**EXECUTIVE COMMITTEE FOR THE YEAR 2011**

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**CIAP’S REPRESENTATIVE:**
Executive Director  
Dr. A. S. Vasudev

**EDITOR-IN-CHIEF**
Indian Pediatrics

**EXECUTIVE MEMBER**

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<tr>
<th>CENTRAL ZONE</th>
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<tr>
<td>Dr. J. S. Bhasin</td>
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<tr>
<td>Dr. V. K. Goyal</td>
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<tr>
<td>Dr. Uttam Pal</td>
<td>9811057237</td>
<td><a href="mailto:uttampal57@gmail.com">uttampal57@gmail.com</a></td>
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<tr>
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<tr>
<td>Dr. R. K. Sinha</td>
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<th>NORTH ZONE</th>
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<tr>
<td>Dr. Rajesh Gupta</td>
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<tr>
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**REPRESENTATIVE FROM SERVICES**
K. S. Rana  
Tel: 9910056737  
Email: kanersinghrana@yahoo.co.in

**MEMBERS CIAP EXECUTIVE BOARD**

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<tr>
<th>Name</th>
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<tr>
<td>Dr. Sunil Mehendiratta</td>
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Organising Committee

A.S. Vasudev
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Anil Bajaj
Secretary
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<table>
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<tr>
<th>Time</th>
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<td>9.30 – 10.00 am</td>
<td>Late Morbidities of Preterm</td>
<td>Dr Sushma Kaul</td>
<td>Dr Harish Chellani, Dr Rajeev Seth</td>
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<td>10.00-10.30 am</td>
<td>Use and abuse of height enhancing methods in children and adolescents with short stature</td>
<td>Dr IPS Kochar</td>
<td>Dr Anju Virmani, Dr Sunil Gomber</td>
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<td>10.30 – 11.30 am</td>
<td>Adolescence… an Age of opportunity.</td>
<td>Dr Suresh K Mohammed (R.C.H., GOI)</td>
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<td>Dr Swati Bhave</td>
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<td>Global Perspectives: Maternal Health Interventions for saving newborns</td>
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<td>Role of Skilled Birth Attendant &amp; ASHA’s in reducing neonatal mortality: Field Experience Jharkhand &amp; UP</td>
<td>Dr Manju Shukla and Dr Dharamendra Panwar (Vistar / USAID)</td>
<td>Dr S K Bhargava, Dr Ajay Gambhir</td>
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### 1.15 -1.30 pm  
**Maternal and Newborn Care Package**  
**Speaker**: Dr Rajiv Tandon (Save the Children)  
**Chair**: Dr Vidya Gupta, Dr D N Virmani

### 1.30-2.15 pm  
**Lunch**

### 2.15 – 2.45 pm  
**Allergic March in Children**  
**Speaker**: Dr Kerstin Wall (Sweden)  
**Chair**: Dr Nalin Nag, Dr A K Sharma

### 2.45 – 3.00 pm  
**Pediatric Urology in 2011**  
**Speaker**: Dr Sujit Chowdhary  
**Chair**: Dr Anurag Krishna, Dr Sunil Mehendiratta

### 3.00 – 3.20 pm  
**Jaundice in a child……… When to worry**  
**Speaker**: Dr Neelam Mohan  
**Chair**: Dr Anupam Sibal , Dr V K Goyal

### 3.20 pm - 5.15 pm  
**Recent vaccines - New Research and Evidence :**

- **3.20 pm – 3.50 pm**  
  **HPV Vaccine**  
  **Speaker**: Dr Subhash Agarwal  
  **Chair**: Dr Anju Aggarwal

- **3.50 pm – 4.20 pm**  
  **Pneumococcal Vaccine**  
  **Speaker**: Dr Krishan Chugh  
  **Chair**: Dr G R Sethi, Dr K C Aggarwal

- **4.20 pm – 4.50 pm**  
  **Rotavirus Vaccine**  
  **Speaker**: Dr Pankaj Garg  
  **Chair**: Dr Arvind Taneja, Dr J S Bhasin

- **4.50 pm – 5.05 pm**  
  **Influenza Vaccine**  
  **Speaker**: Dr P S Narang  
  **Chair**: Dr A P Dubey, Dr J P singh

### Open house (5.05pm – 5.15 pm)  
**Questions from the Delegates on Vaccination (on any Vaccine)**

### 5.15 pm vote of thanks

**Tea**
FROM MINISTER OF HEALTH & FAMILY WELFARE, GOVERNMENT OF INDIA

Dear Dr. Vasudev ji,

I acknowledge the receipt of your letter dated 26th July 2011 inviting the Hon’ble Minister for Health and Family Welfare to the golden Jubilee Conference of Indian Academy of Pediatrics, Delhi to be held on Sunday, the 28th August, 2011 at the Indian Habitat Centre, New Delhi.

It would have been a pleasure for him to attend the function but due to inevitable prior commitments he regrets his inability to attend the function. However, he has conveyed his best wishes for the success of the celebration.

With regards,

Yours sincerely,

(V.S. RAMACHANDRAN)
MESSAGE

I am glad to learn that Indian Academy of Pediatrics Delhi State Branch is celebrating its “Golden Jubilee” and a Conference on the theme of “Pediatric Care 2011 – The Changing Paradigm” is being organized on 28th August, 2011 at New Delhi.

The health services for children and newborn is crucial and it must meet high standards to bring down child death rate. I am sure that the Conference will go a long way in providing a blueprint of effective pediatrics healthcare.

My best wishes for the success of entire endeavour.

(SHEILA DIKSHIT)
MESSAGE

It gives me immense pleasure to know that the Indian Academy of Pediatrics, Delhi State Branch is celebrating its Golden Jubilee and has also decided to organize a Conference on the theme “Pediatric Care 2011...The Changing Paradigm” on 28th August, 2011 at Indira Habitat Centre, New Delhi. It gives me added pleasure to know that a souvenir is also being brought out on the occasion.

I am sure that the conference will be able to deliberate on all issues relating to Child & Newborn care. It is essential to provide a foolproof child healthcare infrastructure to contain child mortality rate. I do hope that the participants have fruitful discussions to update their knowledge on the subject.

I convey my best wishes for the success of the Golden Jubilee Conference and also wish for the successful publication of souvenir.

(DR. A.K. WALIA)

Dr. A.S. Vasudev,
President & Chairman Org. Committee,
Indian Academy of Pediatrics, Delhi
113-114, First Floor, Bank House (P&S Bank),
21, Rajendra Place, New Delhi - 110008.
MESSAGE

Dear Friends,

It is my pleasure and privilege to write a message for the Souvenir to be released in connection with the prestigious Golden Jubilee Conference of IAP Delhi State Branch on 28th August 2011.

Let me at the outset congratulate Dr Anand Vasudev, President of Delhi State Branch of IAP and Organizing Chairman, the organizing Secretary, Souvenir Editor and all members of the organizing team for releasing this souvenir in connection with this State Conference. Also I am very happy that you have selected very good topics for the CME and I think this conference will be very useful for Academicians, Practicing Pediatricians and postgraduate students as well. I wish this Conference and Souvenir all the best.

My dream project for this IAP year is – Family Benefit Scheme (FBS IAP). Even though IAP is doing a lot of work for child survival, this is the first programme for the benefit of the family of IAP members. CIAP has approved the implementation of FBS, to provide financial help to the families of its members in the event of death of the member. FBS is “for the members, by the members, and of the members” a mutually beneficial compassionate and benevolent scheme with Hyderabad as its headquarters. This scheme is launched at Hyderabad on 27th March 2011. Let us all join together and make this programme a grand success, then we will be able to present a cheque of minimum 10 lakh rupees to the family of the member who expires. I request all my fellow paediatrician of IAP to be members of the scheme.

I also request all the IAPians to take active interest in my action plans like Neonatal Hearing Screening Program, Poor Scholastic Performance Program, Allergic Rhinitis & Co morbidities training Program, Child Friendly School Initiative, Anti Tobacco Campaign, Asthma Training Module etc.

I wish this Conference & Souvenir all the best.

Dr. T. U. Sukumaran
National President IAP -2011
Message for Delhi State IAP Branch

It gives me immense pleasure and honor as well to congratulate Dr. Anand Vasudev, Dr. Anil Bajaj, Dr. G. P. Kaushal and all the Executive Board members of Delhi State Branch on the celebration of Golden Year of Delhi IAP through this Annual Scientific Conference.

“Delhi IAP” has undoubtedly academic prowess which is been unchallenged and recognized throughout the country. It indeed may be considered as “gold standard” in the field of pediatric science. It has given innumerable academic wizards to the IAP and the country as well.

I am amused to know that Delhi IAP is one year elder to the Central IAP. I am indeed privileged to get invited for the prestigious conference and the Golden Year celebrations.

