Norditropin®
Committed to GH Therapy

- Stimulates linear growth and increases growth rate\(^1\)
- Regulates metabolism\(^1\)
- Improves psycho-social well-being\(^*\)

Indications & Dosage\(^1\)

<table>
<thead>
<tr>
<th>Indication</th>
<th>Dose in mg</th>
<th>Dose in IU</th>
</tr>
</thead>
<tbody>
<tr>
<td>GHD</td>
<td>0.025 - 0.035 mg/kg/day</td>
<td>0.07 - 0.1 IU/kg/day</td>
</tr>
<tr>
<td>SGA</td>
<td>0.033 - 0.067 mg/kg/day</td>
<td>0.1 - 0.2 IU/kg/day</td>
</tr>
<tr>
<td>Turner Syndrome</td>
<td>0.045 - 0.067 mg/kg/day</td>
<td>0.14 - 0.2 IU/kg/day</td>
</tr>
<tr>
<td>Chronic Renal Disease</td>
<td>0.05 mg/kg/day</td>
<td>0.14 IU/kg/day</td>
</tr>
<tr>
<td>GHDA</td>
<td>0.1 - 0.3 mg/day</td>
<td>0.3 - 0.9 IU/day</td>
</tr>
</tbody>
</table>

Less pain with Norditropin® Nordilet® may aid better compliance\(^*\)

References
Dear colleagues,

First quarter of this year was enriched with lots of new developments in our organization. We actively took part in polio end game planning and implementation. We could even make a short film on immunization, for the first time in the history of State IAP. Kudos to Dr. Jiss Thomas and Team Kottayam. This year’s President’s Action Plan HIT is already commenced at various districts. DIET programmes and NSSK programs are going on with full swing in most of the branches.

Second edition of *Pediatric Companion* of the year 2016 is coming to you with lots of new changes.

In this edition, special articles on management of iron poisoning, NSAID, urticaria, and specific learning disabilities are included which will be useful to practicing pediatricians. Photo quiz updates you with recent happenings in the world of pediatrics while radiology quiz on skeletal dysplasias will refresh your knowledge in interpreting skeletal x-rays. Journal snippets give you the essence of the best among the researches in this field, happened across the globe.

Regards,

*Dr. M. Vijayakumar*

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Govt. Medical College, Calicut
Mob : 94470 71637
Email : drmvijaycalicut@gmail.com

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**Our Motto : ‘Team work for success’**

**Our Vision : ‘Enhancing quality of survival’**

**Our Mission : ‘Ensuring survival’**

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MESSAGE FROM PRESIDENT

Dear IAPians,

It gives me immense pleasure to address you again and thank you all for the support given to the HIT ACTION PLAN 2016. The family get together of IAP EB members was a grand success and it truly resulted in getting to know each other and to socialize and also energize each one of the participants.

I am happy to state that the flag of IAP is flying high, with all district branches and subspecialty chapters active in full strength and competing with each other for excellence. Our members are striving hard to nurture the existing and new branches and subspecialties, enhancing IAP membership, enrolling for e-voting, participating in school and public education sessions, supporting Mission Indradhanush and immunization campaigns including polio eradication end game strategies and so on.

Our IAP members have won laurels as well: Dr T U Sukumaran, senior IAPian, awarded PhD from M.G. University, Kottayam, being truly inspirational to the budding and blooming IAPians. Our salute to Prof. Dr. T U Sukumaran, on this unique and prestigious achievement. The IAP- TV initiative that was discussed by Dr. Sachidanada Kamath is also taking off. Many subspecialty chapters are now headed by Kerala IAPians. The State President of Kerala has been nominated to Indian College of Pediatrics of IAP.

Our attempt to produce an exclusive documentary on immunization has been eminently completed by Dr. Jiss and team from Kottayam and released during the SUMMER PEDICON, May 2016. Let us make it ‘viral’ and reap the fruits of this great effort. Also, let us utilize the IAP TV programme of CIAP by installing the free TV and Telecast for public education. Also, our dream project on book in Malayalam for Mothers and the public has to be executed. All those who can write in Malayalam are requested to send hard and soft copies to IAP Secretary for achieving this goal.

Now that the schools are reopening, and the monsoon is coming, we have a huge responsibility to shoulder. The rainy season illnesses are to be tackled, school based activities are to be resumed.

My plea to all district branches as well as HODs are to adopt a neighborhood school and let us educate the children and parents on D.I.E.T. sessions and also generate data on NCD prevalence, especially among the adolescents. I urge the IAPians, men to generate data on boys and women to generate data on girls. English and Malayalam proforma that are included in this issue shall be used for uniformity and for ease of compilation. Wonderful presents and appreciation are awaiting the best performers.

Another area of emphasis is on taking the module based teaching to all our district branches; for e.g., the Antibiotic Stewardship programme, the Asthma initiative, SOS HOPE, NEP and so on. Empowerment on triaging all the cases that we see into: Red (Very Urgent → ICU care), Amber (Urgent → Evaluation & Management) and Green (Non-urgent → Advice and Home care), the Traffic signal approach is to be achieved at service delivery level as well as UG & PG teaching.

Yours in IAP,
Jai Hind, Jai IAP
Dr. K.E. Elizabeth
Respected Colleague,

Greetings from IAP Kerala State office.

IAP Kerala 2016 activities have already started off with the observation of Days and Weeks celebrations and all the activities were conducted for the awareness of the public. This year’s President’s Action Plan ‘HIT’ will commence once the schools reopen along with last years DIET. As Non Communicable Diseases play a major role in our society, collecting statistics to understand the burden of Non Communicable Diseases among children is being planned.

Immunisation as always been a concern for Pediatricians and this year as part of an IAP Kerala initiative on Immunisation a short film REALISATION was released at the SUMMER PEDICON 2016 at Thalassery, the screenplay and script for the film was prepared by Dr Jiss Thomas from IAP Kottayam.

IAP Kerala Congratulates Dr Shaji Thomas John on being elected as the National President of Adolescent Health Academy 2016, Dr T.M. Ananda Kesavan on being elected as the National President Elect 2016 of Neurology Chapter, Dr M. Narayanan on being elected as the National Joint Secretary 2016 of Disability Chapter, Dr Jiss Thomas for the script of REALISATION.

Congratulations to Dr M. Vijayakumar for his work on Companion and Dr Shibu K on the Website. The President & Secretary of IAP Thalassery has to be congratulated for their project on ADOPT A SCHOOL and conducting largest number of DIET classes. IAP Kottayam too deserves congratulations for the DIET classes and IAP Cochin for the highest number of NSSK Programs under the leadership of Dr Sivaprasad. Special appreciation to IAP Women’s Wing of Kerala for conducting activities during World Women’s Day.

Expecting your wholehearted support and participation in the Sub Chapter Conferences and in all other activities of IAP Kerala.

Thanking you,
Yours sincerely,
Dr. Shimmy Paulose

Golden rules in preterm feeding

1) Minimal enteral nutrition should be initiated in all stable preterm as soon as possible.

2) Progressive enteral feeding is initiated sufficiently early and advanced at 20-30 ml/kg/day.

3) In general, premature babies with gestational age between 28-31 weeks require tube feeding, those with gestational age 32-34 weeks can be fed by paladai/ gokarnam and babies beyond 34 weeks can be fed directly from the breast to begin with.

4) Vitamin D and iron supplementation should be given to all preterm and LBW infants preferably for one year.
# IAP Kerala State President’s Action Plan – 2016

Dr. K.E. Elizabeth, President - 2016, IAP Kerala

<table>
<thead>
<tr>
<th>Name of the School &amp; Place</th>
</tr>
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<tbody>
<tr>
<td>Answer the following by putting a Tick mark</td>
</tr>
</tbody>
</table>

1. **Does your child take breakfast every day?**
   - Yes / No / Not sure

2. **How often does your child take Fast Junk food & Colas?**
   - Daily / Frequently- 4-6 days/week / Occasionally-1-3 days/week / Never

3. **Does your child take iron folic acid tablet every week?**
   - Yes / No / Not sure

4. **Does your child take exercise at least 2 Hrs/week?**
   - Yes / No / Not sure

5. **Do you think your child’s school bag is too heavy?**
   - Yes / No / Not sure

6. **Are there enough clean toilets in your child’s school?**
   - Yes / No / Not sure

7. **Has your child been vaccinated?**
   - Yes / No / Not sure

8. **Does your child share all events in his/her life with you?**
   - Yes / No / Not sure

9. **Does your child continuously use TV / Mobile / I-Pad for > 2 Hrs / week?**
   - Yes / No / Not sure

10. **Are you aware of any habits in your child related to alcoholism, smoking, drugs etc?**
    - Yes / No / Not sure

11. **Do you have any doubt about the safety of your child?**
    - Yes / No / Not sure

12. **Have you appreciated/congratulated your child for achievements other than scoring high marks?**
    - Yes / No / Not sure

13. **Do you have any dreams about your child’s future?**

14. **Do you often compare your child with another child?**

---

**Email / despatch completed sheets to:**

Dr. Riaz I.
Joint Secretary, IAP
Dept. of Pediatrics, SATH, Govt. Medical College, Trivandrum 695 011
Email: dr.riaztvm@gmail.com
Neonatal thyroid screening: Birth right of all newborn babies

Introduction

Congenital hypothyroidism is the commonest cause of mental retardation that can be prevented by early diagnosis, prompt management and regular follow up. Clinical detection is next to impossible in the neonatal period. A dedicated newborn screening program is the only solution to diagnose these children in the neonatal period.

Fetal brain growth and role of thyroxine

80% of brain growth occurs at fetal life and first 3 years of life. Thyroid hormones play a crucial role in early neurodevelopment. Brain development begins at first trimester but fetal thyroids start functioning only at 12 weeks of gestational age. Thyroid hormone required for brain development during this critical period should be supplied by the mother. Mother also contributes a major share of thyroid hormone for the baby even after 1st trimester. If the mother is having hypothyroidism, neurodevelopment of the baby is affected. Hence regular maternal screening for thyroid hormone deficiency also is equally important to prevent mental retardation.

Table 1: Thyroid screening during pregnancy

- Thyroid function tests are performed as soon as pregnancy is confirmed and every 4-6 weeks thereafter.
- TSH levels should be kept < 2.5 mU/L in the first trimester and < 3 mU/L in second and third trimesters.

