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## Inside this Issue



# C A P E NEWS

For Private Circulation Only

## *THE MUMBAI UPDATE*

*Leena Priyambada,*
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The "International Update in Pediatric Endocrinology" held in Mumbai (Feb 14th – 17th 2008), was jointly organized by the Pediatric and Endocrinology Chapter of Indian Academy of Pediatrics (PAEC-IAP) and the Asia Pacific Pediatric Endocrine Society (APPES), with support from the European Society for Pediatric Endocrinology (ESPE). Leading paediatric endocrinologists from all over the world reviewed and discussed the state of the art. It provided an excellent platform for transcontinental exchange of ideas. Interactive case-based discussions and mini-poster sessions enhanced the educative value of the event.

### MOLECULAR GENETICS:

**Role of molecular genetics:**

The increasingly prominent role of molecular genetics in pediatric endocrine clinical practice was discussed in depth by distinguished speakers especially in recognition of linked diseases, targeted therapeutic interventions, disease subtyping, epigenetics (study of changes to the physical structure of DNA that can influence gene expressions).

Use of pharmaco-genomics (anticipating response to treatment based on genotype) to help manage growth hormone (GH) treatment could be an achievable goal within next few years though large collaborative studies will be required. *(PE Clayton)*

**Isolated GH deficiency (GHD) type IB, GHRH-R mutations:**

Genetic analysis of isolated GHD type IB revealed GHRH-R gene mutations (E72X) from west region of India similar to that of Sindh in Pakistan, all patients having a characteristic phenotype; short lean body with lack of frontal bossing or truncal obesity, sharp sculpted features, normal psychomotor development with normal to slightly delayed sexual maturation; described as "miniature adults".

Children with normal levels of GHBP constitute a larger proportion of patients of GH insensitivity. Further molecular characterization will reveal the cause for this different biochemical phenotype. *(MP Desai)*

### GROWTH AND PUBERTY:

**Weekly GH formulation:**

LB03002 is a novel once-a-week subcutaneous sustained release rhGH, being developed in co-operation with Biopartners' and LG Life Sciences' Study Group. In GHD children, once-a-week administration of LB03002 (0.5mg/wk) vs. daily GH (0.03 mg/kg/d) over 24 months was shown to be safe, well tolerated and delivering comparable growth. *(P Saenger)*

**Evolutionary perspectives of growth in infancy & childhood:**

Transition from infancy to childhood is marked by a growth spurt. Adult size is determined to an important extent during *transition* from infancy to childhood. A delay in this growth spurt has a lifelong impact on stature and explains almost half of the idiopathic short stature in developed countries and more in developing ones. Humans evolved to withstand energy crises by decreasing their body size. The evolutionary adaptive

strategies of plasticity in timing of transition from infancy into childhood match the prevailing energy supply, culminating in short stature that is transmitted over 3-4 generations from parents to offspring. *(Z Hochberg)*

**Delayed puberty, chronic illness:**

In a retrospective analysis of causes of pubertal delay in an endocrine clinic in north India, chronic systemic illnesses like malnutrition and tuberculosis were found to be the largest group (40%), followed by hypothyroidism (20%). Since these are potentially treatable causes, care should be taken to recognize them early. *(BK Bhakri, K Biswas)*

## BONE HEALTH:

**Rickets as a manifestation of tissue phosphorous deficiency:**

An interesting perspective on rickets was proposed: that tissue phosphorous deficiency plays a central role in etiology of rickets by failing the apoptosis of the terminal hypertrophic chondrocytes. *(Z Hochberg)*

**Calcium, vitamin D nutrition in adolescent girls:**

Calcium deficiency alone can also cause rickets and osteopenia. Severe dietary deficiency of absorbable calcium leads to elevated PTH, which is known to increase synthesis of $1,25(OH)_2D3$, which in turn degrades 25(OH)D3 to inactive 24,25(OH)D3, thereby depleting body stores of 25 OH D3. *(V Khadilkar)*

**Vitamin D deficiency, pregnant women and neonates:**

Vitamin D deficiency is common in among mothers and their newborns in India even after prolonged sun exposure. Possible contributing factors could be the traditional clothing and inadequate skin exposure, skin pigmentation, pollution, and low dietary calcium. Hypovitaminosis D during pregnancy can lead to neonatal hypocalcemia, infantile rickets, poor growth, lower respiratory tract infections, and fetal programming leading to persisting osteopenia. There is a real need to supplement pregnant women with vitamin D to avoid these problems in their babies. Intervention and possible formulation of public health policy is needed. The minimum effective dose needs to be established through properly designed studies. *(V Bhatia)*