I wish and pray to the almighty to bestow his choicest blessings upon this conference and upon Delhi IAP for grand success and prosperity to the Delhi State IAP.

Dr. Rohit C. Agrawal
President Elect IAP – 2011
Chairperson – IAP ID Chapter
Chairperson – IAP COI
MESSAGE

Dear Esteemed Members of IAP Delhi, Colleagues and Friends,

Greeting from Indian Academy of Pediatrics, Delhi!

It gives me immense pleasure to communicate with you, through this Souvenir, being brought out on the occasion of Golden Jubilee Conference of IAP Delhi “Pediatric Care 2011….The Changing Paradigm” on August 28, 2011 at India Habitat Centre, New Delhi.

IAP Delhi is celebrating its Golden Jubilee this year and it is proud privilege for me to be the President of the Academy in this prestigious year.

IAP Delhi has come a long way since its inception in 1961, and currently has a strength of about 1200 members. The branch has risen to great heights in all aspects, including academics. Our predecessors have toiled hard to bring the branch to its present status and we should all strive to keep the flag flying high.

Since the beginning of the year, we have been planning to celebrate this special year in a special way.

The monthly clinical meetings have been restructured and this has shown good response in attendance.

We had planned to hold a Golden Jubilee conference, and this is happening now. To continue the celebrations, a gala socio-cultural evening with families will be held later in the year. Apart from these, IAP Delhi will collaborate with other organizations in many academic programs.

In this Golden Jubilee Year, we plan to roll out some community programs under IAP Delhi, which will further enhance its image and presence in the city of Delhi. Our aim is to reach out to the community through THE CHILD. The slogan coined for the year is ‘Healthy Living, Healthy Child, Healthier Nation’. We have plans to initiate a Child Friendly School Initiative (CFSI), an Anti Tobacco Campaign in schools and a Poor Scholastic Performance Program, and these will continue over the next 3 years or more.

I request each and every member of IAP Delhi to enroll and participate in these activities.

Your support and patronage will strengthen our endeavor to effectively touch the lives of our young citizens and help achieve our mission of developing a healthier INDIA through a healthy CHILD.

I would like to thank all the Office Bearers specially Dr. Anil Bajaj and all the Executive Board members and all those who have put in their untiring efforts in making this conference a success.

Warmly

Dr A.S. Vasudev
President, IAP Delhi State
Executive Director IAP Central Office 2011
Sr Consultant, Pediatric Nephrologist.
New Delhi
Respected Fellow Academicians,

Greetings from the Central IAP Office!

It is an honour for me to extend my good wishes to the IAP Delhi State Branch for organizing Golden Jubilee Conference on Pediatric Care 2011 to be held on 20th August 2011 at New Delhi.

IAP Delhi State Branch has always been known for its dynamic activities and I am sure that the upcoming conference will also be a real scientific feast for all the delegates, covering many important aspects of child health.

I humbly and sincerely wish IAP Delhi State Branch all the best for its conference, and wish to express that the Central IAP is extremely proud of the IAP Delhi State Branch for all its activities.

With warm regards to one and all.

Yours sincerely,

Dr. Tanmay Amladi
Hony. Secretary General
MESSAGE

August 18, 2011

Dear brothers and sisters of IAP Delhi,

I express my deep appreciation and extend my sincere congratulations to all of you for organizing the Golden Jubilee conference of your illustrious branch. A branch is given birth to, nurtured and given shape and form by several mentors and members with years of hard toil and parental care. It is indeed a proud moment for all members of IAP Delhi who have contributed to its growth, and for those members who have participated in the activities organized by the branch down the years.

I am certain that with the sincere and untiring efforts of the current office bearers of IAP Delhi, and the organizers of the Golden Jubilee conference, the event will be successful if achieving its objectives of providing good science for its delegates, and in making them feel comfortable and well-hosted with the infrastructural arrangements.

I congratulate the office bearers and members of IAP Delhi once again, and wish the Golden Jubilee conference to be a thumping success.

Sincerely,

Sailesh Gupta
Treasurer, Central IAP Office
MESSAGE

Dear Colleagues,

I come to know that IAP Delhi Branch is organizing a conference with the theme of “Pediatric Care 2011....The Changing Paradigm” at India Habitat Centre at New Delhi on Sunday 28th August, 2011 planned as one of the activities on the eve of “IAP- Delhi Branch” celebrating its “Golden Jubilee” this year, 2011. I understand that to make the event memorable, a “Golden Jubilee Souvenir” is brought out. I deem it to be a great privilege to send my greetings.

At the outset let me congratulate the members of “IAP Delhi Branch” for their contribution towards betterment of the IAP and for the service to the pediatric community of our country over a century. I hope this conference apart from quenching the thirst of knowledge of pediatricians, will be a memorable one for the years to come.

I wish the conference a grand success.

Yours in Academy Service

Dr. K. Nedunchelian
Editor-in-Chief,
MESSAGE

It gives me immense pleasure to note that our Academy-The IAP-has reached the stage of Golden Jubilee. During all these years, our peers and other members have worked endlessly and tirelessly to help achieve this milestone. Many hurdles have been crossed.

Let us hope in the years to come, we would be able to achieve a one point figure for neonatal and infant mortality and be able to do away to a greater extent child morbidity due to avoidable reasons.

Deaths due to malnutrition will be negligible; our attention should be on tribal children who have no food or help. They are the ones who have lost a great deal in our prosperous years.

I have no doubt that our enlightened and enthusiastic members in the academy would tackle this formidable problem with understanding, sympathy and fortitude so that the tribals really gain.

Dr. Satya Gupta
MESSAGE

Dear Dr Bajaj

It is heartening to note that the Delhi Branch of Indian Academy of Pediatrics is celebrating its Golden Jubilee this year and the executive has planned year long activities to commemorate the occasion.

Over the years the Delhi Branch has been in the forefront of the IAP activities. In fact the association in Delhi came into existence much earlier than the Indian Academy of Pediatrics and the Delhi stalwarts at that time played a pivotal role in establishing the Indian Academy of Pediatrics at the National level in the year 1962.

Over the years the members of Delhi Branch have played a key role in academic activities of IAP, especially as the editorship of the Indian Pediatrics has vested in Delhi ever since its inception.

The Indian Academy of Pediatrics over the years has been somewhat less focused in defining goals and standards for Children in India. I think we in Delhi are uniquely placed to provide directions and leadership in this direction as Delhi is not only the capital city but also a ‘state’ with a responsive government. In the years to come we must endeavor to increase our interaction with the State (and Central) Government and help define standards of Child care in India which need to be available to all the children of the country.

I wish the Golden Jubilee celebrations a great success.

Dr. S. K. Mittal
Past President IAP, Delhi
Dear

Dr. A.S. Vasudev
Chairman, Organizing Committee
Golden Jubilee conference of IAP Delhi

Date 4 Aug 2011

MESSAGE

Sir,

GOLDEN JUBILEE CONFERENCE OF IAP DELHI

Kindly accept my heartfelt congratulations on the golden jubilee celebration of IAP Delhi.

I congratulate IAP Delhi on reaching the coveted milestone of golden jubilee. Having a rich past full of achievements, I see IAP Delhi having a very bright future and is sure of accomplishing many more academic milestones.

I am sure that the branch will further encourage the bonding of brotherhood between fellow pediatricians.

I wish the conference a grand success.

Dr. M. P. Jain
Joint Secretary-CIAP-2011
Organizing Secretary Pedicon 2012,
Gurgaon.
MESSAGE

I am extremely happy to learn that Delhi Branch of Indian Academy of Pediatrics is celebrating the Golden Jubilee year. It is heartening to note that Delhi IAP has been organizing several activities for the past one year to make the Golden Jubilee year a great success. Delhi IAP is a vibrant body of dedicated pediatricians from Delhi and NCR area and is doing excellent work in the field of pediatric education, advocacy for improving pediatric care in the country and community services. In view of large numbers of under five mortality especially in urban slums and lack of proper health infrastructure, IAP Delhi should come into the forefront and help the Government of Delhi in implementation of the programs related to child health. I urge every IAP member to dedicate at least half a day from their busy schedule and provide free service for the underprivileged and poor children of our society.

Delhi branch of IAP has done excellent work all through out its inception and important National program like Polio eradication with Pulse polio concept was started by our senior IAP members. The new generation of pediatricians under the banner of IAP, Delhi should similarly think of some innovative program which can improve the child health indices of Delhi.

I whole heartedly appreciate the effort made by Dr. A.S. Vasudev and Dr. Anil Bajaj, the dynamic President and secretary of Delhi IAP and all the executive members for the excellent work rendered during their tenure.

