UP Pedinfo

November 2015 Issue

The Official Periodical of UP State Branch of Indian Academy of Pediatrics

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Publication details of UP Pedinfo

Editor in Chief	: Dr. Shrish Bhatnagar
Owner	: Indian Academy of Pediatrics UP
Publisher	: Indian Academy of Pediatrics UP
Place of Publication	: 132/1, Maruti Puram, Faizabad Road, Lucknow
Printing Press	: **Tradmax Printers,** 132/1, Maruti Puram, Faizabad Road, Lucknow - 226 016

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Dear Colleagues,

I am very pleased to present before you the second issue of UP PED INFO in my term as Editor in Chief. I would like to tender my apologies for not being able to come out with regular supplement of the journal as promised previously. Lack of funds was the main barrier in this mission of ours.

The journal this time has not only academic articles but articles of different taste too. Dr Ajay Kalra has taken out time to pen down the "*Past Present and Future"* of UP IAP. We have contributions from Dr. Premasish Mazumdar, Agra, Dr Sarika Gupta, Lucknow and myself on different academic topics. We have interesting article from Miss Sonali Chopra A young PCS officer about her vision of paediatricians of the state of Uttar Pradesh. I hope the readers would have thought provoking and an enriched academic experience after going through the entire issue.

I would like to express my gratitude for the devotion, hard work, and professional acumen of all office executive board member and my co editors Dr. Premasish Mazumdar, Agra; Dr. Anurag Bajpai, Kanpur; Dr. D K Singh, Allahabad

Finally, I would like to acknowledge the generous support of the Johnson and Johnson for sponsoring this academic venture of ours.

Dr. Shrish Bhatnagar

Editor in Chief UP-PED INFO (Year 2015)

Ph 0-9918208555

Dated: 25/11/2015

Kindly send all your suggestion/inputs/articles at : uppedinfo@gmail.com

Message

It is a great pleasure to reach our IAP colleagues through UP Pedinfo

I would like to thank you all for unanimously electing me to this prestigious post of President Elect UP-IAP 2015. Our state Uttar Pradesh is the biggest state in the country and so are its problems, as far as Child Health Problems are concerned. The point of solace is that we have an army of senior pediatricians of national & International repute to guide us and then full brigade of young energetic talented pediatricians spread in all corners of the state willing to take up and overcome all the challenges.

The present office bearers are doing a tremendous work to place UP IAP at a respectable place in the National IAP. We need to be with them in all their ventures.

IAP Jaunpur team needs a special mention and congratulations for hosting the UP PEDICON 2015 at a very small notice. Their first brochure must have reached at all the places, it has been designed meticulously. The optimum utilization of the modern technology like face book, watts app group, emails etc by the Jaunpur team will make the things move faster and also be cost effective.

I request our fellow pediatricians to register in maximum numbers. My special appeal to our colleagues in Medical Colleges to encourage our young budding pediatricians – PG students for Paper presentation, participation in essay competition etc.

With greetings to you all for all the up-coming festivals and hoping to meet you all at Jaunpur.

Regards

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Hon Prof of Paed. Principal Govt Medical College UP

President Elect UP IAP 2015

State Academic Co-ordinator UP IAPNRPFGM

Letter from General secretary desk

Dear IAPians of Uttar Pradesh,

Warm greetings from UP IAP office.

I revisit the vision of our past president late Dr APJ Abdul Kalam that India shall grow as a superpower which leads the world through people who had an enlightened childhood .He has very nicely presented the child awareness through physical,mental and spiritual well being in his last book THE TRANSCENDANCE . The enculcation of moral and spiritual values that make a child to imbibe a crucial role in multicultural and multi linguistic interfaith society .

As physicians we must device a childhood body, mind and wellness procedure and protocols that are applicable throughout the nation and evolve time to time as per the need of society.

As Pediatricians we shoulder a major responsibility of 2030 vision of integrated India providing total Child health care and moving from "Experience to Evidence based Pediatric Practice"

I appreciate the work of Dr Shrish Bhatnagar who is working tirelessly in publishing our Official journal of Academy of Pediatrics , Uttar Pradesh and team Jaunpur who is not leaving any stone unturned in making the state PEDICON a grand success.

Wishing to meet you all at UP state conference at Jaunpur.
Happy new year and Marry Christmas to you and your family

Jai IAP
Jai Uttar Pradesh
Jai Bharat

Dr Ruchira Maheshwari Gupta
General Secretary
Academy of Pediatrics ,Uttar Pradesh

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PEDIATRICS IN UP – PAST, PRESENT AND FUTURE

Dr. Ajay Kalra
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- Nearly 60 years back, the speciality of Pediatrics in Uttar Pradesh, as in the country, was in its formative stages. Most of the practitioners in this discipline were either physicians having a special inclination for treating children diseases or were those, who, having been inspired by child care in Europe and America, had returned to this country with either a qualification in the subject or a desire to serve children. It was a time when departments and hospitals for children were being built and foundations were laid for Pediatric Education. The Unicef was deeply involved in setting up these departments in medical colleges and opening child health centers in the periphery. Fellowships enabled faculty from developed countries to come and train teachers and students in the medical colleges. Also, the faculty from here could get training abroad. Today, we have pediatric education being imparted not only in the medical colleges but in the large number of private institutions as well through the numerous CMEs' conducted by various academic bodies.
- In the past, child care and management was done in medical colleges or government hospitals in the district. They were considered best for the services with the cream of the profession being posted there. They served all strata of society. They also acted as referral centers for expert opinion. Today the private hospitals are more equipped and provide more facilities. With lots of funds, better management and a higher fee structure, they are preferred by those who can afford them. This has also attracted efficient medical professionals to opt for private medical institutions or private practice. Paucity of funds and stagnation of jobs in government institutions have affected the services in general in the government hospitals. On the other hand, the private services have excelled in many fields. Today research data is getting generated even by a practitioner in a remote area.

- Lab diagnosis in the yesteryears was just routine blood investigations and basic radiological examinations. Then came the CTs, MRIs, USGs, and they are now available in every nook and corner of the state. Often they have become the primary investigations. Development of microbiology, molecular diagnosis, radio isometric uses are the best thing that could have happened. Biopsies and drainage of effusions were blind. Now they are ultrasound or video guided. Today many investigations and surgeries are minimally invasive or based on use of endoscope. The future belongs to such innovations.
- Intravenous access was at one time through the femoral vein often resulting into gangrene. In a collapsed child, fluids were injected subcutaneously. The butterfly scalp catheters were the first respectable IV canulas for children. Then we had the intracaths. Now, it is quite common to use even central venous lines. Consequently, the management of shock has undergone a great change.
- Fifty years back it was mainly general pediatrics. Most studies were related to malnutrition, infections, growth and development. Gradually, sub specialities started developing. The state contributed to several studies on these specialities. Since neonates were a subject of curiosity because of their inability to communicate their problems, Neonotology attracted some. Today, it takes a major chunk of studies and occupation in all conferences. Today, we have so many specialities in pediatrics and there are as many chapters of these in the Indian Academy of Pediatrics. The same is true for our state except for the fact that we are moving at a snail's pace.
- Sheldon's book on Pediatrics was read by our teachers. Nelson became the most popular book in our generation. Then came Harrison and Forfar. Now we have so many from Indian authors. They are popular not only in this country but in many other countries of the world.
- Earlier, immunization was available only against small pox. Then came BCG, Polio drops, DPT, Tetanus and Measles. Now we have vaccines of which, it is difficult to keep a count. But these are mainly available in office practice. The UIP

programs in U.P. are far behind the other states. Here, we are still stuck with immunization against six vaccine preventable diseases while in the other states, newer vaccines continue to get added to the UIP.

