MRR Children's Hospital

ADVANCES IN PAEDIATRICS

THE MONTHLY NEWSLETTER

AUGAST 2026

30TH EDITION

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ADVANCES IN
PAEDIATRICS

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(Editor-In-Chief)

Dr Praveen Gokhale

Dear Friends and Colleagues,

As we embrace the vibrant month of August – the season that brings fresh challenges of water-borne infections, vector-borne illnesses and heightened seasonal vulnerabilities for our children – we renew our steadfast commitment to the safety, protection and holistic well-being of every child.

In the inspiring words of **Kailash Satyarthi,** Nobel Peace Prize Laureate, **"It is time for every child to have a right to life, right to freedom, right to health, right to education, right to safety, right to dignity, right to equality and right to peace."** This powerful vision calls us to prioritise compassion, prevention, innovation and family-centred care in paediatric practice.

August offers meaningful opportunities to spotlight child and adolescent health through key worldwide observances. **World Breastfeeding Week (1–7 August)** highlights breastfeeding as one of the most effective interventions for child survival, optimal nutrition, immunity and bonding – especially vital as monsoon-related gastroenteritis and respiratory infections rise. **International Youth Day (12 August)** reminds us of the unique needs of adolescents, urging us to support their mental wellness, responsible screen use and holistic development as they navigate today's complex world and shape the future.

In this edition, we are delighted to present our **Featured Article** on **Disorders of Sex Development (DSD)** – a vital topic that stresses awareness, early recognition and prompt, protocol-driven intervention.

Our **Breaking News** section brings the **Emerging Biological Subtypes of Autism Spectrum Disorder and Their Implications in Paediatric Practice.**

To support clinical excellence at the bedside, we introduce our **Decision-Making Tree** on the **Evaluation of True Vertigo in Children** – a practical algorithm designed to guide swift, confident decisions in both emergency and outpatient settings.

In our **Past Curious segment,** we journey through medical history with the **Evolution of BMT in Children,** tracing its remarkable path of innovation.

Our **Subspecialty Spotlight** shines on a compelling case successfully managed by our NICU team, showcasing the power of multidisciplinary expertise and compassionate, family-centred care in action.

In the **Sampoorna Swasthya Sanwardhan (संपूर्ण स्वास्थ्य संवर्धन)** section, we explore a comprehensive article on **Screen Use in Children and Adolescents and How to Guide Parents**.

Finally, our **PaediaBliss corner** invites us to pause and cherish the pure delight of childhood – the laughter, curiosity and playful adventures that remind us why our work matters so deeply.

Here's to an August brimming with awareness, collaboration and unwavering commitment to paediatric excellence.

Take & Give Care

Warm regards,

Dr Sandeep M. Kelkar MD, Dch
Editor-in-Chief & Director-Department of Paediatrics

Featured Article

Making Sense Of The Basics - Disorders Of Sex Development

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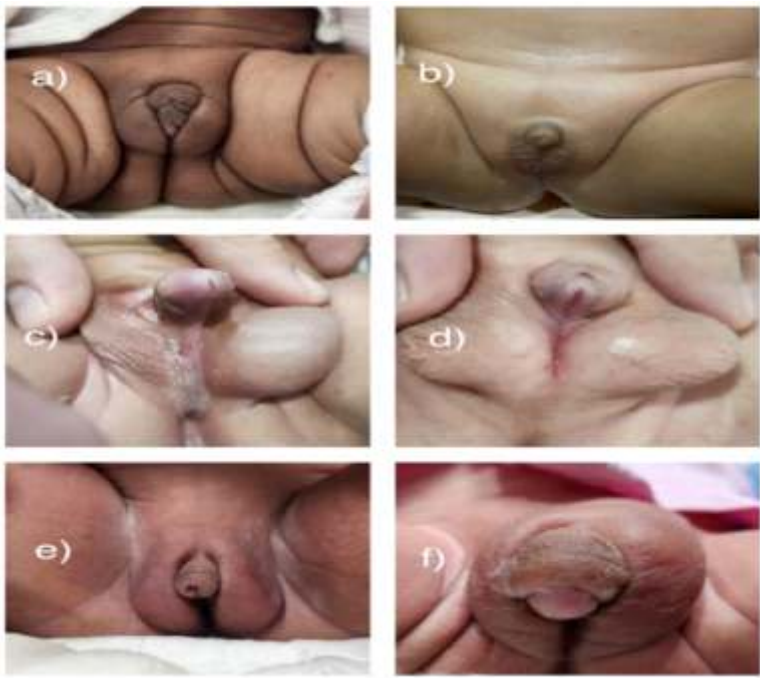
Definition

Disorders of Sex Development (DSD) comprise congenital conditions in which chromosomal, gonadal, or anatomical sex development is atypical.

The following phenotypes fit the descrption of DSD (accompanying figures for reference)

a) Atyical genital appearance
b) Phallus & bilaterally non-palpable testes
c) Unilateral undescended testes with hypospadias
d) Severe penoscrotal, scrotal, or perineal hypospadias, with or without micro- phallus, even if the testes are descended
e) Apparent female appearance with enlarged clitorophallus (clitoromegaly) and/or inguinal hernia(s) or palpable gonad(s)
f) Asymmetry in size, pigmentation and/or rugation



Classification

46XY DSD

Defects in testosterone production

Leydig cell hypoplasia or agenesis [human chorionic gonadotropin/luteinizing hormone (hCG/LH) receptor mutation]

Testicular 17B-hydroxysteroid dehydrogenase type 3 deficiency

Defects in testicular and adrenal steroidogenesis

StAR (steroid acute regulatory) protein deficiency (lipoid adrenal hyperplasia)

3B-hydroxysteroid dehydrogenase type 2 deficiency

17a-hydroxylase/17,20 lyase deficiency

Defects in testosterone metabolism

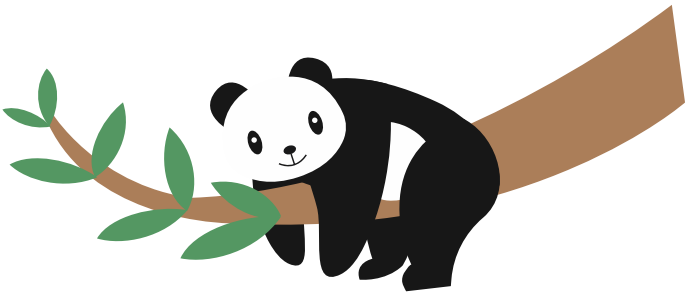
5a-reductase type 2 deficiency

Defects in testosterone action

Complete androgen insensitivity syndrome

Partial androgen insensitivity syndrome

Exogenous maternal steroid exposure



46XX DSD

Excessive androgen production

- 21a-hydroxylase deficiency
- 11B-hydroxylase deficiency

3B-hydroxysteroid dehydrogenase type 2 deficiency

- Defect in androgen metabolism
- Placental-fetal aromatase deficiency

Maternal steroid exposure

- Maternal androgen production
- Progestational agents

Other causes:

Genetic Disorders Of Gonadal Determination

- 45, XO sex chromosome aneuploidy
- Sex chromosome DSD
- 47,XXY sex chromosome aneuploidy
- Ovotesticular DSD
- 46,XX testicular DSD
- 46,XY complete gonadal dysgenesis

Other Abnormalities Of Sexual Differentiation

- Testicular regression syndrome
- Anomalies of Müllerian structures
- Persistent Müllerian duct syndrome
- Rokitansky-Küster syndrome
- Múllerian duct aplasia-renal agenesis- cervicothoracic somite
- dysplasia (MURCS) association.

What Causes DSD?

Normal external genital differentiation depends largely on androgen exposure. In the absence of androgen, the fetus develops “female like” external genitalia. Inadequate androgen production or action in a male fetus results in undervirilisation, whereas excess androgen exposure in a female fetus causes virilisation.

Development Of External Genitalia (Fig)

Androgens promote phallic growth, fusion of labioscrotal folds, and migration of the urethral opening toward the tip of the phallus. Testicular development is controlled by the Y chromosome through SRY, SF1 and SOX9 acting on the bipotential gonad.

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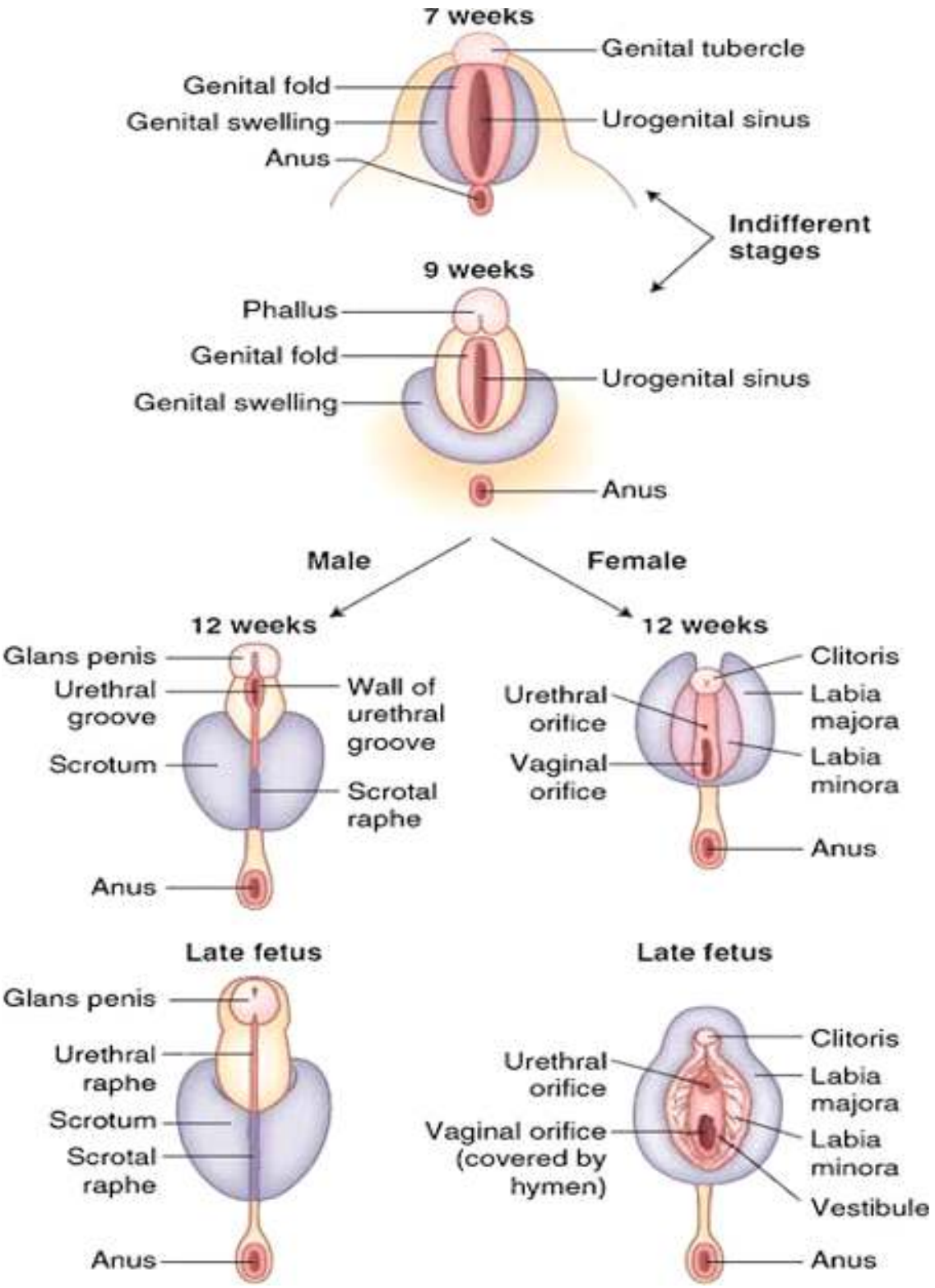

