

Journey of Indian Academic of Pediatrics, Delhi

1949: The leading pediatricians of Delhi met at the office of Dr K L Jain on Thursday afternoon. These pediatricians mostly were Members of the Association of Physicians of India. In that first meeting of pediatrician, the idea of forming an Association of pediatricians of India was mooted. It is interesting to observe that the present practice of our Thursday Meetings seems to have its roots in that first meeting held on a Thursday.

1955: In 1954, the Association of pediatricians of India was christened and it established its Central office at Bombay. The Delhi State Branch came into being in 1955.

1956-1963: during these eight years, Dr K L Jain, Dr P N Taneja, Dr SS Paul, Dr K B Kapur and Dr A K Basu by rotation held the offices of the president and the Secretary of the Delhi Branch. Dr R P Monga remained the Treasurer for many years.

The first Department of Pediatrics was created at the Irwin (Now LNJP Hospital) Hospital and Dr P N Taneja joined as Head of Department in 1952. The Kalawati Saran Children's Hospital came up in 1956 and Dr Sheila Singh Paul was its Head of The department. The next pediatric Department was established at the Safdarjung Hospital in 1957-59 with Dr A K Basu as its Head. The pediatrics Department in AIIMS was started in 1958. Meetings in late fifties were mostly held in these hospitals.

1964: The Indian Academy of Pediatrics was formed in 1964 by the amalgamation of the Association of Pediatricians of India (Bombay) and the Indian Society of Pediatrics (Calcutta). The Delhi Branch of the Academy was opened in 1956-66. The Branch instituted following awards and Orations for its members:

Awards:

1. Dr P N Taneja Award (Started in 1971)
2. Dr Satya Gupta Award (Started in 1975)
3. Dr Sarla Vaishnav Award (Started in 1980)

Orations:

1. Dr. P.N.Taneja Oration (Started on 13th Oct. 1996)
2. Dr. S.C. Khandpur oration (started since Dec. 1990)

Conferences organized by Delhi IAP:

The Delhi IAP has contributed to growth and development of scientific acumen in the country by organizing the several scientific programs for Pediatricians practicing across the Nation. To mention here few of them are as under-

Name of the Conference	Year	Organizing Secretary
		International
8 th Asian Congress of Pediatrics	1994	Dr RK Puri
		National
XIV National Conference of IAP	1977	Dr. O P Ghai
XXIII National Conference of IAP	1986	Dr. S C Arya
XXXI National Conference of IAP	1994	Dr. R K Puri
XXXXIII National Conference of IAP	2006	Dr. A K Dutta
Conf on Adolescent Health	2000	Dr. Sangita Yadav
		Regional/National
Conf. on Resp. Diseases	1993	Dr. Krishan Chugh
-	1994	Dr. Harish Chellani
P.C.N.I.1995	1995	Dr. Shyam Kukreja
-	1996	Dr. Rajesh Mehta
-	1997	Dr. Jagdish Chandra
Ped. Conf. of North India	1998	Dr. A.J.Chitkara

Conf. on Infectious diseases	1999	Dr. Sisir Paul
Conference on Ped. AIDS	2001	Dr. Ajay Gambhir
Environment health	2002	Dr. Anupam Sachdeva
Newer Drugs	2003	Dr.L.S.Arya
Fluid Electrolyte & Arterial Blood Gas Analysis in Ped. Practice.	2004	Dr. Harish K. Pemde

AWARDS :

The Delhi IAP Judged best state branch during 2000-01 by National IAP.

Contribution to CIAP:

The Delhi IAP has played crucial role for growth of Indian Academy of Pediatric to bring up it in present stature since its inception in 1964. The following Members of Delhi IAP served CIAP as President and nurtured it to make it a dynamic organization of the Pediatricians of the Nation.

Name	Year
Dr P N Taneja	1965
Dr Shanti Ghosh	1976
Dr O P Ghai	1978
Dr Satya Gupta	1980
Dr S K Bhargava	1985
Dr Subhash Arya	1991
Dr Meharban Singh	1993
Dr R N Shrivastava	1996
Dr HPS Sachdev	2003

Office Infrastructure:

The IAP Delhi State Branch had no physical office. It usually ran from the Secretary or President's residence/Office. In 1998 a room was provided by then Head of Pediatrics Dr R K Puri in the Department of Pediatrics that was also got vacated by Hospital authorities on pretext of space crunch in the Department. In 1996-97 the executive committee felt that IAP Delhi must have its own office premises to run its activities. Dr Vijay Agarwal then President took initiative and started his effort toward purchase of office space for Academy. Under Delhi Societies Act, a registered society or Association can purchase land or building if it is registered in under Delhi Society Act. Therefore, the Delhi Branch is registered as "Indian Academy of Pediatrics Delhi" with Registration No. S-29229 of 1996. The present office is purchased on 4th June 1996 bearing Municipal No. 4222-A, located at Kala Kunj, No.1 Ansari Road Dariyaganj, New Delhi. Later this office premises renovated and furnished with modern office amenities and computerized.

Delhi City Branches:

Since Delhi has grown rapidly in its length and width, it felt that IAP Delhi also must decentralized its activities and so five City Branches were established. These Branches started functioning from June 2004.

Land for office Building:

The IAP Delhi is striving hard for allotment of the land for construction of its own office building. A proposal to this effect is pending with Ministry of Urban Development, Government of India.

