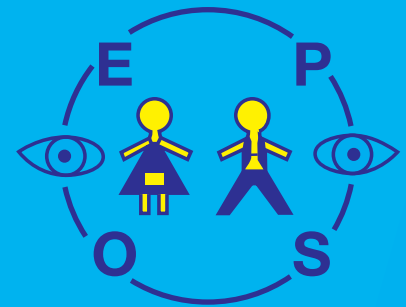




European Paediatric Ophthalmological Society
34th Annual Meeting, Leuven, Belgium, October 23-25, 2008

EPOS 2008



PROGRAMME & ABSTRACT BOOK

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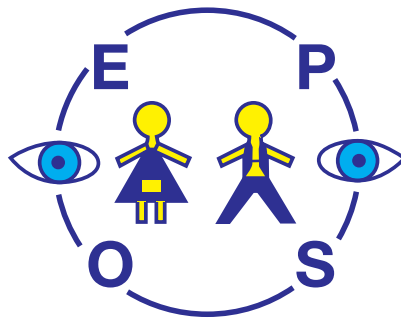
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Programme & Abstract Book

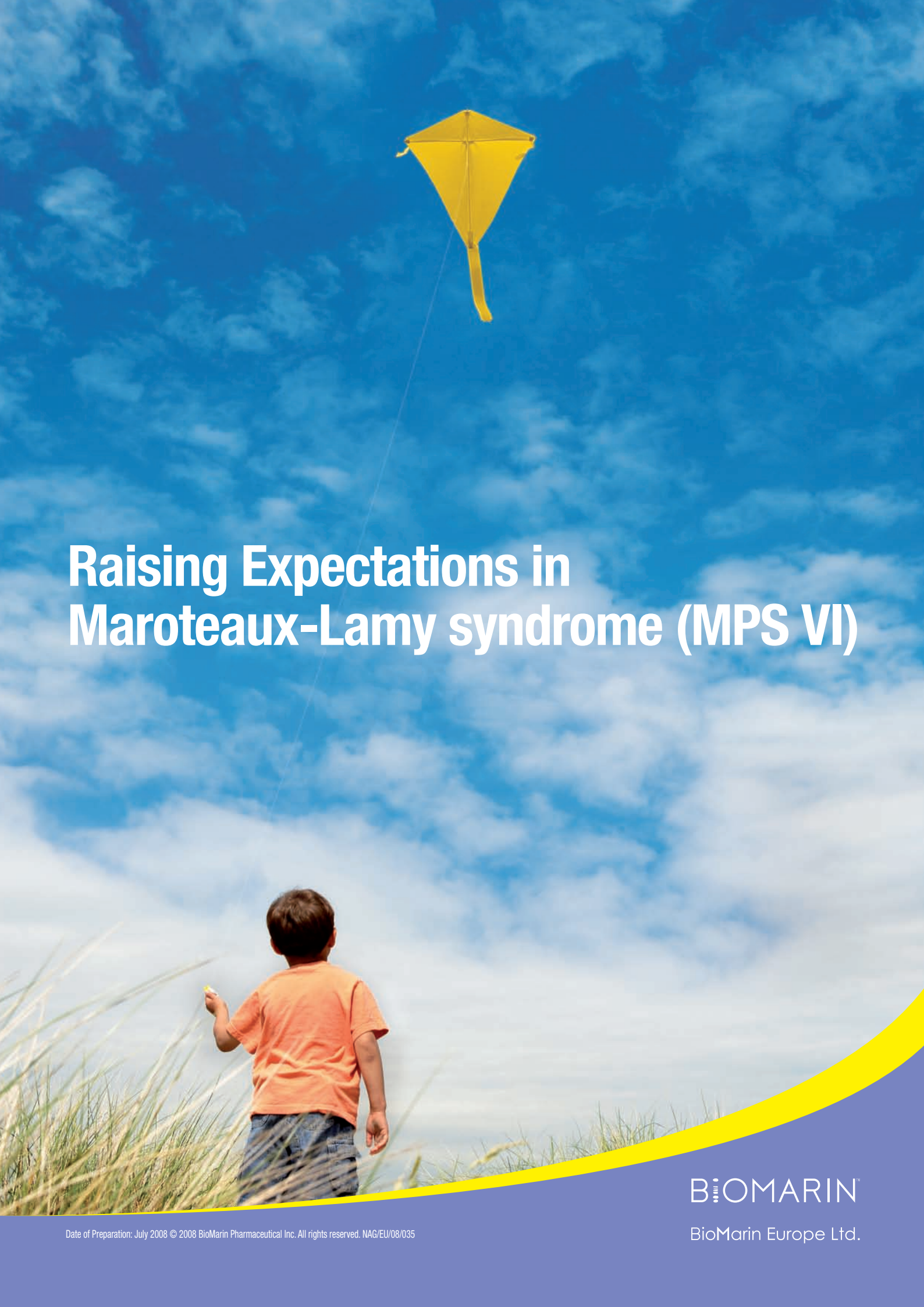
CME Credits

The EACCME has granted
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Raising Expectations in Maroteaux-Lamy syndrome (MPS VI)



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Word from the President

Dear Colleagues, Dear Friends,

It is my pleasure to welcome you to the 34th Meeting of the European Paediatric Ophthalmological Society, EPOS, in Leuven, Belgium. This meeting is hosted by Ingele Casteels and Thomy de Ravel. My sincere thanks go to them and all who helped them put together another very exciting programme, both scientifically and socially. One key to this success is the continuous support from the EPOS Office, namely Markus Preising. The Scientific Committee consisted of Ingele Casteels and Thomy de Ravel as the Congress Presidents and the Board Members of EPOS, H  l  ne Dollfus, Albert Franceschetti, Gerd Holmstr  m, Tony Moore, Nicoline Schalij-Delfos, Branka Stirn Kranjc, and myself.

The main topic is 'The Eye in Systemic Disease', and as a special feature this year there will be a hot topic session on gene therapy in retinal dystrophies. As always free papers and posters from other fields of genetic and paediatric ophthalmology are also scheduled. Outstanding speakers from abroad have accepted the invitation to update us at the meeting: Mr. James Bainbridge, ophthalmic surgeon from Moorfields Eye Hospital, London and vital team member in the gene therapy project, Professor Matthias Becker, leader of the Triemli Hospital Z  rich and active member of the International Ocular Inflammation Society, Professor Stefanie Gr  newald, Great Ormond Street London, a clinician with a keen interest in glycosylation disorders, Professor Hanne Jensen, who has longstanding experience in ophthalmic aspects of glycosylation disorders, Ms Nicola Ragge from Oxford, who has been not only researching but also publishing practical guidelines in the management of anophthalmia and microphthalmia, Professor Renier Schlingemann, ophthalmologist member of the academic medical centre in Amsterdam with a keen interest in angiogenic and anti-angiogenic factors and Professor David Taylor from London, well known for his wide experience and incredible teaching capabilities, highlighted by his book on 'Paediatric Ophthalmology'. Of course we are also very indebted to the outstanding and well-known local Belgian clinicians and researchers.

One mission of EPOS is to promote clinical and scientific paediatric ophthalmology throughout Europe and beyond, and in particular to attract young clinicians and researchers in the field. In this spirit, the concept of the meeting is to have keynote lectures, papers with sufficient time for discussion, and ample poster sessions that encourage a very vivid exchange of ideas. This year, there will be 12 keynote lectures, 25 free papers, and 35 posters. Researchers from as many as 21 countries will present their results.

As in previous years, I would like to thank all exhibitors for their significant efforts in their support of this year's meeting, and encourage all participants to visit the technical exhibit during coffee and lunch breaks.

Since the foundation of EPOS, formerly EPOG, longstanding relations between many EPOS members have been built up and continue to grow and include new clinicians and researchers in the extremely dynamic field of Paediatric and Genetic Ophthalmology. I wish new fruitful collaborations to all participants at this year's meeting and hope that personal contacts will be built up to make the Society grow even further.

Birgit Lorenz - President



Word from the organizing committee

Dear EPOS members, colleagues and friends,

A warm welcome to the 34th EPOS Meeting in Leuven. We hope that you will enjoy this meeting and that you will have some time to visit this magnificent university town.

The main topic of the programme is 'The Eye in Systemic Disease'; there will also be a session on gene therapy in retinal disease.

Special thanks go to Marlene Verlaeckt in Leuven and to Markus Preising and Heiko Klarl of the EPOS office in Germany for their continuous and efficient support in the organization of this meeting; and to the invited speakers and all presenting authors, who will contribute to the scientific programme. We also acknowledge the financial support of our advertisers and exhibitors.

On Friday night we invite you to the Faculty Club for the Congress dinner.



The Faculty Club is a unique conference and banqueting centre. We are very happy to welcome you into the calm and tranquillity of the 13th century Infirmierie of the Grand Beguinage, built in 1561. The ancient historical site of the Grand Beguinage was included in the UNESCO World Heritage List in March 2000, and the Faculty Club manages a part of this unique complex.

We welcome you to Leuven and to EPOS 2008!

Ingele Casteels and Thomy de Ravel
Marlene Verlaeckt
Werner Spileers



Short history of Leuven



Leuven is very proud of both its past and its heritage. Although the first references to the town can be traced back as far as the 9th century and in spite of its strategic location on the river Dyle, it was not until around the 11th-12th century that Leuven began to develop as an important trading centre within the Duchy of Brabant. It was at this time that its first town wall, churches, monasteries and abbeys were built. The town's once flourishing cloth trade had fallen into decline by the 14th century, but a new golden age dawned with the 15th century. The university was founded, various industrial concerns flourished and the main market square with its fine Gothic Town Hall was laid out. Its finely sculptured Town Hall and Saint Peter's church with its magnificent interior and the famous 'Last Supper' painting by Thierry Bouts, are fine examples of flamboyant Gothic architecture. Dating from the same period are the Cloth Hall, 't Sestich House and many churches, including St Gertrude's with its magnificent choir stalls. Other architectural styles are not quite so much in evidence, but they can still be found. The Romanesque Gate, the Saint Lambert's church and the remains of the first town walls are still clearly in evidence. The

13th century beguinage complex has cobbled streets and fascinating 17th century houses. The Baroque style abounds in Leuven, from St Michael's church and the interior of the St John the Baptist's church to the unrivalled stucco of the Park Abbey. Both the Park Abbey and the Arenberg Castle are exciting mixtures of architectural styles. The town still owes much of its character to the numerous university college buildings dating from the 16th and 17th centuries, many of which were renovated in the 18th century. The Van Dale College is pure Renaissance style and the College of the Falcon, Pope's College and the Arras College are neo-classical in style. The paved roads, the canal in the 18th century and the expansion of the brewery created a great impetus for trade and population growth. Unfortunately, this prosperity came to a sudden end under the reign of Joseph II when the French invaded. The most significant development for Leuven in the 19th century was the building of the railway station and the construction of Bondgenotenlaan leading to it. The two World Wars in the 20th century destroyed much of Leuven. Reconstruction works lasted well into the nineteen-sixties. On January 1st January 1995, Leuven became the capital of the province Flemish Brabant.





History of K.U.Leuven (Katholieke Universiteit)

Situated in the heart of Western Europe, K.U.Leuven has been a centre of learning for almost six centuries. Founded in 1425 by Pope Martin V, K.U.Leuven bears the double honour of being the oldest extant Catholic university in the world and the oldest university in the Low Countries.

In its early days, our university was modelled on the universities of Paris, Cologne, and Vienna. In a short time, it grew into one of the largest and most renowned universities in Europe. Its academic fame attracted numerous scholars who made valuable contributions to European culture. In the sixteenth century the humanist Desiderius Erasmus lectured here, where he founded the Collegium Trilingue in 1517 for the study of Hebrew, Latin, and Greek - the first of its kind. The tutor of the young emperor Charles V, Adriaan Cardinal Florensz of Utrecht, was a professor here before being elected in 1522 as the last non-Italian Pope before Pope John Paul II. The philologist, legal scholar, and historian Justus Lipsius taught here for many years. The mathematician Gemma Frisius helped to lay the foundations of modern science and tutored many famous scientists, including the cartographer Gerard Mercator, whose map projection is still in use, the botanist Rembert Dodoens, and the father of modern anatomy, Andreas Vesalius. In a later period, the theses of the Leuven theologian Cornelius Jansenius provoked a large and heated controversy both inside and outside the Church. In the seventeenth and eighteenth centuries, K.U.Leuven was an important training centre for Roman Catholic intellectuals from Protestant countries. At the end of the Age of Enlightenment, in 1783, the chemist Pieter Jan Minckelers discovered the suitability of coal gas for lighting. In the nineteenth century, at the instigation of Pope Leo XIII, K.U.Leuven became an important centre of Thomist philosophy.

Not all has been trouble-free, though, in the university's illustrious history. It has had its share of difficulties during the various social and political upheavals in this region from the sixteenth to the nineteenth centuries. More recently, the two World Wars of the twentieth century deeply scarred the university. In 1914, the University Hall with its precious library was set in flames and 300,000 books were reduced to ashes. Afterwards, an international solidarity campaign with a major American contribution helped construct a new library on the present Ladeuzeplein. Unfortunately, this library was burned down in 1940 during the Second World War and this time only 15,000 of its 900,000 volumes were saved. Since then, the university library, and in fact the entire university, has undergone a thorough reconstruction.

The university is located in Flanders, the Dutch-speaking northern part of Belgium. With the Dutch language's steady rise to renewed prominence, 1968 saw the university split into two new universities. The French-speaking Université Catholique de Louvain moved to the newly built campus in Louvain-la-Neuve. The Dutch-speaking Katholieke Universiteit Leuven remained in the historic town of Leuven.





The Great Beguinage

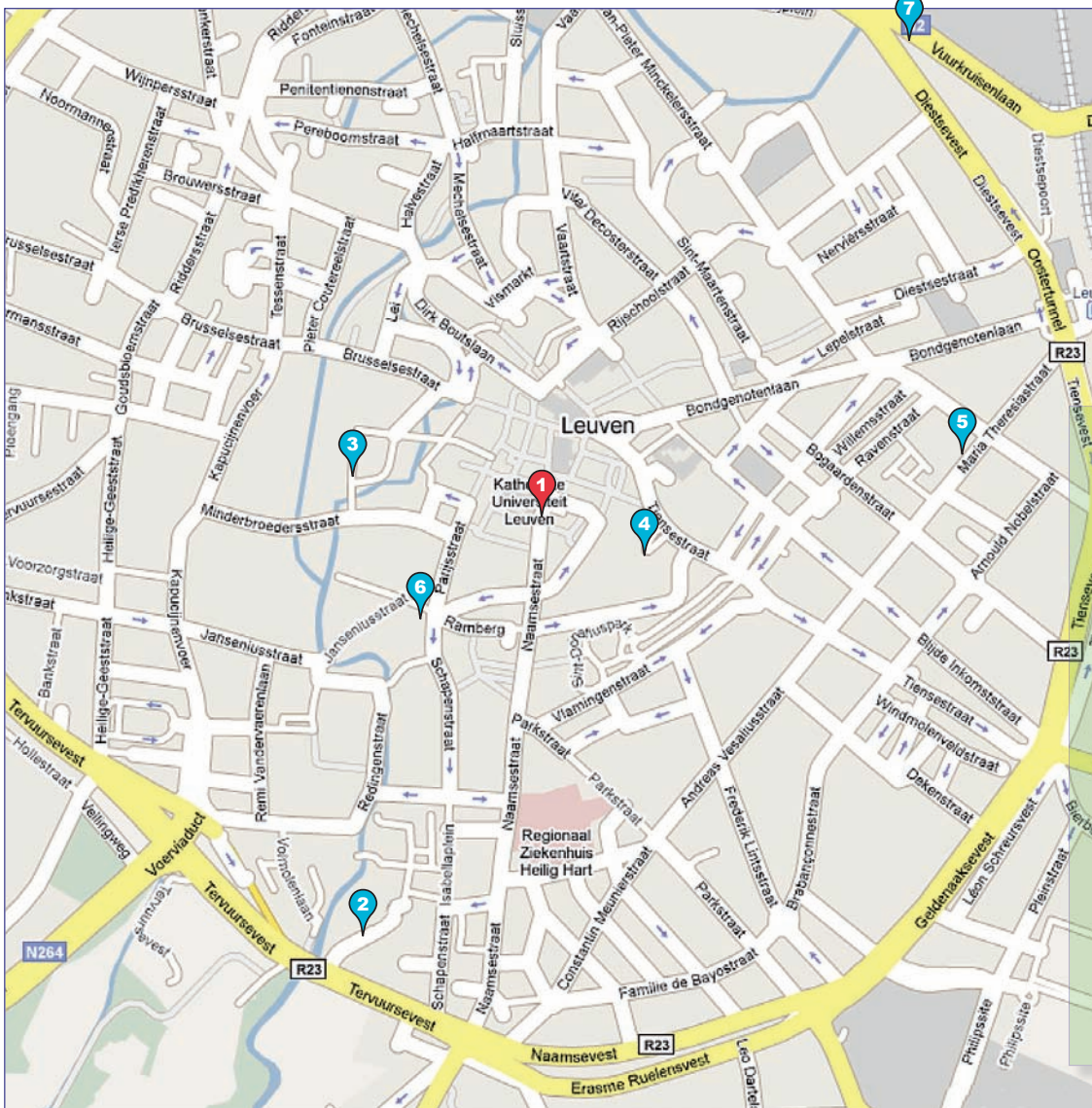
a city within the city



The Great Beguinage (“Groot Begijnhof”) in Leuven is a self-contained community that once housed a female religious foundation. The beguines have long gone; and the complex is now home to students and academics. The oldest houses date back to the 16th century, although most of them are one or-two hundred years younger. They still radiate the same serenity as they did when they were first built. The Beguinage church is the best surviving example of a Gothic building with a Baroque interior added during the Counter-Reformation.



