C A P E NEWS

Pediatric and **A**dolescent **E**ndocrinology **C**hapter of IAP

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CHRONIC ILLNESS: ROLE OF THE DOCTOR ALONG WITH THE CAREGIVER

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Twentieth century advances in medicine have benefited both children and adults. As more children escape or survive life threatening diseases, they run the risk of suffering from chronic illnesses. The burden of chronic illness is not limited to the child alone: there is significant psychological, social and economic impact and burden on the family.

Numerous investigators have documented that children with chronic illness suffer more emotional, behavioral, and educational difficulties (Thompson et al, 1993; Wallander & Varni, 1998). Some of the many factors that make the child vulnerable to psychological disturbances include: the child's external health locus of control, maladaptive coping styles, and poor caregiver psychological adjustment.

Amelioration of these psychological responses within the matrix of the doctor-patient relationship is better for overall and long-term outcome. Ideally, every physician needs to address, in every consultation, the biological, psychological and social components of "physical" symptoms, and therefore of chronic physical illnesses, without caring for one component at the expense of the others (Madhu Sridhar 2001).

The help of trained professionals can be taken to help meet these psychological needs. However, our poor infrastructure makes it impossible to have the psychological needs of the patients catered to, so it becomes crucial for the primary pediatrician to address the psychological needs of the patient/ family along with the biological interventions required, in order to improve the welfare of the patient and family. Unfortunately, while there has been a lot of research in western countries, there is dearth of literature in the field of such interventions for parents and caregivers of such children in the Indian context. More importantly, communication of the results of these studies to specialists looking after children is largely neglected. Till date, during the training our pediatricians undergo mental health is neglected, cure receives more emphasis than care, and when cure is not possible (as with chronic illnesses), they flounder and are left with few options.

The impact of changes in patient behavior has been observed in several disorders, the most significant being seen in diabetes, ischemic heart disease, airway disease and hypertension (Oldenberg 1985; Johnson 1992). For example, a 70% reduction in visits to the doctor was seen in a chronically ill population following a 10 week biofeedback and stress manage-ment program (Kurtz, 1990). As doctors become more aware of the psychological components of physical illness, they realize that a counseling/ behavioral medical intervention can make an impact, which translates into more effective care. Just the process of carefully listening to the patient/ family has also been found to permit the development of trust and enhance the quality of life and well being of the patient (Radha Madhvi, 2001)

Some important reasons for focusing on children with chronic illness and their family members are that along with medical care the family must have:

a) A developmentally appropriate understanding of the illness (both for the child and immediate family),
b) Compliance with treatment regimens,
c) Integration of the illness into family life, including a balance between the needs of other family members,

d) Successful adaptation to important systems, such as the school, peers and the hospital,
e) Mastery of anxiety and fears related to the illness and its management (Koplewicz, 2001).

These pressing psychological, developmental, and social needs have spurred the development of psycho-social interventions to address mental health problems and maximize the functioning of children and families as productive members of society. It has been shown that even an intervention as simple as listening to the patient and family members can help in reducing their psychological disturbances. Other beneficial interventions include:

1. **Psycho-education**: One of the most important components is giving family members complete information about the illness- cause, course, treatment and long-term effects. This information should be honest and appropriate, and given in a form that it is understandable to them, keeping factors such as age and cognitive ability in mind.

2. **Developmental changes**: Children move from the more dependent state of childhood to the more independent state of adolescence. These changes affect their disease management in various ways, sometimes detrimental, eg issues with body image, refusal to take medication or make lifestyle modify-cations, etc. The pediatrician is aware of the phases of normal adolescent development. He/ she must take these into account, make the family aware of them and help the patient and family accept and cope with these changes and their impact. Strategies include:
a) Encouraging adolescents to share their ideas and concerns with healthcare professionals,

b) If the illness reaches an unstable state due to non-compliance, encouraging discussion of what happened rather than reprimanding non-compliance.

c) Teaching and encouraging use of problem-solving skills related to the illness. Ask questions such as: "What do you think you would you do if……?" or "What do you think would happen if……..?" Encourage adolescents to ask you the same kinds of questions.

d) Seeking mental health services when:
i) the adolescent seems overwhelmed with emotional issues related to living with a chronic illness.

ii) a pattern of non-compliance continues.

iii) the adolescent's development regresses, overly dependent behavior continues, and/or the adolescent withdraws from or gives up interest in age-appropriate activities.

