INSTITUTE OF PULMOCARE & RESEARCH

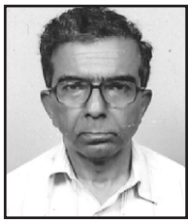
DG-8, Near Rabindra Tirtha, Action Area-I,
(On the way to New Town DPS School), New Town, Kolkata-700 156

Welcomes

ALL DELEGATES
to
PULMOCON - '18

16th All India Update On Pulmonary Medicine

FROM THE PRESIDENT'S DESK



"I am happy to welcome you at Pulmocon 2018. This annual All India update has really become a chosen and admired academic event of our fraternity from different corners of our country.

The organising committee has tried its best to make it a successful event. I would pledge you all to excuse us for shortcomings, if any and enrich us with your suggestions and positive criticisms."

Dr. Dhiman Ganguly
President
Pulmocon - '18

FROM THE SECRETARY'S DESK

"I am happy and honoured to welcome you at Pulmocon '18. This happens to be the 16th Pulmocon in a row – and it gives me pleasure to see that the popularity of the conference has been scaling high and higher over the years.

I welcome all the delegates from across the country and hope that our effort will let you carry home messages to cater better to your ailing patients.

I wish you all continue to bestow with love and affection upon us so that this institute can grow further and the future pulmocons turn better and more meaningful.

With regards and thanks."

Dr. Parthasarathi Bhattacharyya

Dr. Parthasarathi Bhattacharyya
Organising Secretary
Pulmocon - '18

PULMOCON - '18

President :
Dr. Dhiman Ganguly

Organizing Secretary :
Dr. Parthasarathi Bhattacharyya

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Dr. Saikat Nag
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Mr. Sohel Rana Shaikh
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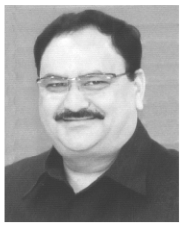
Mr. Tapas Kr. Basu
Mr. Kanai Das

राष्ट्रीय स्वास्थ्य मिशन
जगत प्रकाश नड्डा
Jagat Prakash Nadda

सत्यमेव जयते
स्वच्छ भारत
एक कदम स्वच्छता की ओर

स्वास्थ्य एवं परिवार कल्याण मंत्री
भारत सरकार
Minister of Health & Family Welfare
Government of India



MESSAGE

I am happy to know that the Institute of Pulmocare and Research (IPCR) is going to hold its 16th All India Pulmonary Update - PULMOCON 2018 from 8th – 9th September, 2018 at Kolkata.

This specialty Conference, I am informed, will have sessions addressed by the experts from the field of Pulmonary Medicine and will be attended by delegates not only from India but from across the globe.

I am hopeful that the Conference will provide a platform to exchange the expertise, experience of medical fraternity to update their knowledge and skills improving quality of health service delivery. This will also help in enriching their knowledge regarding the latest and modern technologies in the field of Pulmonary Medicine.

Felicitations to IPCR for its commendable work in research and innovations in procedures & treatment modalities of its field. I also congratulate the organizers and the participants of the Conference and extend my best wishes for its success.

(Jagat Prakash Nadda)

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No. 256A-G Dated : 5/7/18

MESSAGE

Shri Keshari Nath Tripathi, Hon'ble Governor of West Bengal is glad to learn that the Institute of Pulmocare & Research is organizing the 16th All India Pulmonary Update(Pulmocon 2018) on 8th & 9th September, 2018.

The Governor extends his felicitations and best wishes to all those associated with the Institute and congratulates them on the occasion.

SC Tewary
4.7.18
Satish Chandra Tewary

Dr. Parthasarathi Bhattacharyya,
Organizing Secretary,
Pulmocon 2018,
Institute of Pulmocare & Research,
DG-8, Near Rabindra Tirtha,
Action Area – I, New Town,
Kolkata – 700 156.

: CONTENT :

ডাঃ শম্ভুনাথ দে

জন্ম ঃ ১লা ফ্রেবুয়ারী, ১৯১৫ মৃত্যু ঃ ১৫ই এপ্রিল, ১৯৮৫

বাঙালি চিকিৎসক ও বৈজ্ঞানিক, যাঁর পরিচয় জাতির ইতিহাসে প্রায় বিলুপ্ত

স্যার আলেকজাণ্ডার ফ্লেমিং যেমন পেনিসিলিনের আবিষ্কর্তা, স্যার রোনাণ্ড রস্ যেমন ম্যালেরিয়ায় মশার ভূমিকার আবিষ্কারের জনক, তেমনি **ডাঃ শম্ভু নাথ দে কলেরার বিষ (toxin) এর আবিষ্কর্তা।**

সারা পৃথিবী যখন ফ্লেমিং, রস্ কে শ্রদ্ধা জানায় তখন নীরবে বৈপ্লবিক গবেষণা করা এই বাঙালী তথা ভারতীয় চিকিৎসক লোকচক্ষুর অন্তরালেই থেকে যান।

তাঁর মৃত্যুর ৩৩ তম বর্ষে ও জন্ম শতবর্ষে আমরা তাঁকে বিস্মৃতির অন্তরাল থেকে বাইরে সর্বসমক্ষে আনতে চাই।

তাই ২০১৮ সালে অনুষ্ঠিত আমাদের **পালমোকন - ইনস্টিটিউট অব্ পালমোকেয়ার অ্যাণ্ড রিসার্চ** এর বাৎসরিক বক্ষরোগ সংক্রান্ত সর্বভারতীয় সম্মেলনে আমরা প্রয়াত ডাঃ দে - কে আমাদের আন্তরিক শ্রদ্ধা জানানোর চেষ্টা করেছি। তাঁর নামে আমরা ২০০৯ সাল থেকে একটি বাৎসরিক স্মারক বক্তৃতার সূত্রপাত করেছি। এ বছর ঐ বক্তৃতা দিতে আসছেন ভারত বিখ্যাত বৈজ্ঞানিক ও অধ্যাপক প্রফেসর শুভেন্দ্র মোহান্তি।

এক আত্মবিস্মৃত জাতির বর্তমান গ্লানিময় পরিস্থিতির প্রেক্ষাপটে ডাঃ শম্ভুনাথ দে-র উজ্জ্বল জীবনালেখ্য আমাদের চলার প্রেরণা ও পাথেয় হোক। চিকিৎসা বিজ্ঞানে বাঙালীর অবদান আমাদের ও পরবর্ত্তী প্রজন্মকে হারিয়ে যাওয়া সম্মান পুনঃ উদ্ধারে ব্রতী করুক। হীনমন্যতার মলিনতা থেকে বাংলার ও এদেশের চিকিৎসা ব্যবস্থা ঘুরে দাঁড়াক এক নতুন প্রত্যয়ের এবং একনিষ্ঠ প্রচেষ্টার আলোয়।

–ঃ জীবনী ঃ–

১৯১৫ সালে শম্ভুনাথ দে'র জন্ম হুগলীর এক সাধারণ ব্যবসায়ী পরিবারে। ছোটবেলা থেকেই তিনি মেধাবী ছাত্র হিসাবে চিহ্নিত হন - এবং ম্যাট্রিকুলেশনের পর প্রথমে হুগলীর মহসীন কলেজ ও পরে কোলকাতা মেডিকেল কলেজ থেকে ১৯৩৯ সালে MB এবং ১৯৪২ সালে DTM (Diploma in Tropical Medicine) পাস করেন। ১৯৪৭ সাল অবধি কোলকাতা মেডিকেল কলেজে প্যাথোলজি বিভাগে ডেমন্‌স্ট্রেটর হিসাবে কাজ করার পর, তিনি লণ্ডনে PhD করার উদ্দেশ্যে যান। ফিরে আসার পর তিনি কোলকাতা মেডিকেল কলেজের প্যাথোলজি বিভাগে যোগ দেন এবং মূলত কলেরা সংক্রান্ত গবেষণায় মনোনিবেশ করেন।

সে সময়ে কলেরা ছিল একটি মহামারী বিস্তার করা রোগ এবং কলেরা সম্মন্ধে মানুষের জ্ঞান ছিল সীমিত। ১৮৮২-৮৩ সালে রবাট কচ্ (Robert Koch) কলেরার জীবানু আবিষ্কার করেন কিন্তু ঠিক কিভাবে কলেরা হয় তা একটা বিরাট প্রশ্নচিহ্নের মত থেকে যায় – কারন, রবার্ট কক্ এবং তৎপরবর্তী অন্যান্য বৈজ্ঞানিকরা এই রহস্য উদ্‌ঘাটনে ব্যর্থ হন। সাধারণ ব্যাকটেরিয়ারা যে ভাবে মানুষকে আক্রান্ত করে, ঠিক সেই হিসাবে কলেরার আক্রমণ ও রোগ সৃষ্টিকে তারা বুঝতে ও বোঝাতে পারেননি। আসলে কলেরার জীবাণু (vibrio cholerae) রোগ সৃষ্টি করে সম্পূর্ন অন্য উপায়ে - জীবানু নিঃসৃত Toxin বা বিষ অন্ত্রের উপর কাজ করে দাহ্য ঘটায়। অন্ত্রের (ইলিয়াম) এর লুপ মডেলের ব্যবহার করে **ডাঃ দে** দেখান যে কলেরা হয় ঐ বিষ বা Toxin এর জন্য। জীবানু ও তার দেহ নিঃসৃত বিষ যে এভাবে অসুখ সৃষ্টি করতে পারে এ তথ্য প্রতিষ্ঠার জনক হন **ডাঃ শম্ভুনাথ দে** । ১৯৫৯ সালে এই আবিষ্কারের পর, ১৯৬১ সালে তিনি London বিশ্ববিদ্যালয়ের D.Sc. (শরীর বিদ্যা) হন।