I wish the Golden jubilee celebration of Delhi IAP a great success

Dr. A.K. Datta
Director Professor and Head
Department of Pediatrics
Kalawati Saran Children's Hospital &
Vice Principal,
Lady Hardinge Medical College,
Past President Delhi IAP
New Delhi 110001
MESSAGE

I am very glad to note that Indian Academy of Pediatrics (IAP), Delhi is bringing out a Souvenir in the Golden Jubilee Year of the branch. IAP Delhi from its inception has remained in the forefront for various academic activities in the last 50 years. It has produced many stalwarts who has made it proud working on various important positions in IAP like President of Central IAP, Editor-in-Chief of Indian Pediatrics, the official journal of IAP, Vice President of IAP and has hosted many National and International Conferences. I really feel proud to be associated with such a vibrant body as its President in the year 2005. I wish all success for the future of Delhi IAP and send my Best Wishes for the success of its “Golden Year Jubilee Conference” on “Pediatric Care 2011.”

Dr. A. P. Dubey
Director-Professor & Head
Department of Pediatrics
MAMC & LNJP Hospital,
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The Changing Paradigm

Venue: India Habitat Centre, Lodhi Road, New Delhi
MESSAGE

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Golden Jubilee is a milestone and a defining moment for Delhi-IAP. It is an occasion to feel proud for the achievements thus far – and we have a great deal to show in that regard. More significantly, it is an occasion to reflect on the future.

Pediatrics as a discipline is poised for transformational change because child health is in transition: from communicable diseases to non-communicable diseases; from problems of poverty to problems of plenty; from survival to intact survival; and from piecemeal priorities to holistic wellness. The change is also imminent in many other ways: expectations of families from pediatrician are different and greater, cost of care is escalating, technological advances are triggering rapid professional calibration, specialties are emerging and growing, and the accountability of the profession is increasing by the day.

We should ponder on what should be the role of our organization for the next half century? What seeds should we sow today to ensure that we continue to serve the cause of children effectively? We should make sure that our organization remains ever-more relevant in coming years. Delhi – IAP also has the unique responsibility to remain in the forefront of advocacy for children since we are located to the capital of India.

I suggest that we undertake an exercise to develop Vision 2030 during the course of the Golden Jubilee year.

My best wishes for the Golden Jubilee Conference of IAP-Delhi.

Dr. Vinod K. Paul
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Professor & Head
MESSAGE

Respected seniors & esteemed IAP Delhi members,

IAP Delhi is celebrating its golden jubilee this year (1961-2011). IAP Delhi is now 50 years young.

It makes me proud to be working for our esteemed organization in its golden jubilee year.

On this day we remember the contribution made by our esteemed seniors in last 50 years to make it such a vibrant body with high standards in academics.

We are now ready to take more programmes for connecting to community & contribute to the society at large in a positive way. Till now we have been active with updating with state of art of medical profession, though there are individual attempts at helping poor patients. Time has come that IAP Delhi comes to fore front & does work for children & not educating pediatricians only. We also have to document the work so that meaningful Indian data is generated which can be used ultimately in policy making to help raising health standards of children of India.

We also have to work in a way so that many of our colleagues who are not members of IAP Delhi join us as members so that whatever programs IAP Delhi is doing, reaches more pediatricians & our numbers also swell. This will give us more voice & ultimately we reach more number of children for our future programs for community being planned.

For being inclusive we have to network with government & non government organizations, many other national & international agencies so that we partner & draw on each other's strengths & work in symbiotic manner for the welfare of children.

IAP Delhi needs to be more vocal for the cause of children, we have to do active advocacy.

We have started moving on these fronts. Under the guidance of Dr Vasudev, our untiring president, some length has been covered. You will appreciate the movements as time progresses. Many new ideas are germinating with long term programmes.

This year Dr Vasudev has already brought about a change in our monthly meetings & it has been welcomed by members at large. The result is a pleasing increase in attendance at these meetings.

In the end I would like to thank our president Dr A. S. Vasudev for his mature guidance & active help from our Joint Secretary, Dr G P Kaushal, all executive members & office staff of IAP Delhi who are giving their best for success of this conference.

Yours truly,

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MESSAGE

Golden Jubilee year is a special year in the journey of any body's life and in the history of an association. **IAP-DELHI** founded in the year 1961, has been led by great academicians, leaders, visionaries and achievers, whose constant guidance, endless and untiring efforts have brought many laurels to the branch. Being one of the office bearers in the Golden jubilee year of the branch is a great experience and matter of pride for me. This year myriad of events of the branch including “Theme Based” CMEs has been really an attractive and innovative venture by the office bearers. Dr.Vasudev and Dr. Anil Bajaj have really worked hard to make this year really a special year in the history of branch. Kudos to them!

Annual Academic Conference SOUVENIR of this year contain messages and good wishes from Statuary dignitaries and Seniors whose blessings and guidance matter the most for the branch. “Pediatric Endocrinology” the official quarterly academic bulletin is also incorporated in the Souvenir and common endocrinal conditions/problems have been published so as to enable the members have an insight in to these.

JUST a stimulating thought:-

“Deep within man dwell, those slumbering powers; powers that would astonish him, that he never dreamed of possessing; for that would revolutionize his life if aroused and put to action.(Orison Sweet Marden)

Peak performers as opposed to the weak performers have trained themselves to shape events of their life rather than being shaped by them. Recognizing one’s own limitations and over coming, the same by immeasurable and vast potential that one possess is the most simple way to achieve great things and life filled with joy and pure bliss.

With Warm Regards!

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Thyroid Disorders in Day to Day Practice

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Whether the child is short or tall, fat or thin, lethargic or hyperactive, has goiter or not, thyroid dysfunction is often thought of. With the easy availability of lab testing for thyroid hormones (TH), sometimes patients get the test done themselves, or labs offer the tests as part of package deals. Newborn thyroid screening is also fortunately getting done in more centers.

1. **Newborn thyroid screening:** should it be done only in babies born to mothers with thyroid dysfunction? Certainly not! Screening should be done in EACH AND EVERY newborn. Congenital hypothyroidism (CH) is very common (1: 1100 – 1: 3000 newborns), almost impossible to pick up clinically (95% affected newborns look normal), easy to detect, very easy to treat, and very devastating to miss (results in permanent mental retardation). If only CH is to be looked for, then the easiest way to do it is by testing cord blood (sampled from the placental end) for TSH. If other tests are to be added, then the baby must be 2-3 days old, so heel prick sampling on a filter paper can be done. This does make it more expensive, technically demanding, and prone to error.

2. **Normal ranges in the newborn period and later life:** At birth the newborn experiences a sharp thyroid surge. TSH rises rapidly, up to 50 uIU/ml, followed by a rise in T4; then gradually both fall to baseline over 2-3 days. So the cutoffs for the first 72 hours of life for TSH and T4 are much higher, e.g. TSH even up to 50 may be normal at 24 hr of life. Broadly, a TSH up to 25 uIU/ ml in the cord blood is normal, though some use a cut off of even 30. By day five of life, the TSH settles to <10 uIU/ml. By 10-14 weeks of life the TSH settles to the adult range, i.e. < 5 uIU/ml. These values should be kept in mind, so that the infant is not advised unnecessary repeat testing or treatment for say, a TSH of 8 at the age of 3 weeks, unless the T4 is low.

3. **In the preterm baby:** the pattern of the thyroid surge follows the same time pattern, but the peaks achieved may be somewhat lower. When in doubt, TH should be repeated in a week’s time, so replacement is not unduly delayed. At the same time, unnecessary thyroxin does not improve outcomes, and should be avoided if the sick euthyroid syndrome is likely to be present.

4. **When should confirmatory repeat TH testing be done?** Many pediatricians, confronted with a raised TSH, asked for a repeat after 2-4 weeks. This is wrong! If the baby does have CH, treatment should be started as soon as possible, since IQ starts dropping if replacement does not begin by the age of 2 weeks. So please make sure each newborn’s TSH report is ready at discharge from the hospital, advise a repeat test in a venous sample for TSH and T4 if the report is abnormal, and start replacement once the CH is confirmed.

5. **Other tests to be done if CH is diagnosed:** If it is convenient, a thyroid scan and ultrasound should be done before starting replacement, or even within a day or two. This can help make an etiologic diagnosis, e.g. agenesis/hypoplasia/ectopia. Treatment should never be delayed for these tests, so if imaging is not immediately available, replacement should be started without imaging.

6. **Replacement needs in the newborn:** Often the newborn is started on 12.5 or 25 mcg of thyroxin. This may be inadequate, as the newborn can need as much as 12-15 mcg/ kg/ day, in contrast with older children or adults, who need just 1-3 mcg/ kg/ day. Adequacy of the dose requires careful monitoring. Remember that TSH CHANGES VERY SLOWLY, taking as much as 4-5 WEEKS TO STABILIZE completely. So initially, test the infant’s T4 every 2 weeks, to make sure the dose is not too much or too little. Check the TSH only after 4-6 weeks. Thereafter, monitoring of T4, TSH and growth should be done every 2-3 months in the first 2 years of life. If the T4 is in the upper half of the normal range, and the TSH is less but not normal, continue the same dose: occasionally the TSH may take up to a year to normalize.

