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InteRyc-volume 3, July, August and September, 2002

ALL INDIA STRABISMOLOGICAL SOCIETY

JKA Institute of Strabismology and binocular Vision

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President AISS, Director JKAI & Author & Editor of InteRyc:
Sudha Awasthi Patney, MBBS, MS (Ophth), FRCOphth (London)

(NOTE: The following is a repeat for obvious reasons)

A special request to the members: Prevent strabismus and amblyopia in children

This is an appeal to all the members to please start a campaign for prevention of amblyopia. Actually I am of the opinion that a legislation is needed badly, that will make it compulsory that every child's eyes are thoroughly examined by the age of 1 year, so that measures can be taken to prevent amblyopia (strabismic, anisometropic and ametropic) and strabismus. If it could be done for vaccination, it can be done for eye examination also.

At present there is general indifference towards this subject. It is also obvious that pediatricians and ophthalmologists have to be trained not to advise delay in treatment because the patient is a young child / infant. It is tragic that although parents have now become aware of the need for early treatment, the pediatricians only rarely refer them to ophthalmologists who are advising them to wait until the child is 8-10 / old enough for examination. We have to advise them strongly against this practice. If we can not compel the Government to bring in legislation, we can at least alert the public, the pediatric physicians and the ophthalmologists.

It is obvious that many more Institutes of Strabismology are needed in various parts of the country. Would you, dear members, be willing to take on the task of starting a branch of this JKA Institute in your area? *Any help and advice that I am capable of providing will be forthcoming. You will need some basic instruments to start with. Orthoptic instruments are the cheapest of the lot, have you noticed*? *Please let me know at once if you are interested.*

Please try to alert the patients, parents and other relatives, the public and other physicians, particularly ophthalmologists and pediatricians about the dangers of amblyopia, strabismus and other complications if significant refractive errors are not corrected within the first years of life and if strabismus is not treated immediately.

It is very painful to see so many cases of amblyopia. This condition, as you know, is totally preventable if treated early, whatever the age of the patient, the younger the better. The best time is immediately after the start of strabismus. However, it is obvious that to prevent ametropic and anisometropic amblyopia and in many cases strabismus, the children have to be thoroughly checked at least once by the age of 1 year. The saying that prevention is better than cure is *truest* in the case of strabismus and amblyopia.

INFORMATION

1. About the Institute
2. About the Society
3. About the courses
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5. About InteRyc, the News-Letter-Update of the society
6. About the Indian Orthoptic Journal to be restarted soon.

1. *About the Institute*

A) *The need to have a squint treatment center* and a training center for strabismologists and orthoptists in India could not be ignored anymore in *nineteen fifties*. Dr. H.L.Patney felt it most acutely as he had trained as a premedical student, medical undergraduate and postgraduate in ophthalmology in UK. He had been doing orthoptics, contact lenses and all types of surgery as a Registrar in the Ophthalmology department of the Royal Cardiff Infirmary in Cardiff, Wales, UK back in 1942-44. He had the good fortune of being the assistant of Sir Tudor Thomas and used to assist him in his private practice also. Sir Tudor Thomas was a living legend in those days and was a pioneer in keratoplasty. However, he did all types of operations including retinal detachment repair and plastic surgery. Young Dr. Patney was given lots of opportunity to operate even on Sir Thomas' private patients. Sir Thomas was a very famous and busy man and he must have had confidence in Dr. Patney's prowess in surgery as he gave him even major surgeries to do. Sir Thomas' words and signatures on Dr. Patney's old books testify to this.

In 1946 when Dr. H.L. Patney was asked by Dr. Mehrey, the founder of Sitapur Eye Hospital to make a plan for the expansion of the hospital, he did a thorough job. He included in the plan, the name of *a squint / orthoptic department and school* along with those of ocular pathology, instrument factory, blind school, optometry school, postgraduate institute of ophthalmology, trainee's hostels, staff's residences etc. Much later he used to say that everything in that plan materialized except a boundary wall.

Dr. Mehrey who was himself keen on keeping everything upto date in his hospital happily worked hard to realize their dreams. It took them a few years to get a first rate orthoptic department and school.

1) The beginning was with *an orthoptic department* in early fifties by Dr. Patney who taught a smart compounder in the hospital the basic techniques of orthoptic examination and exercises on synoptophore.

2) *The Orthoptic School* was started in 1960 and according to plan Dr. Sudha Awasthi (who was at that time in K.G. Medical College, Lucknow) was asked to join the hospital by Dr. M.K.Mehra, (Dr. Mehrey's son). Dr. Awasthi had just passed her MS (Ophth.) from King George's Medical College, Lucknow, and was known to be specially interested in the subject. She joined Sitapur Eye Hospital and was soon after sent to London in October 1960.

3) A first rate orthoptic department, the first in India, which was on the lines of that at Moorfields Eye Hospital (High Holborn branch where Mr. T. Keith Lyle was the Director), was established after she returned from London after 1 year's training under Mr. Lyle.

B. *The need for imparting training in the subject of strabismology* (including orthoptics), was repeatedly impressed upon Dr. Sudha Awasthi (now Patney) by another living legend of those days, Mr. T. Keith Lyle. He was in 1960 and later for many years, the Dean of Institute of Ophthalmology, London and Director and Surgeon-In-Charge of the famous Orthoptic Department of the Moorfields Eye Hospital (High Holborn branch), London. Dr. Sudha Awasthi was training under him to further her somewhat limited knowledge of the subject, already gained during the running of an orthoptic clinic by her from 1957 to 1959 under the guidance of Prof. M.K.Mehra, a double FRCS.

Mr. Keith Lyle insisted that she should also train like an orthoptist-trainee in their Orthoptic School to gain first hand practical knowledge so that she can train orthoptists and Ophthalmologists / strabismologists with confidence. She stayed at Moorfields Eye hospital for 1 year and was then sent to Germany and Switzerland to learn first hand, pleoptics from the two pioneers (Prof. Cuppers of Giessen, W. Germany and Prof. Bangerter of St. Gallen, Switzerland, respectively). On her return to India in 1961, the ground was ready for her to impart to the ophthalmologists and the orthoptic trainees, special training in strabismology and orthoptics. The *first Squint / Orthoptic department and Orthoptic School of India had already been started at Eye Hospital, Sitapur, which was the premier eye institution of India in fifties, sixties and seventies* (for some more information see the inside of the back page). During her days there she kept on running the squint department, training the orthoptists, DOMS candidates (as Associate Professor in the Nehru Postgraduate Institute of Ophthalmology) and visiting ophthalmologists wishing to learn the subject.

C. *The idea of starting a training institute for strabismology* was conceived soon after Dr. H.L. and Dr. Sudha Awasthi-Patney left Sitapur and came to Rajkot.
The center for squint treatment was being run since their arrival in Rajkot in 1972 but a formal inauguration of a training center was performed in 1983. However, due to Dr. H.L. Patney's serious and prolonged illness the plan had to be kept suspended. The Institute started functioning in real earnest since 1996 but the foundation was being strengthened by Dr. Sudha Awasthi-Patney since 1994. She took a 4.5 months study tour of USA and UK in 1994, followed by annual visits to update her knowledge in preparation for starting and running the Institute and reviving the AISS. New orthoptic instruments were bought and old ones serviced.

D. AS already mentioned, the Institute became functional in 1996 along with the newly revived AISS.

E. At present there are only 29 members in good standing, i.e., the members who have paid up their dues until last year (2001). In all there were 88 registered members. Invitation to join the society was circulated once only. It has never been repeated / sent out again after 1997.

F. The Institute is running a fellowship course by correspondence. A diploma course is soon to be started for people who find the fellowship course too hard.

G. Other activities are the various annual contests, the winners getting trophies and cups and a total of Rs.4350 in cash prizes every year.

H. A free squint camp (diagnostic and surgical) is held every year, usually in collaboration with the Rotary Club of Rajkot Midtown.

I. There is a fellowship (Rs.1000/pm) for members 35 years old or younger during their stay at Rajkot for practical experience. So far nobody has applied for it.

2. *<u>About the Society</u>*

(1) All India Strabismological Society (AISS) was *conceived and started* by Dr. H.L. Patney and Dr. Sudha Awasthi in 1967. The idea came to them during their participation in the founding session of the International Strabismological Association (ISA), which was held in 1966 at Giessen, W. Germany. Prof. Cuppers, one of the pioneers of pleoptics was the head of Ophthalmology at the Universitats Augenklinik (University Eye Clinic) there. Mr. Keith Lyle was the founding president and Dr. G.K. von Noorden, the founding secretary. Dr. Sudha Awasthi was one of the panelists and speaker at the ISA meeting.
One of the 4 aims of ISA is to spread the knowledge of the subject of strabismology. The other three are given on the inside of the front cover.

(2) The founding meeting of the society was held in Calcutta in 1967 during the AIOS conference. Neither Dr. Patney nor Dr. Awasthi wished to be the President. They asked Dr. L.P.Agarwal to be the first president and he accepted. Dr. Awasthi was the founding secretary and Dr. Patney the treasurer. Many senior and well-known ophthalmologists joined the society.

(3) The first regular meeting was held at Ahmedabad during the AIOS congress in 1968. At the executive committee meeting, a proposal to have the *society registered* was passed. This was done same year..

(4) The first activity of the new society was to hold a 7-days refresher course (workshop) on squint and other ocular motility disorders in September 1967 at Sitapur. It turned out to be very successful, probably because it was the first of its kind in India. Members who were mostly senior ophthalmologists attended it; some of them were fairly well known.

(5) Every year new elections were held and the management of the society changed hands. Somewhere around 1976 the society became defunct.
Note: Frankly speaking this is a drawback in the democratic system that a lovingly conceived and nurtured institution / organization may die a premature death if it falls into indifferent hands.