7 weeks
Genital tubercle
Genital fold
Genital swelling
Urogenital sinus
Anus
Indifferent stages
9 weeks
Phallus
Genital fold
Urogenital sinus
Genital swelling
Anus
Male
Female
12 weeks
Glans penis
Urethral groove
Wall of urethral groove
Scrotum
Scrotal raphe
Anus
12 weeks
Clitoris
Urethral orifice
Labia majora
Vaginal orifice
Labia minora
Anus
Late fetus
Glans penis
Urethral raphe
Scrotum
Scrotal raphe
Anus
Late fetus
Clitoris
Urethral orifice
Labia majora
Vaginal orifice (covered by hymen)
Labia minora
Vestibule
Anus

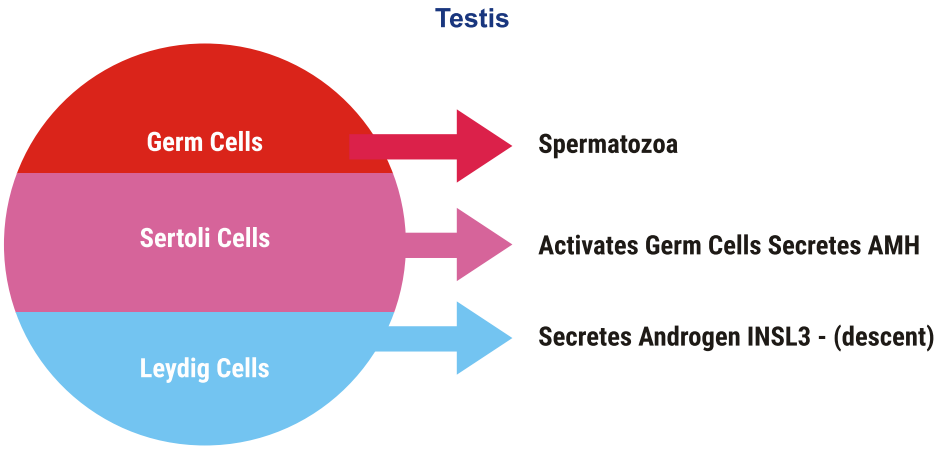
Sex Determinants In 46XY Fetus

Sex development is influenced by genetic sex, gonadal differentiation, hormone production, hormone action, genital appearance and gender identity.

Role Of Testis

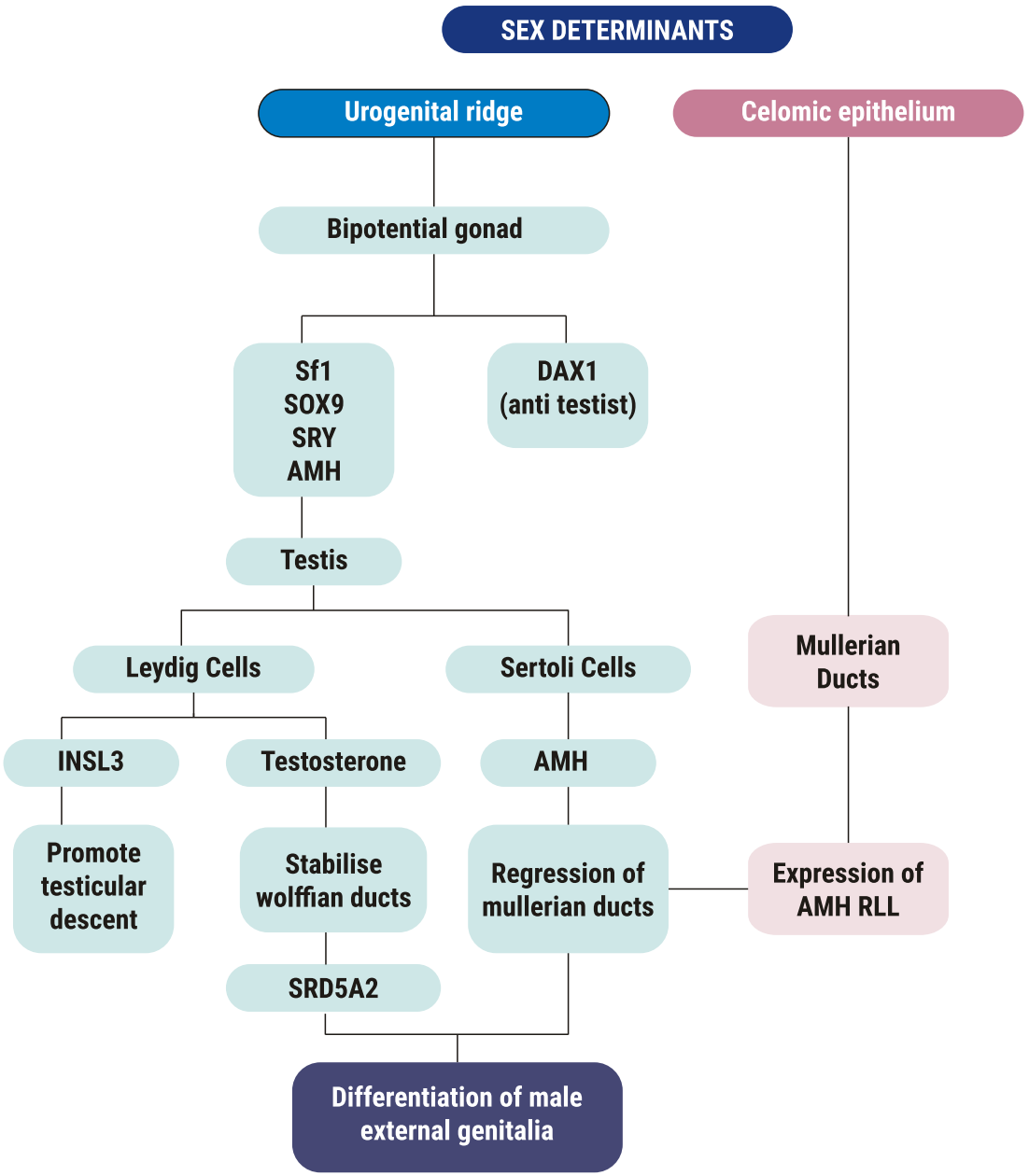
The testis contains Sertoli cells, Leydig cells and germ cells. Sertoli cells secrete anti- Müllerian hormone (AMH), causing Müllerian duct regression. Leydig cells produce testosterone and INSL3, essential for masculinisation and testicular descent respectively.

Failure of these functions leads to various forms of DSD.



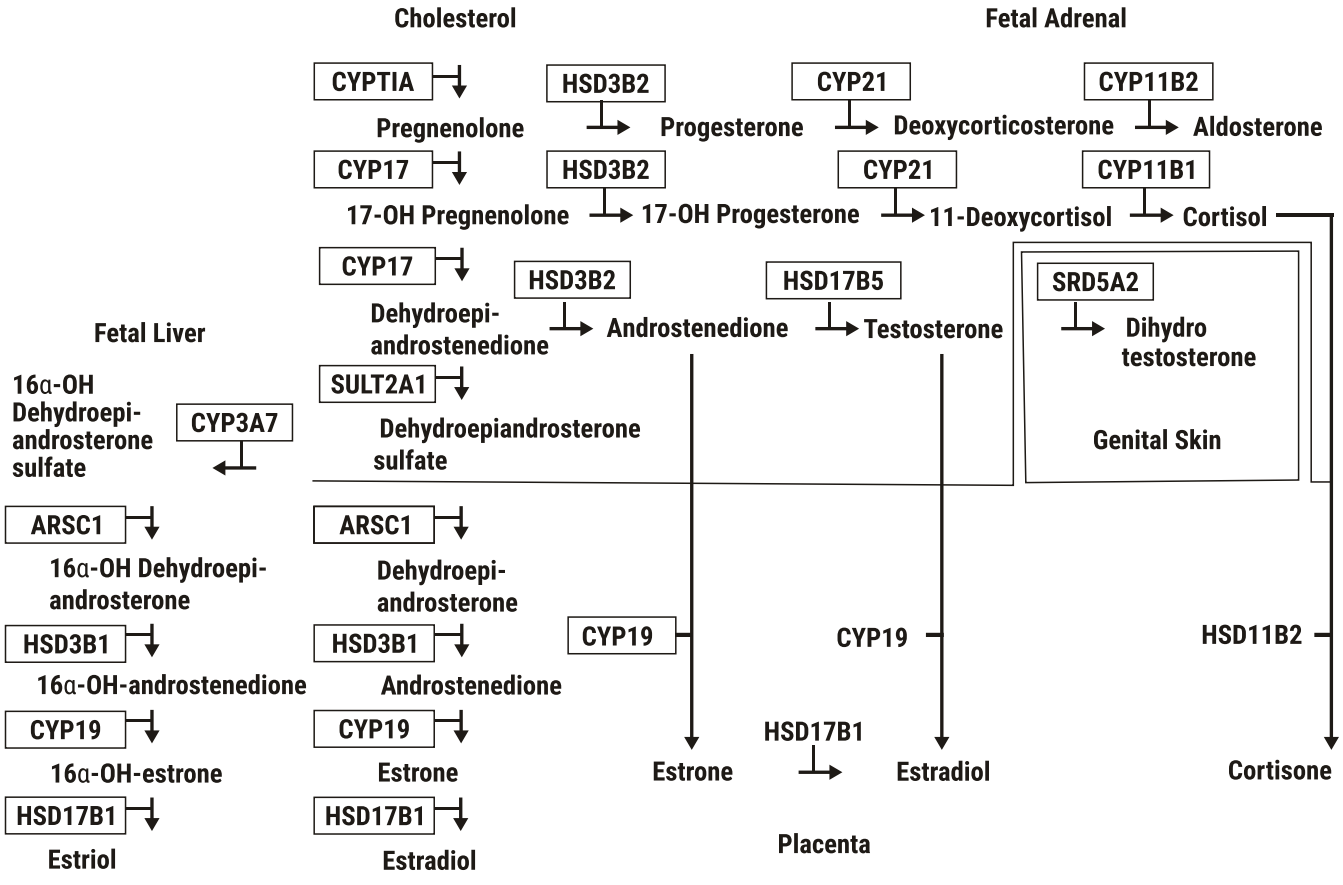
Common Mechanisms In 46 XY DSD

Important causes include androgen insensitivity syndrome, 5-alpha reductase deficiency, 17β-HSD deficiency, testosterone biosynthesis defects, LH receptor defects, gonadal dysgenesis and adrenal steroidogenesis defects.



