediatric residents' continuity clinic: a pilot study. Sleep Med. 2005;6(1):63-69. doi:10.1016/j.sleep.2004.07.015yyAND BEHAVIOUR.

10. Developmental Delay

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A developmental delay happens when a child takes longer than expected to reach key growth milestones compared to other children of the same age.

Main Types of Delays

- **Motor skills:** Trouble with large body movements (gross motor) like crawling or walking, or small movements (fine motor) like holding a small toy. Delays in motor skills interfere with a child's ability to coordinate large muscle groups, such as those in the arms and legs, and smaller muscles, such as those in the hands. Infants with gross motor delays may have difficulty rolling over or crawling; older children with this type of delay may seem clumsy or have trouble walking up and down stairs. Those with fine motor delays may have difficulty holding onto small objects, such as toys, or doing tasks such as tying shoes or brushing teeth. Some motor delays result from genetic conditions, such as achondroplasia, which causes shortening of the limbs, and conditions that affect the muscles, such as cerebral palsy or muscular dystrophy. They may also be caused by structural problems, such as a discrepancy in limb length.
- **Speech and language:** Trouble making sounds, talking, or understanding what others say. Some speech delays are receptive language disorders, in which a child has difficulty understanding words or concepts. Children with this type of speech delay may have trouble identifying colors, body parts, or shapes. Others are expressive language disorders, in which a child has a reduced vocabulary of words and complex sentences for his or her age. A child with this type of speech delay may be slow to babble, talk, and create sentences. Often, a child with a speech delay has a combination of receptive and expressive delays. Children with an oral motor problem - such as weakness in the muscles of the mouth or difficulty moving the tongue or jaw - that interferes with speech production have what is known as a speech production disorder. Children may have speech delays due to physiological causes, such as brain damage, genetic syndromes, or hearing loss. Other speech delays are caused by environmental factors, such as a lack of stimulation. In many instances, however, the cause of a child's speech delay is unknown.
- **Cognitive delays :** Trouble thinking, learning, remembering, and solving simple problems. Cognitive delays affect your child's ability to think and process information, including problem-solving, reasoning, remembering, understanding, and learning. In some cases, a cognitive delay may become more apparent when children enter school. Cognitive delays may affect a child's intellectual functioning, interfering with awareness and causing learning difficulties that often become apparent after a child begins school. Children with cognitive delays may also have difficulty communicating and playing with others. This type of delay may occur in children who have experienced a brain injury due to an infection, such as meningitis, which can cause swelling in the brain known as encephalitis. Shaken baby syndrome, seizure disorders, and chromosomal disorders that affect intellectual development, such as Down syndrome, may also increase the risk of a cognitive delay. In most cases, however, it is not possible to identify a clear reason for this type of delay.
- **Social, Emotional, and Behavioral Delays :** Trouble playing with others, expressing feelings, or understanding social cues. Children with developmental delays, including those with related neurobehavioral disorders such as autism spectrum disorder and attention deficit hyperactivity disorder, often also have social, emotional, or behavioral delays. Due to differences in brain development, they may process information or react to their environment differently than children of the same age. These delays can have an impact on a child's ability to learn, communicate, and interact with others. It is common for children with developmental delays to have difficulty with social and emotional skills. For example, they may have trouble understanding social cues, initiating communication with others, or carrying on two-way conversations. They may also have difficulty dealing with frustration or coping with change. When the environment becomes too socially or emotionally demanding, children with developmental delays may have prolonged tantrums and take longer than other children to calm down. This behavior can be a signal that the child needs more support by modifying his or her environment or learning skills to cope with social and

emotional challenges.

- **COMMON CAUSES**

a. Genetic factors: Chromosomal conditions like Down syndrome or fragile X syndrome.

b. Pregnancy or birth issues: Premature birth, low oxygen at birth, or maternal infections.

c. Environmental factors: Exposure to toxins like lead, or severe lack of proper care and nutrition.

d. Unknown causes: Sometimes, doctors cannot find a specific reason.

WHEN TO CONSULT A DOCTOR :

If you notice your child consistently falls behind on multiple milestones or loses skills they already had, speak to a pediatrician right away for an evaluation and early support.

Developmental Delay in Children : Symptoms, Causes & Outlook

A developmental delay occurs when a child is behind in developing skills that experts expect for their age. Developmental delays.

Types of Developmental Delays in Children

- Developmental Delays in Children
- Find a Doctor & Schedule
- Types of Developmental Delays
- Screening
- Diagnosis
- Therapy for Infants & Toddlers
- Therapy for Older Children
- Support

Often, developmental delays affect more than one area of a child's development. When a child has delays in many or all of these areas, it is called global developmental delay.

Some developmental delays have an identifiable cause. However, for many children, the cause of the delay, or multiple delays, is not clear.

DEVELOPMENTAL DELAYS DEFINED :

As children get older, they develop language, speech, thinking, emotional, and social skills. The progressive steps they take to develop a multitude of skills go in a specific order over a period. For example, babies begin with coos, then speak their first words and add to their vocabulary, before finally speaking in 3-4 word sentences, a skill they don't reach until around two years.

Each step in the process is called a developmental milestone. These milestones occur in the same order but not at the same time for every child.

That's why each milestone has an age range in which it typically occurs. For example, babies typically start walking around 12 months, but it's normal for them to take their first steps anytime between 9 to 18 months.

Knowing the progressive steps toward skill-building and the typical age range allows us to evaluate children and determine if they reach all their milestones on time.

When children don't achieve a milestone, they have a developmental delay. The types of developmental delays include:

DEVELOPMENTAL DELAYS CAUSES

We can't always identify a specific cause for a developmental delay. However, there's a very long list of possible causes, so we carefully evaluate each child to discover if they have a cause that we can target with treatment.

These are only a few of the many possible causes of developmental delays:

Developmental disabilities

In some cases, developmental delays are symptoms of a developmental disability, which means the disability causes the delay. Developmental disabilities include ADHD, autism spectrum disorders, Down syndrome, and cerebral palsy.

Developmental disabilities are life-long conditions that require ongoing intervention. By comparison, most children overcome developmental delays with therapy that teaches them the skills they need to reach their milestones.

Pregnancy And Birth Complications

Children can develop developmental delays if their mother had an infection or drank, smoked, or used drugs while pregnant. Babies who are born prematurely or underweight or who don't get enough oxygen during birth may also have developmental delays.

Environmental Challenges

Environmental challenges that may cause developmental delays include lead poisoning, nutritional deficiencies, trauma, and neglect, to name a few. Trauma and neglect cover many different challenges, from sexual, physical, and emotional abuse to the loss of parents and community violence.

Medical Conditions

Any medical condition limiting a child's daily activities hampers their ability to progress and learn their developmental skills. Health problems affecting development range from serious conditions such as a traumatic brain injury to a wide range of common diseases.

Chronic ear infections, hearing loss, and problems controlling certain muscles can cause a speech delay. Any vision problem interferes with a child's ability to meet developmental milestones. Muscle diseases, whether inherited or acquired, hinder motor skills.

What is Global Developmental Delay (GDD) ?

Developmental delay can be isolated, meaning a delay affects only one area of development. If a significant delay occurs in two or more of these developmental areas, your child may have what providers refer to as global developmental delay.

Autism

Autism is a difference in how your child's brain works that causes them to socialize and behave in unique ways. Early signs of

autism include limited eye contact and body language and repetitive motions or speech. Behavioral therapies and other support can help autistic kids (and adults) make the most of their strengths and manage any challenges.

In the past, healthcare providers used several different names to describe neurodevelopmental disorders with features of autism

- Autistic disorder
- Asperger's disorder
- Pervasive developmental disorder not otherwise specified (PDD-NOS)
- Childhood disintegrative disorder

Providers don't use these names anymore. Instead, they recognize that autism is a spectrum, and that autistic people are neurodivergent. This means they have a wide range of personality traits, strengths and challenges, and they need varying levels of support. So, providers use ASD as the official diagnosis and describe features and needs unique to each person.

Words aren't perfect. And sometimes, "medspeak" that healthcare providers use - like "disorder," "symptoms" or "diagnosis" - doesn't match the lived experience of autistic people or their families. It's important to know that autism isn't a disease that can be "cured," and it's not a mental health condition. Throughout this article, we'll use medical terms as needed to describe available support for your family. But we recognize that autism is an identity, not just a diagnosis. We use the term "autistic" to reflect the identity-first language preferences of the autistic community.

TREATMENT OPTIONS :

Treatment for developmental delays relies on specialized, multi-disciplinary therapies customized to target the child's specific areas of lag.

Core Therapy Options Physical

1. Therapy (PT) : Focuses on gross motor skills like sitting, crawling, walking, balance, and strength.

2 Occupational Therapy (OT) : Addresses fine motor skills (like grasping or writing), sensory processing, and daily self-help tasks (dressing, feeding).

3. Speech and Language Therapy : Improves communication, language comprehension, speech clarity, and oral-motor/feeding challenges.

4. Behavioral and Psychological Support : Helps with emotional regulation, attention, social skills training, and positive reinforcement routines.