Thyroid hormone levels in neonatal period

Within 30 minutes of delivery, TSH level rises to more than 60-70 mU/L. This phenomenon is called as neonatal thyroid surge. This is believed to be due to stress and relative hypothermia of extra uterine environment. TSH surge results in marked stimulation of thyroid gland, resulting in sudden rise in thyroxine levels within 24 hours. TSH level starts falling soon reaching 6-12 mU/L within 72 hours, even though throughout neonatal period, its levels are high compared to other age groups.

Table 2: Thyroid function tests in different age groups

<table>
<thead>
<tr>
<th>Age group</th>
<th>TSH (m IU/mL)</th>
<th>T4 (μg/dl)</th>
<th>FT4 ng/dl</th>
</tr>
</thead>
<tbody>
<tr>
<td>0-3 days</td>
<td>60-80 (TSH surge)</td>
<td>20-24 (T4 surge)</td>
<td>3-5</td>
</tr>
<tr>
<td>4-30 days</td>
<td>0.8-10</td>
<td>8-20</td>
<td>0.8-3</td>
</tr>
<tr>
<td>31 day – 12 mo</td>
<td>0.6-8</td>
<td>7-15</td>
<td>0.8-2.3</td>
</tr>
<tr>
<td>1-5 yrs</td>
<td>0.5-5</td>
<td>7-15</td>
<td>0.8-2</td>
</tr>
<tr>
<td>&gt;10 yr</td>
<td>0.5-5</td>
<td>5-12</td>
<td>0.8-2</td>
</tr>
</tbody>
</table>

Neonatal thyroid screening methods

Neonatal screening tests are TSH based, T4 based or simultaneous T4 & TSH based. TSH based tests are most sensitive and hence popularly performed all over the world. But this test fails to detect central hypothyroidism. It can be done at birth using cord blood sampling or can be done after 48 hours (when neonatal surge has become normalized). If only thyroid
screening is being planned, it can be done on cord blood, since it is painless and we can ensure 100% screening. Screening after 48 hours, using heel prick, is painful. Moreover, many cases may be discharged earlier and hence we cannot ensure 100% screening. But if we are paning it along with screening of other inborn errors of metabolism (phenyl ketonuria, galactosemia etc) late screening at 48-72 hours is preferred.

**Near normal development can be achieved if CH is diagnosed and treatment initiated <14 days of life**

**Biochemical criteria for starting thyroxine treatment**

- Screening TSH > 40 mU/L (for cord blood)– take venous blood for FT4 & TSH and start treatment without waiting for the result (if the results cannot be obtained on the same day).
- After 48 hours usually a cut off of TSH> 20 is taken for further evaluation.
- If the screening TSH < 40 mU/L; wait for confirmation (FT4,TSH) ---if the results are available within 1-2 days.
- FT4 < normal range : start treatment irrespective of TSH value.
- Start treatment if TSH > 20 mU/L, even if FT4 level is normal in the confirmation sample.
- If TSH: 10-20 mU/L, repeat the FT4/TSH again within 2 weeks and if still high, start treatment. These children should be followed up and the dose of thyroxine should be titrated based on subsequent thyroid function tests, since majority of children in this group have transient hypothyroidism. A trial off therapy (will be discussed later) should be considered at 3 years.

T4 can be used in place of free T4. Free T4 is preferred over T4 because this fraction is not protein bound and is metabolically active. In conditions where thyroid binding globulinin levels are low (prematurity, hypoproteinemia, and thyroid binding globulin deficiency) Total T4 levels will be low, but free T4 levels remain normal.

**Indications for second screening**

In some conditions, initial thyroid function results may be normal even though actually baby may be having hypothyroidism. They show TSH elevation once the child recovers from the illness. In these children, a repeat thyroid function is advised, preferably before 14 days of life or as soon as the disease process is over. Major indications are listed below.

- If TSH: 10-20 mU/L,
- Preterm babies (GA < 37 weeks)
- LBW & VLBW babies
- Sick neonates admitted in NICU
- Specimen collection within 24 hours of life
- Multiple birth especially if monozygotic twins

**Other investigations**

X-ray of knee for appearance of distal femoral epiphysis & proximal tibial epiphysis is done mainly to assess the degree of intra uterine hypothyroidism. Ultrasound examination of thyroid gland is a cost effective and commonly available method to detect the presence of the gland as well as its size and echo texture. But it can miss ectopic thyroid (lingual and sublingual). Thyroid isotope studies are not available everywhere. If facilities are available, scintigraphy should be carried out within 7 days of starting the treatment before suppression of TSH. Combining ultrasound and scintigraphy helps to improve diagnostic accuracy.

**Imaging should NEVER be allowed to delay the initiation of treatment. Hence don’t wait for imaging study if the procedure is being delayed.**
Always look for dysmorphic features and other systemic (cardiac or renal) co morbidities

At least 10% of children with congenital hypothyroidism are having congenital malformations. Cardiac renal and neuro-developmental anomalies are common in these children. Many have features of chromosomal anomalies like Down syndrome, Turner syndrome or Pendred syndrome.

Treatment

L-thyroxine is the drug of choice. The medication should be started as soon as possible and IDEALLY NO later than 2 weeks after birth. Initial dose is 10-15 µg/kg per day. Infants with severe disease (very low TT4/FT4) should be treated with highest initial dose. The drug should be given orally preferably in empty stomach same time each day. Avoid taking with Vitamin D, Iron and Calcium along with thyroxine since absorption of thyroxine is hampered by these agents.

Monitoring and Follow up

T4 will be normalized within 3 days and TSH in 2 weeks of initiation of therapy. First follow up should take place 2 weeks after initiation of treatment. TFT should be done at least 4 hours after the last thyroxine administration. T4/FT4 should be kept in the upper limit and TSH in the lower limit of reference range. Baby should be reviewed every 2 weeks until TFT is normalized.

Thereafter child should be reviewed once in 2 months till 1 yr, once in 4 months till 3 years and once in 6 months after 3 years. Additional evaluations should be done at 4-6 weeks after each dose change. More frequent evaluation is carried out if compliance is questioned or abnormal values are obtained.

Developmental assessment, vision and hearing should be assessed in should be done during each visit. Speech assessment is done at 3 years and speech therapy is initiated in selected children. IQ assessment and behavioral scores should be done at school entry. School performances should be assessed at each visit and assistance should be provided in children who are poor in their studies.

Indicators for poor outcome

- Delay in initiation of treatment
- Maternal hypothyroidism – poorly controlled
- Complete agenesis
- Absent knee epiphysis at term
- Very low T4
- Very high TSH

Off treatment

If the chance of transient hypothyroidism is more likely, a trial OFF treatment can be considered at 3 years. Initially decrease the dose of thyroxine by half and do thyroid function tests after one month. If TSH is elevated, re-start the treatment and child may require lifelong replacement. If TSH is normal, taper and stop thyroxine. After stopping thyroxine completely, if TSH is normal, repeat TFT at 1 month interval for 3 months. Then once in 3 months for 1 year, before declaring that child was having a transient hypothyroidism.

If TSH is high after stopping Thyroxine at 3 years of age, before re-starting treatment one could do a Thyroid scan also to find out the etiology of CH.

(Adapted from European Society for Paediatric Endocrinology Consensus Guidelines on Screening, Diagnosis, and Management of Congenital Hypothyroidism - Vol 99, issue 2, 2014)
Urticaria – A Review

Definition

Urticaria, also known as hives affect up to 20% of the population. It is characterized by pruritus and erythematous raised circumscribed lesion with central pallor that blanches with pressure called wheals. When it involves the loose connective tissue it is termed as angioedema.

Historical aspects

The earliest description of urticaria is found in a Chinese tome “the yellow emperor’s inner classic” written about around 1000 BC the word used for the disease was “Feng Yin Zheng” (wind typed concealed rash) which is still the Chinese word used for urticaria¹. The word urticaria means, “aria is wheals and urtica is nettle in Latin”²

Classification

I. Ordinary urticaria (Recurrent or Episodic )

Acute urticaria (up to 6 weeks duration) chronic urticaria (6 weeks or more)

Causes –drugs like Nsaids, Salicylates Idiopathic
ACE inhibitors -Hypothyroidism
-Food and additives -Connective tissue diseases
-Infection and infestations -Malignacies
- Inhalants- Immune complex mediated
-IgE mediated

II. Physical urticaria

Cold contact urticaria
Heat contact urticaria
Solar contact urticaria
Dermographism
Delayed pressure urticaria
Aquagenic uricaria
Exercise induced urticaria

III. Disease related to urticaria

Urticaria Pigmentosa
Urticarial vasculites

Immunology & Pathogenesis

Mast cell is the primary effector cell in urticaria.

There are cellular mediators and non cellular mediators.

CELLULAR MEDIATORS

Mast cells
Basophils
Eosinophils
Lymphocytes

Non Histaminergic Mediators

Bradykinin
ACE inhibitors

Pathogenesis

The binding of histamine to histamine receptors on the cutaneous microvasculature permits vasodilatation and hence vasopermeation.
Capillary Permeability results from the increased release of histamine from the mast cells situated around the capillaries. Other cellular mediators also cause vasodilatation and capillary permeability. This causes wheals in urticaria. Histamine also mediates itch through antidromic stimulation of local C fibers networks. The flare is produced by substance P release from cutaneous nerve endings rather than histamine.

Stimulation of peripheral H2 receptors also causes the same effects in the skin. H2 receptors are also present in gastric mucosa and hence many patients with urticaria also complaints of abdominal pain and gastritis.

About 1/3 of third of patients with Chronic idiopathic urticaria have circulating functional histamine releasing IgG auto antibodies that bind to high affinity IgE receptor (FcERI∞) on mast cells and basophils.

Some patients have IgG that does not bind to IgE receptors, but causes mast cell degranulation.

**Investigations**

Detailed lab investigations are not necessary. A good history usually give a clue starting from duration, time of the day of attack, possibility of food, medication etc.

Autologous serum skin test (ASST) is a non specific screening test. About 50%-60% of cases of chronic idiopathic urticaria show a positive ASST.