Map of Leuven



Map Legend

- CONGRESS VENUE
- Begijnhof
Congres Hotel
- Klooster Hotel
- Holiday Inn
Garden Court
- Hotel Binnenhof
- Hotel
New Damshire
- Novotel



General Information

EPOS Board

Birgit Lorenz - President
Anthony Moore - Past President
Albert Franceschetti - Treasurer
Ingele Casteels - Secretary

Board Members

Hélène Dollfus, Gerd Holmström, Nicoline Schalijs-Delfos, Branka Stirn Kranjc

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THURSDAY • 23.10.2008

16.00	Registration
18.00-20.00	Opening Reception
20.00	Board Meeting

- 08.30 Birgit Lorenz, Presidential Welcome
 08.40 Ingele Casteels, Welcome by the local host

08.50-10.30 **SCIENTIFIC SESSION I. – METABOLIC DISORDERS**

Chairpersons: J Jaeken, D Taylor

L1	08.50	Inherited Errors of Metabolism IEOMs — <i>David Taylor</i>
L2	09.10	Clinical clues to glycosylation disorders — <i>Stephanie Grünewald</i>
L3	09.30	Ophthalmic manifestations in glycosylation disorders — <i>Hanne Jensen</i>
T1	09.50	Eye involvement in lysosomal storage disease — <i>Marije Sminia</i>
T2	10.00	Eye involvement in children with peroxisomal biogenesis disorders — <i>Liesbeth Wenniger-Prick</i>
T3	10.10	The use of the Visante™ non-contact anterior segment optical coherent tomography (OCT) in the monitoring and imaging of corneal crystals in cystinosis — <i>Rachel Pilling</i>
	10.20	<i>Discussion</i>

Topic-Related Posters in Poster Session 1

P1	Retinal dystrophy due to a vitamin E deficiency — <i>Dana Croonen</i>
P2	Long-chain 3-hydroxyacyl-CoA dehydrogenase (LCHAD) deficiency and retinopathy: a case report — <i>Eliane Delouvrier</i>
P3	Gyrate atrophy of the choroid and retina with hyper-ornithinemia: ocular findings in two siblings after reduction of plasma ornithine with diet — <i>Luisa Pinello</i>
P4	Neonatal insulin therapy to drive normoglycemic anabolism — <i>Sophie Vanhaesebrouck</i>

10.30-11.00 **Coffee Break**

11.00-12.00 **SCIENTIFIC SESSION II. – INFLAMMATORY DISEASES**

Chairpersons: P Dureau, R Schlingemann

L4	11.00	Inflammatory diseases — <i>Carine Wouters</i>
L5	11.20	Uveitis / Ocular Inflammation — <i>Matthias Becker</i>
T4	11.40	Cataract surgery in paediatric uveitis — <i>Pascal Dureau</i>
	11.50	<i>Discussion</i>

Topic-Related Posters in Poster Session 1

P5	Papain ferment in treatment of ocular developments of systemic inflammatory diseases of the connective tissue — <i>Igor Aznauryan</i>
P6	Bullous diseases in children – case reports — <i>Erika Maka</i>
P7	Immunosuppressive drug combination therapy in paediatric autoimmune ocular diseases: follow-up from 1991 — <i>Francesca Manzotti</i>
P8	Development of a uveitis screening service for juvenile idiopathic arthritis: use of rapid cycle audit — <i>Rachel Pilling</i>
P9	Orbital cellulitis associated with Adamantiades-Behcet disease — <i>Nikolaos Ziakas</i>

12.00-12.50 **SCIENTIFIC SESSION III. — PAEDIATRIC RETINA**

Chairpersons: W Spileers, B Stirn Kranjc

L6	12.00	Pharmacologic vitreolysis: potential application in vitreous surgery in children — <i>Peter Stalmans</i>
T5	12.20	Optical Coherence Tomography (OCT) in the diagnosis of foveal hypoplasia in children <i>Eva Larsson</i>
T6	12.30	Retinal haemorrhages in the differential diagnosis of child abuse — <i>Alicia Serra</i>
	12.40	<i>Discussion</i>

Topic-Related Posters in Poster Session 1

P10	Optic disc atrophy in children and neuro-imaging findings — <i>Thammanoon Surachatkumtonekul</i>
P11	Choroidal neovascularisation in an adolescent previously treated for nasopharyngeal carcinoma <i>Anna Maria Bilkiewicz-Pawelec</i>
P12	Eye and orbit changes in childhood acute lymphoblastic leukaemia — <i>Elvira Saidasheva</i>
P13	Ophthalmic problems in children with Down syndrome: findings from England <i>Alexandra Louise Creavin</i>
P14	Ophthalmic problems in children with Down syndrome: a comprehensive review <i>Alexandra Louise Creavin</i>
P15	Differences between complete and partial congenital nasolacrimal duct obstruction — <i>Joan Prat</i>
P16	Targeted visual and refractive assessment of children attending special needs schools has a high yield and an effective outcome – an audit study — <i>Meghomala Das</i>

12.50-14.45 **Lunch Meeting sponsored by Thèa**

14.45-15.45 **Poster Session 1**

15.45-16.15 **Coffee break**

16.15-17.50 **SCIENTIFIC SESSION IV. — LEBER CONGENITAL AMAUROSIS: FROM PHENOTYPE TO GENE THERAPY**

Chairpersons: M Preising, T de Ravel

L7	16.15	Leber Congenital Amaurosis anno 2008 – an update — <i>Bart Leroy</i>
T7	16.35	Genetic testing for Leber Congenital Amaurosis (LCA): a 3-year experience — <i>Frauke Coppieters</i>
T8	16.45	The ocular and olfactory phenotype of LCA patients with mutations in CEP290 <i>Suzanne Yzer</i>
L8	16.55	The spectrum of RPE65 mutations in Early Onset Severe Retinal Dystrophies (EOSRD) <i>Birgit Lorenz</i>
L9	17.15	Gene therapy for inherited retinal disease — <i>James Bainbridge</i>
	17.35	<i>Discussion</i>

19.00-22.00 **Congress Dinner at Faculty Club**

08.30-9.55 SCIENTIFIC SESSION V. – GENETICS
Chairpersons: B Lorenz, F Meire, N Ziakas

L10	08.30	Neurofibromatosis type 1 and related syndromes — <i>Eric Legius</i>
T9	08.50	Deletions in COH1 as a cause of Cohen syndrome — <i>Irina Balikova</i>
T10	09.00	Disruptions of distant regulatory elements surrounding FOXL2 in the causation of BPES syndrome — <i>Elfride De Baere</i>
T11	09.10	Functional impact of retinal cysts on ERG and visual fields in patients with X-linked retinoschisis — <i>Christoph Friedburg</i>
T12	09.20	Evaluation of Lyonization in cultured lymphocytes from carriers of choroideremia <i>Markus Preising</i>
T13	09.30	Maculopathies in gyrate atrophy of the choroid and retina — <i>Ingeborgh van den Born</i>
	09.40	<i>Discussion</i>

Topic-Related Posters in Poster Session 2

P17	Duane's retraction syndrome associated with mitochondrial DNA mutation — <i>Remi Macarez</i>
P18	Severe form of tuberous sclerosis in an eight-year-old child — <i>Lígia Ribeiro</i>
P19	Neurofibromatosis type 1 (von Reckingham) with glaucoma and sphenoid-orbital dysplasia in a paediatric patient — <i>Nikolas Ziakas</i>

9.55-10.25 Coffee Break
10.25-11.30 SCIENTIFIC SESSION VI.— RETINOPATHY OF PREMATUREITY
Chairpersons: G Holmström, A Serra

L11	10.25	Vascular endothelial growth factors and angiogenesis in eye disease — <i>Reinier Schlingemann</i>
T14	10.45	Slit lamp-delivered Laser photocoagulation of ROP — <i>Ilse De Veuster</i>
T15	10.55	IGF-I and oxygen-induced retinopathy in mice — <i>Sophie Vanhaesebrouck</i>
T16	11.05	Retinopathy of prematurity in extremely premature infants born before the 27th week – preliminary results from a Swedish national population-based study during three years <i>Gerd Holmström</i>
	11.15	<i>Discussion</i>

Topic-Related Posters in Poster Session 2

P20	Long-term clinical and visual results in extremely preterm patients with retinopathy of prematurity <i>Ludmila Kogoleva</i>
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11.30-12.45 EPOS General Assembly
12.45-13.45 Lunch Break
13.45-14.45 Poster Session 2

14.45-15.35 **SCIENTIFIC SESSION VII. – ANTERIOR SEGMENT DISEASE**

Chairpersons: *H Møller, N Ragge, AC Houtman*

T17	14.45	Surgical treatment of glaucoma associated with Sturge Weber syndrome — <i>Catherine Edelson</i>
T18	14.55	Glaucoma in aphakic and pseudophakic eyes following surgery for congenital cataract in the 1st year of life — <i>Caitriona Kirwan</i>
T19	15.05	ICD3 classification of corneal dystrophies, paediatric ophthalmology aspects — <i>Hans U Møller</i>
T20	15.15	Etiological distribution of paediatric cataracts in Hospital Sant Joan de Déu during the period 1995 to 2007 — <i>Marta Morales</i>
	15.25	<i>Discussion</i>

Topic-Related Posters in Poster Session 2

P21		The examination of the TGF β gene expression activity levels and their receptors in inborn and traumatic cataract post-operative materials –preliminary results — <i>Pawel Banasiak</i>
P22		Bilateral leukocoria in a newborn — <i>Sofie Verstraeten</i>
P23		Orbital cellulitis in children: clinical diagnostic criteria — <i>Joan Prat</i>
P24		Congenital simple ectopia lentis with unusual asymmetric bilateral lens displacement in a paediatric patient — <i>Nikolas Ziakas</i>
P25		LTBP2 mutation in autosomal recessive microspherophakia with lens luxation and megalocornea <i>Françoise Roulez</i>

15.35-16.25 **SCIENTIFIC SESSION VIII. – EYE DEVELOPMENT**

Chairpersons: *E De Baere, L Wenniger-Prick*

L12	15.35	Anophthalmia and microphthalmia — <i>Nicola Ragge</i>
T21	15.55	Anophthalmia and microphthalmia in paediatric population — <i>A Tulin Berk</i>
T22	16.05	FOXC1/PITX2 mutations and copy number changes in a Belgian-Dutch cohort of patients with anterior segment dysgenesis (ASD) — <i>Barbara D'haene</i>
	16.15	<i>Discussion</i>

16.25-16.55 **Coffee Break**



16.55-17.35 **SCIENTIFIC SESSION IX. — NEURO-OPHTHALMOLOGY**

Chairpersons: N Schalijs-Delfos, E Silva

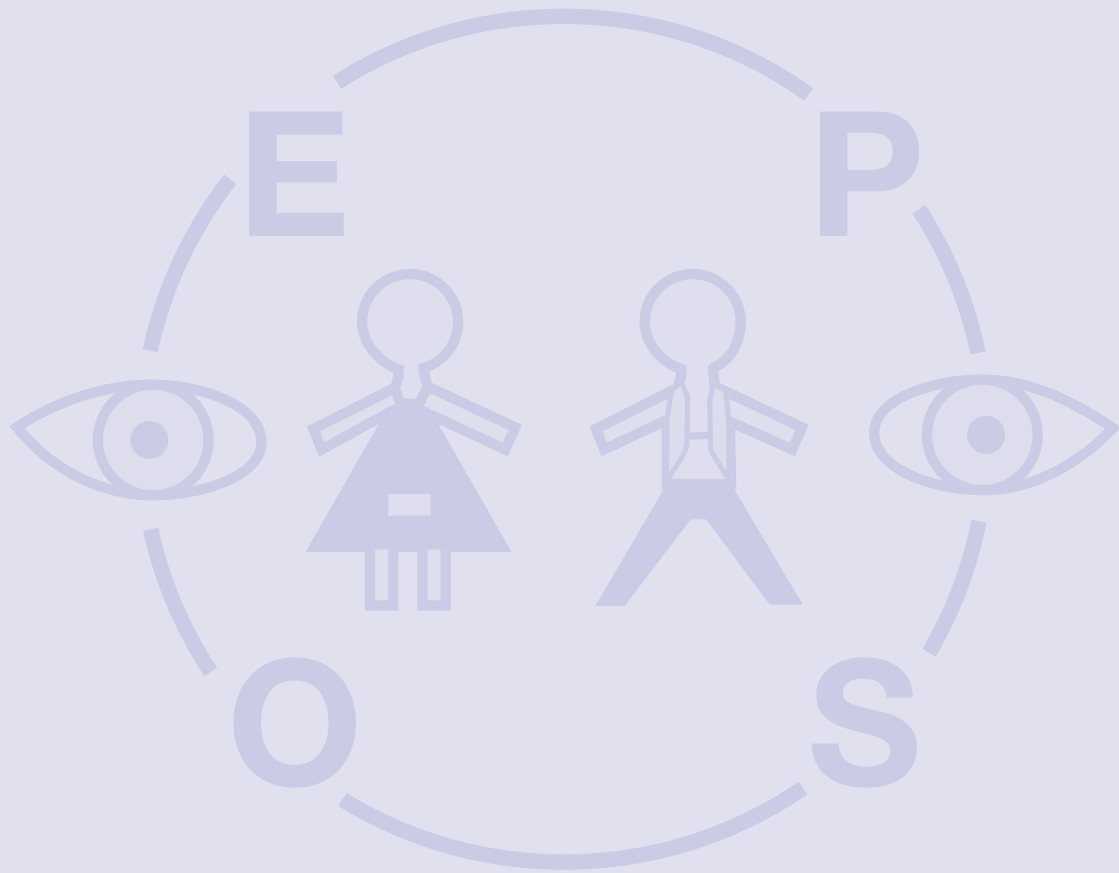
T23	16.55	Ocular features of Lyme borreliosis: an old disease revisited — <i>Arnaud Sauer</i>
T24	17.05	Morphology and function of the central retina In children and young adults with Stargardt Dystrophy — <i>Martina Jarc-Vidmar</i>
T25	17.15	Visual fields in children affected by Periventricular Leukomalacia — <i>Giorgio Porro</i>
	17.25	<i>Discussion</i>

Topic-Related Posters in Poster Session 2

P26	Myelinated retinal nerve fibres and amblyopia: 2 case reports — <i>Ellen Bartholomeeusen</i>
P27	Nutritional deficit and bilateral hyperopic amblyopia — <i>Silvia Gamboa Saavedra</i>
P28	Change of visual field defect between short term intervals in child with migraines — <i>Ungsoo Kim</i>
P29	One brother and sister with mirror image myopic anisometropia — <i>Ungsoo Kim</i>
P30	Misdiagnosis in connection with elevated intracranial pressure — <i>Krisztina Knezy</i>
P31	Ocular tilt reaction and internuclear ophthalmoplegia associated with sinus venous thrombosis: an unusual case — <i>Christina Gerth</i>
P32	Plasticity of visual functions after brain damage — <i>Dienke Wittebol-Post</i>
P33	Retinal dystrophy in CEDNIK syndrome — <i>Jan Willem R. Pott</i>
P34	Secondary glaucoma in children with Vogt-Koyanagi-Harada syndrome — <i>Aleksandra Starikova</i>
P35	Optic neuritis in children - a long term follow-up — <i>Manca Tekavčič Pompe</i>

17.35 **Scientific Awards and Closing Remarks — Birgit Lorenz**

18.00 **End of Meeting**



LECTURES

L01**Inherited Errors of Metabolism IEOMs**

Taylor D¹

¹ Institute of ChildHealth, London, UK

Mechanisms:- Accumulation of substrate Cystinuria, MPS, PKU, Tay Sachs etc Accumulation of normally minor metabolite Galactosaemia, Krabbe's Deficiency of product Congenital hypothyroidism, X-L Rickets Secondary metabolic phenomena • Apolipoprotein B • Vit E malabsorption • β lipoproteinaemia • retinal dystrophy Establishing the pattern of inheritance (AD, AR, X-LR, Mitochondrial) and the behaviour may suggest a template.

5 Steps to Establish IEOMs

1. Think Metabolic! FH/Consanguinity "Intoxication" but no Toxin "Infection" but no Organism Exaggerated response to Intercurrent Infection Poor Response to Rx Catastrophic Illness in the newborn Recurrent Acute Encephalopathy "Two episodes = metabolic"
2. The course of the disease - small molecule vs organelle disease? - regression vs pseudo-regression?
3. The Extent of the disease -anatomy, distribution & pathology?
4. The response to Rx Brisk or poor response to conventional or supportive therapy?
5. The library / web Ophthalmological Presentations and Involvement
1. Neurological disease – Acute encephalopathy – Chronic Encephalopathy: with/without systemic findings • Muscle • Bone disease & dysmorphism • Storage • Skin or Connective Tissue • Behavioural Syndromes
2. Metabolic Acidoses 3. Hepatic Syndromes 4. Cardiovascular Syndromes

L02

Clinical clues to glycosylation disorders

Grünewald S¹

¹ *Great Ormond Street Hospital*

Since the first CDG patients were described in 1984, the progress in the field of human glycosylation disorders has been impressive. So far, more than thirty congenital disorders of glycosylation defects have been characterised on a biochemical and molecular genetic level and were added to the ever growing CDG alphabet. In the first years, after CDG-Ia and CDG-Ib were recognised, CDG patients have been picked up mainly because they presented with the “classical features” of CDG-Ia (inverted nipples, abnormal fat pads, cerebellar hypoplasia) or of CDG-Ib (protein-losing enteropathy). Broadening the screening for CDG, the number of CDG-Ia patients identified with a less typical presentation is increasing, including children with nearly normal psychomotor development and / or normal cerebellum. Extending the screening for CDG in patients with unexplained and mainly neurological problems using the isoelectric focusing of transferrin, the patient group of CDG-x rapidly enlarged, awaiting the characterisation of their underlying glycosylation defect. With identification of hundreds of patient with glycosylation defects over the years, we have learned that any organ system at any time to any degree can be affected. The challenges for clinicians are manifold in the ever growing field of glycosylation disorders: the clinical heterogeneity in-between the different (recognized so far) CDG subtypes is immense, the clinical variation of CDG patients identified with the same CDG subtype can be extraordinary, CDG can mimic other metabolic disorders such as mitochondriopathy and any tissue/cell system can be affected. The add-on challenge anno 2008 is to (re-)structure the work-up of any patient suspicious for a glycosylation defect taking in account the availability of new diagnostic tests and procedures.