3. **Skill training**- The pediatrician has to ensure that the child and family have mastered the skills required for disease management. The family would benefit from finding out from the doctor the changes likely to be seen in the child as a result of the illness.

4. **Socio-economic support-** is often crucial, since chronic illnesses tend to be expensive and emotionally draining. Thus if the doctor can put the family in touch with social support groups which help in getting available resources, give an opportunity to interact with others suffering from the same condition, and share the emotional burden, it would be very beneficial.

5. **Family therapy and group work**- for the parents regarding their role as caregivers. The pediatrician should help the family internalize the roles and responsibility of each member in handling the situation: this would make for a better family environment:
a) Each family member should be actively involved in the care of the child and aware of the disease to the extent that age allows. This helps because certain active changes have to be made in the family due to the child's condition.
b) The patient and family should be encouraged to become self advocates in obtaining information and identifying resources.
c) Contingency management plans should be formulated in case the family faces difficulties.
d) The family should be encouraged to make the life of the sick child as close to normal as possible. This would make the child feel active and involved, rather than isolated from peers and resentful.
e) The family should be constantly counseled that the child should not be made to feel he/ she is a burden or a cause for disturbance for the family.
f) The family should be encouraged to spend enjoyable quality time together.
g) The parents and other elders must be reminded to pay attention to the other siblings also so that they do not feel neglected.

A bio-psycho-social approach to helping patients with chronic illness needs medical practitioners to involve families in their treatment of illness and expand their perspective from an individual medical model to a social systemic model. (Madhu Sridhar 2001).

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A CASE OF IJO

V Bhatia, SGPGI, Lucknow

A 13 yr old girl presented with history of fall from a ladder 2 ½ years back, and onset of lower back pain since that time. The trauma was likely minimal, and she was not immobilised at any time then or thereafter. Spine X-rays taken then showed osteoporosis and anterior wedge fracture of L1-3 and T12. She had no further fractures, and was not given any specific treatment then, but was given a spinal brace about 2 months ago. She gave no history of fever, contact with open TB or past TB herself; no documented anemia or bleeding tendencies; no history suggestive of hyperthyroidism, malabsorption, seizures, or joint disease; no significant medication apart from injections of vitamin D in 2003 and 2004; and no significant family history. She had entered puberty about 1 ½ years before presentation. She was 45 kg, 148 cm, normotensive, afebrile, with no lymphadenopathy or organomegaly, no evidence of Cushing syndrome. Pubertal staging was breast stage 3, pubic hair stage 2. There was no evidence of Marfan syndrome in her body proportions or hand examination (no blue sclerae; no hyperextensibility of joints).

Her lab reports were as follows: hemoglobin 13gm%, TLC 7500/cumm, N70 L26 E2 M2; SGPT 50 U/L; creatinine 0.5 mg%; serum calcium 9.0, 10.1, and 10.8 mg%; PO4 3.4, 4.4, and 5.2mg%; alkaline phosphatase 538 and 594 U/L (normal for adults below 250 IU/L). Serum 25OH Vitamin D was 77.5 ng/ml (normal), PTH 54 pg/ml (normal 9-55 pg/ml), thyroid function tests and abdominal ultrasound examination normal.

The radiologist reported that the spine X-rays (lateral view) showed moderately severe wedge fracture deformities of L1 and L3. The intervening intervertebral disc spaces were of normal height and adjacent end plates were intact. No gross retropulsion was seen at either level. The appearances were those of simple fracture with no suggestion of underlying pathology except osteopenia. A diagnosis of IJO (idiopathic juvenile osteoporosis) was made. Though this is a self-limiting condition, in view of the severe nature of her osteoporosis, we opted to offer bisphosphonate therapy for a short period until we achieved normalization of density. She was given pamidronate infusions at a dose of 45 mg per dose for 3 consecutive days, once in 3 months, for 2 years. The initial bone mineral density as well as that after 2 years is in the table.