The list of the Presidents and the Secretaries of the Delhi Branch from 1961 is given below:

Year	President	Secretary
1961	Dr P N Taneja	
1962	Dr A K Basu	Dr K Kanwar
1963	Dr K Kanwar	
1964	Dr O P Ghai	Dr KPS Verma
1965	Dr G P Vermam	Dr KPS Verma
1966	Dr Sheila Singh Paul	
1967	Dr S Vaishnava	Dr K E Karunakaran
1968	Dr Satya Gupta	Dr G Srivatava
1969	Dr Seeta Sinclair	Dr K Saxena
1970	Dr S Ghosh	Dr S C Arya
1971	Dr S C Arya	
1972	Dr N D Datta Banik	Dr U Saxena
1973	Dr S K Bhargava	Dr U Saxena
1974	Dr H Sehgal	Dr P N Singh
1975	Dr M Singh	Dr P N Singh
1976	Dr V V Gujral	Dr D K Guha
1977	Dr Lata Saini	Dr D K Guha
1978	Dr Anu Gupta	Dr C K Bhalla
1979	Dr U Saxena	Dr I Naryanan
1980	Dr G Srivastava	Dr K Chopra
1981	Dr R N Srivastava	Dr K Chopra
1982	Dr P N Singh	Dr K Chopra
1983	Dr S C Khandpur	Dr S K Mittal
1984	Dr S C Arya	Dr A K Dutta
1985	Dr N K Anand	Dr S Dawar
1986	Dr Daya Sharma	Dr Vijay Agarwal
1987	Dr Man Mohan	Dr T S Jain
1988	Dr D K Guha	Dr Panna Choudhury
1989	Dr S K Mittal	Dr Subhash Agarwal
1990	Dr K Chopra	Dr Arvind Taneja
1991	Dr I C Verma	Dr A K Gulati
1992	Dr R K Puri	Dr Manju Sharma
1993	Dr Sudarshan Kumari	Dr Krishan Chugh
1994	Dr S Thirupuram	Dr Harish Chellani
1995	Dr A K Dutta	Dr Shyam Kukreja
1996-97	Dr Vijay Agarwal	Dr Rajesh Mehta
1997-98	Dr R N Salhan	Dr Jagdish Chandra
1998-99	Dr Anita Khalil	Dr A J Chitara
1999-2000	Dr Arvind Taneja	Dr Sisir Paul
2000-01	Dr T S Jain	Dr Sangeeta Yadav
2001-02	Dr Panna Choudhury	Dr Ajay Gambhir
2002-03	Dr Subhash Agarwal	Dr Anupam Sachdeva
2003-04	Dr L S Arya	Dr C M Khanijo
2004	Dr K Chugh	Dr Harish Pemde
2005	Dr A P Dubey	Dr J P Kapoor
2006	Dr M M A Faridi	Dr K C Tamaria

Dr. K.C. Tamaria, Editor Secretary Elect.

INTERPRETATION OF LIVER FUNCTION TESTS

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A broad array of biochemical tests has traditionally been grouped together as "liver function tests". These tests offer a sensitive and noninvasive method of screening for liver dysfunction. Specific patterns of derangement help ascertain the general type of liver disease, and some tests allow prediction of the severity of disease, while others are used to monitor the course of the disease and the response to treatment. However, liver function tests (LFTs) are not infallible. LFTs lack sensitivity, being often normal or only mildly deranged in chronic liver disease and cirrhosis. LFTs often lack specificity, with aminotransferase elevation seen often with cardiac or skeletal muscle disorders, and hypoalbuminemia being as common with malnutrition and malabsorption as with liver disease.

Optimal utilization of liver function tests therefore requires their interpretation in the light of the clinical context, with an eye to the utility and limitation of each test.

Liver function tests can be grouped in the following categories:

1. Tests that reflect hepatobiliary injury:
 - ❖ Aminotransferases (AST, ALT)
 - ❖ Lactate dehydrogenase
 - ❖ Glutathione S transferase
2. Tests that indicate obstruction to bile flow:
 - ❖ Alkaline phosphatase
 - ❖ Glutamyl transferase,
 - ❖ 5' nucleotidase.
3. Tests that measure the liver's capacity to transport organic anions or to metabolise drugs:
 - ❖ Bilirubin
 - ❖ Serum bile acids
 - ❖ Urine bilirubin and urobilinogen
4. Indicators of synthetic function:
 - ❖ Serum albumin
 - ❖ Prothrombin time
 - ❖ Ammonia
 - ❖ Cholesterol
 - ❖ Urea
5. Indicators of chronic hepatic inflammation, altered immunoregulation, or viral hepatitis:
 - ❖ Immunoglobulins
 - ❖ Specific autoantibodies
 - ❖ Hepatitis serology.

1. Markers of liver cell injury:

Aminotransferases:

Alanine aminotransferase or ALT (serum glutamic pyruvic transaminase, SGPT) and Aspartate aminotransferase or AST (serum glutamic acetoacetic transaminase, SGOT) are sensitive indicators of hepatocellular injury. AST is found in the mitochondria and cytosol, in hepatic as well as extra-hepatic tissues like cardiac muscle, skeletal muscle, brain, pancreas, kidneys, lung, WBCs, and RBCs. The normal levels of the enzymes in serum are low, less than 30 to 40 IU/L. Important points in using ALT and AST to interpret liver disease are as follows:

- ❖ ALT is relatively liver specific compared to AST. A disproportionate or isolated elevation of the AST indicates the possibility of cardiac or skeletal muscle injury, like myositis, muscular dystrophy, and after vigorous exercise etc. Hemolysis may cause a small increase in the AST levels.

- ❖ In most disorders of acute liver disease, AST / ALT ratio is less than or equal to 1. Acute Wilson's disease is also characterized by a disproportionate elevation of AST relative to ALT; AST /ALT ratio of more than 4 in the appropriate clinical setting is highly suggestive of Wilson's disease. The ratio may be more than 2 in cases of Reye's syndrome. A ratio of more than 1 also may suggest cirrhosis.

