NATIONAL COLLEGE OF CHEST PHYSICIANS (INDIA)

Lung Bulletin

NEWSLETTER OF NATIONAL COLLEGE OF CHEST PHYSICIANS (INDIA)
SECOND ISSUE JULY - DECEMBER 2020
THEME - PULMONARY FUNCTION TESTS

HIGHLIGHTS

- PULMONARY FUNCTION TESTS
- MEMBERS CORNER
- MEMBERSHIP BENEFITS
- TRAVEL GRANT
- E - COURSES
- ACADEMIC ACTIVITIES
- PUBLICATIONS
- NEBULIZATION GUIDELINES
- POST - GRADUATE QUIZ
- UPCOMING EVENTS
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<table>
<thead>
<tr>
<th>Sr. No.</th>
<th>Title</th>
<th>Author(s)</th>
<th>Page Nos.</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td><strong>SECTION I</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1</td>
<td><strong>Index</strong></td>
<td></td>
<td>3</td>
</tr>
<tr>
<td>2</td>
<td><strong>Upcoming Events</strong></td>
<td></td>
<td>4</td>
</tr>
<tr>
<td>3</td>
<td><strong>Messages</strong></td>
<td>President</td>
<td>6</td>
</tr>
<tr>
<td></td>
<td></td>
<td>P D Motiani</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Secretary</td>
<td>7</td>
</tr>
<tr>
<td></td>
<td></td>
<td>S N Gaur</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Chairman, Scientific Committee &amp;</td>
<td>8</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Academic Forum</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>S K Katiyar</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Editor, NCCP(I) Lung Bulletin</td>
<td>9</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Nikhil Sarangdhar</td>
<td></td>
</tr>
<tr>
<td>4</td>
<td><strong>Members Corner</strong></td>
<td>National</td>
<td>10</td>
</tr>
<tr>
<td></td>
<td></td>
<td>International</td>
<td>11</td>
</tr>
<tr>
<td>5</td>
<td><strong>NCCP(I) Membership Drive</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>NCCP(I) Membership Benefits</td>
<td>12</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Membership Form</td>
<td>13</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Directory Entry Form</td>
<td>15</td>
</tr>
<tr>
<td>6</td>
<td><strong>NCCP(I) Governing Council</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>NCCP(I) Governing Council Members</td>
<td>16</td>
</tr>
<tr>
<td>7</td>
<td><strong>Academic &amp; Educational Activities of NCCP(I)</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>E- Courses</td>
<td>17</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Indian Guidelines on Nebulization</td>
<td>18</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Therapy</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Books &amp; Publications</td>
<td>20</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Travel Grants &amp; NCCP(I) Young</td>
<td>23</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Scientist Award</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>PG Quiz in Respiratory Diseases</td>
<td>24</td>
</tr>
<tr>
<td></td>
<td></td>
<td>2019</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>NAPCON</td>
<td>28</td>
</tr>
<tr>
<td>8</td>
<td><strong>SECTION II - PULMONARY FUNCTION TESTS</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>8.1</td>
<td>Introduction and History of Pulmonary Function Testing</td>
<td>P. S. Shankar</td>
<td>30</td>
</tr>
<tr>
<td>8.2</td>
<td>Lung Volumes and Capacities</td>
<td>Amita Nene, Neel Thakkar</td>
<td>32</td>
</tr>
<tr>
<td>8.3</td>
<td>Spirometry in Upper Airway Obstruction</td>
<td>Unnati Desai, Ketaki Utpat</td>
<td>37</td>
</tr>
<tr>
<td>8.4</td>
<td>Pitfalls of Spirometry</td>
<td>S. K. Jindal</td>
<td>41</td>
</tr>
<tr>
<td>8.5</td>
<td>Spirometry – Interesting Cases</td>
<td>Rohit Kumar</td>
<td>44</td>
</tr>
<tr>
<td>8.6</td>
<td>Peak Expiratory Flow : Estimation and Clinical Applications</td>
<td>Virendra Singh, Nishta Singh</td>
<td>52</td>
</tr>
<tr>
<td>8.7</td>
<td>Measurement of Diffusion Capacity</td>
<td>Ramakant Dixit</td>
<td>55</td>
</tr>
<tr>
<td>8.8</td>
<td>Pre-Operative Pulmonary Assessment</td>
<td>P. Arjun, Soofia Mohammed</td>
<td>58</td>
</tr>
<tr>
<td>8.9</td>
<td>Assessment of Fitness to Fly</td>
<td>Naveen Dutt, Kunal Deokar,</td>
<td>62</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Shahir Asfahan</td>
<td></td>
</tr>
<tr>
<td>8.10</td>
<td>Body Plethysmography</td>
<td>Lavina Mirchandani</td>
<td>65</td>
</tr>
<tr>
<td>8.11</td>
<td>Cardio-Pulmonary Exercise Testing : Measurement and Clinical Utility</td>
<td>P S Tampi</td>
<td>71</td>
</tr>
<tr>
<td>8.12</td>
<td>Assessment of Respiratory Muscle Strength</td>
<td>Randeep Guleria</td>
<td>76</td>
</tr>
<tr>
<td>8.13</td>
<td>Impulse Oscillometry</td>
<td>Ritisha Bhatt</td>
<td>78</td>
</tr>
<tr>
<td>8.14</td>
<td>FENO : Measurement and Clinical Applications</td>
<td>Saurabh Mittal</td>
<td>81</td>
</tr>
<tr>
<td>8.15</td>
<td>Arterial Blood Gas Interpretation</td>
<td>Supriya Sarkar</td>
<td>84</td>
</tr>
<tr>
<td>8.16</td>
<td>Spirometry Practice in the COVID Era</td>
<td>P. S. Shankar</td>
<td>87</td>
</tr>
<tr>
<td>Sr. No.</td>
<td>DATES</td>
<td>CONFERENCE</td>
<td>VENUE</td>
</tr>
<tr>
<td>--------</td>
<td>-------------</td>
<td>-----------------------------------------------------------------------------</td>
<td>---------</td>
</tr>
<tr>
<td>3.</td>
<td>March 9 - 11</td>
<td>18th World Conference on Tobacco or Health</td>
<td>Dublin</td>
</tr>
<tr>
<td>6.</td>
<td>April 8 - 11</td>
<td>Pulmonary and Critical Care Medicine 2021</td>
<td>Scottsdale</td>
</tr>
<tr>
<td>10.</td>
<td>May 24 - 25</td>
<td>ICRDCCSD 2021 : International Conference on Respiratory Diseases, Critical Care and Sleep Disorders</td>
<td>London</td>
</tr>
<tr>
<td>12.</td>
<td>September 4 - 8</td>
<td>European Respiratory Society (ERS) Congress</td>
<td>Barcelona</td>
</tr>
</tbody>
</table>
DEAR COLLEAGUES,