পরবর্তী কালে ডাঃ দে নীলরতন সরকার মেডিকেল কলেজ ও বসু বিজ্ঞান মন্দিরেও কাজ করেন। প্রভূত প্রতিকূলতার মধ্যে তাকে কাজ করতে হয়। তাঁর কাজে সাহায্য করা দূরে থাক, পদে পদে তাঁকে হেনস্থা হতে হয়। এক সময় হতাশ হয়ে এবং একরকম বাধ্য হয়ে তিনি গবেষণার কাজ বন্ধ করে দেন। একটা ছোট প্যাথলজি ল্যাবরেটরী করে এই মহান বৈজ্ঞানিককে দিন যাপন করতে হত। **জীবদ্দশায় তাঁর ভাগ্যে কোন সম্মান জোটেনি; 'মহান' এই দেশ তাঁকে কোথাও কোন রকম স্বীকৃতি দেয় নি।**

ডাঃ দের গবেষণার ফলশ্রুতিতে আরও অনেক আবিষ্কার যেমন দাস্থ তে ORS চিকিৎসা, কলেরা-টীকা সমস্তই সম্ভব হয়েছে তার আবিষ্কারের সৌজন্যে। **একাধিক বার তিনি বিদেশ থেকে নোবেল পুরষ্কারের জন্য মনোনীত হন, ১৯৭৮ সালে - তিনি নোবেল কমিটিতে বক্তব্য রাখেন।**

মহান এই বৈজ্ঞানিক **১৯৮৫ সালে লোকান্তরিত হন**। যদিও জীবদ্দশায় তিনি তার নিজের দেশে কোন সম্মান ও স্বীকৃতি পাননি, আমেরিকার ও দেশ বিদেশের গবেষকরা কিন্তু ডাঃ শম্ভুনাথ দে কে দুহাতে সম্মান সূচক অর্ঘ সাজিয়ে দিয়েছেন। তার নামে লেখা অনেক প্রবন্ধ, ছাপা হয়েছে বিদেশী বৈজ্ঞানিক পত্র-পত্রিকায়। যদিও তার মৃত্যুর দশ বছর পর **১৯৯৪ সালে কোলকাতা বিশ্ববিদ্যালয় তাঁকে সম্মানীয় D.Sc. উপাধি দেন।**

ডাঃ দের মৃত্যুর ৩৩ বছর পরে তাঁকে সম্মান জানাতে পেরে আমরা নিজেদের কৃতার্থ বলে মনে করছি। আমাদের এই ছোট্ট প্রতিষ্ঠান এই সম্মান প্রদর্শনের মধ্য দিয়ে গভীর ভাবে প্রেরণা অনুভব করছে।

হয়ত বহুশ্রুত নাটকের এই উক্তিই সত্যি - "**সত্য সেলুকাস্, কি বিচিত্র এই দেশ** !"। জাত-পাত, ধর্ম-গোষ্ঠী, রাজনীতির সদা সংকীর্নতা, মানসিক ও আত্মিক দীনতার, আত্মবিশ্বাস হীনতার অন্ধকারে এমনি হারিয়ে যান কত জ্যোতিষ্ক। মৃত্যুর ৩৩ বছর পরও আমরা তাকে কোন মরনোত্তর সম্মান দিতে পারিনি – রাজপথ দূরে থাক, একটা কানা গলি, কি একটা শিক্ষা প্রতিষ্ঠানে কোথাও তাঁর নাম নেই।

আমরা আশা করবো ডাঃ শম্ভুনাথ দে কে আমাদের সমাজ ও সরকার উপযুক্ত মূল্যায়ন করবেন এবং তার মাধ্যমে বাংলা তথা ভারতবর্ষের চিকিৎসা জগৎ - এ গভীর ও সুস্থায়ী পরিবর্তনের সূচনা হবে।

Dr. Sambhu Nath De

Born : 1st February, 1915 **Passed away : 15th April, 1985**

A great doctor and scientist from Bengal – who is forgotten in our national history.

Dr. Sambhu Nath De can be placed in the same row with Dr. Alexzander Flamings (the inventor of penicillin) and Sir Ronald Ross (the inventor of the role of mosquito in malaria). Dr. De discovered that toxins secreted from the cholera germ causes loose motion and this fact changed the face of treatment of diarrhoeal diseases. But Dr. De remains in oblivion when the world pays tribute to the other two and many more.

We wished to bring his name to light in his birth Centenary and after 33 years of his passing away. So, we initiated a memorial oration in his name in our annual update from 2009 onward. This year, Prof. Subhendra Mohanty will deliver the oration.

Let the life and work of Dr. Sambhu Nath De inspire us and make us stride to bring glory and confidence to our efforts in healthcare and research.

ABOUT DR. SAMBHUNATH DE

EARLY CAREER

Sambhu Nath De was born in Hooghly District, West Bengal, India. His father Mr Dasarathi De was a small businessman. Supported by his uncle Asutosh De, De completed the Matriculation examination with distinction that helped him to get the District scholarship as well as to pursue further education in Hooghly Mohsin College. De passed his M.B. examination in 1939 from Calcutta Medical College and completed a Diploma in Tropical Medicine (DTM) in 1942. Soon after graduation he joined Calcutta Medical College as a Demonstrator of Pathology. In 1947, De joined as a Ph.D. student under Sir Roy Cameron at the Department of Morbid Anatomy, University College Hospital Medical School, London, and obtained his Ph.D. degree in Pathology in 1949. After his return, De worked on pathogenesis of cholera and started publishing his findings. In 1955, De became the Head of Pathology and Bacteriology Division of the Calcutta Medical College, which he continued until his retirement.

CONTRIBUTIONS

De made significant contributions to our recent understanding of cholera and related diarrheal diseases. Followed by the discovery of Vibrio cholerae in 1884 by Robert Koch, many works have been carried out all over the world to answer many questions related with its pathogenesis and mode of transmission in causing outbreaks. Three of his works viz., ligated intestinal loop method for studying cholera in rabbit model; ileal loop model to demonstrate the association of some strains of E. coli with diarrhea and lastly but most importantly is his discovery of cholera toxin in 1959 in the cell-free culture filtrate of V. cholerae that stimulated a specific cellular response.

In 1959 De was the first to demonstrate that cholera bacteria secrete enterotoxin. This discovery eventually promoted research to find a treatment aimed directly at neutralizing the cholera enterotoxin. De's paper "Enterotoxicity of bacteria-free culture-filtrate of Vibrio cholerae," while initially unrecognized, today is considered a milestone in the history of cholera research. Biochemist W.E. van Heyningen, professor emeritus, University of Oxford, UK, and John R. Seal, former scientific director, National Institute of Allergy and Infectious Diseases, Bethesda, note that De's paper "deserves to go down as a classic in the history of cholera, and, indeed, as later developments have shown, in the history of cellular physiology and biochemistry."

"An experimental study of the mechanism of action of Vibrio cholerae on the intestinal mucous membrane" is De's most-cited paper, cited 340 times until August 1986. De's most-cited paper has been core to cholera research fronts for many years, especially research fronts on "E. coli and Vibrio cholerae enterotoxin: detection, characterization, and role of adherence" and "Characterization of cholera enterotoxin and other enterotoxins". As noted by John Craig, State University of New York Health Science Center at Brooklyn, De's work was truly creative and novel, and it "forever altered our concepts surrounding the pathogenesis of secretory diarrhoea."

These famous findings came out from the work he carried out at the Nilratan Sircar Medical College, Calcutta Medical College and Bose Institute, Kolkata in extremely modest laboratory settings. Using research methodology that was very simple, easy to perform and inexpensive, he set the highest standards of excellence in novel experimental design and execution.

The oral rehydration therapy (ORT) for replenishing the massive fluid loss in cholera patients, has saved innumerable lives, should be considered as a direct outcome of De's discovery of cholera toxin. His findings on exotoxins set the stage for the modern views of diseases caused by toxin producing bacteria, helped in the purification of cholera and heat-labile (LT) enterotoxins produced by V. cholerae and E. coli, respectively, and in the development of series of cholera and enterotoxigenic E. coli (in short ETEC strains) vaccines.

POST-RETIREMENT

De retired in 1973 from the Calcutta Medical College at the age of 58. After his retirement, he showed no interest in higher positions but continued his research at the Bose Institute, Calcutta. De's desire to purify the cholera toxin did not progress any further as the protein purification technology was not well established in his research settings. In 1978, the Nobel Foundation invited De to participate in the 43rd Nobel Symposium on Cholera and Related Diarrhoeas.