- ❖ Mild elevations of the transaminases (up to 500 IU/L or 2-3 fold) are seen in a wide variety of disorders affecting the liver, including fatty liver, biliary obstruction, alcoholic liver disease etc. Moderate elevations (3 to 20 fold) are seen in most kinds of acute hepatitis including viral hepatitis, chronic hepatitis, infectious mononucleosis, acute cardiac failure, and drug induced liver injury etc. Extreme elevations (more than 20 fold or above 2000 IU/L) are seen in acute viral hepatitis, drug or toxin induced hepatic necrosis, and ischemic hepatitis.

- ❖ Infective causes of elevated transaminases- Hepatitis A-E , Epstein Barr virus(EBV) ,Cytomegalo virus(CMV) , Herpes virus, Toxoplasmosis , Typhoid fever, Malaria , Leptospirosis , Liver abscess

- ❖ Elevated aminotranferases are among the first laboratory abnormalities noted in viral hepatitis, bilirubin elevation lagging behind by about a week. At the time that bilirubin increases, transferases are usually declining. Secondary increases or their

persistent elevation may indicate either recrudescence of disease or development of chronic hepatitis.

- ❖ The absolute values of transferases do not correlate with severity of disease.

- ❖ Decreasing serum values of aminotransferases coupled with a rising serum bilirubin, in the context of fulminant hepatitis, constitute a poor prognostic sign, reflecting the massive destruction of hepatocytes.

- ❖ Levels of aminotransferases are often normal or nearly normal in liver disease without ongoing hepatocyte injury, as in advanced cirrhosis, chronic hepatitis, hemochromatosis, or methotrexate use.

- ❖ Hepatitis C infection is associated with fluctuating levels of ALT and AST, this is called as the "yoyo" phenomenon.

- ❖ Hypoaminotransferasemia, or low levels of these enzymes, is seen in association with azotemia, where levels higher than 20 IU/L may be considered as abnormal and prompt search for occult viral hepatitis, especially hepatitis C which is common in renal disease especially dialysis patients.

- ❖ Elevation of the enzymes is seen with use of drug like erythromycin and drug toxicity due to aspirin, amoxicillin-clavulanic acid, ATT and Antiepileptic drugs particularly valproate

- ❖ In a recently published study (Iorio et al J Gastroenterol 2005; 40:820) of 425 consecutive children with hypertransaminasemia during 6 months of observation, 259 showed normalized liver enzymes. Out of 166 in whom hypertransaminasemia lasted more than 6 months - 75 had obesity related disorders, 51 genetic disorders, 7 autoimmune diseases, 5-cholelithiasis, 3-celiac disease was established.

- ❖ Therefore the interpretation of the values of these liver enzymes must be done appropriately keeping in mind the clinical suspicion of disease.

Lactate dehydrogenase (LDH):

Elevation of LDH is seen with several disorders other than liver disease, like with muscle injury, cardiac disease, hemolysis, stroke, and renal infarction.. An ALT to LDH ratio of more than 1.5 is suggestive of acute viral hepatitis as against ischemic hepatitis. Overall, LDH is not used very often in the diagnosis of liver disease.

Glutathione-S-transferase (GST):

Serum GST-B is a very sensitive indicator of acute hepatocellular injury, as with acute viral hepatitis, and drug induced injury, where the elevations are 5 to 10 times higher than of aminotransferases. Due to its short half-life, cessation of active liver injury is easily recognized by a fall in the serum enzyme level.

2. Markers of biliary obstruction:

Alkaline phosphatase:

- ❖ Alkaline phosphatases are zinc metalloenzymes. The usual sources are bone, liver, intestine and placenta. The enzyme is used as a marker of cholestasis, where its elevation is the result of increased hepatic production with leakage into the serum, rather than failure to clear or excrete the circulating ALP. However, the enzyme must be evaluated in the context of liver disease with the following considerations:

- ❖ Markedly elevated levels of ALP are seen in cholestasis associated with extra-hepatic biliary obstruction, primary biliary cirrhosis, primary sclerosing cholangitis, and drug induced cholestasis.

- ❖ Mild to moderate elevation of ALP is seen with infiltrative disorders associated with granulomatous disorders as tuberculosis and sarcoidosis, amyloidosis, and malignancy. Mild elevations also occur with a variety of liver diseases like viral hepatitis, congestive heart failure, and cirrhosis. Hepatitis A, Hepatitis D, and Epstein-Barr virus may cause disproportionate elevation of ALP.

- ❖ Serum ALP may be elevated up to threefold in normal children and adolescents, due to active bone growth.

- ❖ The hepatic origin of the ALP should be confirmed by concomitant measurement of GGT or 5' nucleotidase.

- ❖ The level of ALP elevation cannot be used to reliably distinguish between intrahepatic and extrahepatic obstruction.

- ❖ Decreased levels of ALP are seen with, hypothyroidism, pernicious anemia, zinc deficiency, and with fulminant Wilson's disease associated with hemolysis. Evaluation of an isolated or predominant elevation of alkaline phosphates should therefore include confirming the hepatic origin of the enzyme, followed by hepatic imaging to look for biliary duct dilatation or biliary disease, and infiltrative disorders.

Glutamyl transferase (GGT):

- ❖ The estimation of glutamyl transferase has 90 % sensitivity and specificity for the purpose of confirming a hepatic origin of an elevated ALP, because though this microsomal enzyme is found in several extrahepatic tissues, it is not produced by bone. There is a good correlation between this enzyme and ALP and 5' nucleotidase in various liver diseases. The normal levels of GGT are higher in infants than in adults. The normal range in children above 4 years of age and in adults is 0 to 30 IU/L.