In a novel and interesting study, two types of doses of vitamin D (60,000 U vs 240,000 U) were supplemented to pregnant females and results compared with unsupplemented controls. Both doses of vitamin D provided protection against neonatal biochemical rickets (babies' cord blood alkaline phosphatase was significantly lower with both doses), and improved neonatal anthropometry (birth weight, length and head circumference were significantly higher, anterior fontanelle significantly lower in supplemented group). The difference in the babies' anthropometry persisted till 9 months of age! *(P Kalra, V Bhatia)*

**Hypercalcemia, hypervitaminosis D:**

In more than 60% of children (1.5-18 months age) presenting with hypercalcemia, the cause was hypervitaminosis D. Mega-doses of vitamin D (9-20 lac U) had been given inappropriately to these children for failure to thrive. Spreading awareness against this practice can easily prevent a majority of cases of hypercalcemia. *(S Rao)*

**1α hydroxylase def. rickets:**

Rickets due to 1α hydroxylase deficiency can present with normal $1,25(OH)_2D$ levels. Therapeutic response is seen with the recommended dose of $1,25(OH)_2D$ supplementation. Therapeutic trial of $1,25(OH)_2D$ should be given in a clinically compatible case where other causes have been ruled out. *(Case discussion: L Priyambada, V Bhatia, Z Hochberg, CJ Munns)*

*** Normal urine calcium/ creatinine ratio is higher in the newborn period (0.9) and becomes 0.25 by 2 years of age.

*** High frequency low magnitude biomechanical bone stimulation is a new and exciting modality with potential to improve both bone strength and muscle function especially in childhood osteoporosis. *(CJ Munns)*

## DIABETES MELLITUS (DM), HYPOGLYCEMIA

**Hyperinsulinemic hypoglycemia:**

Congenital hyperinsulinism is a heterogeneous disorder. $^{18}$Fluro-L-Dopa PET scanning is highly sensitive for differentiating focal from diffuse disease as well as accurately locating the focal lesion. This is crucial, as removal of the focal lesion will result in "cure", while most infants with diffuse disease would require near total pancreatectomy. *(K Hussain)*

**Neonatal DM:**

Some of the persistent and the transient forms are due to activating mutations in the genes (*ABCC8* and *KCNJ11*) encoding the SUR1 and KIR6.2 proteins of the pancreatic beta cell $K_{ATP}$ channel respectively. These patients respond to oral hypoglycemics (sulphonylureas) and their insulin therapy can be discontinued. A subgroup of these patients has developmental delay and epilepsy which also improves with oral sulphonylureas. Hence, all patients with DM diagnosed before six months

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of age should have mutational analysis for the *ABCC8* and *KCNJ11* genes. *(K Hussain)*

Elevated BP at night is the first sign of hypertension in children with DM. Angiotensin receptor II blockers are better used for treatment in hypertension. *(F Chiarelli)*

**Infants of diabetic mothers:**

Correlation was found between infant macrosomia and maternal fatness, serum IGF-I and IGF-2, but not with maternal HbA1c. *(W Cutfield)*

Symptomatic hypoglycemia is more commonly seen in offspring of T1DM mothers than those with T2DM. *(W Cutfield)*

**Infantile onset DM:**

11 cases analyzed; mean age of presentation was 3 months. 6/11 cases had syndromic association (Wollcot Rallison syndrome). It was emphasized (as would have been the practical experience of many of us) that maintaining good glycemic control was difficult as the babies require small doses and there is difficulty in availability and administration of such small doses. Hence there is a need for newer insulin delivery systems for better titration and thereby preventing complications. *(R Ganesh, T Vasanthi)*

**Behavioural and marriage problems in people with T1DM:**

A survey of over 100 T1DM patients reveals that most of tem are not comfortable in social gatherings, and it is relatives who hurt them the most. A significant number felt that they should not make their disease public. Marriage problems were more for the women, and a significant proportion felt they should not get married, which was not the case with the men. Spreading awareness about T1DM and insulin therapy is the need of the hour. *(R Shukla, D Yagnik)*