- The state IAP is nearly 45 years old. The initial constitution of chapter was quite brief. It got a comprehensive one in 1990 which was again updated in 2007, 2008. Guidelines for holding the conferences were also laid down in the same year, but quite often, the constitution and the guidelines remain on paper only and things get done by individual discretions and this can cause indiscretions and unnecessary ill-will. In 2010, the UP Pedinfo replaced the then dormant journal "Child Today". Also in 2010 the state chapter launched its own website. These continue to be vibrant today but the future may belong to the use of Apps and such other inventions.
- The state IAP conferences used to be held in the lecture theatres of medical colleges with some 50 odd delegates being accommodated in the hostels of these colleges. Now, the number of delegates is around 200 to 500 in every conference. They are invariably held in five star hotels and are becoming great extravaganzas.

Inspite of all these developments, it is quite ironical that some of the major child health problems seem to remain the same in the state. Every second child has malnutrition. Diarrheas and respiratory illnesses still top the list of morbidity or mortality. Malaria and encephalitis epidemics continue while newer ones are always knocking at the door. NICUs are still in the developing stage and PICUs are still further behind. Most of the changes in the scenario have been because of the great technological advancements and their utilization by the medical profession. However, the bulging population, lack of basic amenities, poor hygiene/sanitation/environment and inadequate education have continued to be the hurdles in achieving the desired changes. These are things beyond the control of pediatricians. The future perspective of pediatrics in the state would depend upon what changes can be brought about on this painting on our state canvas. While money and funds are always vital, it is only honesty, good governance and compassion which will carry the day for the medical profession, otherwise a status quo or disappointment can be in store.

Upper Gastrointestinal Bleeding in children

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Upper gastrointestinal bleeding (UGIB) is a life threatening condition in children. Bleeding may occur anywhere along the gastrointestinal tract. Children who present with hematemesis constitute 10-15% of referrals to pediatric gastroenterologists (Arain and Rossi 1999).

Gastrointestinal bleeding can be roughly divided into three clinical syndromes.

a. **Upper gastrointestinal bleeding:** UGIB is from a source between the pharynx and the ligament of Trietz. This type of bleeding is characterised by hematemesis and melena.
b. **Lower gastrointestinal bleeding:** Lower gastrointestinal bleeding may be indicated by red blood *per rectum*, especially in the absence of hematemesis. Isolated melena may originate from anywhere between the stomach and the proximal colon.
c. **Bleeding from obscure sources: D**efined as bleeding of unknown origin that persists or recurs, that is recurrent or persistent iron deficiency anemia, occult blood test positivity, or visible bleeding, after a negative initial or primary endoscopy (colonoscopy and/or upper endoscopy) result(Roy and Ozden 2003) Four questions need to be answered by taking history and physical examination(Arora, Mathur et al. 2004). Therefore a complete and thorough history and physical examination is vital.

Is it actually blood? A number of substances such as food coloring agents, vegetables such as beetroot, drugs like ampicillin and phenobarbital may mimic hematochezia. Newborns who have swallowed maternal blood can present with significant melena or hematemesis while appearing stable clinically. The Apt Downey test performed on the emesis identifies the source of bleeding.(Apt and Downey 1955)

Is the child actually bleeding from the gastrointestinal tract? There are certain situations in which the source of the blood might not be the gastrointestinal tract, but can actually come from the respiratory tract, oropharyngeal region, nose and nasopharyngeal area. There might be coexisting conditions like bleeding diathesis or malignancy that may predispose

the child to mucosal bleeding.

What is the site of bleeding? Melena is indicative of a significant blood loss (over 2% of blood volume) most likely taking place distal to the ligament of Trietz. Lesions proximal to the ligament of Trietz presents usually as vomiting of bright red or coffee ground blood.

How much blood has been lost? If bleeding is slow, as much as 13% of blood can be lost without any hemodynamic change. The loss of palmer crease erythema may be seen when the hand is hyperextended as a sign of 50% or more blood volume loss(Arain and Rossi 1999).

Etiology

Newborns	Infants	One year to 12 years
Swallowed maternal blood	Gastritis	Esophageal varices
Hemorrhagic disease of the newborn	Esophagitis	Peptic ulcer disease
Gastritis	Stress ulcers	Gastritis
Vascular malformations	Mallory Weiss tears	
Idiopathic	Vascular malformation	
	Gastrointestinal duplication	

Diagnosis

Microscopic blood loss in the stool can be confirmed with a faecal occult blood test. For upper GI bleeding, a nasogastric tube is placed to confirm the presence of fresh blood and to evaluate the degree of active bleeding.

Endoscopy: Esophagogastroduodenoscopy (EGD) and colonoscopy are currently considered the first-line diagnostic procedures. The site and the cause of bleeding can be identified in 85 to 90% of the patients(Prolla, Diehl et al. 1983).

Radionuclide studies: (99m)Tc-labeled erythrocytes and (99m)Tc sulfur colloid are 2 commonly used techniques to detect active bleeding. It has a false localization rate of approximately 22%, which limits its value as a diagnostic test(Fallah, Prakash et al. 2000). The diagnostic sensitivity of the scans in a retrospective study was 39.1%(Lee, Lai et al. 2008).

Conventional angiography: It is particularly useful in the evaluation of difficult to diagnose cases of recurrent UGI bleeding (Cox and Ament 1979). An accurate angiographic diagnosis is more likely in acute GI bleeding than in chronic GI bleeding, (71% vs. 55%) (Arora, Mathur et al. 2004).

CT angiography: CT angiography is an excellent tool for fast and accurate diagnosis and localization of acute GI bleeding.

C*apsule endoscopy:* A promising new technology introduced to clinical practice in gastroenterology is capsule endoscopy. The capsule is suitable for cases of obscure bleeding from the mouth to the colon. It can successfully image small bowel pathologic features throughout the GI tract. Although this technology cannot be used for biopsy or therapy, it may prove valuable in the assessment of bleeding with negative results on gastroscopy and colonoscopy.

Management of gastrointestinal bleeding

The goals of therapy in a child with UGIB are hemodynamic resuscitation, cessation of bleeding source and prevention of future episodes of GI bleeding.

Pharmacologic management of mucosal bleeding

Therapy in these groups of patients is directed at neutralizing and/or preventing the release of acid. The various agents used include:

Antacids: In children more than five years of age, magnesium and aluminum hydroxide in doses of the 30ml/hr for the first 48 hours followed by same dose at one and three hours after meals throughout the remainder of hospitalization.