GGT is of value in cholestatic diseases presenting in infancy, wherein a normal GGT in the face of an elevated ALP indicates either Byler's syndrome (PFIC type 1) or benign recurrent

intrahepatic cholestasis (BRIC).

GGT may be elevated due to its induction by a variety of drugs including phenytoin, barbiturates and alcohol, while the levels of other enzymes and bilirubin may be normal. Other causes of GGT elevation include GBS, porphyria, and hyperthyroidism.

5' Nucleotidase:

Despite its widespread distribution, significant elevations of this enzyme are seen only with diseases involving the liver. 5' NT is as sensitive as is ALP in the detection of biliary obstruction, infiltration and cholestasis but its levels rises several days after the obstruction and therefore 5' NT levels may still be normal while ALP has risen.

3. Indicators of the liver's capacity to transport organic anions or to metabolize drugs:

Bilirubin:

Normally, the total bilirubin is less than 1 mg/dl and is predominantly unconjugated. Important interpretations pertaining to bilirubin include the following:

Isolated unconjugated hyperbilirubinemia in the presence of normal other liver function tests indicates hemolysis (hemolytic anemia, drug-induced, transfusion reaction, or autoimmune hemolysis), ineffective erythropoiesis (megaloblastic anemia, or thalassemia), or defective bilirubin uptake or conjugation (neonatal physiological immaturity, Gilbert's syndrome, or Crigler-Najar syndrome).

Isolated conjugated hyperbilirubinemia in the presence of normal other liver function tests indicates Dubin- Johnson syndrome, Rotor syndrome.

Elevation of conjugated and unconjugated bilirubin in association with abnormal other liver function tests indicates hepatocellular injury (hepatitis, cirrhosis, or drug induced injury) or obstruction to bile flow (neonatal cholestasis, sclerosing cholangitis, gallstones, stricture, or biliary atresia).

The magnitude of conjugated hyperbilirubinemia does not reliably distinguish between parenchymal liver disease and biliary obstruction.

In fulminant hepatic failure, a rising serum bilirubin when present in conjugation with falling aminotranferases, is indicative of a poor outcome.

A serum bilirubin in excess of 30 mg/dl should suggest renal failure or hemolysis in additional to liver disease.

Serum bile acids:

These are organic acids derived from cholesterol in the liver. Though they sensitively indicate liver disease, they are nonspecific in

differentiating the type of liver disease..

Urine bilirubin and urobilinogen:

Bilirubinuria establishes the presence of hepatic dysfunction, and is very sensitive - it is present even before jaundice appears, while total bilirubin is either normal or is only mildly elevated. The absence of bilirubinuria in the presence of jaundice suggests unconjugated hyperbilirubinemia as with hemolysis.

4. Markers of hepatic synthetic function:

Serum albumin:

Albumin is quantitatively the most important circulating protein synthesized by the liver, and its concentration in the plasma falls in severe acute and chronic liver disease. It is one of the criteria used to stage the severity of liver disease in the Child-Turcotte Pugh classification. However a low serum albumin is not specific to liver disease and may be seen with malabsorption, malnutrition, nephrotic syndrome, with hormonal changes and with increased catabolism. Also, due to the long half-life of albumin (21 days), the measure is poorly sensitive in acute liver disease where PT should be used. Another sensitive measure in the setting of acute liver disease is prealbumin, which has a shorter half-life (1.9 days).

Prothrombin time:

Except for factor VIII, all the clotting factors are synthesized in the liver, and the factors that determine prothrombin time (PT) II, V, VII, and X - are sufficiently short-lived to make PT an important tool in evaluating hepatic synthetic function in the setting of acute liver disease. Therefore PT is used in various scoring systems to grade the severity of fulminant hepatic failure; a prolongation of two or more seconds beyond is considered abnormal. Malabsorption, which may be associated with chronic cholestasis, can be distinguished from liver dysfunction as the cause of the prolonged PT, by measurement of factor V (normal in malabsorption, and decreased in liver disease) or by the administration of vitamin K parenterally (reduces PT prolongation by at least 30% within 24 hours if secondary to malabsorption).

Lipoproteins:

Serum cholesterol tends to be elevated in most forms of cholestasis, and low in parenchymal liver disease. Either cholestatic liver disease or parenchymal dysfunction may be associated with abnormal lipid profile on electrophoresis, with loss of HDL and VLDL, and prominence of the beta band. An abnormal lipoprotein, LP-X has been found to be associated with liver disease, though this is not specific to hepatic dysfunction.

5. Indicators of chronic hepatic inflammation, altered

immunoregulation, or viral hepatitis:

Immunoglobulins:

The elevated levels of immunoglobulins seen in many chronic liver diseases are probably the result of impaired reticuloendothelial function within the sinusoids, or vascular shunting preventing the normal degradation of antigens absorbed from the enteral lumen eliciting a humoral response. Moderate elevations suggest chronic active hepatitis or cirrhosis, while extreme elevations suggest autoimmune liver disease. The level helps monitor response to treatment in autoimmune liver disease.

Procollagen peptide:

This is one of the markers of liver fibrosis that can be detected in the serum. Others include collagens VI and XIV. The intention behind using them is the hope that they may replace the need to do repeated liver biopsies to monitor the course of disease. These tests are still in their experimental stages.