De died on April 15, 1985 at the age of 70. His life's ambition was to make the world a better place to live in through his dedicated selfless services in medical science.

Nobel laureate Prof. Joshua Lederberg had nominated De for the Nobel Prize more than once. Said Lederberg, **"our appreciation of De must then extend beyond the humanitarian consequences of his discovery. . . he is also an examplar and inspiration for a boldness of challenge to the established wisdom, a style of thought that should be more aggressively taught by example as well as precept."**

And yet De was never elected a fellow of any Indian academy and never received any major award. Indeed as Professor Padmanabhan Balaram pointed out in an editorial in Current Science, "De died in 1985 unhonoured and unsung in India's scientific circles. That De received no major award in India during his lifetime and our Academies did not see it fit to elect him to their Fellowships must rank as one of the most glaring omissions of our time. De's heroic story of persistence, dedication and achievement should serve as an inspiration to the many who are increasingly bewildered by the current fashion of mega projects, surrounded by fanfare and publicity and most often surprisingly little discernible scientific output."

Retrieved from "http://en.wikipedia.org/wiki/Sambhu_Nath_De"

A TRIBUTE TO THE MEMORY OF DR. SAMBHUNATH DE

Prof. A. K. Nandy

Dr. Sambhunath De worked and died in this city of Calcutta (now Kolkata), and it was in this city that he discovered the Cholera Enterotoxin - fifty years back in 1959, which as Prof. Van Heyningan of the University of Oxford, noted "deserves to go down as a classic in the history of cholera and, indeed as later developments have shown, in the history of cellular physiology and biochemistry".

This great scientist, though recognised internationally, and by the Nobel Commitee itself for his great work, remained almost unknown and neglected in his own country, in his own State, and even the city he worked in. No wonder then, when Professor P. Balaram in an Editorial in the 'Current Science' journal, wrote "De died in 1985 unhonoured and unsung in India's scientific circles", ... must rank as one of the most glaring omissions of our time. De emerges in retrospect as a moment self-effacing scientist driven by inner compulsions to grapple with a major scientific problem of the time. "De's heroic story of persistence, dedication and achievements should serve as an inspiration to many who are increasingly bewildered by the current fashion of megaprojects - surrounded by fanfare and publicity and most often, surprisingly little discernible scientific output".

Research work of Dr. S. N. De

His work on cholera started when he was the Professor of Pathology in N.R.S. Medical College. His paper published during this period in 1953 has been his most cited paper, cited 340 times until August 1986 and has been designated as a 'Citation Classic' in 1987, indeed a very rare recognition. He continued his work in Calcutta Medical College where he joined as the Director-Professor of Pathology in 1956 and also as a 'Honorary Worker' in the Bose Institute. His work started getting keen attention from the international scientific community, as was exemplified from the words of Dr. John Craig of the State University of New York : "De's work was truly creative and novel, and forever altered our concepts surrounding the pathogenesis of secretory diarrhoea".

By 1960, De established the existence of "CHOLERA EXOTOXIN". In his own words "Vibrio Cholerae has now been promoted to the rank of few exotoxin-producing bacteria. Diphtheria exotoxin was discovered within 4 years of discovery of the bacillus, Tetanus exotoxin within 6 years. It has taken 75 years for cholera exotoxin to be discovered in 1959, after organism was discovered by Robert Koch in 1884" (B.C.Roy Oration).

De wanted to continue with his research for further purification of the toxin ultimately develop that he discovered, and ultimately develop a vaccine against cholera. But, unfortunately various constrains and lack of support for necessary facilities forced him to limit himself. He stated - "by 1963-64, I was forced to discontinue my work and lost all interest in cholera" (B.C.Roy Oration).

But his unfinished work was picked up by scientists abroad in few years' time, when they noted its implications. Extensive work was started on the foundation laid by De; its wider applications in a variety of investigations were also worked out in the proceding years. Applause started reaching him from all corners of the world except his own country. Noble Laureate Professor Joshua Lederberg noted, " ... his findings on exotoxins set the stage for modern views of diseases caused by the toxin-producing bacteria, ... helped in purification of the cholera and E.Coli enterotoxins, ... and in the development of cholera and E.Coli vaccines". He had nominated De for the Nobel Prize more than once.

The Nobel Foundation invited De in 1978 to participate in the 43rd Noble Symposium on "Cholera and Related Diarrhoes". He was highly applauded there by all present there (see letter). Noble Prize seened to be a possibility. But Lady Luck disappointed hid this time also.

He died a brooken-hearted man on April 15, 1985 from Hepatitis-B infection followed by hepatic coma.

The Indian scientific community woke up from its 'Kumbha-Karna Sleep' at long last "Current Science", the pioneer Indian scientific journal brought out a "Special issue on S.N.De and cholera Enterotoxin" in 1990 with contributors from all over the world. This was for the first time the prestigeous journal brought out a special issue on a particular scientist.

IBM Nordic Education Center
FACK 181 20 LIDINGÖ

10th August

My dear Achintyaji

The lecture is over. What a relief! — Congratulated by at least three people as the best lecture in the symposium — all stunned — no questioning. In the workshops — I am keeping silent.

But I do not think this means anything more. However enjoying good and nice holiday under fine environment and [illegible] all conveniences including these letters free ~~[illegible]~~ this afternoon — Boat tour / tomorrow afternoon — hotel — day after tomorrow — Carlisle. 23rd BA 021 London [illegible] [illegible]।

Tulu came to Heathrow — he did not get my letter. Good care in his house. Little Anirban became [illegible]. So has been Diana. Dilip Dipali very friendly. [illegible] — [illegible] Tulu and Dilip — both changed my shabby dress! — Tulu gave a Mack, Dilip a suit — all advise me to enjoy life!

Hope everything is O.K.

[illegible]

IBM

A letter of Dr. Sambhu Nath De to his son-in-law: to display his handwriting

PULMOCON '18

26th and 27th August, 2017

Venue : CII-Suresh Neotia Centre, City Centre, Sector-I, Salt Lake, Kolkata-64

Organised by : **Institute of Pulmocare & Research,** Kolkata.

Acharya Prafulla Chandra Roy Memorial Award, 2018

Bengal has given birth to many great people – Acharya Prafulla Chandra Roy had been one amongst them.

Acharya P C Roy dreamt of self reliance and promotion of self sufficiency in all fields through gaining excellence. The great scientist of his days was a great entrepreneur too. He established Bengal Chemical, the 1st Indian entrepreneurship in Chemical and Pharmaceutical industry over 103 years ago. That was, infact, the beginning of pharmaceuticals industry in India.

The beauty of his personality was in simple living but in extraordinarily thinking, in noble ambitions and the ability to withstand odds. They were admixed with a generosity and extreme love and affection for his students and the countrymen. A story goes as that once he was very angry with a student who spent one penny extra for his professors tiffin but on the same day of the incidence, the apparently miger professor donated ` 50,000/- to the National Congress Party for draught-relief in North India.

To commemorate him and to instill the sprit of innovations amongst ourselves we have incorporated an award in our annual Pulmocon from the year, 2009. The 1st Acharya P C Roy memorial award was offered to Dr. Sujay Guha and the next was confered to famous scientist and innovator Dr. Tinku Acharyya who innovated the digital photography technology with over 150 US patents to his credit. In 2011 we had felicitated Prof. Indu Bhusan Chatterjee for his distinguished contribution in research and innovations in 2012, similarly, in subsequent years doyens in research and innovations have been conferred with this award. In Pulmocon 2018 we are happy to award Dr. Lalit Varshney.