**Screening first degree relatives of PCOS patients:**

191 first degree relatives of 99 PCOS patients were evaluated and compared against matched controls. They were found to have a higher prevalence of diabetes, IGT, metabolic syndrome, and HOMA-IR. BMI and waist hip ratio were found to be strong predictors of IR. *(P Kalra, E Bhatia)*

## DSD:

**Strategies for surgical intervention in DSD:**

Major changes in practice were reiterated: children with minor degrees of clitoral enlargement are less likely to be offered clitoral reduction than they once would have been. Families should be offered the option of deferring reconstructive surgery for their child with ambiguous genitalia. Truthful disclosure makes it possible to have a free and open discussion about the facts with the patient as well as the parents. *(G Warne)*

**Long term outcome studies in congenital adrenal hyperplasia:**

Women with salt wasting forms of CAH have more pain and discomfort during intercourse, are overall less sexually active, and experience a range of emotional disorders, including gender dysphoria. Men with poorly controlled CAH have a high incidence of adrenal rests in their testes; these obstruct the hilum, causing infertility. These adrenal rests may also be mistaken for cancer and testes may be unnecessarily removed. *(G Warne)*

**Genetic analysis of *SRD5A2* (5 alpha reductase enzyme gene):**

The known missense mutation pR246Q in exon 5 of *SRD5A2* was found in 2 patients with 5 alpha reductase enzyme deficiency. PCR RFLP could be used for a quick provisional diagnosis of this mutation where clinically compatible. *(E Bhatia)*

## HYDROCORTISONE AND PREMATURITY:

*** Short courses of antenatal steroids do not cause suppression of the hypothalamo-pituitary-adrenal axis.

*** Transient adrenocortical insufficiency of prematurity (characterized by exaggerated ACTH response and adrenocortical insufficiency during the first week of life in sick VLBW infants) is more responsive to glucocorticoids than to inotropes, and resolves within 14 days. Hypotension has become the commonest indication for the use of hydrocortisone in VLBW infants. Clear benefit of hydrocortisone therapy in treatment of hypotension has been documented in VLBW babies without a concomitant increase in acute adverse effects. Oxygen free survival increased, and need for volume expanders and vasopressor support decreased in babies administered hydrocortisone for refractory hypotension.

*** Healthy VLBW, term infants secrete cortisol at similar rates of 6.6-8.8 $mg/m^2/24hrs$. The secretory pulses are longer and half life shorter in VLBW infants. Hence, cortisol is better administered as an initial iv bolus followed by continuous infusion. Concerns about long term risks require further extensive studies. *(W Cutfield)*

## CUSHINGS DISEASE:

A retrospective analysis of 44 cases of pediatric Cushing's disease has shown comparable sensitivity of clinical features in making a diagnosis as a combination of dynamic tests and imaging modalities. It was interesting to note that of the 4 patients who had to undergo bilateral adrenalectomy, 3

developed Nelson's syndrome within a short average duration of 5 months. *(N Shah)*

**Cushing's disease and growth:**

*** Growth retardation is a hallmark of Cushing's disease; associated with reduction in mean 24 hour levels of GH and the amplitude of its secretory pulses. Hypercortisolemia suppresses the amplitude of GHRH pulses while increasing that of somatostatin. A direct pituitary action may also be present.

*** GH hyposecretion may persist for 1-9 years after remission of Cushing's disease. This results in lack of catch-up growth in these patients.

*** Direct adverse effects are seen on the growth plate also. Expression of the GH receptor is downregulated in chondrocytes, resulting in reduction of local IGF-1 production. Hence, a GH resistant state occurs and this persists after remission.

*** Impact of glucocorticoids on bone mineral density, with resultant osteoporotic fractures especially involving trabecular bone, may be an additional reason for poor linear growth. Close monitoring of growth after documenting cure is necessary for timely intervention. GH±GnRH therapy is indicated at the earliest. *(N Shah)*

## THYROID:

**Congenital hypothyroidism:**

Almost all programs of newborn screening are based on detection of elevated TSH concentrations alone or in combination with T4/fT4.TSH in cord blood samples using similar cut offs as after 3 days of life can be used as an alternative approach. Repeat testing at 2-6 weeks of life can detect an additional 10% of infants with CH. Asymptomatic hyperthyrotropinemia occurs in 1:8000 births, with 50% due to perinatal iodine exposure, eg with use of betadine as disinfectant. This condition is usually treated, but can be managed expectantly if fT4 is in the upper half of normal range.