**Treatment**

**Acute urticaria**

- Try to find out the offending agents like food, drugs or infections and advice to avoid them.
- Oral H1 antihistamine is the main stay of treatment.
- If there is anaphylaxis, subcutaneous Adrenaline injection with or without parenteral H1&H2 antihistamines should be given immediately
- Systemic corticosteroids are sometimes useful
- Measures for preventing airway obstruction and IV fluids in case of shock.

**Chronic Urticaria**

Current International guidelines by European Academy of Allergology and Clinical Immunology (EAACI) guidelines recommend a three step algorithm for treatment.

Standard dose of second generation H1 antihistamines constitute the first line of treatment. If there is insufficient response, the conventional dose can be progressively increased to FOUR TIMES.

**Antihistamines**

H1 antihistamines, especially second generation antihistamines are commonly used in Urticaria. They have longer half life and lesser side effects such as sedation Cholinergic side effects and cardiototoxicity as compared to first generation.

Cetirizine, Levocetirizine, Desloratidine, Fexofenadine, Ebastine are the commonly used ones.

If the urticaria is not getting control with H1 antihistamine alone addition of H2 antihistamine also helps. But there are no randomized controlled trials for this combination.

**Second Line Therapies**

Oral corticosteroids are used in severe exacerbation and should be used for a short period in tapering course.

In resistant cases of Chronic urticaria other drugs like CyclosporinA (4mg/kg body wt/day) for 4-8 weeks can be tried. Leukotriene antagonist, Omalizumab are other agents used.

**References**

Beckwith Weidemann Syndrome in a term neonate

Introduction

In 1964, Hans-Rudolf Wiedemann reported a familial form of omphalocele with macroglossia in Germany. In 1969, J. Bruce Beckwith of Loma Linda University, California, described a similar series of patients. Originally, Professor Wiedemann coined the term EMG syndrome to describe the combination of congenital exomphalos, macroglossia, and gigantism. Over time, this constellation was renamed Beckwith-Wiedemann syndrome (BWS). Although the underlying causes of Beckwith-Wiedemann syndrome remain unclear, approximately 80% of patients demonstrate genotypic abnormalities of the distal region of chromosome arm 11p. Worldwide frequency is estimated at 1 in 13,700 live births. Here we are reporting a case of Beckwith-Weidmann syndrome.

Case Report

A term LGA girl baby, antenatally detected to have an abdominal wall defect, was delivered at 38 weeks of gestation by elective LSCS with a birth weight of 3.96 kg. At birth, baby had a large abdominal wall defect with a huge exomphalos (omphalocele) at the site of attachment of the umbilicus with a covering and contained loops of intestine. She was also noted to have few other features, namely, Naevus Flammeus on the forehead, Macroglossia, Preauricular skin Tag and Helical Pits. These clinical findings along with exomphalos led to the diagnosis of Beckwith-Weidemann Syndrome. The defect was covered with sterile sofratullie and tight dressing done till 30 days of age. We had anticipated that an early reduction of this large sized defect and exomphalos may lead to an increase in the intra-abdominal pressure. So tight dressing of the defect was done on weekly basis up to 30 days of age. Blood sugar monitoring was done strictly throughout the NICU stay and feeding monitored so that there was no episode of hypoglycemia. Thyroid function tests were normal.

At postnatal day 30, surgical reduction of exomphalos was done under general anesthesia. Peri-operatively, there were adhesions within the abdominal cavity which were released and the exomphalos was reduced. Baby had an uneventful postoperative period, and discharged on full breast feeds.

Discussion

Beckwith-Wiedemann syndrome is a pediatric overgrowth disorder involving a predisposition to tumor development. The clinical presentation is highly variable; some cases lack the hallmark features of exomphalos, macroglossia, and gigantism as originally described by Beckwith and Weidmann in 1969. Individuals with BWS may grow at an increased rate during the latter half of pregnancy and in the first few years of life, but adult heights are generally in the normal range. Abnormal growth may also manifest as hemihypertrophy and/or macroglossia. Hypoglycemia is reported in 30 to 50% of babies with BWS. There is an increased frequency of malformations and medical complications, including abdominal wall defects (omphalocele, umbilical hernia, and diastasis recti) and visceromegaly involving liver, spleen, pancreas, kidneys, or adrenals. Fetal adrenocortical cytomegaly is a pathognomonic finding. Renal anomalies may include primary malformations, renal medullary dysplasia, nephrocalcinosis, and nephrolithiasis. There is a predisposition to embryonal malignancies, with Wilms tumor and hepatoblastoma being the most common.

The mode of inheritance of BWS is complex. Possible patterns include autosomal dominant inheritance with variable expressivity, contiguous gene duplication at 11p15, and genomic imprinting.
Superovulation (ovarian stimulation) is an assisted reproductive technology (ART) for human subfertility/infertility treatment, which has been correlated with increased frequencies of imprinting disorders such as Angelman syndrome and BWS. Market-Velker et al. in 2010, examined the effects of superovulation on genomic imprinting in individual mouse blastocyst stage embryos. Superovulation perturbed genomic imprinting of both maternally and paternally expressed genes[1].

Three regions on 11p15 (BWSCR1, BWSCR2, and BWSCR3) may play a role in the development of BWS. BWSCR2 and BWSCR3 map, respectively, 5 Mb and 7 Mb proximal to BWSCR1, which is located 200 to 300 kb proximal to the IGF2 gene on 11p15.5. By sequence analysis of 73 kb containing BWSCR2, followed by screening a cDNA library, cDNAs encoding 2 zinc finger genes, ZNF214 and ZNF215 have been isolated.

Diagnosis is based on clinical findings. A ‘mild’ presentation may include prominent tongue and umbilical hernia (2 ). A careful cytogenetic analysis of the 11p15 region is recommended[2].

Since neonatal hypoglycemia is frequent (1 in 3 cases) and potentially deleterious for the CNS, Martinez-y-Martinez et al proposed monitoring the glycemia in BWS newborns every 6 hours during the first 3 days in order to correct
blood glucose levels below 2.6 mmol/l (46.8 mg/dl)[3].

Adrenal carcinoma, nephroblastoma, hepatoblastoma, and rhabdomyosarcoma occur with increased frequency and justify biannual abdominal ultrasound examinations(4). Wiedemann recommended that children with this syndrome be examined with renal sonography: first, at 3-month intervals, and after the third year of life, at 6-month intervals. Although less frequent, thoracic neuroblastoma occurs. A periodic chest radiograph is necessary (5).

DeBaun and Tucker(6) studied 183 children with Beckwith-Wiedemann syndrome followed for 482 person-years. Thirteen children (7.1%) were identified with cancers before the fourth year of life, and 6 of the tumors were Wilms tumors. The relative risk of Wilms tumor in Beckwith-Wiedemann syndrome patients over the general population was 816. The relative risk for neuroblastoma was 197 and the relative risk for hepatoblastoma was 2,280. Asymmetry of the limbs, or hemihypertrophy, was the only clinical feature associated with an increased relative risk of cancer, the relative risk being 4.6 with 95% confidence interval, 1.5 to 14.2 (6).

**Conclusion**

A diagnosis of Beckwith Weidmann syndrome can be made with a constellation of physical findings including macroglossia, exomphalous, naevus flammeus, preauricular tags and helical pits. Hypoglycemia is seen in 30-50% of cases. The majority of infants with hypoglycemia will be asymptomatic and have resolution of the hypoglycemia within the first 3 days of life. Less than 5% have hypoglycemia persisting beyond neonatal period. In our case, probably because of strict glucose monitoring baby had no documented hypoglycemia.

**References**

Iron poisoning: Management

**Case**

A 3 year old girl was referred from a local primary health centre with history of ingestion of unknown quantity of iron tablets 2 days earlier. The tablets were supplied to the girl’s pregnant mother from the Anganwadi. The presenting complaints were multiple episodes of vomiting and loose stools which started 2-3 hours after ingestion. She was treated in a local health care facility with intravenous fluids on the first day of illness. There was symptomatic relief and she was discharged home on that day itself. At the time of arrival on the third day of illness, she was lethargic with response only to pain. The girl was in hypotensive shock with respiratory failure. Investigations revealed features of multiple organ dysfunction and severe metabolic acidosis. In spite of treatment with supportive measures and desferrioxamine, she died on the 2nd day of hospitalization.

Significant overdose of iron results in multiple organ dysfunction involving almost every organ in the body due to damage caused by oxygen free radicals.

<table>
<thead>
<tr>
<th>Stage</th>
<th>Organ system</th>
<th>Onset of symptoms</th>
<th>Symptoms</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Gastro-intestinal</td>
<td>0-3 hrs</td>
<td>Vomiting/hematemesis, Loose stools, abdominal pain, irritability, lethargy</td>
</tr>
<tr>
<td>2</td>
<td>Apparent stabilization</td>
<td>Up to 12 hours</td>
<td>Symptoms subside</td>
</tr>
<tr>
<td>3</td>
<td>Mitochondrial toxicity</td>
<td>12-48 hrs</td>
<td>Shock, acidosis, coma, seizures, hyper/hypoglycemia, coagulopathy, acute tubular necrosis</td>
</tr>
<tr>
<td>4</td>
<td>Hepatic necrosis</td>
<td>&gt; 48 hrs</td>
<td>Jaundice, encephalopathy</td>
</tr>
<tr>
<td>5</td>
<td>Gastric scarring</td>
<td>2-4 weeks</td>
<td>GI scarring</td>
</tr>
</tbody>
</table>

Child may go directly to stage 3 depending on severity of intoxication. The second stage of apparent stabilization is due to redistribution of free circulating iron from intravascular space into intracellular space or reticuloendothelial cells.