Dr. Lalit Varshney joined Isotope Division, Bhabha Atomic Research centre(BARC) in 1982 after completing 25th batch training school of BARC. He obtained Masters in Science(Chemistry) degree from University of Delhi in 1981and Ph.D from Mumbai University. He is presently, Head Radiation Technology Development Division, BARC; Head School on Applications of Radioisotopes and Radiation Technology, Global Centre for Nuclear Energy Partenership(GCNEP), Delhi and Prof. (Chemistry) Homi Bhabha National Institute, Mumbai. Dr. Varshney started his carrier with research and development on radiation effects studies on Polymers and Pharmaceuticals. Initial years deep investigations on how radiations interacts with drug molecules led him to complete his Ph.D on radiation effects on antibiotic, Chloramphenicol. The findings disclosed that how some drugs are highly resistant to radiation degradation and some susceptible. Thereafter, extensive work on polymers mainly using Radiation Technology promoted use of the technology for sterilization of medical devices in India and developing countries. Presently the country has 15 such facilities engaged in sterilizing medical devices using Gamma Radiation and many more are under construction. Dr. Varshney pioneer work on radiation formation of hydrogels led to the development of technology for indigenous production of hydrogel wound dressing. The dressing is useful for all kind of external wounds including that of burn, ulcers, diabetic ulcers, animals bites and other difficult to heal wounds. The dressing in the form of 5 mm sheet has more than 90% water and water soluble polymers. The technology was transferred to four local companies for wider use. Big cities in our country produce billions of liters of sewage which is treated at Sewage treatment plants. As a by product, thousands of tons of waste sludge which is highly infectious is generated. In absence of non availability of suitable technology, the waste is being disposed in unregulated manner. Dr. Varshney conceptualized and developed a Radiation Technology to hygienise this waste and convert it to bio-fertilizer. Highly penetrating Gamma Radiation kills all the pathogens in the waste, reduce smell and allows inoculation of useful microorganisms to convert it to Bio-fertilizer. The process not only provides organic carbon but also other micro and macro nutrients to soil. It becomes a better product than organic compost. Based on this technology, a 100 tons per day capacity facility has come up in Ahmedabad city and second one is coming up in Indore. Other metropolitan cities are also showing interest to put up such facilities to recycle this waste to resource specially under Swachh Bharat Abhiyan and smart cities. This would finaly help in achieving cleaner rivers and other water bodies. Some of the states in our country and other countries have severe problem of Arsenic contamination. Dr. Varshney and his young team has recently developed a novel technology to selectively fish out Arsenic from Ground Water. The technology is being tested in some areas of Chattisgarh. Using Radiation technology, some ionic polymers are grafted on cellulose which makes permanent attachment of these ionic polymers on to the substrate. This makes reusability of these matrix and ultimately reduction in cost of the treatment. Technologies developed by Dr. Varshney could find several other applications. Dr. Varshneyis currently engaged in the development of advanced materials for Healthcare, industrial and environmental applications using radiation technology. Some of these include, X-ray shield materials, volatile organic compound sensors(VOC) for metabolic disorders, high energy beams for degradation of emerging pollutants, antibacterial cottons and many more. The VOC sensors could rapidly detect minuscule quantity of volatiles released in breath which could help in early detection of diseases. He hasmore than 125international publications, three patents and 4 technology transfers to his credit. Dr. Varshney is also on the panel of International Atomic Energy Experts and has undertaken several assignments for developing countries. Dr Varshney is recipient of prestigious National Award for Technology Innovation in Polymeric Materials, 2015, Indian Nuclear Society award 2004 and BARC Technical Excellence award-2003 and recently DAE Group Achievement Award 2016.

He can be contacted at 022-25593745, email: lalitv@barc.gov.in .

Cough hypersensitivity syndrome

DR. DHIMAN GANGULY

Cough is a part of host defence mechanism, but dysregulated cough is a distressing symptoms. There is enough epidemiological data to show that this is the single most common presenting symptom for seeking medical help. Traditionally, cough has always beenconsidered a consequence of chronic respiratory disease – but a growing concern among physicians in recent years has been that, even after a thorough work up some coughs remained unexplained and also there is a group of patients – where even with an etiological diagnosis and appropriate therapy the cough refuses to settle.

To explain these two groups ofpatients, a novel paradigm was suggested in 2010. Subsequently in 2014 the European Respiratory Society Task force clearly endorsed cough hypersensitivity syndrome as a valid and useful concept.

It now seems chronic cough is also a neurological phenomenon either on its own or most certainly with a trigger of respiratory disease. There is very good published data to show that there is a neuro immunological explanation for a hyperactive cough reflex both at sensory and central phases of the reflex.
Hopefully with clearer understanding of the pathogenic mechanisms newer pharmacotherapy to control cough will evolve in near future.

❖❖❖

Quality Control of Spirometry and DLCO

DR. KAUSHIK SAHA

Assistant Professor Chest Medicine, Burdwan Medical College

Spirometry and DLCO – both are sensitive indices of pulmonary disease progression or response to therapeutic interventions.Quality Control covers the operational techniques and activities that are used to fulfil requirements for quality. It is used to verify that all measured parameters are of acceptable quality and that they are complete and correct.

Quality control of spirometry machine done by i) Calibration check with 3L calibration syringe daily or prior to patient testing, ii) Linearity tests with a 3L calibration syringe weekly, iii) Biological control monthly and iv) Leak test weekly

Quality control of D_{LCO} machine done by i) Flow analyser zeroing before each test, ii) Gas analyser zeroing before/after each test, iii) Volume calibration check daily, iv) Biologic control weekly, v) Calibration syringe D_{LCO} check weekly, vi) Calibration syringe leak test monthly, vii) Linearity check monthly

Variability in some lung function measurements can be very high (>10%). Therefore, unless high-quality measurements, excellent staff training and high-quality assurance processes are needed. The importance of stringent quality control programmes is becoming more apparent in recent times.

PULMOCON-'18

Date: 8th and 9th September, 2018

Theme: Lung Diseases Beyond Lungs

PROGRAMME SCHEDULE (Hall - A)

DAY 1 : 8th September, 2018

Time and Topics	Faculty	Chairperson / co-ordinator
09:30 am - 01:30 pm		
Workshop : Essentials of critical area for physicians -		
• Managing Sepsis in ICU	Dr. Dhruv Chaudhry	Dr. Subhas Todi
Tea to be served at hall		
• Data Interpretation in ICU : Biochemistry/Hematology/Coagulation	Dr. Subhas Todi	
• Basics Hemodynamic monitoring in ICU	Dr. Rahul Guha Biswas	
• Basics of Mechanical Ventilation :	Dr. Dhruv Chaudhry	
• Judicious antibiotic use in ICU	Dr. Subhas Todi	
• Imaging in ICU : Chest Xray/USG	Dr. Susruta Bandyopadhyay	
• Nutrition in ICU	Dr. Saswati Sinha	
• Open forum : Town Hall type discussion (JAMA session : Just Ask Me Anything : with all faculty)		
01:30 pm - 02:30 pm	**Lunch Break**	
02:30 pm - 03:30 pm		
National Pulmo Quiz (Elimination round)	**Quiz Master :** Dr. Sushmita Roychowdhury	**Assist :** Dr. Arindam Mukherjee
03:30 pm - 05:15 pm COPD Symposium :		
COPD : the latest view points and the carry home message:	Dr. Raja Dhar /	
• COPD frequent exacerbations	Dr. P. S. Bhattacharyya	Dr. Rupak Ghosh
• Beta blockers in COPD	Dr. Pawan Agarwal	Dr. Pranab Mondal
• Prescribing ICS in COPD	Dr. Subhasish Ghosh	Dr. Rajeev Ranjan
• Future inhalation therapy in COPD	Dr. Angira Dasgupta	
• COPD mimics	Dr. Ajoy Sarkar	
05:15 pm - 05:30 pm	**Tea Break**	
05:30pm - 06:50 pm		
Lung diseases manifesting outside lungs :		
• Clue for Lung malignancy outside lungs	Dr. Sonia Dalal	Dr. Dhiman Ganguly
• When diseased abdomen reminds a lung disease	Dr. Hindol Dasgupta	Dr. Parthasarathi Bhattacharyya
• When a lung disease licks joints and bones	Dr. M L Gupta	
• When skin changes gives a clue	Dr. Dhruv Chaudhry	
06:50 pm - 08:00 pm		
• **The Dr. S N De Memorial Oration :**		Prof. Subhendra Mohanty
• **The Award giving ceremony : The Acharya P C Roy award will be conferred upon**		Dr. Lalit Varshney
• **The life time achievement award will be conferred upon**		Prof. Siddhartha Majumder
• **The Inauguration program**		
08:00 pm - 09:00 pm	**Dinner with cultural program**	

DAY 1 (Hall - B)

Time and Topics	Faculty	Chairperson / co-ordinator
10.30 am - 12.00 noon (15 minutes each+15 minutes for discussion) **Symposium : Therapeutic issues**		
● Long term azithromycin: beyond 3 years ?	Dr. Shelly Shamim	Dr. Monotosh Khanra
● Systemic effects of COPD	Dr. Ashok Sengupta	Dr. Sumit Tapadar
● Management of refractory wheeze	Dr. Indranil Haldar	
● Preventing drug resistant infection	Dr. Asutosh Ghosh	
● GERD in respiratory disease	Dr. Angshuman Mukherjee (Jr.)	
12:00 noon - 01:30 pm (15 minutes each + 30 minutes for discussion) **Symposium : Ethics and others**		
● Consent for treatment and research : when and how ?	Dr. Tias Sen	Dr. Saibal Moitra
● Reducing mistakes and omissions in prescriptions	Dr. Dhruv Chaudhry	Dr. P S Mondal
● Social media : expectations and manipulations	Dr. Sumit Sengupta	Dr. Jahar Ghosh
● Securing your IP : be an innovator	Advocate B Sarkar	
01:30 pm - 02:30 pm **Meet the professor: difficulties in treatment of tuberculosis** (Packet lunch at the hall for the participants)		Prof. M L Gupta
01:30 pm - 02:30 pm	**Lunch break**	
02:30 pm - 05:00 pm **Workshop : Understanding Spirometry with DLCO**		
● **Basics of spirometry:** (30 minutes+15 minutes for discussion)	Dr. Ritabrata Mitra	Dr. Ritabrata Mitra
● **Basics of DLCO & diagnostic algorithm** (20 minutes+10 minutes for discussion)	Dr. Swapnendu Mishra	
● **Quality control of spirometry and DLCO** (15 minutes+5 minutes for discussion)	Dr. Koushik Saha	
● **Advanced concept in spirometry** (25 minutes+10 minutes for discussion)	Dr. Ritabrata Mitra	
● **Spirometry: past, present and prospect** (15 minutes+5 minutes for discussion)	Dr. Parthasarathi Bhattacharyya	
05:00 pm to 05:45 pm	**Preparation of poster presentation for day 2**	