L03

Ophthalmic manifestations in glycosylation disorders

Jensen H¹

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Introduction: To review the clinical features of congenital disorders of glycosylation with a focus on ophthalmic features and report on longterm observations on 16 cases with CDG 1a.

Methods: Retrospective case series and literature review

Results: CDG 1a is the most common of the more than 30 known types defined by defects in the different steps of the synthetic pathway. The typical phenotype of the CDG 1a regarding ophthalmological findings includes esotropia, refractive myopia and progressive retinal dystrophy. Sixteen cases are presented that illustrate the typical clinical features of the disorder at different ages.

Conclusion: Almost all children had retinitis pigmentosa from an early age and the parents were well aware of the diagnosis. But as the children are wheelchair dependent and moderate to severe mentally retarded the families does not find that the children have severe visual problems despite it can be demonstrated objectively. It is therefore important to demonstrate the problems for caretakers in order to prevent the children from having unnecessary pitfalls.

L04**Eye involvement in systemic inflammatory diseases in children**

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Uveitis can be part of a variety of systemic inflammatory illness in children, most frequently juvenile idiopathic arthritis (JIA) but also pediatric sarcoidosis, Behçet's disease, Crohn's disease, and tubulointerstitial nephritis. In JIA, the estimated prevalence of uveitis is 17-22% of all patients; the risk of uveitis is highest in very young girls with the oligoarticular subtype of JIA and presence of antinuclear antibodies (ANA) with frequencies up to 25%. Complications of uveitis in children with JIA are numerous, including cataract and band keratopathy in up to two thirds of patients, glaucoma and impaired vision in up to one third of children. Guidelines for regular ophthalmologic evaluation comprising a high screening frequency and a long screening period have been developed. Scleritis and retinopathy can be observed in children with systemic lupus erythematosus and systemic vasculitides. Conjunctivitis is seen in acute inflammatory illnesses such as Reiter's syndrome and Kawasaki disease. Optic disc changes associated with anterior uveitis are distinguishing features of CINCA, a hereditary autoinflammatory syndrome. Last but not least to mention are the ocular complications of systemic therapies such as corticosteroids and hydroxychloroquine. Patients with anterior uveitis may be treated with topical therapy alone but patients with posterior uveitis and those with sight threatening complications of anterior uveitis usually require systemic treatment especially if the disease is bilateral. The mainstay of treatment is corticosteroids and additional immunosuppressive agents such as cyclosporin and mycophenolate are used when necessary. There remains a significant cohort of patients in whom this therapy is either not tolerated or is ineffective. The use of the anti-tumour necrosis factor (TNF) antibodies has been very successful in controlling immune-inflammatory diseases including JIA. TNF is known to play a key role in ocular inflammation as shown by animal studies and its detection in the ocular fluids of inflamed eyes in man. The data supporting anti-TNF drugs as a potential treatment option in refractory idiopathic or JIA-associated uveitis in children are presented.

L05**Uveitis / Ocular Inflammation**

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Ocular involvement is common in pediatric rheumatologic diseases. Pediatric rheumatologists should become familiar with the broad spectrums of these ocular disorders. As important members of the interdisciplinary team, they may have to manage more serious cases of inflammatory eye disease. Therefore, a close collaboration between the treating rheumatologist and the ophthalmologist is a prerequisite to prevent potentially devastating visual outcomes. Therapeutic interventions such as topical steroids, systemic immunosuppressants, and biologics must balance the necessity of controlling ocular inflammation and the adverse effects of these treatments on a growing child. The association of systemic corticotherapy and immunosuppressants in pediatric relapsing or steroid-dependent uveitis allows good recovery of visual acuity, fewer complications, and a minimization of side effects. The talk reviews clinical findings and current knowledge on study results for a better understanding of ocular inflammation in childhood.

L06**Pharmacologic vitreolysis: potential application in vitreous surgery in children**

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Presently, several phase II studies have shown the potential of microplasmin to create a posterior vitreous detachment after intravitreal injection. Injection of microplasmin one week before vitrectomy in adult patients with vitreomacular traction has shown that release of traction can be obtained, omitting the need for vitrectomy. In addition, in patients treated with microplasmin without a vitreous detachment present at time of surgery, creating the vitreous detachment during vitrectomy was facilitated. In young children, vitrectomy is often difficult to perform because of the extreme adherence of the posterior vitreous membrane on the retinal surface. Hence, microplasmin could be a promising agent to facilitate vitreous surgery in children.

L07**Leber Congenital Amaurosis Anno 2008 - An Update**

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Introduction: Leber congenital amaurosis (LCA) is a genetically and clinically heterogenous hereditary retinal disorder causing profound visual loss, nystagmus, poorly reactive pupils and a markedly diminished electroretinogram (ERG) due to the loss of photoreceptor function. Theodor Leber first described the condition in 1869 as a severe form of retinitis pigmentosa presenting in infancy or early childhood, with the absence of photoreceptor function.

Methods: An overview of the current status of knowledge regarding phenotypes and genotypes will be presented.

Results: The molecular genetics of LCA has been studied rather intensely over the last decade. All eleven genes so far identified, GUCY2D, RPE65, CRX, AIPL1, CRB1, RPGRIP1, RDH12, IMPDH1, TULP1, CEP290 & LCA5 have different functions in the retina. Together they account for 60 to 70% of all patients. Two additional loci have also been identified, on chromosomes 14q24 (LCA 3) and 1p36 (LCA 9) in consanguineous families. It is also becoming increasingly clear that specific phenotypes can be attributed to specific genotypes. In addition, successful gene therapy of RPE65-related LCA in a Briard dog model of the disease has now been translated into treatment trials in humans in both the UK and the USA, with initial success.

Conclusion: With the discovery of 11 genes to date which account for a majority of LCA patients, and the identification of genotype-phenotype correlations, knowledge about the pathogenesis of LCA is increasing rapidly. In addition, the advent of gene therapy in humans with RPE65-related LCA, makes this an exciting era for patients, parents and genetic ophthalmologists alike.

L08**The Spectrum of RPE65 Mutations in Early Onset Severe Retinal Dystrophies (EOSRD)**

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Introduction: Recently two phase I/II studies reported safe application of Adeno-Associated Virus (AAV) assisted gene transfer to the retinal pigment epithelium (RPE) in patients with mutations in the RPE65 gene coding for the retinol isomerohydrolase. Both studies treated basically patients with an advanced disease stage. A slight positive effect on visual function could be shown even then.

Patients: We identified 13 patients with RPE65 mutations on a molecular level and performed longitudinal and cross-sectional phenotypic studies during up to 16 years using visual acuity, refraction, fundus photography, static and kinetic perimetry, two colour threshold perimetry, electroretinography (ERG, mfERG), optical coherence tomography (OCT), fundus autofluorescence (FAF).

Results: The patients harboured various combinations of RPE65 mutations. These included homozygous and compound heterozygous mutations. The phenotype in early childhood ranged from nightblindness but with normal day vision to both night blindness and severely reduced day vision down to 0.1. Common to all patients was a severely reduced or absent FAF despite an almost normal fundus appearance. Ganzfeld ERG was only measurable in the least severe cases. OCT showed a preserved retinal stratification on STRATUS OCT. Yet, with the high resolution SPECTRALIS OCT clear disorganisation of the outer retina was present even with well preserved cone function.

Discussion: This report presents a collection of data on the variability of the phenotype correlating with different levels of the retinol isomerohydrolase activity. These data are important in view of actual and future gene therapeutic trials.

L09

Gene therapy for inherited retinal disease

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Introduction: Early-onset severe retinal dystrophy caused by mutations in RPE65, which encodes the retinal isomerase RPE65, is associated with poor vision at birth and complete loss of vision in early adulthood. There is no treatment available but the condition may be amenable to gene therapy.

Methods: In a phase I/II clinical trial we administered subretinally to three young adult subjects recombinant adeno-associated virus vector (rAAV-2/2) expressing RPE65 cDNA under the control of a human RPE65 promoter.

Results: There have been no serious adverse events. Examination of the patients up to 12 months after experimental treatment indicates that subretinal administration of rAAV vector is safe in humans and can lead to improved visual function.

Conclusion: These findings support further clinical studies of this experimental approach in other patients with mutant RPE65

L10**Neurofibromatosis type 1 and related syndromes**

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Neurofibromatosis type 1 (NF1) is a frequent autosomal dominant condition caused by heterozygous mutations in the tumor suppressor gene NF1 coding for neurofibromin. Neurofibromin is a negative regulator of an important signal transduction pathway (RAS-MAPkinase pathway) and this is reflected in the many different clinical problems that can be seen in individuals with NF1. Aside from the cognitive and behavioral phenotypes the tumor phenotype is very striking. Loss of the wild type NF1 allele in a variety of cell types results in tumor formation. Benign nervous system tumors such as neurofibromas and gliomas are very frequent in NF1. Malignant peripheral nerve sheath tumors (MPNST) occur with an increased frequency in NF1 individuals with a life time risk of 10%. MPNSTs have an overall bad prognosis and are more frequent in the NF1 patients with a NF1 microdeletion. Abnormalities of melanocyte proliferation are virtually always present and result in café-au-lait spots, skin fold freckling and iris hamartomas (Lisch nodules). Melanocytes from café-au-lait spots show a bi-allelic inactivation of the NF1 gene similar to benign neurofibromas (biallelic inactivation of NF1 gene in Schwann cells). Somatic mutations in RAS genes and several of their upstream and downstream molecules are found in different human malignancies. RAS proteins play key roles in normal cell growth, but also in malignant transformation and learning and memory. In recent years germline mutations in genes coding for components of the RAS signalling cascade have been recognized in a group of phenotypically overlapping disorders, referred to as the neuro-cardio-facial-cutaneous syndromes (Noonan syndrome, Leopard syndrome, CFC syndrome, Costello syndrome, Neurofibromatosis type 1). These syndromes present with variable degrees of psychomotor delay, cardiac abnormalities, facial dysmorphism, short stature, skin defects and increased cancer risk. These findings point to important roles for this evolutionary conserved pathway not only in oncogenesis, but also in cognition, growth and development. We recently identified a new member of this group by reporting SPRED1 mutations in patients with a neurofibromatosis type 1-like syndrome.

L11**Vascular Endothelial Growth Factors And Angiogenesis In Eye Disease**

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Blindness from exudative age-related macular degeneration (AMD), retinopathy of prematurity and proliferative diabetic retinopathy (PDR) is caused by angiogenesis and fibrosis in the subretinal space and vitreous cavity of the eye, respectively. In these conditions a wound healing-like response occurs in which neovascularization is accompanied by influx of inflammatory cells and development of myofibroblasts. Choroidal neovascularization (CNV) in AMD may regress, leaving an atrophic area, or may progress to a fibrotic phase and true scar formation. In ROP and PDR, angiogenesis is followed by a fibrotic phase with fibrovascular contraction causing hemorrhages, retinal detachment and blindness. Several growth factors have been shown to be involved in this train of events e.g. vascular endothelial growth factor-A (VEGF), TGF- β , HGF, PDGF, and the pro-fibrotic connective tissue growth factor (CTGF) [1-5]. In exudative AMD and PDR, VEGF is clearly the primary angiogenesis factor [1]. This has most definitely been shown in human patients. VEGF inhibitors such as Lucentis and Avastin have a marked effect on leakage and growth of subretinal neovascularization in patients with AMD and induce the regression of pre-retinal vascularization in patients with PDR. The clinical introduction of these drugs means a landslide in the options for the treatment of these and other diseases of the retina with neovascularization or vascular leakage, and holds promise for preventing blindness in ROP. The causal factors of fibrosis and scarring, and the factors which regulate the transition from angiogenesis to the fibrotic phase, which we propose to call the angio-fibrotic switch, remain largely unknown. Recent findings from our group support a novel concept where angiogenesis in the vitreous is driven by VEGF, which amongst other factors upregulates the pro-fibrotic factor CTGF in various cell types in the newly formed neovascular membranes. Increasing levels of CTGF inactivate VEGF by reduction of production and by complex formation, and when the equilibrium between these two factors shifts to a certain threshold ratio, the angio-fibrotic switch occurs and fibrosis driven by excess CTGF leads to scarring and blindness. What is the evidence that this hypothesis is true? In a recent study, we found that in PDR patients, vitreous CTGF levels correlated significantly with degree of fibrosis and with VEGF levels, but not with neovascularization, whereas VEGF levels correlated only with neovascularization. The ratio of CTGF and VEGF was the strongest predictor of degree of fibrosis. In addition, in clinical cases of PDR, as predicted by our findings in vitreous, patients with PDR demonstrated a temporary increase in intra-ocular fibrosis after anti-VEGF treatment. This concept identifies CTGF as a major potential therapeutic target in the treatment of ocular fibrosis, in particular in combination with anti-VEGF agents.

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L12**Anophthalmia and microphthalmia - practical management and emerging genetic syndromes**

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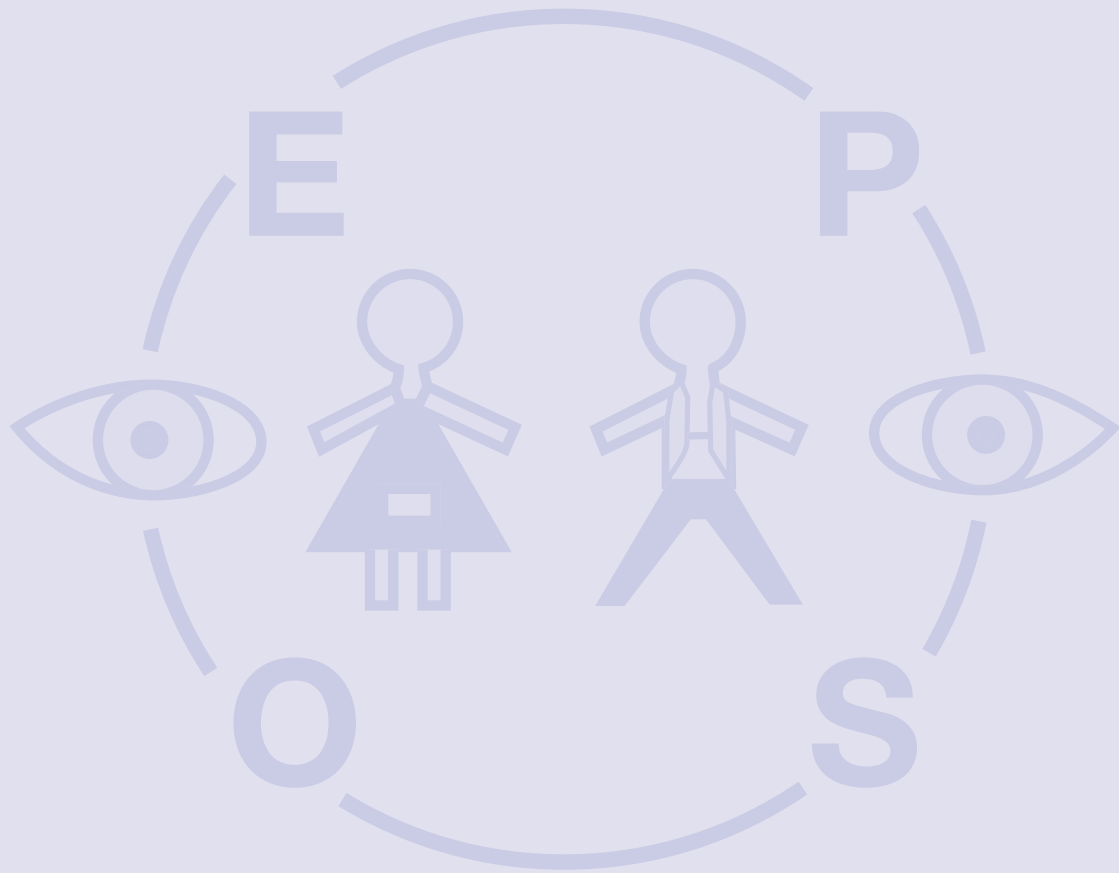
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Introduction: Anophthalmia and microphthalmia (AM) are rare developmental defects that arise as a result of a disturbance of the morphogenetic pathway that controls eye development. They may be isolated, or associated with other ocular manifestations e.g. coloboma, or as part of a syndrome. The management of children with AM often requires multidisciplinary input to allow best visual and prosthetic management together with management of associated conditions. Recent identification of novel genes for AM has important implications in diagnosing syndromes and genetic testing in these families.

Methods: We performed an observational study of hydrophilic expanders in early socket expansion of anophthalmic sockets. We analysed clinical features and molecular genetic investigation of a large cohort of patients with AM ascertained from a national specialist clinic.

Results: Hydrophilic expanders had very little morbidity associated with their use: the main complications being infection or loss, requiring replacement. New genetic syndromes, including those caused by mutations of SOX2, OTX2, BMP4, PTCH and hedgehog (SHH) signalling will be described.

Conclusion: Hydrophilic expanders are a useful adjunct in the early management of the anophthalmic socket. Rapid progress in identification of new AM syndromes means that over 20% of affected individuals can now receive a genetic diagnosis which helps to provide informed genetic counselling and directed medical care.



Paper Presentations

T01

Eye involvement in lysosomal storage disease

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Introduction: The Academic Medical Centre is a tertiary referral centre for lysosomal storage diseases (LSDs). These very uncommon metabolic diseases are often accompanied by eye abnormalities. Enzyme Replacement Therapy has obtained an important place in the treatment of several LSDs, including MPS-I and Fabry disease. In all children eye examination is performed prior to and during ERT in our centre.

Purpose: To present the clinical data of two groups of children with LSD; MPS-1 (14 children) and Fabry disease (15 children). And to report shortly on ERT.

Methods: a retrospective review of the records of these 29 children was performed. The data collected were age of onset, inheritance, presenting symptoms, eye involvement/ symptoms and treatment modalities. The eye examination included visual acuity, slitlamp examination, intraocular pressure measurement and examination of the retina.