5. Special Education : Provides early childhood educational stimulation and structured learning plans tailored to the child's school needs.

Strategies for Home Support

- Incorporate practice into daily routines : Use short, frequent, and playful repetitions of target skills during meals, baths, or play.
- Limit screen time : Keep daily media and device use under one hour for young children to prioritize face-to-face social interaction.
- Maintain consistent schedules : Use predictable routines or visual picture schedules to lower anxiety and build independence.
- **Treatment Challenges**
- Delayed Diagnosis: Many children are identified late because routine developmental screening is missed during early pediatric visits.
- Resource Shortages: Rural and underserved regions lack trained specialists like speech therapists, occupational therapists, and developmental pediatricians.
- High Financial Costs: Long-term specialized therapies and special education place a heavy economic burden on families.
- Social Stigma: Cultural misconceptions or shame often prevent parents from seeking timely behavioral or psychological support.
- System Fragmentation: Poor coordination between primary care doctors, schools, and therapy centers creates gaps in continuous care plans.
- **Treatment Opportunities**
- Early Intervention Impact: Brain plasticity is highest in the first few years of life, making early therapy vastly more effective.
- Multi-Disciplinary Models: Combining speech, occupational, physical, and behavioral therapies addresses every affected developmental domain.
- Government Schemes: Initiatives like India's Rashtriya Bal Swasthya Karyakram (RBSK) provide structured community screening and early management for children.
- Digital Health & Teletherapy: Remote consultations and digital tools help bridge geographical gaps to deliver guidance directly to parents in remote areas.
- Family-Centered Plans: Individualized Family Service Plans (IFSPs) empower caregivers with training and counseling to support daily skill building at home.
- Early childhood development includes acquiring fine and gross motor skills. While both these skill s involve movement, they do have differences :
- Fine motor skills involve movement of the smaller muscle groups in your child's hands, fingers, and wrists.
- Gross motor skills involve movement of the larger muscle groups, like the arms and legs. It's these larger muscle groups that allow babies to sit up, turn over, crawl, and walk.

11. Cerebral Palsy

Dr. KP Sarabhai
Chairperson AOPN 2019 - 20

Cerebral palsy is a permanent disorder of movement and posture due to a non-progressive disturbance in the developing brain.

Red flags :

Persistent head lag beyond the expected age.
Abnormal muscle tone—marked hypotonia or evolving spasticity,
-Persistent fisting ,
-Early hand preference,
-Scissoring of legs
-Delayed rolling, sitting or crawling
-Asymmetry of movements

-Persistent primitive reflexes
-Abnormal movements in early infancy
Pearl:-Early hand preference be considered as hemiplegia.

Absent/abnormal movements at 3–5 months is an important predictor of CP.
General Movement Assessment combined with neurological examination and MRI makes early diagnosis.

If there are clear neurological abnormalities, early referral to a neurorehabilitation team should be done
Early intervention:
Physiotherapy
Occupational therapy
Speech and feeding therapy, Therapeutic
Stimulation of Vision and hearing

Clinical Features:

- Dyskinetic
- Dystonia/choreoathetoid
- Ataxic
- Incoordination, tremor, balance problems
- Mixed

Features of more than one type

- Hemiplegia
- Diplegia
- Quadriplegia/bilateral involvement

Screen For:

- Epilepsy
- Intellectual developmental impairment of
- Speech and communication problems
- Dysphagia and aspiration
- Malnutrition
- Visual impairment
- Hearing impairment
- Sleep problems
- Pain
- Behavioural and emotional difficulties
- Feeding problems are common and
- Choking/coughing during feeds
- Prolonged feeding time
- Drooling
- Recurrent chest infections
- Poor weight gain
- silent aspiration can occur in any child.

Don't treat spasticity because the tone is abnormal.

Possible Interventions Include:

- Stretching/positioning
- Orthoses
- Botulinum toxin for selected focal spasticity
- Oral antispastic medications in selected children
- Intrathecal baclofen in severe generalized spasticity
- Orthopaedic surgery.,may be required.
- Practical pearl: Don't wait for pain or obvious deformity. Hip surveillance with clinical examination and appropriate radiographs should be incorporated into long-term care.
- Sometimes Contractures are preventable—
- Regular assessment of:
- Hip abduction
- Hamstrings
- Gastrocnemius/ankle
- Knee extension
- Upper-limb range of motion
- is important.
- MRI: useful, but interpret it correctly
- MRI can:
- Support the diagnosis
- Help identify the timing/type of brain injury
- Provide prognostic information in some children
- But:
- Abnormal MRI ≠ CP automatically

- Normal MRI ≠ CP excluded
- A progressive neurological disorder should prompt reconsideration of the diagnosis.

Red flags :

Loss of previously acquired skills
Progressive weakness
Progressive worsening beyond expected CP evolution
New movement disorder
Family history of neurological disease
Episodic deterioration
Peripheral signs
Normal early development followed by unexplained regression
CP does not cause true developmental regression.
Prognosis: don't predict only from diagnosis
Functional prognosis is better estimated using:
Age at diagnosis
Early motor trajectory
MRI findings
Cognitive function
Communication
Vision/hearing
Presence of epilepsy
GMFCS level
The GMFCS is particularly useful for communicating expected gross-motor function and planning rehabilitation.
The goal is participation, not "normal tone"
Modern CP management focuses on:
Function → independence → participation → quality of life
rather than trying to make every neurological examination normal.
>⁹ One practical rule for the pediatrician
"See the child, not just the tone."

At every follow-up, ask:

Can the child…
Sit?
Stand?
Walk or move independently?
Use both hands?
Communicate?
Eat safely?
Sleep comfortably?
Attend school?
Play with other children?
Participate in family/community activities?
That approach turns CP care from "treating spasticity" into comprehensive lifelong care.



12. EEG for the Pediatrician: Indications, Pearls and Pitfalls

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Electroencephalography (EEG) is an invaluable investigation in pediatric neurological practice, but its usefulness depends on asking the right question and interpreting the findings in the appropriate clinical context.

Indications

When should a pediatrician ask for an EEG?.

In a child presenting with an unprovoked event suspicious for a seizure, an EEG is obtained primarily to look for interictal epileptiform discharges (IEDs). Their presence provides supportive evidence for an underlying predisposition to seizures and may aid epilepsy classification.

A second important indication arises in the pediatric intensive care unit. Following convulsive status epilepticus, clinical seizures may cease after treatment, while the child fails to regain the expected level of consciousness. EEG is crucial in this setting to detect ongoing electrographic seizures or non-convulsive status epilepticus.

EEG is also useful in encephalopathy. Generalized background slowing in an awake child provides objective evidence of diffuse cerebral dysfunction, while focal slowing may provide a clue to localized cerebral dysfunction or may be seen transiently following a focal seizure.

Finally, video-EEG is particularly valuable when recurrent events of uncertain nature can be captured. Demonstrating an EEG correlate accompanying the habitual clinical event helps establish its epileptic nature and differentiate seizures from movement disorders, functional events and other seizure mimics.

Finally, video-EEG is particularly valuable when recurrent events of uncertain nature can be captured. Demonstrating an EEG correlate accompanying the habitual clinical event helps establish its epileptic nature and differentiate seizures from movement disorders, functional events and other seizure mimics.

Pearls

Timing matters—but there is no single ideal time for an EEG. An EEG obtained soon after a seizure can be informative: focal postictal slowing may provide a localization clue, while ongoing electrographic seizures can be identified despite cessation of clinical convulsions. However, benzodiazepines and other GABAergic medications may suppress interictal epileptiform activity. When the clinical objective is specifically to identify IEDs and the situation permits, allowing an interval after benzodiazepine administration—often 48–72 hours in practice—may improve their detection.

Sleep EEG is important. Sleep reduces movement and muscle artifact, and epileptiform discharges may be more apparent during sleep. In certain childhood epilepsies, abnormalities may be predominantly or even exclusively evident during sleep. Examples include epilepsy with centrotemporal spikes and infantile epileptic spasms syndrome (IESS). Hence, whenever feasible, an EEG recording that includes both wakefulness and sleep improves the diagnostic yield.

A child's EEG is not a small adult EEG. Normal background rhythms and sleep architecture evolve substantially with age. Pediatric EEG interpretation requires familiarity with these developmental changes and is best undertaken by clinicians with appropriate expertise in pediatric EEG.

Pitfalls

A normal EEG does not exclude epilepsy. Epileptiform discharges may be infrequent, arise from deeper regions, involve a small cortical area, or have an electrical orientation that makes them poorly visible on scalp EEG. Even a clinical seizure may occasionally lack an obvious scalp EEG correlate. Epilepsy remains a clinical diagnosis; EEG supports the diagnosis but does not make or exclude it in isolation.

Conversely, an abnormal EEG does not necessarily mean epilepsy. Genuine epileptiform discharges may occasionally occur without clinical epilepsy. Normal variants, developmental patterns and artifacts may be mistaken for epileptiform abnormalities.

