INDIAN MEDICAL ASSOCIATION

VIJAYAWADA
&
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Editor's Pen

It has been indeed a very hectic period for IMA academically and otherwise during the last three months.

It is high time we, as medical professionals must take proactive part in tackling the worst man made malady "Tobacco Consumption". We not only have to educate the public, but we must also practice what we preach.

M/S Raptakos & Brett Co. Ltd helped us in bringing out this issue and on behalf of B M A and I M A, Vijayawada I sincerely thank the management for their contribution.

IS IT ACUTE APPENDICITIS?

A few hints to an acute dilemma.

Dr. B. YOGIRAM, M.S, M.N.A.M.S.,
SRI RAMA HOSPITAL, JAGGAYYAPET. Krishna dist.

When a patient comes to us with acute abdominal pain, many a times we make the diagnosis of acute appendicitis with ease and feel gratified to confirm it on the table. But, sometimes, it puzzles us because of its erratic presentation making us either misdiagnosing or over diagnosing the condition. When the diagnosis is missed or delayed, the patient may face a calamity (peritonitis and its dangerous sequelae), and if over diagnosed, may cause an unnecessary operation to the patient and more serious than that is one may be missing a more catastrophic disease in the abdomen. Hence, all the practitioners encounter this question, *"Is it acute appendicitis?*" frequently and some times may suffer sleepless nights too!!

In such difficult situations to solve the riddle, a few hints are described here. Before presenting these hints, I prefer to refresh few of the anatomical, physiological and pathological aspects of acute appendicitis in order to understand the unpredictable presentation of this disease. It is mainly due to the inconsistency in the anatomical position of the organ and unevenness in the pathological process of the disease and due to the variability in the reflex response of the other parts of the gut, mostly stomach and small bowel.

The position of appendix is so variable (retrocecal, pelvic, paracecal, pre or post ileal) that the clinical presentation could be variable.

The stomach, aptly described as a veritable amplifier and a great sympathiser responds to every acute problem in the abdomen to safe guard the gut from food by reflexly manifesting as nausea and vomiting with upper abdominal discomfort. This gastric presentation sometimes occurs before the diseased organ manifests as pain. In acute appendicitis, the gastric presentation occurs well before the pain appears in the right iliac fossa and most often the disease is diagnosed as acute gastritis in the early part of its course.

The progress of the disease is quite variable. It depends upon the age and the immunological status of the patient, pregnant or otherwise and type of the pathological process (whether it is catarrhal or obstructive appendicitis), and upon the sequelae of the disease. These sequelae may be total resolution, or localization of inflammation with resultant formation of mass or abscess; or spread of infection in the abdominal cavity leading to peritonitis.

How does acute appendicitis presents clinically? The presentation of acute appendicitis varies depending upon the time of onset of the disease. In the first forty-eight hours, the presentation may be of three types – *classical, atypical and misleading*

Classical: *If the patient presents with the following features, a sure diagnosis of acute appendicitis can be made.*

- Shifting pain to R.I.F (pain first experienced in the upper abdomen or umbilical area)
- Pain increased by cough & movement
- Nausea & Vomiting
- Low grade pyrexia (if high grade pyrexia is present, it goes against the diagnosis)
- Focal tenderness /Rebound tenderness in R.I.F
- Guarding in R.I.F

Atypical: *The following clinical features may mislead the physician in a case of acute appendicitis.*

- Absent shifting pain - in pre ileal position of appendix
- Predominant urinary symptoms- in pelvic position
- Loin pain- in retrocecal position
- Vomiting & diarrhea- in pre & post ileal positions
- ***Hence, suspect acute appendicitis in all acute abdominal pains***

Misleading presentation: *Sometimes a misdiagnosis may be made one for the other in the following situations.*

- Acute upper abdominal pain with vomiting may be diagnosed as acute gastritis.
- Shifting of pain to RIF may also be present in **PERFORATED PEPTIC ULCER** due to the tracking of duodenal fluid via right paracolic gutter (Moynahan gutter) to the right iliac fossa causing peritoneal reaction in right iliac fossa.

After two days: The presentation may be 1) abdomen with no or little guarding (in catarrhal retrocecal appendicitis), 2) a lump or 3) peritonitis.

Hints for diagnosis:

In an obscure abdominal pain, the following hints help to solve the riddle of acute appendicitis.

BYWAYS

The road to success is marked with many tempting parking spaces.

→|→|→|

- *Repeated clinical examination (every three or four hours) is the best method of diagnosis. All suspected acute gastritis cases need admission for observation, lest they should be admitted elsewhere as acute appendicitis.* (Because the early cases manifest like acute gastritis).
- *The following investigations may help to a certain extent. There is no investigation, which can positively affirm the diagnosis of acute appendicitis. LABORATORY IS A GOOD SERVANT, BUT A POOR MASTER.* The investigations mainly help to exclude the other acute abdominal conditions rather than confirming the diagnosis of acute appendicitis. The application of five senses of the clinician is far more valuable than a handful of laboratory reports.
  - § Total W.B.C COUNT: Total white cell counts >10,000/cmm is seen usually in acute appendicitis and favour the diagnosis of the disease.
  - § Urine examination: Helps to identify urological diseases
  - § Plain X-ray abdomen: Pneumoperitoneum and fluid levels indicate visceral perforation
  - § U.S.SCAN Abdomen: Non-compressible aperistaltic tubular structure with a dilated lumen and thickened wall in right iliac fossa is suggestive of acute appendicitis. However, it has few limitations –1) the procedure is operator dependent and 2) it has a false negative rate of 20%. It is mainly helpful in excluding gynecological diseases – tuboovarian diseases.
  - § Laparoscopy: It is very useful in diagnosing obscure cases of acute pain abdomen.
  - § Barium enema- its value is doubtful, and sometimes harmful, hence not advised.
  - § C.T. & M.R.I: Spiral CT scan is found to be of help.

When still in doubt – how long to wait doing repeated examinations and later, what to do?