Results: All 14 children (7 girls, 7 boys) with MPS-I had corneal clouding. 5 patients were treated for high intraocular pressure. 6 patients had an abnormal optic disc or VER. The majority of patients had an important visual impairment. All patients in this group were treated with ERT. None of the children in the Fabry disease group (4 boys, 11 girls) had visual impairment. All but 4 children had cornea verticillata. In 8 patients conjunctival or retinal tortuosity was seen. No cataract was found in any of the patients. 9 children had one or two siblings with the same disease. 6 patients in this group were treated with ERT. **Conclusions:** LSDs have a diverse clinical spectrum. Early recognition is difficult but very important. ERT has obtained an important place in the treatment of LSDs. Recognition of the eye symptoms by the ophthalmologist is very important for a timely start with ERT.

T02

Eye involvement in children with Peroxisomal Biogenesis disorders(PBD)

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Introduction: Presentation of two groups of children with PBD: 8 with rhizomelic chondrodysplasia punctata (RCDP), 11 with mild forms of the Zellweger spectrum (ZS)

Methods: retrospective study of neurological and ophthalmological data (age of onset, presenting symptoms, neurological and other organ involvement and survival age) In patients with longer survival, repeated ERG's with fundus photographs are presented.

Results: 4/8 children with RCPD died during follow-up (age between 1 and 6 years). Bilateral cataract extraction with well tolerated contactlens fitting was performed in all children; 2/16 eyes had surgical complications (elevated IOP, shallow anterior chamber, and/or posterior synechiae) In all children VA improved after cataract extraction. Within the ZS patients VA varied from LP to 0,5. RP was always progressively present with night blindness, colour vision- and visual field defects. Refractive errors and sensorineural hearing loss were often present. No serious cataracts were found in this group but strabismus, nystagmus and amblyopia were often present.

Conclusion: Eye involvement is common in children with PBD. Although therapy in these children is only symptomatic, we advocate regular ophthalmic follow-up with monitoring of their visual functions.

T03

The Use of the Visante™ non-contact anterior segment optical coherent tomography (OCT) in the monitoring and imaging of corneal crystals in cystinosis

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Introduction: We demonstrate the use of the Visante® non-contact anterior segment OCT in the monitoring and imaging of paediatric patients attending bimonthly cystinosis clinics. Corneal features of cystinosis include needle shaped crystals found initially in the superficial and peripheral cornea. These subsequently progress centrally and posteriorly to involve endothelium. This process carries a poor prognosis with ensuing corneal oedema and epithelial erosions. Corneal thickness is also increased.

Method: High resolution OCT scans (3mm x 10mm, 512 A-scans in 250 ms with an axial resolution of 18µm) were used for the purpose of recording density and position of corneal crystals.

Results: Cystinosis crystals are clearly visible using high resolution OCT. Furthermore, the amount of reflectivity of the corneal crystals in the OCT images correlates with clinical photographs in terms of number and density of crystals.

Discussion: We highlight the use of non contact anterior segment OCT as a non-invasive tool for monitoring of cystinosis crystals. It confers several advantages to previously reported monitoring systems. It is easier to operate than an anterior segment camera with a very fast acquisition time (250ms) which is particularly important in the paediatric patient. The depth of crystals can be more easily scrutinized than on slit lamp examination. Corneal pachymetry can also be recorded. OCT may be a more objective method of monitoring the effect of topical cysteamine drops potentially removing observer bias. Thus, one potential benefit may be to improve parental compliance with chronic topical therapy, if they are able to visualize a quantifiable reduction in crystal density on OCT.

Conclusion: High resolution OCT is an effective tool for the monitoring and visualization of corneal crystals in cystinosis. To our knowledge the use of anterior segment OCT in imaging corneal cystinosis crystals has not previously been reported.

T04

Cataract surgery in paediatric uveitis

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Introduction: Uveitis in children, often related to juvenile idiopathic arthritis, can lead to a cataract. The aetiology is generally a combination of the inflammation itself and steroid treatment. The surgical technique remains debated. The goal of this study was to illustrate the indications for surgery, the technique and the prognosis in these high risk eyes.

Methods: Retrospective study of 14 eyes of 10 children operated from 2005 to 2007. The following points were noted: Aetiology of uveitis, preoperative visual acuity and anatomy, surgical technique, perioperative treatment, anatomical and functional results.

Results: Mean age at the time of surgery was 6.3 years (2 months-9.5 years). Juvenile idiopathic arthritis was the main aetiology. Preoperative VA was inferior to 0.3 in all cases. Preoperative slit-lamp examination revealed a total cataract with circumferential iridolenticular synechiae in all cases, band keratopathy in 8 eyes. The surgical technique consisted of iris retractors, blue vision, anterior capsulorhexis, bimanual lens aspiration. An acrylic hydrophobic IOL was implanted in 10 eyes. A general and local intensive perioperative steroid therapy was given in every case. Four eyes experienced a severe postoperative inflammation despite this treatment. Visual acuity after a mean 10 months follow-up was poor (<0.2) in 4 eyes, median (0.2-0.4) in 4 eyes, and good (> 0.4) in 6 eyes.

Conclusion: Bimanual lens aspiration is effective for cataracts in paediatric uveitis. In-the-bag implantation improves the visual prognosis but requires a strict perioperative control of inflammation, by the mean of steroids and in some cases immunosuppressive treatments.

T05

Optical Coherence Tomography (OCT) in the diagnosis of foveal hypoplasia in children

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Introduction: The aim of this study was to investigate whether Optical Coherence Tomography (OCT) can be a help in the diagnosis of foveal hypoplasia in children.

Method: Foveal hypoplasia is well known in albinism and aniridia. Children with this diagnosis were examined with Stratus OCT 3 version 4.0.1. Macular thickness was quantified with a “fast macular map protocol”. The average thickness/volume of the macula was measured in nine modified ETDRS areas (A1 – A9). Comparison with a normal population of children of the same ages was performed.

Results: The macular thickness was measured in 13 children with albinism and three children and one adult with aniridia. Children with albinism and aniridia had significantly thicker central macula (A1) and foveola than children in the control group. Children with albinism, however, had a general thinning of the inner (A2-A5) and outer macular areas (A6-A9).

Conclusion: OCT is a useful tool in the diagnosis of foveal hypoplasia in children, even in the case of nystagmus.

T06

Retinal haemorrhages in the differential diagnosis of child abuse

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Introduction: Shaken baby syndrome (SBS) is an extremely serious form of abusive head trauma that occurs when a child is subjected to rapid acceleration, deceleration, and rotational forces, with or without impact, resulting in a unique constellation of intracranial, intraocular, and cervical spinal cord injuries. Retinal hemorrhages (RH) are one of the most characteristic findings, but can be associated with accidental head trauma and with a variety of systemic illnesses, not enough documented in the literature.

Aim: To determine the prevalence of retinal hemorrhages in head trauma due to vertical fall, convulsions, apnoea or whooping cough with the purpose of facilitating the differential diagnosis of the cases of shaken baby syndrome.

Methods: Prospective study on children aged 15 days to 2 years admitted to our hospital with one of the diagnosis listed above over a period of two years (may 2004 - may 2006). A complete neurological examination and dilated fundus examination were performed within 72 hours of admission. If RH were detected, further investigation was undertaken to rule out systemic disorder or maltreatment. Results: 516 children were included, with the following diagnosis: 182 children with first seizure, 154 with head trauma due to vertical fall, 109 with apnoea and 71 with whooping cough. RH were found in 7 patients: 3 after accidental vertical fall (in all cases brain haemorrhage was found in MRI), one after cardiopulmonary reanimation for apnoea, and 3 cases were admitted for first seizure but further investigation confirmed SBS

Conclusion: Accidental head trauma can produce RH in association with brain haemorrhages. Convulsions, cough or apnoea alone are unlikely to cause retinal haemorrhages in children less than 2 years. Therefore, if retinal haemorrhages are detected, investigation into the possibility of non-accidental injury is essential. In addition, it is important to carry out a fundoscopic examination in all infants with an apparent unprovoked seizure, as the documentation of retinal haemorrhages could be the clue to maltreatment diagnosis.

T07

Genetic testing for Leber Congenital Amaurosis (LCA): a 3-year experience

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LCA is genetically highly heterogeneous with an involvement of large disease genes, which hamper genetic testing. The purpose of this study was to determine the prevalence of mutations in 6 common LCA genes in 98 LCA patients, mainly of Belgian origin, in order to optimize a genetic screening strategy for LCA. First, LCA chip screening revealed a mutation in 34% of all patients. Second, direct sequencing of AIPL1, CRB1, CRX, GUCY2D, and RPE65 in chip-negative tested patients revealed causal mutations in 3%. Third, we performed targeted mutation analysis of the CEP290 mutation c.2991+1655A>G. We found this mutation in both homozygous (2/98) and heterozygous (16/98) state. A second mutation was identified through sequencing of the total coding region. Subsequently, the remaining patients were screened for 4 additional recurrent CEP290 mutations. The allele frequencies of the most common mutations p.Lys1575X and c.[3310-1G>A;3310C>A] were respectively 13% and 9%. Finally, sequencing of the total coding region of CEP290 is being performed in the remaining cases. So far, this revealed a homozygous mutation in one case. In addition, we identified RDH12 mutations in 2 families with early-onset retinal dystrophy, and CEP290 mutations in 3 families with Senior-Loken syndrome. In conclusion, we found both disease causing mutations in 59% of all LCA patients (22% in CEP290; 18% in CRB1; 8% in RPE65; 6% in GUCY2D; 3% in AIPL1 and 2% in CRX). A combined genetic testing strategy consisting of LCA chip analysis and targeted mutation screening of 3 recurrent CEP290 mutations represented an efficient first-pass screening, revealing causal mutations in 55% of our LCA population.

T08

The ocular and olfactory phenotype of LCA patients with mutations in CEP290

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Purpose: To study the ocular and olfactory characteristics in Leber congenital amaurosis (LCA) patients carrying homozygous or compound heterozygous CEP290 mutations and to establish a genotype-phenotype correlation.

Methods: 11 LCA patients with proven mutations in CEP290 were identified for phenotyping purposes. Clinical data were analysed retrospectively and all patients underwent ophthalmic examination, including visual acuity (VA), refraction, slit lamp examination and funduscopy. To investigate features of Joubert syndrome we performed neuro-imaging (CT, MRI) and ultrasonography of the kidneys. Olfactory investigation with the brief smell identification test was performed in 2 LCA patients and 7 heterozygous carriers.

Results: Visual acuity ranged from light perception negative to 0.1. All patients were high hypermetropes (ranging from +8.00 to +13.00). Anterior segment exams ranged from normal to full ectatic corneas. Funduscopy ranged from essentially normal in childhood to subtle abnormalities in the posterior pole and periphery with aging. One patient developed Coat's-like exudative vasculopathy. Neuroimaging and ultrasonography of the kidneys did not reveal any abnormalities. Joubert syndrome was excluded in all patients even though three patients had learning and balance difficulties. Both patients and 4 carriers showed significantly reduced values on the brief smell identification test.

Conclusions: Pathogenic mutations in the CEP290 gene lead to a clinically severe form of LCA with mild but progressive funduscopy abnormalities. The CEP290 phenotype seems distinguishable from other types of LCA. Patients with CEP290 mutations might develop significant olfactory dysfunction which may be indicative for the gene involved in LCA patients not previously genotyped.

T09

Deletions in COH1 as a cause of Cohen syndrome

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Cohen syndrome is an autosomal recessive disorder characterized by mental retardation, typical face, truncal obesity, microcephaly and retinal dystrophy. Although mutations in the COH1 gene are detected in the majority of patients, in more than 24% only one or no mutant allele is detected. We hypothesized that some patients with Cohen syndrome carry deletions of the COH1 locus. We designed a high density oligonucleotide array with an average resolution of 300bp and screened DNA of 8 patients with hitherto unexplained Cohen syndrome. Three nullisomies and one heterozygous deletion were detected. In addition we show that the index patients in two different families are nullisomic for the exact same deletion suggesting a founder effect predisposing to Cohen syndrome. The COH1 locus was previously identified as a benign copy number variant (CNV). Our findings pinpoint that such benign CNVs may contain genes causal for autosomal recessive disorders. The COH1 gene locus is, to our knowledge, in addition to that for nephronophthisis, the second locus whereby the disorder is caused by a nullisomy of an apparent benign copy number variant. Given the high frequency of microdeletions in patients with unexplained Cohen syndrome, we strongly recommend that screening for deletions is an essential component in the analysis of DNA in patients with Cohen syndrome.

T10

Disruptions of distant regulatory elements surrounding FOXL2 in the causation of BPES syndrome

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Introduction: Blepharophimosis syndrome (BPES) is a development disorder caused by FOXL2 mutations, total gene deletions or long-range microdeletions. In 12% of patients however, the molecular defect remains unknown. The major aim of this study was to explore the role of subtle copy number changes and variations in conserved non-coding sequences (CNCs) in order to unravel the underlying genetic defect in a panel of 40 patients without intragenic FOXL2 mutations/deletions.

Methods: We developed a combined strategy, consisting of arrayCGH, quantitative PCR (qPCR) and sequencing of CNCs located in the shortest region of overlap (SRO) of previously described microdeletions upstream of FOXL2.

Results: ArrayCGH and qPCR of the FOXL2 region revealed 5 new microdeletions 5' to FOXL2. Interestingly, one of this is a very subtle deletion containing an important regulatory element. Overall, the breakpoints of these new microdeletions are scattered. The phenotypes of the five BPES patients carrying these deletions were indistinguishable from those of patients carrying an intragenic mutation. Sequencing of CNCs in the SRO showed that sequence variations in CNCs do not play a major role in the molecular pathogenesis of BPES.

Conclusion: Our study emphasizes the importance of disruptions of distant regulatory elements surrounding FOXL2 in the causation of BPES, and the need for a highly sensitive copy number screening of the FOXL2 region in this development disorder.

T11

Functional impact of retinal cysts on ERG and visual fields in patients with X-linked retinoschisis XLRS

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Introduction: In XLRS, cystic spaces are found throughout the retina. To evaluate the functional impact of these cystic changes we systematically compared retinal OCT structure to electrical and psychophysical function.

Methods: Seven patients with molecular genetically confirmed XLRS were re-investigated clinically and using Goldmann visual fields with stimuli I/4e and III/4e, Stratus OCT 3 and Ganzfeld-ERG (DTL-electrodes, Nicolet Spirit, ISCEV standard). Retinal structure within the large arcades was analysed quantitatively using OCT 3 and image processing. The relation of cystic to normal retinal areas was compared with amplitudes in scotopic and photopic Ganzfeld ERGs and with areas of Goldmann visual fields.

Results: In 6 patients, cystic areas of OCT cross-sections of the macula measured 20% to 35% of the retinal area, and in one young 4 year-old patient only 10%. Scotopic and photopic ERG b-waves measured about 1/2 to 1/3 the normal size while a-waves were significantly less affected. Accordingly, the b/a-ratio was abnormal except for the 4-y old patient. The areas of visual fields for I/4e were significantly reduced to again about 1/2 to 1/3 the normal size. Though trends were visible no strict correlation of fractional cystic area, visual field and ERG could be demonstrated.

Conclusion: In XLRS, cystic retinal changes visible with OCT are typically accompanied by deteriorated Ganzfeld ERG and reduced light sensitivity as measured with Goldmann visual fields. This correlation, however, is blurred by variance of the measured parameters.

T12

Evaluation of Lyonization in cultured Lymphocytes from Carriers of Choroideremia

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Purpose: Cassels et al. have reported that a REP1 polymorphism partially escapes from Lyonization in unaffected female carriers. The present study investigated the expression level of benign variants and pathogenic REP1 mutations in lymphocyte cultures from females.

Methods: cDNA fragments covering four types of mutations in the REP1 gene (one polymorphism D601S (c.1833C/A), one deletion c.581del14bp, one insertion c.1168ins50bp, one nonsense R270X (c.838C>T)) were amplified from 10 single cells each. The PCR products were analyzed for the frequencies of transcripts from the inactivated allele using the ABI PRISM SNaPshot Multiplex Kit and a capillary sequencer for fragment analysis.

Results: The benign variant was evaluated in two females, and showed predominant transcription of the mutant allele. Transcription of the inactivated allele was seen in 1% to 5% of transcripts. The pathogenic mutations were evaluated in always one carrier for each mutation type. 90% of cells carrying the insertion predominantly expressed the wildtype allele with a mean escape from Lyonization of about 4% (range 20% to 1.6%). Cells carrying the deletion transcribed the minor allele at mean of 25% (range 0% to 50%). Fifty percent of the cells showed predominant transcription of the mutant allele. We could not amplify the expected PCR product from the cells carrying the nonsense mutation.

Conclusion: Cultured lymphocyte cells from females carrying mutant alleles of REP1 show escape from Lyonization, ranging from a few to 50% of transcripts affecting the mutant as well as the wild type allele. We saw differences among the mutations as to the predominantly transcribed allele as well as to the amount of the minor allele. Whether this is due to the situation in cultured cells or to functional consequences of the type of mutation has yet to be evaluated.

T13

Maculopathies in gyrate atrophy of the choroid and retina

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Introduction: Gyrate atrophy (GA) of the choroid and retina is a rare, autosomal recessive disorder observed in patients with an ornithine-delta-aminotransferase deficiency. It is characterised by sharply demarcated circular patches of chorioretinal atrophy in the periphery appearing in the first decade of life. The macular region is usually spared and central vision maintained into the fourth or fifth decade(1). Through the identification of a young GA patient with maculopathy, we were interested in the macular involvement, and thereby the visual acuity prognosis in our own GA patients.

Methods: All clinical data of 8 patients (of 6 six families) were studied retrospectively. Follow-up varied from 1.5 to 20 years. Five patients were on an arginine-restricted diet.