7. **Confirming permanancy:** As much as 50% of CH may be transient, so at the age of 3 years, thyroxin should be stopped for 5-6 weeks, and TH testing and imaging repeated. Testing should not be done too early, e.g. within 4 weeks of stopping. Replacement should be restarted if the TSH rises, and the need for lifelong replacement emphasized to the parents.

8. **Indications for TH testing:** include slow height gain, delayed puberty, precocious thelarche or menarche, chronic anemia, chronic constipation,
poor scholastic performance, irregular periods or menorrhagia, unexplained weight loss, weight gain in a child who is short/ slowly growing, goiter, lipid abnormalities, hyperprolactinemia, etc. In children with autoimmune diseases like type 1 diabetes, vitiligo, celiac disease, pernicious anemia; polyendocrine failure syndromes (type 1 and 2); family history of thyroid disorders; certain medications (see Subclinical Hypothyroidism below); or conditions such as Down syndrome, thyroid function should be tested periodically. Overweight children who are tall for age (and midparental height: MPH) are unlikely to be hypothyroid.

9. **Tests needed for screening**: Whether in the newborn, or the older child, an ultrasensitive TSH alone is usually sufficient for screening for thyroid dysfunction and for monitoring therapy. However, often additional testing of T4 can be useful (as above). In situations where pituitary dysfunction may be present, e.g. a very short child, thalassemia, etc., T4 must be tested with TSH. Serum T3 rarely provides useful information, and may in fact cause confusion. For example, in early hypothyroidism or in iodine deficiency, increased TH causes increased conversion of T4 to T3 as a compensatory mechanism, so the T3 may be high! Please do not order T3 routinely! Thyroid peroxidase antibodies do not alter management in overt primary hypothyroidism, and there is NO role for serial testing or advising corticosteroids even if strongly positive. However, antibodies may be useful in subclinical hypothyroidism.

10. **Primary Hypothyroidism**: is characterized by low T4 and raised TSH. It may be overt (low T4 and raised TSH) or subclinical (normal T4, raised TSH). In childhood, primary hypothyroidism is usually due to chronic lymphocytic thyroiditis (CLT). In **central hypothyroidism**, T4 is low, but TSH can be low, normal, or even slightly raised (bioinactive TSH).

11. **Giving the thyroxin correctly**: The entire dose should be given once daily, as the half life is 72 hours. It is not necessary to give thyroxin in the fasting state; what is important is the timing should be consistent. Make sure substances which interfere with absorption, e.g. iron, soya or calcium, are not given within 4-8 hours of administering thyroxin. Drugs such as carbemazepine, phenytoin, rifampicin, estrogen, may increase the metabolism and thus the dose of thyroxin. Otherwise, higher doses should raise concerns about compliance, medicine quality, and lab errors. Long periods of over-treatment with thyroxin interfere with bone accrual, and can increase the risk of osteoporosis in later life.

12. If multiple endocrine deficiencies exist together, it is important to replace cortisol before starting thyroxin, otherwise an adrenal crisis can be precipitated.

13. **Hyperthyroidism**: is uncommon in young children, and almost always has associated goiter. It could be due to Graves’ disease (over-production of TH), or Hashimoto thyroiditis (increased release of preformed TH). Very rare causes include toxic nodule, McCune Albright syndrome, familial non-autoimmune hyperthyroidism (FNH), or external administration of thyroxin or high doses of iodine. The characteristic profile is low TSH, with high T4 and T3. Here T3 levels may be useful: in T3 toxicosis, T4 may be normal, but T3 is high. A thyroid scan is useful in distinguishing Graves’ disease (uniformly enlarged gland with increased uptake) from thyroiditis (patchy and decreased uptake), or toxic solitary nodule.

14. **Treating Graves’ disease**: requires neomercazole or radio-iodine ablation (rarely surgery). Only symptomatic treatment (e.g. beta blockers) suffices for thyroiditis. Propylthiouracil (PTU) is associated with a higher risk of liver cell failure than NMZ.

15. **Goiter** could be due to iodine deficiency, autoimmune disease (especially CLT or Graves’ disease), or rarely drugs, dyshormonogenesis, nodules, or malignancy. Small goiter during puberty, with normal TH levels, needs no treatment. If it is significant or increasing, ultrasound, TH, and thyroid peroxidase antibodies should be tested, and low dose thyroxin given for 6-12 months to try and reduce gland size. If there is no response, thyroxin should be stopped and TH monitored periodically.

16. **Sources of confusion in interpreting reports**:

(a) One may see a high TSH along with a high T4. This can happens if the thyroxin was missed in the preceding weeks, and just before the test a larger dose than advised is given to the child. While the high dose raises the T4, there was not enough time for the TSH to normalize. In this situation, better compliance should be emphasized, rather than increasing the dose.

(b) One may see a normal TSH, with low normal T4. If the test was done mid afternoon or evening, the diurnal variation in T4 levels may bring it to a low level. Dose increase is not needed.

(c) **Subclinical hypothyroidism** – borderline high TSH (5-10 uIU/ml) with normal T4. If the child is obese and tall for age and MPH, this may be due to just obesity. Treatment with thyroxin is not warranted for simple obesity. If the child is asymptomatic, subclinical hypothyroidism is a very controversial indication for treatment. Most experts agree that if the height is appropriate for age and MPH, and antibody levels are negative, do not start thyroxin; instead follow up carefully, with growth velocity and repeat TH after 3-6 months. Drugs which can increase TSH (iodine administration or iodine containing compounds like topical antiseptics, contrast agents, expectorants; anti-epileptics like valproate; etc.) should be asked for.

(d) **Central hypothyroidism** – is characterized by low T4 and normal or low TSH. In these children, only T4 should be ordered during follow up and used for dose adjustment; TSH should NOT be asked for, as it just creates confusion.
(e) **Severe non-thyroidal illness:** Low T4 and low TSH can be seen. Thyroxin replacement is not usually called for.

(f) **Graves’ disease:** on treatment, initially TSH may remain low for many months; if the T4 has normalized, the dose of NMZ need not be changed. Later, of course, TSH is the better indicator of adequacy of treatment.

In summary, ALL newborns should be tested (preferably cord blood) with TSH and if possible, T4. Replacement should be started early, in adequate doses (usually 50 mcg/day). In older children, TH should be tested with a high index of suspicion, as symptoms can be varied. Thyroxin should not be given unless the indication is clear cut. Careful monitoring and proper treatment are important for a good outcome.
Serum calcium exists in equilibrium with calcium present in extra-cellular and intra-cellular spaces and that in the recently accumulated surface bone. Though accounting for only 1% of total body calcium, these calcium pools are responsible for modulating vital body functions including neural transmission, blood clotting, intra-cellular signal transduction, maintenance of cardiac rhythm, muscle contractility etc. Thus, not surprisingly, serum levels of calcium, especially that of its metabolically active ionized form, is tightly regulated. The mechanisms involved in regulation of serum calcium levels involve calcium sensing receptor (CaSR), PTH and its receptor, vitamin D hormone system and calcitonin.

Definition of hypocalcemia:
Hypocalcemia is defined as total serum calcium level <7 mg% in a pre-term newborn, <8 mg% in a full-term newborn and <8.8 mg% in post-neonatal age group. For Ca²⁺, the corresponding figures are <4.4 mg% for newborns and <4.7 mg% for children beyond neonatal age group.

Causes of hypocalcemia:
Hypocalcemia is most commonly observed in the neonatal period and early infancy. In the neonate there is a physiological dip in serum calcium levels soon after birth. Various situations commonly encountered in the neonatal period exaggerate this dip leading to transient hypocalcemia. Causes of hypocalcemia are best seen in relation to age at presentation.