**Assessment of toxic potential**

Clinical presentation, amount ingested and serum iron level

Patients requiring assessment

- Ingestion of >40mg/kg of elemental iron
- Ingestion of an unknown quantity
- Any symptomatic patient

Lack of symptoms within the first 6 hours makes significant toxicity unlikely

**Management**

- General supportive therapy: Take care of ABC. Treat shock with fluid boluses. Maintain glucose and potassium in the normal range

**Investigations**

**Asymptomatic patients**

- Abdominal X-ray if tablets were ingested

**Symptomatic patients**

- Unknown ingestion or >40mg/kg elemental iron ingestion: Serum iron concentrations 4 hourly until falling

**Serum iron:** Usually peaks at 4-6 hrs post ingestion. Values obtained after 4-6 hours may underestimate toxicity. For slow release or enteric coated tablets: Repeat at 8 hours.
Glucose
Renal and hepatic function
Prothrombin time
Electrolytes

**Decontamination**
Activated charcoal does not bind and hence not indicated

Whole bowel irrigation (WBI) with nasogastric colonic lavage solution (Peglec) 30ml/kg/hr until rectal effluent clear. Contraindicated in bowel obstruction

WBI indicated if abdominal X-Ray reveals tablets and >60mg/kg ingested

**Antidote**

**Desferrioxamine**
Indications:
Clinical toxicity, acidosis, coma, seizures
Iron visible on abdominal X-Ray

If serum iron level is readily available (most often not the case in Indian set up):
Serum iron concentration >90micromol/L
Concentration 60-90micromol/L and tablets visible on X-ray or symptomatic (vomiting, diarrhea, abdominal pain, hematemesis, fever)

Significant symptoms like altered consciousness, shock, acidosis or worsening symptoms. A fall in serum bicarbonate is a surrogate marker of systemic poisoning.

Dose: Begin at 10mg/kg/hour and increase to 15mg/kg/hr IV in 1-2 hours.

If oliguria or anuria develops hemo or peritoneal dialysis may become necessary

**End point of chelation therapy**
Significant poisoning usually requires therapy 12-16 hours

Continue treatment until:
Asymptomatic
Acidosis resolved
Serum iron <60 micomol/L
Return of urine colour to normal
Generally not more than 24hrs

Monitor respiratory status and oxygenation as ARDS has been reported with the use of desferrioxamine

**Discharge guidelines**
If <40mg/kg ingestion, negative X-Ray and asymptomatic 6 hrs postingestion

If ingestion >40mg/kg discharge only if remains asymptomatic and serum iron concentration falling and <60 micomols/L on 2 measurements 4 hours apart

Remember about the latent period in second stage like what happened in the case mentioned!

---

**Remember this formula**

**Swartz formula for estimated GFR (ml/min/1.73 m2 )**

41.3 x (height in meters / serum creatinine in mg/dl )

in pediatric RIFLE criteria for staging acute kidney injury in children
## INDIAN ACADEMY OF PEDIATRICS
### KERALA STATE BRANCH

**NOMINATION FORM FOR THE POST OF PRESIDENT / VICE PRESIDENT / SECRETARY / JOINT SECRETARY / TREASURER**

(PLEASE FILL-UP THE FORM IN BLOCK LETTERS)

<table>
<thead>
<tr>
<th>Name of the Office for which the Candidate is to be nominated</th>
</tr>
</thead>
<tbody>
<tr>
<td>Name of the Candidate (in full)</td>
</tr>
<tr>
<td>Address</td>
</tr>
<tr>
<td>District</td>
</tr>
<tr>
<td>IAP Membership No. of the Candidate</td>
</tr>
<tr>
<td>Mobile</td>
</tr>
</tbody>
</table>

(Self attested copy of Passport / IAP ID Card / PAN number card / Driving license to be attached)

<table>
<thead>
<tr>
<th>Name of the Proposer</th>
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<tbody>
<tr>
<td>Proposer's Address</td>
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<tr>
<td>Membership No. of the Proposer</td>
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<tr>
<td>Mobile</td>
</tr>
</tbody>
</table>

(Signature of Proposer & Date)

(Self attested copy of Passport / IAP ID Card / PAN number card / Driving license to be attached)

<table>
<thead>
<tr>
<th>Name of the Seconder</th>
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<tbody>
<tr>
<td>Seconder's Address</td>
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<tr>
<td>Membership No. of the Seconder</td>
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<tr>
<td>Mobile</td>
</tr>
</tbody>
</table>

(Signature of Seconder & Date)

(Self attested copy of Passport / IAP ID Card / PAN number card / Driving license to be attached)

**DECLARATION**

I hereby declare that I consent for nomination for the post as mentioned above. All information provided by me are true and correct to the best of my knowledge and belief. I shall abide by rule and regulations as per constitution of Indian Academy of Pediatrics. I understand that Election Commission has provided adequate information, in case of any discrepancy rules and regulations of the constitution of IAP shall apply. I also declare to practice the code of conduct prescribed by Election Commission of IAP.

Place:  
Date:  

Signature & name of candidate
INSTRUCTIONS

1. Please make sure about eligibility for the applied post and eligibility of proposer and seconder. [Eligibility- Candidates should have served for 2 complete years in the State EB Presidential candidate should be an IAP Life / Ordinary member for at least 10 years, Vice President candidate should be an IAP Life / Ordinary member for at least 7 years, Secretary/Joint Secretary/Treasurer candidate should be an IAP Life / Ordinary member for at least 3 years]

2. Fill complete form in capital letters. Incomplete form will be rejected.

3. Read carefully all the details given in notice before filling the form.

4. Make sure all particulars given are true and correct.

5. Check list of enclosures-
   - Completed nomination form.
   - Self-attested photo copy of valid ID - (Signature should be same as given on ID)
     a. Candidate- Passport / IAP ID Card / PAN card / Driving license
     b. Proposer- Passport / IAP ID Card / PAN number card / Driving license
     c. Seconder- Passport / IAP ID Card / PAN number card / Driving license
   - DD [for post of President Rs5,000 and post of Vice President Rs2,500] in favor of IAP KERALA CHAPTER payable at STATE BANK OF TRAVANCORE, KOTTAYAM

6. Last date and time for application- The complete nomination form with the nomination fee and ID proof should reach the Chief Election Commissioner on or before July 7th, 2016, 5 PM.

Address: Dr. George F. Moolayil, Emvee House, Kizhathadiyoor PO, Pala

NOTIFICATION

Elections for the post of State President Elect 2017 - 1 Post (Term 1 year)
State Vice President Elect 2017 - 1 Post (Term 1 year)
State Secretary 2017-18 - 1 Post (Term 2 years)
State Joint Secretary 2017-18 - 1 Post (Term 2 years)
State Treasurer 2017-18 - 1 Post (Term 2 years)

Eligibility: Candidates should have served in the State EB for 2 complete years. President Elect candidate should be an IAP Life / Ordinary member for at least 10 years
Vice President Elect candidate should be an IAP Life / Ordinary member for at least 7 years
Secretary/Joint Secretary/Treasurer candidate should be an IAP Life / Ordinary member for at least 3 years

Completed forms to reach the Chief Election Commissioner by 5pm on 7th July 2016
Scrubtiney of forms on 8th July 2016
Withdrawal date on 9th July 2016

Dr George F. Moolayil
Chief Election Commissioner

Dr M. Narayanan
Election Commissioner

Dr Jacob Abraham
Election Commissioner
Inauguration of World Immunisation Week at General Hospital, Ernakulam on 25.04.2016

Dr. Harikumar G, President IAP Kottayam, Prof Dr. Savidha P, HOD Pediatrics, ICH Medical College, Dr. Balachandar D, State Treasurer, Dr. Sunu John, Vice President IAP Kottayam, Dr. Jiss Thomas, Secretary, IAP Kottayam and Dr. Jayakumar P. R. at the Press Conference (World Immunization Week)

Moments of Glory

Dr. S.S. Kamath, National IAP President 2015 along with Dr. Jacob John and Dr. Abraham K Paul at Press Conference on Childhood Immunization

Run @ Kochi- World Immunization Week

Inauguration of World Immunisation Week at General Hospital, Ernakulam on 25.04.2016

Dr. Ashraf T.P, Executive Director, Kerala Social Security Mission & Additional Professor Medical College, Kozhikode receiving SBT Prathibha Samman Award from P.K. Mohanthy, Chief Secretary, Government of Kerala

Dr. Ashraf T.P, Executive Director, Kerala Social Security Mission & Additional Professor Medical College, Kozhikode receiving SBT Prathibha Samman Award from P.K. Mohanthy, Chief Secretary, Government of Kerala
Q 1: Infant with short stature. What are the abnormalities? What is the diagnosis?

Q 2: 8 years old girl with massive splenomegaly, thrombocytopenia, anemia, leucopenia and bone pain of long duration. What is the skeletal abnormality? What is the probable diagnosis?

Q 3: Boy with short trunk, short neck short stature. What is the skeletal finding? What is the probable diagnosis?

Q 4: A 4b years old child with coarse facial features, mental retardation and hepatosplenomegaly. What is the X ray finding? What is the probable diagnosis?

Q 5: 8 years old boy with short trunk short stature and mild mental retardation.

(Answers on Page 24)
1. A 3 month old child with infantile spasms; Connect these 2 pictures and diagnose the disease?

2. Identify the infective pathogen?

3. Identify the logo?

4. Identify this condition in a New born?

5. Identify the snakes

6. Which endocrinological problem is associated with this?

7. Abdominal pain, nausea and bilious vomiting and pallor, Peripheral smear of this boy is given. What is the diagnosis?

8. Identify this newer test?

(Answers on Page 24)
Snaps from the Branches

Dr. Rakesh Raju, Pediatrician taking class for the Asha workers at Vadakara.

Dr. Manoj Mony, Secretary, IAP Kollam talking on the need for compulsory Immunisation at Victoria Hospital.

Dr. Sidek, President, IAP Thalassery, Dr. Sakkariya, Secretary, IAP Thalassery, Dr. Dilkath K. and health workers campaigning for Immunisation of unimmunised children.

Shri Manoj, Circle Inspector of Police, Thalassery flagging off the Immunisation Road Show.

Dr. Sundaran U.G, President Dr. Mukesh, Secretary & Dr. Sugathan M.E., Treasurer, (IAP Thrissur) at the Press Conference.

Dr. Parmeswaran V., State President Elect 2016 addressing the gathering at Manjeri Medical College.

Dr. Mohandas Nair, Addl Prof of Pediatrics addressing the gathering on Immunisation at Mission Mukhti.

Dr. Venkiteswaran M.N., State President Elect 2016 talking on need for Compulsory Immunisation Card.

Dr. Sangeetha, President IAP Alappuzha taking Immunisation class Chikkoos Kaliyarangu on 28.04.2016.

Dr. Padmanabhan T.V. taking part in the Rally at Kanhangad (international women’s day).

Dr. Sister Betty taking a class on prevention of child abuse.