PROGRAMME SCHEDULE (Hall - A)

DAY 2 : 9th September, 2018

Time and Topics	Faculty	Chairperson / co-ordinator
10:00 am - 10.40 am (15 minutes each + 10 minutes for discussion) **Pulmonary interventions**		
● Bronchoscopy in Hemoptysis - when and why?	Dr. Arindam Mukherjee	Dr. Sanjoy Gupta
● Pleuroscopy - diagnostic and therapeutic role	Dr. Ranjan Das	Dr. Sourabh Maji
10.40 am to 11.20 am **Difficulties in making a diagnosis**		
● The heart failure in lung diseases	Dr. Ranjan Sharma	Dr. Nandini Biswas
● Echocardiography for pulmonologist	Dr. Aniruddha Dey	Dr. Debanu Ghosh Roy
11:20 am - 11:30 am	**Tea Break**	
11:30 am - 12:30 pm **National Pulmo-Quiz** (Final round)	**Quiz Master:** Dr. Sushmita Roychowdhury	**Assist :** Dr. Arindam Mukherjee

Time and Topics	Faculty	Chairperson / co-ordinator
12.30 pm - 12.50 pm **Return from death bed: a real life story**	Dr. Suranjan Mukherjee Dr. Soham Majumdar	Dr. Suresh Ramasubbam Dr. Ratan Ghosh
12:50 pm - 01:10 pm **"Igniting innovation in young minds"**	Dr. L Varshney	Dr. P S Bhattacharyya Dr. Subir Banerjee
01:10 pm - 02:00 pm	**Lunch Break**	
02:00 pm - 03:00 pm (15minutes each + 15 minutes for discussion) **Asthma symposium**		
• Airway remodeling and asthma: where do we stand ? • Severe asthma: diagnosis and answers • Cough hypersentivity syndrome	Dr. Aloke Gopal Ghoshal Dr. Sonia Dalal Dr. Dhiman Ganguly	Dr. Somnath Mitra Dr. Soumya Das Dr. Tarashankar Ghosh
03:00 pm - 04:00pm (15 minutes each + 15 minutes for discussion) **Symposium : Difficulties for diagnosis and treatment for Tuberculosis**		
• NTM disease: suspicion and diagnosis • TB molecular diagnosis: pro and cones • Treating TB in diabetes, pregnancy, and chronic renal failure	Dr. M L Gupta Dr. Supriyo Sarkar Dr. Ansuman Mukherjee (Sr)	Dr. Shankar Saha Dr. Anita Mohanty Dr. A Pramanik
04:00 pm - 04:10 pm	**Tea Break**	
04:10 pm - 04:55 pm **Clinical practice**		
• Non resolving pneumonia (15 minutes + 5 minutes for discussion) • Approach to a clinical problems from chest X-rays (20 minutes + 5 minutes for discussion)	Dr. P P Roy Dr. Somnath Kundu	Dr. Arnab Maji Dr. Arunava Dutta Chowdhury
04:55 pm - 05:00 pm	**QUIZ and RESEARCH (POSTER) award and Valedictory session**	
HALL-B		
10:30 am - 12:10 noon **Poster and platform presentation**	**Judges :**	Dr. Angira Dasgupta Dr. Rupak Ghosh Dr. Joydip Deb
12.10 noon - 01.30 pm (15minutes each + 20 minutes for discussion) **Symposium : Medico-legal issues in practice**		
• Medicolegal problem faced by doctors in day to day practice • How to write a response to a complaint and how to face an ethical enquiry • Telephone calls and medical practice - how far and how much? • Media, doctor, medico-legal perspective	Dr. B Sukul Advocate Binota Roy Dr. Ansuman Mukherjee Dr. Krishnagshu Roy	Dr. Saibal Ghosh Dr. Rejaul Karim Dr. Samaresh Saha
01.30 pm - 02:15 pm **Meet the professor: a physician needs a philosophical outlook**	Dr. Dhiman Ganguly	Packet lunch at Hall (only for the participants)
01:30 pm - 02:15 pm	**Lunch Break**	
02.15 pm - 03.15 pm **Symposium : Sleep sessions**		
• Clinical approach to snoring • Interpretation of PSG report • ENT evaluation in sleep disorders	Dr. D J Roy Dr. Arup Haldar Dr. Dipankar Dutta	Dr. Saikat Nag Dr. Tanveer Reja Dr. R. Mishra
03.15 pm - 04.35 pm **Symposium : DPLD**		
• Classification and evaluation • HP : presentation and diagnosis • Treating DPLD: when anti fibrotic and when steroid ? • How to follow up a DPLD patient	Dr. Sujan Bardhan Dr. Anirban Sarkar Dr. Debabani Biswas Dr. Sourabh Maji	Dr. Subhadeep Mukherjee Dr. Saikat Nag

ABSTRACT

Improving diagnosis and treatment of lung diseases using bioinformatics and systems biology approaches

Sreyashi Majumdar[1], Saran N[1], Abhirupa Ghosh[1], Tanmoy Jana[1], Krishnendu Banerjee[1], Debangana Chakravorty[1] and Sudipto Saha[1]*

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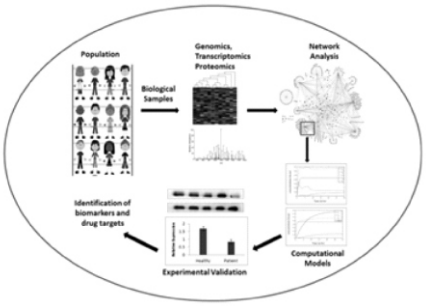
Lung is the primary organ of the respiratory system and the lung tissues can be affected by a number of diseases including tuberculosis, asthma and lung cancer. Bioinformatics and systems biology approaches are used in our lab including database development, machine learning, high throughput data analysis obtained across different omics platform, network analysis and generation of predictive computational models to understand the disease pathway biology to precision therapeutics. Currently, the lab is emphasizing on airway obstructive diseases (like Asthma, COPD), infectious disease (like tuberculosis) and malignant disease (like Lung Cancer). Airway obstructive diseases often tend to exhibit overlapping symptoms. So, our lab uses plasma proteomics based approach, cytokine profiling and network analyses for biomarker identification in asthma and their comparison with COPD patient profiles. A dedicated database (DAAB) was designed as a repositoryfor probablemolecular biomarkers of atopic asthma and other allergic diseases.Emergence of multi drug resistance (MDR-TB) and extensive drug resistance (XDR-TB) leads to complication in TB treatment. Resistance to Fluoroquinolones leads to transition from multidrug resistance to extensively drug resistance. Mycobacterial fluoroquinolone pentapeptide protein (MfpA) along with a small GTPase protein MfpB, upon expression in Mycobacteria confers low level resistance to fluoroquinolones. Our lab also focusses on screening novel small chemical modulators to disrupt MfpA-MfpB interaction. A database DRAGdb have also been created with mutations in 12drug resistance associated genes related to 6 anti-tuberculosis drugs across organisms including *Mycobacterium tuberculosis*, ESKAPE bacteria like *Staphylococcus aureus, Klebsiella pneumoniae* and other bacterial species.Recently, specific protein-protein interaction (PPI) interfaces are being targeted by small chemical modulators for drug discovery. So, specific PPI and small chemical modulators for PPIs including Myc-Max, Mdm2-p53 and Bcl2-Bax are being studied in our lab. Dedicated databases and servers (PPIMdb, PPIMpred and PPIM-IC50Pred) have been designed for high-throughput screening of small molecules targeting protein–protein interaction in cancer. Recently, the lab has initiated study on regulatory networks in pluripotent embryonic stem cells and their correlation with lung cancer stem cells.The overall approach of the lab is to employ computational tools and systems biology approach for a better understanding of disease pathobiology and identification of clinical biomarkers, drug targets and potential lead molecules for different lung diseases.

Graphical abstract:



HILAR MASS: A RARE THORACIC MANIFESTATIONS OF WEGENER'S GRANULOMATOSIS

Dr. Bijaya Ku Meher, Dr. Pragyan Rout, Prof. Dr. M R Pattnaik, Assoc. Prof. Dr. T Mohanty

Introduction:

Pulmonary manifestations of Wegener's granulomatosis (WG) range from asymptomatic nodules, fleeting infiltrates to alveolar haemorrhage.[1] A mediastinal mass was considered an unlikely feature of WG and its presence was regarded as an alternative diagnosis.[2]

Case Report:

A 48 year hindu female was admitted to our hospital with complains of dry cough, intermittent fever, shortness of breath and recurrent scanty hemoptysis since 4 months. On examination, vital signs were stable except pallor and chest findings were unremarkable. At hospital chest radiography showed a right perihilar mass and chest computed tomography (CT) disclosed a cavitating mass in the right middle lobe abutting the great vessels. Besides plenty of RBCs in Urine microscopic examination, all other routine blood tests were within normal limit. Bronchoscopy revealed ulcerative tracheobronchitis with inflammation. The antineutrophil cytoplasmic antibody was positive (cytoplasmic type) and other autoantibody tests were negative. Thus, we diagnosed it as a case of granulomatosis with polyangitis (GPA, Wegner's granulomatosis). The patient was initiated treatment with corticosteroids and cyclophosphamide.