Usually, the shifting of pain to right iliac fossa from upper abdomen or manifestation of guarding in right iliac fossa occurs within the first twelve hours. Hence, waiting for this period in obscure cases is justifiable and even after that period, if the diagnosis is inconclusive, *it is better to look and see rather than to wait and see,* especially if guarding is present.

After opening the abdomen (McBurney's incision is preferable even in doubtful cases), the appendix may be found normal, or some unexpected wonder may be seen (alas! Abdomen is like a Pandora's box). Then what to do? Let us discuss it in our next IMA newsletter.

ENHANCEMENT OF SUBSCRTIPTION

The General Body of IMA, Vijayawada and BMA met on 27th May 2001 and unanimously resolved to increase the membership subscription consequent to the increase in the Headquarters Fund Contribution(HFC).

The following is the new subscription fee :

Individual Annual membership	-	Rs 500/-
Couple Annual membership	-	Rs 700/-
Individual Life membership	-	Rs 5000/-
Couple Life membership	-	Rs 7000/-

Registration fee extra.

FORTHCOMING EVENTS

June 10th - Dr Ravi Botla, Gastroenterologist from San Antonio, USA, will be addressing our branch about "What is new in management of viral hepatitis" and "Cirrhosis and its complications".

June 16th - Clinical meeting.

June 17th - Guest lecture session on Cardiology

Sponsored by Poorna Cardia Centre, Vijayawada

i "Pacemakers- Recent concepts" by Dr. M. Srinivasa rao, Care Hospitals, Hyderabad.

ii "Pediatric cardiac interventions" -by Dr. Nitin rao, Care Hospitals, Hyderabad.

iii. Modern Trends in Surgery for Congenital Heart Disease by Dr S Pratap, Care Hospitals, Hyderabad.

June 24th - Workshop on Medico-legal aspects an CPA organised by Vijayawada obstetric and gyenecological Society. (Entry only for registrants)

July 1st - Doctors' Day (Dr B C Roy's Birthday)/Family get together and Felicitations to Dr T Anjaneya Sarma.

July 15th - Dr Chalasani Pitcheswara Rao Memorial 1st Oration for IAP, Krishna by Dr B Amdekar on "Management of Common Respiratory Problems".

July 29th - Dr (Maj) K.N.Rao and Dr.T.Srinivasan Memorial Oration. Dr.Tanikachalam will speak on "Timing of surgery in valvular regurgitation"

ANNUAL CME PROGRAMME - 2001

IMA Vijayawada conducted the annual CME programme on 6th May 2001 at our IMA hall. Soumya Apollo Hospitals had sponsored this year's programme.

The Programme began with a brief inaugural session. The chief guest was Dr Narasimha Reddy, Registrar, NTR University of health sciences. Dr C Suresh, MD of Soumya Apollo Hospitals spoke on the occassion. Dr T Anjaneya Sarma presented a wall clock to our association.

The topics covered were ***"Reconstructive surgery in head and neck cancer", "Oxygen therapy". "Common urological emergencies", "Use of drugs in patients with renal failure", "Termination of pregnancy - Recent developments" and "Mangement of Anaphylaxis"***.

Do you know what is DOTS?

DOTS is an accronym for Directly Observed Treatment, Short course.

It is a strategy for the control of TB. DOTS is based on research done over the past 40 years. DOTS combines 5 elements

1. Government commitment,
2. Diagnosis primarily by microscopy,
3. Regular supply of good quality drugs for short-course treatment,
4. Direct observation of treatment, at least in the intensive phase, and
5. Systematic monitoring and accountability.

DOTS ensures that patients take medicines regularly untill they are cured. During the intensive phase a health worker watches as the patient swallows the drugs in his/her presence.

Sputum microscopy is done at defined intervals to monitor patient's progress.

The key to the success of DOTS strategy is that it places the responsibility for curing TB on the health workers - not the patients.

CENTRE FOR DNA FINGERPRINTING AND DIAGNOSTICS

Dept. of Biotechnology, Govt. of India, Diagnostics Division

7-18 ECIL Road, Nacharam, HYDERABAD: 500076

Charges for Genetic Diagnosis &

Counseling Services available at CDFD

CLINICAL GENETICS:

Clinical diagnosis of genetic diseases,Pregnancy counseling,Syndrome identification, Management of genetic disorders in terms of Genetic counseling,therapeutic and nutritional interventions.

CYTOGENETIC SERVICES:

Chromosomal analysis from

Peripheral blood	Rs: 500/-
Amniotic fluid	Rs1250/-
Fetal cord blood sample,	Rs:750/-
Bone marrow/ leukemic blood	Rs: 500/-
Chorionic Villi	Rs:1250/-
Products of conception	Rs:1250/-

MOLECULAR DIAGNOSIS SERVICES (Proband/Carrier/Prenatal diagnosis)

Spinal muscular Atrophy (Werdnig-Hoffman syndrome) Rs: 1500/-

Duchenne/ Becker muscular Dystrophy DMD carrier analysis Rs: 1500/-

Myotonic Dystrophy Rs: 1000/-

Ataxia panel: SCA1, SCA2, SCA3, SCA6 and SCA7. Rs 1000 /3500 per panel

SRY and DAZ Analysis Rs: 1000/- each(Both-1500)

Fragile -X Syndrome Rs: 500/- persample/1500 per family

Cystathionine beta synthase and MTHFR mutation analysis for CAD Rs: 1500/-

Methylene tetra hydro folate reductase and CBS mutations for Neural tube defects.Rs: 1500/-

Homocystinuria	Rs: 1000/-	Heamophilia-B	Rs: 1500/-
Freidreich ataxia	Rs: 1000/-	Factor 5 Leiden	Rs: 1500/-
Huntington's ataxia	Rs: 1000/-	Sickle cell anemia	Rs: 1500/-
Heamophilia-A	Rs: 1000/-	Beta-Thalassemia	Rs: 1500/-

BIO-CHEMICAL DIAGNOSIS SERVICES:

Total plasma Homocysteine assay for Neural tube defects, Coronary artery diseases, Down's syndrome, Bad obstetric history etc. Using HPLC with fluroscencedetection. (Pre column derivitisation) Rs: 550/-

Plasma Amino-acid assay for all individuals Amino-acid disorders using HPLC with Fluorescence detection, pre column derivitisation, Gradient separation. TLC Rs: 200/-

HPLC:Rs: 500/-

Maternal serum alfa-fetoprotein (MSAFP), amniotic fluid alfa-fetoprotein (AFAFP) and acetylcholine esterase activity in amniotic fluid. Rs: 250/- AMF: 350/ -

Triple marker Screening: AFP, intact HCG and unconjugated estriol screening serum.