(6) *Revival of the society* was proposed during a meeting (of old members and some other ophthalmologists), that was hastily arranged at the request of Dr. Sudha Awasthi-Patney in 1981 just after the conclusion of Dr. Nagpal's very successful National Symposium on squint. It was decided to revive the society during the next conference of AIOS and Dr. Sudha Awasthi-Patney was asked to be the convener and do it. New and old members gave their names to be enrolled again.
Dr. Awasthi-Patney unfortunately failed to attend the next AIOS conference in 1982 due to the sudden serious illness of Dr, Patney. She requested Dr. B.T. Maskati, the Hon. Gen. Secretary of AIOS to make an announcement that Dr. Awasthi-Patney can not come now but she will be sending circulars

for a meeting of the society to be held later at Rajkot. She never knew what happened but Dr. Prem Prakash started a new society. It is no use going into the details now.

(7) At last the AISS was revived in 1996. At present there are 88 members but out of them only 29 *are members in good standing (having paid at least upto year 2001***)**. Only 11 members have paid for 2002.

3. *About the courses*

(a) *Fellowship*: Theory part is sent in 15 installment of 50-100 pages each as the old X installment (last but one) having 5 parts was extended to 334 pages. The number of installments was raised from 11. Apart from the theory part, some practical experience has to be gained at the Orthoptic / Ocular Motility Clinic, Rajkot. The period of the practical experience has to be determined by the fellows themselves but a minimum of 1 month is preferable. The very minimum for somebody with some working knowledge of the subject is 1 week.

(b) *Diploma* (to be started soon): Detailed information on request.

(c) *Certificate of* Proficiency: If the ophthalmologists / strabismologists wish to get some practical experience only, as many of them did when I was at Sitapur Eye hospital, they are welcome. They will be given a testimonial (Certificate of Proficiency) for the period of their stay here. There will be no fees.

4. *About the workshops / Refresher Courses*

Some of the members who could not attend the September workshop have asked me to hold another soon. I shall see if it can be done.

5. *About InteRyc, the News-Letter-Update of the society:*

(A) At present it is being published every three months. Previously it was coming out every two months. If we revive the Indian Orthoptic Journal that had been started by Dr. Sudha-Awasthi Patney and Dr. J.M. Pahwa in 1963 at Sitapur, the InteRyc may have to be discontinued. We have an alternative plan also. Please read the item 6 below.

(B) It is sent free to every member of the AISS and JKAI but the subscription for membership must be sent every year for it to be economically viable.

(C) If the subscription for 2002 (that was due on January 1) is not received by December 31, 2002, I am sorry to say that it will not be possible to send the InteRyc until the subscription is received. I wish I had enough money to keep on sending them despite nonpayment.

6. *About the* ***proposed revival*** *of the Indian Orthoptic Journal*: Action on this proposal is being delayed because I had requested the members to give their names for the editorial board. We wish the journal to be started soon but at the moment not enough members have come forward to take part. We shall have to invite outsiders. At present 4 persons in all, myself, DR. S.K. Pal, Dr. Meenakshi Bajpai, Dr. R.M. Sahai. I wonder if Dr. (Prof.) N.C. Singhal would like to help out. Let's find out.

ATTENTION

1. *The CME quiz-No*.3, 2002 is included in this volume. Please answer it, cut along the dotted line and send it back by conventional mail. The answers to the CME quiz- No.2, 2002 are also included in this volume.
2. *The questions in each quiz* are drawn from the material given in that particular issue of the InteRyc under the headings of Strabismus Summary Series, Update, InformIT and Short Review article on Strabismus etc.
3. *Member of the year is chosen on the basis of overall performance during that year, including the answers to the quiz.*

4. The *update questionnaire* is printed on the back of the CME quiz. *Please do answer it if there is any change or addition in the information about phone No., FAX number, mobile phone number, pager number, E-mail address or a web-site address. When I try to call the members on phone I find that many numbers have been changed.*

5. *Please express your opinion on whether Indian Orthoptic Journal should be restarted and the other details. A poll form is given on page 33.*
Background of the Indian Orthoptic journal: Dr. Sudha Awasthi (Patney) was inspired greatly by her teacher Mr. T. Keith Lyle (read about him under the heading of "In fond memory" on the inside of back cover). He stressed the need of making the subject of strabismus popular among ophthalmologists and campaigning for early diagnosis in infants and children to prevent amblyopia. After coming back to Sitapur Eye Hospital in 1961, she conceived the idea of bringing out an Indian Journal of Orthoptics on the lines of the British Orthoptic Journal. Dr. J.M. Pahwa (who liked the idea and agreed to look after the practical aspect) and Dr. Awasthi (Patney) started the journal in 1963 and looked after it as the editor and the joint editor respectively until her departure from Sitapur in 1972. Dr. Pahwa continued publishing it until a few years back. About years back he asked Dr. Sudha A. Patney if she would like to restart publishing the journal to which she replied in the affirmative. Late Dr. Pahwa then sent some old papers relating to the society sometime back. The journal would probably replace the InteRyc, as it will be difficult to publish both unless there is a managing editor assisted by an editorial board.

6. *Fellowship course fees:* Now the total amount to be paid in one lump sum is Rs.1500 *including the mailing charges*. The mode of mailing each installment is either by registered A.D. post or by couriers, mostly by the latter as it is faster. However, couriers do not go to all the places. Moreover, once an installment sent by the courier did not reach a fellow and I sent another one by registered A.D. post. Now therefore, we shall have to send them by post despite more expense involved.

7. *The usual procedure of sending the installments*: Installments are sent one by one accompanied by the relevant question paper. The fellow has to answer the questions and send the answer sheet back, on receipt of which the next installment of the course is sent. Previously the fees had to be sent for one installment at a time. This has been changed to save the fellow's time, effort and postal expenses. It is now payable in one lump sum, in advance in the form of a demand draft for Rs1500, in the name of Dr. S.A. Patney, S/B account No.4256 at UCO bank. As explained in earlier InteRycs this is a no profit-some loss venture.

8. *The membership subscription for year 2002* became due on 1st January 2002. Members, who do not pay the subscription for the year 2002 by the end of December 2002 (the final extended date) will not be sent future InteRycs. This is because of financial constraints. Despite subsidizing the expenses we are finding it hard to keep afloat. The members, therefore, *are requested to send it soon.*

(Please see the Update questionnaire on the back of the Member of the Year 2002 Quiz 3).

Information about subscription:

(a) *All the members who have not paid for 2001 are requested to send two years subscription (for 01 and 02). It can be in the form of a demand draft for Rs.200 OR cheque for Rs.220, in the name of Dr. S.A. Patney, UCO bank S/B account No. 4256, Rajkot.*

(b) *Members who have paid for 2001 but not for 2002, are requested to send one year subscription only, DD for Rs.100 / cheque for Rs.120 only.*

NEWS

The only news is about the hands-on workshop on strabismus and Amblyopia that was held from September 13 through 19, 2002. Comments on the workshop are given in the letter on page 2. Details will follow in InteRyc volume 4, 02.

COMING UP

1. 12/3/2002**:** New York Society for Clinical Ophthalmology Winter Meeting: New Evidence, New Treatments: The Impact of NEI Clinical Trials on Glaucoma Management, New York, NY; contact Kim Corbin at (212) 979-4444; fax: (212) 982-1395; e-mail: kcorbin@nyee.edu.

2. 12/6/2002 - 12/7/2002**:**14th Biennial Cornea Conference: Current Topics in Corneal Diseases and Refractive Surgery, Philadelphia, PA; *contact:* Lucia M. Manes, Department of Continuing Medical Education, Wills Eye Hospital, 900 Walnut Street, Philadelphia, Pennsylvania 19107; Phone: (215) 440-3168; Fax: (215) 440-3176; Email: willseyecme@hslc.org

3. 12/7/2002**:** Advanced Excellence in LASIK, Los Angeles, CA; *contact:* Tollfree Phone: (800) 862-5266, ext. 6930.

4. 12/7/2002**:** Fugo Blade Certification Course, Norristown, PA**;** Sponsored by MediSURG Research & Managament Corp. Contact Dawn DelCampo at (610) 277-3937; fax: (610) 277-7256; e-mail: fugoblade@fugoblade.com.

5. 12/7/2002**:** Uveitis Update, Cleveland, OH; Sponsored by the Cleveland Clinic Cole Eye Institute. Contact Kelli Meeks, Education Coordinator at (216) 444-2010; fax: (216) 445-3676; e-mail: Meeksk@ccf.org.

6. 12/7/2002**:** "Basics By the Bay": Current Concepts in Ophthalmology for Ophthalmic Medical Personal, San Francisco, CA**;** contact Denice Barsness, CRA, COMT, ROUB, FOPS, California Pacific Medical Center, Department of Opthalmology, Eye Medical Services, 2100 Webster Street, ste. 219, San Francisco, CA 94115; (415) 923-3937; fax: (415) 923-6563.

7. 1/19/2003 - 1/24/2003**:** Hawaii 2003 The Royal Hawaiian Eye Meeting, Maui, HI; To be held at the Grand Wailea Resort. *Contact:* Registration Manager, Tollfree Phone: 1-877-307-5225; or Phone: 856-848-1000; Email: meetingregistration@slackinc.com

8. 1/23/2003 - 1/25/2003
   Phaco, Foldable and Refractive Results, Park City, UT;
   For more information or to register, contact: Education Department, Alcon Laboratories Inc., P.O. Box 6600, Fort Worth, TX 76115; Tollfree Phone: (800) 862-5266, ext. 6930.