I. Causes of hypocalcemia in infants <72 hrs:
1. Neonatal illness:
   - Prematurity
   - Birth asphyxia
   - Intra-uterine growth retardation
   - Sepsis
   - Respiratory distress
   - Citrated blood/alkali
   - Photo-therapy
2. Maternal illness:
   - Diabetes mellitus
   - Toxemia of pregnancy
   - Hyper-parathyroidism

II. Causes of hypocalcemia in infants >72 hrs:
1. High dietary phosphate load
2. Maternal vitamin D deficiency
3. Hypomagnesemia: transient/metabolic disorder
4. Hypo-parathyroidism; transient/permanent
5. Chronic renal insufficiency
6. Pseudo-hypoparathyroidism

III. Causes of hypocalcemia in older children/adolescents:
1. Parathyroid related:
   - Various causes of hypo-parathyroidism: congenital/acquired
   - Target organ resistance: hypomagnesemia or pseudo-hypoparathyroidism
2. Vitamin D related:
   - Deficiency of vitamin D
   - Increased losses: anti-epileptics
   - Metabolic defects in vitamin D action
   - End organ resistance

Clinical features of hypocalcemia:
Early neonatal hypocalcemia is often asymptomatic. When present, the symptoms are neither specific for hypocalcemia, nor related to its severity. These include jitteriness, irritability, tremulousness of limbs, focal or generalized seizures. Other uncommon features include stridor due to laryngospasm, apnea and cyanosis and disturbances of cardiac rhythm. On the other hand, late neonatal hypocalcemia is often symptomatic and presents in full term, usually healthy newborns towards the end of 1st week of life or beyond, with convulsions. Older children with hypocalcemia may have numbness, tingling in circum-oral region, fingers or toes or frank tetany. Chronic hypocalcemia may lead to dental hypoplasia, basal ganglia calcification and cataract if associated with hyperphosphatemia (as in hypoparathyroidism or PTH resistance), and skeletal changes of rickets if associated with hypophosphatemia (as in vitamin D deficiency). Clinical examination may reveal twitching of ipsi-lateral angle of mouth on gently tapping the facial nerve just below the maxilla (Chvostek sign). Carpo-pedal spasm may be elicited by inflating a blood pressure cuff around an arm and maintaining pressure at 10 mm Hg above systolic for 3 minutes (Trousseau sign)

Diagnostic approach to a case with hypocalcemia:

Important issues in history:

Neonate:
Age at onset of symptoms (early/late hypocalcemia), gestational age (prematurity), peri-natal events (birth asphyxia, jaundice, respiratory distress etc), type of feeding (high phosphate load), maternal illness (diabetes, hypoparathyroidism, toxemia) and drug intake, family H/O hypocalcemic seizures (hypoparathyroidism), Older child:
Age, clinical symptoms, H/O chronic disease like renal failure, mal-absorption states, thalassemia, other endocrine disorders (auto-immune poly-glandular syndrome), H/O drug intake (anti-epileptics), surgery, irradiation (parathyroid gland destruction), repeated infections with cardiac disease (Di-george syndrome), family H/O seizures/hypocalcemia (hypoparathyroidism, auto-immune poly-glandular syndrome, pseudo-
Examination:

Newborn:
Gestation, systemic sickness, facial dysmorphism, other malformations specially cardiac Di-george syndrome

Older child:
Rickets, features suggestive of other chronic disease: anemia, hypertension, short stature (renal failure), jaundice (liver disease), alopecia, oral candidiasis/ectodermal dystrophy (auto-immune polyglandular syndrome), heart disease (Di-george syndrome), cataract, brachydactyly, round face with obesity (Albrights hereditary osteo-dystrophy)

Investigations:
All cases: Serum tCa, Ca²⁺, Mg, P, ALP, total protein, albumin.
Venous blood gas analysis, kidney function test, X-ray chest (thymic shadow, cardiac disease).
Older children: X-ray wrist, LFT.
Second line investigations (required in some cases depending upon initial evaluation):
Serum PTH, 25 OHD, 1,25 (OH)₂D
Maternal Ca, P, PTH, ( for neonatal hypocalcemia only)
Urinary Ca/Cr ratio, P, creatinine

Characteristic biochemical features of important disorders leading to hypocalcemia are as follows:
- ↓ Ca, ↓ P, ↓ PTH, ↓ ALP, ↓ 25OHD: Vitamin D deficiency
- ↓ Ca, ↓ P, ↓ creatinine, ↓ Renal failure
- ↓ Ca, ↓ P, ↓ creatinine, ↓ PTH: Hypoparathyroidism
- ↓ Ca, ↓ P, ↓ PTH, ↓ Mg: Hypomagnesemia
- ↓ Ca, ↓ P, ↓ creatinine, ↓ PTH: High dietary P load /PHP

Treatment of a child with hypocalcemia:
Asymptomatic hypocalcemia in the newborn should be treated at tCa level <6mg% in PT and <7mg% in FT infant with oral calcium supplement 75 mg/kg/day given 6 hourly for 3-4 weeks. Patients with hypocalcemic seizures/tetany require therapy with I/V 10% calcium gluconate. The dose required is 0.2-0.5 ml/kg in older children (maximum 10 ml) and up to 2 ml/kg in neonates. The injection is given slowly under cardiac monitoring over a period of 10-20 minutes. This is followed by infusion of calcium gluconate @ 50mg/kg/day for neonates or 20 mg/kg/day for older infants and children titrated to achieve eucalcaemia monitored 12 hourly. Once eucalcemia is achieved the infusion is tapered over a period of 48 hours with simultaneous introduction of oral calcium 50-75 mg/kg/day (upto 100 mg/kg/day) in neonates.

Further treatment depends upon underlying etiology. Newborns with early neonatal hypocalcemia usually require no further therapy. Most patients with hypomagnesemia, apart from those rare subjects with primary hypomagnesemia/ Mg wasting states, respond well to two doses of 50% magnesium sulphate 0.2 ml/kg given 12-24 hours apart. Patients with hypo-parathyroidism require 20-60 ng/kg/day of calcitriol with oral calcium supplements 30-75 mg/kg/day. These patients require careful, ongoing monitoring of therapy to prevent hypercalcemia and hypercalciuria. Neonates with Vitamin D deficiency require about 1000 IU vitamin D/day (given as 30,000 IU/month) with oral calcium supplements for 2-4 months. Older infants can be given up to 3000 IU/day of vitamin D (a sachet of 60,000 IU every month) for 2-4 months. Older children and adolescents with vitamin D deficiency can be given 3-6 lac IU vitamin D as a single dose or 3000-6000 IU/day for 3-4 months along with calcium supplements. A preventive dose of 400 IU for infants or 1000 IU/day for older children should be given subsequently to prevent recurrence of deficiency and building up of body stores.
Recent trends in use of Growth Hormone Therapy

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Abstract Until the mid 1980's, growth hormone (GH) therapy was only prescribed to treat children with severe growth hormone deficiency (GHD). Today, however, with abundance of recombinant human GH (rhGH), it is used to treat a wide range of conditions. rhGH can be used to treat short stature from GH deficiency (GHD), insufficiency and other disorders leading to poor growth. Currently it is also used for patients with Chronic renal failure (CRF), Turner syndrome (TS), Prader Willi syndrome (PWS), Small for gestational age (SGA) without catch up growth by 2 years, Idiopathic short stature (ISS), SHOX Deficiency, Noonan Syndrome and some Dysmorphic syndromes with short stature. With GH therapy many children can achieve adult height better than the anticipated based on their pretreatment growth pattern.

Key words Growth hormone, Turner syndrome, Growth hormone deficiency, Idiopathic short stature

INTRODUCTION

Human growth hormone (hGH) or somatotropin is a single chain of polypeptide comprising of 191 amino acids, that circulates either complexed to a binding protein or in the unbound (free) state. At all ages, fetal through adult, GH is secreted in an intermittent, pulsatile pattern, largely as a result of reciprocal interactions of two hypothalamic peptides i.e. Growth hormone releasing hormone (GHRH) and somatostatin (SS) or Somatropin release inhibiting factor (SRIF). Growth hormone interacts with its receptor to generate IGF-1 (insulin like growth factor), the main mediator of GH action, in the liver and in most other tissues, including epiphyses. Many of the effects of GH are mediated by IGF-1, which circulates in the plasma, bound to one of a series of binding proteins, called IGFBPs. These proteins circulate and modify IGF-1 action, either as stimulators or as inhibitors. IGFBP-3 is the major circulating form of the binding protein. This complex system subserves the process of growth. At puberty, the pulsatile release of GH is increased 2-3 fold, predominantly by increased amounts of GH released at each secretory episode along with increasing amounts of sex steroid hormones, this accounts for the much of the pubertal growth spurt following which the secretion of GH returns towards prepubertal values.

The process of growth also depends on adequate nutrition, normal bone structure and biochemistry, normal thyroxin levels and other endocrine secretions, as well as general health. Disruption of normal growth may therefore be an indication of many pathologies. After the establishment of the National Pituitary Agency in 1961, pituitary derived GH was used till mid 80s, when its use was prohibited with the emergence of Creuzfeldt- Jacob in 1985. Around the same time recombinant human GH, which was ready for use by the late 80s (1978), was approved by the FDA and introduced for the treatment of GHD in 1985. Subsequently it was found to be beneficial in patients with chronic renal failure and Turner syndrome, a decade later (1995) FDA approved its use for these disorders. Currently GH therapy has been approved by FDA for additional conditions i.e. Prader Willi Syndrome – PWS (2000) , Small for gestation age – SGA without catch up growth by 2 years (2001), Idiopathic short stature – ISS, (2003) SHOX Deficiency(2006) Noonan Syndrome(2007) and some of the Dysmorphic syndromes with short stature.