Individual test: 150/- Total: 400/-

Galactose assay for Galactosemia. Rs: 150/-

BIOCHEMICAL SCREENING FOR NEWBORN AND SICK NEWBORN

1. **Amino-acid Disorders: (TLC** Rs: 200/-, **HPLC** Rs:500/-)

Homocystinemia	Maple syrup urine disease
Phenylketonuria	Hyperleucinemia
Hyper-phenylalaninemia	Tyrosinemia
Hyper glycenemia	Histidinemia etc.
Urea cycle disorders	Alkaptonuria

2. **Carbohydrate Disorders:** Rs: 150/-

Galactosemia (Using CODA System)	Glycosuria

3. **Hematological Disorders:** Rs: 150/-

Hemoglobinopathies	Glucose 6 phosphate dehydrogenase

4. **Hormone assays using CODA system:** Rs: 150/-

Neonatal TSH	17-hydroxy progesterone

For further information contact:

Dr.Seyed E.Hasnain, Dr. Radha Ramadevi

Director Head, Diagnostics division

Phone: 7155604 Fax: 7155610 Phone:7155608 Fax:7155610

Collection and Transport of Samples:

For Cytogenetic Analysis:

Peripheral blood: 3 ml of blood in heparin tube.

Bone marrow/leukemic blood: 2-3 ml bone marrow/leukemic in heparin tube.

Amniotic fluid: 15-20 ml in sterile centrifuge tubes.

Chorionic villi: About 30-mg chorionic villi tissue in transport culture media (supplied on demand).

Homocysteine and Molecular analysis:

5 ml of blood in EDTA tube should be sent without lysis for molecular analysis of listed disorders.

For Homocysteine after collection of 5-ml blood in EDTA tube immediately centrifuge at 3000 rpm and separate plasma in to 2-ml appendorf and send within 24 hours. (Separation of plasma from RBC or Whole blood should be done within 10 mints after collection of blood)

Biochemical Analysis:

TSH, GALT, G6PD and TLC amino acids, Carbohydrate Chromatogram requires Blood spots on S&S specimen collection paper or Whatman No: 3 filter paper. (The blood spots should be 4 and it should be broad and round).

IMA VIJAYAWADA GOES ONTO WEB

Vijayawada IMA now has a Website which will be inaugurated formally on Doctors' Day ie. July 1st.. Members who wish, can log on to

`http://imavja.20m.com.`

Members can kindly help by contributing speciality wise links so that they can be incorporated in our web page. Members having their own web pages can also give their URLs and they will be gladly included. Any suggestions about improvement of the Website are most welcome.

Take it easy

Telegram : "Missed the train. Will leave tomorrow same time."
Reply : "Don't leave same time. will miss it again."

MEASURE PLEASURE - CONVERSION UNITS

Basic unit of laryngitis	:	One hoarsepower.
Half of large intestine	:	Semi colon.
Time between slipping on a peel and falling	:	one bananosecond.

GLEANINGS FROM INTERNET

ORAL CONTRACEPTIVES REDUCE COLORECTAL CANCER RISK
Oral contraceptives offer significant protection against colorectal cancer, according to the results of a meta-analysis of 20 studies, published in the current issue of the British Journal of Cancer.
http://familymedicine.medscape.com/36293.rhtml?srcmp=fm-042001

ELEVATED PULSE PRESSURE IN EARLY PREGNANCY LINKED TO PREECLAMPSIA
Women whose pulse pressure is elevated in early pregnancy are at increased risk for developing preeclampsia, according to a report by researchers from Massachusetts General Hospital in Boston.
http://familymedicine.medscape.com/36218.rhtml?srcmp=fm-042001

MODERATE ALCOHOL CONSUMPTION REDUCES AMI MORTALITY, RISK OF HEART FAILURE
Compared with nondrinkers, patients who consume moderate amounts of alcohol in the year before an acute myocardial infarction (AMI) have reduced mortality, and older individuals who consume moderate amounts of alcohol have a lower risk of heart failure.
http://familymedicine.medscape.com/36342.rhtml?srcmp=fm-042001

NEW TYPHOID VACCINE SAFE AND IMMUNOGENIC IN YOUNG CHILDREN
A newly devised Salmonella typhi Vi conjugate vaccine (Vi-rEPA) is safe and immunogenic in children aged 2 to 5 years, according to a report published in the New England Journal of Medicine.
http://familymedicine.medscape.com/37050.rhtml?srcmp=fm-042701

DESPITE IOM REPORT, CONGRESSMAN WANTS RECALL OF MMR VACCINE, CITING AUTISM RISK
The chair of the House Government Reform Committee today blasted science and health officials for not recalling the combination measles, mumps, and rubella (MMR) vaccine that he says may be causing autism.
http://familymedicine.medscape.com/37159.rhtml?srcmp=fm-042701

PRENATAL ZINC SUPPLEMENTS IMPROVE HEALTH OF LOW-BIRTHWEIGHT OFFSPRING
Maternal zinc supplementation during pregnancy reduces morbidity during the first 6 months of life in low-for-gestational-age infants, according to results of a study conducted in Dhaka, Bangladesh.
http://familymedicine.medscape.com/35968.rhtml?srcmp=fm-041301

It is now proven beyond doubt that smoking is the leading cause of statistics

-Fetcher Knebel

CLINICAL MEETING - MARCH 2001

The clinical meeting for the month of March, 2001 was held on 17-3-2001, at IMA hall. The following
cases were presented and discussed.