Finally, do not order an EEG without a clinical question. Syncope, breath-holding spells, stereotypies, tics, sleep phenomena and behavioral events may mimic seizures. Like any other investigation, EEG is useful only if used in the appropriate clinical context and it should never be used to "rule out epilepsy."

13. Neurometabolic Disorders: A Panoramic View

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Dr Mohammad Anas
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Although individual neurometabolic disorders are rare, as a group they are not uncommon and may be masquerading as usual neurological conditions seen in pediatric practice. Their clinical spectrum is remarkably broad, with considerable variation in age at presentation and phenotype.

Behaviour and Developmental Conditions

- Autism when associated seizures or dystonia consider cerebral creatine deficiency. Plasma guanidinoacetate (GAA) and urine creatine/creatinine provide useful screening clues. MRI with MRS shows a low creatine peak. Diet and supplements are available including creatine supplementation.
- Autism with non-familial hypopigmented hair and microcephaly to think PKU. Plasma phenylalanine confirms the diagnosis. Treatment includes phenylalanine-restricted diet and sapropterin (Kuvan) targeting phenylalanine levels between 120–360 µmol/L
- Developmental delay with failure to thrive may appear nutritional, when associated with elevated homocysteine. But always consider intracellular cobalamin disorders if B12 normal despite elevated homocysteine. Treatment with hydroxocobalamin and betaine can improve brain atrophy.
- Autism when associated seizures or dystonia consider cerebral creatine deficiency. Plasma guanidinoacetate (GAA) and urine creatine/creatinine provide useful screening clues. MRI with MRS shows a low creatine peak. Diet and supplements are available including creatine supplementation.
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- Developmental delay with failure to thrive may appear nutritional, when associated with elevated homocysteine. But always consider intracellular cobalamin disorders if B12 normal despite elevated homocysteine. Treatment with hydroxocobalamin and betaine can improve brain atrophy.
- Intellectual disability can have various causes and one amongst them is classical homocystinuria. Eye exam is paramount in all such children for iridonesis, ectopic lens and progressive myopia. High total homocysteine and methionine are features. Treatment is pyridoxine and some require methionine restriction and betaine. Target plasma total homocysteine is <50 µmol/L in pyridoxine-responsive patients
- Cerebral palsy without a obvious sentinel event should be reviewed for metabolic mimics, including arginase deficiency and biotinidase deficiency. Especially late-onset biotinidase deficiency may lack the classic alopecia and skin manifestations. Biotinidase activity is readily testable and neurological symptoms improve with early treatment with biotin 5-20mg/day.

Neonatal conditions

- Refractory neonatal seizures are not always HIE or encephalitis. Vitamin responsive conditions like pyridoxine-dependent epilepsy, PNPO deficiency and folinic-acid-responsive seizures must be kept in differentials. A therapeutic trial of pyridoxine (30mg/kg/day), PLP or folinic acid (3–5 mg/kg/day) is required for refractory seizures.
- A severe HIE-like presentation with MRI showing cystic changes need to reviewed for Sulfite oxidase deficiency and molybdenum cofactor deficiency (MoCD). Low uric acid is an important clue to MoCD. Importantly, MoCD type A now has disease-modifying treatment with cPMP (fosdenopterin)

Neuromuscular

- Not all floppy infants are SMA.
- Pompe disease has to considered and ECG pattern (high voltage QRS complexes) can be a quick screening tool for cardiomyopathy. Alpha-glucosidase activity provides rapid diagnostic direction, and early enzyme replacement therapy is disease-modifying.
- Tk2 deficiency, a mitochondrial myopathy also has treatment options including nucleoside supplementation and Kygevvi.

Pediatric ER

- Recurrent vomiting followed by encephalopathy is not always cyclical vomiting. Check ammonia for Urea cycle disorders during the episode. Children with partial enzyme deficiencies remain completely well between episodes and present during fasting, infection or increased protein intake.

Clinical Pearls

- A normal newborn screen does not close the metabolic chapter. In an unexplained neurological phenotype, targeted add-ons such as ammonia, lactate, ketones, uric acid, total homocysteine, methylmalonic acid and creatine/GAA, guided by the phenotype, can uncover diagnoses missed by routine screening.
- Avoid broad diagnostic label and early closure without thorough historical review, as many children may remain undiagnosed and undertreated.

14. TIC Disorders: Practical Clinical Pearls

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(As Tribute to Late Dr. PAM Kunju Sir - immediate past Chairperson AOPN)

Tic disorders are common neurodevelopmental conditions of childhood, ranging from mild, self-limiting tics to Tourette syndrome with significant functional consequences. Although tics are often the most visible feature, ADHD, OCD, anxiety and social difficulties may contribute more to disability. Management therefore requires recognition of the phenomenology, appropriate assessment of impact and comorbidities, and treatment tailored to the individual child.[1–4]

Recognise the clinical features

Tics are brief, repetitive motor movements or vocalisations, usually beginning in childhood and characteristically waxing and waning.[1]

Ask about the premonitory urge

- Older children may describe an uncomfortable sensation or urge before the tic, followed by relief. Younger children may not recognise or report this experience.[1]

Tourette syndrome

- requires both motor and vocal tics for more than one year. They need not occur simultaneously. Coprolalia is uncommon and is not required for diagnosis.[1]

Phenomenology is central to diagnosis

Functional tic-like behaviours should be considered when onset, complexity, variability or other features are atypical for a developmental tic disorder. Careful observation is often more useful than extensive testing.[1]

Avoid unnecessary investigations

A child with typical tics and a normal neurological examination generally does not require routine EEG, MRI or laboratory testing. Investigate when movements are atypical, progressive, painful or associated with altered awareness, focal neurological signs or diagnostic uncertainty.[1]

Assess impact rather than tic frequency alone

Pain, injury, sleep disturbance, school difficulties, social embarrassment and family distress may be more important than the number of tics.[1,2]

Screen for comorbidities

ADHD, OCD, anxiety, mood and behavioural problems, learning difficulties and sleep disorders should be actively sought, as they may cause greater disability than the tics themselves.[1,2]

Psychoeducation is often the first intervention

Explain the fluctuating nature of tics and discourage repeated requests to suppress them. Mild tics without functional impairment may require only reassurance and follow-up.[2,4]

Use behavioural therapy when tics are . troublesome

BIT, incorporating habit-reversal strategies, is an established first-line treatment. ERP is another evidence-based option. Remotely delivered behavioural therapy can improve access where specialist services are limited.[2,4]

Reserve medication for significant impairment

Clonidine or guanfacine may be particularly useful when ADHD coexists. For more disabling tics, aripiprazole and other dopamine-modulating agents are established options; treatment should begin with low doses and be titrated according to response and adverse effects.[3]

Treat ADHD when it is impairing

The presence of tics alone should not lead to withholding ADHD medication. Methylphenidate, atomoxetine or an α2-adrenergic agonist may be considered according to the clinical situation.[2,3]

Treat OCD independently

CBT with Exposure and Response Prevention is an important first-line treatment; SSRIs are appropriate when pharmacotherapy is required.[5]

Reassess periodically

Tic severity and treatment needs change during childhood and adolescence. Medication should therefore be reviewed regularly, with reduction or withdrawal considered when sustained improvement allows.

Clinical Pearl

The most visible symptom is not always the most disabling one. In a child with tics, ask, “What is interfering most with this child's life?”and treat that first.

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15. Autoimmune Encephalitis (AE)

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Autoimmune Encephalitis (AE) in children is an increasingly recognized neuroinflammatory condition where the immune system mistakenly targets healthy brain cells, altering synaptic function and neural excitability. Presentations in children are often different from adults, hence pediatricians must maintain a high index of suspicion to ensure rapid diagnosis and treatment, which is essential for maximizing long-term neurocognitive recovery.

Key Clinical Messages

- Predominance of Neurological Signs: Children are much more likely than adults to initially present with distinct neurological symptoms.
- Symptoms and Signs: The typical clinical picture involves a rapid, subacute onset (usually developing within weeks) of multi-system central nervous system dysfunction in a previously healthy child. Symptoms frequently cross multiple domains, including:
- Seizures: Often new-onset, multifocal, or generalized, and highly refractory to standard anti-seizure medications.
- Movement Disorders: prominent orofacial dyskinesias, chorea, dystonia, or tremors.
- Behavioral and Cognitive Regression: acute developmental loss, speech impairments or mutism, severe irritability, and sleep alterations.
- Psychiatric Disturbances: Including intense aggression, uncharacteristic temper tantrums, or fulminant psychosis.
- Pathogen Triggers: AE can be secondary to an infection. Up to 27% of Herpes Simplex Virus (HSV) encephalitis patients can develop post-HSV anti-NMDAR encephalitis; within three months of the initial viral infection.