AS MEMBERS / FELLOWS OF NATIONAL COLLEGE OF CHEST PHYSICIANS (INDIA),

ARE YOU GETTING ..... 
► E-voting form sent to Your E-mail to Vote in Yearly Elections to NCCP(I) Governing Council ?
► Communications through E-mail and invitations to attend NCCP(I) Annual General Body Meeting (AGM) ?
► Indian Journal of Chest Diseases and Allied Sciences (Quarterly issues) by Post ?
► NCCP(I) National Directory of Chest Physicians (Every 5 years) ?

HAVE ANY OF THESE CHANGED ..... 
► E-mail ID or Address ?
► Mobile Number ?
► Postal Address ?
► Residence ?
► Clinic or Hospital ?
► Institution or Place Where You Work ?

IF THE ANSWER TO ANY OF THESE IS 
NO

THEN IT’S TIME TO UPDATE YOUR COMMUNICATION DATA IN OUR RECORD !

Please send an E-mail to Prof. Dr. S. N. Gaur, Secretary, NCCP(I) to sngaur9@gmail.com mentioning Your Name and NCCP(I) Life-Membership or Life-Fellowship Number and providing the contact details below with a request to update Your information in NCCP(I) record :

1. Active Mobile Number
2. Working E-Mail ID
3. Complete Postal Address (with Landmark, Street, District, City and PIN-CODE)

Should You need any assistance or have any queries regarding Your NCCP(I) Membership or Benefits, feel free to contact us. Our Support is always available to help You !

COMMUNICATE WITH US

www.nccpindia.org

Dr. S. N. Gaur, Gaur Clinic, 130 - A, Patparganj Village, Delhi – 110091

sngaur9@gmail.com , ncsarangdhar@rocketmail.com

(+91) 9811271916 , (+91) 9029429015
Dear Colleagues,

It is a matter of pleasure that second newsletter of National College of Chest Physicians (India [ NCCP (I) ] is going to be released. It is dedicated to pulmonary function testing. Pulmonary function tests are non-invasive tests that show how well the lungs are working. Tests measure lung volumes, capacities, rate of flow, and gas exchange in the subjects for diagnostic and prognostic purposes in symptomatic lung problem, subjects exposed to substances at workplace, and also to monitor chronic lung diseases. It has limitations with heart diseases, surgeries, obesity, pregnancy and other respiratory infections. It requires trained personnel to perform, equipment and pulmonary labs.

The new techniques and tests are now available. The second issue of newsletter of NCCP (I) will be informative to all of us, contributed by eminent pulmonologists on various Pulmonary Function Tests.