Serum TSH in some treated infants with proven CH may remain relatively elevated for a few months despite normal T4, due to a resetting of the feedback threshold for T4 suppression of TSH release. The resetting occurs prematurely but the mechanism is obscure. *(A Greuters)*

**Thyroid function in the premature infant:**

VLBW, ELBW infants are predisposed to development of transient primary hypothyroidism and the syndrome of transient hypothyroxinemia of prematurity due to their immature hypothalamic- pituitary- thyroid system and high neonatal morbidity. Transient primary hypothyroidism (low T4, high TSH) varies geographically, relative to iodine intake. Premature infants require higher iodine levels than term babies to maintain adequate T4 production in extrauterine environment. Fetal thyroid is also inordinately sensitive to inhibitory effects of iodide on hormone synthesis (perinatal iodide exposure, eg betadine use). This will require treatment if the hypothyroidism persists for several weeks. *(A Greuters)*

**Transient hypothyroxinemia of prematurity:**

In ELBW, VLBW infants serum TBG, T4, T3, and fT4 are lower, with an obtunded TSH surge. This immaturity is inversely proportional to their gestational ages. Nadir values are reached by 7-10 days of life, with re-equilibration with cord values by 3-4 weeks. The concomitant high prevalence of neonatal morbidity leads to characteristic changes of nonthyroidal illness. The impact on brain maturation is not clear. *(A Gruters)*

**Primary hypothyroidism (PH) and Histiocytosis:**

In an unusual case, PH with goiter was an early presentation of Langerhans cell histiocytosis in a five year old girl, followed by progressive CNS involvement, hypothalamic mass, hypocortisolemia, and GHD, and subsequent death. Provisional diagnosis made by FNAC of thyroid and confirmed by electron microscopy of thyroid tissue (birbeck's granules). Infiltrative disorders might need to be ruled out in primary hypothyroidism with unusual presentations, such as firm goiter at an early age, as in this child. *(L Priyambada, V Bhatia)*

**RESOURCES:**

'POSSUM' (http://www.possum.net.au/about_us/about_us.htm): on line computer reference for searches on syndromes.

'CLAN' (http://cahclan.org/): An Australian based international support organization for "maximizing quality of life for all patients with CAH by supporting access to affordable medication, optimal medical treatment, CAH Support Group networking; so that all members of the international CAH community can enjoy healthy and happy lives, and know that their neighbors care."

## NEW MEMBERS

**We extend a very warm welcome to our new members:**

1. **ANISH AHAMED**, Kochi
2. **SUBODH BANZAL**, Indore
3. **IP CHOUDHRI**, Delhi
4. **PRASUN DEB**, Hyderabad
5. **NILESH DETROJA**, Rajkot
6. **GANESH HK**, Mangalore
7. **JOE GEORGE**, Kannur

8. **RAJESH JOSHI**, Mumbai
9.**NAVEEN MITTAL**, Ludhiana
10. **MINI G PILLAI**, Kochi
11. **COL P PRUSTY**, Kendrapara
12. **VM RAO**, Kakinada
13. **AP REDDY**, Tirupati
14. **SM SARDANA**, Ropar
15. **RAMANBIR SINGH**, Ludhiana
16. **AS VERMA**, Hyderabad
17. **VIJAYAKUMAR M**, Calicut.

**WEBSITE:** We would be starting our website soon. Please give us ideas about what should be featured in it. –Ed.

## PUBLICATION NEWS

It is always important to know our own data. We propose to start a new column listing publications in the recent past by Chapter members. For a start, I found these papers by Council members. Please do send me references of your papers or others you come across with pertinent Indian data.- Ed