1. Multiple Hydatids of the liver, Surgical excision by thoraco abdominal approach - Dr RV Sivarama Prasad
2. Imaging spectrum of Hydatid disease - Dr VN Vara Prasad
3. A case of leptospirosis - an unusual presentation - Dr NV Narayana Rao

Later interesting orthopedic XRays were presented by Dr Y Poorna chandra Rao and Dr V Somanadham.

The case presented by Dr TV Narayana Rao - A 45 yr old male presented with increasing breathlessness and oedema of feet for the previous 2days. There was H/O fever with chills.MP was positive and SBR was 3.5 mg%. X-Ray chest showed consolidation of the right lower lobe. There was intense conjunctival congestion with significant sub conjunctival hemorrhages and herpes simplex labialis at time of admission. After admission investigations revealed PF and QBC for MP were negative. Liver function tests revealed increased SBR but normal SGPT and SGOT. Leptospirosis IG M antibody test was positive.

Hence the patient was given besides supportive treatment Inj Crystalline Penicilline and Doxycycline. There was gradual improvement and the patient was dscharged on the 8th day after admission.

Interesting interaction followed.

CLINICAL MEETING - APRIL 2001

The clinical meeting for the month of April, 2001 was held on 21-4-2001, at IMA hall. The following
cases were presented and discussed.

1. Two cases of tuberculosis of larynx, diagnosed as Carcinoma of larynx -Dr Manne Venkataratnam

   These are cases which came with progressive hoarseness of voice. There were masses seen on vocal cords and one was diagnosed on biopsy as Sq cell carcinoma. The second case was clinically diagnosed as malignant tumor but X-Ray chest revealed TB lesions.
2. Two interesting cases of cervical cord compression with quadriparesis, modalities in management - Dr SV Ranga Rao

   i. A male 45 year old with a massive tumor in the cervical region resulting in progressive quadriparesis. A cervical laminectomy and subtotal cord tumor decompression was done and the patient regained power of 4/5 from 2/5 within 2 months.

   ii. A female 65 year old with a high cervical cord tumor at C2-C3 level with progressive quadriparesis was operated with removal of the tumor and significant improvement in the power.

Interesting interaction followed.

MANAGEMENT OF ANAPHYLAXIS

Dr Thota Phaneendranadh
Soumya Apollo Hospitals

Definition:

The symptom complex that accompanies the acute reaction to a foreign substance to which the patient had been previously sensitized (Immediate or Type I hypersensitivity)

The term anaphylactoid reaction is used to describe reactions that are clinically identical to anaphylaxis, but the mechanism is either non-immunological or has not been determined

Aetiology:

Drugs, Blood products, Plasma substitutes, X-ray contrast media, Foods or food additives, Insect stings

Pathophysiology:

This is IgE modulated. Histamine is responsible for early signs and symptoms. There is production of vasodilators. Smooth muscle contraction, increased glandular secretion and Increased capillary permeability are seen.

Anaphylactoid reaction:

The direct histamine-releasing properties of some drugs may produce severe reactions due to histamine alone

Clinical anaphylaxis is usually seen in fit patients!

Does the adrenal response to stress “pretreat” sick patients? It is a possibility.

Asthma seems to be an exception to this protection.

Clinical presentation:

Onset is variable, usually <10 min to parenterally administered drugs. Duration and severity are also variable.

Cutaneous, respiratory, GI and cardiovascular manifestations occur either singly or in combination

a. Cutaneous:

Erythematous flush, general urticaria, Angioneurotic oedema, Conjunctival injection, Pallor, Cyanosis.

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b. Respiratory:

Rhinitis, Coughing, Choking sensation, Laryngeal oedema, Bronchospasm, Pulmonary oedema.

c. Cardiovascular:

Most common. Often it is the only system to be affected. There is increased heart rate, decreased arterial blood pressure and shock.

d. Cardiovascular collapse:

Vasodilatation, Capillary leak, Endogenous hypotension, Decreased Venous returns, Decreased Cardiac output, Decreased Arterial blood pressure? Myocardial dysfunction.

e. Others:

Apprehension, Paraesthesia, Arthralgia, Convulsions, Loss of consciousness, Disorders of haemostasis.

Management:

Withdraw/discontinue the agent causing the reaction, maintain airway, administer oxygen, assisted ventilation if necessary, monitoring of heart rate & rhythm, CVP, arterial blood pressure, SpO2.

Adrenaline is the drug of choice. It increases cyclic AMP in leucocytes and mast cells. It inhibits histamine release. It has beneficial effects on vasomotor tone, broncho-motor tone and contractility. Dosage: Adrenaline 0.5 mg IM or bolus + infusion IV.

Rapid infusion of colloids & crystalloids is necessary. Noradrenaline may be life saving. IV aminophylline and Nebulized salbutamol are useful. Antihistamines' usefulness is doubtful. Corticosteroids are useful only in refractory broncho-spasm

Diagnosis:

Mast cell tryptase level is increased one hour after an anaphylactic reaction begins. This increase persists for 4 hours. It is highly specific & sensitive. It can be used for postmortem diagnosis.

Follow-up:

Determination of agent that is responsible is a must.

? Hyposensitization, medic alert bracelet, etc. education of patient and family in use of drugs, adrenaline, cromoglycate, antihistamines, salbutamol, these are all useful in preventing a further episode.

Frequently asked questions about second-hand smoke

What is second-hand smoke?

Second-hand smoke results from the "sidestream" smoke that comes from the burning tip of a cigarette and the "mainstream" smoke that is exhaled by the smoker. Second-hand smoking, passive smoking, involuntary smoking or exposure to environmental tobacco smoke (ETS) all refer to the phenomena of breathing other people's smoke.

What is found in second-hand smoke?

Second-hand smoke is the smoke that individuals breathe when they are located in the same air space as smokers. Second-hand smoke is a mixture of exhaled mainstream smoke from the tobacco user, sidestream smoke emitted from the smoldering tobacco between puffs, contaminants emitted into the air during the puff, and contaminants that diffuse through the cigarette paper and mouth end between puffs.[1] It is a complex combination of over 4000 chemicals in the form of particles and gases. It includes irritants and systemic toxicants poisons such as hydrogen cyanide, sulphur dioxide, carbon monoxide, ammonia, and formaldehyde. It also contains carcinogens and mutagens such as arsenic, chromium, nitrosamines, and benzo(a)pyrene. Many of the chemicals, are reproductive toxicants such as nicotine, cadmium and carbon monoxide, damage reproductive processes. Second-hand smoke is also an important major indoor air pollutant. It has been classified by the United States Environmental Protection Agency as a "class A" or human carcinogen for which there is no safe level of exposure.