Results: The most recent recorded visual acuity varied from light perception to 1.0 (median 0.6) at ages varying from 10 to 47 years (median 23 years). Funduscopy revealed a normal macular region in 1 patient, atrophic maculopathy in 1, subfoveal choroidal neovascularisation in 1, and cystoid maculopathy in 4. All of these lesions were bilateral. Besides cataract extraction in 5 eyes (of 3 patients), 3 eyes (of 3 patients) required posterior segment surgery because of rhegmatogenous retinal detachment in one, pronounced vitreoretinal traction in one, and vitreous haemorrhage with Coats'-like exudative vasculopathy in one.

Conclusion: Maculopathies occurred in 7 out of 8 GA patients, and so more frequently than described in the literature. Furthermore, 3 out of 8 patients required posterior segment surgery due to complications of their disease. In conclusion, central vision in GA patients may be affected at a relatively young age.

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T14

SLITLAMP-DELIVERED LASER PHOTOCOAGULATION OF ROP

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Introduction: To retrospectively study the outcome after slitlamp-delivered laser photocoagulation for threshold ROP using general anesthesia and topical anesthesia with sedation.

Methods: Review of the medical records of premature infants consecutively treated for ROP using slitlamp-delivered 532 nm laser photocoagulation and a contact lens between August 2001 and October 2007.

Results: In 40 eyes of 20 premature babies zone II threshold ROP was treated. Ten babies were treated under general anesthesia; the last 10 infants were treated under topical anesthesia and sedation. Because of disease progression 2 infants received bilateral supplemental treatment. ROP regressed in all eyes. Postoperative recovery was less eventful when treatment was performed under topical anesthesia compared to general anesthesia (need for increased respiratory support, analgesia, time to regain full enteral nutrition). Twelve infants were followed for 40 months (11 - 61 months). Mean refraction (spherical equivalent) was -1,04 D. Astigmatism of 2 D or more was present in 4 eyes (mean 2,4D). At the last examination (mean age 49 months) a mean visual acuity of 0.3 (log MAR, LEA symbols) was recorded (14 eyes of 7 children).

Conclusion: Slitlamp-delivered laser photocoagulation for ROP can be safe and effective. The mild myopia possibly reflects that this treatment modality interferes less with ocular development.

T15

IGF-I and Oxygen-Induced Retinopathy in Mice

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Introduction: In premature newborns, low levels of circulating insulin-like growth factor (IGF-I) correlate with the risk of retinopathy of prematurity (ROP), suggesting that upregulation of IGF-I may contribute to prevent ROP. In mice, IGF-I is a prerequisite for normal development of retinal vessels. We explored the link between IGF-I and murine oxygen-induced retinopathy (OIR).

Methods: In order to assess the role of endogenous IGF-I, pups were randomly assigned to small or large litters; in one subgroup, we measured body weight and circulating IGF-I on postnatal day (P) 14; in another subgroup, we applied hyperoxia (75% oxygen) from P7 to P12, and assessed the presence of retinal neovascularisation (NV) on P17. In order to screen for the potential of exogenous IGF-I, we administered a single dose of rhIGF-I (1 µg vs placebo) to P4 pups from equal litters, and applied hyperoxia from P7 to P12; body weight was measured and maturation was scored on P7, P14 and P17; NV was assessed on P17.

Results: Mice in small litters had a higher body weight than mice in large litters; they also had higher levels of circulating IGF-I, and developed less OIR, as judged by retinal NV (p=0.002). Mice who received rhIGF-I, weighed more than placebo-treated mice; they also matured faster and developed less OIR (p=0.00001).

Conclusion: Our findings in mice support the notion that a higher IGF-I availability reduces OIR risk, thereby sharpening the perspective that human ROP may be preventable, either by upregulating endogenous IGF-I or by administering exogenous rhIGF-I.

T16**Retinopathy of prematurity in extremely premature infants born before the 27th week – Preliminary results from a Swedish national population-based study during three years**

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Background / aim: Retinopathy of prematurity (ROP) remains an important cause of blindness among children. Today there is a new population of extremely premature infants, previously not surviving. Consequently, there is an interest in evaluating the incidence of ROP among these infants.

Method: A national project was initiated by the Swedish Association of Perinatology and the Swedish National Board of Health and Welfare with a general aim to investigate the neonatal morbidity in infants born before the 27th week of gestation, during three years (2004 – 2007). The ophthalmological part of the study was separately organized, and a network of paediatric ophthalmologists screening for ROP was created. When screening was completed, screening and treatment protocols were registered (DA, GH) in a national database, the Perinatal Quality Register.

Preliminary results: During the study period, there were 495 live-born infants who survived at least 35 days. ROP was noticed in 72% of whom 37 % had mild ROP and 35 % severe ROP, ie ROP stage 3 or more. The risk to develop ROP increased with decreasing gestational age (GA) at birth. Treatment was performed in 18 %. Six infants progressed to stages 4 and 5. Despite surgery five of them are severely visually handicapped. Comparison with results from other population-based studies will be made.

Conclusion: A new population of extremely prematurely born infants is today surviving, as a result of improving neonatal care. Preliminary results from a Swedish national study reveal that these infants have a high risk for severe ROP requiring treatment.

T17

Surgical treatment of glaucoma associated with Sturge Weber syndrome

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Introduction: Sturge-Weber syndrome is characterized by naevus flammeus of the face and ipsilateral angioma of the meninges responsible for epilepsy. Ocular manifestations consist in a choroidal angioma and glaucoma due to an episcleral haemangioma decreasing filtration. The aim of this study was to evaluate the outcome of glaucoma treatment in pediatric patients with Sturge-Weber syndrome.

Methods: This retrospective case series analysis included all patients, operated for glaucoma associated with Sturge-Weber syndrome between 2004 and 2007. The surgical technique, the control of intra-ocular pressure (IOP), the need for additional medical treatment, and the surgical complications were analysed

Results: Ten eyes of eight patients were operated. Median age at surgery was 11 months. All children presented with facial angioma and three with meningeal angioma. Deep sclerectomy was performed in 9 eyes and cyclodiode in 1 eye. Two eyes were retreated with cyclodiode and received anti-glaucoma medication, in the eye primarily treated with cyclodiode a second session was performed. One eye had a retinal detachment due to choroidal hemangioma. After 23.8 months of follow-up, mean final IOP was <20mmHg in 9 eyes (81.8%), of those 4 eyes (36.3%) needed anti-glaucoma medication.

Conclusion: In Sturge Weber syndrome, treatment is difficult with an important rate of failure. Cyclodiode is inconstantly efficient and needs several procedures while hypotony induced by filtering surgery is responsible for retinal detachment or choroidal effusion. Deep sclerotomy is associated with a lower retinal risk. The use of antimetabolites has to be evaluated in this surgery. Functional prognosis is correlated to the control of IOP, but also to the cerebral lesions and the uni or bilaterality of the condition. Management of glaucoma associated with Sturge-Weber has to be specified, results being often transient.

T18

Glaucoma in aphakic and pseudophakic eyes following surgery for congenital cataract in the 1st year of life

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Introduction: To determine the incidence and risk factors for glaucoma in pseudophakic and aphakic eyes following surgery for congenital cataract within the first year of life.

Methods: A review was performed of surgery performed over a 23 year period. Age at surgery, corneal diameter, intraocular lens implantation and presence of persistent fetal vasculature were documented. Time to development of glaucoma, management and outcome were determined.

Results: 7 (10.6%) pseudophakic and 25 (32.5%) aphakic eyes developed glaucoma. Cataract extraction was performed on all eyes of children with glaucoma ≤ 2.5 months. Microcornea and removal of visual axis opacification were not associated with an increased risk of developing glaucoma. Duration of follow up was significantly longer in the aphakic (107.9 ± 68.6 months) compared with the pseudophakic eyes (60.7 ± 44.9 months), ($p < 0.001$). Ahmed valves proved effective in controlling intraocular pressure but visual outcome was poor in the majority of cases.

Conclusion: Surgery for congenital cataract ≤ 2.5 months of age increases the risk of glaucoma development, regardless of whether the eye is aphakic or pseudophakic. Intraocular pressure control with Ahmed valves is frequently required. Glaucomatous damage and dense amblyopia contribute to poor visual outcome in these eyes.

T19

ICD3 classification of corneal dystrophies, pediatric ophthalmology aspects

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Introduction: ICD3 classification of corneal dystrophies Pediatric ophthalmology aspects H U Møller Viborg, Denmark C Bredrup Bergen, Norway An international group of cornea specialists has made a classification of corneal dystrophies: The IC3D Classification is a new classification system that incorporates many aspects of the traditional definitions of corneal dystrophies with new genetic, clinical, and pathologic information. Standardized templates provide key information that includes a level of evidence for their being a corneal dystrophy. The present presentation describes the use of this classification for pediatric ophthalmologists.

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T20

Etiological distribution of paediatric cataracts in Hospital Sant Joan de Déu during the period 1995 to 2007

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Introduction: To study etiological distribution of congenital/infantile cataracts treated at our hospital during the period 1995 to 2007.

Methods: The medical records of 315 children (454 eyes) who were medically or surgically treated for cataracts at our hospital during the period 1995 to 2007 were retrospectively reviewed. Secondary cataracts due to uveitis were excluded. MAIN OUTCOMES MEASURES: Classification of congenital/infantile cataracts according to presumed aetiology, gender and laterality.

Results: A total of 454 eyes with congenital/infantile cataract, 175 boys and 140 girls, were included, of whom 139 were bilateral (278 eyes) and 176 unilateral. 39, 4% of all cases (179 eyes) were idiopathic cataracts, with no aetiology and without any ocular dysmorphism or systemic anomalies associated. 14.7% (67 eyes) were familial-hereditary cataracts, 7.9% (36 eyes) were anterior polar cataracts, 7.7% (35 eyes) were traumatic cataracts, 7.7% (35 eyes) were associated to other ocular dysmorphism, 4.6% (21 eyes) were associated to neurological delay 3.9% (18 eyes) were associated to persistent fetal vasculature, 3% (14 eyes) were anterior pyramidal cataracts, 2.6% (12 eyes) were associated to Down's syndrome, 1.3% (6 eyes) were associated to corticotherapy, 1.3% (6 eyes) to leukaemia, and finally we found 1 children (2 eyes) 0.4% with Alagille's syndrome, 1 with Stickler syndrome, 1 Cystic fibrosis, 1 Hereditary hyperferritinemia syndrome, 1 Cerebrotendinous xanthomatosis syndrome, 1 Galactosemia, 1 Lowe syndrome, 1 children with a previous heart surgery, 1 eye (0.2%) with congenital toxoplasmosis, 1 eye with a congenital rubella, 1 eye with a Tuberous sclerosis syndrome and 1 eye with a Rubinstein-Taby syndrome.

Conclusion: When including traumatic cataracts and considering anterior polar cataracts and persistent fetal vasculature the aetiology of the congenital/infantile cataracts, the cause of 39, 4% of all cataracts remain unknown.

T21

Anophthalmia and microphthalmia in pediatric population

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Introduction: The aim of the study is to document the associated ocular and systemic findings in patients with anophthalmia and microphthalmia and find out the visual prognosis and results of cosmesis.

Methods: Records of 57 cases (83 eyes) with anophthalmia and microphthalmia were retrospectively reviewed. Results: 67 eyes (80.7%) had microphthalmia, 16 eyes (19.3%) had anophthalmia. Forty-five percent of the cases had bilateral involvement. Mean age of the cases was 34.6 years at the time of diagnosis. The associated ocular findings were: cataract (19.3%), ocular coloboma (52.6%) and PHPV (3.5%). Twenty four percent of the patients had nystagmus, 21 % had strabismus, 17.5% had refractive error. Prematurity was found with a mean gestational age 32 months in 12.2 % of the cases. 17.5% cases had consanguinity and 28% of the cases had associated systemic and syndromic findings.

Conclusion: Treatment in these cases should be directed towards maximizing existing vision and improving cosmesis through simultaneous stimulation of both soft tissue and bony orbital growth.

T22**FOXC1/PITX2 mutations and copy number changes in a Belgian-Dutch cohort of patients with anterior segment dysgenesis (ASD)**

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Anterior segment dysgenesis (ASD) refers to a group of rare developmental disorders of the anterior eye segment, which display mostly an autosomal dominant inheritance pattern. Disease-causing mutations or copy number changes of FOXC1 and PITX2 account for 40% of the molecular defects in ASD patients. A primary purpose of this study was to determine the prevalence of disease-causing FOXC1/PITX2 mutations and copy number changes in a Belgian-Dutch cohort of ASD patients. Sixty-four probands, mainly of Belgian-Dutch origin, were examined for copy number changes of FOXC1/PITX2 with MLPA and screened for subtle FOXC1/PITX2 mutations by sequencing. MLPA revealed 5 FOXC1 deletions and 4 PITX2 deletions. Furthermore sequencing revealed 1 known and 7 novel FOXC1 mutations and 1 known and 6 novel PITX2 mutations. In this cohort the underlying genetic cause was revealed in 38% of the patients, which sustains the major role of the FOXC1/PITX2 genes in the molecular pathogenesis of ASD in the Belgian-Dutch population. The molecular data are currently being complemented by phenotypic information in an attempt to establish genotype-phenotype correlations. A second aim was to delineate FOXC1 and PITX2 deletions in order to unravel their underlying mechanism. ArrayCGH with a tiling BAC array for the FOXC1 region was carried out for 4 patients with a known FOXC1 deletion. In addition, 3 PITX2 deletions were further delineated with SNP chip copy number screening. Subsequently, 1 PITX2 deletion of maximum 2.5 Mb in size was further defined using quantitative PCR (qPCR), which enabled us to pinpoint the deletion breakpoints to a region of 4639 bp at the centromeric end, and of 786 bp at the telomeric end of the deletion. The precise location of the breakpoints is currently being characterized by long-range PCR and sequencing. Overall, these results show a scattered location of the breakpoints, which is in line with previous findings. These non-recurrent rearrangement events propose non-homologous end joining (NHEJ) or Fork Stalling and Template Switching (FoSTeS) as possible underlying mechanisms.

T23

Ocular features of Lyme borreliosis: an old disease revisited

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Introduction: The human eye can offer critical clues to the presence of systemic disease. The various ophthalmologic manifestations of Lyme borreliosis (LB), which is the most common tick-transmitted disease in the northern hemisphere, are discussed in this article. The disease is also common in children.

Methods: Twenty-one patients, including 5 children, with LB-associated ocular manifestations were treated between 2000 and 2007 in the ophthalmology department of Strasbourg (an endemic area). The diagnosis was based on medical history, ocular and systemic clinical findings. Determinations of antibodies related to *Borrelia* and/or the detection of the bacteria as well as exclusion of other causes were diagnosis criteria.

Results: Eight uveitis (2 children), 3 chorioretinitis, 5 abducens palsy (2 children), 2 optical neuropathies (1 child) and 2 orbital myositis associated with LB were diagnosed. Systemic findings, such as arthritis, rash, or erythema migrans were mentioned for all cases. The most common systemic manifestations in childhood were flu-like symptoms with fever and rash. One child also complained about severe knee and hip arthritis, which are known to give a very serious prognosis, leading to total knee replacement. Determination of antibodies was positive in all patients. A patient also presented with anterior uveitis, detection of *Borrelia* DNA was realised in his aqueous humor. All patients were treated by antibiotics adjusted to individual circumstances. All patients with uveitis, chorioretinitis or myositis underwent an anti-inflammatory treatment based on topical or systemic corticosteroids. Resolution of ocular signs, without any relapse, was observed in all patients within 2 weeks to 3 months.

Conclusion: Patients with any of these ocular manifestations should be questioned for exposure to an area endemic for LB, tick bites, skin rash, or arthritis. Such patients should undergo serological testing. If the clinical presentation is suggestive of LB, a course of oral antibiotics should be used. Corticosteroids can be used for anterior segment inflammation, chorioretinitis and myositis. For any ocular symptom, even for children, LB should be taken into account especially in endemic areas. All in all, the presence of an articular syndrome determines the prognosis of LD, which remains good in cases of early treatment. Thus, in childhood permanent defects are extremely rare, even following long-term manifestation at an early age.

T24

Morphology and function of the central retina in children and young adults with Stargardt Dystrophy

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Introduction: The aim of our study was to evaluate central retinal function in children and young adults with Stargardt dystrophy by correlating retinal morphology (autofluorescence, OCT) with functional (visual acuity, microperimetry) and electrophysiological tests (photopic, scotopic ERG, PERG, mfERG).

Methods: 6 patients (3 F, 3 M, VA: $0,2 \pm 0,1$, VA range from 0,3 to 0,1; age range from 10 to 30 yrs, average onset of problems at age of 10yrs) were included in the study. Autofluorescence was recorded by HRA. The central 10° visual fields were tested with microperimetry (MP1, Nidek technologies), that enables one to compare central retinal sensitivity and fixation patterns in relation to the fundus image. The morphology of the retina was recorded by OCT (Topcon 3D OCT-1000). ISCEV pattern, photopic and scotopic and mfERG were recorded in all the patients.

Results: AF showed mottled central hypo and hyperfluorescent areas with peripheral hyperfluorescent flecks in 4 patients and a central hypofluorescent region with hyperfluorescent ring in 2 patients. OCT showed transverse loss of the central photoreceptor layers (average OCT thickness: $38,9 \pm 14,8$ microns, average OCT volume $6,1 \pm 0,5$ mm³). Microperimetry showed shift of fixation to the preferred retinal locus (superior or nasal to the lesion) with relatively unstable or unstable fixation in most of the eyes. Pattern P50 and N95 responses were abnormal in 8 eyes. Photopic and scotopic ERG were normal in all except two patients with abnormal cone and 30 Hz responses. mfERG showed reduced responses mostly in the inner three rings (ring 1: 22,6% of mean normal value, ring 2: 27,3%, ring 3: 38,9%, ring 4: 57,9%, ring 5: 64,2% of mean normal value).

Conclusion: AF and OCT showed central atrophy of the retina, with shift of the fixation to the preferred retinal locus in most of the patients. Cautious interpretation of mfERG as well as PERG is needed in patients with eccentric and nonstable fixation.