Approach to Characteristic biochemical patterns

Use of a few tests, including the total and direct bilirubin, aminotransferases, alkaline phosphatase, albumin, globulin, prothrombin time, and urine for bilirubin, in the form of a battery, increases their sensitivity and specificity. A few examples are listed below:

1. Bilirubin elevation of upto 5 mg/dl, with > 85 % being unconjugated, in the presence of normal aminotransferases, alkaline phosphatase, albumin, globulin and PT, without any bilirubinuria: suggests either hemolysis or Gilbert's syndrome. Further test for confirming hemolysis are needed for definitive diagnosis.
2. Bilirubin elevation(both fractions elevated), with aminotransferase elevation preceding bilirubin elevation (usually exceeding 500 IU/L), with ALP elevation (upto three times normal) , a normal albumin and globulin, normal or prolonged PT and bilirubinuria: suggests **acute hepatocellular necrosis**, this may be due to viral hepatitis, drug toxicity, congestive heart failure or autoimmune insult. PT prolongation indicates a poor prognosis.
3. Bilirubin elevation (both fractions elevated), aminotransferase elevation usually within 300 IU/L, ALP elevation upto three times normal, often with decreased albumin and increased -globulin, presence of bilirubinuria, with prolonged PT that does not get corrected with parenteral vitamin K: suggests either **chronic hepatitis or cirrhosis**. An AST/ALT more than 2 suggests cirrhosis.
4. Bilirubin elevation (both fractions elevated), with aminotransferase elevation usually mild to moderate and not exceeding 500 IU/L, ALP elevated often over four times normal, with bilirubinuria, normal albumin, normal -globulin and sometimes elevated -globulin, with normal or prolonged PT that corrects with parenteral vitamin K: suggests **obstructive jaundice or intrahepatic cholestasis**
5. Normal or near-normal bilirubin, with normal to slightly elevated aminotransferases, elevated ALP often over four times normal, with normal albumin and globulin, with normal PT: suggests partial bile duct obstruction or infiltrative disease like granuloma or tumor, if the hepatic origin of the ALP has been confirmed by using either 5'NT or GGT. -globulin may be elevated in cases of granulomatous diseases.

The above examples amply demonstrate the ability of these LFTs to suggest the diagnosis; once this information is obtained, specific tests like hepatitis serologies, ultrasound or CT, and liver biopsy, may be employed to arrive at the actual diagnosis.

Thus, the liver function tests can be employed in conjunction with a good history and physical examination to arrive at a judicious estimate of the type and extent of liver injury. Currently available tests are limited by problems regarding their sensitivity and specificity. Possibly in the future more tests, including better quantitative assays will find widespread application overcoming the shortcomings of the currently available tests.

Five things That one Should Know About Pediatric Leukemia

1. TYPES OF LEUKEMIA

Leukemia (blood cancer) is the most common cancer in children. There are two main subtypes in leukemia : i.) acute lymphoblastic leukemia (ALL) and ii) acute myeloid leukemia (AML). There is also a small percentage of case of chronic myeloid leukemia (CML). Acute lymphoblastic leukemia is the commonest single type of childhood cancer and accounts for almost 80% of cases of leukemia.

2. WHAT CAUSES PEDIATRIC LEUKEMIA?

In general, parents can be reassured that they could not have prevented the leukemia. In the majority of childhood cancers it is not possible to identify any specific reason which could be considered to be the cause of cancer. Irradiation received by pregnant women during the early stages of pregnancy slightly increases the risk of their offspring developing leukemia. Although very rare, other predisposing or contributing causes include environmental and genetic factors, viruses, chemical carcinogens and immunodeficiency states. Leukemia is not an infectious disease.

3. DIAGNOSIS OF LEUKEMIA

A child suffering from leukemia may present with anemia, fever, fatigue, burising and bleeding from various sites and bone pain. Examination may reveal pallor, fever, petechiae, bone tenderness and enlargement of lymph nodes, liver and spleen. Leukemia can

usually be diagnosed by presence of blast cells in the peripheral blood. However the diagnosis is confirmed by examination of the bone marrow.

4. PEDIATRIC LEUKEMIA IS CURABLE

Because of the major advances in diagnosis, development of rational use of combination chemotherapy and improved supportive care, the cure rate in childhood leukemia has increased tremendously and at present over 70% of children with leukemia are cured.

5. WHERE SHOULD LEUKEMIA BE TREATED?

These children should be treated by a specialist in pediatric oncology in a cancer centre or hospital with all the necessary pediatric supportive care facilities including pediatric nursing care, good diagnostic and laboratory support. Survival rates of children with leukemia or any cancer are significantly enhanced through access to state-of-the-art treatment given according to well defined protocols in specialised centres.

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The Lactation Consult: Problem Solving, Teaching, and Support for the Breast feeding Family

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"It's all in the latch." Lactation consultants preach about a good latch at the breast. Many pediatricians working with new mothers know it is important. But how can you confirm that a mother has achieved a good latch when breast feeding her newborn? Very simply, the latch is correct when the infant is receiving colostrums or milk and the mother is free from pain. The helping professional must focus on both of these questions simultaneously when working the nursing couple.

The gold standard for proof of milk transfer from the breast to the infant is audible swallowing. In the first few days of life, infants often suck six or seven times before swallowing the thick colostrums. By the time the infant is 3 to 5 days old, a mother's milk has "come in," and many swallows may be heard following each letdown. A mother will have several letdowns with each feeding as oxytocin stimulates the letdown of breast milk every 6 to 10 minutes during a feeding. This is evidenced by burst of swallowing, pauses, and more bursts of swallowing. It is important to remember that infants may appear high on areola and have visible jaw movements but not be transferring milk from the mother's breast. The presence of a drop of milk on the end of the nipple when infant releases the breast also may fool the practitioner. Only when you hear repetitive swallowing deep in infant's throat can you be reasonably certain that the infant is receiving milk. You may need to place your ear close to the infant's head to ascertain quiet swallowing. Nutritive nursing is confirmed by a steady weight gain of approximately 1 oz per day in the newborn period. Conversely, increasing weight loss confirms that a problem does indeed exist.