Site	Density (gm/cm2) 2004	Z score 2004	Density (gm/cm2) 2006	Z score 2006	Rate of change over 2 years
L1	0.663	-0.9	0.863	0.0	
L2	0.568	-2.8	0.904	- 0.4	59%
L3	0.485	-4.3	0.920	- 0.5	
L4	0.326	-6.7	0.835	- 1.2	156%
Total hip	0.484	- 3.4	0.679	- 2.2	40%
Wrist	0.335	NA	0.445	NA	33%

Comment: This case highlights several points worth remembering:

a. IJO is a diagnosis of exclusion, and a thorough history and examination should be performed to rule out important causes of osteoporosis.

b. When some vertebrae are collapsed, the conventional L1-L4 mean bone density should not be considered, and only individual vertebrae which are spared should be taken into account (as can be appreciated in the discrepantly high density in L1 in 2004).

c. The rate of change of density is higher than that seen in a 2 year period in this age group. The improvement could be spontaneous, or treatment related, or both. Rates of change in the pediatric age group in patients on anti-resorptive therapy are far higher than those obtained in adults, and the natural gain with age should be kept in mind while interpreting improvements. The normal rates of change in childhood are of the order of 4-5% and during puberty 8-10%.

d. T scores are not mentioned in pediatric bone density reporting, as peak bone mass has not yet been achieved. (T score is the number of standard deviations (SDs) the BMD is below reference peak bone mass for sex whereas Z score is the number of SDs the BMD is below reference bone density for age and sex). Even interpretation of Z scores may be fraught with danger. BMD depends on the size of the bone. The size of the bones is a girl with chronic disease, and possibly delayed puberty, is likely to be smaller, and thus may not be comparable to that of a normal girl of the same age.

A recent study published in J Bone Miner Res (Hartikka et al 2005) has thrown some light on the etiology of IJO. Three of 20 patients had mutations of the LDL receptor-related protein 5 (LRP5). Affected family members of one proband also carried the mutation. No patient carried the collagen gene Coll 1 A1 and A2 mutations seen in osteogenesis imperfecta.

For those interested in pediatric bone densitometry, there is an excellent book: "Bone Densitometry in Growing Patients" by Sawyer, Bacharach & Fung (Humana Press, 2007).

CLIPPINGS FOR CAPENEWS

Ram Murty Shastry, Goa

Iodine Nutritional Status of exclusively breast-fed infants and their mothers in New Delhi. Rishi Gupta, Anju Seth, CS Pandav, MG Karmarkar, S Aneja. JPEM; 2006; 19 (12).

Iodine deficiency during the period of brain growth (intrauterine period and first 2 years of life) has a profound effect upon brain development since the synthesis of thyroxin is dependent upon an adequate supply of iodine. Iodine deficiency is an important public health problem in India. However, there are no published data on iodine nutritional status of pregnant/ lactating women and infants.

This cross-sectional study, with the objective of assessing the iodine nutritional status of exclusively breast-fed infants and their mothers, was conducted in the Department of Pediatrics, Kalawati Saran Children's Hospital & Lady Hardinge Medical

College, New Delhi, in collaboration with the International Council for Control of Iodine Deficiency Disorders. Spot urinary iodide (UI) and serum TSH levels were measured in 175 healthy, exclusively breast-fed infants (aged 4-24 weeks) and their mothers (aged 16-40 years). Iodine content of salt used by the participants for domestic consumption was also analyzed.

89.7% of mothers had BMI in the range of 18.5-24.9; of 175 infants, 90% had weight by height >=80% of the expected value, suggesting normal nutritional status in both mothers and infants. Median UI levels in mothers and infants were 124ugm/l and 162ugm/l respectively, with 34% mothers and 21% infants having UI levels <100ugm/l (indicating iodine deficiency). Of the mothers, 44 had mild deficiency (UI 50-99ugm/l), 14 had moderate (UI<50ugm/l) and 1 had severe iodine deficiency (UI<20ugm/l). Of the infants, 23 had mild, 11 moderate and 3 infants severe iodine deficiency, respectively. Serum TSH was elevated in 29% mothers (>5mIU/l) and 2% of infants (>9.1mIU/l). No correlation was observed between individual mother-infant UI or serum TSH levels. 96% of the salt samples tested had adequate iodine concentration, ie >15ppm.