How does second-hand smoke affect health?

There is substantial scientific evidence that second-hand smoke is a serious health threat. Non-smokers who breathe second-hand smoke suffer many of the same diseases as regular smokers. Heart disease mortality deaths as well as lung and nasal sinus cancers have been causally associated with second-hand smoke exposure. Second-hand smoke also causes a wide variety of adverse health effects in children including bronchitis and pneumonia, development and exacerbation of asthma, middle ear infections, and "glue ear", which is the most common cause of deafness in children. Exposure of non-smoking women to second-hand smoke during pregnancy causes reduction in fetal growth, and there is also evidence that postnatal exposure of infants to second-hand smoke contributes to greatly increased risk of sudden infant death syndrome (SIDS). Tobacco smoke also causes immediate effects such as eye and nasal irritation, headache, sore throat, dizziness, nausea, cough, and respiratory problems.

What is the extent of the problem of second-hand smoke?

It is a Exposure to second-hand smoke is a widespread ubiquitous problem that af-

fects people from all cultures and countries. This exposure occurs throughout ordinary situation in daily life: in homes, at work and school, on playgrounds and public transport, in restaurants and bars—literally everywhere people go.

Surveys conducted in countries around the world confirm widespread exposure. One survey estimated that 79 % of Europeans over age 15 were exposed to second-hand smoke. Another estimated that 88% of all non-smokers in the United States were exposed to second-hand smoke. Recent data from South Africa shows that 64 % of children below age five in Soweto live with at least one smoker in the house. The Cancer Society of New Zealand reports that second-hand smoke is the third largest killer in the country, after active smoking and alcohol use.

Are well-ventilated non-smoking sections the answer?

No. Although good ventilation can help reduce the irritability of smoke, it does not eliminate its poisonous components. When smoking sections share ventilation with non-smoking areas, the smoke is dispersed everywhere. Smoking sections only help protect non-smokers when they are completely enclosed, have a separate ventilation system that goes directly outdoors without re-circulating air in the building, and when employees are not required to pass through them.

So how can we protect people from second-hand smoke?

Governments can regulate and legislate smoking bans in public places, educate people about the dangers of second-hand smoke, and provide support for those who wish to quit smoking. Employers can initiate and enforce smoking bans in workplaces. Parents can stop smoking in the house and car, particularly around children, and ask others to do the same. They can also ensure that their children's day-care, school and after-school programs are smoke-free. Individuals can let their family, friends and co-workers know that they do mind if they smoke near them.

Work with your local organizations to initiate actions on second-hand smoke.

Are smoking restrictions hard to enforce?

Most of the public — even smokers — supports smoke-free spaces. Smoking bans in workplaces and public places work when people are aware of them. The public should know in advance that smoking bans are being implemented, and they should know the health reasons for smoking bans. Good education and advance planning lead to self-enforcement and success of smoking restrictions.

Do smoking restrictions hurt business?

No. Most employers who go smoke-free save money by increasing productivity, lowering maintenance and cleaning costs, and lowering insurance coverage. Studies of sales receipts from restaurants and bars in the US before and after smoking bans have found that sales usually stay the same or go up after a smoking ban.

...then why are smoke-free places so rare?

The tobacco industry spends millions to fund misinformation campaign on second-hand smoke. Scientists and consultants have been hired to not only confuse the public about the validity of scientific data, but to also create doubt about the researchers who produce the data and about the science itself. In addition to attacking legitimate studies, bogus research projects that downplay the seriousness of second-hand smoke are funded and promoted.

Tobacco lobbyists and lawyers deflect government regulation of second-hand smoke, and this has been supplemented, aided by huge tobacco contributions to political campaigns. When money and misinformation don't work, the industry promotes false solutions to control second-hand smoke.

Although evidence shows that ventilation is not an effective solution to the problem of second-hand smoke, the industry continues to push for this option, even forming indoor air consulting "front groups" who downplay the risks of second-hand smoke.

A campaign to promote "courtesy of choice" as an alternative to banning smoking in public places has been launched worldwide. This implies that the serious problem of second-hand smoke can be solved merely by smokers asking for permission before they light up, or by having separate smoking and non-smoking sections. Second-hand smoke is thus portrayed as a mere annoyance for non-smokers, rather than as a health issue. The industry also funds smokers' rights' movements to create so-called independent opposition to smoking bans. People concerned about second-hand smoke are then branded as zealots.

Fortunately, tobacco industry opposition to clean air can be defeated. Your actions will make a difference. Become a leader in your workplace, your organization, your community, and your home. Speak up for clean air and make your voice heard! Let's clear the air.

[1] *Environmental Protection Agency. Respiratory health effects of passive smoking: Lung cancer and other disorders. Washington, D.C.: Office of Health and Environmental Assessment, 1992.*

A man who wants to do something will find a way
A man who doesn't, will find excuses

Every man has three characters -
that which he exhibits,
that which he has and
that which he thinks he has.

Thus spake the wise

TIME

Time goes, you say? Ah, No!
Alas, Time stays, We go!!
- Austin Dobson

MAIL

I have received no more than one or two letters in my life that were worth the postage.
- Henry Davi Thorreau

ELECTION

The election is not very far off when a candidate can recognize you across the street.
- Kin Hubbard

The only new thing we learn from new elections is we learned nothing from the old.
- American proverb

VALUE

Now a days we know the price of everything and the value of nothing.
- Oscar Wilde

NEWS IN BRIEF

The Annual Conference and Continuing Medical Education on "Changing face of Radiology and Imaging" was organised by Indian Radiological & Imaging Association, Andhra Pradesh State Chapter in association with I M A Vijayawada branch on 11th March 2001 at Swarna Vedika A/C Conference Hall, Vijayawada. The daylong conference was preceded by a brief inaugural function. It was presided over by Dr Kakarla Subba Rao, President IRIA and Organising Committee chairman. The Chief Guest was Dr S Aruna, Minister of Health, Andhra Pradesh. The faculty included Dr Kakarla Subba Rao, Dr Bhavin Jankharia, Dr B Srinivas Desai, Dr B S Rama Murthy, Dr C Joga Rao, Dr T L N Praveen, Dr J Jagan Mohan Reddy and Dr S Rama Murthy. In the night, Banquet was preceded by a hilarious skit by Dr N V K Prasad and friends and old melodious songs.