The second key to a good latch at the breast can be determined by asking the mother one question: Is the breast feeding painful? If a mother denies pain, appears comfortable, and is willing to relax and allow the infant to determine the length of the feeding, latch is correct from the mother's perspective. Often in the first few days of nursing there is nipple tenderness at the initial moment of latch on to the breast. When the latch is correct, pain should begin to subside within a few sucks and essentially disappear within 1 to 2 minutes. Many mothers seek lactation help after discharge from the hospital with cracked, bleeding or scabbed nipples. They may report that "Many nurses saw me breast feed and they all said the baby was latched on right!" The presence of damaged nipples proves this was not correct. A common mistake occurs when the helping professional focuses on the appearance of the infant at the breast and milk transfer but fails to confirm that the breast feeding is comfortable. Painful nursing causes nipple damage that may not be visible for two to three days. Hospital staff may never know that their breast feeding assessments were incomplete or leading to future problems. So, if mother says it hurts, the latch is not correct even if the infant is swallowing milk. Only the mother can confirm a good latch from the standpoint of her personal comfort.

Who Needs Lactation Consult After Discharge from the Hospital?

Breast feeding is a codependent relationship. The infant's ability to Breast feeding affects the mother's milk supply. Maternal problems affect the infant's ability to receive adequate milk for growth. All concerns must be addressed promptly to avoid a rapid downward spiral toward premature weaning. The primary pediatrician can identify breast feeding infants with potential problems by reviewing the hospital history. Increased weight loss, hypoglycemia, infrequent nursing, or maternal reports of poor feeding are predictive of early problems. The normal newborn often is sleepy during the hospitalization period but should begin to wake for more frequent feedings soon after discharge. When real or potential problems are suspected, a lactation consult is required. This assessment of the infant, mother, and breast feeding couple working together is the essence of the lactation.

When one or more of following circumstances are present, lactation consult is warranted.

- ❖ Inability of the infant to latch on to the breast within 8 to 12 hours after birth

- ❖ Absence of audible swallowing during breast feeding (in the first 3 to 4 days of life, several sucks may be noted before colostrums is swallowed; when the mother's milk comes "in," a swallow should follow many sucks)

- ❖ Infants output of fewer than 6 wet diapers and fewer than three 3 stools in 24 hours (output may diminish briefly around the third day before the onset of milk production)

- ❖ Newborn weight loss near or exceeding 10% of birth weight during the first week of life

- ❖ Maternal pain with breast feeding and/or visible nipple damage.

- ❖ Maternal history of flat or inverted nipples, endocrine and/or fertility problems, breast surgery, or the absence of breast changes during pregnancy.

- ❖ Presence of a medical problem likely to affect normal breast feeding, for example ankyloglossia (tongue tie), neurological impairment, Down syndrome, cleft lip or palate, or other congenital anomalies.

- ❖ Begin by creating a comfortable Environment for the Mother it is helpful to remember that a woman may have little exposure to family members or friends who breast fed their infants. She may view her breast primarily in a sexual context and so feel uncomfortable receiving help with breast feeding. She needs time to adjust to her new role of breast feeding mother. New mothers are also tired and overwhelmed. An unwillingness to accept lactation help may actually reflect a need to hide feelings of inadequacy.

- ❖ It is challenging for the busy pediatrician to allow adequate time for the mother to move comfortably through the lactation assessment. Obviously, the relaxed mother is better able to focus and absorb the information provided. When the father has joined the mother, include him in your conversation. Suggest that he observe and listen so that he can help the mother at home. Strive to solidify

each parent's role as caretaker of the new baby.

Proceed Step by Step through a Lactation Consultation

Just as each provider finds a comfortable and efficient approach to perform a well child assessment, practice helps the Pediatrician proceed smoothly through a lactation consultation. It may be easier for a new mother to begin the evaluation by focusing on her infant. Listen to the mother's opinion about how baby feeds. Take your time and examine the infant carefully. Finally, help the mother to breast feeding, observing for clues that might direct you to the cause of any problems.

The following steps suggest a logical strategy for the health pediatrician to obtain information and problem solve during the lactation assessment:

- ❖ Obtain the infant's weight. Compare the current weight to birth weight, and if possible, discharge weight. Continuing weight loss suggests a problem for most infants 5 days of age or older unless the infant demonstrates frequent and sustained swallowing during the consult.

- ❖ Obtain the mother's feeding history. Listen carefully to what the mother believes to be the problem(s).

- ❖ Examine the infant, focusing on alertness, hydration, neurologic tone, the presence of jaundice, or the existence of congenital anomalies.

- ❖ Put on a glove and examine the infant's tongue, palate, and lingual frenulum. Assess the infant's ability to suck, noting the position of the tongue and any rigidity or poor muscle tone.

- ❖ Query the mother about any breast changes during her pregnancy and since the delivery of her baby. Ask about the existence of any thyroid or fertility problems. If she has other children, inquire if any breast feeding difficulties occurred with these infants.

- ❖ Note the current condition of the mother's breast. Check for the presence or absence of colostrums or milk, signs of nipple damage, and asymmetric or v-shaped breast that might indicate glandular insufficiency.