GROWTH HORMONE DEFICIENCY (GHD)

The classical indication for GH treatment is growth hormone deficiency, irrespective of the underlying cause which leads to the GH deficient state. From a clinical prospective; GHD it can be subdivided on the basis of etiology into 2 categories: organic and isolated idiopathic GHD.

Variability in the diagnosis of GH deficiency remains a clinical challenge and is related to the continuum between severe GHD, insufficiency and normalcy[1-3]. Marked variability in GH assays in the tests used, arbitrary cut offs to define GHD based on stimulation tests have been some of the problems in arriving at the diagnosis of GHD. Prior to proceeding with the investigative evaluation, careful clinical history, clinical and auxologic examination, the relationship among chronologic age, height age, bone age and height evaluation in relation to the relevant population based charts and midparental based target height is important.

The diagnosis of GHD is a challenge in the absence of the classic phenotype. A significant proportion of short, slowly growing children have no overtly obvious clinical features. A three step approach to diagnosis is:

(j) Comprehensive clinical and auxological assessment to differentiate non-endocrine and non-GHD states, and select patients most likely to have GHD (jj) biochemical investigations of the HP-GH axis in carefully selected patients and (iii) neuroimaging to define pituitary morphology [4].

Investigations of the HP-GH axis should
be undertaken in a centre with expertise in pediatric endocrinology. There is no single perfect test to assess the HP-GH axis but peak GH responses are more reproducible with arginine or pyridostigmine which stimulate GH releasing hormone secretion and control endogenous somatostatin tone. The agents most commonly used for the GH provocation test are clonidine and insulin. Insulin is the gold standard for GH provocation test. In addition to GH levels following provocation, the GH-dependent peptides IGF-1 (and IGFBP3 if available) should be measured because low levels of all strongly support a diagnosis of GHD, although normal levels would not exclude a diagnosis. Levels of IGF-1 and IGFBP-3 should be interpreted against age, gender and pubertal stage matched normal ranges. Acute and chronic malnutrition, intercurrent illness or liver disease may all affect IGF-1 levels and complicate interpretation in the context of possible GHD. rhGH in children with GHD provides physiological replacement[5-7]. Titrating the dose of rhGH to maintain IGF-1 levels in the normal range, while normalizing growth can be considered to approximate physiological replacement. Younger age at the beginning of treatment, longer duration of treatment, smaller height deficit at the start of treatment and greater catch up in height in the first year of treatment are an advantage for final height. The height velocity in 1st year of rhGH is 8-12 cm/year Before starting rhGH, sleep studies should be done in all children who have PWS or are obese, and an ENT evaluation in those with a history of snoring and disturbed sleep.

TURNER SYNDROME (TS)

Turner Syndrome occurs in approximately 1/1500 to 2000 of female births and is a common pathological cause of short stature. Of the numerous manifestations recognized, the only consistent ones are short stature and ovarian failure. Although the stature of girls with TS varies considerably, the pattern of growth is characteristic. There is a gradual decline in HV (height velocity) throughout childhood, absence of pubertal growth spurt and adult height deficit of about 20cms.

Girls with TS do not have GH or IGF-1 deficiency, but levels of both are relatively low, particularly during adolescence, which can be attributed to estrogen deficiency and increased adiposity. A degree of GH and IGF1 insensitivity is considered to contribute to growth failure and this forms the basis of treatment with supraphysiological doses of rhGH. Unlike GHD and PWS, GH provocation testing is not required in girls with TS. Much of the defect in height is caused by haploinsufficiency of the short stature Homeobox-containing gene (SHOX) located on the X-chromosome. There are several factors which may influence the effect of GH treatment, age and height at start, GH dose and injection frequency, non compliance, genetic factors, the addition of oxandrolone, the estrogen dose regimen and the timing of puberty induction. The height velocity in 1st year is 5.5-8 cm/year. Overall the safety profile of this treatment is good; however long term follow up of the girls using the supraphysiological doses of growth hormone is required.

SHORT CHILDREN BORN SMALL FOR GESTATIONAL AGE (SGA)

SGA is the term used to describe infants with birth weight and/or length less than -2SD for the gestational age. Up to 90% of children born SGA, experience catch up in linear growth during infancy, have height above -2SD by the first birthday and reach adult height approximately 1 SD below the normal population. Children born SGA require evaluation to identify the underlying cause and also require regular growth monitoring to identify the 10% who do not have significant catch up growth and thus remain exceptionally short [8]. This is more likely in premature infants, severe IUGR and recognized syndromes (e.g., Silver Russel). Multiple factors influence growth in this heterogeneous group of children and a relative resistance to GH and IGF-1 is likely to contribute. There is a growing body of evidence that GH therapy improves final height in short SGA children. In Europe, SGA children aged 4 years or older, with failure to catch up height below -2.5 SD, HV below 0 SD and height SDS more than 1 SD below midparental height are eligible for rhGH treatment. A wide dose range is recommended and higher doses may be considered for a limited period initially in children with marked growth retardation (height below -3 SD). The positive effects of GH therapy extend beyond linear growth and include potentially important effects on body composition, muscle mass and function, bone mass, metabolism, behavior and cognitive function. The height velocity in the 1st year of rhGH is 8-10 cm/year. Regarding the use of GH in Silver Russell Syndrome (RSS) little data is available. Many studies suggest a good response in younger children. Limited data suggest that GH does not exaggerate limb asymmetry.

CHRONIC RENAL INSUFFICIENCY (CRI)

Impaired growth, short stature, delayed puberty and an attenuated pubertal
growth spurt leading to reduced adult height are common in children with CRI. The factors contributing to growth failure are primary renal disorder, uremia, under nutrition, metabolic acidosis and bone disease. The growth outcome, post transplantation is influenced by the dose of corticosteroid, allograft function, age of the child, pubertal status and height deficit at the time of transplantation. Patients with CRF have relative GH insensitivity reflected not only by raised GH levels but also by raised or normal IGF-1 levels and by raised IGFBP-3 levels, reducing IGF-1 bioactivity. Thus high doses of rhGH are recommended to overcome this scenario. Treatment is indicated in those with significant growth impairment (height < 3rd or HV < -2SD) and CRI (GFR < 75 ml/1.73 m² BSA) before or during dialysis or following renal transplantation.

To get the best results, treatment should be initiated at an earlier age and earlier in the course in CRI [9]. Before starting growth hormone therapy, nutritional and metabolic status should be optimized and steroid treatment should be minimized. In children with CRI, initial catch-up growth followed by relatively normal growth and attainment of normal adult height can be anticipated. The height velocity in the 1st year of rhGH is 5.5-8 cm/year.

IDIOPATHIC SHORT STATURE (ISS)

In the last two decades, growth hormone (GH) therapy has expanded to include many children with non-GH deficient short stature such as idiopathic short stature (ISS), skeletal dysplasia, genetic syndromes and other chronic diseases associated with short stature.[10] The term idiopathic short stature is used to describe a child or adolescent with height more than 2 SD below corresponding mean height for given age sex and population group, in whom with current diagnostic tools, no etiological diagnosis can be made.

ISS now appears to be the most common indication for GH treatment. It is difficult to differentiate GHD from ISS with conventional growth hormone testing alone. There are subtle abnormalities of GH secretion and GH sensitivity in patient of ISS. There is no consensus on which all disease must be excluded and how during a diagnostic evaluation a child should be labeled as ISS. GH in a supra physiological dosage generally increases height velocity in children with ISS and increases the adult height up to 7 cm. The average effect on final height is modest. The final gain varies between 5.4 cm to 7.2 cm.

It is difficult to predict the height gain for an individual child. GH injection is well tolerated without significant side effects. However the theoretical risk of unwanted long term sequelae of elevated serum GH and IGF-1 levels have not been evaluated yet. The psychosocial benefits and cost effectiveness need meticulous evaluation to justify GH therapy.

SKELETAL DYSPLASIA (SD)

It is a heterogeneous group of diseases affecting the skeleton. The most prevalent is achondroplasia with an incidence of 1 in 25,000 births. Other skeletal dysplasias include hypochondroplasia, Dyschondrosteosis, congenital spondylephysyal dysplasia, pseudoachondroplasia and many others. Some mucopolysacharidosis like Morquio Syndrome exhibit bone dysplasia. Most of the SD cause moderate to severe disproportionate short stature. Most patient with SD have a normal growth provocative test. The final height differs between various disorders but often in the range of 110-130 cm. The result in skeletal dysplasia with rhGH has been less rewarding. GH therapy does not benefit patients with Achondroplasia much but a subgroup of patient with hypochondroplasia may benefit significantly. Other uncommon forms of skeletal dysplasia have not benefited from GH treatment. X-linked Hypophosphatemic rickets is characterized by rickets, short stature, impaired renal phosphate reabsorption and vitamin D metabolism, most children with XLHR demonstrate reduced height. Poorly growing children benefit from GH therapy.

rhGH is given as a daily subcutaneous injection. A variety of needle and needle free pen devices are available. Parents and older children can learn the injection technique without much difficulty. Although the preference is to administer rhGH in the evening to crudely mimic endogenous GH secretion, the timing can be altered to accommodate family.