Diagnosis

- Antibody testing in serum and /or CSF is needed. Anti-NMDA receptor (anti-NMDAR) encephalitis is the most common form in children. Myelin oligodendrocyte glycoprotein antibody-associated disease (MOG-AD) is also a frequent pediatric neuroinflammatory condition, expanding beyond classic ADEM to present as encephalitis. Overlapping Encephalitis is known where two or more types of encephalitis may occur concurrently .
- Criteria for the diagnosis of anti-NMDAR encephalitis have been proposed by Graus, et al. A probable anti-NMDAR encephalitis can be made if all three of the following criteria have been met: (i) Rapid onset (less than three months) of at least four symptoms among psychiatric/behavioral dysfunction, speech abnormalities, seizures, movement disorders, decreased consciousness or autonomic dysfunction; (ii) Abnormal EEG/CSF; and (iii) Exclusion of other causes
- MRI may show unilateral or bilateral T2/ FLAIR signal hyperintensities involving the mesial temporal lobe. However it is usually normal and helps in exclusion of other conditions.
- EEG -a typical pattern called ‘extreme delta brush’ may be seen in a third of patients. Others may show diffuse slowing or epileptiform activity.
- • Differential Diagnosis
- • Acute Infectious Encephalitis- often presents acutely with fever, seizures and altered consciousness. Systemic symptoms and rashes may be seen in some cases. Depending on the pathogen, certain clinical and MRI features may provide clues to etiology.

- PANDAS/PANS: AE is characterized by a consistently progressive decline in cognitive and neurological functioning, differentiating it from the distinct episodic, relapsing-remitting course seen in PANDAS/PANS.

Clinical Pearls for Pediatricians

- Normal Imaging Does Not Rule Out AE: A normal brain MRI is common at presentation, especially in anti-NMDAR encephalitis, and does not exclude the diagnosis.
- Antibody-Negative Cases Occur: Only up to 44% of patients with AIE have an antibody-positive status. Do not withhold treatment while waiting for antibody panels, or if the panels return negative. Validated consensus criteria allow for a "probable" diagnosis based entirely on clinical features and proof of neuroinflammation if other causes are excluded.
- Look for Red Flags: A history of pre-existing developmental delay, an insidiously chronic presentation (>3 months), a lack of multi-system involvement, or a completely normal EEG should strongly point you toward a metabolic, genetic, or primary psychiatric condition rather than AE.
- Initiate Treatment Empirically: If suspicion is high and alternative causes (such as active viral or bacterial infections) have been reasonably excluded, initiate empirical first-line immunotherapy immediately. First-line agents include high-dose intravenous corticosteroids, intravenous immunoglobulin (IVIG), or plasmapheresis.
- Escalate Quickly: If there is a poor clinical response within 1 to 2 weeks, escalate to second-line therapies. International consensus recommendations for pediatric NMDARE prefer rituximab over cyclophosphamide as a second-line agent.
- Since anti-NMDAR encephalitis may be associated with ovarian teratomas in older children and with neuroblastoma or ganglioneuroma is younger children screening for these is recommended.

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16. MRI PEARLS in Pediatric Neurological disorders

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MRI of the brain is an essential part of the neurological evaluation in children, and characteristic imaging patterns can provide crucial diagnostic clues.

1. Interpretation requires
a. Knowledge of brain anatomy
b. MRI acquisition slice orientation and direction principles
(The AC–PC line is the imaginary line connecting the anterior commissure (AC) to the posterior commissure (PC) on a mid-sagittal brain MRI. It is used to orient the images in different cuts)
• The right side of the image is the left side of the brain, and vice versa
• Axial Images: taken from inferior to superior (slices aligned parallel to the AC–PC line)
• Coronal Images: Anterior to posterior (Slices aligned perpendicular to the AC – PC line)
• Sagittal Images: oriented in the midline, with the AC–PC line lying within the mid sagittal plane. The slices move from right to left (Siemens and GE scanners) and left to right (Philips scanners)

Correlation of different structures in different planes on the MRI is not possible to put in detail here , but can be seen on :
https://jhpmribrainatlas.rcc.uchicago.edu/sites/default/files/midata/swf/allAtlas.html

c. Knowledge of MRI sequences, which are a particular way of acquiring MRI images that shows different tissues in different shades from black to white, in different sequences. Eg Water, fat, blood products, cellularity or myelin, may look different in different sequences eg CSF looks bright on T2 and dark in T1

2. MRI sequence protocol is chosen depending on the clinical scenario. A routine brain MRI should include at least T1, T2, FLAIR, DWI/ADC and susceptibility-sensitive imaging (SWI/GRE).
3. Contrast enhancement (always done on T1), angiography/ venography, spectroscopy or other dedicated sequences are used as per clinical requirement.
4. Information from different sequences /protocols
a. Epilepsy protocol: Thin-slice 3D T1, high-resolution T2 and FLAIR in coronal plane, preferably with dedicated hippocampal imaging, improves detection of focal cortical dysplasia, hippocampal sclerosis and subtle malformations of cortical development.
b. Diffusion Weighted images (DWI) : Restricted diffusion can identify acute ischemia, encephalitis, hypoxic-ischemic injury and some metabolic disorders very early, before other sequences become abnormal. MUST ALWAYS BE EVALUATED WITH ADC maps.
c. Susceptibility Imaging(SWI)/ Gradient Recall Echo (GRE) : Identifies hemorrhage, calcification, venous abnormalities and iron deposition
5. Sequences and protocol for difference scenarios
a. Neonatal encephalopathy: DWI/ADC show early changes of hypoxic-ischemic injury. T1/T2 evolution is informative later.
b. Metabolic disorders : Distribution of signal abnormalities provide clues: basal ganglia involvement, white-matter pattern, cortical involvement and characteristic tract changes can suggest the diagnosis
c. White-matter disorders : Assessed by pattern and distribution eg involvement of U-fibres, corpus callosum, cerebellum, corticospinal tracts. Serial MRI may be useful..
d. ADEM/ autoimmune encephalitis: Multifocal T2/FLAIR lesions, deep grey involvement or characteristic patterns may be seen. Correlation with clinical picture and CSF/serological findings is important .
e. Acute encephalitis : changes on DWI and distribution of lesions is important . eg Temporal, insular and cingulate abnormalities raise suspicion for HSV encephalitis; thalamic involvement may suggest Japanese B encephalitis
f. Posterior fossa disorders: thin-section imaging required for accurate assessment
g. Age adjustment is required during interpretation, as myelination changes rapidly in infancy upto 2 years age. Myelination is assessed on T1WI till 6 months of age and from 6 months to 2 years on T2 WI.

THE FINAL PEARL

MRI is an essential extension of the neurological examination in children. When interpreted in the context of age, with clinical correlation and disease evolution, characteristic imaging patterns from different sequences and protocols can provide crucial diagnostic clues.

Tissue characteristics in different sequences

MRI sequence	CSF	White matter	Grey matter	Key appearance / what it shows	Main clinical usefulness
T1-weighted	Dark	Bright	Intermediate–dark	Excellent anatomical detail; fat is bright	Brain anatomy, myelination, cortical development, atrophy; post-contrast T1 for enhancement
T2-weighted	Bright	Darker than grey matter	Intermediate–bright	Fluid-sensitive sequence	Oedema, gliosis, inflammation, infection, demyelination, many structural lesions
FLAIR	Suppressed/dark	Relatively dark	Relatively bright	T2 effect with CSF suppression	Particularly useful for periventricular, cortical/subcortical and white-matter lesions; gliosis, demyelination, encephalitis
DWI	Usually relatively dark	Intermediate	Bright	Restricted diffusion = bright	Acute infarction; encephalitis; abscess; hypoxic-ischaemic injury; status epilepticus; some metabolic disorders
ADC map	Bright	Intermediate	Dark	True restriction = low ADC (dark)	Confirms restricted diffusion and distinguishes it from T2 shine-through
SWI	Dark	Variable	Variable	Blood products, calcification and veins appear dark (susceptibility)	Microbleeds, haemorrhage, cavernoma, venous structures; useful in trauma and vascular disorders
GRE/T2*	Bright	Intermediate- dark	Bright	Susceptibility from blood products	Haemorrhage, calcification, cavernomas; older alternative to SWI
Post-contrast T1	Dark	Slightly brighter than grey matter	Intermediate-dark	Areas of contrast enhancement become bright	Tumour, infection, inflammation, active demyelination, meningitis, vascular lesions
T2*/perfusion	Variable	Variable	Variable	Signal changes related to blood flow/haemodynamics	Tumour characterisation, vascular lesions and selected epilepsy/tumour protocols
MR angiography (MRA)	—	—	—	Visualises arterial flow	Arterial stenosis/occlusion, aneurysm, AVM, vasculopathy, stroke
MR venography (MRV)	—	—	—	Visualises venous channels	Cerebral venous sinus thrombosis, venous anomalies

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17. Childhood Epilepsy: A Bird's-eye View

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(In Fond Memories of Late Dr. S. P. Shrivastava - Chairperson AOPN2001-2003)

- Epilepsy is diagnosed after two unprovoked seizures, or after one unprovoked seizure when the risk of recurrence is sufficiently high.
- A carefully obtained history and eyewitness description often provide more diagnostic information than the EEG.
- Not every staring spell, fall, or abnormal movement is epileptic.
- Smartphone videos recorded by caregivers can be extremely helpful in distinguishing seizures from their mimics.
- EEG supports the clinical diagnosis but should never be interpreted in isolation.
- A normal EEG does not exclude epilepsy, while an abnormal EEG does not necessarily establish it.
- MRI is particularly important when focal epilepsy, developmental abnormalities, neurological deficits, or an underlying structural lesion is suspected.
- Antiseizure medication should be selected according to the epilepsy syndrome and the child's clinical profile—not merely the apparent seizure type.
- Rescue medication and a clear seizure-action plan should be provided for children at risk of prolonged seizures.
- Failure of two appropriately chosen and adequately used antiseizure medications should raise the possibility of drug-resistant epilepsy.
- Children with drug-resistant epilepsy should be referred early to a specialised paediatric epilepsy centre for comprehensive evaluation.
- Development, behaviour, schooling, sleep, and psychosocial well-being should be reviewed alongside seizure control.
- Many childhood epilepsy syndromes have an excellent long-term prognosis with appropriate diagnosis and management.
- Clinical pearl: Treat the child, not the EEG.
- Take-home message: The goal is not merely freedom from seizures, but seizure control while preserving development, learning, and quality of life.