Dr. Rakesh Raju, Pediatrician taking class for the Asha workers at Vadakara.

Dr. Venkiteswaran M.N., State President Elect 2016 talking on need for Compulsory Immunisation Card.

Dr. Padmanabhan T.V. taking part in the Rally at Kanhangad (international women’s day).

Dr. Krishna Kumar, Director NIMHANS, Kozhikode giving a talk at Kasargod.

Dr. Jayakrishnan M.P., President IAP Kozhikode inaugurating the State Level Autism Day at Govt School of Nursing, Kozikode.

Release of the journal AUTISM VOICE by handing over first copy to Dr. Joshi K.K, President IAP Malappuram.

Prof Dr. Santhosh Kumar A, HOD Pediatrics lighting the lamp (World Autism Day).
1. Incorrect umbilical vein catheterization is associated with severe periventricular hemorrhages and mortality in extremely premature newborns.


What they did : They investigated the relationship between umbilical vein catheter (UVC) placement and death in extremely premature newborns (<29 weeks gestation) using a retrospective, case-control study design.

What they found : Death rate was 30% in infants with low lines versus 16% in those without. High UVC tip placement significantly increased severe periventricular hemorrhages (p = 0.014). In extremely premature newborns, incorrect UVC placement is significantly associated with death due to severe periventricular haemorrhage

2. Role of corticosteroids on survival orduration of mechanical ventilation among children with acute lunginjury.


What they did : This was a prospective cohort study from Children’s Hospital Philadelphia. They assessed the association between corticosteroid exposure and outcomes in children with ARDS. They prospectively screened patients admitted to the PICU between 2011 and 2014. Children 1 month to 18 years of age who were mechanically ventilated for >1 day and met the criteria for acute lung injury (PaO2/FiO2 ≤300 and bilateral parenchymal infiltrates) were enrolled.

What they found : In multivariate analysis, mortality did not significantly differ between the S- and S+ groups. Prolonged use of corticosteroids offers no benefit for pediatric ARDS.

3. Maternal vitamin D supplementation versus infant supplementation in ensuring adequate vitamin D levels in breastfeeding infants.


What they did : In this RCTMother/infant dyads were randomized at 4-6 weeks postpartum to either 400 IU of vitamin D3 per day to the mother and infant, 2,400 IU only to the mother, or 6,400 IU only to the mother.

What they found : Maternal vitamin D supplementation with 6,400 IU/day is as effective as infant vitamin D supplementation of 400 IU/day and offers an alternative strategy to infant supplementation.

4. Role of intravenous magnesium sulfate in pediatric patients with sickle cell disease admitted to the hospital for the management of pain crisis to find its effect on shortening the length of hospital stay, decreasing narcotic administration, and improving the quality of life


What they did : Children aged 4-21 years with sickle cell disease (including homozygous hemoglobin [Hb]SS and HbSb0 thalassemia) hospitalized for a pain crisis were enrolled.
Participants were randomized to receive standard therapy plus either magnesium (dosed at 40 mg/kg IV every 8 hours for up to 6 doses or until discharge) or placebo (an equivalent volume of normal saline given IV).

**What they found:** Magnesium sulfate did not provide a measurable benefit when added to standard care of children with sickle cell pain crisis.

5. Effects of oxygen saturation target ranges (85%-89% vs 91%-95%) on death or disability at 2 years corrected age in premature infants <28 weeks gestational age.


**What they did:** In this RCT infants born at <28 weeks gestational age in the UK and Australia were enrolled and randomly assigned to either a lower (85%-89%) or higher (91%-95%) target oxygen saturation range. Disability was defined as Bayley-III scores <85, visual impairment, cerebral palsy, or severe auditory impairment. These outcomes were compared in infants in the 2 treatment groups.

What they found: Death occurred more commonly in the lower-target group versus the higher-target group, but disability was not significantly different between the oxygen targeting groups. An oxygen saturation target range of 91%-95% is safer than a target range of 85%-89%.

### REFRESH UR RADIOLGY ANSWERS (PAGE 20)

1) Achondroplasia (Trident shape ileum (spinous process), Lucent area at upper end of femur, Interpedicular distance decreases from L1 to L5, Flared metaphyseal ends of long bones)

2) Gaucher disease (Erlenmeyer flask appearance - expanded lower end of femur)

3) Pear shaped lumbar vertebrae. Spondyloepiphyseal dysplasia congenital

4) Hook shaped (lower anterior beaking) vertebra and gibbus. MPS I (Hurler disease)

5) Double hump shaped vertebrae. Lacing of the ileac wings. Dyggve Melchior Clausen syndrome

### PHOTO QUIZ - ANSWERS (PAGE 21)

1) Aicardi syndrome: Triad of features: Agenesis of corpus callosum, chorioretinal lacunae, and infantile spasms a) Partial Agenesis of corpus callosum in MRI (grey matter heterotopia and abnormal gyration/polymicrogyria also seen) b) Chorio retinal lacunae

2) Zika virus. Zika virus got its name from Zika forest in Uganda. Transmitted by Aedes Aegypti. Newborn with Microcephaly

3) Logo of “The switch” to Bivalent OPV from trivalent OPV. (On 25th April 2016). Continued use of type 2 strain in vaccine carries the risk of VDPV. New recommendation: Combined IPV + bi OPV. Endgame strategic plan to withdraw OPV by 2019-20

4) Riga-Fede Disease, Laceration in ventral surface of tongue due to presence of natal teeth

5) a) Sand boa b) Lychodon (wolf snake) – Both are Non poisonous, (Mistaken for viper and krait)

6) Shortening of the fourth/fifth metacarpals: Pseudohypoparathyroidism. Idiopathic, post-traumatic, Turner syndrome. pseudopseudohypoparathyroidism

7) Lead poisoning (Basophilic stippling and pancytopenia)

8) CBNAAT (Cartridge Based Nucleic Acid Amplification Test) for rapid diagnosis and to find resistance of MDR/XDR TB. Freely available in Govt TB centre.
From the newborn nursery

Preterm baby born at 30 weeks of gestation, baby weight 1100 grams. Mother had gestational diabetes. As patient was referred with labour pain and it was an emergency admission did not get time to give antenatal steroids. Delivery was normal and the baby was not asphyxiated. Baby developed dyspnea within one hour. Made working diagnosis of Hyaline membrane disease, surfactant was administered and was put on CPAP. Dyspnea gradually improved and work of breathing came down and was maintaining oxygen saturation with room air.

At 36 hours suddenly dyspnea worsened
Three main possibilities were considered
1. Patent ductus arteriosus
2. Early sepsis and
3. Intracranial bleed.

There was no risk factors for sepsis
Anterior fontanel not bulging, no pallor or jaundice to support the possibility of IC bleed.

Pulse volume normal, a systolic murmur at the second left space supported the first possibility. X Ray chest showed cardiomegaly with increased vascular markings. We were not in a position to do ECHO as it was a Saturday evening

Baby was given ibuprofen 10 mg per kg followed by 5mg per kg two doses 24 hour apart.

Dyspnea worsened.

ECHO done next day, showed a ductus dependent systemic circulation. baby died within hours.

So in a scenario where ductal patency is essential for life we were trying to close it which must have contributed to death.

Our analysis and decisions were justified in the clinical context. Available evidence does not recommend routine ECHO for at risk neonates before giving Indomethacin.

Guidelines say clinical diagnosis of PDA should preferably be confirmed by ECHO prior to medications. But if this is not possible medications may be done on clinical diagnosis. Still once in awhile such a decision taken to save a baby may lead to totally opposite result.

Should we modify the above statement like this?

“Medications like indomethacin and ibuprofen should be done only after ECHO”

Before concluding,

Another equally important therapeutic decision, in another but similar scenario. Another drug “OXYGEN “, whether to give or withhold.

A baby born at term, cried normally after a short time gap develops dyspnea severe enough to cause cyanosis. Here we consider respiratory causes, as first possibility and some malformations top in the list. we’ll try to rule out all respiratory causes by clinical examination and x ray. Then only we’ll consider cardiac reasons especially few entities which are likely to be missed by clinical examination. This is typical scenario of presentation of ductus dependent pulmonary/ systemic circulation, (ie presentation after a gap of few hours as shock and distress in ductus dependent systemic circulation and dyspnea and cyanosis in ductus dependent pulmonary circulation). Most of these cases when clinical examination and X Ray are normal we ll put baby on oxygen naturally.

Ductal patency is keeping the baby alive and our life saving drug here will hasten the closure of Ductus and unknowingly worsens the situation.

Here again ECHO is ideal.

But how much practical?
Clinical Club

5 year old girl brought for growth and developmental retardation by elderly parents.
Clinical Diagnosis – Gestalt diagnosis was made in view of certain constellation of findings
Apert syndrome.-acro-cephalo-syndactyly
Birth prevalence: 1/65000

Etiology
• majority-sporadic, Autosomal Dominant
• Association-older paternal age
• FGFR2 gene mutation
Recurrence rate-
• unaffected parents : negligible, affected parents:50%

Abnormalities
Craniofacial:
• short AP diameter with high full forehead&flat occiput
• Irregular craniosynostosis
• Flat facies
• supraorbital horizontal groove,,shallow orbits,hypertelorism,
• Downslanting palpebral fissures,
• small nose,maxillary hypoplasia
• Dental anomalies
Limbs:
• osseous or cutaneous syndactyly
• complete fusoin of 2nd,3rd&4thfingers-MC
• short fingers
• distal hallus  broad & malformed
• cutaneous syndactyly of toes with or without osseous syndactyly

Growth
mean growth & length may be initially above 50th percentile, deceleration of growth in childhood.

Dr. Elizabeth K.E.
Professor of Pediatrics
Sree Mookambika Institute of Medical Sciences
Kanyakumari Dst.