H2 receptor antagonists: are used in the treatment of gastritis, peptic ulcers and superficial mucosal erosions.

Proton pump inhibitors: Targeting the terminal step in acid production, as well as the irreversible nature of the inhibition, results in their ability to reduce gastric acid secretion by up to 99%.

Sucralfate: It is a sucrose sulfate-aluminum complex which serves as protective barrier at the ulcer surface, preventing further damage from acid, pepsin, and bile.

Endoscopic management of mucosal bleeding

A meta-analysis on the role of injection therapies for bleeding ulcers has found no difference between various techniques like thermal therapy, sclerosant therapy, clips, and thrombin/fibrin glue(Laine and McQuaid 2009).

Pharmacologic management of variceal bleeding

Start all patients on H2 receptor blocker drugs or Proton pump inhibitors. A vasoactive drug should also be started to decrease the splanchnic pressures. There is very little to choose between octreotide and somatostatin except that the latter is costlier.

Pharmacologic Therapy of Gastrointestinal Bleeding

Drug	Indication	Dosage(Boyle 2008)
Ranitidine	Control of active bleed and prevention of rebleeds	Continuous infusion, 1 mg/kg followed by infusion of 2 to 4 mg/kg per day Bolus infusions, 3 to 5 mg/kg per day divided every 8 hours
Pantoprazole	Control of active bleed	Children <40 kg: 0.5 to 1 mg/kg per day IV once daily Children >40 kg: 20 to 40 mg once daily (maximum, 40 mg/d)
Octreotide	Control of active bleed	1 mcg/kg IV bolus (maximum, 50 mcg) followed by 1 mcg/kg per hour May increase infusion rate every 8 hours to 4 mcg/kg per hour (maximum, 250 mcg per 8 hours) When bleeding is controlled, taper 50% every 12 hours. May stop when at 25% of starting dose
Somatostatin	Control of active bleed	250 µg IV bolus followed by 250 µg/hour continuous infusion Can be maintained from 2-5 days, if successful Monitor for hyperglycaemia every 6 hourly Side effects: abdominal discomfort, flushing, nausea, bradycardia, steatorrhoea, dyspepsia
Glypressin (Terlipressin)	Control of active bleed	2 mg IV every 4 hours till a bleeding free interval of 24 – 48 hours is achieved Side effects same as somatostatin
Vasopressin	Control of active bleed	0.002 to 0.005 units/kg per minute X 12 hours, then taper over 24 to 48 hours (maximum, 0.2 units/min)
Sucralfate	Coating of ulcerated mucosa	40 to 80 mg/kg per day in 4 divided doses (maximum, 1,000 mg/dose in 4 divided doses)
Propranolol	prevention of rebleeds	1 mg/kg per day in 2 to 4 divided doses May increase every 3 to 7 days to maximum of 8 mg/kg per day to achieve a 25% reduction from baseline pulse rate

Endoscopic Management

Endoscopy should be performed when the patient has been stabilized and preferably within 24 hours of admission or onset of hemorrhage(Cox and Ament 1979). The modalities available for controlling acute variceal bleeding are either variceal ligation or sclerotherapy. Sclerotherapy can control acute variceal bleeding in 70-100% of cases. However, in a meta-analysis endoscopic variceal ligation therapy significantly reduced rebleeding, mortality, frequency of esophageal strictures and the number of sessions required to achieve variceal eradication when compared with injection sclerotherapy (Laine and Cook 1995).

Surgical Management

In cases where conservative management fails with combined pharmacotherapy and endoscopic treatments, shunt and nonshunt surgeries are the definitive treatment. For intrahepatic portal hypertension, transjugular intrahepatic portosystemic shunting (TIPS) provides temporary decompression of the intrahepatic portal vein into the hepatic veins. Surgical portosystemic or portoportal shunts for GI bleeding are now reserved for refractory cases and/or when liver transplantation is not an option.

Prognostic factors associated with increased mortality(Cox and Ament 1979)

The coexistence of another severe medical disorder
Coagulation disorder
Failure to identify the bleeding site
Hemoglobin level <7 g/dL, and/or a hematocrit value of <20% at presentation
>85 ml/kg blood loss without surgical intervention

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Retinopathy of prematurity: clinical perspectives

***Shefali Mazumdar, ** Premasish Mazumdar**

Introduction: Retinopathy of prematurity (ROP) is a multifactorial retinal disorder primarily of low birth weight premature infants. It can be mild with no visual defects, or it may become aggressive with new vessel formation (neovascularisation) and progress to retinal detachment and blindness. The fundamental pathological process underlying ROP stems from incomplete vascularization at birth. Normal retinal vascularization progresses in-utero from the disc margin (16 weeks) and reaches the nasal ora serrata (by 36 weeks) and then temporally (by 39-41 weeks) to complete a mature vascular retina. Term infants have completely vascularized retina and hence are not at risk for developing ROP[1]. Premature infants have avascular or incompletely vascularized retina at birth; ROP evolves over 4-5 weeks after birth. This relatively slow evolution is however usually asymptomatic and the onus of whom to send for screening lies primarily with the neonatologist/childspecialist in order to effectively conduct retinal examinations and timely interventions to improve visual outcome and avoid irreversible blindness. The incidence of ROP in India is reported to vary between 38 – 51.9 % in low birth weight infants [2,3,4]. Out of the approximate 26 million annual live births in India, approximately 8.7% of newborns in India are < 2000 grams in weight [5]. This would imply that almost 2 million newborns are at risk for developing ROP

Risk factors (1)

Birth weight and gestational age

Infants with very low birth weight are at significantly higher risk of developing severe ROP that requires treatment. Similarly, the severity of ROP is inversely proportional to gestational age. Present evidence shows that low birth weight and gestational age are the most predictive risk factors for the development of ROP.

Oxygen Use

Oxygen therapy has been previously implicated in the aetiology of ROP. The use of supplemental oxygen neither caused progression of pre-threshold ROP nor significantly reduced the number of infants requiring peripheral ablative therapy. Recent evidence suggests that repeated hypoxic and hyperoxic episodes may be an important factor in the pathogenesis of ROP. Strict management of oxygen delivery without fluctuations and monitoring may be associated with decreased occurrence of ROP. One should also avoid SPO2 >94% in preterm babies. Although the exact relationship between oxygen therapy and ROP is currently not well established,

oxygen therapy seemed to play an important role in the pathogenesis of ROP.

Other Risk Factors

The other risk factors that have been implicated in the development of ROP include use of, glucocorticoids , surfactant, indomethacin , xanthine and dopamine. In addition, ROP has also been associated with intra-ventricular haemorrhage, ante-natal blood loss requiring blood transfusions and surgery under general anaesthesia, sepsis , candidemia , hypo/hypercarbia, raised serum bilirubin levels, and assisted conception.

However, there is insufficient evidence to determine the degree of importance of these risk factors in contributing to the pathogenesis of ROP.

There is no relation between ROP and bright light exposure, maternal smoking andmaternal PIH.