9. 1/25/2003**:** VitreoRetinal Surgery Presents: Retinal Update 2003, Minneapolis, MN;
   For information, contact Mary Nordenstrom at (952) 259-3448; e-mail: mnordenstrom@visi.com

10. 1/30/2003 - 2/02/2003**:** 22nd Annual Squaw Valley Retinal Symposium, Squaw Creek, CA; contact Laura Wendel or Robert Wendel, MD at (916) 483-6299; fax: (916) 483-6297; e-mail: laurawendel@attbi.com.

11. 1/30/2003 - 2/2/2003**:** Current Concepts in Ophthalmology, Puerto Rico Meeting, Dorado, Puerto Rico; Sponsored by the American Society of Cataract and Refractive Surgery (ASCRS) and the Wilmer Eye Institute of Johns Hopkins University. Contact Lucy Santiago, ASCRS, (703) 591-2220.

12. 1/31/2003 - 2/1/2003**:** Glaucoma Summit 2003: Vision for the Fututre, Cleveland, OH; Sponsored by the Cleveland Clinic Cole Eye Institute. Contact Kelli Meeks, Education Coordinator at (216) 444-2010; fax: (216) 445-3676; e-mail: Meeksk@ccf.org.

13. 2/7/2003 - 2/9/2003**:** Seventh European Society of Cataract & Refractive Surgeons (ESCRS) Winter Refractive Surgery Meeting, Rome, Italy**;** *contact:* ESCRS, (353) 1-209-1100; Fax: (353) 1-209-1112; Email: escrs@agenda-comm.ie

14. 2/7/2003 - 2/8/2003: Bascom Palmer Eye Institute Mid-Winter Glaucoma Symposium, Miami, FL; *contact:* Nancy Fernandez, Bascom Palmer Eye Institute, 900 NW 17 Street, Miami, FL 33136; Phone: (305) 326-6110; Fax: (305) 326-6417; Email: nfernandez@med.miami.edu.

15. 2/8/2003**:** Seventh Annual Glaucoma Symposium, San Francisco, CA; *For information*, contact Glaucoma Research & Education Group, 490 Post Street, suite 644, San Francisco, CA 94102; (415) 986-0835; fax: (415) 986-0876; e-mail: greg@glaucomausa.org.

16. 2/8/2003**:** The Association for Research in Vision and Ophthalmology (ARVO) Annual Meeting, Ft. Lauderdale, FL**;** contact the Association for Research in Vision and Ophthalmology, 12300 Twinbrook Parkway, ste. 250, Rockville, MD 20852; (240) 221-2900; fax: (240) 221-0370; e-mail: mem@arvo.org.

17. 2/13/2003**:** Australasian Society of Cataract & Refractive Surgeons (AUSCRS) 2002 Meeting, Capital Gains-Canberra, Australia; contact Prof. Ingrid Kreissig, MD, (49) 7071-29-8-50-64; fax: (49) 7071-29-52-09; e-mail: ingrid.kressig@med.uni-tuebingen.de.

18. 2/13/2003**:** South Pacific Educators in Vision Impairment Biennial Conference: "An Eye to the Future," Gold Coast, Australia**;** contact the Conference Secretariat, P.O. Box 3496, South Brisbane Q 4101, Australia; (61) 7-3844-1138; fax: (61) 7-3844-0909; e-mail: spevi2003@icms.com.au.

19. 2/13/2003 - 2/15/2003**:** American College of Eye Surgeons' Quality Surgery XVII Program, "Ophthalmology 2003...United in Quality," Kissimmee, FL; contact Continuing Education, Inc. at (800) 422-1571; e-mail: contactus@continuingeducation.net.

20. 2/14/2003: Redefining Glaucoma and its Management in the 21st Century, Baltimore, MD; contact Nancy K. Cook at (410) 328-5929; fax: (410) 328-6346; e-mail: ncook@aol.com

21. 2/14/2003 - 2/16/2003**:** New Orleans Academy of Ophthalmology's 52nd Annual Symposium: At the Crossings - An Update on Amblyopia, Strabismus, Cataracts, Oculoplastics and Refractive Surgery, New Orleans, LA; contact Amber Howell at (504) 899-9955; fax: (504) 899-4948; e-mail: ahowell@noao.org

22. 2/14/2003 - 2/16/2003**:** Fourth International Congress of Wavefront Sensing and Aberration-Free Refractive Correction, San Francisco, CA; *contact:* COR Communications, Phone: (760) 603-1171; (760) 603-1181; Email: corcocommun@aol.com

23. 2/15/2003 - 2/16/2003**:** Sixth Annual Ocular Drug & Surgical Therapy Update Meeting, Dana Point, CA; at the St. Regis Monarch Beach. Contact: Registration Manager toll-free, 1-877-307-5225; phone: (856) 848-1000; e-mail: meetingregistration@slackinc.com.

24. 3/18/2003 - 4/1/2003**:** 24th Pan-American Congress of Ophthalmology, San Juan, Puerto Rico. *Contact:* Spectrum Negroni & Associates, Phone: (787) 708-2100; Email: mjlandrau@spectrumdms.com

25. 3/19/2003 - 3/23/2003: *Fourth Annual International Glaucoma Symposium (I.G.S.), Barcelona, Spain;* contact the Symposium Secretariat, Kenes International, 17 Rues du Cendrier, P.O. Box 1726, CH-1211, Geneva 1, Switzerland; (41) 22-908-0488; fax: (41) 22-732-2850; e-mail: glaucoma@kenes.com.

26. *3/21/2003 - 3/23/2003: International Vision Expo, New York, NY;*
contact Liz Lollis, Registration Manager, (203) 840-5954; fax: (203) 840-9954; e-mail: elollis@reedexpo.com

STRABISMUS SUMMARY SERIES

This series will be continued in the InteRyc volume 4, 02.

InformIT

By: Mr. Sameer Shah, Technical IT advisor to the JKA Institute of Strabismology

(NOTE: Mr. Shah is a teacher at the NIIT, Rajkot, one of the famous institutions that is imparting training in the subject of Information Technology (IT). He was my teacher at NIIT. We are fortunate to have his help in this series on IT. Here he describes, in short, three of the up to date techniques of identification, called "Biometric techniques".

Face Recognition

The human ability to look at a face, memorize it, and recall it when you meet the same person again is entering the domain of the computer. Called facial recognition, this technique enables the computer to use your face as your password, or identify criminals by looking at photographs of a crowd and matching these to an existing database.

The advantages of face recognition over *other biometrics techniques* are that it's non-intrusive and less expensive to set up. The subject of recognition needn't click on anything or give his fingerprint, for instance, his photograph can be captured even without his knowledge. It's less expensive in the sense that databases—of employees, national citizens, criminals, etc—may already exist. The hardware required for a small home or office setup is also not very expensive—all you need are standard video cameras with a resolution of at least 320x240 and a frame rate of at least 3–5 fps, a good video card, and a processor with enough speed. After this, all you need to buy is the software. Like other biometrics techniques, recognizing a face involves taking pictures of that face, extracting its features, creating a template from these features, and comparing this to existing templates in a database.

Retina scanning and iris scanning are two biometrics technologies that use the characteristics of human eyes for authentication

Retina scanning

Like fingerprints, the retina and iris of the human eye exhibit uniqueness for each human. The retina is an internal part of the eye, while the iris is the outer colored part. The retina is located at the back of the eye, and is a set of thin nerves, which senses the light coming

through the cornea, pupil, eye lens and vitreous humor, in that order. The pattern of blood vessels which make up the retina are unique for each individual.
The unique pattern of the blood vessels can be recorded by a retina scan device. The individual whose retina pattern has to be scanned, must have his eye located at a distance of not more than a half inch. Also the position of the eye must not move while it is scanned. While scanning the individual looks at a rotating green light. For recognizing the patterns about 400 unique points on the blood vessels are recorded. For individual authentication, the recorded pattern is compared against the blood vessel pattern of the retina. If they match, access is allowed to the individual otherwise it is denied. Since the retina is an internal portion of the eye, retina scanning is considered intrusive. Thus the individual may be hesitant to get exposed to the scanning. Moreover, retina scanning is a costly and sophisticated process.

Iris scanning

The iris has colored streaks and lines that radiate out from the pupil of the eye. The iris provides the most comprehensive biometrics data after DNA. And the chance that any two people may have the same pattern is one in 10 to-the-power-78, which is way above the current population of the Earth. In this scanning, the characteristics of the iris are taken into account. About 266 unique points (compared to 40 in finger prints) are recorded and converted into a 512 byte IrisCode (somewhat similar to barcode). For recording the iris pattern, a monochrome camera is used and the distance between the eye and the camera can be at most 3 feet. The patterns located at the inner edge of the Iris outlining the pupil, are recorded. The IrisCode constructed contains information about the characteristics and position of the unique points.
Iris scanning can be done at day or night, with glasses or contact lenses on. Since iris scanning can be done from up to 2 feet away, it is not considered intrusive.

SHORT REVIEW ARTICLE ON STRABISMUS

Nystagmus is the subject of this installment of short review article on strabismus. It is being presented in three parts. The first part appears in this volume. The references will be given at the end of the third part.

NYSTAGMUS: PART 1

Nystagmus is among the oculomotor disorders that are relatively more difficult to manage. In most cases it is congenital. Its etiology, mechanism and other factors are not yet well understood.

In this series on nystagmus we shall include the more important points relating to nystagmus, mainly congenital, as strabismologists have to deal with the congenital form. The acquired nystagmus is mainly due to neurological causes and concerns the neurologist and neuro-ophthalmologist.