- ❖ Using a comfortable armchair or a breast feeding pillow, help the mother position the baby for nursing. Evaluate the latch; listen for audible swallowing and carefully observe the mother's comfort level.

- ❖ Allow time for the mother to demonstrate that she can implement your suggestions without your participation.

- ❖ Provide written instructions to reinforce your teaching and serve as a reference for the mother when she returns home. Follow up is essential, plan a subsequent office visit.

Helping the Mother and Newborn Achieve a good Latch

- ❖ Undoubtedly there are merits in many techniques and drawbacks to some for individual mothers. The recommendations below are meant to provide a simple yet tried and true approach that is successful for many mothers. Remember, if the baby is swallowing milk and the mother is breast feeding comfortably, you have accomplished your goal.



Figure1 : The Football Hold.

- ❖ The football hold and modified cradle hold often are easiest for the struggling mother or baby. Below are description of these holds. To avoid confusion, the feminine gender will refer to the mother; the male gender will refer to the infant. You may need to practice with a doll and memorize the basic steps to each technique before attempting to help a mother.

THE FOOTBALL HOLD

The football hold is often best for the mother and infant experiencing difficulty achieving an effective latch on. This position allows the mother to support both her infant's head and her breast simultaneously. It is easier for her to visualize the latch on process. The sleepy or disinterested infant may be more alert in this position. The football hold is often more comfortable for mothers who had a cesarean section.

Assist the mother with the football hold as follows:

1. Provide a comfortable armchair with pillows or a nursing pillow. Tuck the infant into a "V" position at his mother's side with his legs parallel to her back.

2. The infant's head should rest in the palm of his mother's hand. The infant's head rests in his mother's right hand when nursing at the right breast; his head rests in her left hand when feeding at the left breast. The mother's forearm supports her infant's neck and back.

3. Have mother support her breast with her free hand?

4. Use multiple pillows if necessary to support the mother's hand and arm. The mother may remove the hand supporting her breast as long as she remains comfortable.

MODIFIED CRADLE HOLD

The modified cradle hold position works well when the mother prefers the cradle hold but has difficulty controlling the infant's head or supporting her breast for latch on. Women with larger breast as well as those with shorter arms may feel more comfortable with a modified cradle hold.

1. Position the infant on his side, stomach with his mother. The baby should be resting against the mother's upper abdomen, not lying low near her lap. Use a nursing pillow or multiple smaller pillows for comfort and support.

2. If nursing at her right breast, have the mother support her infant by placing his head in the palm of her left hand. Her left forearm should support the baby's back and maintain the stomach to stomach position.

3. When nursing on her right breast, have the mother use her right hand to support her breast. Have her support her left breast with her left hand when feeding on the left breast.

4. Once the infant has latched on, the mother may remove the hand supporting her breast if she remains

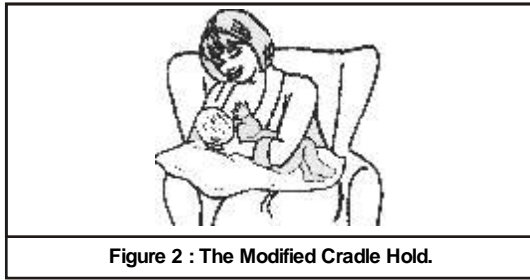


Figure 2 : The Modified Cradle Hold.

comfortable.

THE MOMENT OF LATCH ON

No matter how “perfect” the mother's positioning for breast feeding, the infant primarily is responsible for a good latch and effective nursing. The mother can be a critical facilitator by making the nipple and areola easily available to the infant, but in the end only the baby can latch and breast feeding.

You may help the mother to facilitate a good latch by following these suggestions:

1. Have the mother support her breast using a “C” hold. Be certain that her thumb and fingers are positioned well behind her areola.

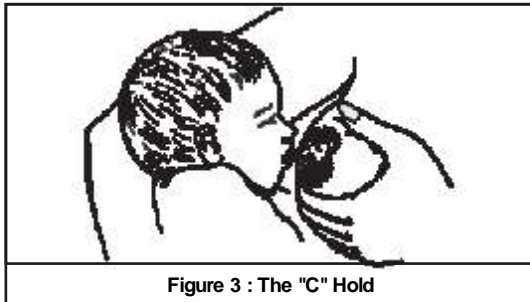


Figure 3 : The "C" Hold

2. The mother can lightly touch the tip of her nipple to the center of her infant's mouth. Have her squeeze a drop of milk onto the nipple tip if possible. It is best to avoid over stimulating the infant's mouth and to “invite” the infant to latch on.

3. Wait until the infant opens yawn wide. His tongue should be down on the floor of his mouth. Note his lingual frenulum and see if the infant can extend the tip of his tongue past the lower lip. The tongue must be able to curl around the bottom of the areola in order to suck effectively and avoid pain for the mother.

4. Point the nipple tip straight toward the back of the infant's throat. Mothers often aim their nipple up so they can SEE the infant latch on around the nipple. Injured nipple tips occur when the roof of the infant's mouth bangs on the upturned nipple during feeding. Red, abraded, or scabbed nipple tips may suggest this problem.

5. Confirm that infant's lips are rolled out so that a thin lip line is visible around entire areola at latch on. Sucking is less effective as well as painful when either lip is rolled inward.

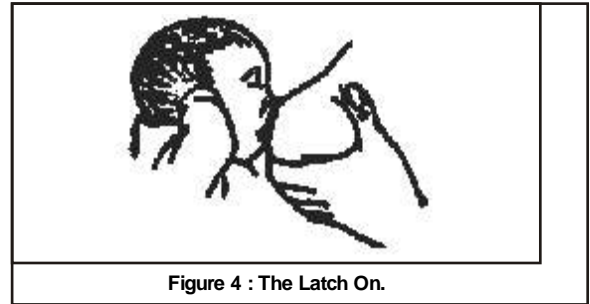


Figure 4 : The Latch On.