46 XX DSD

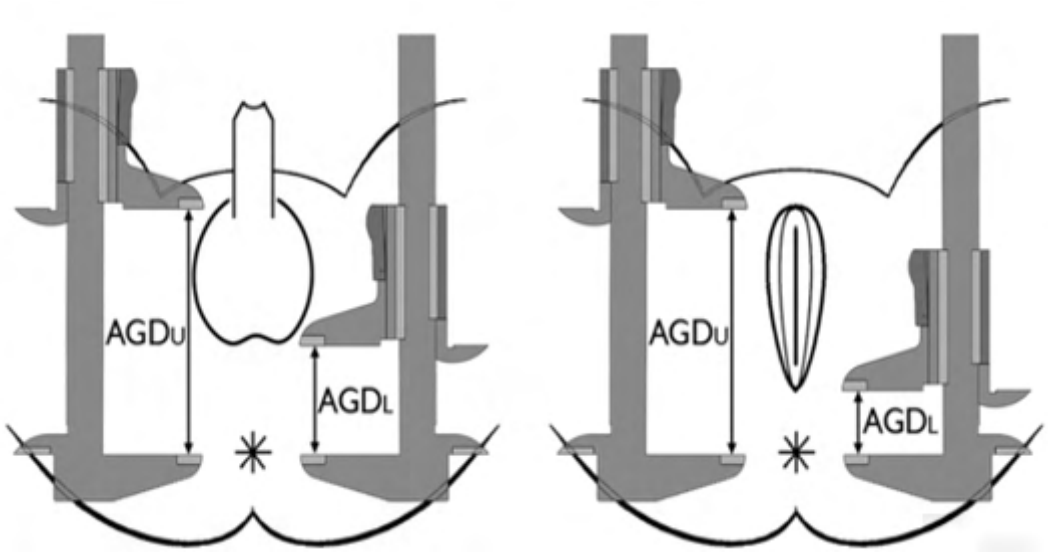
Females are generally evaluated by identifying the source of excess androgen. Causes include congenital adrenal hyperplasia (CAH), maternal androgen exposure, androgen- producing tumours, exogenous hormones and placental aromatase deficiency.


Cholesterol
Fetal Adrenal
CYPTIA
HSD3B2
CYP21
CYP11B2
Pregnenolone
Progesterone
Deoxycorticosterone
Aldosterone
CYP17
HSD3B2
CYP21
CYP11B1
17-OH Pregnenolone
17-OH Progesterone
11-Deoxycortisol
Cortisol
CYP17
HSD3B2
HSD17B5
SRD5A2
Dehydroepi-
androstenedione
Androstenedione
Testosterone
Dihydro
testosterone
Fetal Liver
Genital Skin
16α-OH
Dehydroepi-
androsterone
sulfate
CYP3A7
SULT2A1
Dehydroepiandrosterone
sulfate
ARSC1
ARSC1
16α-OH Dehydroepi-
androsterone
Dehydroepi-
androsterone
HSD3B1
HSD3B1
CYP19
CYP19
HSD11B2
16α-OH-androstenedione
Androstenedione
CYP19
CYP19
HSD17B1
16α-OH-estrone
Estrone
Estrone
HSD17B1
Estradiol
Cortisone
HSD17B1
Estradiol
Estriol
Placenta

History And Examination

History should include consanguinity, family history of ambiguous genitalia or infertility, infant deaths suggesting salt-wasting CAH, maternal virilisation and drug exposure during pregnancy. Examination includes assessment for congenital anomalies, careful palpation of gonads, stretched penile length, localisation of the urethral meatus and assessment using Prader staging (not preferred anymore), External Masculinization Score (EMS) or External Genital Score (EGS).

Neutral terminology such as genital tubercle and labioscrotal folds should be used. Referral is recommended for infants with abnormal EGS



EGS	Labioscrotal Fusion	Genital Tubercle Length (mm)	Urethral Meatus	Right Gonad	Left Gonad
3	Fused	>31	Top of the GT		
2.5		26–30	Coronal Glandular		
2			Along the GT		
1.5	Posterior fusion	21–25	At the GT base	Labioscrotal	Labioscrotal
1		10–20	Labioscrotal	Inguino-Labioscrotal	Inguino-Labioscrotal
0.5				Inguinal	Inguinal
0	Unfused	< 10	Perineal	Impalpable	Impalpable

Abbreviations: EGS, External Genitalia Score; GT, genital tubercle

External Genital score describes Phenotypical features at 5 anatomical landmarks of the genitalia: Degree of labioscrotal fusion, length of genital tubercle, position of the urethral meatus, and locations of right and left gonads. Final score is the sum of points allocated to features 1 to 5.

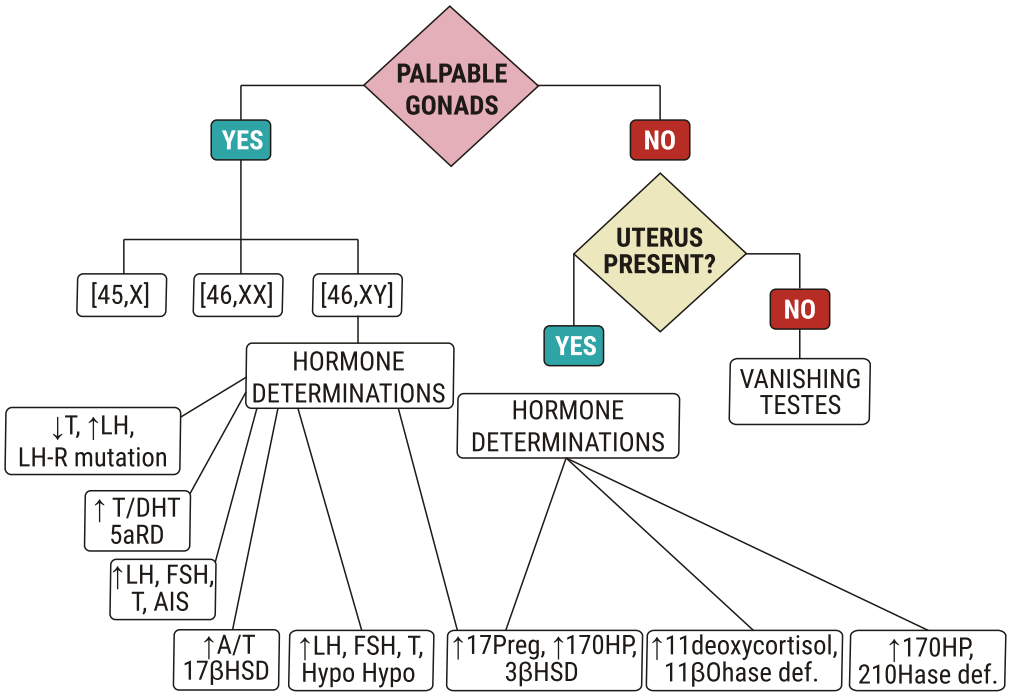
Investigations

Initial investigations include pelvic ultrasound to identify internal genitalia, early Y chromosome detection by FISH/PCR followed by karyotyping, plasma glucose, serum electrolytes, 17-hydroxyprogesterone, FSH, LH, AMH and androgen levels. Dynamic tests include hCG stimulation and ACTH stimulation tests.

Interpretation Of 46 XY DSD

AMH, testicular androgen production and adrenal precursor levels help narrow the diagnosis. Low AMH with low testosterone suggests gonadal dysgenesis. Normal AMH with low testosterone suggests testosterone biosynthesis defects or LH receptor defects. High AMH with androgen resistance suggests androgen insensitivity syndrome or 5-alpha reductase deficiency.

AMH	Testicular Androgen	Adrenal Precursors	First Think Of
Low	Low	Normal	Gonadal dysgenesis
Normal	Low	Normal	Testosterone biosynthesis defects LH Receptor defect
Normal/ above range	Low	Deranged	Adrenal steroidogenesis defects
High	High	Normal	AIS, 5ARDase, PMDs



Conclusion

A structured approach integrating history, examination, hormonal evaluation, imaging and genetic testing enables accurate diagnosis and timely management of DSD. Multidisciplinary care involving endocrinologists, surgeons, geneticists, psychologists and experienced counsellors is essential for optimal outcomes.

BREAKING NEWS IN PAEDIATRICS

In this section, we share the latest practice-changing updates or recent innovations in the treatment from the field of paediatrics.

Emerging Biological Subtypes Of Autism Spectrum Disorder: Insights From Cross-Species Functional Neuroimaging – Implications For Paediatric Practice

[A concise review based on Pagani et al., Nature Neuroscience (published online 15 May 2026)]

Autism Subtypes: What This Means for Your Practice

Evidence-based summary of Pagani et al. Nature Neuroscience 2026 - foundational research, not yet clinical diagnostic tool

Key Biological Insights

- Two reproducible subtypes identified in~25% of idiopatic autism via fMRI connectivity (hypoconnectivity linked to synaptic dysfunction; hyperconnectivity to immune/transcriptional pathways)
- Supported by 20 mouse models + large human ABIDE/CMi cohort (n=940 ASD)
- Cross-species validation strengthens findings. Distinct network architectures and behavioural profiles (hyperconnectivity subtype shows moderately higher ADOS social affect severity)

Current Clinical Implications

- Reinforces profound biological heterogeneity of autism - one size does not fit all.
- Standard care remains essential: early intensive behavioural intervention, speech/OT, medical comorbidity screening (GI, sleep, epilepsy)
- Family-centered, multidisciplinary approach critical regardless to subtype. Supports holistic view including emotional intelligence and mental wellness support.

Emerging & Future Directions

- Potential for precision stratification in research trials als/ta (targeted therapies e.g. synaptic modulators or immunomodulation)
- May explain variable treatment response.
- Need for non-invasive biomarkers to replace fMRI subtyping. Encourages participation in research registries.

Take-Home Message: Autism is biologically diverse. Early intervention and supportive care save lives and potential -continue evidence-based practice while advocating for research into mechanisms and targeted treatments.

For educational purposes; consult full paper for details.

Autism spectrum disorder (ASD, hereafter autism) has long been recognised as highly heterogeneous in clinical presentation, genetics and neurobiology. A groundbreaking study published in *Nature Neuroscience* provides the first direct empirical evidence that this heterogeneity reflects biologically dissociable subtypes, identified through innovative cross-species functional MRI (fMRI) connectivity analyses.

This work bridges rodent models and large human cohorts, revealing two dominant, reproducible brain connectivity subtypes: one characterised by widespread **hypoconnectivity** (linked to synaptic dysfunction) and another by **hyperconnectivity** (linked to immune and transcriptional alterations). These findings offer a new framework for understanding autism's biological diversity and have important implications for how we conceptualise, counsel families about and ultimately approach care for children on the spectrum.

Background: The Challenge Of Heterogeneity

Autism affects approximately 1 in 36–100 children globally (with rising recognition in India). Core features—social communication difficulties and restricted/repetitive behaviours—vary enormously in severity, language, cognition and co-occurring conditions (ADHD, anxiety, GI issues, epilepsy, sleep disturbances). Genetic studies show hundreds of risk variants, rare high-penetrance mutations (modellable in mice) and common polygenic contributions. Environmental factors, including prenatal immune activation, also play roles.

Until now, direct links between specific biological mechanisms and observable brain phenotypes (such as fMRI connectivity patterns) were lacking. Most subtyping efforts relied on clinical or statistical clustering without strong neurobiological validation. This new research changes that.

The Study: Cross-Species Approach

Researchers aggregated resting-state fMRI data from **20 distinct autism-relevant mouse models** (genetic mutations such as *Shank3*, *Fmr1*, *Cntnap2*, *Chd8*, *Tsc2*, *Ube3a*, *16p11.2*, *22q11.2*; plus immune models like prenatal IL-6 exposure and *Trem2* deficiency; and the BTBR inbred line). Each model was compared to wild-type littermate controls using voxel-wise weighted degree centrality (global connectivity), a metric translatable across species.