The common association of nystagmus and infantile esotropia is well known and there may be reason to suspect that primary brain dysfunction may be responsible for both, the strabismus and the congenital nystagmus (latent and manifest). The primary brain dysfunction may have been due to brain injuries[1] caused during delivery of the infant. However, other authors have indicated that latent nystagmus may be secondary to strabismus[2], which in turn may have been a consequence of optokinetic asymmetry.

Definition

Nystagmus is involuntary, rhythmic and pendular/jerky to and fro (oscillatory) movement of the eyes.

Incidence

Prevalence: An incidence[3] of 1 in 6550 was reported by Hemmes in 1927.
A preponderance of males over females has been reported repeatedly in literature.[4, 5]

Heredity

Congenital nystagmus, particularly the *sensory type*, is not uncommonly hereditary. We have a family on record that has about 9 members that have oculocutaneous albinism and nystagmus in first and second cousins. Two of them have esotropia also.

The mode of transmission in some of the cases of sensory nystagmus is given in table 1 below.

Table 1

Type of sensory lesion associated with nystagmus	Mode of transmission (inheritance)
Oculocutaneous albinism	Autosomal recessive
Ocular albinism	X-Linked[38]

Terminology related to nystagmus

1) Type: A. *Pendular*: Phases (to-and-fro movements) are of equal velocity
   B. *Jerky*: Phases are of unequal velocity.

2) Direction: The side (direction) of the fast component. However, the pathological movement is the slow one.

3) Frequency: Rapid / Slow

4) Amplitude: Coarse (large) / Medium / Fine

5) Trajectory: Horizontal / Vertical / Diagonal / Rotary / Circular / Elliptical

6) Dissociated: The amplitude in the two eyes is different.

7) Null Zone: The field of gaze in which the intensity is minimal.

8) Conjugacy: (a) *Conjugate*: The nystagmus in both eyes is similar.

(b) *Disjugate*: The movements in the two eye are different, e.g., horizontal in one eye and circular in the other eyes.

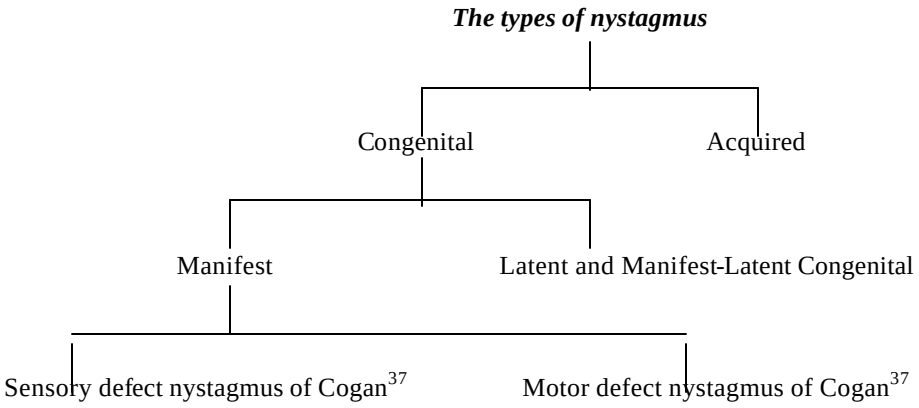
9) Neutral Zone: The field of gaze in which the bilateral jerky nystagmus reverses its direction.

Classification

There are various ways of classifying nystagmus. Different books give different classifications. The following are a few examples:

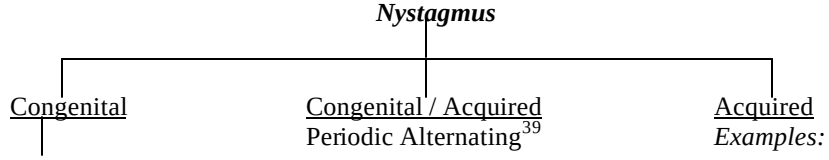
Classification example 1:

Noorden[6] has classified nystagmus as: Congenital and Acquired. The latter not being of concern to ophthalmologist / strabismologist, he has divided the congenital variety as summarized below.



Classification example 2:

Pratt-Johnson[36] mentions the following types of nystagmus in his classification:



Manifest Motor & Sensory	Latent and Latent-Manifest	*Causes*: Posterior fossa lesions Congenital Hydrocephalus Arnold Chiari Malformations Myelomeningocele	Seesaw nystagmus Downbeat nystagmus Others (Note: Such cases must be referred to neurologist)

Classification example 3:

A neurological classification is given in table 2. Although strabismologists are not concerned with other than congenital type of nystagmus, but having an idea of the huge variety of nystagmus and related movements may not be out of place here.

The traditional classification of congenital nystagmus into motor and sensory types is not recognized now by neurologists.[40] The reason for this is as follows:

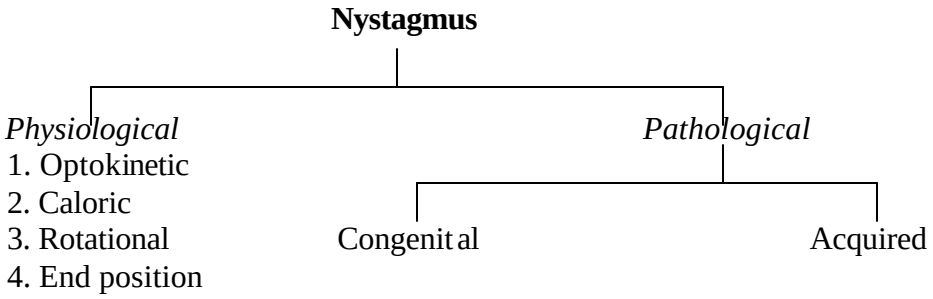
The recording of the eye movements in congenital nystagmus is the same in the case of congenital nystagmus associated with visual dysfunction and that without it. A causal relationship between the visual defect and the nystagmus can only be proved if the nystagmus has been proved to follow the visual loss. Otherwise the two of them can coexist without the loss of vision being responsible for nystagmus.

Table 2

Neurological Clinical classification of Nystagmus[40]				
Physiological, e.g., 1. End position nystagmus 2. Opto-kinetic 3. Caloric 4. Rotational	*Specific, localizing, recognizable types, e.g.,* 1) Congenital (Manifest) 2) Latent 3) Manifest-Latent 4) Spasmus Nutans 5) Dissociated & Disjugate 6) Downbeat 7) Upbeat 8) See-Saw 9) Convergence-retraction 10) Periodic alternating 11) Vestibular 12) Voluntary 13) Rebound	*Gaze evoked* • No nystagmus in PP • Down beating nystagmus on down gaze (different from Downbeat nystagmus) • Upbeating nystagmus in upgaze (different from Upbeat nystagmus) *Etiology*: 1. Drug-induced (e.g., Dilantin, sedatives etc.) 2. Posterior fossa disease	*Saccadic intrusions and oscillations* a Square wave jerks b Ocular flutter c Opsoclonus d Ocular dysmetria e Ocular myoclonus f Ocular bobbing g Reverse ocular bobbing h Ocular dipping i Reverse ocular dipping j Superior oblique myokymia (see chapter 49)	*Periodic deviations* I. Periodic alternating gaze deviation II. Periodic alternating ping-pong gaze III. Periodic alternating skew deviation

Comments: In my opinion none of the existing classifications that have come to my notice are comprehensive. But I would like to suggest a *new classification* of nystagmus as given below.

Classification example 4:



Note: *Under the congenital and acquired nystagmus we can then list the various types of each of them*.

Physiological nystagmus

1. Optokinetic nystagmus (OKN)

   When a patient looks at alternate black stripes as in the case of an optokinetic drum, the eyes show nystagmus, known as Optokinetic nystagmus. The main points are:

   - OKN is a combination of pursuit movements (SEM) and Saccade (compensatory FEM) to take up fixation of the next target on the drum.
   - The function of the OKN is to facilitate image stabilization when looking at a constantly moving field.
   - Normal OKN is symmetric.
   - Asymmetric OKN is found in some cases of deep parietal lesions with hemianopia and in infantile esotropia. In the former OKN is decreased when the stripes are moving towards the side of the lesion. In the later the OKN has a nasal bias.
   - OKN is used to test the visual acuity in an infant.
   - It can also be useful in cases of functional blindness where it can show the presence of vision if present

2. Caloric nystagmus[82]

   - It is a combination of SEM (vestibular / pursuit) and compensatory FEM (saccade).
   - SEM is produced by stimulation or inhibition of one or more of the semicircular canals.

- ✞ Depending on the position of head, unilateral irrigation leads to horizontal, circular or oblique nystagmus.
- ✞ Bilateral simultaneous irrigation leads to vertical nystagmus. With cold water the fast phase is upwards and with warm water it is downwards.

3. Rotational nystagmus

- It is a jerky nystagmus, which is produced as a result of rotation or fast movements of head.
- It is useful in estimating the oculomotor status in infants.
- The eyes deviate tonically in the direction of the movement during the nystagmus.
- The quick phase of nystagmus is towards the opposite side.

4. End position nystagmus

- ✓ Fatigue nystagmus: It occurs after rather prolonged deviation of eyes in side gaze (about 10-15 seconds).
- ✓ Sustained end position nystagmus: It comes along when eyes are in extreme side gaze. It is symmetrical and fine. It stops when eyes come back to PP.
- ✓ Unsustained end position nystagmus: Occurs initially in extreme side gaze. It stops spontaneously in 5-10 seconds.

Etiology of nystagmus

The etiology varies according to the type of nystagmus. The main types are given in the following text.