We look forward to your valuable contribution to this commemorative issue.

Thanks and Regards
Editorial Team
JAOPN

18. Genetic Testing In Pediatric Neurology

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INTRODUCTION

Advances in DNA sequencing have improved the identification of genetic causes of paediatric neurological disorders and the guidance of treatment, thereby enhancing patient care. Early and accurate diagnosis is crucial, as many childhood neurological disorders may have genetic origins and require treatment tailored to the specific genetic findings.
(1) This chapter provides a brief overview of different genetic techniques and their utility in child neurology practice. Diagnostic genetic testing is performed in individuals presenting with symptoms to confirm or rule out an underlying genetic disorder.(2)

KARYOTYPING (KT)

White blood cells, particularly T-lymphocytes that proliferate in culture, are commonly used for chromosomal analysis in clinical practice. A variety of other tissues, including lymphoblastoid cells, fibroblasts derived from skin biopsy, bone marrow samples, and fetal cells derived from amniotic fluid or chorionic villi sampling, can also be used for long-term cell cultures.(3) For cytogenetic analysis, a small volume of peripheral blood is collected, white blood cells are separated, and the cells are placed in tissue culture medium and stimulated to divide. The dividing cells are arrested in metaphase, and then treated with a hypotonic solution that releases chromosomes. The chromosomes are spread on slides and stained by one of several techniques. G-banding (Karyotyping) is the first-line test in patients with:

• Aneuploidy syndromes (Trisomy 21, Trisomy 13 and Trisomy 18)
• Sex chromosome aneuploidy
• Chromosomal mosaicism
• Infertility or multiple miscarriages

Fluorescent in situ hybridization (FISH)

Fluorescence in situ hybridization (FISH) is a cytogenetic method that employs fluorescently labelled DNA probes to identify specific chromosomal abnormalities. In neurological disorders, it is useful for detecting targeted changes, such as microdeletions, microduplications, and translocations, that may be difficult to identify with conventional karyotyping. The resolution of karyotyping is 5-1 Mb, while the resolution of FISH is 50 kb-1 Mb.(4) However, FISH is a targeted technique and can detect only abnormalities corresponding to the probes used; hence, chromosomal microarray (CMA) and sequencing-based approaches are generally preferred for

Chromosomal microarray (CMA)

Two major types of microarrays are used for detecting copy-number variants (CNVs): genome-wide single-nucleotide polymorphism (SNP) arrays, which analyse SNPs throughout the genome, and array comparative genomic hybridization (aCGH), which uses larger oligonucleotide probes distributed across the genome, often supplemented by a backbone of SNP probes. The resolution of SNP array is 10-100 Kb, and chromosomal microarray may be indicated in patients with unexplained intellectual disability and congenital anomalies.(4)

Multiple Ligation-Dependent Probe Amplification (MLPA)

Multiplex ligation-dependent probe amplification (MLPA) is a semi-quantitative method used to determine copy-number changes through multiplex fragment-size analysis. Sequence-specific probe pairs are hybridized to the target DNA, ligated, and subsequently amplified, with each target containing a unique-length stuffer sequence that enables identification of individual fragments.(4) MLPA is particularly useful for detecting small CNVs, including single-gene deletions and recurrent microdeletion or microduplication syndromes.(4) MLPA has been widely used in the diagnostic testing of patients with spinal muscular atrophy, Duchenne muscular dystrophy and imprinting disorders. MLPA is a targeted approach for detecting copy-number variants (CNVs). Although it can identify a CNV involving the region of interest, additional genomic techniques may be required to determine the size and precise breakpoints of larger CNVs.

Gene sequencing technologies

Gene sequencing technologies are widely used in children with suspected monogenic disorders that follow Mendelian pattern of inheritance.

First-generation sequencing technology: Sanger sequencing employs dideoxynucleotides to terminate DNA strand extension during replication, enabling the generation of sequence reads several hundred nucleotides long.(5) Sanger sequencing became widely established and facilitates the rapid and efficient determination of DNA and RNA sequences. Sanger sequencing has been widely used for segregation studies.

Second-generation sequencing technology: It enables the concurrent sequencing of thousands to millions of DNA fragments, distinguishing them from conventional Sanger sequencing through their high-throughput, massively parallel sequencing capability. The key applications include targeted genetic panels and whole-exome sequencing (WES). WES focuses on sequencing exons, which represent about 1-2% of the genome, and detects single-nucleotide variants (SNVs), insertions, deletions, and copy-number variations (CNVs) within protein-coding regions.(5) Whole-genome sequencing uses either short-read or long-read platforms to sequence the whole genome. The second-generation NGS platforms include 454 pyrosequencing, Ion Torrent, Illumina, SOLiD, and PacBio Onso system.(5)

Third-generation sequencing: Third-generation sequencing offers long-read sequencing technologies, such as PacBio single-molecule real-time (SMRT) sequencing and Oxford Nanopore DNA sequencing.(5) Whole-genome sequencing with long reads covers all genes, regulatory regions, and non-coding regions.

Mitochondrial genome sequencing: This approach typically begins with enrichment of mitochondrial DNA (mtDNA) from total genomic DNA, commonly achieved through long-range PCR amplification, followed by high-throughput next-generation sequencing (NGS) to analyse the mitochondrial genome.

Repeat-Primed PCR

Repeat-Primed PCR can typically quantify 1-220 repeats and detect up to 1,000 repeats. This testing is helpful in the diagnostic workup of children with repeat expansion disorders.

Conclusion

When genetic testing is clinically indicated, its application requires consideration of several factors, including established knowledge of the disease, available genetic testing techniques, and the sensitivity and specificity of each approach.

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19. Autism Spectrum Disorder

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Chairperson AOPN 2024-25

- **1. Think Autism Early**
- ASD may become clinically apparent in the second year of life. Lack of response to name, poor joint attention, limited pointing or showing, reduced social reciprocity, limited pretend play and atypical language should prompt closer developmental assessment.
- **2. Language Delay Alone Is Not Autism**
- The quality of social communication is more informative than the number of words. A child may have words or even fluent speech but still have marked difficulty with reciprocal conversation, sharing interests and understanding social cues.
- **3. Look for Joint Attention**
- Failure to point to share interest, follow another person's point or consistently bring an object to show someone can be an early and clinically useful clue.
- **4. Regression Deserves Attention**
- Loss of previously acquired language, social interaction or adaptive skills warrants prompt evaluation. Ask specifically about regression rather than waiting for parents to volunteer the history.
- **5. Screen but Do Not Diagnose by Questionnaire**
- Autism-specific screening at 18 and 24 months, along with ongoing developmental surveillance, is recommended. M-CHAT-R/F can identify children who need further assessment, but a negative screen should not override significant clinical concern.[1]
- **6. Diagnosis Remains Clinical**
- A detailed developmental history and direct observation, interpreted using DSM-5 or ICD-11 criteria, remain central. No blood test, EEG, imaging or single instrument can independently establish or exclude ASD.[2]
- **7. Do Not Delay Intervention for Diagnostic Perfection**
- A child with clear difficulties should receive appropriate developmental intervention while assessment is progressing. Early, evidence-based intervention can improve communication, adaptive functioning and participation.[1,3]
- **8. Search Actively for Comorbidities**
- Intellectual disability, language disorder, ADHD, anxiety, sleep disturbance, feeding difficulties, constipation, epilepsy and behavioural difficulties may substantially influence functioning.
- **9. Investigate Selectively**
- Hearing assessment should be part of evaluation. EEG is indicated only when seizures are suspected, not routinely. Genetic evaluation should be considered when the phenotype or family history suggests an underlying genetic cause.
- **10. Avoid the Autism Explains Everything Trap**
- New neurological symptoms, regression, seizures, gait abnormalities, focal deficits or unusual changes in behaviour should be evaluated on their own merits.
- **11. Management Should Be Individualized**
- Speech-language therapy, occupational therapy, developmental and behavioural approaches, parent-mediated interventions and educational support should be selected according to the child's functional needs, strengths and family priorities.[3]
- **12. Medication Does Not Treat Core Features**
- Pharmacological treatment may have a role for selected associated problems such as severe irritability, aggression, ADHD symptoms, anxiety or significant sleep difficulties.
- **13. Parents Are Partners Not Observers**
- Families often notice subtle differences before professionals do. Their observations are an essential part of both diagnosis and follow-up.
- **14. Follow the Child Longitudinally**
- Development, communication, adaptive skills, behaviour, sleep, nutrition, schooling, family functioning and quality of life should be reviewed over time.