T25

Visual fields in children affected by Periventricular Leukomalacia

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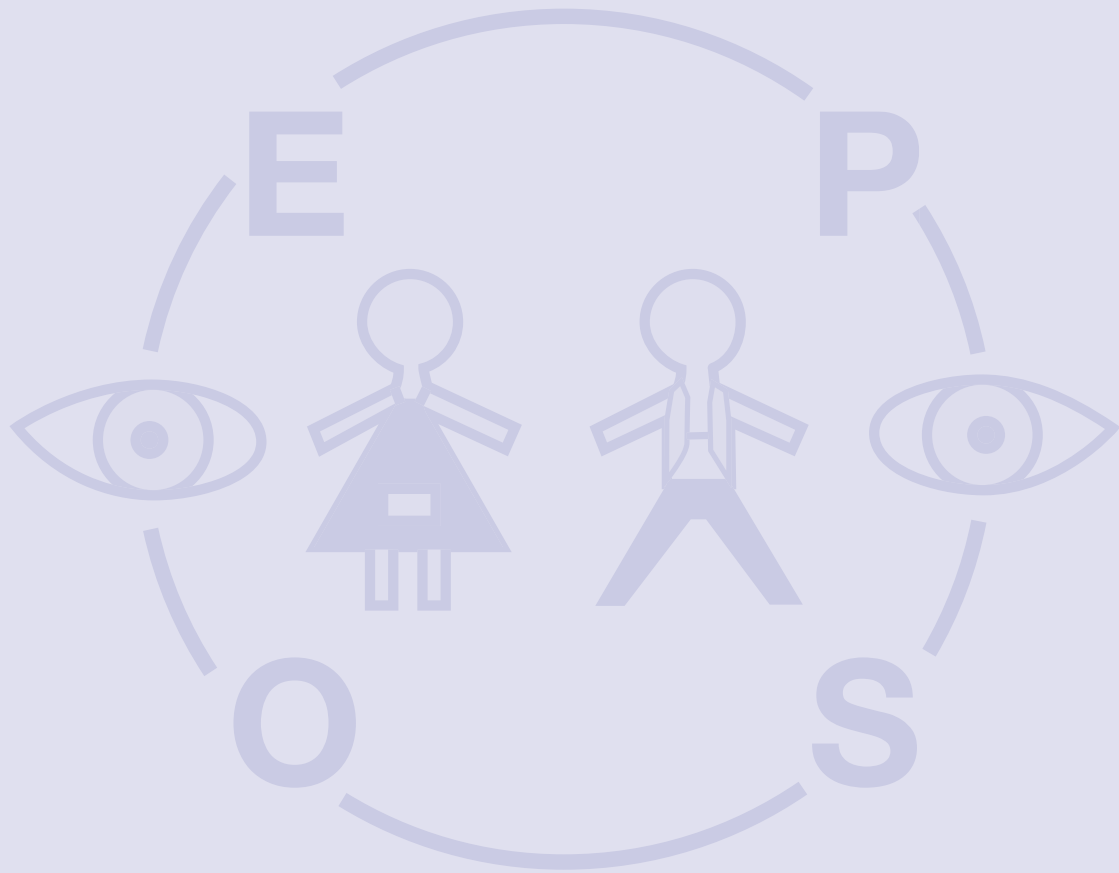
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Aim of the study. Periventricular Leukomalacia (PVL) is a lesion of the immature brain which may involve the optic radiations. Aim of this retrospective study is to search for signs of injury of the visual field (VF) in neurologically impaired children affected by PVL.

Material and Methods. The monocular or binocular VF of 44 children (17 girls and 27 boys) aged from 6 months to 12 years was tested using standard- or behavioural perimetry.

Results. VF could be performed in 95% of the children, mainly using behavioural perimetry (Befie test). In 19 subjects (43%) VF was normal, while 7 subjects (15%) showed an unilateral homonymous inferior quadrantanopia, 6 subjects (14%) showed a bilateral inferior homonymous quadrantanopia, 5 subjects (11%) showed a concentric VF defect, 2 subjects (5%) showed an homonymous hemianopia and one subject (2%) showed an unilateral homonymous superior quadrantanopia.

Conclusions. The present study shows that VF defect are common in neurologically impaired children affected by PVL.



Posters

P01**Retinal dystrophy due to a vitamin E deficiency**

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Introduction: Vitamin E (α -tocopherol) is a potent lipid-soluble antioxidant. Deficiency of vitamin E can cause neurological disorders, the eyes may also be involved. We will present a case in which vitamin E deficiency has led to a retinal dystrophy without any neurological abnormalities.

Case report: A 13 year old boy without a medical history noticed a decreased vision and nyctalopia after a minor contusion to his right eye. Examination showed best corrected visual acuity (BCVA) of hand motion in his right eye and 20/40 in his left eye. Fundus examination revealed an atrophic optic nerve and atrophic retinal pigment epithelium with pigment clumping. The electroretinogram (ERG) showed normal photopic and 30Hz responses OD. However, there was an amplitude reduction and a lowered a/b ratio under scotopic conditions. Kinetic perimetry showed visual field restrictions. After half a year his BCVA OS deteriorated to 20/100. A second ERG now showed amplitude reduction under photopic and 30Hz conditions. The implicit time under scotopic conditions was prolonged. Visual fields with kinetic perimetry further deteriorated. Neurological examination was normal. Extensive laboratory tests showed lowered blood and intra-erythrocyte vitamin E levels. After supplementing with vitamin E laboratory tests normalised and his clinical ophthalmological findings showed some improvement.

Conclusion: Vitamin E deficiency as a cause of retinal dystrophy has been described in combination with ataxia (AVED syndrome; ataxia with vitamin E deficiency). Our patient has been diagnosed as having AVED syndrome due to his specific blood abnormalities. Interestingly, our patient had no neurological abnormalities on examination. To the best of our knowledge there are no known cases with an isolated retinal dystrophy in combination with vitamin E deficiency. In our patient, vitamin E supplementation therapy seemed to halt the progression of retinal disease and may have prevented ataxia. This emphasizes the need of metabolic screening in children with an atypical retinal dystrophy.

P02

Long-chain 3-hydroxyacyl-CoA dehydrogenase (LCHAD) deficiency and retinopathy. A Case report

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Background: LCHAD deficiency is a rare disorder of the mitochondrial long-chain fatty acid oxidation and a genetic syndrome inherited as a recessive trait. Affected patients can present hypoglycaemia, rhabdomyolysis and cardiomyopathy. In few cases, they may suffer from retinopathy.

Case report: an eleven year-old girl was diagnosed as suffering from LCHAD deficiency with recurrent muscle problems at seven months old by an inaugural coma with hypoglycaemia and hepatomegaly. Appropriate diet and fatty acid supplementations were managed regularly. Retinopathy was observed as she was two years old. Physical, biochemical and ophthalmological assessments including visual acuity and ERG recordings were conducted over a ten year-period.

Results: Visual acuity was stable. Eye fundi showed a stable appearance of chorioretinopathy. A photophobia was noticed four years before photopic-ERG amplitude decreased with normal scotopic-ERGs and abnormal EOG light rise. These results suggest that RPE and cone system dysfunction as noticed by others. Comments are conducted in relation with the mitochondria role in the retina.

P03

**Gyrate atrophy of the choroid and retina with hiper-ornithinemia :
ocular findings in two siblings after reduction of plasma ornithine with diet**

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Introduction: Gyrate atrophy of the choroid and retina is a rare autosomal recessive inherited disease, characterized by progressive chorioretinal dystrophy that begins in childhood and leads to total blindness in the fourth or fifth decade of life. The primary defect is a deficiency of ornithine –delta- amino-transferase. The purpose of the study was to evaluate the ocular findings and the visual function in two siblings affected by gyrate atrophy, to study the impact of a precocious diagnosis on visual impairment in gyrate atrophy, and the protective effect of diet against choroideal and retinal dystrophy.

Methods: We examined 2 siblings (a girl and boy). The girl came to our attention when she was 8 years old, presenting typical gyrate retinal and choroid lesions in midperiphery, moderate myopia and high astigmatism. Her best corrected visual acuity was 5/10 in both eyes. Plasma aminoacid profile showed a level of ornithine 982 µmol/l. She started therapy with B6 vitamin (600 µmol/day) and low protein diet (1g/kg/day). The younger boy , 2 years and 9 months old, underwent an ophthalmological examination showing only mild retinal depigmentations in far periphery. The ornithine level was 850 µmol/l and he was put a diet (1g/kg/day) and B6 vitamin (300 mg/day). The follow-up consisted in periodic ophthalmological evaluations of visual acuity, visual field, color vision, contrast sensibility, fundus examination, retinography, and electrophysiological exams.

Results: They were followed-up for 37 months. The level of ornithine showed a decreased but not a normalization in both siblings (respectively 430 µmol/l in female and 498 µmol/l in male). At last evaluation visual acuity was stable in the girl (5/10), and normal in the boy (8/10). Electroretinography showed abnormalities at an early stage; it didn't worsened in both cases. Visual field showed a more severe restriction in the girl.

Conclusions: The younger boy who started receiving the diet at an early stage ,without typical gyrate lesions, had much less ocular disease and visual function impairment. The genetic heterogeneity may play a role in the phenotypic variability of gyrate atrophy but the chronic reduction of ornithine seems markedly to slow the progression of the chorioretinal dystrophy

P04

Neonatal Insulin therapy to drive normoglycemic anabolism

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Introduction: In very low birth weight (VLBW) newborns, retinopathy of prematurity (ROP) is one of the sequelae associated with neonatal hyperglycemia. The effects of early insulin therapy were recently explored in a multicenter trial (NIRTURE). We performed a NIRTURE subanalysis, focusing on ROP in the two largest study centers, which apply similar types of standard care.

Methods: ROP was assessed in VLBW newborns with gestational age (GA) ≥ 26 weeks, randomized to receive either standard care during the first postnatal week (control arm, N=94) or insulin infusion (0.05 U/kg.hr) titrated with glucose to aim for normoglycemia (insulin arm, N=88). Glycemia was measured continuously. Main analyses were control vs insulin; non-ROP vs ROP; and ROP prevalence during vs after NIRTURE.

Results: Across the first week, newborns in the insulin arm received more glucose ($p < 0.0001$) than controls, experienced fewer episodes of hyperglycemia ($p = 0.005$). Median glycemia and interquartile ranges (IQR) did not differ between study arms. In the insulin arm, recovery of BW was faster ($P = 0.001$), and ROP was less prevalent ($p = 0.01$). ROP development was associated with a higher glycemia on each of the first 7 days, the apparent threshold ranging around 6.0 mmol/L. Study analysis pending, both centers returned for 9 months to standard care, and ROP prevalence returned in both centers to control levels ($P = 0.01$).

Conclusion: An insulin-driven increment of first-week glucose infusion improved glycemic control, accelerated neonatal anabolism, and reduced ROP prevalence in the two largest NIRTURE centers.

P05

Papain ferment in treatment of ocular developments of systemic inflammatory diseases of the connective tissue

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Introduction: Ocular developments of systemic inflammatory diseases of the connective tissue compose from 1 to 5%. Established localization of a pathological process, which influences vision, is the area of optical mediums and optic nerve sheath. Thus, the choice of a method of amputation of postinflammatory connective tissue accrementitions has primary significance when selecting the ways of treatment of these states.

Methods: 21 patients were observed with subacute type of optochiasmic arachnoidite and 13 patients with persistent vitreous opacities after rheumatic fever. The average age of patients was 10.8 ± 1.5 years. Evaluation criteria of the state of vision before and after the treatment were: • vision accuracy • field of vision • visual evoked potentials • degree of optical and ultrasonic transparency of vitreous body Proteolytic ferment papain (papaya extract) was used for treatment. Papain was applied by endonasal electrophoresis in case of optochiasmic arachnoidite. Treatment of rheumatoid vitreous opacities was performed by transpalpebral electrophoresis method.

Results: Normalization of visual evoked potentials and fields of vision was observed on 17 patients with optochiasmic arachnoidite. Vision accuracy was 0.7-1.0. In 9 cases the increase of vision accuracy to 0.7-0.8 and optical and ultrasonic transparency of vitreous body was observed on patients with rheumatoid vitreous opacities.

Conclusion: papain ferment effectively resolves pathological postinflammatory connective tissue accrementitions. In treatment of the above mentioned states in ophthalmology the treatment efficiency depends on the way the medicine is administered and on the phase of disease.

P06**Bullous Diseases in Children – Case Reports**

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Introduction: Toxic epidermal necrolysis (Lyell's syndrome) and erythema exsudativum multiforme major (Stevens-Johnson syndrome) are rare but very serious dermatological lesions characterized by the mucocutaneous exfoliation. Various etiological factors (infections, drugs, systemic diseases) have been implicated as causes of these diseases. The most serious, often fatal complications are infection and sepsis. Ocular complications are very frequent, ranging from mild conjunctival hyperaemia to ankylosyblepharon and corneal ulcer.

Case reports: We report three cases with these bullous diseases observed last year.

Case 1: The boy was born on 30th gestational week with 1050 grams birth weight. Bowel surgery was performed because of necrotizing enterocolitis. On 12th chronologic week Klebsiella sepsis induced Lyell's syndrome. Necrolysis of the eyelids skin developed and recovered after effective systemic treatment. Unfortunately an other sepsis was fatal for the boys life.

Case 2: 15.5 years old girl was treated with lamotrigine for epilepsy. This drug induced erythema exsudativum multiforme major. On 3rd day conjunctival hyperaemia was observed. Every hour arteficial tears, and five times per day steroid drops were applied. Despite local and systemic treatment symblepharon developed on both side.

Case 3: 4.5 years old boy was referred to our department who was treated with ibuprofen for fever and Stevens-Johnson syndrome developed. Ocular complications were symblepharon and recurrent corneal erosion on both side. We performed amnion membran transplantation and later perforating keratoplasty on the left side. Soft contact lens was fitted against the recurrent erosion.

Conclusion: Ocular complications of bullous diseases can be various and sometimes serious. Treatment of ocular surface disease connected with systemic disorders can be very difficult.

P07

Immunosuppressive drug combination therapy in paediatric autoimmune ocular diseases. Follow-up from 1991

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Introduction: to assess the effectiveness of a steroid-sparing immunosuppressive treatment protocol (IST) in the control of autoimmune ocular inflammatory diseases in paediatric age.

Methods:retrospective clinical study. Paediatric patients presenting with ocular inflammations seen at the Autoimmune Ocular Disease Service, Ophthalmology Department, University of Parma from 1991, inadequately responsive to topical and/or systemic steroids, were offered the option to switch to a combined IST. The protocol consisted of different immunosuppressive drugs added in a stepladder sequence; each drug (including the steroids) administered discontinuously. Only patients with a minimum follow-up of 1 yr at 31st Dec 2005 were included in the study.

The outcomes were: the control of the inflammation; the visual acuity; the treatment safety. Data were summarized and computed by appropriate statistics: mixed and multilevel linear models performed using SAS version 8.2 Proc MIXED (Marvin Research srl, Milan, Italy). Results:50 patients affected by different autoimmune ocular diseases were included in the study. Mean follow up was 56 months (range: 24 – 151 months). 36 % of patients experienced at least one relapse episode during follow-up. Visual acuity showed a statistically significant improvement during follow-up. Some laboratory tests (Uric acid, Bilirubin, Gamma GT) had a statistically significant increase, while others (WBC and PLT) had a decrease during follow-up. None of them was outside safety limits. Growth parameters were inside normal limits.

Conclusion:In our follow-up, ICT appeared to be a valid alternative for paediatric patients affected by ocular autoimmune diseases, inadequately responsive or suffering from severe side effects coming from long- term steroid monotherapy.

P08

Development of a uveitis screening service for Juvenile Idiopathic Arthritis: use of rapid cycle audit

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Introduction: Leeds Teaching Hospitals is a tertiary referral centre for both Paediatric Rheumatology and Paediatric Ophthalmology. We aim to provide a same day ophthalmic review for new patients presenting with Juvenile idiopathic arthritis and present our journey in developing this service during a time of change in location of hospital services.

Methods: During the period 2005-2008, we undertook small sample, rapid cycle audit to monitor our screening service. Initially we looked at new referrals in region. We aimed to see these children within 6 weeks of presentation, setting a standard of 100%. Changes were made after each audit cycle and then reaudited. Methods of data collection were also altered to allow for easier retrieval.

Results: Audit outcomes varied from 35% to 90% assessment within 6 weeks of diagnosis.

Conclusion: Rapid cycle sampling is an efficient way monitoring and developing a service. It allows quick response to changes in service arrangement to ensure patients needs are met. It requires regular changes in sample assessment and data retrieval for best results.

P09**Orbital cellulitis associated with Adamantiades-Behcet disease**

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Introduction: Adamantiades-Behcet disease is a chronic, systemic vasculitis with unknown cause, although an association with HLA-B5 and B51 is clear.

Methods: An 11 year old girl with swelling and redness of left eye was admitted to our Hospital. She had mild irritation, photophobia, lacrimation and pain of the left eye for 4 weeks. A CT scan showed orbital inflammation.

Results: Her visual acuity was 4/10, she had findings consistent with anterior and posterior uveitis and swelling of the optic disk. A subsequent MRI scan confirmed the orbital cellulitis. The patient was treated with intravenous antibiotics and prednisolone. Serologic tests for viruses were negative and HLA screening was positive for B51 and DR3 positions. The genetic analysis revealed MTHFR C677T, Endothelin Receptor A-231 G/A and Endothelin-1 138 insA AAA/AAAA polymorphisms. Her symptoms and signs regressed totally within 3 months.

Conclusion: To our knowledge orbital cellulitis is a feature, never been reported before in a paediatric case of Adamantiades-Behcet disease.

P10**Optic disc atrophy in children and neuro-imaging findings**

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Objective: To evaluate neuro-imaging findings in children presented with optic disc atrophy.

Methods: Retrospective medical record charts were reviewed from January 1998 to December 2007. Patients aged less than 17 years old who presented with optic disc atrophy and performed neuro-imaging examinations (magnetic resonance imaging or computed tomography) were included.