Etiology of manifest congenital nystagmus

Among the commonest causes of manifest congenital nystagmus are:

1) Oculocutaneous albinism[6, 7]
2) Congenital cataracts
3) Congenital glaucoma
4) Down's syndrome[8]
5) Aniridia
6) Achromatopsia
7) High myopia
8) Optic nerve hypoplasia[7]
9) Leber's amurosis[7]
10) Coloboma of choriod (in our experience)
11) Other conditions causing congenital defective vision (in our experience)

Etiology of Sensory defect nystagmus of Cogan

1) The basic cause in these cases is insufficient image formation on the fovea. This in turn is caused by some disorder of the anterior visual pathway that interferes with the formation of a clear image on the fovea. The feedback from the foveal region is thus inadequate /abnormal, resulting in disruption f normal oculomotor control of the fixation mechanism, leading to nystagmus.

According to Cogan the normal oculomotor stabilization requires that normal sensory input be there. This is only possible if the afferents are normal. His view was supported by the work done by Doesschate[10], which demonstrated the occurrence of pendular nystagmus after the need and thus the stimulation for fixation was removed by making the foveal image stable.

2) But the mechanism of production of nystagmus seems to depend on more than just one factor. No doubt that establishment of normal oculomotor stabilization depends on the feedback from normal sensory afferent, but there must be other factors also because the congenital sensory nystagmus does not go away in dark nor when Fresnel lenses are used (when all stimulation to fixate is removed) [11, 12].

Etiology of Motor defect nystagmus of Cogan

The cause of motor defect (congenital) nystagmus is a defective efferent system. The seat of lesion could be either in the centers or the pathways of the binocular (conjugate) ocular movements. No ocular anomalies are found. The visual acuity may be better in one of the directions of gaze because of the nystagmus being less or absent in that direction.

Etiology of latent and latent-manifest nystagmus

Various factors have been held responsible for this type of nystagmus. The better known theories / hypotheses are summarized below.

- *A difference in the quality of the two retinal images*[32]: It is considered to be the main etiological factor, in the presence or absence (as in strabismus) of binocular vision.
- *A lack of stability of oculomotor coordination*[33]: It may have been caused by a defective development of monocular and binocular fixation reflexes.[34]
- *A nasal drift bias originating centrally*: This factor is supposed to be caused to a defect of spatial localization and directionalization.[28, 35]

Symptomatology

Age of onset: Usually manifest congenital nystagmus starts between 3-4 months of age. However, it can occur at any age, later in infancy, childhood or teenage. It has even been reported to start in adult age[9].

Clinical course: Manifest congenital nystagmus is found to disappear after some time in quite a number of cases. The percentage of cases in which nystagmus disappears varies in

various clinical reports in the literature. In one report[10] it has been found to be from 30% (in cases with strabismus and neurological disorders) to 70% where no other anomaly is present. These workers found that nystagmus went away in 50% of cases by the age of 5 years. Other authors find these figures to be on the higher side[6].

Clinical characteristics

General features of nystagmus are given in short below followed by special features each of the main types.

1) *Visual acuity and Amblyopia*

Nystagmus amblyopia is one of the main types in von Noorden's classification of amblyopia (see chapter 23, page327-328. It can be easily realized how constant movements of the eyes will adversely affect the visual acuity. However, it is not clear if the nystagmus is the cause or the result of reduced visual acuity. In every case of bilateral amblyopia one should always look out for the presence of nystagmus that is not visible to naked eye. To the naked eye, in some cases, the nystagmus is not visible. However, when the patient is asked to look at the fixation object in the visuscope or the ophthalmoscope, the fine fast nystagmus can be easily seen. Micronystagmus particularly is detected in this way. The underlying causes of amblyopia in these cases can be multiple as elaborated a little later on.

The main points are given below.

- *It is extremely important* to examine both the binocular and uniocular *visual acuity* (VA) for near and for distance, with and without CHP, in cases of nystagmus. In latent and manifest-latent nystagmus the visual acuity is greatly reduced when one eye is occluded. In these cases binocular VA is significantly better than uniocular VA.

- However, in some cases uniocular VA is found to be better when one eye is occluded to examine the VA the nystagmus is seen to decrease leading to improvement in VA.[25]

- In cases of *sensory defect* nystagmus the VA is reduced according to the type and severity of the organic disease. In the *motor defect* type of nystagmus the VA is reduced due the nystagmus and is often found to be severely affected, e.g., 6/60 or even sometimes lower.[26, 2].

- But in most cases there is a combination of factors that is responsible for the reduction of VA. Apart from the constant movement due to nystagmus, refractive error, sensory defects (organic disease like congenital cataract), underdevelopment of fovea (particularly in cases of oculocutaneous albinism, strabismus amblyopia, ametropic amblyopia and visual (stimulus) deprivation amblyopia may all co-exist.

- Patients' whose null point is situated in the periphery improve their VA by adopting a compensatory (anomalous) head posture (CHP).

- If a compensatory (anomalous) head posture (CHP) is present the *visual acuity should be tested with as well as without the CHP*. If the CHP is due to nystagmus, the VA will be found to be better with CHP. Without CHP and with the test types placed in primary position the VA is found to be severely reduced.

- Visual acuity is found to be reduced in all kinds of nystagmus Even in latent nystagmus as soon as one of the eyes is occluded to test the VA the nystagmus manifests and VA is reduced. However, it varies according to the particular type of nystagmus and in the directions of gaze in which nystagmus becomes worse. The visual acuity (VA) improves when the nystagmus is reduced either by placing the eyes in the "null point" or, in the case of latent nystagmus when both eyes are open. In latent- manifest nystagmus the reduced visual acuity due to the manifest component decreases further when one eye is occluded.

- If the nystagmus is fast and jerky the decrease in VA is more marked because the *foveation time (the time during which the image of the object of attention stays on the fovea)* in these patients is quite short.

- However, the motor features of nystagmus, its frequency, its amplitude, and velocity do not always co-relate with the visual acuity. Often it is difficult to explain the degree of visual defect. This may be due to the presence of a *nystagmus amblyopia* that is obviously due to *visual deprivation* as is the case in ametropic amblyopia. The nystagmus amblyopia further complicates an already complicated clinical picture.

- In my practice I have found that some of these cases have an underdeveloped fovea. This is particularly true of patients of oculocutaneous albinism with nystagmus. Also, refractive errors are fairly common in cases of congenital idiopathic nystagmus.

- In every case of nystagmus it is important to measure the near as well as distance VA. The former is often much better than the latter. These patients often hold the print very close but may be able to read N5! Like distance VA, near VA should also be measured with each eye in turn and also binocularly.

The fact that near VA is often significantly better than distance VA, has been attributed to the effect of convergence on nystagmus. It has been suggested that convergence innervation has a dampening effect on nystagmus.[27]

- Correction of refractive error helps therefore it should always be done before starting any other therapy.

- In the patients of nystagmus with strabismus, presence of a strabismic amblyopia complicates things further. If the nystagmus does not become exacerbated with one eye occluded, occlusion therapy for it can still be carried out.
- The visual acuity can be improved by prisms (Fresnel's). This is done in one of the two ways given below: *Firstly*, by moving the null point to primary position and *secondly*, base out prisms in front of both eyes stimulate convergence as the patient has to converge to fuse the images (that have been displaced nasally by prisms) to avoid diplopia.[28] The fact that convergence reflex suppresses nystagmus is well known.

- It can also be bettered by surgery that places the eyes in the neutral zone (at null point). Details are given under the heading of surgery for nystagmus.

2) Oscillopsia

- This symptom of seeing even stationary objects moving is rarely present in cases of *congenital* nystagmus. Usually it accompanies *acquired* nystagmus.
- The reason could of absence of oscillopsia in congenital nystagmus cases may be one of the following two:
  (1) Well-developed foveation periods lead to visual stability thereby suppressing the oscillopsia.[47]
  (2) Extraretinal signals may be playing a role in preventing oscillopsia in congenial nystagmus.[48]

3) *Compensatory or anomalous head posture* (CHP)

♦ Quite often the nystagmus is not there in all the directions. For instance, it may be present in horizontal, vertical or oblique direction only, in more than one of these. For instance, nystagmus may occur in dextroversion only and absent in levoversion and so on (see case report 51-1).

Case report 1
A girl aged 6 years came to us for defective vision in both eyes. The number of corrective glasses was: OD: -3 sph.-3.5 cylinder axis 20 degrees and OS: -3 sph.-1.5 cyl. Axis 150 degrees. No significant deviation is there. She has a rather noticeable *face turn to right, slight head tilt to right and some chin depression.* The cause is a nystagmus which is *only present in Dextroversion, dextroelevation and dextrodepression*. The nystagmus is of maximum intensity in dextrodepression. There is a latent mild nystagmus in primary position, which is not always demonstrable but as the eyes turn towards right, it goes on increasing. The null point is, naturally, in levoelevation (opposite of dextrodepression) and therefore the eyes are maintained in that position by adopting the CHP.

♦ CHP is adopted to place the eyes at the null point where the nystagmus is either absent or markedly reduced. It is only in cases where null point is to one side or in elevation or depression that CHP can be of use. It is obvious that if the null point is in primary position (PP) the best visual acuity is obtained in PP. It is only when the null point is situated in the periphery that the head has to be placed so that the eyes are

looking towards the direction in which the nystagmus is absent/reduced. This is done to get the best possible visual acuity.

- If the null point is only slightly displaced to one side, the CHP is also slight and the patient needs no therapy to correct the anomalous head posture. However, if the CHP is marked because the null point is right at the periphery a marked CHP can be present that is uncomfortable and is cosmetically intolerable. If the head is straightened the visual acuity is severely reduced as the foveation (the time the image of the object of attention stays at the fovea by a steady fixation) time is drastically reduced. In such cases if the null point is moved to the center by prisms or surgery, the CHP disappears/improves leading to marked improvement of VA in primary position.