Hierarchical clustering of connectivity difference maps (Cohen's *d*) revealed two dominant subtypes:

Hypoconnectivity subtype (11 models)

Hyperconnectivity subtype (9 models)

These were not arbitrary; they mapped to distinct molecular pathways via protein–protein interactome and gene ontology analyses.

The team then translated these rodent-derived “dysconnectivity priors” (specific brain regions showing consistent hypo- or hyper-connectivity) to a large human dataset: **n = 940 individuals with idiopathic autism** and **n = 1,036 neurotypical controls** (ages 5–30 years) aggregated from ABIDE I/II repositories plus a Child Mind Institute collection. Using regional masking in evolutionarily conserved areas, they identified analogous subtypes in the human data.

Key Findings

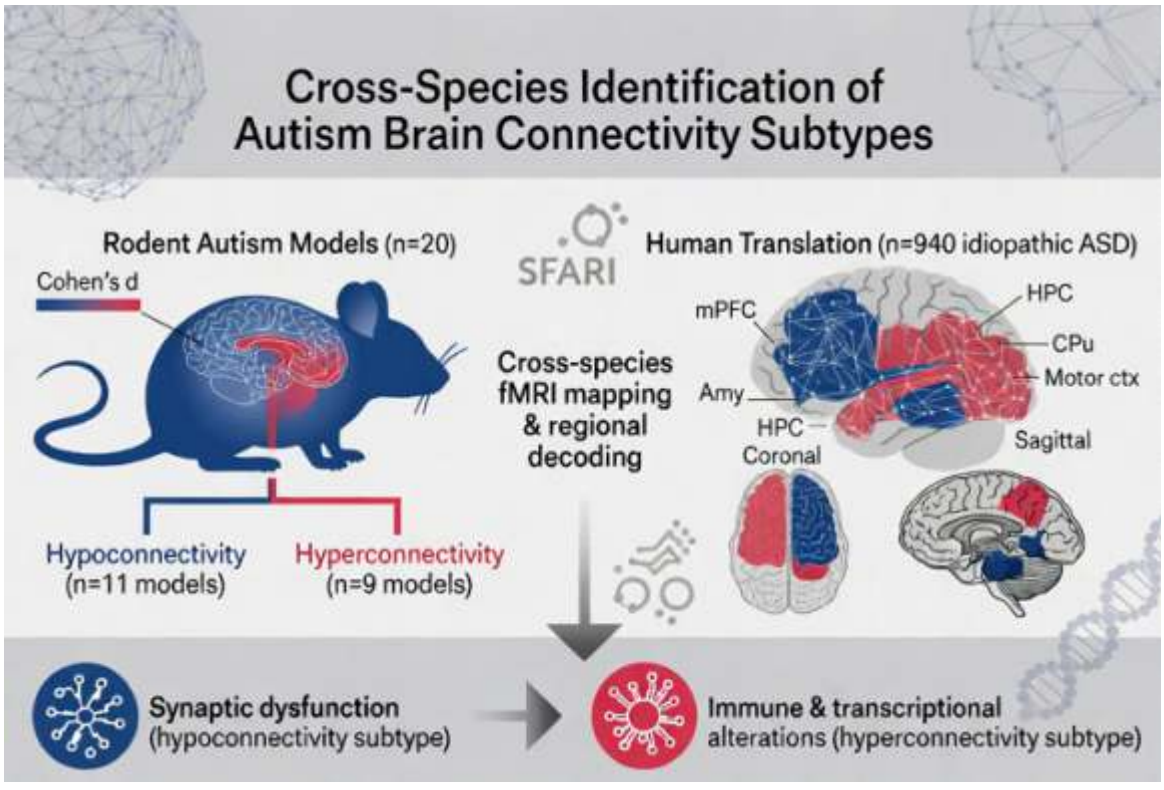
Rodent Phase: The hypoconnectivity subtype showed prominent reductions in connectivity involving salience, default mode and hippocampal networks, with vulnerability in regions such as the medial prefrontal cortex, striatum and hypothalamus. Gene enrichment strongly implicated **synaptic mechanisms** (protein–protein interactions at the synapse, chemical synaptic transmission, SynGO ontology, MAPK signalling, WNT signalling, membrane trafficking and protein translation).

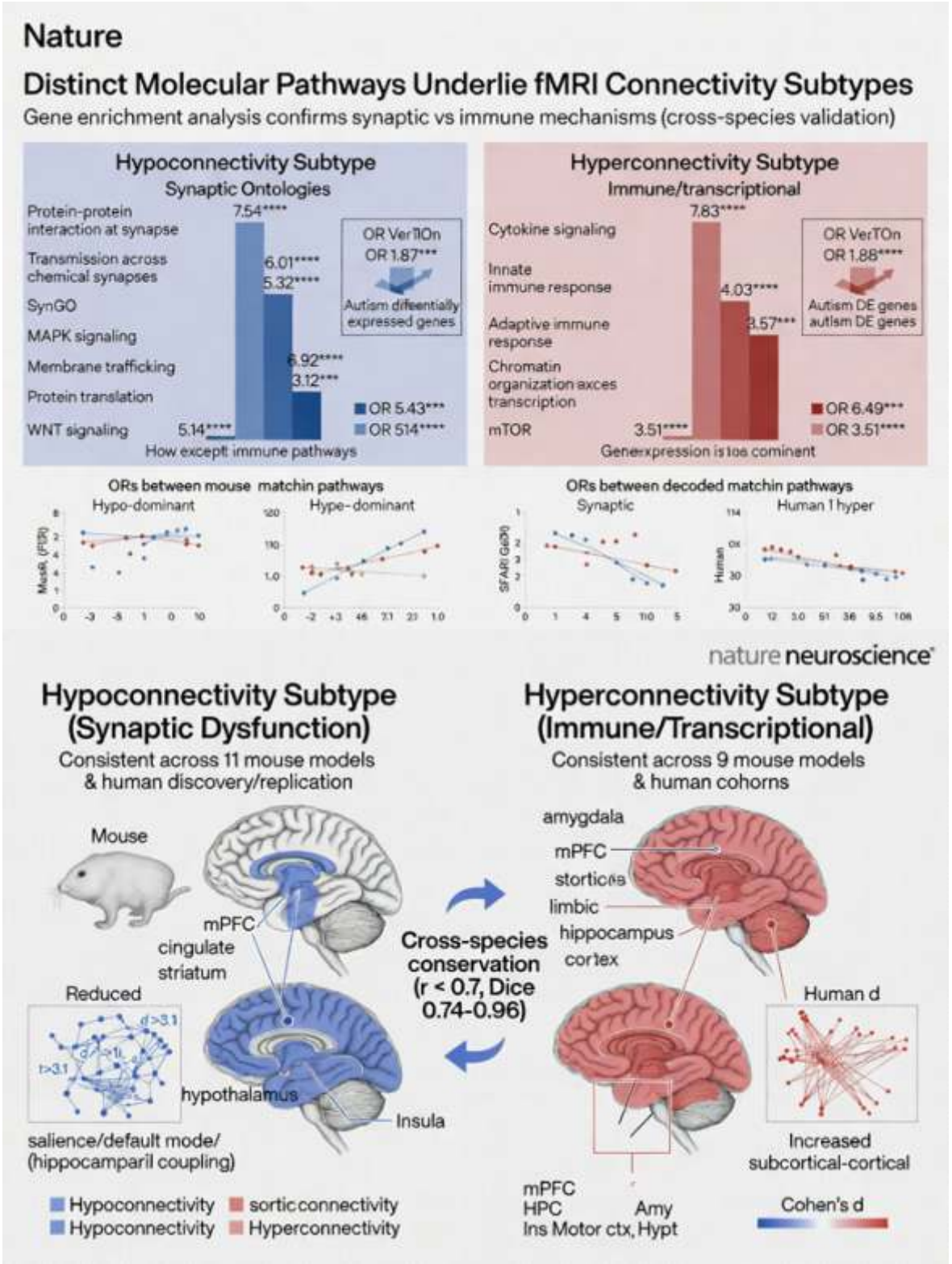
The hyperconnectivity subtype showed increased connectivity, particularly in limbic, salience and subcortical systems, with prominent involvement of amygdala and hippocampus. Enrichment pointed to **immune-related and transcriptional pathways** (cytokine signalling, innate and adaptive immune responses, chromatin organisation and gene expression transcription). mTOR signalling appeared in both but was more dominant in hyperconnectivity.

Human Translation: Analogous hypoconnectivity (~8%) and hyperconnectivity (~17%) subtypes were identified, together accounting for ~25% of the idiopathic autism cohort. Findings were highly replicable across discovery (78.5% of sample) and replication (21.5%) splits, with excellent spatial overlap (Dice coefficients 0.74–0.96).

Network analyses confirmed distinct architectures: hypoconnectivity featured more cortico-cortical reductions; hyperconnectivity showed increased subcortico-cortical coupling. The hyperconnectivity subtype was associated with moderately higher autism symptom severity on ADOS-2 calibrated severity scores (particularly the social affect domain).

Gene decoding of the human fMRI maps (using Allen Brain Atlas spatial expression data) recapitulated the rodent pathways—**synaptic enrichment in the hypoconnectivity subtype** and **immune enrichment in the hyperconnectivity subtype**—with significant overlap with genes differentially expressed in postmortem autism cortex.




Nature
Distinct Molecular Pathways Underlie fMRI Connectivity Subtypes
Gene enrichment analysis confirms synaptic vs immune mechanisms (cross-species validation)
Hypoconnectivity Subtype
Synaptic Ontologies
Protein-protein interaction at synapse
Transmission across chemical synapses
SynGO
MAPK signaling
Membrane trafficking
Protein translation
WNT signaling
7.54****
6.01****
5.32****
6.92****
3.12***
5.14****
OR VerTOn OR 1.87***
Autism differentially expressed genes
OR 5.43***
OR 514****
How excepti immune pathways
Hyperconnectivity Subtype
Immune/transcriptional
Cytokine signaling
Innate immune response
Adaptive immune response
Chromatin organization axces transcription
mTOR
7.83****
4.03****
3.57***
3.51****
OR VerTOn OR 1.88****
Autism DE genes autism DE genes
OR 6.49***
OR 3.51****
Gene expression is too cominent
ORs between mouse matchin pathways
Hypo-dominant
Hype-dominant
ORs between decoded matchin pathways
Synaptic
Human 1 hyper
nature neuroscience
Hypoconnectivity Subtype (Synaptic Dysfunction)
Consistent across 11 mouse models & human discovery/replication
Mouse
mPFC
cingulate
striatum
Reduced
hypothalamus
Insula
salience/default mode/ (hippocamparil coupling)
Cross-species conservation (r < 0.7, Dice 0.74-0.96)
Hyperconnectivity Subtype (Immune/Transcriptional)
Consistent across 9 mouse models & human cohorns
amygdala
mPFC
limbic
hippocampus
cortex
Human d
Increased subcortical-cortical
mPFC
HPC
Ins Motor ctx, Hypt
Amy
Cohen's d
Hypoconnectivity
Hypoconnectivity
Hyperconnectivity

Clinical Relevance For Paediatricians

This is **foundational mechanistic research**, not yet a clinical diagnostic or subtyping tool. fMRI is not part of routine autism evaluation, and only about one-quarter of idiopathic cases were classified here; the remainder likely lie on a continuum or represent additional subtypes. Nevertheless, the findings carry immediate and forward-looking value:

What Stays The Same (and is reinforced)

- Autism is biologically diverse. Variable presentation, treatment response and outcomes are not merely "spectrum" noise—they have biological roots.
- Early, intensive, family-centred intervention (behavioural, speech-language, occupational, educational) remains the cornerstone, regardless of subtype.
- Comprehensive medical evaluation for co-occurring conditions (GI, sleep, epilepsy, nutritional) and genetic testing (when indicated by dysmorphic features, regression, or family history) continue to be essential.
- Holistic support—including emotional regulation, social skills and family mental health—is critical.