Results: Eighty cases were met the criteria. Seventy seven cases were presented with bilateral optic disc atrophy. Neuro-imaging examinations were abnormal in 54 cases (67.50%). Twenty two cases (40.74%) were found brain tumors. Craniopharyngioma was found in 9 cases (40.91%) and was the most common brain tumor in this study. Thirty two cases had abnormal neuro-imaging findings such as hydrocephalus 10 cases, cerebral malformations 8 cases and encephalomalacia 4 cases. Two of 3 cases with unilateral optic disc atrophy were traumatic optic disc atrophy.

Conclusion: Children who presented with optic disc atrophy may need to do neuro-imaging examinations to find central nervous system anomalies and life- threatening causes.

P11**Choroidal Neovascularisation in an adolescent previously treated for nasopharyngeal carcinoma**

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Introduction: A seventeen-year-old male presented to the eye casualty with a two-week history of blurred central vision in the right eye. The patient had a history of nasopharyngeal carcinoma diagnosed at age twelve which was treated with chemotherapy and radiotherapy. He had required right cataract extraction and intraocular lens implantation at age fourteen for a radiation induced posterior sub capsular cataract.

Methods: Ocular examination revealed the visual acuity in the right eye as counting fingers and the left eye 6/9. Posterior segment examination of the right eye revealed a macular lesion suggestive of choroidal neovascularisation, with an epiretinal membrane and sub retinal fluid. This was imaged with fundus photography, fluorescein angiography and optical coherence tomography.

Results: Treatment with an anti-VEGF is being considered and the outcome is to be closely observed.

Conclusion: The mechanism of CNV development in ARMD and radiation retinopathy is compared.

P12

Eye and orbit changes in childhood acute lymphoblastic leukaemia

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Introduction: There is a possibility of the eye structures' involvement as a relapse of childhood acute lymphoblastic leukaemia (ALL). The features involving the eye can have high polymorphism (Gallin, 2000; Chang, Moshfegni, Alcorn, 2006; Melnikova, Rummyantsev et al., 2007) and include infiltration by leukaemic blasts, hemorrhages and ischemic changes. Chemotherapy drugs used for leukaemia management can cause eye problems due to its toxicity. Immune suppression plus infection can bring to intraocular inflammation.

Methods: Children (n=74) with ALL were being treated at an oncology-haematology department of the Municipal Children's Hospital #1, Saint Petersburg, Russia, between January 2006 and December 2007 inclusive. Of 74 babies with ALL 40 were boys (54%), 34 were girls (46%). All patients included were receiving treatment in accordance with COALL92 protocol for ALL. Ophthalmologic examinations included routine investigations, fundus investigations and picturing with the RetCam II ®, and ultrasound scanning of the eyes. In needed cases magnetic resonance imaging was performed.

Results: Of the 74 children with ALL, 15 patients (20,3%) had specific changes in the eyes. The most often observed changes were retinal hemorrhages and partial hemophthalm: 5 patients (33.3%). Another intraocular manifestation of ALL was infiltration of the optic nerve by leukaemic blasts (4 cases, 26,6%). Iridocyclitis was suspected as possible adverse effect of therapy in two patients (13,3%) as well as the dry eye syndrome in three patients (20%). One patient (6,6%) developed one-sided candidal endophthalmitis with the following enucleation of the eye. Dynamic observations in this group of patients revealed two cases (2,7%) of intraocular relapse of neuroleukaemia. One of these patients died despite of local radiotherapy treatment.

Conclusion: Adequate diagnosis of the eye and orbit diseases as relapses of ALL is of high importance for management and prognosis of patients with acute lymphoblastic leukaemia.

P13**Ophthalmic problems in children with Down syndrome:
Findings from England**

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Introduction: Down syndrome is a common congenital abnormality with characteristic features that include ocular manifestations. Guidelines for medical surveillance of ophthalmic problems in people with Down syndrome exist (1).

Methods: A systematic literature review was conducted to determine the frequency of ophthalmic disorders in Down syndrome patients. Hospital records were examined retrospectively to determine how many children with Down syndrome were seen by ophthalmic services. For those children that were assessed ophthalmologically, records were reviewed to determine the adequacy of examination and frequency of ocular disorders. Locally there is no formal ophthalmic screening program for children with Down syndrome.

Results: Of all 96 children (aged 0-16 years) known to have Down syndrome, 59 had been assessed ophthalmologically. The majority of referrals were from either orthoptists (43%) or paediatricians (42%). Subjects had a mean (S.D) age of 2.5 (2.4) years; 51% were male. Nearly all patients (96%) were found to have at least one ophthalmic abnormality, most commonly hypermetropia (83%); astigmatism (74%); strabismus (65%) and visual acuity below 6/18 (51%). Myopia (17%); nystagmus (8%) and cataract (4%) were found less often. Glasses were prescribed for 38% of children with hypermetropia. The maximum refractive error in a child not given glasses was +3.50D. Compared to children with normal hearing, the 47% (n=25) with hearing problems were more likely to have a low visual acuity (50% c.f 54%), astigmatism (71 c.f 77%), strabismus (56% c.f 74%) and nystagmus (4% c.f 13%). They were less likely to have a cataract (8% c.f 0%). Ocular disorders that were found frequently in six asymptomatic children referred only because they had Down syndrome included poor visual acuity (3), strabismus (3), astigmatism (2) and refractive error (6). Glasses were prescribed for one of these children.

Discussion: Approximately 2/3 of children with Down syndrome are seen by ophthalmic services. Of those that are assessed, many have ophthalmic disorders, particularly refractive errors and strabismus. Screening may therefore be appropriate for all children with Down syndrome to avoid missing these abnormalities. Hearing problems are common in children with Down syndrome and may be associated with increased ocular pathology. The empirical findings warrant further study.

REFERENCE:

1) <http://www.dsmig.org.uk/library/articles/guideline-vision-5.pdf>

P14**Ophthalmic problems in children with Down syndrome:
A comprehensive review**

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Introduction: Down syndrome is an increasingly common congenital abnormality with characteristic features.

Methods: A systematic review of the available literature was performed to determine the common ophthalmic disorders in children with Down syndrome. Seven electronic databases, including Medline and EMBASE, were searched using the UK National Library for Health interface. The search strategy was Down syndrome (OR synonyms) AND ophthalmic (OR synonyms) AND paediatric (OR synonyms).

Results: The search strategy yielded 230 articles. After application of exclusion criteria 23 were included in the review. Synthesis of the literature demonstrated that children with Down syndrome are at risk of a number of disorders. Strabismus was a frequent finding, particularly esodeviation, and refractive error was also reported regularly, particularly hypermetropia. Other common findings included poor visual acuity, blepharitis and nystagmus, among others. Glaucoma and congenital cataract were less common but with potentially serious implications for future vision. Only one study compared routine paediatric examination with that of a paediatric ophthalmologist.

Discussion: The findings of this review suggest that an ophthalmic screening program for children with Down syndrome is advisable. Further work is required in a number of areas, particularly to assess the adequacy of paediatric examination compared to that of the specialist ophthalmologist. Additional work should investigate how a screening program could be best implemented.

P15

Differences between complete and partial congenital nasolacrimal duct obstruction

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Introduction: Children with clinical signs of congenital nasolacrimal duct obstruction are divided in 2 groups: complete or partial obstruction according to the result of low pressure irrigation of 1ml of serum. We study the differences between both groups.

Methods: Inclusion criteria: children with constant epiphora, secretion, mucocel or acute dacryocystitis. Sample size: 272 patients between 1 and 5 y.o. and 467 lacrimal pathways from april-2003 to may-2007. Minimum follow-up of 6 months. Parameters: Sex, laterality, bilaterality, previous managements and procedures, clinical manifestations and result of probing as exploration, surgical management used and their general efficacy. Statistics: Prospective study. The chi-square test was the method for compare both groups.

Results: Complete % Partial %
o Sex, laterality and age Female/Male 42 / 49 57 / 42 Right/left 47 / 53 56 / 43 Bilateral / Unilateral 70 / 30 85 / 15 Age 1y 56 44 2y 49 50 3y 34 66 4y 20 80
o Number of surgery 1 st 90 79 2on 9 18 3 rd 1 3
o Clinical manifestations Epiphora: only/+secretion 97 / 3 93 / 7 constant/intermittent 74 /26 68/31 Secretion only/+secretion 66 / 34 54 / 45 constant/intermittent 74 / 26 51 / 49 Signs Acute dacryocystitis 0, 5 0 Only epiphora 34 45 Epiphora + secretion 57 47 Only secretion 3 7 Mucocel 5 0, 4
o Previous management Conservative 49 51 Probing 49 51 Intubation 16 84
o Exploratory probing Normal 6 65 Hasner membranous 71 30 Hasner hard 19 3 Lacrimonasal multiple 5 2

Conclusion: The clinical differences between complete and partial congenital nasolacrimal obstruction are several, most of them with statistical value. We should consider these 2 sorts of lacrimal obstruction to optimize the future management.

P16

Targeted Visual and refractive assessment of children attending special needs schools has a high yield and an effective outcome – an audit study

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Introduction: Epidemiological research and data concerning visual impairment (VI) in children with learning disabilities (LD) are scarce. Unrecognized and often preventable VI adversely affects development and social behaviour in this already vulnerable group, and potentially constitutes a missed therapeutic opportunity. This study aims to determine the frequency and severity of VI and refractive errors (RE) in children with LD.

Methods: A protocol-based, ophthalmological assessment of 240 children (5-19 years) attending schools for LD (Glasgow) was performed between 2004-2006.

Results: 215/240 (89.5%) participants were able to co-operate with the visual assessment. 140/240 (58.3%) participants were found to have VI (according to WHO Criteria); 101/240 (42%) had a correctable RE. The prevalence of RE in this population was found to be similar to that of the adult LD population and significantly greater than that of the normal child population.

Conclusion: With enough patience, suitably adapted methods and a skilled investigator, visual assessment is feasible in the majority of children with LD and the detection rate of correctable refractive error is significant.

P17

Duane's retraction syndrome associated with mitochondrial DNA mutation

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Introduction: Duane's retraction syndrome (DRS) is a rare condition resulting from a paradoxical innervation of rectus lateral muscle by branches of the oculomotor nerve. We report a case of rare association of Duane's retraction syndrome with mental retardation associated with mitochondrial DNA mutation in a seven-year-old child.

Methods: Case report Results: She has been referred to our department because of an abnormal eye position associated with a slight torticollis. When first seen by us, her visual acuity was 20/20 in each eye and the ocular motility examination revealed type 1 left Duane's syndrome. It was characterized by a left globe retraction with normal adduction in right gaze and narrowing of the left interpalpebral fissure. In left gaze, restricted abduction of the left eye was noted. Primary position showed no deviation. This oculomotor condition was associated with a mental retardation of an unknown origin. A muscle biopsy was performed which disclosed an important decrease of mitochondrial DNA related complexes. Mitochondrial DNA testing showed two homoplasmic mutations with a methionine to threonine transformation. Molecular biology could not be performed in any other member of her family as she lives in a welcome family because of a difficult social situation since she was born.

Conclusion: To our knowledge, this would be the first case reporting such an association which may be fortuitous. However, we cannot eliminate the possibility of a mitochondrial origin of this unilateral DRS associated with mental retardation.

P18**Severe form of tuberous sclerosis in an eight-year-old child**

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Tuberous sclerosis complex (TSC) is a multisystemic disorder characterized by the existence of tumor-like lesions (hamartomas) at multiple organs. The severity and symptomatic expression of TSC is highly variable, from the classic triad described by Vogt (epilepsy, mental retardation and adenoma sebaceum) to the involvement of the major organ systems. The authors present the case of an eight-year-old child with a severe and precocious form of TSC, with involvement of CNS (epilepsy since the seventh month of living, mental retardation, cortical tubers and periventricular subependymal lesions, including one at the foramen of Monro), skin (hypopigmented spots at birth, facial angiofibromas, shagreen patch on the forehead extending to the right eyelid) and eye (multifocal retinal hamartomas bilaterally). These retinal hamartomas have the three basic morphologic types already described in the literature, which leads us to think they are in different evolutionary stages. We performed photography and OCT of the retinal hamartomas and, in the future, we pretend to see their evolution repeating these imaging studies periodically.

P19

Neurofibromatosis type 1 (von Recklinghausen) with glaucoma and sphenoid-orbital dysplasia in a paediatric patient

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Introduction: NF-1 is an autosomal dominant disorder with high index of spontaneous mutations and extremely varied and unpredictable clinical manifestations.

Methods: We present a 6-year old boy suffering from NF- 1 with unilateral congenital glaucoma (buphthalmos) in the right eye detected at birth, plexiform neurofibroma involving the right upper eyelid with associated mild ptosis and ipsilateral facial hemihypertrophy. Multiple café-au-lait spots were detected in the trunk. There was no positive family history.

Results: His visual acuity was light perception in the buphthalmic right eye with dense corneal opacity and dense amblyopia. The left eye was normal. Biopsy of the right lid confirmed the plexiform neurofibroma. The CT of brain and orbits revealed an unusual type of sphenoid-orbital dysplasia.

Conclusion: This interesting case demonstrates multiple typical NF-1 features but the specific sphenoid-orbital dysplasia is a feature of the disease rarely described.

P20

Long-term clinical and visual results in extremely preterm patients with retinopathy of prematurity

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Purpose: To analyse clinical findings and visual functions in ROP patients with birth weight less than 1000 g during follow up.

Methods: Clinical records, visual acuity, refractive errors, electroretinograms, visual fields, OCT and fluorescent angiograms for 23 patients (41 eyes) aged 6 years and older were reviewed.

Results: The clinical and Fluorescent findings of the peripheral retina were: the abnormal and incomplete vascularisation were at 8 eyes; pigmented chorioretinal atrophic scars, retinal thinings, pre- and intraretinal fibroses – at 15 eyes; local retinal detachment – at 6 eyes. The dragging of disk and central retinal vessels with peripheral retinal changes were detected in 12 eyes. Median best-corrected visual acuity was 0,6 (20/30) with a median refractive errors of – 5,6 diopter spherical equivalent (range, - 16,25 to + 0,5 D). 9 eyes with no any clinical macular changes and visual acuity 20/30 and less had OCT findings: an abnormal contour in the foveal area, neurosensory detachment or vitreomacular traction. Subnormal max-ERG was registered in 85% cases, subnormal flicker-ERG – in 79%. Reduce of waves of macular ERG (especially, on red stimulus) was detected in 90%. Changes of multifocal ERG (abnormal configurations and amplitudes, decreasing of retinal response of P1 component) were detected on 32 eyes (78 %). In 63% cases central and peripheral defects of visual fields were founded.

Conclusions: Extremely preterm patients had a high frequency of the clinical and functional disorders of different retinal layers, defects of visual fields, and refractive errors, that possibly take part in the pathogenesis of decreased visual acuity in ROP.

P21

The examination of the TGF β genes expression activity level and their receptors in inborn et traumatic cataract post-operative materials – preliminary results

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Introduction: Transforming growth factor is one of the most important factor influencing the regulation of cell behaviour in ocular tissues in physiological or pathological processes. In human lens three isoforms of TGF β - β 1, - β 2 and β 3 are known. Previous studies have established that all three TGF β isoforms are cataractogenic for lens cells.

The aim of this study is to determine the role of the TGF β and its receptors in childhood cataract formation.

Methods: The material from 20 patients after cataract surgery has been examined. The patients were between 3 months and 20 years of age at the time of operation, averaging 7.9 years of age. The examined group consisted of 14 patients who underwent inborn cataract surgery –including 13 patients with binocular cataract, while the comparative group consisted of 6 patients after traumatic cataract surgery. For molecular examination the fragments of tissue of front bag and lens, which be removed from the eye during cataract surgery have been used. At the next stage, the taken tissues were kept at temperature of -70°C and the RNA extraction was conducted. The transcriptional activity of TGF- β 1, - β 2, - β 3, and their receptors in genes was assessed on the basis of mRNAcopy number/1 μ g of total RNA by the use of the real-time QRT-PCR technique.

Results: Expression of all three isoforms TGF β genes and their receptors was detected in all examined patients. A comparative analysis of results was conducted. The quantitative relations between three TGF β isoforms are as follows: inborn cataract TGF β 1(13402) > TGF β 2(2932) > TGF β 3(2779), traumatic cataract TGF β 1(14079) > TGF β 2(8387) > TGF β 3(2357). The quantitative relations between three isoforms TGF β receptors are as follows: inborn cataract TGF- β 1Rec(7400) > TGF- β 2Rec(7934) > TGF- β 3Rec(1328); traumatic cataract TGF- β 1Rec(23708) > TGF- β 2Rec(12541) > TGF- β 3Rec(9453).

Conclusion: In inborn cataract, the low level of TGF β 2 and TGF β 3 expression has been observed, whereas in traumatic cataract only TGF β 3 has been observed. In inborn cataract, the level of expression at all three TGF β receptors was smaller but for the TGF β 3 receptors expression was 7 times smaller in comparison to traumatic cataract. The results show the differences in expression of TGF β isoforms and their receptors in both types of cataracts. This finding suggest that each of TGF β isoforms may play a different role in cataract formation.

P22

Bilateral leukokoria in a newborn

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Introduction: Leukokoria in an infant or child is an alarming clinical sign. Parents have glimpsed an unusual appearance of the eye, depending on the source of illumination. Referral and investigation in these children are to be carried out urgently. Retinoblastoma is the first diagnosis to be excluded; many other underlying causes have been described.

Methods: We describe the case of a 5 week old boy presenting with bilateral leukokoria. He is the first child born to healthy non-consanguineous parents after a normal pregnancy and delivery. A complete ophthalmological examination with additional B-scan ultrasonography was performed. We also referred the patient for a neuropsychiatric investigation, Computer Tomography (CT) and Magnetic Resonance Imaging (MRI) of the brain. A blood sample for DNA-analysis was taken for genetic mutation analysis of the retinoblastoma and Norrie disease (pseudoglioma) genes.