- If surgery is decided upon, it is better to try appropriate prisms prior to surgery to see if the CHP can be corrected by placing the eyes at the null point and whether the visual acuity improves.
- The type of CHP depends on the situation of the null point (the point at which the nystagmus is either absent or significantly reduced resulting in marked improvement of visual acuity (VA). For instance, if the null point is situated in levoversion, the head has to be turned to right to place the eyes in levoversion. This leads to a face turn to right. Other CHPs are as follows:
  Null point in dextroversion: Face turn to left
  Null point in deorsumversion (depression): Chin elevation
  Null point in sursumversion (elevation): Chin depression
  Null point in an oblique direction: Face-turn associated with head tilt to have the eyes looking in that direction.

    - Face turn is by far the most common type of CHP in cases of nystagmus.

4) *Strabismus*

⇒ Strabismus is quite often found to be present in cases of nystagmus, especially in motor defect type. Even sensory defect nystagmus can be associated with strabismus as is seen in cases of congenital cataract and other conditions causing defective vision.

⇒ Latent / manifest latent nystagmus is commonly associated with infantile esotropia while manifest nystagmus is not.

⇒ A thorough orthoptic examination (ocular motility workout) is important in every case of nystagmus as a small strabismus can be easily missed.

⇒ It is rather difficult to conduct an orthoptic examination in a case of nystagmus because of the constant movements but keen observation on repeated cover test is usually successful in detecting even a small strabismus.

⇒ An association of nystagmus, strabismus and oculocutaneous albinism is not an uncommon occurrence.

⇒ In my practice there have been many cases of nystagmus associated with strabismus and in some of them at least, correction of refractive error and/or surgery for strabismus reduced the nystagmus.

⇒ Unilateral amblyopia can be explained easily if a small strabismus can be detected. The latter is rather difficult as small movements on cover test with the eyes constantly moving due to nystagmus are usually always missed or can be confused with the refixation movement of the eye.

⇒ In some patients esotropia is brought on to block the nystagmus: Also called as the "*Nystagmus blockage syndrome*", it is discussed under a separate heading in this chapter.

A short description of the various types of nystagmus follows.

Sensory defect nystagmus of Cogan

- Presence of anterior ocular (visual pathway) disease, leading to indistinct/defective image formation on the fovea.
- Bilateral
- Horizontal
- Pendular (velocity of oscillations is equal in both directions)
- Becomes jerky in extreme gazes.

Motor defect nystagmus of Cogan

- No ocular disease present
- May be asymmetrical/unequal in the two directions getting better/absent in one direction leading to
- Improvement in visual acuity in that direction and adoption of:
- Compensatory (anomalous) head posture to place the eyes in the direction of the "Null point[13]/neutral zone[14]/priviledged area[15] (direction in which the nystagmus is least/absent)

Manifest congenital nystagmus

- Usually starts during early infancy(first 3-4 months of life) but onset can be at any age, even adult age.[9]

- Often associated with one of the following conditions: congenital cataract, oculocutaneous albinism, congenital glaucoma, Leber's amaurosis, aniridia, achromatopsia, high myopia Down's syndrome and optic nerve hypoplasia, the most common of these being oculocutaneous albinism.

- *Heredity*: Congenital nystagmus may be sporadic or it may follow X-linked, autosomal dominant or autosomal recessive inheritance patterns. Chromosome 6p 12 is the first reported genetic locus in cases of autosomal dominant congenital nystagmus.

- *Natural history*: In some patients the nystagmus disappears by the age of 5 years, especially if strabismus or a neurological disease does not accompany it. The incidence of disappearance of nystagmus in this group varies.[6, 19]

- *Uniocular and binocular visual acuity are the same*, as the nystagmus remains the same with uniocular and binocular fixation.

- *Main characteristic* of the congenital manifest nystagmus is a slow drift of the visual target off the fovea followed by a correctional movement in the form of a rapid saccadic movement.[20, 21]

- *Congenital* nystagmus is generally horizontal but it may be[60] vertical, rotary (circular), elliptical / oblique.

- *Congenital* nystagmus may be pendular or jerky.

- *Amplitude and frequency* of the congenital manifest nystagmus remain the same when one eye is closed or both eyes are open.[22]

- Sometimes the manifest nystagmus is associated with latent nystagmus and in such cases occlusion of one eye (as for instance, for examining VA) makes the nystagmus worse.[23]

- *Electronystagmography*: In cases of manifest congenital nystagmus nystagmograph (recording of the eye movements in nystagmus) usually shows equal frequency regardless of whether the fixation is maintained with one or both eyes. It is biphasic, usually pendular and has *an increasing velocity slow phase*. This last is the most important feature distinguishing it from latent nystagmus. No change is observed when one eye is covered.
  Difference between a decreasing and increasing velocity slow phase cannot be made out without electronystagmography.[22, 24]

- *Direction* of the nystagmus remains the same despite a change of fixation from one eye to the other.

- *Association with infantile esotropia is uncommon.*

- According to Dell'Osso and Daroff'
- Needs treatment if CHP is significant.

Latent and Manifest-latent congenital nystagmus[28]

* True latent nystagmus is only present when one eye is occluded or becomes amblyopic or blind due to some cause. *With both eyes open there is no nystagmus*.

* The manifest-latent nystagmus is there when both eyes are open but becomes worse / more intense when one eye is covered. This means that occluding one eye leads to manifestation of the latent part of the nystagmus. The manifest-latent nystagmus is also known as "manifested latent nystagmus".[29]

* The amplitude of manifest-latent type of nystagmus increases in abduction and decreases in adduction. Its fast phase is invariably towards the side of the fixating eye.

* There has been, in literature, some confusion between true latent nystagmus and manifest-latent nystagmus. This is so because sometimes the manifest element is very fine and is not visible to the eye. In such cases the correct diagnosis can only be made by *electronystagmography* (recording the nystagmus electrographically).

The main difference between the latent nystagmus and the manifest-latent nystagmus is only quantitative (that is, in degree).[31] Otherwise findings as visible to the naked eye and in electronystagmography are similar. As already mentioned earlier, the true latent nystagmus is only there when one eye is occluded. However, in some cases the nystagmus is so fine with both eyes open that it is invisible clinically and is only found on nystagmography.

* It has been generally agreed upon that the differentiation between manifest nystagmus and latent nystagmus (or manifest-latent nystagmus) is sometimes difficult or even impossible without *electronystagmography*.

* The main difference between the manifest nystagmus and latent (or manifest-latent) nystagmus is the slow phase velocity. Manifest nystagmus has an increasing velocity slow phase while latent (or manifest-latent) nystagmus has a decreasing velocity slow phase. *This is the most important differentiating feature between the two types of nystagmus.*

* Latent or manifest-latent nystagmus is usually accompanied by infantile esotropia.[30] It is not so with manifest nystagmus, which only coexists with infantile esotropia in occasional cases.[32]

Ciancia's syndrome: Association of esotropia with latent or latent -manifest nystagmus is known as Ciancia's syndrome. Also there is a head turn towards the adducting eye and bilateral limitation of abduction.[46]

Nystagmus blockage syndrome

- Definition: Nystagmus blockage syndrome (NBS) was so named by Adelstein and Cuppers.[33] It was defined by them as a condition with the following components:

  1. *Infantile esotropia* (with onset in infancy),
  2. *Frequent history of nystagmus* starting before esotropia comes on,
  3. *Pseudopalsy* of lateral recti,
  4. *Straight eyes under deep anesthesia* and a large manifest nystagmus during the induction,
  4. *Nystagmus is present* when eyes are straight and infant is *not attentive* but *absent / significantly reduced with the infant attentive and esotropic.*
  5. *Manifest nystagmus* when fixing eye is moved out of its adducted position and goes into abduction,
  6. *Anomalous head-posture* on occlusion of either eye so that adduction is maintained.

- *According to present thinking*, it includes all the ocular movements (vergences and versions) that are used to reduce nystagmus[41]. For instance, when the nystagmus is worse in dextroversion the eyes are turned to reduce nystagmus and consequently improve visual acuity. Adopting a face turn to right does this. This is an example of compensation by versions. When nystagmus is reduced by exerting convergence leading to esotropia it s called compensation by vergence.
- The only *aim* of Nystagmus blockage syndrome is to improve the visual acuity.
- *This is done by*, either turning the eyes in a direction (compensation by version), in which nystagmus is reduced / absent or by inducing vergence, e.g., convergence. The latter is typically identified as *Nystagmus blockage syndrome.*
- As it is difficult to keep the eyes turned in version and walk, a compensatory head posture is adopted with the head placed in a direction opposite to that of the eyes. Thus nystagmus is a common cause of *anomalous head posture*.
- It is important to remember that though nystagmus is often blocked by excessive convergence causing esotropia consequent upon nystagmus, the two may co-exist coincidentally. Thus every case of esotropia with nystagmus should not be diagnosed as "Nystagmus blockage syndrome".
- *Terminology*: It is also known as " Nystagmus compensation syndrome", "Nystagmus blocked syndrome" and "Nystagmus dampening".
- *Clinical features*: Nystagmus blockage syndrome is a clinical entity with certain *special features*, which are as follows (case reports 50-2 and 50-3) :

  1) There is a pre-existing congenital nystagmus.

2) An esotropia develops to block the nystagmus. Bringing in the convergence mechanism tends to dampen the nystagmus by augmenting the adduction of the fixing eye.
3) Usually the esotropia is unilateral but it could be alternating, with the infant changing the compensatory head posture (CHP) from one side to the other.
4) There is an eccentric "Null Zone" in the converged position.
5) As soon as the eye moves out of the adducted position, one can see a congenital horizontal nystagmus, with the fast phase towards the abducted eye. That means the nystagmus is jerky in waveform and the jerky phase is in the direction of the gaze.
6) The esotropia is non-accommodational and therefore the corrective glasses do not make any difference in the angle of deviation.
7) Constriction of pupil during the esotropic phase has been reported[42, 43] by some while others deny its presence.[44]
8) *Base out prisms* fail to produce abduction of the fixing eye.
9) *Surgery* may not be effective.