Conclusion: The study demonstrated significant iodine deficiency in both mothers and infants despite consumption of adequately iodized salt. The iodine nutritional status of the infants was better compared to the mothers, indicating a preferential iodine supply to the infants over the mothers.

Comments:

1. The results of this study emphasize that any program of salt iodization in a population must pay particular attention to these vulnerable groups.
2. In countries known to be iodine deficient, specific iodine supplementation should be considered for pregnant/ lactating women and non-breast-fed infants to ensure optimal brain development.
3. No correlation was observed between maternal and infant UI levels. This is understandable: UI excretion, while a good indicator of iodine nutritional status of a community, has been found to have poor sensitivity for individuals.

Variable presentation of precocious puberty associated with the D564G mutation of the LHCGR gene in children with testotoxicosis. George S Jeha et al, J Pediatr 2006; 149:271-4.

The authors have reported for the first time a family with familial male-limited precocious puberty (FMPP) due to D564G mutation of the LHCGR (luteinizing hormone/ choriogonadotro-pin receptor) gene, wherein the family members showed a varied phenotypic expression (severe precocity unresponsive to therapy with compromise of the predicted final height in some members, to attainment of tall final stature in others who never received medical treatment). DNA amplification and sequencing of exon 11 of the LHCGR gene was done for the affected members and their mother, which showed an A→G nucleotide substitution at position 1691 leading to an aspartic acid to glycine substitution in the receptor at amino acid 564.

The index case presented with precocious puberty at 4½ yrs. His height was 122.9 cm (1.9 SD above the mean for age), bone age 10 yr, predicted adult height 163 cm while his mid-parental height was 182 cm. His testes were 6 cc in volume. His serum testosterone level was 254 ng/dl, while gonadotropin and HCG levels were undetectable. GnRH/ LHRH stimulation test revealed a prepubertal response. High dose ACTH-stimulation test was normal, ruling out congenital adrenal hyperplasia.

The index case has 2 maternal half-brothers with precocious puberty. One of the half-brothers, 14 yr old and 187.9 cm, began puberty between 4-5 yr. The other half-brother, 17 yr and 180.3 cm, had obvious signs of puberty at 2½ yrs of age. The mid-parental height for the half-brothers was 184 cm and both had bone ages of 18 yr. The index case's maternal grandfather had pubertal onset before the age of 5 and achieved a final adult height of 162.5 cm, which was unusually short for his family. This man's brother, who reportedly did not have precocity and achieved final stature of 185.4 cm, had a son with precocious puberty and short stature. The index case's maternal grandfather also had a son who had puberty at 2 yr and is said to be tall.

The index case was put on testolactone and spironolactone, which was later replaced by flutamide. Lupron DEPOT-PED was added at 6 yr after a GnRH stimulation test showed central activation of puberty. The index case continued to experience rapid growth and sexual maturation despite therapy.

This report puts forth the possibility that the effect of mutant LHCGR gene, D564G, on phenotypic expression of FMPP, such as final adult height, is modified by other genetic factors. Thus, more studies are needed to correlate genotype with phenotype.

CHAPTER NEWS

We extend a very warm welcome to the members who joined our peds endo family in 2007:

Dr CM BATRA, Delhi
Dr RAVI BHELONDE, Nagpur
Dr SUBRATA DEY, Kolkata
Dr RK GANGWAR, Bareilly
Dr IPS KOCHAR, Delhi
Dr PRAVIN MISHRA, Nagpur
Dr RAJIV MOHTA, Nagpur
Dr RAJ PAL SINGH, Kanpur
Dr PAWAN SUREKA, Mumbai
Dr NARESH P THAKKER, Mumbai

FROM THE SECRETARY'S DESK

Minutes of the Annual GBM: Pediatric & Endocrinology Chapter, January 12, 2007, at Mumbai.

Eight members attended the meeting, chaired by Dr. Anju Virmani.