What This Adds

- It provides the strongest evidence yet that synaptic and immune mechanisms can drive opposing large-scale brain connectivity patterns in autism.
- It extends the excitation–inhibition (E/I) imbalance hypothesis to the systems level and suggests why some children may respond differently to the same interventions.
- It validates cross-species modelling for autism and opens doors to mechanism-targeted research (e.g., synaptic stabilisers or immunomodulatory approaches in appropriate subgroups).

Key Takeaways

- Counsel families that autism's heterogeneity has biological meaning—this can reduce self-blame and stigma.
- Continue advocating for early identification and intervention; these remain the most evidence-based actions we have.
- Consider participation in research registries or biomarker studies when families are interested.
- Stay attuned to emerging literature on precision approaches; non-invasive proxies for these subtypes (blood, EEG, or other imaging) may become available in the coming years.

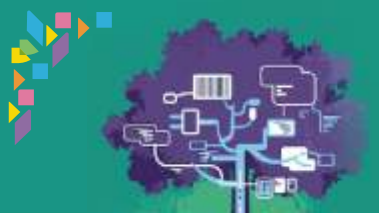
Limitations & Future Directions

The study used a regional, cross-species-guided approach rather than fully data-driven clustering in humans. Transcriptional signals prominent in rodents were not strongly recovered in human fMRI maps. Sex-balanced analyses and longitudinal trajectories are needed. Most importantly, these subtypes are observational; clinical translation will require validation against treatment response, outcomes and development of accessible biomarkers.

Recommended Reading: Pagani M, et al. Autism subtypes identified using cross-species functional connectivity analyses. *Nat Neurosci*. 2026 May 15. doi:10.1038/s41593-026-02287-z (Open access).

This summary is prepared for educational purposes for paediatric healthcare professionals. Always refer to the primary literature and current clinical guidelines for patient care decisions.

If you would like a printable PDF version, slides for departmental teaching, Marathi-language key messages for families, or further discussion on integrating these insights with emotional intelligence and child mental wellness programs, please let us know.



DECISION-MAKING TREE

Algorithm for the 'Evaluation of True Vertigo In Children'

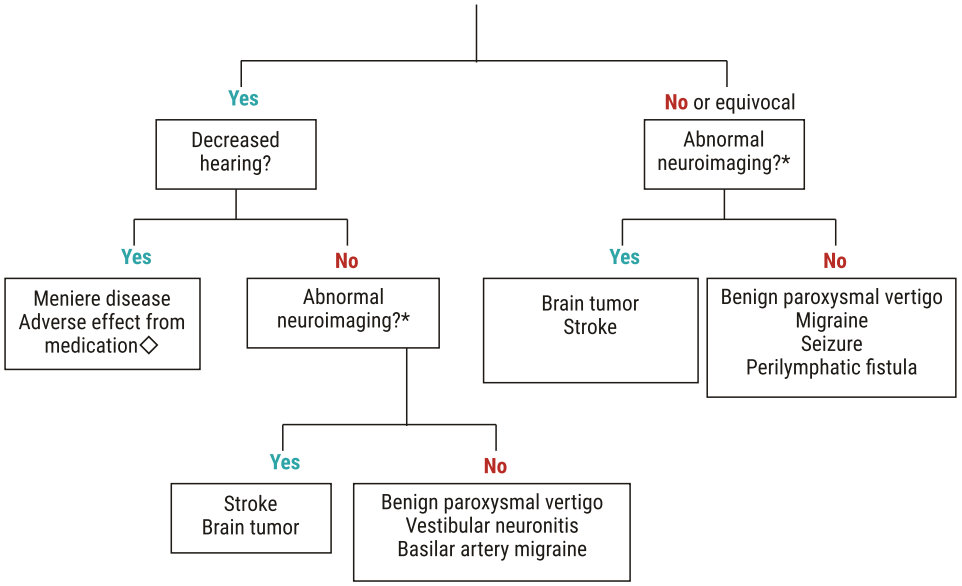
Approach to the Child with True Vertigo

Clinical findings of vertigo (two or more of the following)?
Physical sensation of spinning or moving
Nystagmus
Nausea and/or vomiting

- **Yes** → Prior head trauma?
  - **Yes** → Abnormal neuroimaging?
    - **Yes** → Temporal bone fracture
      Increased ICP
    - **No** → TemporaaPostconcussion syndrome
      Labyrinth concussion
      Posttraumatic migraine
      Postictal state after seizurel
  - **No** → Altered mental status?
    - **Yes** → Fever?
      - **Yes** → Meningitis¶
        Encephalitis¶
      - **No** → Abnormal neuroimaging?*
        - **Yes** → Brain tumor
          Stroke
        - **No** → Drug overdose (eg, ketamine, PCP, barbiturates, or ethanol)
          Migraine (confusional)
          Postictal state after seizure
    - **No** → Abnormal otoscopy?
      - **Yes** → **Abnormal canal**:
        Cerumen impaction
        Foreign body
        Herpes zoster oticus (Ramsay Hunt syndrome)

        Middle ear effusion:
        Serous or purulent labyrinthitis
        Distorted TM:

        Cholesteatoma
        Perilymphatic fistula
        (post-traumatic perforation or ossicle disruption; TM normal if spontaneous)
      - **No** → Associated with travel in a car, boat or plane?
        - **Yes** → Motion sickness
        - **No** → Abnormal vestibular testing (eg, positive head impulse test)? A
- **No** → Dizziness without vertigo
  (refer to UpToDate topics on syncope or ataxia in children)



TM: tympanic membrane; PCP: phencyclidine; ICP: intracranial pressure.

* Emergency imaging of choice for trauma patients is computed tomography of the head with temporal bone windows. Magnetic resonance imaging of the brain is preferred when brain tumor or stroke is suspected if it can be rapidly and safely performed.

Patients with signs of meningitis or encephalitis and focal neurologic deficits or concern for increased intracranial pressure also warrant neuroimaging.

Δ For descriptions of physical examination testing of vestibular function, including the head impulse test, refer to UpToDate topics on vertigo in children. Some patients may require specialized testing by an otologist.

◇ Medications that may cause hearing loss with vertigo include aminoglycosides, furosemide, minocycline, and salicylates.

Causes of dizziness in children and adolescents

Dizziness with vertigo*	Dizziness without vertigo (pseudovertigo)
Life-threatening	
CNS infection"	Arrhythmia
Head trauma	Heat stroke
Poisoning or adverse medication effect	Hypoglycemia
Stroke	Poisoning or adverse medication effect
Brain tumor	
Common	
Benign paroxysmal vertigo of childhood	Anaemia
Labyrinthitis (vestibular neuritis)	Anxiety
Migraine	Ataxia
Motion sickness	Depression
Otitis media complicated by labyrinthitis	Hyperventilation
	Orthostatic hypotension
	Pregnancy
	Presyncope
Other	
Benign paroxysmal positional vertigo	Somatic symptom disorder
Cholesteatoma	
Congenital defects	
Mastoiditis	
Meniere disease	
Middle ear trauma	
Multiple sclerosis	
Perilymph fistula	
Ramsay Hunt syndrome	
Seizure	

CNS: central nervous system.
* True vertigo refers to dizziness with a sense of spinning.
¶Meningitis, encephalitis, or intracranial abscess.
A Eg, Mondini dysplasia, Usher syndrome, Joubert syndrome, Schiebe deformity, enlarged vestibular aqueduct syndrome.

SUBSPECIALITY SPOTLIGHT

Here we focus on a short article on a paediatric subspeciality topic.

In this issue, we are publishing an interesting rare case managed by our NICU team.

From Encephalopathy To Exome:
Decoding A Lethal Metabolic Crisis In A Newborn

Dr Khushboo Kapasi
Head Of Department, NICU
MRR Children's Hospital

Abstract

Urea cycle disorders are rare but life-threatening causes of neonatal encephalopathy. Early diagnosis and prompt metabolic management are critical for survival. We describe a neonate with severe hyperammonemia whose diagnosis was confirmed through whole exome sequencing.

Case Presentation

A 36-week late preterm male neonate, born to second-degree consanguineous parents, presented in the early neonatal period with decreased activity, poor feeding, hypotonia and encephalopathy. Initial evaluation revealed severe hyperammonemia (ammonia 2005 µmol/L), strongly suggesting an inborn error of metabolism.

The baby required intensive care support including ventilation, ammonia scavenger therapy (sodium benzoate, L-arginine, carnitine) and early peritoneal dialysis. Ammonia levels showed a rapid decline with treatment, accompanied by significant neurological improvement. Tandem mass spectrometry demonstrated elevated citrulline levels, indicating a probable urea cycle disorder.

Neurosonography revealed diffuse cerebral edema, which resolved with treatment. The infant was gradually stabilised, extubated and transitioned to specialised metabolic nutrition, including UCD formula and protein-restricted feeds.

Discussion

Whole exome sequencing identified a homozygous likely pathogenic variant in the *ASL* gene (c.436C>T; p.Arg146Trp), confirming argininosuccinic aciduria, an autosomal recessive urea cycle disorder.

This case highlights the classical presentation of urea cycle disorders with hyperammonemic encephalopathy in the first few days of life. Consanguinity increased the index of suspicion for an inherited metabolic disorder. Early multimodal therapy—including dialysis—played a crucial role in preventing irreversible neurological damage.

Genomic confirmation enabled precise diagnosis, prognostication and appropriate genetic counselling for the family.

Key Learning Points

Neonatal encephalopathy with severe hyperammonemia should prompt urgent evaluation for urea cycle disorders.

Ammonia >1000 µmol/L is a metabolic emergency requiring rapid intervention.

Early initiation of ammonia scavengers and dialysis can dramatically improve outcomes.

Elevated citrulline levels help narrow the diagnosis among urea cycle defects.

Whole exome sequencing is essential for definitive diagnosis and genetic counselling.

Consanguinity is a key risk factor for autosomal recessive metabolic disorders.

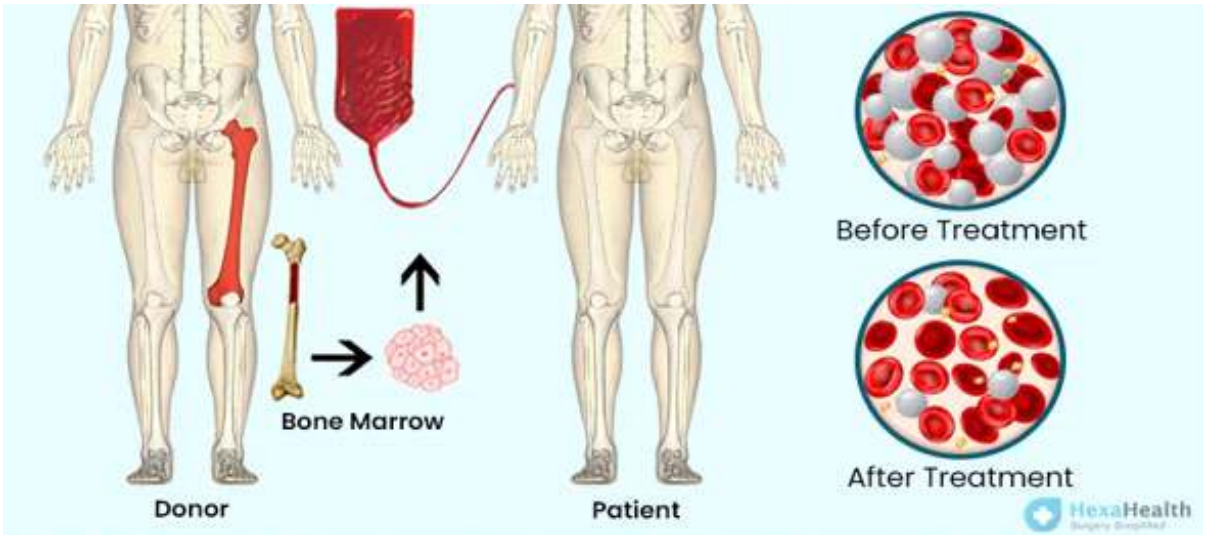
Conclusion

This case underscores the importance of early recognition of hyperammonemic encephalopathy and rapid intervention in neonatal urea cycle disorders. Integration of metabolic and genomic diagnostics enables precise diagnosis, guides therapy and facilitates long-term family counselling, ultimately improving survival and neurodevelopmental outcomes.