Discussion:

Granulomatosis with polyangitis is characterized by necrotizing granulomatous inflammation of upper and lower respiratory tracts, glomerulonephritis, and necrotizing vasculitis of the lungs and a variety of systemic organs and tissues. Tracheobronchial involvement with GPA has several manifestations, including tracheal and bronchial stenosis, mass lesions. Tracheal stenosis is frequently reported, however, bronchial ulcerations are less common. So it is important to recognise the presenting features of WG, because early treatment is essential for a favourable outcome.

References:

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Pleural effusion in Silicosis: A rare case report

Dr. Pragyan Rout, Dr. Bijaya Ku Meher, Prof. Dr. M R Pattnaik, Assoc. Prof. Dr. T Mohanty

Silicosis is a diffuse pulmonary interstitial disease characterized by a fibrotic response in lung parenchyma caused by continual inhalation of crystalline silica (SiO2).[1] It is one of the primary pneumoconiosis diseases caused by inhalation of mineral dust and its presentation, clinical course, and severity are variable. Several forms of the disease can be identified from the clinical, radiological and functional data. These are classified as chronic silicosis (simple, complicated, and interstitial pulmonary fibrosis), accelerated silicosis and acute silicosis.[1]

Various pleural involvements such as pleural thickening and progressive multifocal fibrosis (PMF) associated pleural invaginations are well-recognized complications associated with silicosis, particularly advanced pulmonary silicosis. However, pleural effusion is not a well-recognized finding in patients with silicosis and extremely rare presentation. To our best knowledge, there have been only few cases of silicosis with pleural effusion reported in medical literature.[2] Herein, we describe a case of a 60-year-old man, who presented with shortness of breath and Left sided pleural effusion. The patient had undergone extensive workup and was diagnosed with pulmonary silicosis.

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PULMONARY HYDATID CYST MISDIAGNOSED AS LUNG METASTASIS: A CASE REPORT

25years male presented with dry cough & fever for 6 months, bilateral chest pain for 3 months, malaise for 2 months. Patient had history of dog exposure at home. Initial chest x-ray showed bilateral well defined homogeneous coin leisons. USG thorax done which revealed bilateral cystic lung lesions. CT thorax reported as bilateral cystic lung lesions HU(1-10) with membrane like structure suggestive of lung hydatid cyst. Serum IgG for echinococcusgranulosus was positive. Finally diagnosis of bilateral pulmonary hydatid cyst was established & patient was advised oral albendazole 400mg BD for 3 months and surgical excision of lesions.

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Sensitivity of Cartridge Based Nucleic acid Amplification Test (CBNAAT) in diagnosing Extra-pulmonary tubercular cervical lymphadenopathy (EPTCL) & its correlation with cytological picture & Acid-FastBacilli (AFB) staining

Dr. Sutapa Das*, Prof. (Dr.) Dipanwita Nag, Dr. Shinjan Patra*#**

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Introduction/Background:
Cervical Lymphadenopathy is a commonly encountered clinical problem which has a multitude of causes, among them Tuberculosis is the most important in a developing country like us. Tuberculous lymphadenitis is the most common site of extrapulmonary tuberculosis. The hallmark of tuberculosis histologically is the presence of caseating necrosis associated with epithelioid cell granuloma. Cartridge Based Nucleic acid Amplification Test (CBNAAT)has emerged to enable clinicians for early recognition of M. tuberculosis from a variety of extra-pulmonary clinical samples, with very good positive predictive value.

Methods/Objectives:
Our main objective in this study was to detect the sensitivity of the CBNAAT technique to diagnose EPTCL by Fine Needle Aspiration (FNA) technique. We have performed FNA on suspected EPTCL patients & divided the aspirate into 3 parts; One for depicting the cytology, another for AFB staining & the remaining for CBNAAT technique. The patients whose sample came negative for tuberculosis with all these 3 methods were thoroughly followed-up with other clinical methods like Mantoux test or culture for tubercular bacilli from re-FNA sample from the lymph-node. Finally the correlation between all three described techniques had been done.

Results:
A total of 82 patients of EPTCL were chosen for FNA technique where lymphnode can be aspirated in 69 cases only. Among them 52 samples (75.36%) were detected tuberculosis by CBNAAT technique; where only 7 of them (10.14%) reported AFB bacilli & 11 of them (15.94%) had classical caseating granuloma. No patients with CBNAAT negativity (17 cases out of 69: 24.64%) had been detected as AFB positive or with classical granuloma. Among these only 2 patientswere subsequently diagnosed as tuberculosis by culture method.
Conclusion: From this study we have substantially proved the efficacy of CBNAAT technique for early diagnosis of EPTCL beyond doubt & most importantly it can be substituted for culture technique which is very time-taking & cumbersome also. So we are recommending sending every lymph node sample for CBNAAT to diagnose EPTCL early &commencing efficient treatment.

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Classification of Normal, Asthma and COPD using Multichannel Lung Sound Signals

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Background:
Lung sound (LS) auscultation is a primary noninvasive procedure to appreciate the respiratory pathologies. However, this process depends on the hearing ability of the physicians, which varies among them. In the literature of LS analysis, no study has been performed as yet for classification of normal, asthma, and COPD in a single platform using LS signals, may be due to their several common manifestations. There is an urgency to develop a simple, noninvasive, and cost-effective diagnostic system to diagnose both asthma and COPD in a single stage.

Methods:
In this study, a total of 60 subjects (20 normal, 20 asthma, and 20 COPD) participated. Lung sound signals (LSSs)were acquired from four different positions, i.e., right apical, right posterior basal, left apical, and left posterior basal, using a newly developed 4-channel data acquisition system. Next, LSSs were preprocessed,andtotal 240 LS cycles were extracted for further analysis. Power spectral density (PSD) was estimated using Welch's method of each LS cycle and then decomposed into uniform subbands. Four statistical features were extracted from each subband and fed to the support vector machine (SVM) classifier to classify the normal, asthma, and COPD subjects.

Results:
The classification performances for all possible channel combinations were estimated to investigate the effectiveness of the recording positions. We achieved the maximum classification accuracy of 70%, the sensitivity of COPD class of 75%, the sensitivity of asthma class of 55%, and the specificity of 80% for the combination of right apical and both the posterior basal segments.
Conclusion: In this study, we have segregated normal, asthma, and COPD subjects based on the changes in their posterior LSSs where the presence of wheeze is not a requirement. The proposed approach provides reasonable classification performance, where the classification accuracy is 26.67% more than the empirical chance level, despite the several common manifestations of asthma and COPD.

Estimation of Respiration signal from Photo plethysmography (PPG) signal

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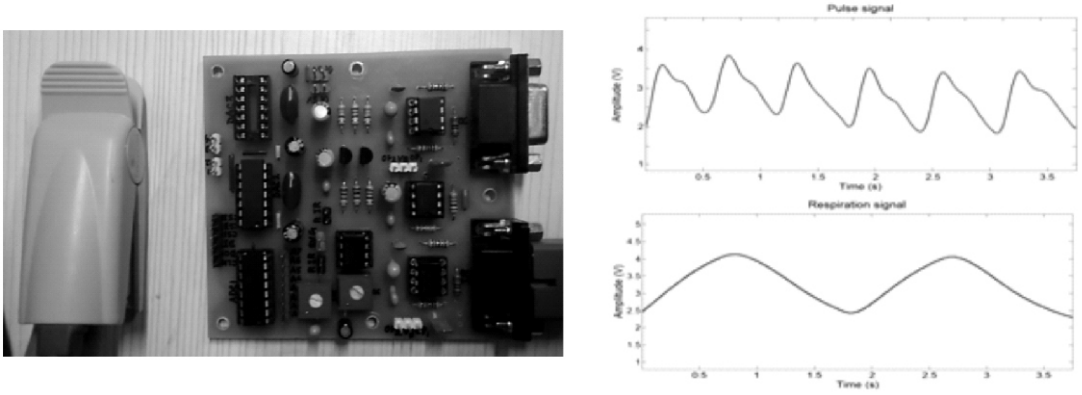
Background:
Heart rate, Respiratory rate and Blood Oxygen Saturation (SpO2) are three most significant vital parameters of the body needed to be continuously monitored since in case of any abnormality or diseases they get altered. Nowadays, the commercially available pulse oximeters only provide pulse rate and blood oxygen saturation. In this work we propose an algorithm for estimation of respiration signal from the fingertip pulse signal and further calculation of respiratory rate, pulse rate and SpO2 from a single pulse sensor. This technique can be very useful for continuous monitoring of patients with better comfort and lesser number of wired connections.