Clinical Pearl

When a child does not respond, do not simply ask, "How many words does the child have?" Ask, "How does the child connect, communicate and share the world with others?"

Take Home Message

Recognize early, diagnose thoughtfully, investigate selectively and intervene according to need. Autism is a diagnosis, but the child's individual strengths, abilities and support needs should always remain the priorities.

20. Current Management Scenario Of Neuromuscular Disqaorders In Children

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- The way we look at neuromuscular disorders has changed. These were traditionally considered largely supportive-care disorders; today, for several conditions, an early diagnosis can open the door to disease-modifying treatment.
- In India, the first challenge is still reaching the diagnosis. Delayed recognition, limited awareness, variable access to genetic testing and the scarcity of specialised centres continue to affect outcomes, particularly outside major cities.
- Think neuromuscular when a child is weak, not just when the child is hypotonic. Motor regression, difficulty getting up from the floor, Gowers sign, frequent falls, calf hypertrophy, fatigability, ptosis, bulbar weakness or unexplained respiratory problems should prompt evaluation.
- A CK is a simple but powerful first test. A very high CK should immediately raise the possibility of muscular dystrophy. However, a normal CK does not rule out SMA, congenital myopathy, neuropathy or congenital myasthenic syndrome.
- Genetic testing has moved from being a confirmatory test to becoming part of first-line diagnosis. With targeted testing, gene panels and exome/genome sequencing increasingly available, the molecular diagnosis may also tell us whether a child has a treatment option.
- SMA is perhaps the clearest example of this change. Nusinersen, risdiplam and onasemnogene abeparvovec have transformed the natural history of SMA. The important message is that earlier treatment gives better outcomes—making newborn screening, rapid diagnosis and referral increasingly important.
- The Indian SMA story, however, is not simply about availability of drugs. Affordability, timely access, awareness, limited centres with multidisciplinary expertise and long-term follow-up remain major challenges. In India, these practical issues can determine whether a child actually benefits from an available therapy.
- Duchenne muscular dystrophy is similarly moving beyond steroids alone. Corticosteroids remain an important part of treatment, while mutation-specific therapies and gene therapy have added new possibilities. At the same time, cardiac, respiratory, orthopaedic and rehabilitation care remain essential.
- New therapies should not make us forget supportive care. In LMIC settings especially, good respiratory care, nutrition, physiotherapy, contracture prevention, cardiac surveillance and appropriate assistive devices can make a major difference even when advanced therapies are not accessible.
- Respiratory deterioration can be insidious. Sleep-related hypoventilation, ineffective cough and recurrent chest infections may precede obvious respiratory failure. Anticipatory respiratory assessment,airway-clearance strategies and timely sleep studies are therefore important.
- Multidisciplinary care is the dictum. Neurology, pulmonology, cardiology, nutrition, rehabilitation, orthopaedics, genetic and psychosocial support need to work together, particularly for children with progressive disorders. This remains an important gap in many parts of India.
- What is new is not only more drugs, but a change in the therapeutic philosophy. newborn screening, early molecular diagnosis, disease-specific therapy and increasingly personalised treatment are becoming possible.
- The challenge for the next decade is to make these advances equitable. For India and other LMICs, the goal should not only be to bring new therapies into the country, but also to build systems that allow children to be diagnosed early, treated on time and followed throughout childhood.

Clinical pearl: In paediatric neuromuscular disease, the most important treatment decision may be made before the treatment is even started—recognising the disease early.

Take-home message: Diagnose early, confirm genetically, treat when a disease-modifying option exists, and provide good multidisciplinary care to every child—whether or not the newest therapy is accessible.

21. CNS INFECTIONS

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Central nervous system infections are potentially life-threatening, particularly in children, and may result from a wide range of infectious agents. They can present as diffuse involvement of the meninges, brain, or spinal cord, or as localized space-occupying lesions. Diagnosis relies on integration of epidemiological factors, clinical presentation, and cerebrospinal fluid findings, with syndromic classification into acute bacterial or aseptic meningitis, chronic meningitis, or focal CNS infection guiding further evaluation.

Common clinical Presentations

CNS infections commonly present with the classical triad of :fever, headache, and altered mental status. Classical triad may be absent if the child was treated with antibiotics before the hospital admission.
In Infants-hyperthermia or hypothermia, poor feeding, irritability or excessive crying, lethargy,seizures and a bulging anterior fontanelle.
Classical features of meningitis may be absent, making a high index of suspicion essential.

Other symptoms: Seizures, Autonomic disturbances.

Red Flags

Severe or complicated infections may be associated with focal neurological deficits, features of raised intracranial pressure, septic shock, or a characteristic rash, particularly in meningococcal infection.

Features of raised Intra Cranial Pressure

Headache: Typically more pronounced in the morning and may worsen with lying flat, bending, or coughing, reflecting raised intracranial pressure.

- Vomiting
- Altered Mental status/Behavioural changes
- Vision Changes-Diplopia/Blurred vision
- Examination
- Vitals-Blood pressure,Pulse Rate.
- Signs of Meningeal irritation- Neck Rigidity,Kernigs,Brudzinski Signs.
- Fundus-Pappiledema
- Cranial Nerve Palsies-Commonly seen in CNS TB

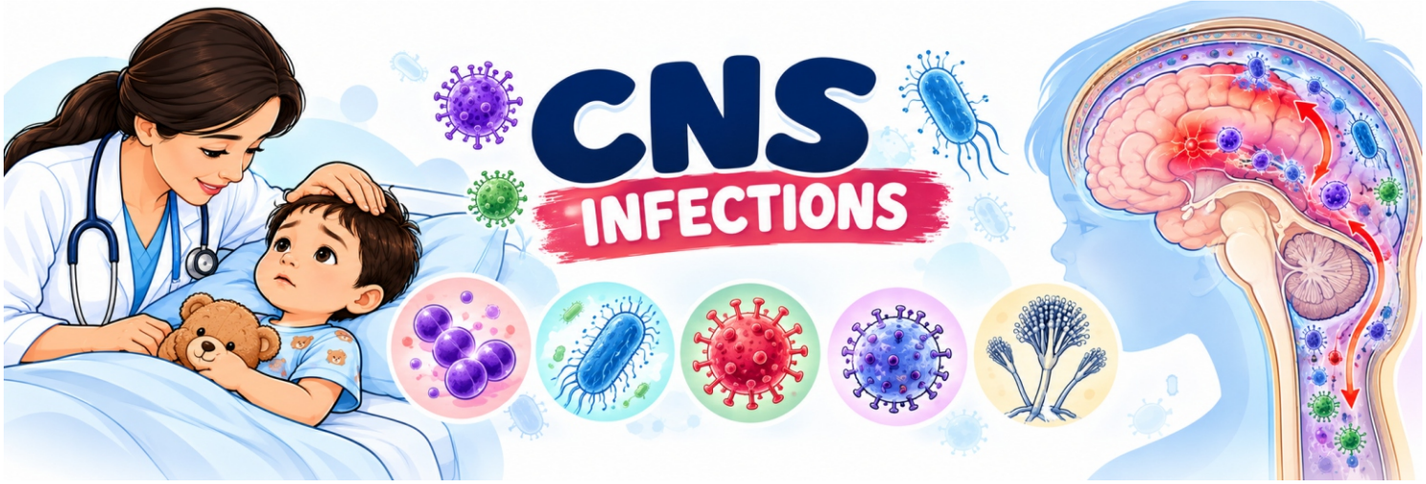
Specific Clinical Presentations

Neurocysticercosis (NCC):
Seizures are the most common presentation; headache and focal neurological deficits may also occur.

Tuberculous meningitis:
Subacute headache, low grade fever, vomiting, altered sensorium, cranial nerve palsies, and features of raised intracranial pressure are characteristic.

Viral encephalitis:
Fever, headache, altered mental status, behavioral changes, seizures, and focal neurological deficits are typical.

Fungal meningitis:
Usually presents with a subacute or chronic headache, fever, nausea/vomiting, altered mental status, and features of raised intracranial pressure, particularly in immunocompromised individuals.



Essential Diagnostic Investigations

- Complete Blood count, CRP, BloodCultureESR, Liver function Test, Urea, Creatinine, S.Electrolytes, Procalcitonin.
- CSF Analysis-Protein,Sugar, Cell Count, Cell Type, Gram stain,CSF culture
- CNS tuberculosis suspected – GeneXpert, MTB/RIF ultra PCR assay
- Neuroimaging-MRI Brain with contrast is the Investigation of choice, MR Angiography-Suspected Vasculitis.