PAST CURIOUS: THE SAGAS OF HISTORY OF MODERN MEDICINE

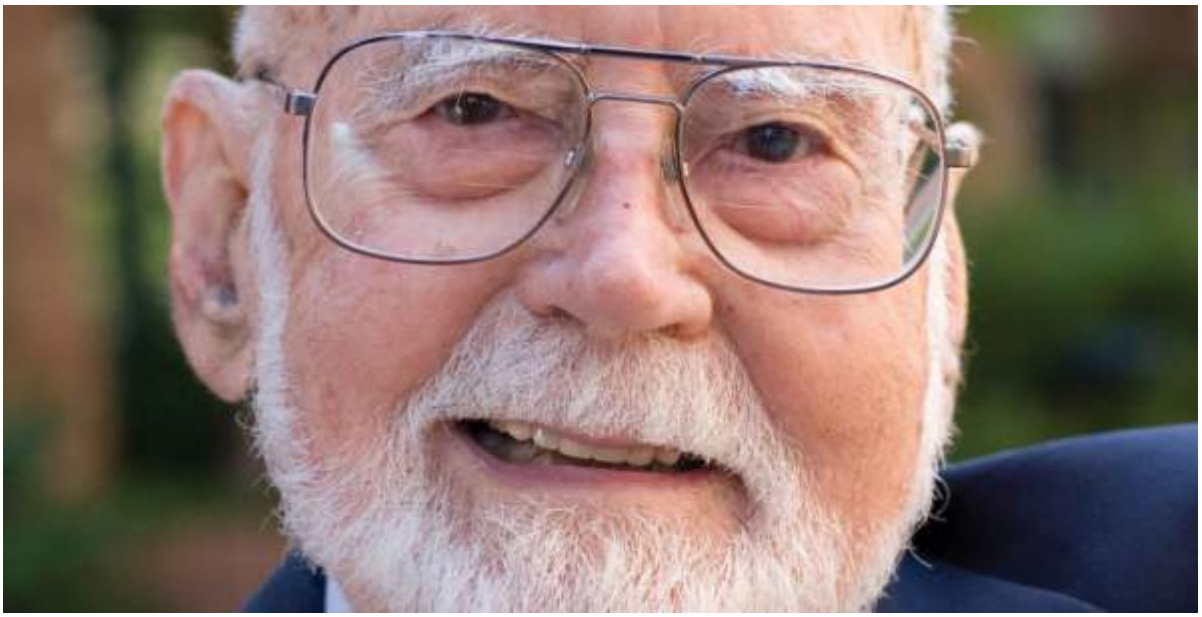
Here we share a short informative article on the study & evolution of medical treatments, practices & knowledge over time.

The Evolution Of Bone Marrow Transplant (BMT)



Pioneers Of Early Paediatric BMT

Dr E. Donnall Thomas – "Father of Bone Marrow Transplantation." He performed the first successful paediatric BMT in 1956 on a child with leukaemia using an identical twin donor and received the Nobel Prize in 1990.



Dr E. Donnall Thomas receiving the Nobel Prize (1990) for his pioneering work in cell transplantation.

Dr Robert A. Good – Performed the landmark 1968 allogeneic BMT that cured an infant with severe combined immunodeficiency (SCID) using a matched sibling donor – the first successful curative allogeneic transplant in a child. His legacy is honoured at the University of Minnesota Center for Immunology.

Cord Blood Transplantation Pioneer

Dr Eliane Gluckman – Performed the world's first successful umbilical cord blood transplant in 1988 on a child with Fanconi anaemia in Paris. This opened a new donor source for children without matched siblings.

BMT in children evolved from 1940s–1950s animal radiation-protection studies into a curative therapy for leukaemia, immunodeficiencies and genetic blood disorders.

In **1956**, Dr **E. Donnall Thomas** performed the first successful BMT on a child with leukaemia using marrow from an identical twin, proving hematopoietic stem cells could rescue patients after myeloablative therapy. HLA compatibility, discovered by Jean Dausset (1958; Nobel 1980), proved essential for success.

The landmark paediatric milestone came in **1968** when Dr **Robert Good** at the University of Minnesota cured an infant with severe combined immunodeficiency (SCID) using HLA-matched sibling marrow—the first curative allogeneic BMT in a child.

Unrelated donor transplants succeeded in 1973. A major advance for children arrived in **1988** when Dr **Eliane Gluckman** performed the world's first umbilical cord blood transplant in Paris on a child with Fanconi anaemia, greatly expanding donor options.

Thomas shared the **1990 Nobel Prize**. Later developments—reduced-intensity conditioning, haploidentical transplants and gene-therapy combinations—improved safety and access. Today BMT cures thousands of children annually worldwide.

Further Insights: Latest Advances in Paediatric BMT & Gene-Edited Stem Cell Therapies (2024–2026)

Building on the historical arc (Thomas 1956 twin transplants → Good 1968 allogeneic cure in SCID → Gluckman 1988 cord blood → Nobel 1990), the field has entered a new evolutionary phase. Allogeneic hematopoietic stem cell transplantation (HSCT) continues to improve, but **autologous CRISPR and lentiviral gene-edited stem cell therapies** now offer transformative, donor-independent options for children with genetic hemoglobinopathies.

Gene-Edited Autologous Therapies — The 2026 Paediatric Leap

Casgevy (exagamglogene autotemcel/exa-cel), the first CRISPR/Cas9-edited therapy, received FDA approval in late 2023/early 2024 for patients ≥12 years with severe sickle cell disease (SCD) or transfusion-dependent β-thalassemia (TDT).

In **July 2026**, the FDA expanded approval to children **as young as 2 years old** — a landmark for paediatrics.

Phase 3 data (CLIMB-151 and related trials) in children aged 5–11 with severe SCD showed **all evaluable patients remained free of vaso-occlusive crises (VOCs)** for up to two years post-infusion, with a safety profile consistent with earlier studies. Similar durable transfusion independence is seen in young TDT patients. These one-time therapies edit the patient's own hematopoietic stem cells to boost foetal haemoglobin or correct β-globin, eliminating the need for a donor and the risk of graft-versus-host disease (GVHD).

Other approved options include **Lyfgenia (lovotibeglogene autotemcel)** for SCD and **Zynteglo (betibeglogene autotemcel)** for TDT.

Key paediatric trial leadership includes

Dr Haydar Frangoul and international collaborators. These therapies use BMT-like infrastructure (stem cell mobilisation, conditioning, infusion) but represent a paradigm shift toward personalised, autologous cures.

Advantages Over Traditional Allogeneic BMT: No donor search, no GVHD, potentially lower long-term immunosuppression.

Current limitations: High cost, complex manufacturing/logistics, requirement for myeloablative conditioning (improving with reduced-toxicity regimens) and still-maturing very-long-term durability data (>5–10 years).

Advances In Allogeneic Paediatric HSCT

For children who still require donor HSCT (e.g., high-risk leukaemia, certain immunodeficiencies, or when gene therapy is unavailable), progress continues:

- **Haploidentical transplants with post-transplant cyclophosphamide (PTCy)** have become a reliable standard for patients lacking matched donors. Recent data support extending PTCy-based platforms to mismatched unrelated donors (MMUD) with encouraging GVHD control and survival.
- **GVHD prophylaxis innovations**: Abatacept-inclusive regimens in children have reduced acute and chronic GVHD incidence/severity while lowering neurotoxicity (PRES). Combinations with PTCy are under active study.
- Emerging supportive strategies include microbiome modulation (faecal microbiota therapy trials such as MaaT013) and targeted agents (belumosudil, axatilimab) highlighted at ASH 2025 for GVHD prevention and treatment.
- CAR-T cell therapy is increasingly integrated — used as a bridge to HSCT in relapsed/refractory paediatric ALL or for post-transplant relapse management.

Future Directions & Relevance

The trajectory points toward **hybrid and personalised approaches**: choosing allogeneic HSCT, gene therapy, or combinations based on donor availability, disease biology, patient age and comorbidities. Reduced-toxicity conditioning, in vivo gene editing and improved vectors are active research areas. Long-term survivorship programs emphasising growth, fertility, neurocognitive outcomes and psychosocial support (including emotional intelligence frameworks) are becoming standard.

For regions with high burdens of thalassemia and SCD (including India), these advances offer immense hope, though equitable access, cost reduction and local infrastructure development remain critical next steps.

The evolution from 1950s twin transplants to 2026 CRISPR therapies in toddlers illustrates one of the most rapid and impactful progressions in modern medicine. Ongoing trials will further refine who benefits most from each modality.

Latest Advances: CRISPR Gene-Edited Therapies (2023–2026)

Casgevy (exa-cel) — The first CRISPR/Cas9 gene-edited autologous stem cell therapy. In July 2026, the FDA expanded approval to children as young as 2 years old with sickle cell disease or transfusion-dependent β-thalassemia, based on strong Phase 3 data showing durable VOC-free survival and transfusion independence in young children.

PaediaBliss

The Joys, Laughter & Heartwarming Tales of Paediatric Practice

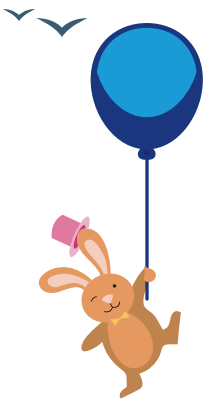
In this section, we share some delightful stories that capture the joy and humour of our daily interactions in paediatric practice with children & their families.

Chikitsak, Chikitsak: The Day My Patient Renamed Me

A 4-year-old entered my cabin chanting 'Chikitsak, Chikitsak'. Puzzled, I asked what he was chanting. His parents explained that the school community health worker had taught him this new Hindi word for a 'Doctor'. I was amazed and amused.

On the next visit, he announced 'Aushadh, Aushadh!' – he had learnt a new word for medicine! What began as a hilarious mix-up has now become our cherished ritual. Every follow-up, I eagerly ask what new word he has learned in school.

These moments remind us that in the world of paediatrics, the most delightful lessons often come from our little visitors.