Listen to confirm audible swallowing and ask the mother if she has any breast pain. Initial latch on pain should resolve quickly when the latch is correct. Even when a mother has visible damage to her nipples, latch on pain should ease significantly when the positioning and latch are correct.

LACTATION SUPPORT

Many mother baby pairs experience at least one concern or problem requiring assistance from a knowledgeable health provider. Prompt assistance can determine whether breast feeding is successful or the mother weans prematurely. The principle always holds: the better the latch, more easily the baby gets the mother's milk”. From maternal perspective, the most common concern often is how to resolve painful breast feeding. To help achieve effective and comfortable breast feeding, the focus of this discussion has been the early assessment of the breast feeding couple and achieving an efficient and comfortable latch at the breast.

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Clinical Pharmacology Cell Adverse Drug Reaction Monitoring Programme

A. Patient Information

- | | | | | |
|-----------------------|--|--------------------|-----------|-----------|
| 1. Patient Identifier | 2. Age at time of reaction | 3. Sex | 4. Height | 5. Weight |
| Chart No. |Or
Date of Birth
DD MM YYYY | • Male
• Female | Cm | Kg |

B. Adverse Reaction

- | | |
|---|---|
| 1. Outcome attributed to adverse reaction | |
| • Death (Dd/mm/yyyy) | • Disability |
| • Life threatening | • Congenital malformation |
| • Hospitalisation | • Needed intervention to prevent damage |
| • Hospitalisation - prolonged | • Other |
| 2. Date and time of reaction | 3. Date of this report |
| DD MM YYYY | DD MM YYYY |
| 4. Adverse reaction or poroblem | |

5. Relevant tests / laboratory data (including dates : dd/mm/yyyy)

6. Other relevant history, including pre-existing medical conditions
(Eg. allergies, substance abuse, hepatic, renal, cardiac, CNS dysfunction)

C. Suspected Drug Product/s, Vaccine

- | | |
|--|------------------|
| 1. Name (give, generic name, labelled strenfth & namufacturer) | |
| # 1. | # 2. |
| 2. Dose, frequency and route used | |
| # 1. | # 2. |
| 3. Therapy dates (if unknown, give duration) | |
| # 1. From (dd/mm/yyyy) to (dd/mm/yyyy) | # 2. |
| 4. Indications for the use of suspected drug/ product vaccine | |
| # 1. | # 2. |
| 5. Reaction abated after use of drug / product stopped | |
| 6. • Yes • No | • Does not apply |
| 7. Lot Number | 7. Expiry date |
| # 1. | # 1. |
| # 2. | # 2. |
| 8. Reaction reappeared after reinstitution of drug / product | |
| # 1. • Yes • No | • Does not apply |
| # 2. • yes • No | • Does not apply |
| 9. Concomitant drugs used (name, does, route) and therapy dates (dd/mm/yyyy) | |

D. Reporter

- | | | | |
|-------------------------------------|-----------------|--------------|---------------|
| 1. Name, address, phone no., E-mail | | | |
| 2. Occupation | Office practice | Nursing home | Govt facility |

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Dr R N Salhan, Additional Director General of Health Services and Medical Superintendent, Vardman Mahavir Medical College and Safdarjung Hospital, New Delhi, has been honoured with the Fellowship of Indian Academy of Pediatrics (FIAP) for the year 2005.

Dr Arvind Saili, Professor of Pediatrics, Lady Hardinge Medical College and Kalawati Saran Children's Hospital, New Delhi has been honoured with Fellowship of Indian Academy Pediatrics (FIAP) for the year 2005.

Dr Ajay Gambhir, Consultant Pediatrician, Saroj Hospital, Apollo Clinic and Brahm Shakti Hospital, New Delhi, has been honoured with the Fellowship of Indian Academy of Pediatrics (FIAP) for the year 2005.

Dr Tarsem Jindal, Senior Consultant and HOD, Department Of Pediatrics, Jaipur Golden Hospital, Delhi has been honoured with Fellowship of Indian Medical Association's Academy of Medical Specialities (FIAMS) in Pediatrics at Acadima 2005, 39th annual conference of IMA held at Ludhiana, Punjab on 22nd October 2005.



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Dear Doctor,

FINAL ORDER FOR FDA ON JOHNSON'S BABY PRODUCTS

We are pleased to inform you that the Maharashtra state FDA as well as the Drug Controller of India have accepted the company's position that our Johnson's baby products are safe, pure, mild and gentle for use on sensitive skin of babies. This welcome outcome is based on our detailed response with supporting scientific and technical data, submitted to FDA in reply to its notice to the company march 16, 2005.

We have now received a final order from FDA with directions to make some changes on our product labels such as declaration of ingredients and usage safety information. These will be incorporated in batches manufactured after September 2005. With this order and our willingness to fully comply with it, the unfortunate controversy created in the media around johnson's baby products has now come to an end.

We are grateful to you for your strong support to the organization. We have been overwhelmed with the trust and confidence that our consumers, customers and esteemed doctors like you have reposed in the company. This in itself is a testimony to the rich heritage and goodwill that Johnson & Johnson India has earned over the years.

I would like to take this opportunity to once again thank you for your support and look forward to your support in serving our customers.

Sincere regards,

Sd/-

Rajeev Nagi
PROFESSIONAL MARKETING MANAGER
CONSUMER PRODUCTS DIVISION



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