I congratulate Dr. Nikhil Sarangdhar for his passion and hard work in bringing out second issue of Newsletter of NCCP (I).
Dear Colleagues,

The National College of Chest Physicians (India) [NCCP(I)] is a registered body functioning to promote the cause of Chest Diseases and Allied Sciences in India and to take this specialty forward in the field of Medicine. It was formed originally with 58 founder members as the Indian Association of Chest Diseases (IACD) in 1959 at the Indian Science Congress. The IACD in its meeting held on November 15, 1979 subsequently ratified by the General Body meeting held on November 6, 1979, unanimously decided to change the name of IACD to National College of Chest Physicians (India) and to make consequential changes/amendments in the memorandum of the Association, and its rules and regulations by a sub-committee, duly constituted for this purpose and the recommendations were confirmed and approved by the prescribed authority and confirmed at a subsequent special meeting of the General Body held on August 14, 1980. The National College of Chest Physicians (India) thus came into being in January, 1981. Since then, it has grown from strength to strength and currently has on its roll more than 1800 Members and 300 Fellows, making it one of the largest national registered professional medical associations, contributing to the development of the specialty of Pulmonary Medicine since its inception. The mission of NCCP(I) is to promote academic growth, partnership and collaboration for education in a rapidly developing world and develop strategies for better clinical practice in Pulmonary Medicine.

The NCCP(I) official website is www.nccpindia.org. The Indian Journal of Chest Diseases and Allied Sciences is the official publication of NCCP(I) and is published jointly with Vallabhbhai Patel Chest Institute, Delhi. This journal is indexed and has been widely acclaimed at both national and international levels. In addition, the NCCP (I) publishes a Directory of Chest Physicians, which is updated every 5 years.

Under the convenorship of Dr. Rajesh Chawla, Past President of NCCP(I), the College has launched two E-courses - Comprehensive Pulmonary Medicine E-course (CPMeC) and Interventional Pulmonology E-course (IPeC) for the benefit of post-graduates and clinicians practising in the specialty. The CPMeC was the first online course in Pulmonary Medicine in India accredited by the National Board of Examinations, New Delhi and met with resounding success, having nationwide enrolment of more than 1400 doctors. IPEC has been launched last year. NCCP(I) in collaboration with ICS has also developed guidelines for Pneumonia, Vaccination, ILD, COPD, Bronchoscopy, Spirometry, and the progress is going on for Guidelines of Pleural Diseases, Medical Thoracoscopy and revised COPD guidelines. NCCP(I) has also developed National guidelines on Nebulization therapy. NCCP(I) also encourages original research by young scientists and consultants by providing travel grants to all members and fellows for upgrading their knowledge by attending national and international conferences (ACCP, ATS, APSR, Gulf Thoracic and others).

Ever since its inception, the College held 33 conferences with the Association of Physicians of India and since the 28th conference, it has organized its annual conferences (NACCON) independently. These conferences were highly successful and were chaired by the President of NCCP(I). From 1999, the NCCP(I) with ICS is having Joint National Conference on Pulmonary Diseases – NAPCON. I am happy to inform you that all the last twenty-one NAPCONs were a grand success, appreciated by the delegates and international faculty. I am sure that the same spirit will continue and we will have more and more participation as well as better conferences in the future. This year, NAPCON-2020 is being organized and as in the past, we are expecting a good number of foreign faculties from ACCP, ATS, ERS, APSR, Gulf Thoracic Society and from neighboring countries. I have full confidence that NAPCON-2020 will be organized with best efforts in a manner to make it a most memorable event.

NCCP(I) also started under the leadership of then President Dr. Rajesh Chawla, a Newsletter titled “Pulmonary Communications” in 2016, which was continued as “Lung Bulletin” in 2020 with Dr. Nikhil Sarangdhar as Editor, NCCP(I) Newsletter. The NCCP(I) Newsletter is aimed at updating current knowledge about various respiratory diseases, to acquaint our young enthusiastic Post-graduates and Chest Physicians with events of interest occurring in our specialty and also provide them a platform to interact with each other and to participate in the exchange of knowledge. The newsletter will be published twice-yearly with each issue dedicated to a different topic. The First issue of Newsletter, dedicated to Pulmonary Hypertension was very successful and well received and appreciated by all members and fellows of the college. On behalf of the National College of Chest Physicians (India) as well as on my personal behalf, I congratulate Dr. Nikhil Sarangdhar, who is the Editor, NCCP(I) Newsletter for his hard work in bringing out the second issue of NCCP(I) Newsletter, dedicated to “Pulmonary Function Tests” which I am confident will be appreciated by all our colleagues in the field. I am sure that this endeavour will be useful and I wish the NCCP(I) Newsletter all success.
Dear Colleagues,