1. Age-related changes in bone turnover markers and ovarian hormones in premenopausal and postmenopausal Indian women. Desai MP, Bhanuprakash KV, Khatkhatay MI, Donde UM. *J Clin Lab Anal. 2007; 21(2): 55-60.*
2. Subclinical hypovitaminosis D among exclusively breastfed young infants. Bhalala U, Desai MP, Parekh P, Mokal R, Chheda B. *Indian Pediatr. 2007 Dec; 44(12): 897-901.*
3. Serum leptin levels in obese Indian children relation to clinical and biochemical parameters. Dubey S, Kabra M, Bajpai A, Pandey RM, Hasan M, Gautam RK, Menon PS. *Indian Pediatr. 2007; 44(4):257-62.*
4. High prevalence of glucose intolerance even among young adults in south India. Raghupathy P, Antonisamy B, Fall CH, Geethanjali FS, Leary SD, Saperia J, Priya G, Rajaratnam A, Richard J. *Diabetes Res Clin Pract. 2007;77(2):269-79.*
5. Etiology of early-onset type 2 diabetes in Indians: islet autoimmunity and mutations in hepatocyte nuclear factor 1alpha and mitochondrial gene. Sahu RP, Aggarwal A, Zaidi G, Shah A, Modi K, Kongara S, Aggarwal S, Talwar S, Chu S, Bhatia V, Bhatia E. *JCEM 2007 Jul; 92(7): 2462-7.*
6. Intensive glycemic control in diabetic pregnancy with intrauterine growth restriction is detrimental to fetus. Parikh RM, Joshi SR, Menon PS, Shah NS. *Med Hypotheses. 2007; 69(1): 203-5.*
7. Pituitary tuberculosis. Sunil K, Menon R, Goel N, Sanghvi D, Bandgar T, Joshi SR, Menon P, Shah N, Goel A. *J Assoc Physicians India. 2007 Jun;55:453-6.*
8. Management of diabetic ketoacidosis in PICU. Jahagirdar RR, Khadilkar VV, Khadilkar AV, Lalwani SK. *IJP 2007;74(6):551-4.*
8. IAP growth monitoring guidelines for children from birth to 18 years. Khadilkar VV, Khadilkar AV, Choudhury P, Agarwal KN, Ugra D, Shah NK. *IP 2007;44(3):187-97.*
9. Multicentric study of efficacy and safety of growth hormone use in growth hormone deficient children in India. Khadilkar VV, Khadilkar AV, Nandy M, Maskati GB. *IJP 2007; 74(1):51-4.*
10. Insulin like growth factors and growth hormone deficiency. Yadav S, Krishnamurthy S. *IP 2007; 44(5): 349-53.*

## PEDICON 2008: BHUVANESWAR

*Vandana Jain, Dept of Pediatrics, AIIMS, New Delhi*

The Pediatric Endocrinology Chapter symposium was held on 19th January (8-9.30am). Dr Subrata Dey from Kolkatta spoke on ***21st century technology - how it is helping the diabetic child***. It was followed by Panel Discussion on ***Growth and GH*** with Prof Sangeeta Yadav from Maulana Azad Medical College, New Delhi, as moderator. The panelists spoke briefly on ***All short stature is not endocrine*** (Dr Vandana Jain, AIIMS); ***Difficulties and pitfalls in the diagnosis of GHD*** (Dr Rajesh Khadgawat, AIIMS); ***GH therapy for GHD*** (Dr Shaila Bhattacharya, Bangalore) and ***GH therapy for Turner syndrome and idiopathic short stature*** (Dr Kavita Bhat, Bangalore). The talks as well as the subsequent panel discussion were very well received by the audience. Immediately after the symposium, the annual GBM was held with Dr V Bhatia in the chair.

During the pre-conference CME on 16th January, Dr Shaila Bhattacharya spoke on ***Obesity***, and Dr Vijayalakshmi Bhatia on ***Rickets***. Both talks were appreciated.

## PRACTICAL PEDIATRIC ENDOCRINE UPDATE: KOLKATA

*Subrata Dey, Apollo Gleneagles, Kolkata*

The 1st Practical Pediatric Endocrine Update (PPEU) was organized by Dr Subrata Dey at Apollo Gleneagles Hospital, Kolkata in collaboration with the IAP West Bengal Branch on 20th April, 2008. A power one day program was designed to address the common queries of practicing pediatricians and post graduates. There was a variety of topics ranging from newborn thyroid screening and goiter, to PCOS, childhood metabolic syndrome, rickets and puberty. Speakers included Dr Margaret Zacharin (Australia), Dr Vijayalakshmi Bhatia Dr Nalini Shah and Dr Anju Virmani. The attendance was excellent with several delegates from Bangladesh and all over the northeast, and the audience was very enthusiastic in its participation. This first of its kind, this pediatric endocrinology CME in Eastern India was a resounding success.