On 7th April 2001, IMA, Vijayawada organised a meeting along with Vijayawada Psychiatric society to commemorate World Health Day. The theme of this year is "Mental Health - Stop exclusion: Dare to Care". The speaker was Dr Karri Rama Reddy from Rajahmundry.

On 10th April 2001, IMA, Vijayawada along with Human development foundation conducted a meeting on "Importance of Chakra Dhyanas" and "Yoga for better health". Sri Ananya Bhagavad Dasaji(Lena Larsen) from Sweden spoke on these subjects.

On 1st April 2001, a CME session on "Organophosphorus poisoning" and a panel discussion on "Management of poisoning cases" were conducted. Dr TBKV Prasada rao from Ponnur spoke about Organophosphorus poisoning and the panelists for the latter session were Dr TBKV Prasada rao, Dr. N Ammanna, Dr S Bhanu Prabhakar and Dr G Eswar.

On 15th April 2001, Dr Radha Rama devi, from Centre for DNA Finger Printing and Diagnostics, Hyderabad, Spoke on "Human Genetics in the new millennium - How genetics help practicing physicians". This was a combined meeting of IMA and IAP,Krishna.Later Dr C Sudha spoke on "Down's syndrome, prenatal risk assessment and management"

On 17th April on the occasion of World Hemophilia day IMA, Vijayawada along with Hemophilia society, Vijayawada chapter, conducted a programme for hemophilia patients. This started with interaction with hemophilia patients, followed by a lecture by Dr Maganti Prasad and workshop on Physiotherapy by Dr Sajja Koteswara rao.

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IRON POLYMALTOSE COMPLEX

Iron complexed with carbohydrate molecules, including ferric hydroxide-polymaltose complex (Fe-PM), have been surounded by controversies to some extent because of questions on the bioavailability of iron administered through them. It may therefore be worth while taking a closer look at their kinetic behaviour, particulrly because of their higher cost of therapy.

Based on a few rat model studies, it was once believed that the non-covalently bound polymaltose molecules would allow entry of the ferric form straight into the body without having to go through conversion into the ferrous form. However, recent studies have made it cler that iron from Fe-PM is also absorbed only in the ferrous form after its release and reduction from the high molecular weight complex. It must be noted that the rat, unlike humans, is capable of synthesising ascorbic acid in the gut lumen which reduces the ferric iron to ferrous form before absorption. This might have been responsible for some of the earlier controversial claims on Fe-PM.

Almost no haemoglobin increase was observed in 9 patients during a 40weeks treatment period when given Fe-PM (100-300 mg Fe/d) on empty stomach, whereas subsequent treatment with ferrous sulfate (100-200 mg Fe/d) was therapeutically effective (0.15-0.23 g/dl Hb-increased).

Enhancers of iron absorption present in the diet play a significant role in accelerating recovery of the anaemic patient. It is obvious that iron supplements alone cannot do the job, which has to be completed by overall dietary management.

No matter what form of iron is presented to the gastrointestinal tract through food or pharmaceutical formulations, it has to be ultimately converted to and absorbed only as the ferrous form.

The clinician will no doubt keep an overall perspective of cost-safety-efficacy considerations before prescribing therapy for an individual patient.

Because of its very limited bioavailability feric polymaltose, like all other feric iron preparations hitherto known, should not be used in oral iron therapy in man. The intestinal iron absorption

of a single oral dose (100 mg Fe) of Fe-PM was very low as compared with ferrous sulfate. The low absorbability of iron from Fe-PM was confirmed by an almost negligible therapeutic efficacy of commercial Fe-PM in patients with severe iron deficiency anemia.

During and after a treatment period of 4 weeks (100-300 mg Fe/d), the effect on the haemoglobin concentration and the iron utilization (taken into account the individual blood loss and the food iron absorption) was low or almost negligible. In addition, all other parameters of iron metabolism (serum iron, tranferrin iron saturation, and serum ferritin were not significantly increased under oral treatment with Fe PM. A similar low iron bioavailability was recently reported for the use of Fe-PM in a group of iron deficient children.

A measurable amount of ionic iron was released from Fe-PM complex at low pH. Iron from Fe-PM is absorbed in humans only in form of ferrous iron after its release and reduction from the high molecular weight complex. No evidence was found for a special absorption mechanism different to ionic iron as suspected in rats.

Therefore, ferric polymaltose, similar to ferric polysucrose and probably similar to other ferric carbohydrate complexes shoud not be used in the oral iron therapy in humans.

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Swamy Chinmayananda said

The youth are not useless, they are used less,
Youth are not careless, they are cared for less.

Parents give you your body. You bring your mind yourself. To cultivate that mind and grow from within is your own responsibility.

Don't fall in love, but rise in love.

In life you may never get what you desire but you never fail to get what you deserve.

A truly successful man is one who can lay a firm foundation with the bricks that others throw at him.

Disappointment can come only to those who make an appointment with the future.

TREATMENT OF SEIZURE DISORDER CURRENT CONCEPTS

DR.V. JAYAKUMAR, MD, DCH, DM (NEURO).,

The management of Seizure Disorder involves consideration of several factors of which some of the more important practical aspects are outlined here. The choice of the drug and its duration of therapy depend on the correct identification of the seizure type and the epileptic syndrome.

1. WHEN TO START TREATMENT?

Seizure provoked by metabolic insults, infection etc., need to be treated in the acute phase. Long term anticonvulsant therapy may not be necessary in most of the occasions except when there is a specific risk of recurrence.