It is my great pleasure to extend heartfelt greetings to the readers of the Newsletter of National College of Chest Physicians (India). I congratulate Dr. Nikhil Sarangdhar, Editor, NCCP(I) Newsletter for his efforts in bringing out the second issue of this bi-annual publication, which is not just a newsletter, but much beyond, in terms of its rich and varied academic content. Each issue is being dedicated to one disease of clinical interest, this time 'Pulmonary Function Tests', with the aim to have a comprehensive coverage of all its clinical aspects and making it more case oriented also. This will not only attract our younger members, but all others too, to update their knowledge on diagnosis and management of respiratory illnesses. Efforts of the entire Editorial Board and the Governing body are commendable and need appreciation for this splendid effort.

I am sure the NCCP(I) Newsletter – Lung Bulletin will also prove useful to all members to keep them informed and provide them with updated news, events, reports and other information. The bulletin will also bridge the gap between the members and the College and become a medium of communication between the two, who otherwise, can only learn about the achievements and activities of the College during the annual general body meeting held once a year during the conference. The newsletter is a media for the members through which they can share their information, knowledge, experiences and concerns. Further it will also help to connect the members promoting better understanding and cooperation.

As Chairman of the Scientific Committee of NAPCON – 2020, it is my proud privilege and honour to invite and welcome you all to this year’s conference and I can assure that you are going to witness one of the best scientific programs, which will be rich in its contents and purposeful too, to entirely change the perspective of your day to day clinical approach.

I wish all the readers of the NCCP(I) Newsletter an enriching and informative reading experience and will welcome their feedback on this new communication. My best wishes to Dr. Nikhil Sarangdhar and his editorial team for grand success of this venture.

Please Take Care, Stay Safe and Healthy during this COVID-19 crisis.
From The Desk of Editor, NCCP(I) Lung Bulletin

Dr. Nikhil Sarangdhar  
Editor, NCCP(I) Lung Bulletin  
Former Assistant Professor, Department of TB & Chest diseases, K. J. Somaiya medical college, Mumbai  
Organising Secretary, NAPCON 2016 & Member, Scientific Committee, NAPCON 2016, 2018, 2019 & 2020  
Young Scientist Awardee of the Indian College of Allergy Asthma and Immunology (2011, 2014 & 2015),  
Association of Physicians of India (2015), Indian Chest Society (2015),  
National College of Chest Physicians-India (2017)  
E-mail: ncsarangdhar@rocketmail.com

Dear Colleagues,

National College of Chest Physicians (India) [NCCP(I)] recognizes the ever growing scope and potential of Pulmonary Medicine in India and seeks to tap the pool of knowledge, skill and talent available to keep up with the growth of our field nationally and globally. The news that all students and practising specialists of Pulmonary Medicine, both the young and the elderly, are going to be provided a common academic platform ignites a scientific temper which spreads like a raging inferno, inculcating a deep academic interest to learn by interaction by sharing their ideas, knowledge and experiences with each other. To keep up this scientific temper with a focus on our young dynamic chest physicians of today, the National College of Chest Physicians (India) launched the first issue of its Newsletter ‘NCCP(I) Lung Bulletin’ dedicated to “Pulmonary Hypertension” and today we are privileged to launch the second issue, dedicated to “Pulmonary Function Tests”

Besides providing a platform for interaction with all our colleagues, NCCP(I) Lung Bulletin also aims to update our knowledge and keep us acquainted with current events of interest in Pulmonary Medicine through articles written by senior colleagues as well as young experts across the length and breadth of India, from Kashmir to Kanyakumari and Dwarka to Dibrugarh for a truly national outlook. Each issue is bifurcated into two sections, a general section abundant in academic content including editorials and reviews on guidelines, practice changing research and other developments in our field, followed by a specific section dedicated to one disease or area of interest in Pulmonary Medicine with technical information about basic sciences supplemented by interesting real-life case reports to ensure a unique amalgamation of knowledge, experience and skill. Lung Bulletin is meticulously compiled with an integrated and systematic approach to ensure that every issue is unique, with the ultimate goal of providing a comprehensive all-in-one review and up-to-date source of information on the subject to the reader.

NCCP(I) Lung Bulletin will be regularly published on a biennial basis with the aim to make it a highly popular and sought after publication. We are confident that it will be received with unparalleled excitement and enthusiasm by all members and fellows of our fraternity who eagerly await its launch. We strive to maintain our Indian tradition of the ‘personal touch’ at all levels while compiling Lung Bulletin and welcome inputs from all post-graduate medical students, teachers and consultants in the field of Pulmonary Medicine. Please feel free to write to me at ncsarangdhar@rocketmail.com.