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****Consultant Neonatologist, Agra.**

Classification of ROP [11]

ROP classification is based on the location of the disease into **3 zones** (**1-3**), extent of the disease based on **clock hours** (**1-12**), **stage** (**1-5**) and the presence of plus disease.

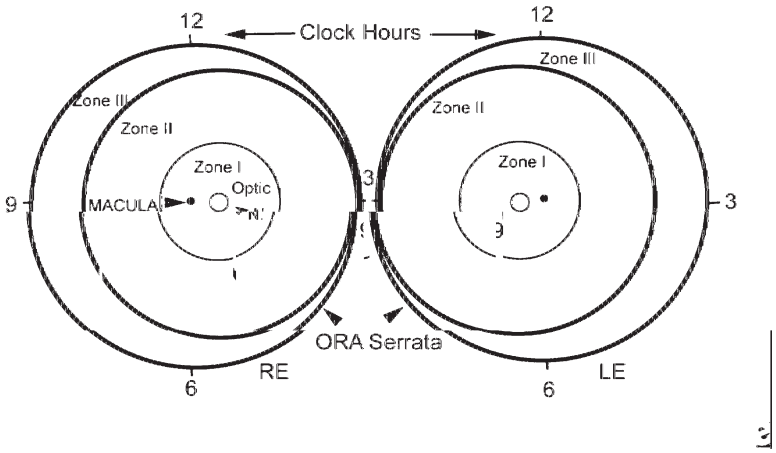
Location of ROP shown in figure 1.

a. Zone 1: innermost Zone , the radius of which is twice the distance from the centre of optic disc to macula

b. Zone 2: extends from Zone 1 to ora serrata of nasal side and about half the distance from ora serrata on temporal side.

c. Zone 3: residual crescent of retina on temporal side

Fig.1.



Extent (figure 1): it refers to the circumferential location of the disease and is reported as clock hours (1-12) in the appropriate zone.

Stage : it is divided into 5 stages

a. Stage 1: demarcation line that separates avascular retina anteriorly from the vascular retina posteriorly
b. Stage 2: ridge of scar tissue between the avascular retina and vascular retina
c. Stage 3: ridge with extraretinal fibrovascular proliferation or neovascularisation. Abnormal blood vessels extend into vitreous
d. Stage 4: partial retinal detachment due to pull of scar tissue. 4A- if detachment involves outside the fovea. 4B- if detachment involves fovea
e. Stage 5: total retinal detachment

Plus disease: it implies venous dilatation and arterial tortuosity of posterior retinal vessels, and later may incude iris engorgement, rigid pupil and vitrous haze.

Pre-Plus disease: intermediate level of vascular dilatation and tortuosity between normal appearing posterior pole vasculature and frank plus disease

AP-ROP (Aggressive posterior ROP)

A rapidly progressive and severe form of ROP. The characteristic features are its posterior location, prominence of Plus disease, its ill-defined nature and rapid progression to stage 5. It is more common in Indian babies and carries a worse prognosis as compared to classical ROP[6].

Threshold ROP

Threshold ROP is pre sent if 5 or more contiguous or 8 cumulative clock hours(30-degree sectors) of stage 3 with plus disease in either zone 1 or 2 are present. This is the level of ROP at which risk of blindness is predicted to be at least 50% and at which the CRYO-ROP study showed that risk of blindness could be reduced to approximately 25% with treatment[7]

Pre threshold ROP

Any ROP in zone less than threshold ROP, and in zone 2, stage 2 ROP with plus disease, stage 3 without plus disease, or stage 3 with plus disease but fewer than the requisite clock hours that define threshold ROP.

Type 1 prethreshold ROP includes

i. In zone 1, any ROP and plus disease or stage 3 with/without plus disease

ii. In zone 2,stage 2 or 3 ROP with plus disease

Type 2 pretreshold ROP includes

i. In zone 1, stage 1or 2 without plus disease
ii. In zone 2, stage 3 without plus disease

Screening of ROP: ***The onus for referring patients for screening lies solely with the Neonatologist / Paediatrician.*** The ideal setting for screening is under a radiant warmer in the NICU, under the guidance of the neonatologist. Discharged and stable babies may be screened in the trained ophthalmologist's clinic or in the NICU itself. The treating team should not forget to communicate with the parents regarding the risk of ROP; the need for screening preterm babies must be addressed along with the initial admission counseling itself. Documentation of such a communication is highly desirable. The baby should be swaddled and preferably fed one hour prior to examination. Pupillary dilatation should be performed about an hour prior to screening. A combination of **cyclopentolate 0.5% and phenylephrine (2.5%) drops is used two to three times about 10-15 minutes apart. Tropicamide 0.5-1% is an alternative to cyclopentolate**. The examination is carried out under topical anesthesia without any sedation, using the indirect ophthalmoscope and a 20 D or 28 D condensing lens. It must be remembered that retinal examinations are stressful and may be even painful to the infant. Swaddling the infant firmly in a thin blanket and administering 0.5-1 ml of 24% sucrose orally by syringe 1-2 minutes prior to the examination will help to provide comfort and relieve pain. Apnea and bradycardia may rarely develop during the examination in very premature babies. Resuscitation measures should be readily available. The pertinent questions regarding screening are **(1)** which neonates should be screened for ROP? **(2)** When should such screening be initiated? **(3)** How frequently should the infants be screened? **(4)**When is the screening complete?

Which infants should be screened for ROP? Screening for ROP should be performed in all preterm neonates who are < 34 weeks gestation and / or < 1750 grams birth weight. Apart from these infants, those preterm infants between 34 to 366/7 weeks gestational age or a birth weight between 1750 and 2000 grams with risk factors for ROP should also be screened[8,9,10] .Risk factors for ROP in larger infants have not been clearly established. Multi-centre studies need to be undertaken to determine the incidence, risk factors and natural course of ROP in the larger preterm infants.

When should the first screening be done? The first screen should be performed not later than 4 weeks of age or 30 days of life in infants ≥28 weeks of gestational age. They may also

be screened by the third week of life to enable diagnosis of AP-ROP[6] .Infants <28 weeks or <1200 grams birth weight should be screened early at 2-3 weeks of age, to enable early identification of AP-ROP.

How frequently should the infants be screened? Follow up examination intervals are based on the retinal findings; these findings are classified according to the revised International classification of ROP (ICROP)[11] Based on the retinal findings, the follow up examination schedule is suggested.

1 Week or less follow up

Stage 1 or 2 ROP : zone I
Stage 3 ROP : zone II

1 to 2 weeks follow up

Immature vascularisation : zone I – no ROP
Stage 2 ROP: zone II
Regressing ROP : zone I

2 weeks follow up

Stage 1 ROP: zone II
Regressing ROP : zone II

2 to 3 weeks follow up

Immature vascularization : zone II – no ROP
Stage 1 or 2 ROP: zone III
Regressing ROP: zone III

When should the screening be terminated? The following are the recommendations to guide when to stop further examinations[9].

a) Full retinal vascularization; this usually occurs at about the 40th week of postmenstrual age and mostly completes by the 45th week
b) Regression of ROP noted It is advisable to screen the baby every 1-2 weeks at least until the infant is 38-40 weeks of postmenstrual age.
c) When ROP has progressed to a stage when treatment is indicated.