- *Summary*: Doubts have been raised about the existence of NBS by some authors.[44] *Present reviewed definition of NBS is as follows*:[45]

  The acute phase of NBS shows an esotropia of varying angle, in which whenever the patient is concentrating and is exerting visual attention, there is a manifest convergent deviation with no nystagmus. However, when the child is not concentrating (visual inattention) there is orthotropia with manifest nystagmus. Thus the nystagmus intensity is inversely proportional to the degree of esotropia.

  However, Dell'Osso[44] wonders if NBS is congenital (manifest) nystagmus (CN), manifest-latent nystagmus (MLN) or both? After a careful study involving quantitative oculography on one patient of NBS and two of suspected NBS, he is of the opinion that NBS has been diagnosed indiscriminately in the past. His patient with NBS demonstrated the presence of two different kinds of nystagmus. For distance there were waveforms typical of CN while for near the convergence innervation did not dampen it. However, when one of the eyes became esotropic for near, the nystagmus suddenly changed to MLN.

  The differentiation between the two types can only be diagnosed by oculography. This indicated that there are two different mechanisms causing the nystagmus this case. This conclusion has been reached on the basis of the findings in the only case with ongoing CN where oculography has been done. There was no change from CN to MLN.

Some of the cases of Nystagmus blockage syndrome (Nystagmus blockage syndrome) reported in the past may not have had NBS at all, as the diagnosis was only made clinically and congenital nystagmus (CN) and manifest-latent nystagmus (MLN) can only be differentiated on the basis of oculography and not clinically.[45]

NOTE: This article will be continued in InteRyc volume 4, 2002.

UPDATE

Note: Update contains abstracts/short outline of the articles that are of clinical interest and that have been recently published in the medical/ophthalmic literature. The abstracts given below have been taken from the Internet.

Update-General ophthalmology

1. *Potential of pigment epithelium transplantation in the treatment of AMD (By Gabriele Thumann: Graefe's Arch. Clin. Exp. Ophthalmol. 2002, 240: 695-697):* The author points out that transplantation of pigment epithelium has not been thoroughly evaluated as immunosuppressive drugs have not been tried. (Copyright 2002 S. Karger AG, Basel)

2. *Deep Lamellar Keratoplasty: (Fernando Trimarchi, Elisa Poppi, Catherine Klersy, Cesare Piacentini: Ophthalmologica 2001;215:389-393):* The authors describe a new technique of lamellar surgery and compare the results of 150 cases treated with this technique with those observed in as many cases of penetrating keratoplasty (PK). Results: No rejection episodes were recorded in the 150 patients who underwent DLKP surgery, whereas rejections occurred in 4% of the patients after PK. A statistically significant higher density of endothelial cells was found in DLKP cases, who also experienced a lower degree of astigmatism than PK patients. A slight but significant improvement in visual acuity was achieved in DLKP patients. Conclusion: Our results confirm that DLKP is the preferential procedure when no endothelial damage is involved. (Copyright 2002 S. Karger AG, Basel)

3. *Anesthesia with EMLA((R)) Cream for Botulinum A Toxin Injection into Eyelids (By Soylev MF, Kocak N, Kuvaki B, Ozkan SB, Kir E.: Ophthalmologica 2002 Sep-Oct;216(5):355-8):*
The authors investigated the efficacy of an eutectic mixture of local anesthetics (EMLA((R))) in reducing the pain of a botulinum toxin injection into eyelids. 17 patients with facial dyskinesia (9 blepharospasm and 8 hemifacial spasm) who had been treated regularly with botulinum toxin injections received EMLA cream and placebo. The pain was assessed by the ophthalmologist and the patient. Injection with EMLA cream showed lower pain scores (mean 1.82 +/- 2.13, median 1) than injection with placebo (8.76 +/- 2.17; p = 0.0001). Percutaneous anesthesia induced by EMLA cream is an effective and safe method which improves the comfort in patients who need repeated botulinum toxin injections for their facial dyskinesia. (Copyright 2002 S. Karger AG, Basel)

Update-Strabismology

1. *Consecutive exotropia following strabismus surgery (By Oguz V, Arvas S, Yolar M, Kizilkaya M, Tolun H.: Ophthalmologica 2002 Jul-Aug;216(4):246-8):* We investigated the clinical factors affecting the development of consecutive exotropia following esotropia surgery. The development period of consecutive exotropia,

amblyopia and limitation of adduction were evaluated in 89 patients with primary esotropia that changed to consecutive exotropia after surgery. In the presence of deep amblyopia, consecutive exotropia developed earlier. When two horizontal muscles were operated, limitation of adduction was more frequent in symmetrical rather than asymmetrical surgical procedure. Since consecutive exotropia may develop many years after esotropia surgery, a long-term follow-up period in patients without consecutive exotropia in the early postoperative period is advised. (Copyright 2002 S. Karger AG, Basel)

2. *Treatment of Vomiting after Paediatric Strabismus Surgery with Granisetron, Droperidol, and Metoclopramide (By Fujii Y, Tanaka H, Ito M.: Ophthalmologica 2002 Sep-Oct;216(5):359-362)* :
   The authors have compared the efficacy and safety of granisetron, droperidol, and metoclopramide in the treatment of postoperative vomiting (POV) in children scheduled for strabismus surgery. After experiencing POV during the first 3 h after recovery from anaesthesia, 120 patients received intravenously, in a randomized, double-blind manner, granisetron 40 &mgr;g/kg, droperidol 50 &mgr;g/kg, or metoclopramide 0.25 mg/kg (n = 40 in each group). The patients were then observed for 24 h after administering the study drugs. Emesis-free episodes were more often observed in patients who had received granisetron (88%) than in those who had received droperidol (63%) or metoclopramide (58%; $p < 0.05$). No clinically serious adverse events were observed in any group. In conclusion, granisetron is more effective than droperidol or metoclopramide in the treatment of POV after paediatric strabismus surgery. (Copyright 2002 S. Karger AG, Basel)

3. *Pseudotumor cerebri in a patient with Goldenhar's and Duane's syndromes (By Tillman O, Kaiser HJ, Killer HE: Ophthalmologica 2002 Jul-Aug;216(4):296-9):*
   A 4-year-old boy presented with Goldenhar's syndrome, Duane's syndrome and bilateral papilledema. Magnetic resonance imaging of the brain was unremarkable. On lumbar puncture, the cerebrospinal fluid (CSF) pressure measured 36 cm H(2)O. CSF examination was normal. The diagnosis of pseudotumor cerebri was made, and treatment with acetazolamide was started. As the papilledema did not resolve, steroids were added to the treatment. Lumbar puncture was repeated after 1 month, and pressure was found to be 30 cm H(2)O. Because medical treatment was not effective in lowering the CSF pressure, optic nerve sheath fenestration was performed. Papilledema resolved over the next 2 months. To the best of our knowledge, this is the first case of Goldenhar's syndrome associated with pseudotumor cerebri. (Copyright 2002 S. Karger AG, Basel)

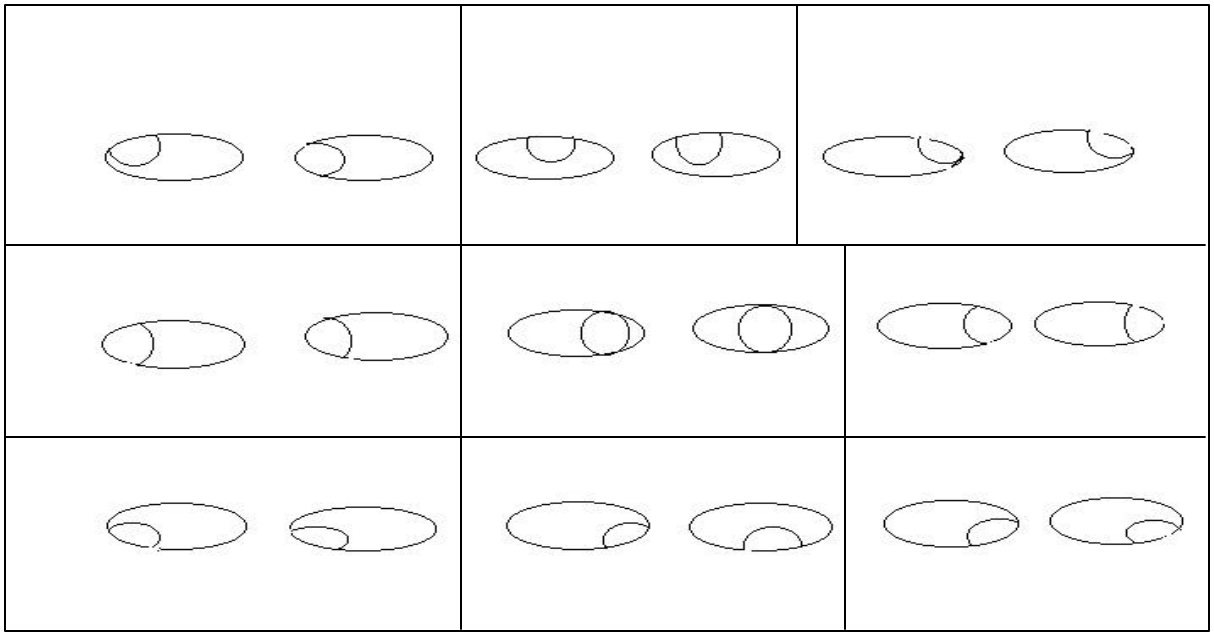
4. *Normal magnetic resonance contrast enhancement of extraocular muscles: a quantitative analysis (By Karakas HM, Tasali N, Cakir B: Ophthalmologica 2002 Mar-Apr;216(2):85-9):*
   The purpose of this study was to evaluate quantitatively the magnetic resonance contrast enhancement of normal extraocular muscles and the use of temporal muscles as a reference of enhancement. Eighty extraocular and 20 temporal muscles were taken into analysis. Before contrast administration, mean intensity of extraocular

muscles was found to be higher than that of temporal muscles ($p < 0.000$). With contrast agent, all extraocular muscles were enhanced more (111% enhancement) than the temporal muscles (45% enhancement, $p < 0.000$). Lateral recti had the lowest signal intensity, both in pre- and postcontrast images ($p < 0.005$). Normal extraocular muscles showed prominent enhancement on contrast-enhanced T(1)-weighted images. Temporal muscles were also enhanced in all subjects, urging the observers to compare the enhancement of extraocular muscles not with the latter. Copyright 2002 S. Karger AG, Basel

SPOT THE DIAGNOSIS (5)

Note: Please have a good look at the composite diagrams given below and write to us your diagnosis, your name and JIM number.