As the treatment is for a prolonged period of time, first seizure doesn't require long-term anticonvulsant therapy unless there is a risk of recurrence.

The risk of recurrence after the first unprovoked generalised tonic clonic seizure is 42% at 2 years and after a second seizure risk is 76-96%

Following risk factors are associated with increased recurrence risk.

Etiology (Birth asphyxia, CNS infection Phakomatosis etc.,)

Abnormal EEG

Type of seizure (eg. partial)

The risk of recurrence at 2 years is as follows

Idiopathic seizure with Normal EEG - 24%

Symptomatic seizures or Abnormal EEG - 48%

Symptomatic seizure and Abnormal EEG - 65%

Misdiagnosis of other conditions for seizure is greater in patients who had a single episode hence long-term treatment is usually deferred until there is a recurrence.

2. **WHICH DRUG TO BE USED AND HOW?**

It is always better to start with a single drug, which is ideal for the particular type of seizure, as given below:

GENERALISED

1.Tonic Clonic -	Carbamazepine, Sod. Valproate, Phenytoin,Phenobarbitone, Primidone
2.Absence	Ethosuximide, Sod. Valproate, Clonazepam
Atypical Absence	Clobazam.
3. Myoclonic	Sod. Valproate, Clonazepam, Nitrazepam, ACTH, Clobazam, Zonisamide.
4. Tonic	Phenytoin, Phenobarbitone
5. Atonic	Sod. Valproate, Clobazam
PARTIAL	Carbamazepine, Phenytoin, Phenobarbitone, Gabapentin, Oxcarbazine

INFANTILE SPASM	ACTH
LENNOX GASTAUT'S	Sod. Valproate, ACTH, Felbamate
PHOTOSENSITIVE EPILEPSY	Sod. Valproate.
HOTWATER BATH EPILEPSY	Carbamazepine

Wrong selection or wrong combinations can worsen the seizure for eg.

1. Carbamazepine	Can worsen absence, myoclonic seizure and atypical absences.
2. Phenobarbitone	Increases absence seizure, atonic seizure.
3. Ethosuximide	not effective for partial seizure.
4. Phenytoin, Carbamazepine	not effective for febrile fits.
5. Vigabatrin	may aggravate myoclonic, absence seizures.

Although the selection of drug is based on the seizure type, sometimes it may be necessary to go by the type of epileptic syndrome.

For eg. Although Carbamazepine is effective against GTCS it may worsen the overall condition when tonic clonic seizure occur in Juvenile Myoclonic Epilepsy Syndrome.

3. **WHICH DOSAGE IS TO BE FOLLOWED?**

Recent studies indicate that in most of the occasions initial low dosage schedule is sufficient as the incidence of side effects increase with increasing drug dosage.

Sometimes we may to go to a dosage, which is almost nearer to the toxic level. Hence each patient has to be individualised. The detailed drug dosage of most of the anticonvulsants and their toxicity are given in every textbook and may be referred.

4. **HOW FREQUENTLY THE DRUG HAS TO BE GIVEN**

In general drugs like phenobarbitone and phenytoin are given once a day as their half-life is around 12-24 hours, whereas drugs like Sod. Valproate, Carbamazepine, etc., whose half-life is 8-12 hours have to be given at least twice or thrice a day.

5. **HOW TO INCREASE THE DOSAGE?**

After starting the drug at a low dose gradually the dose is increased at periodical intervals till it reaches a steady state (which refers to the time taken for a steady drug level to be reached after each dose administration.)

To achieve 'steady state' for a drug it takes about to assess whether the drug is effective or whether further increase is necessary. Too frequent increase in the drug dosage before this time can result in toxicity and too early withdrawal of the drug or addition of a new drug before this period can result in replaces. The following table gives the half-life of various anticonvulsants and the time taken for the steady state to be reached.

	HALFLIFE	TIME TAKEN FOR STEADY STATE
1.PHENOBARBITONE	48-96 hours	2-4 weeks

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2.PHENYTOIN	12-24 hours	5 days
3.CARBAMAZEPINE	8-12 hours	3-4 days
4.SODIUM VALPROATE	6-8 hours	2-4 days
5.PRIMIDONE	6-18 hours	4-7 days

6. **WHAT ABOUT DRUG INTERACTION?**

Although numerous drug interactions can occur some of the important interactions are considered.

In a patient who is already on phenytoin.

Addition of Sod. Valproate, INH (for primary complex) chloramphenicol, may increase the level of phenytoin resulting in toxicity.

Addition of phenobarbitone will lower the level of phenytoin resulting in recurrence of seizure.

Erythromycin can elevate carbamazepine level resulting in toxicity.

7. **IS PROGNOSIS AFFECTED BY EARLY CONTROL OF SEIZURE?**

It has been found out that repeated uncontrolled seizure if not treated early may result in reduced response to the available anticonvulsants. The prognosis also depends on the epileptic syndrome.

BAD

Epilepsies associated with neurological handicaps from birth.

Progressive disorders

GOOD

Childhood absence

GTCS on awakening,

EXCELLENT

Benign partial epilepsies

Benign myoclonic epilepsy of infancy.

Some forms of reflex epilepsies.

Uncertain:

Juvenile myoclonic epilepsy

Localisation related epilepsies.

8. **WHAT TO DO WHEN MONOTHERAPY FAILS?**

We have 2 options

To add another drug

To substitute the initial drug with another with subsequent gradual withdrawal of the first drug.

The second option is favored by many epileptologists.

Sometimes we may have to add one more drug in certain refractory cases. (Usually not more than two anticonvulsants are given to the patient at a time).

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For eg.

In myoclonic absence - Sod. Valproate + Ethosuximide.

In refractory complex partial seizure - Phenytoin + Carbamazepine.

In some GTCS - Sod. Valproate + Carbamazepine.

Vigabatrin acts by increasing GABA level Lamotrigine acts by decreasing excitation by glutamate level, thereby both have complimentary action.

Similarly Sod.Valproate and Lamotrigine may be effective in combination.