Treatment of ROP when and how? Prior to December 2003, the CRYO-ROP treatment guidelines were followed. Only 'threshold disease' was treated. The Early Treatment for Retinopathy of Prematurity study (ETROP) [12] study showed that early treatment of Type 1 pretreshold ROP significantly reduced unfavorable outcomes to a clinically important degree. The guidelines from the above study are the currently recommended indications for ablative treatment and are summarized in table1. AP-ROP also needs early and aggressive laser treatment, often in multiple sessions to prevent retinal detachment .

Tab1.Treatement guidelines adopted from ET-ROP guidelines

ZONE 1	NO PLUS	Stage1	Follow
		Stage2	Follow
		Stage3	Treat
	PLUS	Stage1	Treat
		Stage2	Treat
		Stage3	Treat
ZONE 2	NO PLUS	Stage1	Follow
		Stage2	Follow
		Stage3	Follow
	PLUS	Stage1	Follow
		Stage2	Treat
		Stage3	Treat

Treatment of ROP

The aim of the treatment is to ablate the entire avascular retina up to the ora serrata in a near confluent burn pattern getting as close to the edge of the ridge as possible. Laser photocoagulation delivered by the indirect ophthalmoscopic device is the mainstay of ROP treatment. Laser has supplanted cryotherapy due to better structural and functional outcomes. The child can be fed after about 30 minutes following completion of the procedure. Vital signs must be monitored. It is preferable that the child be under the supervision of the neonatologist or an anesthesiologist for at least 2-3 hours following the procedure. Post-treatment hypothermia and hypoglycemia are common and must be prevented. Mild conjunctival chemosis and hyperemia following the procedure may last for a few days and the parents must be counseled regarding this.

Stage 4 or 5 ROP requires vitreo-retinal surgical intervention; retinal detachment carries a high risk of irreversible blindness. Visual rehabilitation must be offered to all visually challenged ROP babies.

Followup of ROP babies. . This may be typically scheduled after week 1, 2, 4 and 12 following treatment based on the findings recorded by the treating ophthalmologist. Infants with ROP, regardless of whether they have required treatment, are at risk for developing visual disorders such as strabismus, amblyopia, myopia and cataract;[13]. Retinal detachment may also occur during adulthood in infants with ROP. Moreover, prematurity may itself predispose to refractive errors, strabismus and lenticular opacities. Appropriate follow-up for these potential problems after discharge from the NICU is essential.Babies need to be under more intensive follow up for the first 6 months followed by a less intensive follow up schedule until young adulthood period to identify long term complications promptly.

Future of ROP screening: Photo-documentation and Tele-ophthalmology

The use of retinal wide field digital imaging (WFDI) using a portable pediatric fundus camera such as the RETCAM II, III and RETCAM shuttle (Clarity MSI, CA, USA) has become a useful adjunct to the documentation of ROP and as a screening and teaching tool[14] . The PHOTO-ROP study reports have shown that WFDI compares well with indirect ophthalmoscopy with a high diagnostic sensitivity[15]. In our country where trained ophthalmologists for ROP management are so few in number when the need is much more, the role of tele-ophthalmology in screening infants in peripherally situated semi-urban and rural centers by ROP experts in the tertiary care centers seems promising. This may enable timely referral of the affected infants to appropriate centers for further evaluation and treatment.

Summary:

- ROP is emerging as one of the leading causes of preventable childhood blindness in India.
- The responsibility of recognition of infants for screening lies with the pediatrician/neonatologist.
- Screening for ROP should be performed in all preterm neonates who are born < 34 weeks gestation and/or < 1750 grams birth weight; as well as in babies 34-366/7 weeks gestation or 1750- 2000 grams birth weight if they have risk factors for ROP.
- The first retinal examination should be performed not later than 4 weeks of age or 30 days of life In infants born ≥28 weeks of gestational age. Infants born < 28 weeks or < 1200 grams birth weight should be screened early, by 2-3 weeks of age, to enable early identification of AP-ROP.
- Communication with the parents regarding timely screening for ROP, seriousness of the issue, possible findings and consequences is extremely important.

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Preschool wheezing in children

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Recurrent wheeze in children under five years of age is a very common clinical problem facing practitioners throughout the world. The understanding of preschool (children under five years of age) wheezing illness has evolved in the last decade, largely by several cohort studies, which have generated evidence on pathophysiological mechanisms, existence of different wheezy phenotypes and reseach attempt to optimise management in these children.

Population studies have shown that the cumulative prevalence of wheeze is almost 50% at the age of 6 years [1].

Wheeze:

Wheeze is a sign of airway obstruction in intrathoracic airways. It is a continuous sound that has a musical quality to it. Intrathoracic airways are naturally at their narrowest in expiration, when the entire chest cavity reduces in volume. Air is expelled from the intrathoracic airways through the extrathoracic trachea and upper airway into the atmosphere. On inspiration the lungs and intrathoracic airways are pulled open by negative intrapleural pressure and draw air out of the extrathoracic trachea, which narrows. This see-sawing of air between the intrathoracic and extrathoracic airways means that any pathological obstruction of the intrathoracic airways (for instance asthma) has its greatest effect on airflow during expiration, whereas obstruction of the extrathoracic trachea (for instance viral croup) has its greatest effect during inspiration. However as obstruction becomes more severe, the other, less affected phase of respiration may become noisy as well.

Intrathoracic airway obstruction also results in 'air-trapping' when all the air that is inhaled cannot be exhaled before the airway closes. Air-trapping leads to progressive hyperexpansion of the lungs and intrathoracic airways. The increased diameter of the

airways due to hyperexpansion acts to reduce the degree of obstruction in expiration. Expiration becomes easier, but the patient now has to work harder to breathe in. This increased effort is not due to obstruction, but to the elastic recoil of the already stretched, hyperexpanded alveoli and chest wall. Hyperexpansion stops progressing when a new state of balance is reached: expiration is mostly passive and inspiration involves a huge amount of work. Most of the clinical signs of increased work of breathing (accessory muscle use, indrawing, tracheal tug) reflect this increased inspiratory effort. Patients with asthma themselves complain of difficulty getting air in, even though we observe that it is their expiratory phase that is prolonged.

The clinical effect of these phenomena is to reduce breath sounds (decreased inspiratory flow and transmission to the chest) with a mixture bilaterally of inspiratory and expiratory wheezes from different airways, and prolonged expiratory phase. The effect of hyperexpansion is most visible in the equalization of diameter of the upper and lower chest in both the lateral and frontal dimensions, so that instead of an inverted funnel shape, the chest becomes more cylindrical or barrel shaped. (This can be difficult to detect in infants who may be naturally somewhat barrel-chested.) The clavicles and upper ribs are elevated medially and so are more horizontal.