Ocular motility chart (diagrammatic representation) of a patient:



EYE-RHYME
(Dr. S.A. Patney)

"The eyes"

"The soul's windows to the world", people call them,
But they are windows to the soul too.
In Sanskrit poetry they are compared to Lotus,
And believe me some of them can bewitch you.

CARTOON-EYE

(Dr. S.A. Patney)

HISTORY-A FEW FIRSTS IN STRABISMOLOGY

Worldwide

(a) Chevalier John Taylor (1703-1772) who performed a successful operation on a boy did first surgery for squint. He was half surgeon and half quack. He must have realized that squint was a disturbance of muscular equilibrium and conceived the idea that dividing a muscle or a nerve can cure it. However, he earned a bad name through many failures, one of them being on the eyes of Bach, the famous musician.

(b) In 1743 George L. Buffon recognized amblyopia and recommended occlusion for it.

(c) In 1839 Johann F. Dieffenbach performed the first successful tenotomy.

(d) du Bois-Reymond (1952) and Mackenzie (1954) were the first to suggest orthoptic treatment but it was elaborated and established as a technique by Javal (1864-96).

(e) Prof. A. Bangerter of Switzerland and Prof. C. W. Cuppers of Germany first advocated pleoptic treatment for amblyopia. However, their approach was different.

(Continued overleaf on page 26)

CME (Member of the year) Quiz no.3, 2002:

(NOTE: Please encircle the appropriate number or letter, fill in the blanks or describe as required. Then cut along the black line and return by mail. Turn over for the update-questionnaire)

1. *Please enumerate the advantages of various "Biometric techniques" of identification*:
   (1) .
   (2) .
   (3) .
   (4) .
   (5) .

2. *Please circle the correct answer:*
   (a) Face recognition is a invasive technique: Yes / No
   (b) Manifest nystagmus is the same as Latent-Manifest Nystagmus: Yes / No
   (c) In disjugate nystagmus each eye shows different movements: Yes / No
   (d) Consecutive exotropia is more common in cases of esotropia with amblyopia: Yes / No
   (e) History of heredity is common in cases of nystagmus: Yes / No

3. What are the main features of Nystagmus Blockage Syndrome?
   (a) .
   (b) .
   (c) .
   (d) .
   (e) .

4. *Name the* main types of physiologic nystagmus*:*
   (A) .
   (B) .
   (C) .
   (D) .

5. *The clinical characteristics of nystagmus are:*
   (1) .
   (2) .
   (3) .
   (4) .

HISTORY-A FEW FIRSTS IN STRABISMOLOGY

In **India**

(Continued from previous page)

(A) Dr.H.L.Patney started running an orthoptic clinic with the help of a compounder at Sitapur Eye Hospital whom he taught orthoptic exercises, in early nineteen fifties.
(B) Dr. M.K. Mehra and Dr. Sudha Awasthi (now Patney) started the first Orthoptic clinic at K.G. Medical College, Lucknow in 1957. She ran it for 2 ½years.
(C) Dr. H.L. Patney started the first Orthoptic Department and the first Orthoptic School of India at Eye hospital, Sitapur, U.P. in 1959 and Dr. Awasthi (now Patney) Pleoptic dept. in 1961.
(D) Dr.Sudha Awasthi and Dr. J.M. Pahwa started the first Indian Orthoptic Journal in 1964.
(E) Dr. H.L.Patney and Dr. Sudha Awasthi started the All India Strabismological Society in 1967 and held India's first workshop on strabismus in 1967.

Please answer the questions or encircle the correct answers, cut along the black line and send by return mail)

Update questionnaire

1. I have been receiving InteRyc regularly, sent 2 monthly in 1998 (6 volumes) and 3 monthly (4 volumes) since 1999: Yes / No
2. My address remains unchanged: Yes / No
3. My email address: My web address:
4. My phone No.: My FAX No.:
5. My pager No.: My mobile phone No.:
6. I am enclosing herewith a demand draft for Rs100 / *cheque* for Rs118 (year 2002 subscription) / DD for Rs200 or *cheque* for Rs218 (for the years 2001+2002) / DD for Rs 300 or *cheque* for Rs318 for 2000+2001+2002.
7. I would like to resign from the membership of AISS and JKAIS: Yes / No
   If answer is yes, please write the reason if you don't mind. It may help to improve our system.
8. My membership No. is: JIM-
9. My name and present address are:

For fellowship candidates only:

10. I have paid for …….. installments.
11. I have received ……..Installments.
12. I have sent back solved question papers of …… installments.
13. I have the following problems with the course (please attach a sheet if required):
14. I have paid membership subscription for the years 98 / 99 / 00 / 01/02 / all (97-02)
15. I would like to come for the hands on experience in the month of …… 2002.
    (*Please inform at least 3-4 months in advance for arrangements to be made*)

RATE YOUR PERFORMANCE YOURSELF

The results of the "CME Quiz NO.2, 02 and those of "Spot the Diagnosis" No.2, 02 are given on this page.

CME (Member of the year) Quiz no.2, 2002:

The correct answers are given below:

Q.1. Please enumerate the advantages of WAP technology:

(1) .Don't need a PC each time you want to check mail
(2) Cheaper to purchase as compared to a laptop
(3) Ideal for getting specific information in the form of tickers or Web clippings
(4) As easy to use as a mobile phone
(5) You can store site addresses and username and password details without having to type them again and again

Q.2. Please circle the correct answer:

1) Maddox Wing is meant for measuring the deviation in Heterotropia: No
2) Removal of refractive error by surgery can help in controlling certain types of squints: Yes
3) Unstable rectus pulleys can cause incomitant strabismus: Yes
4) Surgery for Duane's retraction syndrome should wait until the patient is at least 6 years old: Yes

Q.3. What are the main features of Duane's retraction syndrome type II:

A. Compensatory head posture.
B. Exotropia in primary position.
C. Absence or marked limitation of adduction.
D. Abduction normal or slightly limited.
E. Electromyography: electrical activity in lateral rectus muscle during adduction and abduction.

Q.4 *Name the* main features of Duane's retraction syndrome type III *:*

1) CHP: absent / mild away from the affected side
2) Large Exotropia in PP, worse in adduction
3) Severe limitation of adduction and abduction, adduction being usually worse
4) Retraction of globe and narrowing of palpebral fissure in adduction
5) Electromyography: Electrical activity in medial and lateral recti during adduction and abduction

Q.5. The main procedures used in the surgery for Duane's Retraction syndrome are:

1. . Recession of one (affected) or both medial rectus muscle/muscles in type 1
2. . For large ET in type1 additional effect obtained with lateral rectus resection of opposite side
3. .Transposition of insertions of vertical recti to those of affected horizontal recti
4. . Recession of one or both lateral recti for exotropia
5. . Recession of lateral and medial rectus muscles of affected eye for marked narrowing of PF

SPOT THE DIAGNOSIS No.2, 2002

Correct answer:

Operated case of CFEOM (Congenital fibrosis of extraocular muscles) OD

Poll-Form to be filled in to decide the fate of Indian Orthoptic Journal

(The journal is not being published at present)

NOTE: Please fill in the form given below. Encircle the correct answers (yes / no)

Name of the member:

Membership No.: JIM-

Should the Indian Orthoptic Journal be restarted ?: yes / no

Should the name be changed to Indian Journal of Strabismology and Binocular Vision?: yes / no

Should the InteRyc (the Newsletter-Update) be discontinued ?: yes / no

Are you willing to be a member of the editorial board ?: yes / no

Would you like to suggest somebody else's name for the editorial board ?: yes / no

Please mention the name if the answer is yes:

Any special comments / suggestions

(Info for myself: Please see next page for info re. Sending it to Sameer Bhai)

The following message was sent to Sameer Bhai along with the file of InteRyc Vol.3, 02 as attachment on 30-1-02

Dear Sameer Bhai,

I am sorry to bother you with this but there is no other way. Please open the attachment for the InteRyc volume 3, 2002, print it and give it to Rekha or call her and tell her to collect it from you at NIIT. Do as is convinient to you.

Also please let me very soon what you need from here. I remember you saying something about a software you wanted from here.

I would like to have the following info please:

1. Should I buy a hard disc with 30 GB RAM?
2. Does my CPU have space for CD rewriter drive?
3. What is the RAM in my computer?

I trust you and your family are fine.

With regards,

S.A. Patney