Results: Ophthalmological examination showed a fixed non-reactive pupil, a clear cornea with normal diameter, a narrow anterior chamber, an atrophic mydriatic iris with synechiae, a clear lens and a retrolenticular membrane comprising of dysplastic retina with exudates and haemorrhages. The intraocular pressure was 20 and 26 mm Hg in the right and left eye respectively. On B-scan ultrasonography a closed funnel retinal detachment was seen; there were no calcifications. Clinical neuropsychiatric and hearing investigation were normal. CT and MRI examination showed a bilateral retinal detachment, a focal retrolenticular mass and contrast captation of the optic nerves. There were no other cranial abnormalities. The diagnosis of retinal dysplasia was postulated.

Conclusion: Leukokoria is an alarming sign in an infant or child. A complete investigation is urgent. The differential diagnosis in our patient includes: Norrie disease, Osteoporosis-pseudoglioma syndrome, Walker-Warburg syndrome, incontinentia pigmenti and bilateral severe familial exudative vitreoretinopathy.

P23**Orbital cellulitis in children: clinical diagnostic criteria**

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Introduction: Differential diagnosis between orbital (OC) and preseptal cellulitis (PC) in children based on clinical manifestations can be difficult. A CT was used to differentiate both groups. We present a retrospective study of these differences to achieve as good as possible majors (x 10 or more) and minors (x 2 or 3) diagnostic criteria.

Methods: Inclusion criteria: children with severe eyelid oedema enough that went into our hospital from January 1st 2005 to December 31st of 2007, especially when oral antibiotics don't improve the disorder. We include 103 children from 0 to 17 years. Parameters: month of the year, age, laterality, beginning of symptoms, oedema, chemosis, pain, proptosis and displacement, diplopia, visual acuity, fever, signs of superior respiratory pathways infection, leucocytes account and related data. Statistics: Prospective comparative study. Test chi-square was the method for compare both groups.

Results: • Low differences in the month of disease $p=0,851$ • Low differences in age $p=0,851$ • Beginning of symptoms (days) $p=0,773$ • One eyelid edema $14,3 < 44,4$ $p=0,035$ • Chemosis $p=0,335$ • Pain $20,3 > 1,9$ $p=0,063$ • Proptosis $28,6 > 1,9$ $p=0,002$ • Lateral displacement $12,2 > 0$ $p=0,071$ • Diplopia $8,1 > 0$ $p=0,205$ • No high leucocytes account $81,6 > 74,1$ $p=0,394$ • Fever $20,4 < 42,6$ $p=0,029$ • Upper respiratory disease $63,3 > 22,2$ $p=0,000$ • Related antecedents with PC $0 < 42,6$ $p=0,014$ • Way in of infection of PC present $0 < 33,5$ $p=0,000$ • Hemoculture positive $4/49 > 0/41$ $p=0,006$ • Culture of secretion negative $2/2 < 2/6$ $p=0,000$ We define the diagnosis criteria according to this data: • Major criteria of OC (more frequent than 10 times): painful ocular movements, proptosis, lateral displacement of the globe, diplopia and blood culture positive. • Major criteria of PC (more frequent than 10 times): pathologic antecedents related to a preseptal disorder, way of entrance of the pathogenic agent recognizable and culture of local secretion positive. • Minor criteria of OC (2 to 3 times more frequent): associated clinical manifestations of superior respiratory pathways infection (x 2, 85) • Minor criteria of PC: one eyelid oedema (x 3, 1), no fever ($< 38^{\circ}\text{C}$) (x 2, 08).

Conclusion: Some clinical signs can intensely orientate to OC or PC. It could obviate the irradiation of CT in small children

P24

Congenital simple ectopia lentis with unusual asymmetric bilateral lens displacement in a paediatric patient

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Introduction: Ectopia lentis is a significant diagnostic clue in a number of ocular and systemic comorbidities.

Methods: We present an interesting case of congenital simple ectopia lentis with unusual asymmetric bilateral lens displacement in a three-year old girl. She presented with progressive reduction of visual acuity. The personal and family history was negative of any ocular and systemic comorbidity.

Results: She had an abnormal head posture, a remarkable iridodonesis in both eyes, minimally dislocated downward and nasally lens in the right eye bisecting the pupil and totally dislocated free-floating in the vitreous lens in the left eye. Axial length, corneal diameters, keratometric measurements and fundus examination were normal. Systemic disorders such as homocysteinuria, Marfan's or Weill-Marchesani's syndromes were ruled out by paediatric evaluation of general health including metabolic screening and echocardiography.

Conclusion: In congenital simple ectopia lentis the ocular anomaly is usual manifested as bilateral, symmetric, upward and temporal displacement of the lens. In our case an unusual and very asymmetric lens displacement was observed, features that to our knowledge have not been reported before.

P25

LTBP2 mutation in autosomal recessive microspherophakia with lens luxation and megalocornea

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We observed a large consanguineous family with three children affected with microspherophakia, progressive lens luxation, axial myopia and megalocornea. They also all presented ocular hypertony, two of them suffered retinal detachment and the two eldest children had low intelligence (low IQ). They did not meet the diagnostic criteria for Marfan (MFS) and Weill-Marchesani (WMS) syndromes. No mutation was found in the MFS-associated gene FBN1. Locus mapping by homozygosity to a 12.6 cM region of chromosome 14q2 was performed, using a 10K GeneChip SNP array in the affected siblings. It was followed by microsatellite analysis, with a multipoint LOD of 2.57. The linkage interval contained one conspicuous candidate gene, LTBP2, encoding a latent transforming growth factor-beta binding protein. LTBPs are extracellular matrix proteins with multiple domain structures bearing strong homologies with fibrillins. They may play several roles, including fine control of TGF- β activity in the matrix, a structural role in microfibrils, and a role in cell adhesion. A truncating mutation g.76339dupC (p.Pro599ProfsX4) was found, homozygous in the affected siblings, heterozygous in the parents, and absent from 100 unrelated control subjects from the same ethnic group. Using a polyclonal antibody, LTBP2 was found to be strongly expressed in the calf ciliary zonule. Fibroblast cultures and lymphoblast cell lines from the affected siblings are under study. We conclude that the LTBP2 truncating mutation reported here is a rare cause of autosomal recessive microspherophakia with lens luxation.

P26**Myelinated Retinal Nerve Fibers and Amblyopia : 2 case reports**

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Introduction: Report of two cases.

Methods: Review of the medical records of two children with extensive myelinated retinal nerve fibers in one eye, anisometropia and amblyopia. In both cases the treatment consisted of occlusion and soft contactlens wear.

Results: A marked improvement in visual acuity was obtained.

Conclusion: Good results can be achieved with aggressive amblyopia treatment.

P27**Nutritional Deficit and bilateral hyperopic amblyopia**

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Introduction: purpose: to present a case of bilateral amblyopia in an Indian boy with history of nutritional deficit and to differentiate among organic causes of low vision

Methods: to study the medical history of a boy aged seven years with nutritional, development and neuropsychological defects

Results: the patient is using bilateral hyperopia correction and he has a multidiscipline follow up.

Conclusion: we suspect of an organic cause when we have not improvement of visual acuity but generally the continuous use of the optic correction improves vision little by little in bilateral hyperopic amblyopia.

P28

Change of Visual Field Defect between Short Term Intervals in Child with Migraines

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Introduction: To report a spreading visual field defect in a child with migraines.

Methods: A 14-year-old boy presented with headache with visual disturbance. He had been suffering from a headache. No abnormal findings were noted in the pediatric clinic, and brain magnetic resonance imaging was normal. On ophthalmologic examination, best corrected visual acuity was 1.0 in both eyes, and no abnormal findings were noted on slit lamp examination. A central scotoma was found in the right eye on visual field testing. Therefore, we rechecked the visual field test nine days later.

Results: The visual field test changed at intervals. The first test showed peripheral depression, which changed to left incongruous hemianopsia after 1 hour. One hour later, the visual field defect was normalized.

Conclusion: Temporary, especially spreading visual field defects occurring in succession may be seen in migraine patients. This characteristic visual field defect is helpful when analyzing the clinical features of migraine and interpreting the migrainous visual aura.

P29**One brother and sister with mirror image myopic anisometropia**

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Introduction: To report one brother and sister with mirror image myopic anisometropia

Methods: Sister was 10 years 11 months old and brother was 8 years 4 months old. There was a myopic anisometropia in the right eye of sister and myopic anisometropia in the left eye of brother.

Results: The full ophthalmic examinations were performed and the cycloplegic refraction was done. The differences in refractive power (spherical equivalent) between both eyes were -12.5 in sister and -6.975 diopter in brother, respectively. The anisometric amblyopia was found in both of them. The abnormal ocular movement was not recognized and fundus was shown no specific findings.

Conclusion: The co-occurrence of severe myopic anisometropia in brother and sister is extremely rare. The present case suggests that severe myopic anisometropia may be related by genetic inheritance.

P30**Misdiagnosis in connection with Elevated Intracranial Pressure**

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Introduction and Aims: Two cases are presented in order to focus attention on neuro-ophthalmological entities that are rarely encountered in pediatric ophthalmological practice.

Patients and Methods:

Case 1 - Girl, aged 6, otherwise healthy presented with headache lasting for four weeks, which started after the healing of a purulent otitis media. Her first diagnosis was Benign Idiopathic Intracranial Hypertension. Although dismissed from a Pediatric Hospital, her complaints did not cease.

Case 2 – Nine-year-old girl with a strong headache and diplopia was examined on a Pediatric Ophthalmological Ambulance. She has been living with a ventriculo-peritoneal shunt from almost her birth. The child was first diagnosed with convergent strabismus and bilateral optic disc swelling but was sent for consultation to the University Ophthalmological Department. Routine ophthalmologic examination and funduscopy were performed on a serial basis in both cases. Other examinations comprised MRI brain, MRV and consultations with specialists from connected fields.

Results:

Case 1 – True optic disc swelling still measuring 3 diopters was seen on both sides. The girl was sent to a second, more detailed neurological consultation and MRV revealed the cause of raised intracranial pressure.

Case 2 – Bilateral optic disc swelling of 2 diopters were found on fundus examination accompanied by bilateral sixth nerve paresis. Neuro-surgical consultation established the etiological diagnosis.

Discussion: Final diagnosis proved to be Sigmoid Sinus Thrombosis in case 1 and Shunt Malfunction in case 2. Only full history of the patients could give clues to what was happening to the patients. These are shown in details on the poster. Quality communication is essential between medical subspecialities.

Conclusion: Ophthalmologist should be well informed about general and ophthalmological history of the examined patient and should be aware about the significance of the findings. Also they should be able to make it clear to colleagues.

P31**Ocular tilt reaction and internuclear ophthalmoplegia associated with sinus venous thrombosis: an unusual case**

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We report about a 11 year old previously healthy girl, who presented with an acute history of vomiting, neck and back pain, diplopia and photophobia after a 4 day treatment for bilateral otitis media. Cerebral CT revealed a sinus thrombosis, which was treated with anticoagulation therapy. She developed a head tilt and unusual oculo-motor findings, progressive worsening of papilloedema with subsequent retinal nerve fiber layer and optic nerve atrophy. The case underlines the importance of early and regular ophthalmological assessment and surveillance in the diagnosis and management of patients with complicated venous sinus thrombosis and intracranial hypertension. In children too ill or young for reliable perimetry progressive optic disc and RNFL changes may be more useful in monitoring the effectiveness of treatment. INO and OTR may reflect marked degrees of intracranial hypertension and can serve as additional clinical parameters for the clinician to monitor.

P32

Plasticity of visual functions after brain damage

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Introduction: "Plasticity is a universal feature of all (developing) living things. It allows adaptation, i.e. change in response to the challenges of life". Defects of visual functions are often considered irreversible, when they have not recovered 6 to 12 months after brain damage.

Methods: Long-term (3 – 15 years) follow-up data of three patients with post-chiasmatic damage to the visual pathways was analysed with special reference to visual acuity and visual fields.

Results: Progressive recovery of visual acuity and visual fields was documented many years after brain damage.

Conclusion: Visual acuity and visual fields can recover after damage to the retrochiasmatic visual pathways, not only in the very young, but also in older children and young adults.

P33

Retinal Dystrophy in CEDNIK-syndrome

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Purpose: to describe the retinal dystrophy in a brother and sister who probably have CEDNIK syndrome.

Methods: observational case reports

Results: both children were born from consanguineous parents. The boy is 9 years old and the girl 6 years. They are mentally retarded, have dysmorphic features, acrodermatitis, MRI abnormalities, nystagmus with a high myopia and a retinal dystrophy. The MRI of the brain showed a partial agenesis of the corpus callosum in the brother and a complete agenesis in the sister. Myelinisation was delayed in both children. Both children made no eye contact during the first month of their live and had roving eye movements. Visual responses improved after the age of one year, and a nystagmus developed. They both had a high myopia (-10 D and -13 D). Funduscopy in the brother at half year of age showed dark pigmentation in the macular area. At one and a half year the macula showed linear pigmentations and the peripheral retina a more scattered pigmentation. The sister also had small circular dark pigmentation of the macula at three month of age. She also developed peripheral pigmentation at later age. At five years of age the macular linear pigmentation had the shape of a 'Mercedes-star' and the pigmented lesion of the peripheral retina were circular. Electrophysiological testing showed in both children a negative waveform under scotopic conditions, with prolonged implicit time of the fotopic responses.

Conclusion: CEDNIK-syndrome is a neurocutaneous syndrome caused by an abnormal SNARE-protein involved in vesicle fusion, characterised by cerebral dysgenesis with corpus callosum abnormalities, neuropathy, ichtiosis and keratoderma. In the only report on this syndrome, Sprecher et al. (Am J Genetics 77:2005; 242-251) only give a confined description of the ophthalmological features. The ERG responses in our two patients suggest a defect in the second order neurons or their synapses in the rod visual pathways. Both patients had a high myopia. The pattern of retinal pigmentation is unlike classical retinitis pigmentosa or any other known retinal dystrophy. Confirmation of the clinical diagnosis of CEDNIK syndrome in both children by identification of a SNAP29 mutation will be started in the immediate future.

P34

Secondary glaucoma in children with Vogt-Koyanagi-Harada syndrome

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Objective: To evaluate frequency, clinical features and efficiency of treatment secondary glaucoma in children with Vogt-Koyanagi-Harada (VKH) syndrome.

Methods: 11 children (2 boys, 9 girls) aged 3 – 17 years (mean 7.7 years) with chronic bilateral panuveitis, related to VKH syndrome were examined. Besides of typical ocular findings of panuveitis neurological (severe headache) and dermatological (poliosis, vitiligo) symptoms presented in 27% and 46% patients correspondingly.

Results: Glaucoma occurred in both eyes of 4 children (36%) during acute or subacute phase of the disease. The intraocular pressure (IOP) ranged from 26 to 42 mm Hg and signs of acute glaucoma were revealed in 3 eyes. The most prominent sign was diffuse iris thickening confirmed by ultrasound biomicroscopy. Elements of pupillary block due to reduced pupil movement, posterior synechias and fibrous prelental membranes formation were also founded. As a result shallow anterior chamber, narrow and partially closed anterior chamber angle took place in all cases. Treatment with β -adrenergic antagonists, topical carbonic anhydrase inhibitors and mydriatics was effective in 2 of 8 eyes. Repeated YAG laser iridotomies performed in 4 eyes were unsuccessful in all cases because of iris thickening. Peripheral iridectomies combined with posterior synechias dis-section and prelental membranes removal reduced IOP in 2 of 6 eyes. In 4 eyes steady IOP compensation was achieved only after lensectomy with anterior vitrectomy.

Conclusion: Uveitis in pediatric VKH syndrome often causes secondary angle closure glaucoma resulting from iris thickening and elements of pupillary block. When topical hypotensive medical therapy is unsuccessful to reduce the IOP lensectomy with anterior vitrectomy should be performed for management of this complication.

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P35**OPTIC NEURITIS IN CHILDREN - A LONG TERM FOLLOW-UP**

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Aim: clinical and electrophysiological investigation years after acute optic neuritis in childhood.

Methods: 7 persons (1 male and 6 female) from 15 to 31 years of age were investigated in average 10.5 years (8-18 years) after acute optic neuritis. At the acute phase 4 had both eyes affected, while 3 only one eye (11 affected eyes). 5 clinical parameters were compared with the acute phase: visual acuity, pupillary reactions, optic disc appearance, visual fields and colour vision. Electrophysiological parameters included VEP P100 wave latency and amplitude. Standard tests (Snellen charts, swinging flashlights, direct/ indirect ophthalmoscopy and fundus photography) were utilized to test visual acuity, pupillary reactions and optic disc appearance. Visual fields were tested with Goldman and Octopus G2Top perimetry. Colour vision was investigated with Ishihara plates, Nagel anomaloscope, D15 saturated and desaturated test and Farnsworth-Munsell 100 hue test.

Results: None of our patients had multiple sclerosis over the follow-up period and they also didn't have any registered recurrences of optic neuritis. Their final visual acuity was normal in 10/11 eyes, pupillary reactions were normal in all eyes, optic disc was pale in 10/11 eyes, minor visual field defects were observed in 4/11 eyes and residual colour vision deficiencies were present in 6/11 eyes. Four eyes still showed prolonged P100 wave latency.

Conclusions: Despite excellent visual acuity residual colour vision deficiency and optic disc pallor were often observed in persons with optic neuritis in their childhood.



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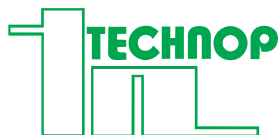
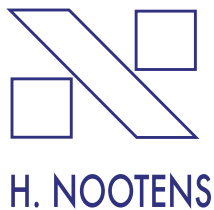
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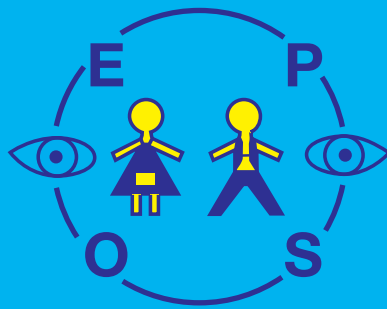
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