Performance
Intellectual disability is common
CNS :
Corpus callosum  agenesis, Non progressive ventriculomegaly
Progressive hydrocephalus, Absent / defective septum pellucidum
Gyral abnormalities, Megalencephaly

Skin:
• Acne
Others:
• Fusion of cervical vertebrae, Short humerus, Genu valgum
• Synostosis of radius and ulna, GI abnormalities (1.5%)
• Respiratory anomalies (1.5%), Cardiac defects (10%)
• Genitourinary anomalies (10%)

Natural history
• May develop raised ICT-early surgery
• When thumb immobilised-may needearly surgery for pincer grasp
• Moderate to severe language problems
• Social withdrawal
• Hearing loss, Risk of corneal ulcers
• Early death can occur due to airway compromise
Specific Learning Disability

Ian, a 27 year old Australian, has severe difficulty reading and writing, but he passed his SSLC-equivalent examination by taking it orally. He is now married and gainfully employed. When Ian and his mother visited us in Fort Cochin, they stayed in a nearby flat. One afternoon his mother took a nap, after putting a sign outside the flat door: I AM HOME. When Ian returned from shopping and saw the sign, he came to my house instead, as he had read IAN HOME.

Specific learning disability usually manifests as an inexplicable discrepancy between a child’s IQ and his scholastic performance. The commonest and most serious type is Reading Disorder. Ian has all the classic features: HE READS EXTREMELY SLOWLY, word by word, often spelling out larger words, misreads simple words, omits words, and ignores punctuation. His speech is not fluent and he may search for words. You suspect mental retardation – but when you talk to him his comprehension is excellent, and so is his memory, while his vocabulary and grammar are adequate.

Those with severe Reading Disorder may struggle even at the start of schooling. Persons with milder problems may manage an average performance in school, though they have to struggle thrice as much as their peers to do so. But they just cannot manage in college, as reading is too intensive at this stage. They tend to do very badly in science fields, as subjects like Physics or Biology require a massive accumulation of available knowledge. (Contrary to popular myth, Einstein and Edison were astonishingly prolific readers and writers throughout life. Einstein mastered Euclid at 13 years. Edison read Gibbon’s 3790-page “Decline and Fall of the Roman Empire” at 12 years. Edison’s written output – he kept detailed notes of every experiment he performed – came to about 10,000 notebooks of 100 pages each, and there is no evidence of any difficulty in writing. Bill Gates dropped out of college because he found it boring and irrelevant to his great talents).

While those who read well tend to go (or be pushed) into the usual fields like Medicine or Engineering or Teaching, those with reading disorder are forced to diversify and use their available talents – which is sometimes a blessing in disguise. Thus a learning-disabled Picasso, a Rajan Tata, an Abhishek Bachhan, a Sachin Tendulkar, an AR Rahman are permitted and even encouraged to explore their talents. A UK study of small businessmen showed that they had a three-fold higher prevalence of Reading Disorder than the general population.

Writing Disorder is less common (1%) than Reading Disorder (4%), and often accompanies it. The child WRITES VERY SLOWLY and illegibly, with irregular spacing, and makes terrible spelling mistakes.

Mathematics Disorder (1%) presents as children who FEAR MATHS, and make the silliest errors (19+17=2, 70-12=62, 25+36=511) and count on their fingers even at 9 years. In ‘Balyakalasakhi’ Basheer humorously describes a severe case, possibly a fictionalized version of a true-life incident. Two thin streams join to form a river at Vaikom. When the Master sarcastically asks his victim, “you moron, how much is one plus one?” the trembling student answers “a very large ONE.”

Why do learning disorders occur? Why not? If many of us are pathetic at singing or dancing or painting, if some among us cannot catch a ball that is tossed gently at us, or throw a ball straight – is it surprising that others have difficulty in reading or math? We are lucky; a few centuries ago the ability to throw a spear straight and far was vital while the ability to read was irrelevant.
Anti-inflammatory drugs with focus on NSAIDs

Anti-inflammatory drugs were discovered by accident while analyzing content of plants and their extracts that were being used for the relief of pain, fever and inflammation. The leaves and bark of the willow tree, used as a remedy for aches and fever (1) contained salicin which metabolized into salicylic acid in the human body (2). The non-steroidal anti-inflammatory drugs (NSAIDS) were developed from organic acids – the pre-prostaglandin period before the 1970’s and thereafter when effects on prostaglandin production formed part of the screening in the drug-discovery process. Aspirin, indomethacin and phenylbutazone were chosen from the anti-inflammatory drugs in animal studies because they produced the least gastro-intestinal (GI) side effects.

In the 1990’s the two cyclo-oxygenase (COX) enzyme systems controlling the production of prostanoids [prostaglandins (PGs) and thromboxane (TxA2)] were discovered that changed the approach to dealing with inflammatory processes; COX-1 produced PGs and TxA2 that regulate gastrointestinal, renal, vascular and other physiological functions, and COX-2 regulated production of PGs involved in inflammation, pain and fever. Drugs that selectively controlled COX-2 and spared COX-1 - that was responsible for the adverse effects - were then sought. Research resulted in discovery of the highly selective COX-2 inhibitors - the coxibs (celecoxib and rofecoxib) - which had negligible GI side effects. Alarm bells rang when in late 2004 rofecoxib was withdrawn worldwide because of serious cardiovascular side-effects causing concerns and initiating research into the CVS and CNS (stroke) effects of the coxibs. The importance of COX isoforms in the pathogenesis of non-arthritic or non-pain states such as cancer and other neurodegenerative diseases; and the applications of NSAIDs and the coxibs in the prevention and treatment of these conditions as well as aspirin and other analogues in the prevention of thrombo-embolic diseases now constitute one of the major therapeutic developments of this century.

Though steroids are the most potent anti-inflammatory agents in children and adults alike, this article will focus on use of NSAIDs in children.

Choice of anti-inflammatory drugs in children

There is very little difference in the anti-inflammatory effect of various NSAIDs but response to the drugs and their tolerance varies from child to child. Most children respond to any NSAID and some who do not respond to one would respond to another. Though analgesic effect starts almost immediately after the first dose and full effect occurs within a week, anti-inflammatory effect may not manifest/ not be clinically demonstrable even after 3 weeks – effect in JIA takes 4-12 weeks (3). If adequate effect is not seen within the duration mentioned another NSAID needs to be started. Further, it is not possible to predict which child would respond to which NSAID. The child friendly formulations of various NSAIDs need to be used.

NSAIDs reduce the production of prostaglandins by inhibiting the enzyme COX-2. Since GI side-effects are rare in children taking NSAIDs for short periods, highly selective COX-
2 inhibitors are not mandatory and their role in children is undetermined.

Ibuprofen and naproxen are propionic acid derivatives used in children: Ibuprofen combines anti-inflammatory, analgesic, and antipyretic properties. It has fewer side-effects than other NSAIDs but its anti-inflammatory properties are weak. Most studies highlight its antipyretic and analgesic effect. Naproxen combines good efficacy with a low incidence of side-effects (4).

Diclofenac, indometacin, mefenamic acid, and piroxicam have properties similar to those of propionic acid derivatives: Diclofenac is similar in efficacy to naproxen (5). Indometacin has an action equal to or superior to that of naproxen, but with a high incidence of side-effects including headache, dizziness, and gastro-intestinal disturbances. It is rarely used in children and should be reserved for when other NSAIDs have been unsuccessful. Mefenamic acid has minor anti-inflammatory properties. It has occasionally been associated with diarrhoea and haemolytic anaemia which require discontinuation of treatment and must be used with extreme caution in children less than 14 yrs of age (6).

Piroxicam is as effective as naproxen and its half life of 30 hrs in children permits a once daily dosing of this drug (7). GI side effects and skin reactions limits its use.

Meloxicam and Etoricoxib are the only 2 selective inhibitors of cyclo-oxygenase-2 that have been licensed for use in adolescents (8) when they are intolerant to other NSAIDS. They are not to be used in children.

Aspirin has limited use in children because it has been associated with Reye’s syndrome. Aspirin-containing preparations should not be given to children and adolescents under 16 years, unless specifically indicated, such as for Kawasaki syndrome, Rheumatic fever, Henoch Shonlien purpura, prophylaxis of clot formation after cardiac surgery, or for prophylaxis of stroke in children at high risk. If aspirin causes dyspepsia, or if the child is at a high risk of gastro-intestinal bleeding, a proton pump inhibitor or a H2-receptor antagonist can be added.

References
1. An aspirin a day keeps the doctor at bay. The world’s first blockbuster drug is a hundred years old this week. http://www.nobelprizes.com/nobel/medicine/aspirin.html Accessed on 25/9/13
Allergy Skin Testing - An Overview

Allergy is defined as an abnormal clinical reaction towards substances which are harmless to most individuals. The typical allergic reaction is an IgE-mediated response resulting from activation of TH2 pathway (instead of TH1 pathway). Eventhough used interchangebly, the terms ‘Atopy’ and ‘Allergy’ are not the same. ‘Atopy’ is only an increased tendency to produce specific IgE to common environmental allergens.

The term allergen refers to an antigen that can trigger an allergic reaction. Allergens can sensitize the immune system via respiratory tract (aeroallergens), gastrointestinal tract (food allergens) or skin(contact allergens). A thorough history including details of clinical features, trigger factors, family history, environment history, diet history and medication history along with a focused clinical examination are important in making a diagnosis of an allergic disease.

Various in-vivo and in-vitro tests are available to help diagnosis of allergic disease and identification of the allergen(s). However, it is very important to remember that, in fact, all “allergy tests” can detect only “sensitization” and cannot diagnose “allergy” as allergy is a clinical syndrome. Hence, diagnosis of allergy is always clinical.

Allergy Skin testing is an important invivo test for detecting the allergen specific IgE. It is done by introducing a very small quantity of allergen in to the epidermis by pricking or puncturing. The introduced allergen will link up with the mast cell bound specific IgE antibodies if present. This leads to release of chemical mediators,causing a wheal and flare reaction.

**Indications of Allergy Skin Testing:**
- Allergic Rhinitis, Allergic Conjunctivitis,
- Allergic Asthma
- Suspected Food allergy.
- Insect bite Venom Allergy
- Suspected Drug Allergy
- Suspected Latex Allergy

**Contraindications of Allergy Skin Testing**
- Recent Anaphylaxis
- Severe Eczema,

**Procedure**

Prick test (Epicutaneous Test) can be performed on the volar surface of forearm or upper back. Skin testing areas should be atleast 5 cm from the wrist and 3 cm from the ante cubital fossa. There should be atleast 2-2.5 cm gap between each applied allergen to avoid false positive reaction between adjacent skin test sites.