Brain imaging should be done before LP :

- GCS score <12
- Status epilepticus or any focal neurologist deficits.

Neuroinfection	Typical MRI lesion pattern	Key distinguishing features
Neurocysticercosis	**Ring Enhancing lesion Different stages are seen**	**Eccentric scolex; edema and enhancement vary with stage; calcified lesions may show susceptibility on MRI**
Tuberculous meningitis	**Ring Enhaning lesions-Conglomerate. Basal meningeal enhancement**	**Basal exudates, hydrocephalus, infarcts, and tuberculomas; often involves basal cisterns**
Viral encephalitis	**T2/FLAIR hyperintense parenchymal lesions**	**1.Temporal and insular involvement is characteristic of HSV; cortical/ deep gray-matter involvement may occur. 2.Dengue Encephalitis-Bi thalamic Involvement with hemorrahgic foci-Double doughnut Sign Japanese Encephalitis-Bithalamic Involvement.**
Fungal meningitis	**Meningeal or parenchymal lesions**	**Leptomeningeal enhancement, granulomas/abscesses, infarcts, and vasculitic changes; more common/severe in immunocompromised children.**
Brain abscess	**Well-defined ring-enhancing lesion**	**Central diffusion restriction, surrounding edema, and mass effect; capsule may be smooth and well defined**

CSF BioFire FilmArray is a rapid multiplex PCR-based molecular assay used for the detection of common pathogens causing meningitis and encephalitis. It simultaneously identifies multiple bacterial, viral, and fungal pathogens from a small CSF sample, providing results within approximately 1 hour.

First Line management

Supportive Management:
IV fluids,Osmotherapy for Raised ICT -3 %Nacl or Mannitol,Antiseizure Medication in case of Seizures.

Corticosteroids:
Better to be given before or within 4 hours of first antibiotics dose and can be considered in presence of focal neurological deficits. (Not useful if child has received either oral or injectable antibiotics prior to steroid administration).
Antibiotics should be started as early as feasible even empirically.
Duration of antibiotics therapy. They should be given intravenously.
Unspecified bacterial meningitis: 10–14 days
Neisseria meningitidis: 5–7 days
Haemophilus influenzae: 7–10 days
Streptococcus pneumoniae: 10–14 days
Gram-negative bacillary and pseudomonal meningitis: 21–28 days

Antiviral medications- IV Acyclovir-10 mg/kg body weight every 8 hours.
AntiTubercular Therapy for CNS TB

Neurocysticercosis-Albendazole 15 mg/kg/day PO in 2 divided doses; if >2 viable parenchymal cysts, add praziquantel 50 mg/kg/day.Coricosteroids -Dexamethasone (3-6 mg/kg/day for 3-5 days.maximum 16 mg/day).

Prognosis and outcome

- Outcome depends on the causative organism, age, severity, site of CNS involvement, and timely and appropriate treatment.
- Early recognition and accurate differentiation from non-infectious neurological disorders are crucial to reduce mortality and neurological sequelae.
- Poor prognostic factors include:
  - Delayed treatment
  - Altered consciousness
  - Seizures
  - Focal neurological deficits
  - Raised intracranial pressure
  - Hydrocephalus
  - Septic shock
- Complications may include:
  - Hearing or visual impairment
  - Epilepsy
  - Cognitive and developmental impairment
  - Motor deficits
  - Cranial nerve palsies
  - Hydrocephalus
- Persistent neurological disability:
  Early Neurorehabilitation: including physiotherapy and early intervention therapy—should be initiated in children with neurological sequelae to maximize functional recovery and developmental outcomes.

22. ADHD: Diagnosis and Management: Attention to action!

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Chairperson AOPN 2022 - 23

1. ADHD is a neurodevelopmental disorder characterized by persistent, developmentally inappropriate hyperactivity–impulsivity and /or inattention causing significant functional impairment.
2. Common presentations include hyperactivity, distractibility, inattention, impulsivity, emotional dysregulation, poor task completion, academic difficulties and peer problems; symptoms must be present but may differ between different settings like home and school.
3. ADHD can be diagnosed from 4 years of age, but diagnosis in preschool children requires caution because higher activity levels and limited attention span overlap substantially with normal development. Thus, in younger children, establish that symptoms are clearly excessive for developmental age, persistent, present across settings, and associated with meaningful impairment.
4. Essentially, diagnosed with information from two settings eg. parents and teachers. Vanderbilt, Conners, and INDT-ADHD are a few rating scales that can quantify symptoms and impairment. Questionnaires should not be used in isolation but coupled with clinical context.
5. Detailed history and assessment should include ruling out mimics and evaluating comorbidities, including sleep disorders, language/learning difficulties, autism, intellectual disability, hearing/vision problems, epilepsy, anxiety, and psychosocial adversity.
6. Consider developmental correlates, family stressors, inconsistent routines, excessive screen exposure, inadequate sleep, classroom/environmental factors, child's abilities, and parental/curriculum/cultural expectations mismatch, particularly in younger children.
7. Routine EEG, brain MRI or extensive laboratory investigations are not indicated in uncomplicated ADHD unless history or examination suggests seizures, developmental regression, neurological disorders, or another specific disorder necessitating investigations.
8. Management should be individualized, combining psychoeducation, positive parenting behavioural strategies, school-based interventions and combined with medication when clinically indicated.
9. In preschool children, parent training and environmental/behavioural modifications are first-line. Medication should generally be considered only for persistent, significant functional impairment despite adequate behavioural intervention.
10. In school-aged children with significant impairment, the medication plan may include stimulants, particularly Methylphenidate where appropriate and available. Atomoxetine or α2-adrenergic agonists may be considered as suitable.
11. Prior to use and during medication follow-up visits, monitor height, weight, appetite, sleep, blood pressure, pulse, and adverse effects with review of functional goals. Medication should be reviewed periodically with reassessment of ongoing need.
12. ADHD may persist into adolescence and adulthood, although symptoms and impairment will vary with age and situational demands. Family counseling regarding academic functioning and limitations/demands, self-esteem, peer relationships, safety, and substance-use risk as they transition into adolescence should be included as relevant.

Clinical pearl: Not every hyperactive child has ADHD. First, understand the child's developmental stage, family, school, and environment; modify what is modifiable, strengthen behavioural strategies, and use medication when a well-established diagnosis and significant impairment justify it.

23. Neurodevelopmental monitoring of NICU graduates

Dr. Vilas Jadhav
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- Neurodevelopmental impairment (NDI) includes one or more cognitive, motor, sensory (vision and hearing loss), behavioral, attentional and/or psychological impairments.
- Risk factors at discharge for NDI include gestational age <30 week, multisystem congenital malformations, neonatal course that includes - severe asphyxia, postnatal hypoglycemia, intrauterine growth retardation, severe intraventricular hemorrhage (IVH), periventricular leukomalacia or infarction, meningitis, seizures, respiratory failure requiring mechanical ventilation, and poor growth (including head growth).
- The available clinical tools to predict outcome include - neuroimaging and early childhood clinical assessment during the first two years of life.
- Neuroimaging should not be used as the sole accurate predictor of long-term neurodevelopmental outcome for individuals as it poorly predicts cognitive function.
- Studies have shown correlation between MRI findings and long-term neurodevelopmental outcomes, particularly if the MRI is grossly abnormal (predictive of poor neurodevelopmental outcome) or normal (predictive of favourable neurodevelopmental outcome).
- Early clinical assessment can identify moderate to severe impairments. The formal clinical assessment for NDI includes detailed neurologic examination focusing on identifying motor deficits, visual assessment, hearing assessment and use of standardised tests for cognitive and motor function, language, social development, and behaviour.