Methods:
For developing the proposed system we will be using Nelcorr pulse oximeter probeand ATMEGA 328P Microcontroller unit for data acquisition (Figure 1for data analysis and heart rate, SpO2 and respiratory rate calculation and display unit for displaying the patient's pulse rate respiratory rate and SPO2value.



Results:Fig. 1. (a) Developed Hardware (b) Acquired respiration and pulse signal

Conclusion: In this work we have developed an algorithm for estimation of respiration signal from pulse signal which eliminates the need of using an extra sensor for monitoring respiration. Hence the vital parameters like respiratory rate, heart rate and SpO2 can be monitored from a single fingertip sensor.

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Comparative Study between features extracted from Respiration Signal and ECG Derived Respiration Signal

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Background:
Chronic obstructive pulmonary disease (COPD) becomes a global cause of morbidity and mortality. According to WHO, COPD is the 3rd leading cause of death and a major cause of disability worldwide. It is a type of progressive lung disease which is characterized by airway obstruction along with airflow limitation. Changes in pulmonary mechanism thus affect the respiration pattern of COPD from that of normal subject. Various ECG changes are also reported by researchers in case of COPD patients. Even the respiration signal derived from the ECG i.e. ECG derived respiration (EDR) signal also has these effects induced in it. Features extracted from both this signals may help in establish the effectiveness of EDR signal for COPD classification from normal population.

Methods:
A group of 15 COPD patients and 10 normal subjects were selected based on their chest X-ray and pulmonary function test after they were examined by expert pulmonologists. ECG and respiration signals were collected for 300 seconds using Biopac MP-45 data acquisition device from the subjects while they were resting in supine condition. Raw signals were denoised and normalized and features were extracted from both signals for each individual. Area ratio, time ratio and respiration rate were calculated for five randomly selected cycles and their mean values were taken as features. A comparative study on the derived features was performed afterwards to classify COPD subjects from normal population.

Results:
Respiration signal of 15 COPD patients (mean age 64.5±6.3 years) and 10 normal subjects (49.8±3.4 years) were taken for this study. It has been observed that area ratio (1.75±0.64), time ratio (1.85±0.59) of COPD calculated from respiration signal are much different from the area ratio (0.92±0.08), and time ratio (0.92±0.13) of normal subjects calculated from the same signal. Similarly, the area ratio (1.52±0.31) and time ratio (1.57±0.26) of the COPD patients calculated from the EDR signal are higher than the area ratio (0.94±0.13) and time ratio (0.88±0.14) of normal subjects. Whereas, the respiration rate calculated from both the signals did not vary much for COPD patients and normal subjects. Individual feature calculated from both EDR and respiration signal for both the data groups are not statistically significant (>0.1).

Conclusion:

Extracted features (like area ratio and time ratio) using both the signals showed much difference between the values measured for COPD patients and normal subjects. But each individual feature calculated from respiration signal does not vary much from that of the same feature calculated using EDR signal. This may establish the fact that EDR signal also can be used for classification of COPD from normal population. Study on more number of subjects for further validation is needed for better result.

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Does FEF_{25-75} correlate with FVC to indicate the course of DPLD?

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Background:
The course of diffuse parenchymal lung disease (DPLD) is marked by change in FVC. FEF_{25-75} has also been recently claimed to be an independent marker of DPLD in relation to differentiating it from obstructive airway disease. It will therefore, be worthwhile to evaluate the change in FEF_{25-75} in comparison to FVC longitudinally in DPLD patients on therapy.

Materials and methods:
Random selection of DPLD patient's serial spirometry from the archive is done with follow up cases of improvement and worsening. The spirometry variables were charted and FEF_{25-75} is correlated with the change in FVC for both improved and worse subjects.

Result:
196 patients (Mean age= 58.9±11.3 years; Male: Female= 94:102) were evaluated with 97 of them showing improvement and 99 showing worsening after 400±463 days of treatment. The change in percentage predicted value of FVC for the improved and worse groups were 7.33± 0.98 (p value= 0.005) and 7.85± 1.42 (p value=0.002) respectively. The percentage predicted value of FEF_{25-75} also changed parallelly; the corresponding change of FEF_{25-75} in improvement and deterioration were 4.58± 3.45 (p value= 0.11) and 4.50±0.02 (p value=0.4). The change in FEF_{25-75} correlates with FVC in both the situations (p value= 0.01 and 0.02 respectively).

Conclusion:
The FEF_{25-75} correlates with FVC for improvement and deterioration; however the relation does not appear strong. Further study is needed in this field.

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Can spirometry differentiate between predominant fibrosis vs inflammatory variety of DPLD: a pilot observation

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Background:
The patients of diffused parenchymal lung disease (DPLD) show varying degree of fibrosis and inflammation in lung parenchyma. The predominant fibrotic form of DPLD is marked by honeycombing while the predominant inflammation is marked by ground glass opacity (GGO) as apparent from HRCT chest. FVC and recently postulated FEF_{25-75} are thought to represent the course of DPLD. Their association separately with fibrotic and inflammatory groups that needs to be looked for.

Materials and method:
The retrospective spirometry data of DPLD patients as per the predominant fibrosis and inflammation determined by HRCT chest were analysed and compared with normal subjects. The HRCT features were determined by joint decision of two experts. The change of FVC and FEF_{25-75} were evaluated for the two groups.

Result:
163 patients with haze alone (inflammatory type) and 88 patients with honeycombing (±other changes) (fibrotic type) were included with 39 normal subject's spirometry data. The FVC and FEF_{25-75} appears significantly reduced ($p<0.05$) in DPLD (both the groups) compared to normal. However FVC and FEF_{25-75} cannot significantly differentiate fibrotic and inflammatory DPLD groups ($p=0.13$ and $p=0.14$ respectively).

Conclusion:
Both FVC and FEF_{25-75} are reduced in DPLD (both inflammatory and fibrotic type) compared to the normal group. The reduction in FVC appears more than FEF_{25-75}. None of them can effectively differentiate predominantly fibrotic from predominant inflammatory DPLD.

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Correlation of Transthoracic Echocardiography with Right Heart Catheterization in Pulmonary Hypertension patients

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Background:
the presence of PH is a common complication in patients with different respiratory disorders.

Objective:
to correlate estimated pulmonary arterial pressure (PAP) by echocardiography with right heart catheterization (RHC) measurements, correlate left ventricular ejection fraction with the cardiac output and to correlate the TR- jet velocity with the PVR and the PAP with RHC measurements.

Method:
in a screening survey for PH in patients presenting to a referral respiratory OPD services, we diagnosed PH from a clinico-radio- (HRCT chest)-echocardiographic work up. Screening for the etiological factors was accomplished with spirometry, collagen profile, V/Q scan in selective and suspected situations, polysomnography, liver function tests and other in case of obvious suspicion by the treating physicians for a specific etiology.

Results:
25 consecutive patients with Pulmonary Hypertension from varied etiologies underwent RHC. The correlation between right ventricular mean pressure (RVP) and mean pulmonary arterial pressure (PAP) was r=0.71,(p<0.0001), the maximum TR-jet velocity and the pulmonary vascular resistance (PVR) was r=0.67,(p<0.0006) and between TR- jet velocity and the mean PAP (RHC measurement) was r=0.764, (p<0.0001). However, the correlation of echo measured ejection fraction (EF) and cardiac output (CO) was poor, r=0.278, (p <0.2224).

Inference:
Single echocardiographic operated hemodynamic parameters revealed acceptable correlation with RHC measured variables for PH. Although RHC is regarded as a gold standard, echo-derived measurement for PAP may be acceptable as a suitable parameter for screening patients.

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The prevalence of sleep apnea in patients with Class III PH: a pilot observation

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Background:
Sleep apnea is a cause of Class III PH and it is usually looked for only on clinical suspicion.

Aim:
the aim was to identify the presence of sleep apnea in patients of Class III PH, where OSA is not clinically forthcoming or suspected.

Method:
In a screening survey for PH in patients presenting to a referral respiratory OPD services, we diagnosed PH from a clinico-radio- (HRCT chest)-echocardiographic work up. Screening for the etiological factors was accomplished with spirometry, collagen profile, routinely monitoring with VQ scan, polysomnography, liver function tests, HIV serology, peripheral smear examination and other, in case of obvious suspicion by the treating physicians for a specific etiology. All the patients were routinely evaluated for the presence of OSA with apnea link. They were evaluated by measuring pulse rate (PR), Apnea Hypopnea Index (AHI), Respiratory Index (RI), and Oxygen Desaturation Index (ODI).

Results:
32 consecutive patients with chronic respiratory disease and PH were sent for sleep study. The results reflected that the presence of sleep apnea is higher in these patients (26 out of 32) i.e. 60.23%. The profile of the underlying respiratory disease was as, i) COPD= 10 (41.66%), ii) asthma = 5 (16.66%), iii) PAH=3 (4.1%), iv)others= 8(33.33%). The severity of the OSA was decided on the basis of certain parameters as mentioned above. (Table 1)

Inference:
The presence of covert OSA in the screened class III PH is high (60.23%) in patients not suspected of the condition by expert physician. However, the casual association needs to be further elucidated.