Shared & Compiled by

Dr Sandeep M. Kelkar

Director-Department Of Paediatrics,

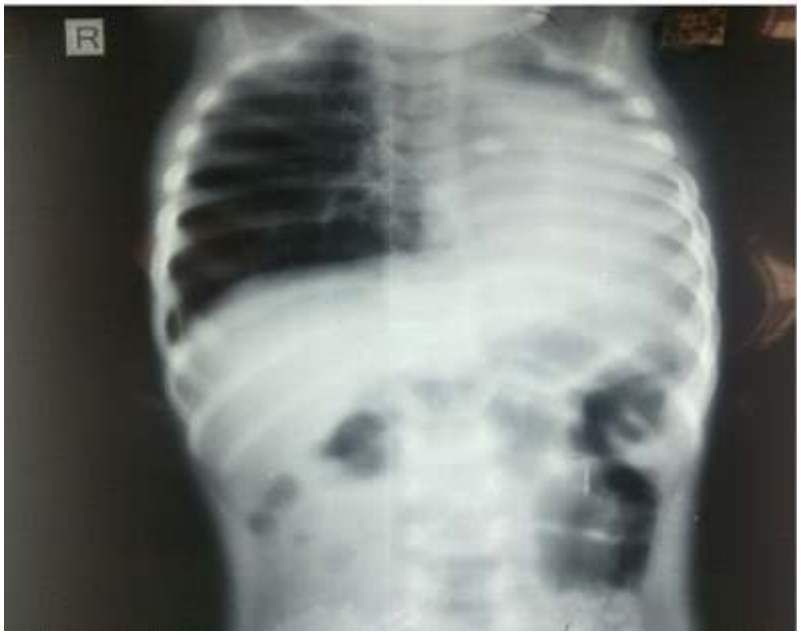
MRR Children's Hospital

CASE CHALLENGE

An 18-month-old boy presented with cough and tachypnea for 5 days, unresponsive to OTC remedies. No witnessed choking. History of pica.

Exam: Tachypnea, nasal flaring, intercostal/subcostal retractions, diminished air entry and rales on left side.

Labs: Hb 7.1 g/dL, MCV 52.7 fL, microcytic hypochromic anaemia; WBC 42 430/mm³ (82% polymorphs); platelets 834 000/mm³.



What's your spot diagnosis?

(For Answer: Please refer to the last page)

Compiled by

Dr Sandeep M. Kelkar,

Director-Department Of Paediatrics,

MRR Children's Hospital

Sampoorna Swasthya Sanwardhan
(संपूर्ण स्वास्थ्य संवर्धन)

In this section, we share a short article on child psychology, mental health & wellness, neuroscience, emotional intelligence & effective parenting for the holistic health of children

Dr Sandeep M. Kelkar
Director- Department Of Paediatrics
MRR Children's Hospital

Screen Time Use In Children And Adolescents
Evidence-Based Protocols For Paediatricians To Guide Parents

This resource equips paediatricians with concise, evidence-informed content and practical protocols to counsel families on screen time, promoting digital wellness as part of holistic child development. It aligns with IAP guidelines while incorporating recent meta-analyses and AAP 2026 updates emphasising quality, context and family-centred approaches over rigid limits alone.

1. Introduction And Rationale

Digital devices are ubiquitous in Indian households, with increasing screen exposure among children from infancy. The Indian Academy of Pediatrics (IAP) responded with dedicated parent guidelines in 2021, recognising screens as a 'double-edged sword' — beneficial when used appropriately, harmful when excessive or inappropriate. Paediatricians are uniquely positioned to integrate screen time assessment into well-child visits, similar to nutrition, sleep and immunisation counselling.

Recent evidence (2023–2026) strengthens the case: excessive screen time is associated with obesity, sleep disruption, language/cognitive delays in young children, increased anxiety/depression risk, externalising behaviours and academic decline. Bidirectional relationships exist — mental health challenges can drive more screen use for coping. Parental screen use in the child's presence independently predicts poorer child outcomes. Conversely, high-quality, co-viewed, time-balanced media can support learning, creativity and social connection, especially for children with special needs.

This article provides paediatricians a ready-to-use framework: scientific summary, IAP-aligned age recommendations, visual protocols for digital hygiene, counselling steps (adaptable 5Cs/5As), red-flag identification and family media planning tools.

Scientific Evidence: Harms And Benefits
Physical Health Effects

• Obesity & Sedentary Behaviour: Meta-analyses consistently link >2 hours/day recreational screen time with higher BMI, reduced physical activity and unhealthy snacking. Displacement of active play is a key mechanism.

Sleep Disruption: Blue light from screens suppresses melatonin; evening use delays sleep onset, shortens duration and fragments sleep. Adolescents need 8–9 hours; screens often crowd this out. Strong evidence from systematic reviews.

Ocular & Musculoskeletal: Computer Vision Syndrome (CVS) — eye strain, dryness, headaches — is common. Poor posture leads to neck/back pain ('text neck'). The 20-20-20 rule and ergonomic setups are evidence-supported preventive measures.

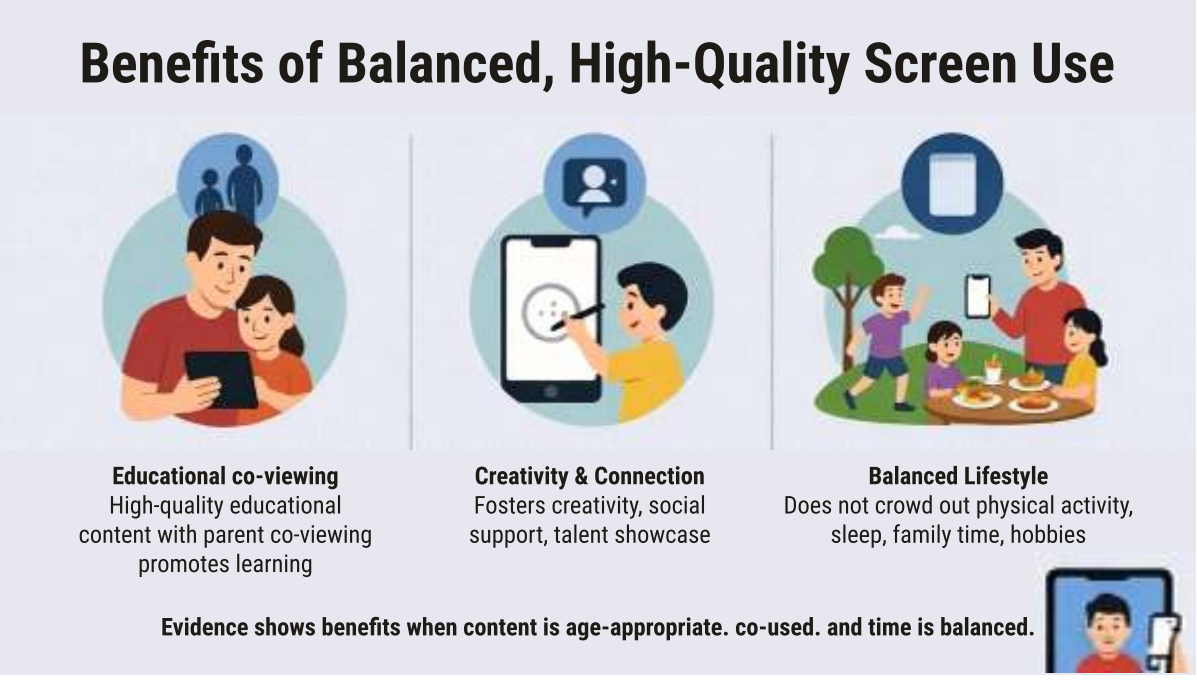
Cognitive, Language & Psychosocial Development (Especially <6 years)

Systematic reviews and meta-analyses (JAMA Pediatrics 2024) show program viewing and background television are associated with poorer cognitive outcomes; age-inappropriate content and caregiver screen use during routines are linked to poorer psychosocial outcomes. Solo viewing reduces parent-child interaction critical for brain development. Co-use (joint engagement) shows positive associations with cognition and bonding.

Mental Health & Behavioural Outcomes

2025–2026 meta-analyses and large cohort studies demonstrate significant associations between excessive screen time (>3–4 hrs/day recreational) and higher risks of anxiety (aOR ~1.45), depression (~1.61), conduct problems, ADHD symptoms and emotion dysregulation. Physical activity and sleep quality are major parallel mediators (explaining 30–40% of effect). Bidirectional vicious cycles are evident: emotional problems more screens for escape worsening problems. Social media adds unique risks: cyberbullying, social comparison/FOMO, sexting and predatory exposure.

Benefits Of Balanced, High-Quality Use



Evidence-supported benefits emerge when screens are high-quality, co-used by caregivers, age appropriate and do not displace essential activities.

- Educational apps and co-viewed programs (e.g., Sesame Workshop style) can reinforce learning and language when actively discussed.
- Video calls maintain family bonds (grandparents, relatives) — explicitly permitted even for <2 years.
- Customised programs benefit children with autism (social skills) and learning disabilities (study tools).
- Creative platforms allow talent expression under guidance; social support for chronic illness families.

3. Age-Specific Recommendations

The IAP guidelines remain foundational for Indian practice. AAP 2026 shifts emphasis toward the '5 Cs' framework (Child characteristics, Content quality, Context/co-use & timing, Calm—not for soothing, Crowding out other activities, ongoing Communication) while retaining core time-and-balance principles. Paediatricians should individualise: stricter for high-risk children (obesity, ADHD, sleep issues, anxiety).

Age Group	IAP Recommendation	Key Evidence & Notes
<2 years	No screens except occasional video calls with relatives. Social interaction is essential for brain development.	Language delay, hyperactivity and poor social skills risk. Background TV is harmful even if the child is 'not watching'.
2–5 years	1 hour/day of high-quality, supervised, interactive/educational content. Less is better. Co-viewing is preferred.	Meta-analyses: program viewing + background TV linked to poorer cognition /psychosocial outcomes. Co-use is protective.
6–12 years (School-going age)	Balance screen time so it does NOT displace: 1 hour of physical activity, adequate sleep (9–11 hours), schoolwork, meals, hobbies, family time. Monitor content & duration.	Excess linked to obesity, sleep problems, academic decline and externalising behaviours. Parental modelling is critical.
13–18 years (Adolescents)	Prioritise balance with sleep (uninterrupted 8–9 hours), physical activity, studies, real-world social interaction. Night-time curfews on devices. Parental monitoring + open communication.	Highest risk for social media anxiety, cyberbullying, sleep displacement, mental health issues. 4+ hours of recreational screen time is associated with worse outcomes.

Table 1: Age-specific screen time recommendations (Adapted From IAP 2021 Guidelines for Parents).

Social Media & Online Gaming — Minimum Permissible Ages (IAP)

Children and young adolescents lack full capacity to weigh risks/benefits or manage digital footprints. IAP recommends avoiding social media platforms before the platform's stated minimum age, with parental permission and monitoring where applicable.

Platform	Minimum Age (IAP)	Notes for Paediatricians
Facebook, Instagram, Twitter/X, Snapchat, Google+	13 years	Many users are underage. If access is permitted, ensure appropriate privacy settings and parental supervision.
WhatsApp	16 years	Commonly used before the recommended age. Group chats may expose children to inappropriate content and cyberbullying.
YouTube	18 years (13–18 years with parental permission)	Use supervised mode. Avoid unrestricted autoplay and access to comments for younger users.
PUBG/Similar Battle Royale	18 years (13–18 years with restricted play time and parental permission)	High potential for addiction, exposure to violence and sleep disruption. Screen time should be monitored strictly.
Clash of Clans/ Similar games	13 years	In-app purchases, social features; monitor spending and interactions.

Table 2: Permissible ages for social media and gaming platforms (IAP Guidelines). Always verify current platform policies.