24. AFP in Children

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Secretary General AOPN 2025 - 27
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- Acute Flaccid Paralysis (AFP) is defined as acute onset of flaccid weakness in any child <15 years — a notifiable public health emergency under WHO surveillance, not merely a clinical diagnosis.
- Despite polio elimination, every AFP must be treated as suspected polio until proven otherwise. Notify immediately to District Immunization Officer / WHO-NPSP and collect two stool samples (8-10g each), 24-48 hours apart, within 14 days of onset in reverse cold chain.
- History is the best localizer: Clarify fever at onset, tempo of progression (ascending vs descending), symmetry, sensory complaints, bladder-bowel involvement, back pain, preceding GI/respiratory illness or vaccination, and history of animal bite / toxin exposure.
- Examination pattern defines etiology: Look beyond flaccidity. Assess cranial nerves (especially extraocular movements, facial and palatal palsy), respiratory effort (single breath count), autonomic signs (hypertension, tachycardia, sweating), and sensory level.
- Etiology in India has shifted: Guillain-Barré Syndrome (GBS) is now the commonest cause of AFP, followed by transverse myelitis, non-polio enteroviral myelitis, traumatic neuritis, hypokalemic periodic paralysis, and acute flaccid myelitis associated with encephalitis.
- GBS: Acute symmetric ascending weakness, areflexia, relative fever-free at onset, prominent radicular pain/cramps, minimal sensory loss. CSF shows albuminocytologic dissociation after first week. Consider Miller Fisher variant if ataxia, areflexia and ophthalmoplegia coexist.
- Transverse Myelitis: Look for acute sensory level, early bladder-bowel involvement, back pain and brisk reflexes after initial spinal shock. MRI spine with contrast is diagnostic and urgent.
- Non-polio enteroviruses and Acute Flaccid Myelitis: Often asymmetric, patchy weakness with preceding febrile illness, pleocytosis in CSF, MRI shows longitudinal cord grey matter hyperintensity.
- Never miss in first hour: Blood pressure, single breath count, gag reflex and GCS. Respiratory failure and autonomic dysfunction are the leading causes of mortality in AFP.
- Focused workup in first 24 hours: Stool for polio virology (mandatory in all), serum electrolytes with K+, Ca++, CPK, MRI spine if myelopathy, Nerve Conduction Study after day 5-7 if GBS considered, CSF analysis once stable.
- Common pitfalls: Administering corticosteroids empirically in GBS worsens outcome, intramuscular injections in a child with evolving AFP can provoke paralysis, and normal early NCS/CSF does not exclude GBS.
- Management is etiology-specific: GBS requires IVIg 2g/kg over 2-5 days or plasmapheresis; transverse myelitis requires high-dose IV methylprednisolone; hypokalemic paralysis needs prompt K+ correction with cardiac monitoring; physiotherapy is contraindicated in acute phase.
- Indications for urgent PICU referral: Rapidly ascending weakness, bulbar involvement, single breath count <15, autonomic instability, or urinary retention with sensory level.
- Prognosis: GBS and hypokalemic paralysis have excellent recovery with timely therapy, whereas delayed diagnosis of myelitis or polio results in residual deficits.
- Clinical pearl: Symmetry guides you — symmetric ascending weakness suggests GBS, asymmetric patchy weakness suggests polio / non-polio enterovirus, symmetric weakness with a sensory level suggests myelitis.

- Take-home message: Every AFP is polio until proven otherwise — notify early, collect two adequate stool samples early, and think GBS first, non-polio enteroviruses next, and myelitis always.



25. Pediatric Stroke

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Senior Child Neurologist ,Bangalore

- How is it different: Pediatric ischemic stroke presents distinct challenges, like a higher frequency of stroke mimics (seizure, migraine, trauma, tumor) obscuring diagnosis, less validated prehospital and hospital screening tools, variable evidence for efficacy and safety of hyperacute therapies, and a greater need for multidisciplinary coordination among pediatricians, neurologists, radiologists, and interventionalists.
- Stroke or not a stroke: It is important to know the difference between stroke and stroke-like episodes. Stroke-like episodes are not directly caused by a vascular event, and the neurological deficit is attributed to a non-vascular cause. This is in stroke contrast, which is believed or proven to be due to a vascular etiology.
- Localization: Above the brainstem, contralateral weakness and cranial nerve involvement; below the brainstem is characterized by ipsilateral cranial nerve involvement and contralateral weakness to the side of the lesion. Depending on the site of the brainstem involved, corresponding cranial nerve nuclei and involved structures are affected.
- Screening Tools: Use of stroke screening tools helps emergency physicians to effectively identify stroke and transport such patients to appropriate facilities. New pediatric-specific tools, like the pediatric modification of the National Institutes of Health Stroke Scale (PedNIHSS) and Pediatric Rapid Arterial Occlusion Evaluation (PedRACE), show good interrater reliability.
- Imaging: MRI Brain is the investigation of choice. However, a computerised tomography (CT) brain can be performed within one hour of arrival at hospital in every child with a suspected stroke, including CT angiography (CTA) from vertex to neck.
- MR Angiography (MRA)? Vessel anatomy/ flow voids could be visualized clearly. MRA can confirm vascular occlusion and suggest possible arteriopathy if it involves large/medium vessels (often normal in small vessel vasculopathy).
- Intravenous Thrombolytics (IVT). The newer 2026 AHA guideline recommends IVT with alteplase in children aged 28–18 years presenting within 4.5 h of a confirmed AIS, and with neurological deficits. TIPS trial confirmed the safety of IV alteplase in children at a dose of 0.9 mL/kg; however, its efficacy remains uncertain.
- Endovascular Thrombectomy (EVT). The 2026 AHA/ASA guideline recommends that EVT can be effective in improving long-term functional outcomes for pediatric patients ≥ 6 years of age with large vessel occlusion, presenting within 6 h of symptom onset, and for patients presenting between 6 and 24 h of symptom onset with neuro-imaging suggestive of a salvageable penumbral region. For younger children aged 28 days–6 years with AIS due to large vessel occlusion, EVT may improve functional outcome in carefully selected patients.
- Secondary Preventive Therapies. The 2019 AHA suggests using either an antiplatelet or an anticoagulant, depending on the cause, for at least 2 years post-stroke.
- Antiplatelet agents like aspirin are recommended in children with ischemic cerebral infarction.
- Anticoagulation with warfarin is recommended if there is a substantial risk of recurrent cardiac embolism, arterial dissection, and hypercoagulable states.
- Cerebral Sinus Venous Thrombosis (CSVT): Low molecular weight heparin or unfractionated heparin, followed by switching to oral warfarin to maintain long-term anticoagulation for 1- 2 years or even lifelong, depending on etiology.

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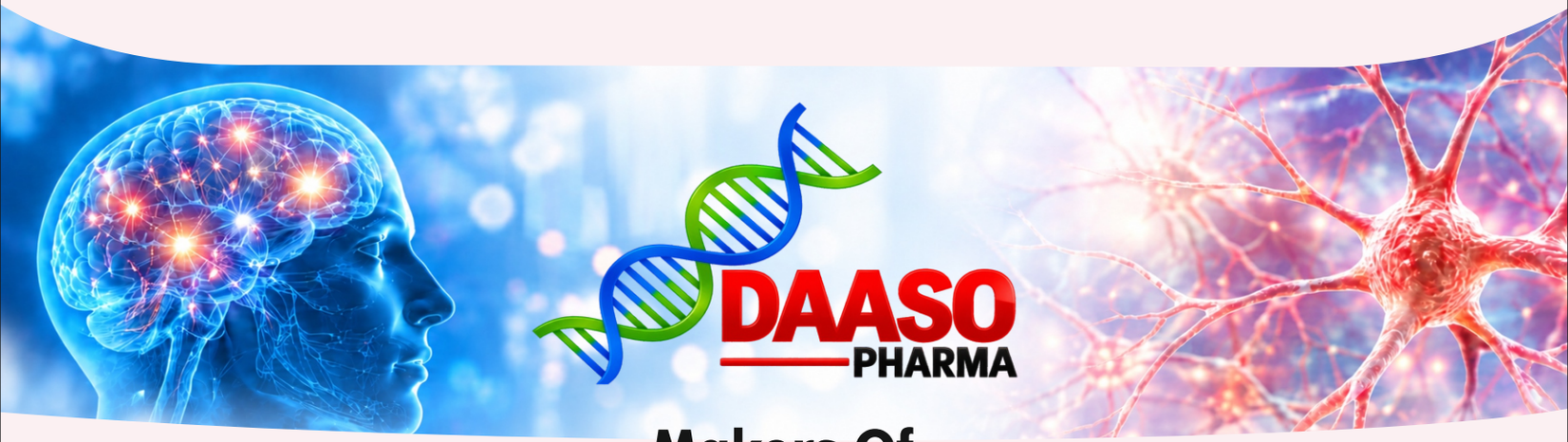
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Each 5ml Contains : Omega 3 FA : 700mg
EPA : 320mg DHA : 265mg Vit D3 : 400 IU

Q-CARNI10 Tab. / Syrup
Each: 5ml Contains : Co enzyme Q 10-50mg
& L carnitine - 500mg

Glocarno 400
(Each 5ml Contains : L Carnosine 400 Mg)
L-Carnosine Syrup | Disp.Tablet

Significant Advance in the Treatment of Schizophrenia
ARIPASO
aripiprazole 2/5/10 mg TABLETS

MIBASO
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Each Tablet Contains: Methylcobalamine 1500mcg And Folic Acid : 5mg)

TINASO
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(Biotin 5 mg / 10 mg)

Hexyfen-2
Trihexyphenidyl Hydrochloride 2mg Tablet

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Tablet
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CARNIPED Syrup
Each: 5 ml Contains : L Carnitine - 500 mg

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(Each ml contains: PYRIDOXAL 5 PHOSPHATE – 5 mg)
(PYRIDOXAL 5 PHOSPHATE – 50 mg Tablet)

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Dasovit
Multivitamin Multimineral & Lysine syrup

DASOVORIN
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Each 5 ml contains - L-Carnosine 200 mg.
EPA 320mg,DHA 265g,CoQ10-50 mg
SYRUP

DASOMID
Midazolam 1.25 mg/0.1ml Nasal Spray

Paintings by Neurodiverse Children.
(Courtesy: Dr. Jasodhara Chaudhari and Divyayogi Foundation)