Children wheeze more often than adults because of physical differences. Infants' and young children's bronchi are small, resulting in higher peripheral airway resistance. As a result, diseases that affect the small airways have a proportionately greater impact on total airway resistance in these patients. Infants also have less elastic tissue recoil and fewer collateral airways, resulting in easier obstruction and atelectasis. The rib cage, trachea, and bronchi are also more compliant in infants and young children, and the diaphragm inserts horizontally instead of obliquely. All of these factors increase the likelihood of wheezing and respiratory distress in both of these groups

Phenotypes of wheeze in infants and young children:

When faced with a recurrently wheezing infant/preschool child, the criticl question that

needs to be ascertained at the outset is: What is the underlying cause for this wheezing?

Wheezing in preschool children has several causes and many different etiopathogenetic mechanisms are described in these disorders.

1. Typical wheezy infant/preschool child has symptoms that usually begin during the first year or two of life and may continue into childhood. Some have a clear atopic background, while others wheeze only in association with viral infection. *None of them have any significnt underlying airway or lung or systemic illness.*
2. Atypical wheezy infant/preschool child has symptoms that may begin at any age that are not ascribed to atopy or viral infections, but have an underlying airway or lung or systemic illness.

Features suggestive of Atypical wheezing in infants or preschool child include:

History:

1. Onset from birth or shortly afterwards.
2. Wheeze is prdominantly continuous. No discrete symptom free intervals periods.
3. Associated with symptoms pertaining to other systems of the body: vomiting/diarrhoea/recurrent infections/skin lesions/seizures/developmental delay.
4. Marked upper respiratory tract disease or ear disease.

Examination:

1. Failure to thrive
2. Anemia
3. Clubbing
4. Upper respiratory tract signs, stridor, ear discharge
5. Asymmetry of the chest wall or bony anomalies (pectus excavatum)
6. Unilateral or localized wheeze or educed air entry, In infants, wheezing that is audible without a stethoscope and that is not associated with respiratory distress is usually a sign of a congenital airway lesion
7. Crepitations, persistent tachypnea or cyanosis beyond acute exaccerbations
8. Neurological impairement that may cause recurrent aspirations

If there are any features suggestive of atypical wheezing, patient need to be worked up for the underlying suspected cause and accordingly treated.

Table 1: Causes of atypical wheezing, key features and diagnostic tests

Diagnosis	Key features	Diagnostic tests
Viral infection	Features of bronchiolitis-coryza, hyperinflation and basal crackles; seasonal pattern	Nasopharyngeal aspirate for immunoflorescence, PCR or viral culture
Gastro-oesophageal reflux	Vomiting or poor weight gain; Associated with feeding, cough, and vomiting	pH study, contrast swallow, bronchoscopy for lipid laden macrophages
Inhaled foreign body	Prior episode of coughing or choking (not always present), chronic cough	Chest radiograph, bronchoscopy
Immunedeficiency	Wheeze with infections which are Severe, Persistent, Unusual or Recurrent (SPUR)	Immunoglobulin profile, CD counts
Cystic fibrosis	Recurrent cough, poor weight gain, clubbing	Sweat test, mutation analysis
Bronchomalacia Tracheomalacia, anomalies of the great vessels	Harsh monophonic expiratory wheeze Wheezing associated with positional changes	Bronchoscopy Angiography, bronchoscopy, ECHO, CT/MRI
Aspiration syndrome	Neurological impairement	
Cardiac anomaly especially those causing left to right shunt	Tchycardia, crackls	Chest X-ray, ECG, ECHO
Primary ciliary dyskinesia	Rhinorrhoea in the first week of life	Chest radiograph to look for dextrocardia (in 50%), ciliary studies
Bronchopulmonary dysplasia or chronic lung disease of prematurity	Premature birth, home oxygen	

Wheezy phenotypes amonh typical wheezers:

Tucson classification:

In the Tucson Children's Respiratory Study (TCRS), four different wheezing phenotypes were identified among 1246 newborns followed for lower respiratory tract infections based upon the presence of wheezing symptoms during the first three years of life and again at six years [1]. The epidemiologic phenotypes generated from this prospective longitudinal study included:

- **Never wheezers** – Healthy children who never wheezed
- **Early, transient wheezers** – Children with wheezing that began before three years of age and resolved by six years of age. Transint early wheezers tend to wheeze with lower respiratory tract infections and often have no family history of asthma or allergen sensitization.

Interestigly, those with transient early wheezing are noted to have reduced lung function that remains low at age 6 years although wheezing has ceased, when compared with children who have never wheezed. They are born with airways that are smaller than average, possibly as a direct influence of nicotine (maternal smoking)

- **Nonatopic persistent wheezers** – Children with wheezing that began before three years of age and was still present at six years of age. They typiclly wheeze with viral infections and their lung functions are slightly lower than in control subjects from birth to 11 years of age, but they do not have allergic sensitization and methacholine hyperresponsiveness. These findings are thought to be secondary to alterations in regualation of airway motor tone.
- **Immunoglobulin E (IgE)-associated/atopic persistent wheezers** – Children who developed wheezing between three and six years of age. Can begin in infancy, b u t increases in prevalence with age; associated with personal and family history of atopy, methacholine hyperresponsiveness, and poor growth of lung function. This phenotype may represent a classic allergic asthma phenotype, but it is unknown if children with this phenotype will have symptoms that persist into adulthood.

This classification system although has conrtributed significantly to our undrstanding of the pathophysiology, risk factors and long term outcome of the wheezing infants or preschool children. However the greatest drawback of this classification is its reterospective nature and cannot be of much help in giving either point of care or prognostiction to that individual child who reports to a physician with recurrent wheezing in preschool days. Also there could be children fitting into intermediate patterns of wheezing ilness.

To overcome this limitation, The European Respiratory Society defined two symptom-based phenotypes [2] for recurrent wheezing children <5 years, to guide clinicl management.

1. **Episodic (viral) wheeze** – Wheezing during discrete time periods, with absence of wheeze between episodes; usually associated with viral respiratory tract infections

2. **Multi-trigger wheeze** – Wheezing during discrete exacerbations and have intermittent symptoms between episodes. Symptoms include nocturnal wheze or cough and triggers include cold air, viruses, allergens, exercise, laughing or crying and cigarette smoke.

Airway functionw was lower in multiple-trigger than episodic wheeze, suggesting that these are functionally different phenotypes [3]. However, in one study, over half of children classified into these two phenotypes based upon their wheezing history in the previous year switched to the other phenotype in the ensuing year [4], suggesting that these phenotypes are not stable over time.