I take this opportunity to express my gratitude to the National College of Chest Physicians (India) for entrusting me with the responsibility of compiling and publishing NCCP(I) Lung Bulletin. I am personally grateful for the unconditional support and encouragement extended by the Governing Council of NCCP(I), particularly Prof. Dr. P. D. Motiani (President), Prof. Dr. S. N. Gaur (Secretary) and Prof. Dr. S. K. Katiyar (Chairman, Scientific Committee and Academic Forum) as well as all our Members and Fellows. I thank our authors and contributors for being accessible, enthusiastic, cooperative and supportive like a family throughout this endeavour. Lastly, I place on record the personal feedback and appreciation given by everyone in support of our endeavour which made it possible for us to publish Lung Bulletin in the most scientific and professional manner. I am confident we will together succeed in driving Lung Bulletin to greater heights to propel the ever-expanding future of Pulmonary Medicine in our country.
I consider it my privilege to share my message in NCCP(I) News Letter and I thank Dr. Nikhil Sarangdhar for giving me the opportunity. Updation is a mandatory requirement in the medical field and there is no better platform for this than learning from the experience and views of stalwarts. The News Letter will serve as a treasure house of information and rapid reference module for the practising clinician. Devoting each issue of the News Letter to a focused arena in pulmonary medicine allows for in-depth understanding and refreshing of the basic concepts topped by recent advances. I am sure that, with guidance from the senior teachers in NCCP(I) like Prof. Dr. S. N. Gaur, Prof. Dr. S. K. Katiyar and others, Dr. Nikhil Sarangdhar will ensure this News Letter to be a concise learning forum appreciated by consultants and trainees alike. I wish the NCCP(I) News Letter all success.

Rajesh V
Sr. Consultant and Head, Department of Pulmonary Medicine, Rajagiri hospital, Kochi, Kerala
Organizing Secretary, NAPCON 2019

NCCP (I), a national academic body, has constantly aimed to achieve academic brilliance by means of journal, conferences and now newsletters. It was a proud privilege for me to contribute an article for the inaugural issue of NCCP newsletter. I am extremely happy to learn that the second issue of NCCP(I) News Letter is going to be released. It will be dedicated to Pulmonary Function Tests and will have contributions from various experts in the field of respiratory medicine from all over the country. I wish the Editorial board all the success and congratulate in this academic endeavour. I am sure, under the dynamic leadership of Dr. Nikhil Sarangdhar, the newsletter will continue to grow and evolve.

Pranav Ish
Assistant Professor - Pulmonary, Critical care & Sleep Medicine,
VMMC & Safdarjung Hospital, New Delhi

I am very happy to learn that the second News Letter of National College of Chest Physicians (India) focused on Pulmonary function tests has come out. Teaching and learning go hand in hand. It’s a great initiative by NCCP(I) especially for the young pulmonologists for learning and training. I was also very happy and lucky as well to be a part of the inaugural issue of NCCP(I) News Letter. I am very sure that under the able leadership of Dr. Nikhil Sarangdhar and the NCCP(I) editorial team, this News Letter series will be a huge success and will guide and teach everyone in their routine practice.

Piyush Arora
Assistant Professor
Department of TB and Respiratory Diseases, JLN medical college, Ajmer, Rajasthan
MEMBER’S CORNER (INTERNATIONAL)

Dear Dr. Nikhil Sarangdhar,

It is heartening to learn that the National College of Chest Physicians (India) [NCCP(I)] is publishing its Newsletter aimed at updating the professional knowledge of physicians on various respiratory diseases.

National College of Chest Physicians (India) is an Institution exceptional in its diversity and expertise of professionals in the wide arena of Pulmonary Medicine and the NCCP(I) Newsletter is a long-awaited event. I am sure, rather I have a firm belief that the NCCP(I) Newsletter will provide a comprehensive platform to share knowledge among our colleagues and provide updates on Pulmonary Medicine.

It is matter of great honor and privilege to write a message for the first issue of the NCCP(I) Newsletter. I gratefully appreciate your commendable efforts towards this endeavour. As a fellow of NCCP(I), I extend My Heartfelt Congratulations and Best Wishes to You and the Editorial Team in bringing out this Newsletter and Wishing You all the Best on your next project!

Dr. Narendra Bhatta - Professor & Head, Department of Pulmonary, Critical Care & Sleep Medicine
B. P. Koirala Institute of Health Sciences (BPKIHS), Dharan, Nepal

Dear Dr. Nikhil Sarangdhar,

I am delighted to learn that the National College of Chest Physicians (India) [NCCP(I)] is bringing out its Newsletter aimed at promoting professional collegiality among the clinicians and updating their knowledge about different respiratory illnesses.