The test should be performed only after cleaning the skin properly. A small lancet or 25/26 gauge needle is used to pierce through a drop of allergen extract placed over the prepared skin. Always make sure that only good quality allergen extracts are used. skin is pierced at an angle of 45-60 degree. The needle should be lightly pressed in to the epidermis and then lifted creating a break in the epidermis with out causing bleeding. After 15-20 minutes, the pricked site should be looked for wheal and flare. A 3 mm or more wheal diameter is taken as positive. Glycerinated buffered saline and histamine are used as negative and positive control respectively.

**Prick to Prick test**

Used in diagnosis of food allergy. The commercially obtained food extracts are less reliable as the heat labile proteins are destroyed during preparation. The lancet/needle is inserted in to the suspected fresh food, and then the patient’s skin is pricked immediately.

**Intradermal Skin Tests:**

Intradermal tests are more sensitive than the prick tests. But they are only done if the epicutaneous test is negative, and there is high clinical suspicion based on history. The allergen extracts used for intradermal testing are 100 to 1000 fold less concentrated than extracts used in epicutaneous tests. A small amount of allergen
extract (0.01-0.02 mL) is injected into the dermis of the arm using 25-27 gauge needles. Intradermal tests are never done with food allergens because of the risk of anaphylaxis.

Medications to be avoided before Allergy Skin Testing:
Anti histamines, both H1 & H2 blockers, should be discontinued for 3 days, Fexofenadine for 5-7 days, Loratidine & Cetrizine for 7 days. Tricyclic antidepressants should be discontinued for 7 days as they have anti histaminic effects. Beta blockers increase the risk of allergic reactions and hence they should be discontinued for 1-2 days.

Allergy Skin testing is a rapid, sensitive and cost effective way of detecting IgE mediated disease. Along with a thorough history and detailed clinical examination it forms a perfect adjunct in the clinical management of allergy.

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**Realization**

Realization, a short film is an initiative of IAP Kerala.

Dr. Jiss Thomas Palukkunel and his crew have done a commendable work to bring out this film which contains a strong message against anti vaccine campaigns.

Youtube link: [http://youtu.be/I0pyMi4b0po](http://youtu.be/I0pyMi4b0po)
1. **What is “puerile breathing”**?

Vesicular breath sounds in children up to the age of 9 years are higher pitched than those of adults, and hence may be mistaken for bronchial breathing. This was referred to as “puerile breathing” by none other than Laennec.

2. **What is egophony? How will you imitate egophony?**

The term ‘egophony’ is derived from Greek and means ‘voice of the goat’.

The quality of the spoken voice is altered so that it has a nasal quality which resembles the bleating of a goat.

Ask the patient to say ‘one, one’ loudly and then suddenly pinch his nostrils with the fingers, while he continues to count.

3. **What are the differences between bacterial pneumonia and viral pneumonia?**

<table>
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<tr>
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<th>Bacterial pneumonia</th>
<th>Viral pneumonia</th>
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<tbody>
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<td>Gradual</td>
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<tr>
<td>Epidemic pattern</td>
<td>Absent</td>
<td>Present</td>
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<tr>
<td>Course</td>
<td>Progressive</td>
<td>Self-limited</td>
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<td>Temperature</td>
<td>++++</td>
<td>+/-</td>
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<td>Toxemia</td>
<td>++++</td>
<td>Absent</td>
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<td>Respdistress</td>
<td>+++</td>
<td>+ (infants)</td>
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<td>Crackles</td>
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<td>+/-</td>
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<tr>
<td>Wheeze</td>
<td>+/-</td>
<td>++</td>
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<td>Radiological</td>
<td>Confluent infiltrates</td>
<td>Diffuse infiltrates (RSV infection)</td>
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<td>(Consolidation)</td>
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<tr>
<td>Hyperinflation</td>
<td>+/-</td>
<td>+</td>
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<tr>
<td>Pleural effusion</td>
<td>++</td>
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<tr>
<td>Pneumatocele</td>
<td>May be seen in</td>
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4. **What are the differences between pleural rub and crepitations?**

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<th>Crepitations</th>
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<td>Associated pain</td>
<td>Present</td>
<td>Absent</td>
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<tr>
<td>Palpability</td>
<td>May be palpable</td>
<td>Never palpable</td>
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<tr>
<td>Pressure with stethoscope over chest</td>
<td>Increases</td>
<td>No change</td>
</tr>
</tbody>
</table>

5. **What are the physical findings of fluid overload?**

- Edema
- Hypertension
- Elevated jugular venous pressure
- Abdomino-jugular reflux
- S3 gallop
- Basal crepitations
- Ascites

6. **What are the genetic diseases that cause bronchiectasis?**

- Cystic fibrosis
- **Williams-Campbell syndrome** - a developmental defect of bronchial cartilages.
- Mounier-Kuhn syndrome - a developmental defect of elastic and muscular tissues of trachea and bronchi (tracheobronchomegaly).
• Kartagener’s syndrome is a form of immotile cilia syndrome characterized by bronchiectasis, chronic sinusitis or frontal sinus agenesis and situs inversus.

• Chandra-Khetarpal syndrome: Levocardia, sinusitis and bronchiectasis; no ciliary abnormality

• Young’s syndrome - an overlap syndrome between immotile cilia and cystic fibrosis. Spermatogenesis and sweat chloride are normal.

• Yellow nail syndrome - characterized by yellow nails, bronchiectasis, pleural effusion and lymphedema.

• Alpha-1- antitrypsin deficiency

• Various primary immunodeficiency syndromes like Bruton’s disease

7. **Why is the LLL more prone to develop bronchiectasis?**

• The diameter of the left main bronchus is only two-third of that of the right main bronchus.

• The left main bronchus crosses the mediastinum at an acute angle behind the aorta by which it can be easily compressed.

• The apical and posterior basal segments of the LLL are always in dependent position in all common positions such as erect, semi-recumbent and lying.

8. **What is Brock syndrome?**

Brock syndrome or middle lobe bronchiectasis is characterized by recurrent atelectasis of the right middle lobe, in the absence of endobronchial obstruction.

After several episodes of atelectasis, the patient finally develops bronchiectasis of RML.

It is usually a complication of primary pulmonary tuberculosis resulting from obstruction of RML bronchus by TB lymph nodes.

9. **What is three-layered sputum?**

When the sputum from a patient with bronchiectasis is collected in a conical flask, it forms three layers:

• Upper thin frothy layer

• Middle thick and mucopurulent layer

• Lower opaque purulent layer

10. **What is the best view for evaluating a pleural effusion?**

Lateral decubitus view is helpful to determine how much fluid is present and whether it is free flowing or loculated.

11. **What is the most common congenital anomaly of the lung?**

The most common congenital anomaly of the lung is congenital lobar emphysema, the most common site being the left upper lobe.

The second commonest anomaly is congenital cystic adenomatoid malformation. This comes in the radiologic differential diagnosis of diaphragmatic hernia

12. **What is Boerhaave’s syndrome?**

This refers to the barogenic rupture of the esophagus, which follows an intense episode of retching and vomiting and usually drains into the left pleural space or peritoneum. The tear usually occurs longitudinally and lies on the left posterolateral wall of the esophagus.

It was first described by Boerhaave in 1724, after autopsy of the High Admiral of the Dutch navy, the Baron van Wessenaer.

13. **What percentages of the lung volume are provided by the right and left lungs?**

The right lung provides 55% and left lung 45% of the total lung volume.

14. **What is the most common cause of hoarseness in children?**
Singer’s nodule or screamer’s nodules. These are vocal cord nodules caused by vocal abuse like screaming and shouting, and coughing.

15. **What are the risk factors for aspiration pneumonia?**
- Gastro esophageal reflux
- Comatose patient
- Seizure disorder
- Periodontal infections
- Bulbar palsy

16. **Which finding in bronchospasm is most ominous?**
A silent chest in a tired and lethargic patient with airway obstruction signifies exhaustion and impending respiratory arrest. Previously heard wheezes disappear because air flow velocity is decreased in obstructed airways and no sounds are produced. Such a situation requires prompt intervention with intubation and mechanical ventilation.

17. **What are the skin findings in pulmonary diseases?**
- Central cyanosis—in severe hypoxemia
- Erythema nodosum in many pulmonary diseases, especially tuberculosis
- Lupus vulgaris in tuberculosis
- Lupus pernio in sarcoidosis
- Vesicles in varicella pneumonia
- Ulcers in tularemia pneumonia
- Horder’s spots in psittacosis pneumonia—faint pink macules on the trunk

18. **What are the ocular findings in pulmonary diseases?**
- Tuberculosis—phlyctenularkerato conjunctivitis, chorioretinitis, iridocyclitis, choroid tubercles
- Sarcoidosis—optic nerve involvement with gradual loss of vision
- SLE—episcleritis and uveitis
- Sjogren’s syndrome which causes interstitial lung disease – keratoconjunctivitis
- Wegener’s granulomatosis—lid edema, nasolacrimal duct obstruction, proptosis, and conjunctivalchemosis

19. **What are the sputum microscopy findings in asthma and bronchiectasis?**
Asthma- Curschmann spirals and Charcot Leyden crystals
Bronchiectasis- Dittrich plugs

20. **What are the conditions in which breath sounds are absent?**
- Massive pleural effusion
- Collapse with occluded bronchus
- Pneumothorax
- Near fatal asthma
- Pneumonectomy
- Agenesis of lung

21. **When should heli-ox be used in asthma?**
Heli-ox is a blend of helium and oxygen that has a gas density less than that of air.

Reduced gas density has the potential to reduce airway resistance. Further studies are required before this gas can be recommended in the routine management of acute asthma.

In the meantime, heli-ox should be used in patients with very severe exacerbations of asthma who do not respond to the usual vigorous therapy.

22. **What is “supermarket cough”?**
“Supermarket cough” is heard in tracheomalacia.

This is a condition in which a part of the trachea is unusually soft. The soft area of the trachea is more collapsible and causes a coarse inspiratory stridor. When the child coughs, the more easily collapsible soft area of trachea
generates a loud brassy cough—best described as the “supermarket cough”.

This is because the child gets immediate attention from other shoppers in the supermarket when they hear the loud harsh cough.

23. What is hyperoxia test?

Hyperoxia test helps to differentiate an infant with congenital cyanotic heart disease from one with lung disease.

The infant is given 100% oxygen, and an arterial blood gas level is obtained.