Another study compared epidemiologic phenotype definitions identified by latent class analysis (persistent, late onset, intermediate, transient, and none/infrequent) with clinical phenotypes based upon patient histories from one to six years of age in an international multicenter birth cohort [5]. Four clinically-defined wheeze phenotypes (recurrent unremitting wheeze, unremitting wheeze, asthma diagnosis, and frequent wheeze) were found to have a high sensitivity and specificity. These findings suggest that these clinical phenotypes may be useful asthma definitions for future epidemiological studies. The clinical phenotypes were characterized as follows:

1. Asthma diagnosis – Clinician diagnosis of asthma ever or recurrent diagnoses of spastic, obstructive, or asthmatic bronchitis by age six
2. Multi-trigger wheeze – At least two common asthma triggers leading to wheeze between ages three to six
3. Unremitting wheeze – Having symptoms between wheezing episodes or wheeze without a cold at least once between ages one to six
4. Recurrent unremitting wheeze – Having symptoms between wheezing episodes or wheeze without a cold reported for two or more years between ages one to six
5. Frequent wheeze – Wheeze on a monthly basis for at least one year between ages one to six
6. Episodic wheeze – Wheezing episodes associated with viral respiratory tract infections only between ages one to six

As there are several early-childhood wheezing phenotypes described, accurate classification of young children at high risk to develop persistent asthma may help predict long-term outcomes and identify children who may benefit from secondary prevention interventions.

Various predictive models or clinical indicators of risk have been studied to help the clinician identify those children who will continue wheezing into older childhood. These models have employed various risk factors associated with the development of asthma in epidemiologic studies, such as parental history of allergic sensitization and asthma, wheezing history, atopic disease in the child, immunoglobulin E (IgE) levels, and cytokine secretion profiles. The most commonly used predictive scoring system is the Asthma Predictive Index (API) [6].

Asthma Predictive Index:

The *major criteria* were clinician-diagnosed eczema or parental asthma. The *minor criteria* were clinician-diagnosed allergic rhinitis, wheezing apart from colds, and eosinophilia $\geq$4 percent.

A *positive loose index* was defined as *any* parental report of wheezing on the surveys at two or three years of age and either one major criteria or two minor criteria. A *positive stringent index* was defined as frequent wheezing on these same surveys plus the same combination of major or minor criteria.

Children with a positive loose index were four times more likely to have active asthma during a subsequent survey at 6, 8, 11, or 13 years of age (sensitivity 42 percent, specificity 85 percent). Children with a positive stringent index were seven times more likely to have active asthma in at least one of these school-aged surveys (sensitivity 16 percent, specificity 97 percent).

A modified version of the API, which replaces provider diagnosis of allergic rhinitis with allergic skin testing, has been endorsed by the US National Asthma Education and Prevention Program Expert Panel Report 3 for use in the diagnosis of asthma.

Management:

Treatment of acute wheezing episodes:

Short acting β-2 agonists:

Inhaled rapidly acting β-2 agonists are the most effective bronchodilators available, and therefore the drug of choice for acute symptoms of wheeze. Infants possess functional β-2 receptors from birth, therefore stimulation of these receptors can produce the sam effects as in older children. Oral administration of β-2 agonists is also effective but is limited by systemic side-effects.

Inhaled anticholinergic agents:

According to a Cochrane review, the combination of ipratropium bromide and β-2 agonists as compred to β-2 agonists alone was associated with a reduced need for additional tretment but no difference was seen in tratment response, respiratory rate, oxygen saturation improvement in the emergency department and length of hospital stay.

Inhaled corticosteroids:

Based on the current evidence, it appears that intermittent high dose inhaled corticosteroids are effective in children with frequent episodes of moderately severe episodic wheeze or multiple trigger wheeze, but this is associated with short term effects on growth and cannot be recommended as a routine [7].

Maintenance treatment:

Episodic viral wheeze:

Randomized controlled trials have shown that inhaled corticosteroids in preschool children with episodic viral wheeze children do not alter the natural history of the disease nor reduce the risk of later asthma and the symptoms return when steroid therapy is discontinued[8, 9].

The recommendation for episodic viral wheeze is:

1. Montelukast 4 mg once daily should probably be given for the treatment of episodic viral wheeze.
2. Trial of inhaled corticosteroids may be considered in preschool children with episodic viral wheeze, in particular when episodes occur frequently or if the family history of asthma is positive [10].

Multi-trigger wheeze:

The recommendation for multi trigger wheeze is [10]:

1. A trial of 3 months of inhaled corticosteroids (daily dose of 400ug Beclomethasone equivalent) may be tried.
2. During the trial of treatment parents should document symptoms and response daily. If however there is no improvement after a few weeks, the treatment should not be stepped up but stopped and further investigations should be carried out in order to identify the cause of symptoms.
3. If preschool children with multi trigger wheeze respond well to inhaled corticosteroids therapy, it is unclear whether this is due to treatment or the natural resolution of symptoms. It is recommended therefore, that treatment be withdrawn in children who become almost completely free of wheeze after inhaled corticosteroids thearpy.

If symptome recur after withdrawl and respond to reintroduction of therapy, it further supports the beneficial response in muti trigger wheezers.

Short term systemic steroid therapy should be reserved for excaerbation of wheezy symptoms where hospitalization is necessary.

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A Message for Builders of Nation Builders of Tomorrow…..

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My 10 months experience as a District Savings Officer has made me gain insights into the lives of multitude of people belonging to different strata of society. One good old adage that I always share with them is that one should always save for a rainy day, as savings, though not entirely, are very much linked to the uncertainties of life and are our best friends in the hour of need. However, one thing which I noticed all through my small experience was that, for majority of people, that hour of need is a health emergency. Therefore, though, theoretically we believe that people's savings are utilized for education and marriage of their children, more often than not, they end up losing all of it when confronted with a health issue in the family. This is primarily due to two reasons. One is the increasing burden of diseases in our country and the second is widespread poverty which renders healthcare a luxury for majority of Indians. Numerous studies have shown that healthcare expenses account for a major portion of an individual's out of pocket expenses.

Without going into the financial solutions to the problem at hand, let us focus on the root of the problem. Why so many diseases in the first place? One of the youngest nations in the world (by virtue of average age of populace) is also a lead nation in terms of malnutrition and diseases of its young ones. This certainly is an unfortunate situation and does not bode well for a country with global aspirations. Children are the most valuable resource for a country. We proudly claim to reap demographic dividend in the years to come but given the health statistics of our children, we should better brace ourselves up for a demographic disaster.

To my limited understanding, just as a building with a strong foundation can withstand tremors of a high intensity earthquake, so can a person face and fight any health complication in adulthood, if his formative years were adequately attended to by a doctor. This analogy may sound bizarre to many pediatricians reading this article, but for an outsider to medical community of doctors like me, it sounds logical. Why not strengthen the base then? Notwithstanding the sorry state of education in our country, the health aspect of the foundation has to be managed by the doctors. Children are the future of our nation.

That is precisely the reason I titled this article as a 'message for builders of nation builders of tomorrow…'

About 0.76 million neonates die every year in India. Whether it is infant mortality ratio or maternal mortality ratio or any other medical statistical jargon, we are lagging behind. We all know where the problem lies. In spite of plethora of state and centrally sponsored high decibel programmes for a healthy India like NRHM which in the twelfth plan has been revised into NHM, the results are far from satisfactory. One does not ignore the good work done by the government or the medical fraternity, but when we talk of health, it is better to be a cynic than an optimist. A healthy body is indispensable for a meaningful life. Even the Supreme Court in its various judgements has done a liberal interpretation of right to life as enshrined in Article 21 of the Indian Constitution as something more than mere survival. Right to good health and Right to doctor's assistance is very much a part of the fundamental right to life.