I congratulate the Editorial Board for bringing out this newsletter. NCCP(I) has the reputation of being an Outstanding Academy of Pulmonary Medicine. I gratefully remember the academic contents of many NAPCON’s organized under NCCP(I) leadership. I am sure that this Newsletter will provide the platform to connect Pulmonary professionals worldwide and provide recent advances and updates occurring in the field of Pulmonology.

I feel honored to write a message for the first issue of the NCCP(I) Newsletter. I send My Best Wishes to You and All Members of the Editorial Board on the occasion of publication of this Newsletter and extend My Greetings!

Dr. Nisha Kesahry Bhatta - Professor & Chair, Division of Neonatology, Department of Pediatrics,
B. P. Koirala Institute of Health Sciences (BPKIHS), Dharan, Nepal

Dear Colleagues,

As a Fellow of National College of Chest Physicians (India), I feel elated to know that NCCP(I) is going to launch its very first Newsletter. NCCP(I) is one of the oldest association of Chest Physicians with an outstanding reputation nationally as well as internationally and has played a key role in fostering the growth of many budding Pulmonologists during their professional careers in India and abroad.

The objectives of NCCP(I) Newsletter are many, for one, it will acquaint our colleagues with the academic endeavours and activities of NCCP(I), keep them updated about different respiratory diseases and current events as well as bring us all together on a single platform to exchange views, ideas and achievements for professional growth like a fraternity.

I congratulate NCCP(I) and the Editor, NCCP(I) Newsletter Dr. Nikhil Sarangdhar for their efforts in bringing out the first issue, which I am sure will prove to be a very popular publication rich in academic content that will benefit all our colleagues and post-graduate students alike and will acquire an outstanding momentum which will be kept up with subsequent issues dedicated to specific topics. My Best Wishes for the grand success of this novel venture.

Dr. Vikram Sarbhai - Specialist in Pulmonology, R.A.K. Hospital, United Arab Emirates
Senior Consultant, Pulmonology Critical Care and Sleep Medicine, National Heart Institute, New Delhi
**NCCP(I) MEMBERSHIP DRIVE**

President : Dr. P. D. Motiani  
Secretary : Dr. S. N. Gaur  
Convenor, Membership drive : Dr. S. K. Katiyar  
Co-Convenor, Membership drive : Dr. Nikhil Sarangdhar

**BECOME A MEMBER TODAY**

**COMMUNICATE WITH US**

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Dr. S. N. Gaur, Gaur Clinic, 130 - A, Patparganj Village, Delhi – 110091
sngaur9@gmail.com, nc3sarangdhar@rocketmail.com
(+91) 9811271916, (+91) 9029429015
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**MEMBERSHIP BENEFITS**

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<td>1.</td>
<td>Discounted Registration for NCCP(I) Members and Fellows at NAPCON.</td>
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<td>2.</td>
<td>Discounted Course fee for NCCP(I) Comprehensive Pulmonary Medicine E-Course (CPMeC) and NCCP(I) Interventional Pulmonology E-Course (IPeC) [Course Website: <a href="https://chestcourses.org">https://chestcourses.org</a>].</td>
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<tr>
<td>3.</td>
<td>Opportunity to participate and present your original research work at national conference (NAPCON) with travel grant for NCCP(I) - Prof. Dr. S. N. Gaur young scientist award.</td>
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<td>4.</td>
<td>Travel Grant for International Conferences (Rs. 80,000/- for U.S &amp; Canada &amp; Rs. 60,000/- for other countries) and National Conferences (Rs. 20,000/-) each year.</td>
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<td>5.</td>
<td>Lifelong subscription to quarterly issues of Indian Journal of Chest Diseases and Allied Sciences, one of the top rated and cited indexed journals of Respiratory Medicine.</td>
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<td>7.</td>
<td>Lifelong subscription to Directory of Chest Physicians (updated every 5 years).</td>
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<td>8.</td>
<td>Opportunity to avail of the Prestigious NCCP(I) Fellowship.</td>
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<td>9.</td>
<td>Opportunity to participate in Research Activities conducted under aegis of NCCP(I).</td>
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<td>10.</td>
<td>Upgradation of Knowledge and Technical Skills by attending accredited Conferences, Workshops and CME programmes organised under the aegis of NCCP(I).</td>
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<tr>
<td>11.</td>
<td>Opportunity to Associate, Collaborate and have One-to-One interaction with the top level practising Clinicians and Researchers in Pulmonary Medicine in India.</td>
</tr>
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<td>12.</td>
<td>Vote during Elections and Introduce New Members at Annual General Body Meeting during NAPCON.</td>
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**TAKE A TOUR OF OUR WEBSITE www.nccpindia.org**
National College of Chest Physicians (India)
(Formerly Indian Association for Chest Diseases)
V. P. Chest Institute, University of Delhi, Delhi - 110007

MEMBERSHIP ENROLMENT FORM

Regd No.: S/1421 (1981)

Send both Membership Form and the Directory Entry Form (see overleaf), completed and signed along with supporting documents (Degree & Medical Council Registration Certificate), photograph and payment by DD/Cheque for Rs. 7080/- in favour of "National College of Chest Physicians (India)" by post to:

Dr. S. N. Gaur, Gaur Clinic, 130-A, Patparganj Village, Delhi - 110091.