## APPES NEWS

*PSN Menon, Member, Governing Council, APPES.*
*psnmenon@hotmail.com*

**Fellows Meeting, Taipei, Dec 2007**

Every year before the APPES regional meeting, a Fellows' Meet is organized to impart education and professional guidance to young fellows interested in the

field of pediatric endocrinology. The last Fellows' meeting was held in Taipei, Taiwan (3-5 December 2007). We got the great opportunity to represent India among about 40 fellows from Asia Pacific region.

The esteemed teaching faculty comprised of Dr Xiaoping Luo (China), Prof Louis Low (Hong Kong), Prof Ho Seong Kim (Korea), Prof Sei Won Yang (Korea), Prof Wayne Cutfield (New Zealand), Dr Shu San-Ging (Taiwan), Dr Fu-Sung Lo (Taiwan) and Dr Maria Craig (Australia). All the faculty members were friendly and approachable. The talks given by them were up to date, lucid and very informative.

The three day meeting was divided into various sessions of Fellow case presentations, case discussions and talks by faculty. Prior to the meeting, we were asked to submit interesting and problematic pediatric endocrine cases in our clinical experience. These were further re-allotted among other fellows for detailed discussions on various subtopics. Thus we had a basket of rare and interesting cases from all over the Asia Pacific region, discussed on one podium with excellent inputs from the teaching faculty and other fellows. There were very interesting brainstorming sessions covering all the fields of pediatric endocrinology. The interactive sessions helped us to clear our doubts and problems faced in managing patients. In the quiz, which was held on the last day of meeting, we managed to get 2nd and 3rd positions.

It was also a wonderful opportunity to interact with the fellows from other areas of the region and share information about different health systems, teaching facilities, patient profiles and common problems faced in pediatric endocrine practice.

This meeting was very well organized in terms of visa, travel and hospitality arrangements. It was a good opportunity to get the taste of original oriental cuisine. The social event, a visit to a skyscraper, Taipei101 was full of fun and enjoyment.

It was very productive for young fellows in terms of information, approach and updates. Overall we can say it is a perfect type of event that every aspiring pediatric endocrinologist should attend.

**Dr Bhanu Kiran Bhakhri,** Senior Resident, Dept of Pediatrics, Vardhman Mahavir Medical College & Safdarjung Hospital, New Delhi.

**Dr C Sadish Kumar,** Registrar, Dept of Endocrinology, Metabolism & Diabetes, King Edward VII Memorial Hospital and Seth GS Medical College, Mumbai.

**Dr Deepti Chaturvedi,** Specialist Pediatric Endocrinologist, New Medical Center Hospital, Abu-Dhabi, UAE.

## FORTHCOMING MEETINGS

**1. TYPE 1 DIABETES MEET:** 1st June 2008, organized by IAP-BPS and manipal Hospital, Bangalore. Contact: Dr Shaila Bhattacharya, 9000655552; (080) 2502 3263/ 4444.

**2. ISPAD 2008:** 34th Annual Meeting: 12-16 Aug 2008, Durban, South Africa. Contact: Kuben Pillay, West Ville Hospital Westville, 7 Spine Road, Suite 561, IDurban 3600 4013, South Africa. Tel: +27 31 2655377, Fax: +27 31 2655378, kubenpillay@worldonline.co.za, www.ispad2008.com

**3. ESPE 2008:** 47th ESPE Meeting: 20-23 Sep 2008, Istanbul, Turkey. Contact: Atilla Buyukgebiz, Prof Dokuz Eylul Faculty of Medicine, Dept of Paediatric Endocrinolgy & Adolescence, Inciralti, IZMIR, TR-35340, Turkey. Tel: +90 2322788411 (telefax)/4644540, Fax: 4649240, atilla.buyukgebiz@gmail.com www.congrex.com/espe2008/

**4. JSPE 2008:** 42nd Annual Meeting: 2-4 Oct 2008, Yonago, Tottori, Japan. Contact: Susumu Kanzaki, E-mail: smkanzak at grape.med.tottori-u.ac.jp

**5. APPES 2008: 5th Bie**nnial Meeting of the Asia Pacific Pedistric Endocrine Society: 29 Oct- 1 Nov 2008, Seoul, Korea. Email: appes@willorganise.com.au.

**6. ICE 2008:** 13th International Congress of Endocrinology: 8-12 Nov 2008, Rio de Janeiro, Brazil. Contact: Ruth Clapauch, rclapauch@uol.com.br. www.ice2008rio.com/

**7. RSSDI 2008: 36th** Annual Scientific Meeting of the Research Society for Study of Diabetes in India: 21-23 Nov 2008, Hyderabad. Contact: Dr Vasanth Kumar, registration@rssdi2008.org; www.rssdi.com.