Practical Protocols For Paediatricians

Routine Screening Protocol (Integrate Into Every Well-Child Visit)

Ask (2–3 minutes):

1. 'How many hours per day does your child spend on screens (TV, mobile, tablet, computer, gaming) for entertainment or non-school use?' (Differentiate schoolwork vs recreational).

2. 'What devices and apps/games are used? Is there co-viewing or supervision? Any rules at home?'

3. 'Does screen time interfere with sleep, outdoor play, family meals, homework or hobbies?'

4. 'Any concerns about behaviour, attention, mood, bullying online or sleep problems?'

5. 'How much screen time do parents/caregivers use in front of the child?' (Parental modelling is a strong predictor).

Counselling Framework (5 Cs + Brief Motivational Approach)

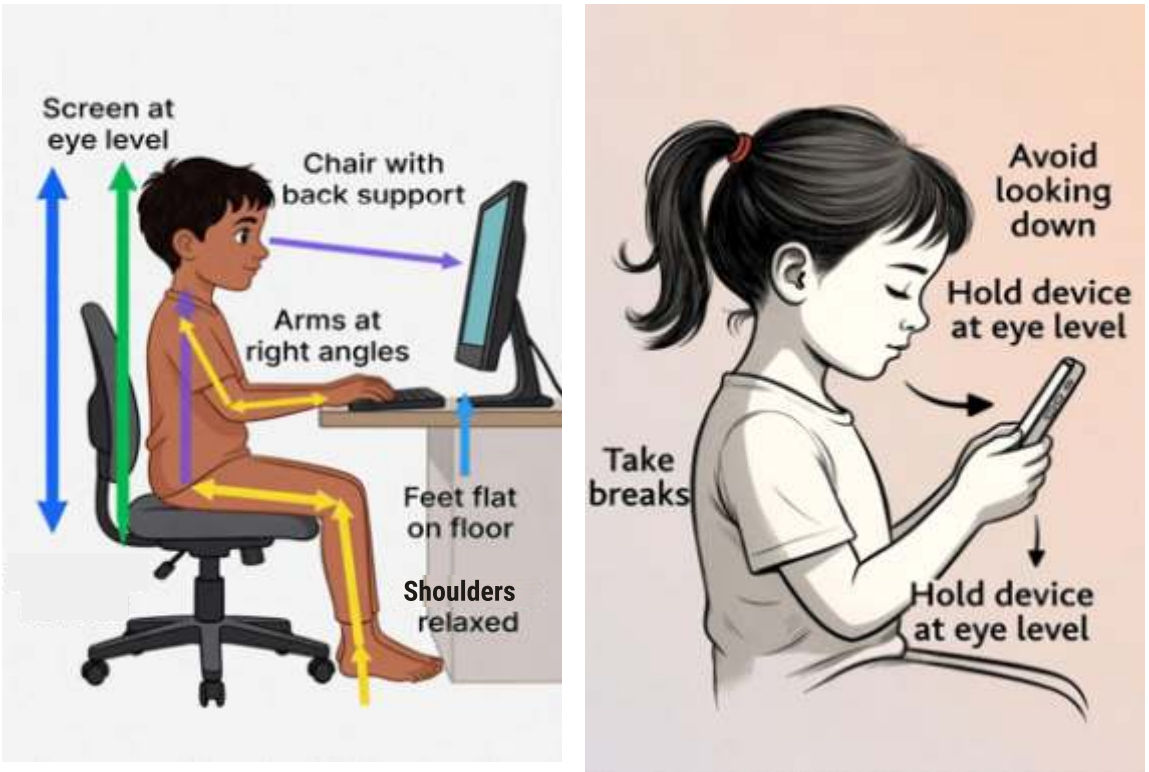
AAP 2026 emphasises conversation over prescription. Use a collaborative, non-judgmental style. For families exceeding limits or showing issues, apply brief 5A's: Ask (above), Advise (share IAP recs + personalised risk), Assess (readiness to change, barriers), Assist (co-create Family Media Plan), Arrange (follow-up in 1–3 months or referral)

Family Media Plan — Simple Template to Share with Parents

- Screen-free zones: Bedrooms (no devices in bedroom after 8–9 pm), dining table/mealtimes, during homework.
- Screen-free times: 1 hour before bedtime (blue light + wind-down); device curfew for adolescents.
- Daily balance rule: Screen time only after physical activity, homework and family time are done.
- Content rules: Only age-appropriate, non-violent apps/games; parental controls + safe search on; review history together weekly.
- Co-use for young children: Watch/play together and discuss content (teachable moments).
- Model healthy use: Parents put phones away during family time; verbalise 'I need a break from screens too'.
- Digital fasting: One screen-free evening/week or 'unplugged' family activity (park, board games, cooking).
- Review & revise: Revisit plan every 3–6 months as child grows or issues arise.

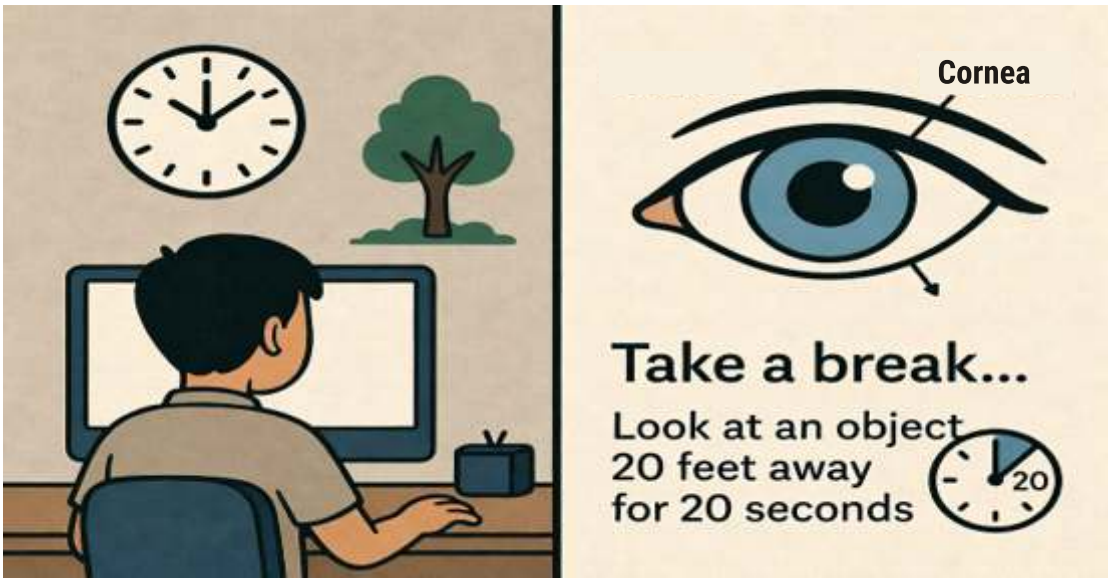
Practical Family Media Plan Template — print and give to parents (adapt from AAP Family Media Plan tool).

Digital Hygiene Protocol — Posture, Eye Health and Sleep Protection



Left: Correct ergonomic posture at desktop/laptop. Right: Proper mobile phone use to prevent 'text neck' and eye strain.

20-20-20 Rule (Evidence-Based For Reducing Computer Vision Syndrome)



Every 20 minutes of screen time, look at an object 20 feet away for 20 seconds. Also: blink consciously, adjust lighting/brightness, use artificial tears if dry eyes.

Additional rules: No screens in bedroom; charge devices outside bedroom overnight; night mode/blue-light filter after sunset; consistent bedtime routine without screens.

Red Flags for Problematic Media Use/Addiction & Referral Protocol

Internet Gaming Disorder is recognised in ICD-11. Media addiction shares features with other behavioural addictions. Screen for these in adolescents and high users:

- Preoccupation/craving: Constant thinking about or planning next screen session.
- Loss of control: Unable to limit time despite repeated attempts or promises.
- Withdrawal: Irritability, anxiety or low mood when devices are restricted.
- Tolerance: Needing more time or intense content to feel satisfied.
- Interference: Declining school performance, loss of interest in hobbies/sports/friends, sleep deprivation, family conflict.
- Continues despite harm: Persists even when it worsens anxiety, aggression or physical symptoms.
- Deception/hiding use or sneaking devices.
- Cyberbullying involvement (victim or perpetrator) or exposure to inappropriate/sexual content.

Action: If 3–4 red flags or significant impairment, refer promptly to a child & adolescent psychiatrist/psychologist or mental health professional experienced in digital addiction. For milder cases or prevention: motivational counselling, family media plan and follow-up. In India, also consider IAP-trained PEICH (Promoting Emotional Intelligence in Children), paediatricians or Equipoise-style programs for holistic support. Report cybercrimes to cybercrime.gov.in or Childline 1098.

5. Parental Role Modelling & Family Context

Parents who model balanced, mindful device use and prioritise face-to-face family time create the strongest foundation for children's healthy digital habits.

Recent meta-analysis (JAMA Pediatrics 2025) found parental technology use in a child's presence associated with poorer child cognition, lower prosocial behaviour, reduced attachment/security, increased internalising/externalising problems and higher child screen time. Children learn by watching. Paediatricians should gently highlight this: 'Your phone habits teach your child how to use (or not use) technology.' Encourage parents to verbalise boundaries ('I'm putting my phone away now so we can enjoy dinner together') and create device-free rituals.

6. Key Messages For Parents

1. Screens are tools, not babysitters or emotional regulators. Avoid using screens to calm or distract young children.
2. Balance is everything: Screen time must not displace sleep, physical activity (1 hr/day), family interaction, meals or studies.
3. Quality> Quantity: Choose educational, non-violent, age-appropriate content. Co-view and discuss with young children.
4. Protect sleep: No screens 1 hour before bed; devices out of bedroom at night.
5. Model the behaviour you want: Children copy parental screen habits more than rules.
6. Create family rules together and review regularly. Use Family Media Plan as a living document.
7. Watch for warning signs (red flags above) and seek help early — media addiction is treatable with professional support.
8. Digital citizenship matters: Teach online manners, privacy, reporting bullying and critical thinking about content.

7. Conclusion

Screen time management is now a core component of paediatric preventive care, akin to nutrition counselling or injury prevention. By combining the clear, practical IAP 2021 framework with emerging evidence on mechanisms (sleep, physical activity, parental modelling, bidirectional mental health pathways) and AAP 2026's emphasis on conversation and context, paediatricians can provide nuanced, family-centred guidance. Routine screening, visual protocols (posture, 20-20-20), Family Media Plans and timely referral for red flags will help children develop healthy digital habits while preserving time for play, relationships and real-world learning.

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HOSPITAL HIGHLIGHTS AND SUCCESS STORIES

Basic Life Support (BLS) Training For Associated School Staff

Over 25 school staff members participated in a Basic Life Support (BLS) training session, gaining essential knowledge in Cardiopulmonary Resuscitation (CPR), emergency response and managing medical emergencies. The session equipped them with the confidence to respond promptly and appropriately in emergency situations.

Self-Defence For Kids

In collaboration with our associated sports club, we organised a Karate Certification Programme for children. Approximately 120 children and their parents participated in the programme, celebrating the importance of fitness, discipline and self-defence.




Doctor's Day Celebration With Kids

This year, we celebrated Doctor's Day with our little champions. Over 170 children joined the celebration and expressed their appreciation by presenting handmade greeting cards to our doctors.



CASE CHALLENGE

SPOT DIAGNOSIS:

Left Main Bronchus Foreign-Body Aspiration

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