Instructions:
1. Entries in Boxes should be in Capital letters Only.
2. Information in Cols 1 to 5 and Cols 15, 16 are Mandatory and should be in Capital Letters only.
3. DD/Cheque should be drawn in favour of “National College of Chest Physicians (India)” payable at Delhi.
4. All correspondence and the IJCDAS (Journal) will be dispatched at your Mailing address.
5. Filled applications to be sent to Prof. S. N. Gaur, Gaur Clinic, 130-A, Patparganj Village, Delhi – 110091.

To,
The Secretary,
National College of Chest Physicians (India)

Dear Sir,

I request that I may be enrolled as a Member of National College of Chest Physicians (India). The Annual Subscription of Rs. 7080/-, Life Membership fee Rs.5000/- and Enrolment fee of Rs. 1000/- + GST 18% (Rs.1080) (Total Rs.7080/-) is enclosed herewith by Cash / Cheque / Demand Draft.

DD/Cheque No: ……………………… Date: ………………..  Amount Rs.7080/-

Name of the Bank and address)

1. Applicant’s Surname
   First Name
   Middle Name

2. Marital Status

3. Date of Birth
   Place of Birth
   D  D  M M Y Y Y

4. Permanent Address:
   State
   City
   PIN

5. Mailing Address*
   State
   City
   PIN

6. Telephone / Fax (with Area Code)
   Residence:
   Office:
   Fax:
   Mobile:

7. E-mail Address:
8. Medical Education: (ENCLOSE COPIES OF DEGREE / DIPLOMA)

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9. Experiences in Chest Speciality:

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10. Any Other Experience:

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11. Affiliation to other Scientific Bodies:

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12. Present Appointment and Office Address:

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13. Research Activities & Publications: (ENCLOSE LIST)

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14. Any other Relevant Information:

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15. Proposed and Seconded by:

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<th>Name</th>
<th>NCCP(I) Fellowship/ Membership No.</th>
<th>Address</th>
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Proposer:

Seconder:

16. Signature of Candidate (Applicant):

________________________________________________________________________________________________________________

Remarks of Credential Committee:

President NCCP(I) ..............................  Secretary NCCP(I) ..............................

For any difficulties encountered while filling up Membership form, write to ncsarangdhar@rocketmail.com
# NCCP (I) DIRECTORY ENTRY FORM

**Instructions:**
1. Please use Capital Letters or Type.
2. Please mention your Membership / Fellowship number for all future correspondence with College.
3. All correspondence and the IJCDAS (Journal) will be dispatched at your Mailing address.
4. Filled applications to be sent to Prof. S. N. Gaur, GAUR Clinic, 130-A, Patparganj Village, Delhi – 110091.

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| Present Designation & Organisation: |

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<th>Degrees:</th>
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| Affiliation to other Scientific Bodies: |

| Specialties: 1 2 3 4 |

| Interest Section: |

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<th>Spouse Name:</th>
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<td>Spouse Profession:</td>
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* Please let us know about any other information / suggestion or out of date information printed in the last Directory.
** Enclose any other information to be added in the Directory on a separate sheet.
NATIONAL COLLEGE OF CHEST PHYSICIANS (INDIA)
GOVERNING COUNCIL (2020-2021)

President (2020-21) – Dr. P. D. Motiani
Secretary (2019-22) – Dr. S. N. Gaur
Organizing Chairman, NAPCON 2020 – Dr. B. O. Tayade
President-Elect (2021-22) – Dr. Surya Kant
Immediate Past President (2019-20) – Dr. Surya Kant

Vice-President (2020-21) – Lt. Gen. Dr. B.N.B.M. Prasad
Joint Secretary (2020-22) – Dr. Raj Bhagat
Treasurer (2018-21) – Dr. V. K. Singh
Editor, IJCDAS (Director, VPCI) – Dr. Raj Kumar

Zonal Chairman (North) – Dr. J. C. Suri
Zonal Chairman (South) – Dr. R. Narasimhan
Zonal Chairman (East) – Dr. Narayan Mishra
Zonal Chairman (West) – Dr. V. K. Jain