There is no rationale in giving primidone and phenobarbitone as primidone is ultimately converted to phenobarbitone.

9. **HOW LONG TO CONTINUE ANTICONVULSANTS?**

When the seizure free period is sufficiently long for eg. 2 years then the possibility of discontinuing the drugs gradually can be considered (withdrawal should be done gradually over a period of not less than 6 months).

However there is a risk of relapse, which is about 25% at 1 year and 29% at 2 years. Factors, which may help to predict the relapse, are

Type of Seizures: - Myoclonic, tonic, atonic and symptomatic partial seizure.

In Juvenile myoclonic epilepsy 85-95%. Hence the treatment is for life long.

GTCS on awakening 30-90%

Symptomatic partial epilepsy 25-75%

Childhood absence epilepsy 5-25%

Benign rolandic epilepsy 0%

Abnormal EEG

Onset of seizure before the age of 2 years.

Presence of a structural lesion and or neurological deficit.

Prolonged duration of epilepsy or high number or seizures before control.

History of status epilepticus.

Short duration of seizure free period.

More than one seizure type.

Poly therapy at the time of discontinuation.

Fast rate of drug withdrawal.

10. NEWER ANTICONVULSANTS: -

They are quite expensive

They are mostly useful as 'add on' Drugs in refractory cases.

Some of them are poorly tolerated.

As detailed review is not possible only their clinical indications are outlined

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Politicians are same all over. They promise to build a bridge where there is no river

- Nikita S Krushchev

DRUG	SEIZURE TYPE
1. ***GABAPENTIN*** *(GABA ANALOGUE)*	Partial
2. ***VIGABATRIN*** *(inhibits GABA Transaminase)*	Partial, Infantile spasm due to tuberous sclerosis,LGS.
3.***GLOBAZAM*** *(Benzodiazepine)*	Partial, LGS, inf.spasm, startle and eating epilepsy
4.***OXCARBAZEPINE*** (Keto analogue of Carbamazepine)	Partial, Generalised
5.***LAMOTRIGINE*** *(Inhibits glutamate)*	Secondary generalised, atypical absence, partial and generalised.
6.***FELBAMATE*** *(Aplastic anaemia, Hepatotoxicity are* limiting its use).	LGS, Refractory partial seizures.

MEMBERS' DIRECTORY

I M A Vijayawada is bringing out a latest "Members' Directory". Member's name, Address and Telephone numbers will be given as per the available data with us.

Members are requested to go through the database in our office and make suitable corrections if any. Change in address and telephone numbers may be intimated to the office in writing so that latest and near accurate information will be printed in the directory.

We donot wish to print the mobile phone numbers of the members to preserve their privacy. Members who wish their mobile numbers to be included must inform in writing to the office.

We also wish to print the residence numbers of the members. If any member doesn't want his/her residence number included, also must inform the office in writing.

Advertisements are welcome in the directory. Prime slots will be allotted on first-come-first-served basis.

For tariff details contact IMA office.

Thanks Galore

This year's Annual CME programme is the combined effort along with Soumya Apollo Hospitals. Big Thanks to Dr C Suresh, MD and to the excellent faculty from Soumya Apollo comprising of ***Dr.Vijaya Kumar, Dr.Manikanth Lodaya, Dr.M. Anand, Dr.A. Srinivas and Dr.Thota Phaneendranadh***. Their zeal and enthusiasm for sharing their knowledge and experiences with our members is really laudable.

Dr R S Rama Devi, spoke on "Medical termination of pregnancy" on the CME day. Thank you very much madam, for your delightful lecture.

The new big wall clock in our IMA Hall is donated by Dr T Anjaneya Sarma garu and is probably meant to remind us of the need for punctuality (the earlier piece is rather small). We thank you sir, for your **"time"ly** gesture.

The 6th piece in our series of patient education handouts "Garbhinee streelu - Jaagrattalu" was the effort of Dr V Padmaja and with fine-tuning from Dr C Sudha and Dr R S Ramaa Devi, the issue came out beautifully. Thank you so much Madams.

The eloquent drawings in our patient education handout series number 7 on the occassion of "World No Toacco Day" about the bitter facts of smoking were the contribution from Shri Y V Krishna, Principal, Vidyardhi English medium school, Vijayawada.

TAKE IT EASY

* A bald headed man was complaining to his barber about the price of the haircut. "You ought to cut my hair cheaper because there is so little of it to cut."

  "But you don't understand, sir," explained the barber. "In your case we don't charge for cutting the hair; we charge having to search for it."
* When he gets home, a man finds his pregnant wife in labour, so he phones up the hospital.."My wife is having contractions and they are only two minutes apart, what should I do?" he asks frantically.

  "Is this her first child?" asks the doctor.

  "No!" the man shouts, "this is her husband!"
* In any argument between a husband and wife, the wife will have the last word. Anything the husband says afterwards starts a fresh argument.

Often, the treatment of anaemia is begun only when the symptoms manifest...

FRANK
IRON DEFICIENCY ANAEMIA
Hb levels reduced and symptoms manifest

↑

LATENT IRON DEFICIENCY
Hb levels normal, but iron stores exhausted

↑

PRE-LATENT IRON DEFICIENCY
Hb levels normal, but iron stores depleted

↑

NEGATIVE IRON BALANCE
Body mobilises iron stores

↑

- Poor intake of iron
- Decreased bioavailability of iron
- Blood loss
- Increased requirement of iron

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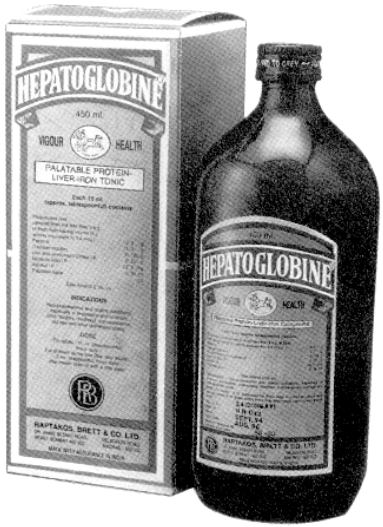

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