This is not to say that our Doctors are in anyway doing less than what they ought to. In fact they are the ones who are constantly under scrutiny for honouring the Hippocratic Oath and doing "selfless service". Probably the government takes note of the enormity of the problem and blesses us with healthy health budgets. Official data tells us that health expenditure by the Centre and States, both Plan and Non Plan stood at 0.94% of GDP in the Tenth Plan and 1.04% in the Eleventh Plan. One only hopes it increases to at least 2.5% by the end of Twelfth Five Year Plan period. With increased resources, the public and private health sector should come together to achieve the dream of Universal Health Care because a MAKE IN INDIA…SKILL INDIA…DIGITAL INDIA…IS ONLY POSSIBLE when we have A HEALTHY INDIA.

My role as a small savings officer brought me face to face with the real aam aadmi and get a firsthand experience of their struggle for as basic a right as good health. I realized that though we proudly talk about Medical Tourism and super speciality corporate hospitals next to none in the world, what about many fellow Indians standing in serpentine queues of OPDs in various hospitals? The idea is not to sound didactic or preachy. It is only to kindle the goodness which is inherent in each one of us, especially Doctors who are God personified on earth for their patients. I salute all the Doctors for being there for us. We need you and we need you more every day.

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Dr. A K Kaushik, Meerut
Dr. Atul Srivastava, Bareilly
Dr. J K Gupta, Kanpur
Dr. M Irshad, Moradabad
Dr. Om Chaurasiya, Jhansi
Dr. Raghavender Dwivedi, Agra
Dr. Sanjay Niranjan, Lucknow
Dr. Shirish Bhatnagar, Lucknow

Ex Officio
Dr. Vivek Saxena, Kanpur

TIME LINE for UP IAP ELECTIONS

Last date of receiving Nomination paper by Chief Election Officer (CEO): 19th December, 2015, 5.00 pm

Last date of withdrawal of nomination paper : 25th December, 2015

Last date of posting ballot paper by CEO : 30th December, 2015

Last date of recovering request for duplicate ballot paper : 7th January, 2016

Last date of posting duplicate ballot paper : 12th January, 2016

Last date of receiving all ballot paper by CEO : 20th January, 2016

Counting and declaration of results : 22nd January, 2016

Kindly send your nomination for the post of President elect. 2017. Joint Secretary and Executive Board Member 2016 to Chief Election Officer Dr. Ajay Kalra (Add.: 58, Khandari Road, Agra-282002, Ph No.-0562-3249822, Mail-ID - kanwal.kalra@yahoo.in)

The nomination from and all other details are available at UP IAP official website : www.upiap.org.

In case of any query kindly contact General Secretary, UP IAP Dr. Ruchira Gupta.

Office: B-190, Sector-19, Noida - 201301 (U. P.)

INDIAN ACADEMY OF PEDIATRICS – U.P. STATE BRANCH

MEMBERSHIP FORM

Personal Information

First Name _____ Middle Name _____Last Name _____

Complete Mailing Address:_____

City: _____ Pin Code: _____ STD Code: _____

Phone Residence: _____ Phone Clinic: _____

Mobile No.: _____Email ID: _____

Are you a member of Central IAP: Yes/No: _____ Central IAP Number: _____

Qualifications:

Qualification	Year of Completion	Institute
MBBS		
DCH		
MD		
DM		
Other (specify)		

Present Attachment: ☐ Teaching(1), PMHS(2), Corporate Hospital(3), Private Practice(4), Other(5)

Remarks (if any)_____

Date: **Signature**

Place:

Attach a DD for Rs. 2000/- in favour of"Indian Academy of Pediatrics UP State Chapter" payable at Agra, U.P.

Please mail the completed form to:

DR. RUCHIRA M. GUPTA
Sr. Consultant Dept. of Pediatrics
Kailash Hospital & Heart Institute
H-33, Sector – 27, Noida – 201301
E-mail: ruchiramaheshwar@gmail.com
Mobile: 9910316347

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extra burden

Ease the burden and treat symptoms of cough with only the required ingredients[1]

In dry cough & sore throat, recommend a targeted approach for a symptomatic relief.

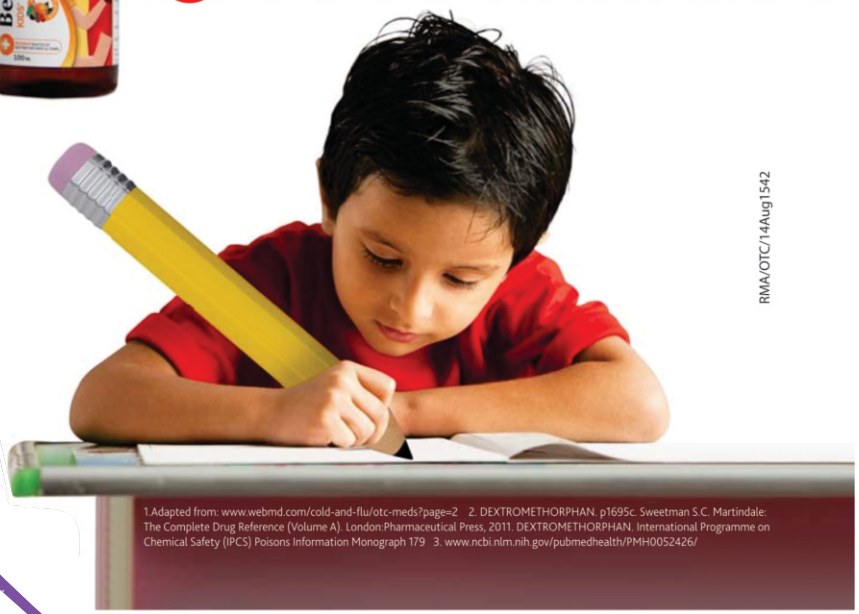
Benadryl® DR KIDS'

For targeted approach

Each 5 ml contains Dextromethorphan Hydrobromide I.P. 7.5 mg

- Acts within 30 minutes[2]
- Reduces severity & frequency of cough[3]

- ALCOHOL FREE
- DELICIOUS MIXED FRUIT FLAVOUR
- NON-DROWSY FORMULA



1.Adapted from: www.webmd.com/cold-and-flu/otc-meds?page=2 2. DEXTROMETHORPHAN. p1695c. Sweetman S.C. Martindale: The Complete Drug Reference (Volume A). London:Pharmaceutical Press, 2011. DEXTROMETHORPHAN. International Programme on Chemical Safety (IPCS) Poisons Information Monograph 179 3. www.ncbi.nlm.nih.gov/pubmedhealth/PMH0052426/

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2 Mild

3 Effective

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care

JOHNSON'S® baby products in India carry the international seal of JOHNSON'S® commitment.

SAFE

We only use ingredients that are proven to be appropriate for babies and have passed through rigorous safety checks

MILD

Our formulas are rigorously developed and tested to avoid irritation of baby skin

EFFECTIVE

Our products are designed to respect the skin barrier, and the delicate scalp, to support good hair condition and healthy skin development

RMA/JB/10Sept2015344