The dose of rhGH should be calculated according to the body weight (in obese children according to body surface area) and individualized according to the growth response and IGF-I levels adjusted as the child grows. IGF-1 routines I and IGF-BP-3 levels should be maintained within age dependent normal ranges, bearing in mind that the oncogenic potential is likely to be greatest with long term supra-physiological doses, high IGF-I levels and low IGFBP-3 levels. For patients who show a good response, rhGH is continued until significant further growth is unlikely (HV < 2 cm/year indicates near final adult height) or satisfactory height is attained.

SIDE EFFECTS OF GROWTH HORMONE THERAPY

Recombinant Human Growth hormone (rhGH) has proved to be a safe medication and relatively free of untoward side effects. The reported side effects occur with a frequency of about 2-5% per patient year of treatment. Adverse effects are generally seen in less than 3% of the recipients. As the use of GH has expanded to include other indications such as idiopathic short stature, small for gestational age (SGA) babies and Prader Willi Syndrome, it becomes even more important to continuously monitor the safety of rhGH [11]. After the initiation of therapy with rhGH, transient edema due to fluid retention, transient headaches and even benign intracranial hypertension is reported. BIH is generally reversible with discontinuation of GH treatment. Severe edema and carpal tunnel syndrome are rare in pediatric patients. These effects are usually transient, reverse when treatment is stopped for a short time and generally does not recur on re-initiation of therapy. Enhanced risk of leukemia or brain neoplasia in children without specific risk factors is not proven. GH marginally increases the risk of slipped capital femoral epiphyses in children with GHD, return of limb edema and worsening of kyphoscoliosis in some patients with Turner syndrome. There is some concern about the effects of growth hormone on carbohydrate metabolism in SGA small for gestational age children but frank diabetes is very rare. Recent studies have not substantiated increased
risk of transplant rejection in patients with renal failure. rhGH/IGF-1 may worsen the probability of sleep apnea in patients with Prader-Willi Syndrome, hence careful pretreatment evaluation and monitoring is advocated. Minor side effects such as injection site pain, numbness, redness, swelling, bleeding and sweating at the local site as well as generalized pruritus are reported. Other reported effects include prepubertal gynecomastia and increased growth rate of cutaneous nevi. There are very few published studies done in India, reporting the safety and efficacy of rhGH. The side effects experienced by Indian children were headaches, urticarial rash and local reaction in the form of itching and erythema. rhGH should also be used with caution in Fanconi anemia and Bloom syndrome due to the inherent tendency for malignancy in these conditions.

**FUTURE PROSPECTS**

GH is recommended in catabolic wasting states such as HIV infections. In many conditions GH therapy is being tried on an investigational basis such as cystic fibrosis, steroid dependent states and chronic diseases which retard growth but where the use of GH proved to be safe. Pathophysiology of ISS is gradually being unraveled by the development of new genetic tools.

GH can be used in Thalassemia with short stature. It is still in the experimental stages. The anabolic effects of GH have also led its use in many catabolic states like severe burns, HIV induced cachexia, chronic high dose glucocorticoid treatment, chronic obstructive pulmonary disease, surgery, trauma, cancer, organ failure etc. In severely burnt children, GH has shown to decrease whole body catabolism, increase protein synthesis, accelerate wound healing, and reverse growth arrest. GH is approved by the food and drug administration for administration to adult patients with HIV associated cachexia. The GH treatment in these patients resulted in a positive nitrogen balance, increase lean body mass, decrease body fat, and improved work output.

In the future we anticipate the following things in growth hormone development

1. The availability of GH in weekly doses or monthly doses, instead of daily injections
2. GH being available in dermal patches form, inhaled form and tablet form.
3. GH combined with LHRH analogues for early puberty with short stature.

In future the use of GH is likely to include many more conditions beyond those proven at present. Although generally safe, potential side effects of GH need to be carefully noted. Children receiving GH must be monitored closely by physicians who are experienced in the use of this pharmacological agent.

**REFERENCES**

Approach to a Child with Ambiguous Genitalia

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The birth of a child with ambiguous genitalia is a challenging event to handle, both for the family and the treating pediatrician. It is estimated that genital anomalies occur in 1 in 4500 births. The severe forms with associated medical issues (adrenal crises) usually present in neonatal period however, as this condition is viewed as social stigma in Indian society, presentation may be delayed till childhood or adolescence in a significant proportion of children.

Starting from potential medical emergency in a neonate to an array of unusual and complex issues (medical, psychological, social, ethical and legal) all through out the life, the condition prompts a long-term management strategy that involves a myriad of professionals working with the family.

Revised nomenclature
Several labels have been used to describe such patients (intersex, hermaphrodite, hijras) influenced with conventional beliefs and societal misconceptions. The currently used scientific nomenclature divides these patients based upon morphological features and finding on evaluation. The term Disorders of Sex Development (DSD) is proposed for all such patients, as defined by congenital conditions in which development of chromosomal, gonadal or anatomical sex is atypical.

Who needs evaluation?
Minor variations and malformations in genital morphology are not uncommon in population. The criteria for need of evaluation for DSD in an infant are:

1. Overt genital ambiguity (eg cloacal extrophy).
2. Apparent female genitalia with an enlarged clitoris, posterior labial fusion, or an inguinal/labial mass.
3. Apparent male genitalia with bilateral undescended testes, micropenis, isolated perineal hypospadias or mild hypospadias with undescended testis.
4. A family history of DSD such as complete androgen insensitivity syndrome.
5. Discordance between genital appearance and a prenatal karyotype.

Later presentations in older children and young adults include:
1. Previously unrecognized genital ambiguity.
2. Delayed or incomplete puberty.
3. Inguinal hernia, primary amenorrhea or virilization in a female.
4. Breast development or gross and occasionally cyclic hematuria in a male.

Newborn with DSD
First-line testing in newborns with 46,XX DSD includes:
- Karyotyping with X and Y-specific probe detection.
- Imaging (ultrasound) to look for mullerian structures and localize gonad.
- Serum electrolytes, blood sugar and blood gas analysis.
- Measurement of 17-hydroxyprogesterone, testosterone, cortisol (before and after ACTH stimulation).

For other type of DSDs, initial evaluation includes pelvic imaging, karyotyping and estimation of androgens, estrogen and gonadotropins. Lapsoscopic and histological evaluation of gonads may be required in some cases.

Emergency management of a newborn with suspected or proven salt loosing crises includes:
- IV fluids- 5% dx with NS, 1.5-2 times the maintenance rate.
- Hydrocortisone IV bolus 25mg followed by 15 mg every 6 hrs for first 2-3 days, gradually tapered to 5 mg twice a day.
- Oral FC 100-300 µg/day.

The baby should be discharged on replacement dose of glucocorticoids (hydrocortisone ~15 mg/m2/day) and minirelocorticoids (100-200 µg/day), with stress cover advice and salt replacement.
Management of an older child encompass following

- Establishing the etiological diagnosis and evaluation of psychological make up of the child
- Assignment of the gender.
- Surgical reconstruction to get best possible genital morphology.
- Supplementation of deficient hormones to get desired phenotype.
- Monitoring and follow-up

Establishing the diagnosis

The simplified flow diagrams indicate the stepwise evaluation of a child with DSD with or without palpable gonad.

Evaluation of psychosexual makeup

Psychosexual development is traditionally conceptualized as three components.

- Gender identity refers to a person’s self-representation as male or female.
- Gender role describes the psychological characteristics that are sexually dimorphic within the general population, such as toy preferences and physical aggression.
- Sexual orientation refers to the direction(s) of erotic interest (heterosexual, bisexual, homosexual) and includes behavior, fantasies, and attractions.

Psychosexual development is influenced by multiple factors such as exposure to androgens, sex chromosome genes, and brain structure, as well as social circumstance and family dynamics.

Gender Assignment

Factors that influence gender assignment include the diagnosis, genital appearance, surgical options, need for lifelong replacement therapy, the potential for fertility, views of the family and sometimes, circumstances relating to cultural practices.

More than 90% of 46,XX CAH patients and all 46,XY CAIS assigned female in infancy identify as females. Approximately 60% of 5α-reductase (5αRD2) deficient patients assigned female in infancy and virilizing at puberty (and all assigned male) live as males. Among patients with PAIS, androgen biosynthetic defects, and incomplete gonadal dysgenesis, there is dissatisfaction with the sex of rearing in about 25% of individuals whether raised male or female.

Surgical Management

It is anticipated that surgical reconstruction in infancy will need to be refined at Puberty.