1. The minutes of the 2006 Annual General Meeting held at New Delhi, which were already circulated to the GB and accepted on email, were formally passed.
2. The honor accorded to Dr Meena Desai by the Asia Pacific Pediatric Endocrine Society (APPES) in recognition of her lifetime service to pediatric endocrinology in the region, was informed to the GB, as was Dr PSN Menon's appointment as member of the executive council of APPES, representing our Chapter.
3. The annual report of the Chapter was presented by Dr V Bhatia.
4. The audited statement submitted by Dr Nalini Shah was found to be incomplete, and she was requested to get the auditor to complete the audit expeditiously.
5. Dr Desai informed the GB of the proposed international pediatric endocrinology meeting to be held in Mumbai in February 2008, in coordination with APPES. Dr Desai will be Chairperson of the meeting, and Dr Nalini Shah its organizing secretary. The format of the meeting is to include a one day CME for pediatricians, and a 2-3 day meeting aimed at pediatric endocrinologists and endocrinologists.
6. The GB was informed of IAP's decision, communicated to the Chapter only in September 2006, to request all Chapters to register themselves independently with Registrar of Societies and maintain independent accounts. The GB gave the responsibility of carrying out these changes to the office bearers of the Chapter, and approved the draft Memorandum of Association and Rules and Regulations of the proposed new Society, which had already been circulated by the Secretary V Bhatia, by email, to all members of the Chapter.

INDIAN ACADEMY OF PEDIATRICS

Pediatric & Adolescent Endocrinology Chapter

APPLICATION FORM FOR LIFE MEMBERSHIP

(PLEASE USE CAPITAL LETTERS ONLY)

NAME:_____**SEX**:_____
(first middle surname)

DATE OF BIRTH_____ **NATIONALITY**_____

IAP MEMBERSHIP NUMBER:_____

PROFESSIONAL DESIGNATION:_____

OFFICE ADDRESS:_____

_____

_____

PIN_____**PHONE #**:_____

HOME ADDRESS:_____

_____

_____

PIN:_____**PHONE #**:_____

E-MAIL: _____

QUALIFICATIONS: University Qualifying Year
MBBS
DCH
MD
Other

SPECIAL TRAINING IN ENDOCRINOLOGY (Please give details):
_____

SPECIAL AREA OF INTEREST IN ENDOCRINOLOGY:

Thyroid ( )	Pituitary ( )	Adrenal ( )	Growth ( )
Diabetes ( )	Gonadal ( )	Adolescence ( )	Calcium metabolism ( )

PRESENT WORK:

(a) Full time ( )	(a) Teaching ( )
(b) Private practice( )	(b) Research ( )
(c) Honorary ( )	(c) Non-teaching( )

NUMBER OF PUBLICATIONS:_____

_____

MEMBERSHIP OF MEDICAL SOCIETIES_____

MEMBERSHIP OF OTHER IAP CHAPTERS_____

DETAILS OF PAYMENT Cheque/DD #_____ ___dt_____

Bank_____ Amt_____

(Demand draft Rs 750; Outstation cheque Rs 850: in favor of Pediatric & Adolescent Endocrinology Chapter of IAP)

DECLARATION: I agree to abide by the rules and regulation of the Pediatric & Adolescent Endocrinology Chapter of IAP, in force from time to time.

PLACE
DATE APPLICANT'S SIGNATURE

(Please send/ mail to: Dr Vijayalakshmi Bhatia, Secy-Treasurer, Dept of Endocrinology, SGPGI, Rae Bareli Road, Lucknow 226001)

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7. The GB appreciated the efforts of Drs Anuradha and Vaman Khadilkar towards formulating guidelines for universal growth assessment, and getting IAP involved in this.
8. Two academic and patient care activities were proposed for the Chapter to take up in this year: (a) Guidelines on neonatal thyroid screening and treatment of congenital hypothyroidism – proposed by Anju Virmani and to be further taken up by Anju Virmani, with Meena Desai as advisor and Vaman Khadilkar, P Raghupathy and Anju Seth as co-authors (b) Creation of Turner growth charts for Indian Turner patients, using pretreatment Turner height data pooled from as many centers as possible in India – proposed by V Bhatia, coordination by V Bhatia, Nalini Shah and Vandana Jain.
9. V Bhatia informed the GB of efforts under way to fashion indigenous orchidometers as well as of efforts to get pediatric endocrinology recognized on the schedule of pediatric fellowship programs of DNB. Anju Virmani informed the GB of efforts to get needle cutters to be routinely used by diabetes patients and clinics. The GB endorsed the importance of this effort and pledged to take up this issue as a body.
10. The GB permitted the office bearers to explore setting up of a website of the Chapter, and approved appropriate advertisements in CAPENEWS against payment.
The meeting ended with thanks to the Chair.