**8. ESICON 2008:** 38th Annual Meeting of the Endocrine Society of India: 18-20 Dec 2008, Kochi. Contact: Dr Unnikrishnan. unnikrishnanag@aims.amrita.edu.

**9. PEDICON 2009:** 46th National Conference of the IAP: 22-25 Jan 2009, Bangalore. Contact: Dr R Nisarga, cimblr@cimindia.net; www.pedicon2009.org

**10. PEDS ENDO WORKSHOP:** 21st Jan 2009, at Sagar Apollo Hospital, Bangalore. Contact Dr P Raghupathy, p.raghupathy@gmail.com.

**11. ESPE/LWPES:** 8th Joint Meeting: 9-12 Sep 2009: New York, USA. Contact: Paul Saenger, Fax: +856.439.0525. phsaenger@aol.com. & lwpes-espe2009@ahint.com. www.lwpes-espe2009.org

**12. ISPAD 2009:** 35th Annual Meeting: 16-18 Sep 2009, Ljubljana, Slovenia. Contact: Tadej Battelino, E-mail: tadej.battelino@mf.uni-lj.si

## NEWS YOU CAN USE

### THE NATIONAL INSTITUTE OF OPEN SCHOOLING (NIOS)

*(Courtesy Dr Chittaranjan Andrade)*

The National Institute of Open Schooling (www.nos.org) is an important initiative of the Government of India, created to cater to the educational needs of students who cannot attend regular schools. This may be a godsend to those of our patients who have lost school years because of chronic illness. It offers a national board examination at two levels: secondary and higher secondary. The secondary level is equivalent to the ICSE, CBSE, SSLC,

and other national or state Standard 10 board examinations. The higher secondary level is equivalent to the PUC and other Standard 12 board examinations. Many boards, institutions and universities across the country recognize the NIOS certification for continued or higher studies; these are listed in the NIOS prospectus and at the NIOS website. Patients who cannot attend regular college either can continue education after NIOS at the Indira Gandhi National Open University (IGNOU).

NIOS requires students to study a minimum of 5 and a maximum of 7 subjects. There is a very wide range of subjects on offer, and students are allowed to change subjects midway through the course if they are unhappy with their choice. They can choose from among 3 languages for their medium of instruction.

Students who fulfil the specified minimum age criteria can register for the secondary or higher secondary NIOS courses during July and August in each year; the exact dates are specified at the website. The cost of the registration and of the courseware is low; all sections of society will be able to afford an NIOS education. Instruction is also available from priced audio and video cassettes, CDs, radio and television broadcasts.

Exams are conducted twice a year: in April-May, and in October-November. Examiners tend to be lenient in their standards, understanding the special needs of the students who appear. Students require to score a minimum of 33% to pass an examination.

The NIOS website (www.nos.org) is not very well constructed but serves its purpose. It provides the full text of the prospectus and information about the courses. Courseware is available online, as are sample question papers and the question papers of previous years. Important events and results are also announced online. While there is scope for improvement in the efficiency of the NIOS system, it is overall a scheme of which the country can be proud. Disadvantaged children can at last have an opportunity for pride in having completed their basic education.

The **Greulich and Pyle bone age atlas** is available for on-line purchase from Amazon.com. The cost of a new book is US $ 145. Shipping to India costs about $ 10 (shipping to a US address is free). It takes a month to arrive. Dr. Leena Priyambada.

Growth charts (based on KN Agarwal reference data) and locally made orchidometers are available with Dr Vijayalakshmi Bhatia.

*The Pediatric Endocrinology list serve is a lively international forum for discussing a wide range of interesting topics. To subscribe, send an email to* peds-endo-subscribe@yahoogroups.com. *or go to* health.groups.yahoo.com/group/peds-endo *for more information.*

BOOKS FROM KARGER

1. Practical Algorithms in Pediatric Endocrinology. Ed Zeev Hochberg: CHF 53, Eur 38, USD 48.
2. Yearbook of Pediatric Endocrinology 2007. Ed Jean-Claude Carel, Zeev Hochberg: CHF 70, Eur 50, USD 48.

*** For ordering or information on other Karger publications see: www.karger.com

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