If the child has a primary lung disease, a PaO2 > 100 mmHg is usually achieved. If he has a CCHD, PaO2 is < 100 mmHg.

Typically, children with CCHD also have a low or normal pCO2, whereas children with lung disease have an elevated pCO2.

Unfortunately, the hyperoxia test does not usually distinguish children with cyanotic heart disease from those with persistent pulmonary hypertension.

24. What are endothelin receptor antagonists?

Endothelin receptor antagonists reverse the effect of endothelin1, which is a powerful vasoconstrictor. As a result, these drugs also produce vasodilatation and hence are useful in pulmonary hypertension.

They are of 2 types:

A) Oral non-selective:

Bosentan and macitentan: nonselective dual receptor antagonists - approved for use in pulmonary hypertension

B) Selective antagonists

• Sitaxentan
• Ambrisentan

25. What is the role of omalizumab in asthma?

Omalizumab is a recombinant monoclonal antibody that was developed by immunizing mice with human IgE.

It forms a complex with unbound IgE, interferes with its binding with cell receptors, and thus prevents the subsequent release of inflammatory and chemical mediators, including histamine.

This drug which has a very good safety profile, is useful in the management of moderate and severe asthma with hyper IgE.

Points to remember while treating febrile seizures

1) Iron deficiency is associated with increased risk of febrile seizures. Hence all children with febrile seizures should be screened and effectively treated for iron deficiency anemia to get a better result.

2) In majority of cases, continuos therapy with valproate or phenobarbitone do not give much long term benefits and they are having life threatening adverse effects.

3) In a child with first attack of febrile seizure, if he is otherwise neurologically normal, EEG would not predict future recurrence even if the result is abnormal. Hence EEG need not be performed in all cases.
IAP THALASSERY

Report by Dr Sakkariya PP

01.01.2016: New Year Celebration IAP Thalassery chapter celebrated New Year with the students of Special School Dharmadam Thalassery.

05.01.2016: Installation Ceremony
Installation ceremony started with family get together in Pearl View Regency Thalassery. Dr. Divakaran.K, Past State President, IAP Kerala was the Installing Officer. He installed Dr. Siddique.K.P.A as new branch President, Dr. Sakkariya.P.P as new branch Secretary and Dr. Ashok.M as new branch Treasurer.

12.01.2016: CME on Paediatric Oncology conducted at IMA Hall Thalassery. Speaker was Prof. Rani George, Prof. of Paediatric Oncology, Dana Farber Cancer Institute Harvard Medical School, Cambridge.

15.01.2016: School Health Camp IAP Thalssery conducted a Health camp at Govt. L.P. School Chettamcoon, Thalassery. Camp was inaugurated by Saiitha, Ward Counselor.

15.01.2016: DIET Programme IAP Thalassery conducted a Diet programme in association with Kazhcha Sports & Arts Club, Thalassery at Govt. School Chettamcoon, Thalassery. The programme was inaugurated by Mrs. Najma Hasheem, Vice Chairperson, Thalassery Municipality.

17.01.2016: Pulse Polio Immunisation Motivation activities for the public started on 17th January 2016, prior to pulse polio day. Numbers of polio camps are conducted in various hospitals and public places. Mrs. Najma Hasheem, Vice Chairperson Thalassery Muncipality.

21.03.2016: observed World Down Syndrome day: CME on “Fetal Hydronephrosis How to Tackle?” taken by Dr. Abhilash Antony.

26.03.2016: observed World TB Day and conducted several awareness programmes.

31.03.2016: state level inauguration of IAP School Adoption programme conducted at Chettamcoon Govt. L.P. School, Chettamcoon, Thalassery.

IAP KASARAGOD

13-2-16: Adolescent nutrition class conducted at GHSS kasaragod. 20-2-16 Monthly meeting held on at Krishna nursing home, Vidyanagar Kasaragod. Dr Harsha Prasad, Consultant pediatric haemologist & oncologist, KMC spoke about primary immunodeficiency disorders.
Kasaragod. 21-2-2016: Pulse polio second round district level inauguration held at Malikdeenar charitable hospital. Hon.MLA N A Nellikunnu inaugurated the programme.11-3-2016: World measles day and vaccination awareness class conducted at General hospital Kasaragod. Programme inaugurated by municipal chairperson smt Beefathima Ibrahim. Dr Harikrishnan junior consultant pediatrician GH Kasaragod conducted class about measles and vaccination awareness.22-3-2016 IAP kasaragod in association with general hospital Kasaragod, day care centre Kasaragod and IMA Kasaragod observed world down syndrome day GH conference hall.Dr Ranjith superintendent GH Kasaragod inaugurated function.. Dr Harikrishnan discussed about Down syndrome with parents of kids 23-3-2016: Observed world TB day. IAP president Dr Narayana naik participated in TB day rally flaged off by Dr Dinesh Kumar DMO Kasaragod participated. TB day inauguration done by Hon district collector. TB day message given by former DTO Dr Ceriyac Antony. 24-2016: World autism awareness day observed at IMA hall Kasaragod. Dr P Krishnakumar, Director IMHANS Calicut was guest speaker. 26-3-2016. Conducted a class on switch from TOPV to BOPV and IPV. Dr Divakara rai took class. World health day 2016 observed by IAP Kasaragod in association with IMA Kasaragod, Govt general hospital kasaragod, PPUNIT kasaragod, Quiz master Dr Janardhana naik conducted quiz on diabetes. Dr Abdul Sathar MRCP conducted class on world health day theme beat diabetes.

IAP KOTTAYAM

Reported by Dr. Jiss Thomas (Secretary)

27-1-2016. DIET program at Mangalam school by by secretary Dr. Jiss Thomas.February 8-2-2016. Disability camp Dr. Sukumaran Dr. Jiss Dr. Hari Dr. Sunu Dr. Balachander at Pediatric house.

February 10 DIET class at NSS school changanassery by Dr. Anush.February 10 DIET and PFT camp at Mount carmel school Dr. Jiss. February 10 Healthy life style marathon at Pala Dr. Sebastian Lukose.February 14 installation ceremony by Dr. T.U. Sukumaran v Dr. Harikumar president Dr. Jiss Thomas secretary Dr. Anush joint secretary Dr. Sunu John vice chairman Dr. Bindhu K. P treasurer followed by CME. February 19 DIET at Belmont school Kottayam Dr. Jiss.February 24 DIET at marian school Dr. Shaji. March 8 Women s day at Caritas Health talk Dr, Sunu John Message Dr. Savida.March 8 Women s day at ICH Dr. Savida Dr. bindhu. March 9 Women s day talk at Pala Alphonsa college Dr. Savida. March 13 CME talk by Dr. Vijayakumar Dr. Sara Chandy at Pediatric house. March 14 DIET at Baker school Kottayam Dr. Shaji. March 21 Workld Down syndrome day at ICH Kottayam Dr, Savida Dr. Jiss Dr. Bindhu competitions and health talk.

March 22 World TB day at KIMS Dr. Jiss health talk. March 23 World tb day at ICH Dr, Savida Dr, Jiss class for PG s and case presentation. March 30 world TB Day at District hospital Kottayam Dr. Murari and Dr. Laly Rally flag of by Collector. April 2 World autism day Dr. Jiss at RAAAHPA Kottayam. April 7 World Health day marathon. April 19 World liver day marathon Dr. Jiss from Kumaranelloor to kottayam. April 26 Press release on immunisation week Dr. Savida Dr. Jayakumar PR Dr. Harikumar Dr. Jiss Thomas Dr. Sunu John Dr. Balachander. April 27 immunization week talk Dr, Sunu Caritas Hospital. April 29 Immunization Poster exhibition Caritas Dr. Sunu. April 30 Health talk in Pala Dr. Harikumar

IAP PARIYARAM

Reported by Dr MTP Muhammad (Secretary)

Immunization week is observed with various activities like awareness classes to mothers, demonstrations, posture displays etc. Secretary Dr Mohammed MTP conducted Vaccination - FAQS to Pediatricians of Payyanur and Taliparamb. Associate Professor of Pediatrics Dr KV Urmila conducted awareness class to mothers on Measles Day. On world Diabetes day Prof. of Medicine Dr Balakrishnan Valliot delivered a talk. tOPV to BOPV switch and IPV Introduction calls taken by SMO Calicut, Dr Srinath Ramamoorthi and RCH Officer Dr Jyothi, on 22.4.2016..A new illuminated Flux box with a cartoon display stressing about the importance of vaccination is raised at the immunisation clinic of Pariyaram medical college.
2. Eating a large amount of food quickly
4. Tooth shaped
5. Of sound mind
7. Mute is a more polite word
8. Cannot speak
11. In autism earliest repetitive movements are often seen in
12. The science of medicine dosage
13. Sharp or sudden
14. Phobia- fear of going to bed
15. Alkali used in the treatment of distal renal tubular acidosis
17. 24 hour heart monitor
19. Overproduction of aldosterone leads to ——— syndrome
20. Medical prioritizing
21. A form of communication
22. Developmental scale used to assess maturation

ACROSS
1. A risk factor that impacts health
3. Ascending limb of nephron is site of action of ……… diuretics
6. A benign epithelial tumor with a glandular organization
9. Autosomal Recessive congenital pancytopenia
10. Dull continuous pain
13. Evolutionary tendency to revert to ancestral type
18. Hepatic stellate cells are called ——— cells
19. Overproduction of aldosterone leads to ——— syndrome
20. Medical prioritizing
21. A form of communication
22. Developmental scale used to assess maturation

PEDI-CROSSWORD
Please send in your answers by 1st October 2016.
An attractive prize awaits the winner.

JANUARY 2016 ISSUE SOLUTION

Winner
Dr. Jessy P.L.
Consultant Pediatrician
Lisie Hospital, Ernakulam
Mob 9656110305
## Conversion Table

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Less pain with Norditropin® Nordilet® may aid better compliance.

References
1. Norditropin® Nordilet® India Prescribing Information.
Committed to Innovation

Nordilet® is liquid, prefilled, multidose pen

Available in 3 different strengths: 5, 10 and 15 mg
No refrigeration after first use – stable up to 25°C
Prefilled multidose pen
Ready to use – no mixing required
Using NovoFine® needles and histidine buffer minimises pain


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