- Surgery should only be considered in cases of severe virilization (Prader III-V) and be performed in conjunction, when appropriate, with repair of the common urogenital sinus.
- Vaginal dilatation should not be undertaken before puberty.
- In the case of a DSD associated with hypospadias, standard techniques for surgical repair such as chordee correction, urethral reconstruction and the judicious use of testosterone supplementation apply.
- The testes in patients with CAIS and those with PAIS, raised female, should be removed to prevent malignancy in adulthood.
• The streak gonad in a patient with MGD raised male should be removed laparoscopically (or by laparotomy) in early childhood. Bilateral gonadectomy is performed in early childhood in females (bilateral streak gonads) with gonadal dysgenesis and Y chromosome material.

Sex Steroid Replacement

Hormonal induction of puberty should attempt to replicate normal pubertal maturation to induce secondary sexual characteristics, a pubertal growth spurt, and optimal bone mineral accumulation, together with psychosocial support for psychosexual maturation. Intramuscular depot injections of testosterone esters are commonly used in males. Females with hypogonadism require estrogen supplementation to induce pubertal changes and menses. A progestin is usually added after breakthrough bleeding develops or within 1-2 years of continuous estrogen.

Monitoring and follow-up

These children require regular follow up and need monitoring for
• Growth and pubertal status.
• Compliance to the prescribed medications and reminders for stress cover.
• Evaluation of desired effects of replacement steroids and monitoring for adverse effects.
• Addressal of psychological and social issues.

Open communication with patients and families is essential and participation in decision-making should be encouraged. Patient and family concerns should be respected and addressed in strict confidence. Optimal care for children with DSD requires an experienced multidisciplinary team. Ideally, the team includes paediatric subspecialists in endocrinology, surgery and/or urology, psychology/psychiatry, gynaecology, genetics, neonatology and, if available, social work, nursing and medical ethics. Support groups complement the work of the health care team and, together, can help improve services. Initiatives by support groups have led to improvements in management of DSD and research directed towards clinically relevant issues.
Hypocalcemia due to vitamin D deficiency presenting as heart failure in an infant

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Background:
Hypocalcemia due to vitamin D deficiency is an important reversible cause of cardiomyopathy and cardiac failure in infants.

Case
A 2 ½ month female infant was brought to the emergency with cough since 4 days and rapid breathing since a day. She had difficulty in feeding and sweating during feeds. There was no fever. She was born to a 22 year old mother without any antenatal complications. Her birth weight was 2 kg and the neonatal period was uneventful. The mother had not received any calcium and vitamin D supplements during and after pregnancy.

On examination, the child was irritable. She had pulse of 150/minute with regular rhythm and normal lower limb pulses. There was tachypnea (respiratory rate 60/minute) with intercostal retractions. There was no cyanosis and capillary refill time was less than 2 seconds. Cardiovascular examination revealed muffled heart sounds with apical grade 2/6 systolic murmur. Bilateral crepitations were present over lung fields. Liver was enlarged 2 cm below the right costal margin. Oxygen saturation was 96% on room air.

A clinical diagnosis of congestive cardiac failure was kept. The child was started on oxygen, IV fluids, dobutamine, frusemide and digoxin.

Electrocardiogram showed prolonged corrected QT interval (0.5 s) and sinus tachycardia with normal axis. Radiograph of chest showed cardiomegaly and rachitic changes in the rib ends. Echocardiogram showed dilated cardiac chambers, grade 2 mitral regurgitation and low ejection fraction (25%).

The biochemical investigations showed hypocalcemia (ionized calcium- 0.77 mmol/L), raised alkaline phosphatase (725 U/L) and normal phosphate (4.4 mg/dL). Further evaluation showed hypovitaminosis D (4 ng/mL) with secondary hyperparathyroidism (574 pg/mL). Hemoglobin was 93 g/L.

Hypocalcemia was managed with IV calcium gluconate infusion and oral calcitriol. There was remarkable clinical improvement with treatment. Oral calcium carbonate was started after initial stabilisation. Vitamin D supplementation was done with cholecalciferol 30,000 IU every 10 days. Mother had hypovitaminosis D, which was appropriately treated.

At 3 month follow up visit, she was asymptomatic, her serum calcium was 10.4 mg/dL and ejection fraction had improved from 25% to 45%.

Discussion
Vitamin D deficiency is very common in India despite of abundant sunshine. Hypocalcemia is uncommon due to secondary hyperparathyroidism, but can occur in inadequate calcium intake and increased demands of infancy and adolescence. Hypocalcemia commonly presents as seizures but cardiac failure secondary to dilated cardiomyopathy can be a less common presentation. Identifying this is very important as with adequate treatment, the cardiac dysfunction is completely reversible [1].

Maternal hypovitaminosis D, exclusive breast feeding, low birth weight and no supplementation of vitamin D puts the child at the risk of vitamin D deficiency as in the case described above. Investigation and treatment of mother must be done in such cases.

Additionally maternal primary hyperparathyroidism, which can cause hypocalcemia in the newborn can also be picked up by investigating the mother.

There are no adequate studies on the dose and duration of vitamin D treatment. However megadoses of 3 lac or 6 lac units can cause hypercalcemia and renal injury and should not be given in infants. Misra et al, in one of the review have recommended to use dosess of 1000 IU/day for infants <1 month, 1000-5000 IU/day for 1-12 month and >5000 IU per day for >12 month old [2].

American Academy of Pediatrics recommends 400 IU of vitamin D to be supplemented to all the newborn babies and infants for the prevention of vitamin D deficiency. There are no specific recommendations for Indian population.

Key messages:
1. Hypocalcemia is an important reversible cause of dilated cardiomyopathy.
2. Vitamin D deficiency is common at all ages and must be considered in infants presenting with hypocalcemia.
3. Investigation and treatment of mother is very important.

Key References:
A Case of Diabetic Ketoacidosis

Background:
Diabetic ketoacidosis is seen in 30-70% percent of cases of type 1 diabetes at onset. The incidence is higher where delay in diagnosis is common due to various reasons. Cerebral edema is the most common cause of mortality in diabetic ketoacidosis.

Case History:
A 12½ yr boy was transferred from a hospital with history of polyuria & polydipsia of 2 weeks duration. He was admitted to the referring hospital on the prior evening and was found to have blood glucose of 450 mg/dL. Pending further tests (fasting and postprandial glucose) he was started on IV fluids. Subsequently he developed respiratory distress following which a SC dose of insulin was given and an insulin infusion was started. He was referred to the tertiary center because he developed 2 episodes of tonic posturing of limbs. On admission to the tertiary center, the boy was had sunken eyes and dry oral mucosa. He had tachycardia (Pulse - 150/min), peripheral pulses were palpable and had normal volume and BP of 130/80 mm Hg. Urine output was normal. In the emergency department he had another episode of tonic posturing.

The random blood glucose was 395 mg/dL and the blood gas showed a pH of 7.0, pCO2 of 9.8 mm of Hg and HCO3 of 5.4 mmol/L. Urine ketones were positive, Serum sodium was 124 mEq/L and corrected sodium was 130 mEq/L.
He was clinically diagnosed to have tonic posturing due to sudden lowering of osmolality (secondary to large SC dose and infusion of insulin). He was given 4 ml/kg of 3% sodium chloride following which there was significant improvement in his sensorium.
He was subsequently managed with IV fluids (with potassium) and insulin infusion and the acidosis was resolved in 24 hrs. Subsequent fluid correction was done orally and SC insulin with glargine and regular insulin was started.

Learning points
1. Diagnosis of type 1 diabetes is usually simple as the symptoms and the level of blood sugar is typical. In such cases there is no need to delay the treatment to await any other confirmatory tests as this can have dangerous consequences. Diabetes is diagnosed with a random blood glucose of more than or equal to 200 mg/dL in presence of classical symptoms.
2. The clinical signs of dehydration underestimate the severity in DKA. Being a hyperosmolar dehydration, the intravascular blood volume is maintained till very last stages.
3. If the urine output is normal or high even in presence of severe dehydration, diabetes should be suspected.
4. Bolus SC or IV doses of insulin and rapid administration of fluids can cause rapid decline in osmolality causing cerebral edema and raised ICP. This can be fatal, hence recent guidelines do not recommend giving any boluses of insulin. The insulin therapy is started 1 hour after the fluids and as a continuous infusion in normal saline.
5. For the same reason rapid boluses of IV fluids are avoided in DKA. Fluid boluses are reserved for lack of pulses or hypotension and should be given slowly. The fluid correction also should be done slowly over a period of 36-48 hours.
6. Sodium bicarbonate therapy can cause hyperosmolality, paradoxical CNS acidosis and hypokalemia. The acidosis of DKA most often gets corrected with fluids and insulin therapy. Bicarbonate therapy is reserved for severe acidosis (pH < 6.9) despite of adequate fluids and insulin or in cardiac compromise due to acidosis.

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