FORTHCOMING MEETINGS:

1. ESPE 2007: 46th ESPE Meeting: 27-30 June 2007, Helsinki, Finland. Contact: Raimo Voutilainen, Dept of Paediatrics, Kuopio University Hospital, PO Box 1777, KUOPIO, FIN-70211, Finland. Tel: +358 17 172 391, Fax: +358 17 172 410, E-mail: Raimo.Voutilainen@uku.fi, www.eurospe.org/meetings.html
2**. BSPED 2007:** 35th Annual Meeting: 11-13 Sep 2007, Cambridge, UK. Details: www.bsped.org.uk/professional/meetings/index.htm
3. ISPAD 2007: 33rd Annual Meeting of the International Society for Pediatric & Adolescent Diabetes: 26-29 Sep 2007, Berlin, Germany. Contact: Olga Kordonouri, Hannover, Germany. Kordonouri@hka.de, www.ispad2007.com
4. APEG 2007: Annual Scientific Meeting: 15-18 Oct 2007, Broome, Western Australia. Email: apegasm@willorganise.com.au www.willorganise.com.au/apeg07
5. JSPE 2007: 41st Annual Meeting: 7-9 Nov 2007, Yokohama, Japan. Contact: Susumu Yokoya, Dept of Pediatrics, Toranomon Hospital, 2-2-2 Toranomon, Minato-ku, Tokyo 105-8470, Japan. Tel: +81-3-3588-1111, Fax: +81-3-3582-7068 Email: yokoya@toranomon.gr.jp
6. MBD: International Symposium on Metabolic Bone Disorders: 29 Nov, Tirupati, AP. Contact: Dr Harinarayanan.
7. ESICON 2007: 37th Annual Meeting of the Endocrine Society of India: 30 Nov-2 Dec 2007, Tirupati, AP. Contact: Dr CV Harinarayanan, Dept of Endocrinology, SVIMS, Tirupati 517507, AP. 0877- 2287777 ext 2315, 2314, 2312. Fax 0877- 2286803. email: esicon@rediffmail.com
8. PEDICON 2008: 45th National Conference of the IAP: 17-20 Jan 2008, Bhuvaneshwar. Contact: Dr Gadadhar Sarangi, Baidyanath Memorial Hospital, Kanan Vihar Phase I, Chandrasekharpur, Bhubaneswar 751031, Orissa. Tel: 0674 2741740, 09338415073, Fax 0674 2744231. email: iap@pedicon2008.org, www.pedicon2008.org
9. 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, email: kubenpillay@worldonline.co.za, www.ispad2008.com
10. 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: +90 232 278 8411 / 4644540, Fax: +90 232 278 8411/ 4649240, E-mail: atilla.buyukgebiz@gmail.com www.congrex.com/espe2008/
11. 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
12. APPES 2008: 5th Biennial Meeting of the Asia Pacific Pedistric Endocrine Society: 29 Oct- 1 Nov 2008, Seoul, Korea. Email: apegasm@willorganise.com.au.
13. ICE 2008: 13th International Congress of Endocrinology: 8-12 Nov 2008, Rio de Janeiro, Brazil. www.ice2008rio.com/
14. ESPE/LWPES: 8th Joint Meeting: 9-12 Sep 2009: New York, USA. www.lwpes-espe2009.org
15. ISPAD 2009: 35th Annual Meeting: 16-18 Sep 2009, Ljubljana, Slovenia. Contact: Tadej Battelino, E-mail: tadej.battelino@mf.uni-lj.si
16. ISPAD 2010: 36th Annual Meeting: 5-11 Sep 2010, Buenos Aires, Argentina. Contact: Olgar Ramos, E-mail: ramoso@interlink.com.ar.

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