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Obstructive airway disease in tribal and non-tribal populations in rural Bengal, India

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Background:
Studies regarding the respiratory diseases suffered by the people of both tribal and non-tribal population of rural Bengal, India are lacking. It is important to look into their respiratory status to find out the prevalence of respiratory diseases in them.

Method:
We screened 713 subjects (n= 302 tribes and n=411 non-tribes) of Rajnagar block of Birbhum district, West Bengal, India for the presence of respiratory ailments using Chest X-Ray, Spirometry, and clinical evaluations. The status of the obstructive lung disease (OLD) was analyzed in them.

Results:
The frequency of OLD was 20(6.69%) and 50 (9.70%) among the tribal and non-tribal population. The mean age of the OLD patients was 38.37±12.94 and 42±13.00 with the male and female ratio being 14:7 and 38:12 for the tribal and non-tribal population respectively. The severity of OLD (GOLD III and IV) was higher in nontribal population (58%) compared to the tribal population (45%). The predicted percentage of FEF_{25-75} was significantly low in non-tribal population (24.52±9.40) compared to the tribal population (30.85±7.74).

Conclusion:
The tribal population appears to have lesser frequency and severity of OLD compared to the non-tribal population in the same geographic area of West Bengal, India. The reason behind this may be related to the differences in their smoking habits and use of fuels by them. Further researches are being conducted to elucidate the proper reason for such differences.

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Diagnostic clue in lung function test of clinical and HRCT suspected constrictive bronchiolitis

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Background:
Constrictive bronchiolitis is a small airway fibrotic respiratory disease usually diagnosed using a transbronchial biopsy. High Resolution Computed Tomography (HRCT)gives clue to it from the 'mosaic' pattern and air trapping. HRCT being an expensive diagnostic tool we searched for marker of the disease, if

any, in the lung function test.COPD and Asthma is the closest differential diagnosis, so our aim was to distinguish the constrictive bronchiolitis from asthma using spirometry parameters.

Materials and methods:
11 patients out of 307 selected subjects were studied serially and clinico-radiological diagnosis for constrictive bronchiolitis was done. In these patients we looked at the different variables in the lung function test such as FEV_1/FVC, % predicted FEV_1 and % predicted FEF_{25-75}.

Results:
Spirometry of the suspected constrictive bronchiolitis patients showed evidences of a. obstructive airway disease, b. moderate to severe air flow limitations and c. disproportionate reduction in % predicted FEF25-75 compared to % FEV1 as $\geq$ 40%.

Conclusion:
Out of proportion reduction of FEF25-75 compared to FEV1 may indicate towards a possibility of chronic bronchiolitis in patients of obstructive airway disease. The observation demands further validation.

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Possible Role of Rad50 in lung infection mimicking Acute Lung Injury (ALI)

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DNA repair protein Rad50, encoded by rad50 gene, plays a major role in repairing double strand DNA damage, recombination, and meiosis. The Genome Wide Scan Association Studies identified Rad50 as one of the genes associated with asthma. Studies done in our lab found that Rad50 knocked down mice showed increased levels of IL17 and severe neutrophilia. Thus we hypothesized that Rad50 have a role in lung infection as both IL-17 and neutrophils are also involved in lung infection. Through neutrophil chemotaxis assay, we mimicked the neutrophil migration in blood vessel and we found that Rad50 deficient bronchial epithelia secrete something that attracts neutrophil. LPS (the outer lipopolysaccharide wall of bacteria) when given, mimics lung infection in mice and serves as a model for acute lung injury. Importantly, we found a reduction in Rad50 expression in lungs of LPS administered mice. Further, when LPS administered mice were given Rad50 plasmid (intravenous route), there was a reduction in the features of ALI. A decrease in the thickening of the alveolar wall septum is seen along with reduction in the neutrophil level of alveoli in these mice compared to control plasmid given mice. Whereas in the LPS treated control mice, alveoli were surrounded by neutrophils and thickening of alveolar wall due to neutrophil infiltration. However, these features were reduced in Rad50 plasmid given mice. This indicates that Rad50 have a role in preventing neutrophil migration and could be a therapeutic target in neutrophil dominant lung diseases like acute respiratory distress syndrome.

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Status of Key DNA Repair Proteins in Asthma

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Abstract:
Various genetic and functional studies indicate the importance of DNA repair proteins in respiratory diseases like asthma and COPD.In this study, DNA damage levelsof control and asthmatic individuals and SHAM, OVA &CE exposed mice were determined. The levels of 8-OHdG, an oxidative DNA damage marker was determined in control and asthmatic lungs of human and mice.The status of major DNA damage sensors such as ATM, ATR and DNAPkcs and Ku proteins, involved in double strand break repair were measured incontrol and asthmatic mice lungs. γ-H2AX levels, a biomarker for double strand breaks was determined incontrol and asthmatic micelungs. The asthmatics show an increase in DNA damage, compared to controls. 8-OHdG was increased in asthmatic lungs. There was a great reduction in ATM and Ku80 and modest reduction in Ku70 in asthmatic lungs.However, there was no change in γ-H2AX levels in asthmatic lungs; this could be due to reduction in ATM levels in asthmatics. These results suggest that there is increased DNA damage and reduced expression of key DNA repair proteins in asthmatic lungs.

Abbreviations used:
OVA: Ovalbumin, CE: Cockroach allergen extract, 8-OHdG: 8-Oxo-2'-deoxyguanosine, ATM: Ataxia telangiectasia serine/threonine kinase, ATR:ATM and Rad 3 related serine/threonine kinase, DNAPkcs: DNA dependent protein kinase - catalytic subunit, γ-H2AX: Gamma H2A histone family member X.

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Antiphospholipid syndrome (APS) with pleural effusion

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Antiphospholipid syndrome (APS) with pleural effusion with pericardial effusion is extremely rare. A 32-year-old female was admitted to our hospital for spreading breathlessness ,pain and swelling of extremities, as well as dyspnea. One month back she had history of spontaneous abortion. Her chest X-ray demonstrated bilateral pleural effusion left side greater than right site of lung. She underwent repeated drainage of the pleural effusion but the effusion recurred. Ultrasonography showed thrombosis in inferior venacava and complete thrombosis of interna jugular vein.We added oral prednisolone 30 mg daily to his prior anticoagulant therapy. Her pleural effusion rapidly improved and disappeared without any complication. Corticosteroids might be a choice of treatment for intractable pleural effusion in APS patients.

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A novel role for Rad50 in Pulmonary fibrosis

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Abstract:
Idiopathic pulmonary fibrosis (IPF) is the most common type of interstitial pneumonia of unknown etiology in which chronic fibrosis may lead to irreversible damage of lung architecture. It is the life threatening disease of median survival less than 3 years. There is no promising therapy available that can completely cure the disease. Our understanding of pathophysiology of IPF is still incomplete and there is an urgent need of effective therapy that can eradicate IPF. Our lab has first time demonstrated the role of Rad50; a crucial DNA repair protein in asthma. In this study we have observed Rad50 knock downed mice has shown some features of pulmonary fibrosis like increase in TGF-β, collagen deposition in lungs. With these observations we hypothesized Rad50 may be involved in pathogenesis of IPF. Surprisingly, expression of Rad50 was found to be increased in human lungs of IPF patients as compared to normal individuals. We also found similar up-regulation in lungs of mice that have been administered bleomycin that showed the features of human IPF like pulmonary fibrosis. We are in the process of elucidating the detailed role of Rad50 in pulmonary fibrosis and to target Rad50 in pulmonary fibrosis.

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Possible role of SMAR1, a nuclear matrix protein, in COPD pathogenesis

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Abstract:
The chronic obstructive pulmonary disease (COPD that is characterized by chronic bronchitis (mucus hyper secretion) and emphysema (alveolar disruption)) is known to be the fourth leading disease of death (expected to be third by 2030 -WHO reports) worldwide affecting over 329 million people (almost 4% of entire world population). In general, it is understood that smoke irritants and subsequent oxidative stress lead to release of pro-inflammatory mediators and matrix digestive enzymes from T_H17 cells and neutrophils such as IL-17, elastase and matrix metalloproteinase 9 (MMP9) to cause alveolar disruption (emphysema). Our lab had found a reduction in the levels of SMAR-1 [nuclear scaffold /matrix- associated region-binding protein 1 which was first identified in T_H double positive mouse thymocytes] in human and mice COPD lungs. It is to be noted that IL-17 is found to be suppressed by SMAR-1. SMAR-1 siRNA treated mice had shown the increase of IL-17A levels and total MMP activity, an indirect index of emphysema, in the lungs. Hence, modulating the levels of SMAR-1 by genetic strategy or using small molecules might reduce the features of emphysema. We are in the process of elucidating the detailed role of SMAR-1 in COPD and pharmacologically target SMAR-1 in COPD.

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