Councillor (2019-21) – Dr. S. K. Katiyar
Councillor (2019-21) – Dr. Rakesh Chawla
Councillor (2019-21) – Dr. Ramakant Dixit
Councillor (2019-21) – Dr. Salil Bhargava

Councillor (2020-22) – Dr. K. B. Gupta
Councillor (2020-22) – Dr. Rajendra Prasad
Councillor (2020-22) – Dr. Gajendra Vikram Singh
Councillor (2020-22) – Dr. Nikhil Sarangdhar

Chairman, Academic Forum – Dr. S. K. Katiyar
Member, Academic Forum – Dr. Rajesh N. Solanki
Member, Academic Forum – Dr. Rajesh Chawla
Member, Academic Forum – Dr. K. B. Gupta

Organizing Chairman, NAPCON 2020 – Dr. S. K. Katiyar
Organizing Secretary, NAPCON 2020 – Dr. Rajesh N. Solanki
Chairman, Academic Forum – Dr. S. K. Katiyar
Member, Academic Forum – Dr. Rajesh N. Solanki
COURSE HIGHLIGHTS

► CPMeC has been meticulously prepared by National College of Chest Physicians (India) with the help of Eminent National and International Pulmonology Experts

► CPMeC is useful for Students as well as Practising Pulmonologists for updating themselves with latest recommendations and standards of care for the management of various respiratory diseases

► CPMeC consists of 50 online modules to cover all aspects of Pulmonary Medicine over a span of 150 days

► Each module contains Master Class, Take Home Points, Suggested Reading and Feedback

► More than 1400 Doctors have successfully enrolled in CPMeC accredited by National Board of Examinations, New Delhi (II-A)

► NCCP (I) E-Courses will issue online certificate after successful completion of the course

Website - https://chestcourses.org  Support - https://support.chestcourses.org , +91 - 84540 94444

Course Fee :  NCCP(I) Members - 4000 INR ;  Non-NCCP(I) Members - 6000 INR ;  Foreign Nationals - 149 USD

COURSE HIGHLIGHTS

► Nowadays, Interventional Pulmonology has progressed from simple Bronchoscopy to highly advanced diagnostic and therapeutic Bronchoscopy and Thoracoscopic procedures

► IPeC has been meticulously prepared by National College of Chest Physicians (India) with the help of Eminent National and International Experts in International Pulmonology

► IPeC is useful for Students as well as Practising Pulmonologists for to acquaint and update themselves with the skills required to perform a variety of diagnostic and therapeutic procedures including Bronchoscopy, Endobronchial Ultrasound (EBUS), Medical Thoracoscopy, Cryobiopsy, Airway Stenting , Management of Air Leaks and Hemoptysis and Percutaneous Tracheostomy

► IPeC consists of 30 online modules to cover all aspects of Interventional Pulmonology over a span of 180 days

► Each module contains Master Class, Take Home Points, Suggested Reading and Feedback

► NCCP (I) E-Courses will issue online certificate after successful completion of the course

Website - https://chestcourses.org  Support - https://support.chestcourses.org , +91 - 84540 94444

Course Fee :  NCCP(I) Members - 4100 INR ;  Non-NCCP(I) Members - 6100 INR ;  Foreign Nationals - 137 USD
Dear Colleagues,

You will be happy to know that we are soon going to publish ‘Indian Guidelines on Nebulization Therapy’ under the aegis of the National College of Chest Physicians (India). These guidelines are the first of their kind in our country and their compilation a pioneering achievement by the College in the field of Medical Education.

To formulate, compile and publish the Indian Guidelines on Nebulization Therapy under the aegis of the National College of Chest Physicians (India) was the brainchild of Prof. Dr. S. K. Katiyar. The Meeting of Experts for the Indian Guidelines on Nebulization Therapy was convened at Delhi on 3rd and 4th November 2018. A total of 67 Experts in Pulmonary Medicine across India, including members from states like Jammu & Kashmir and Assam were invited to ensure unique pan-Indian representation of ideas, expertise and opinion. Dr. S. K. Katiyar planned and convened the meeting, which was chaired by Dr. Rajesh Solanki [President, NCCP(I), in chair] and Dr. S. N. Gaur [Secretary, NCCP(I), in chair] and coordinated by Dr. Nikhil Sarangdhar.

The expert members were allocated into five groups consisting of a Group Convenor, Chairpersons, Advisor and Expert Members to cover different aspects of Nebulization therapy as follows :

1. Group A - Introduction, basic principles and technical aspects of nebulizers, types of equipment, their choice and maintenance.
2. Group B - Nebulization therapy in obstructive airway diseases
3. Group C - Nebulization therapy in the intensive care unit
4. Group D - Use of various drugs (other than bronchodilators & inhaled corticosteroids) by nebulized route and miscellaneous uses of nebulization therapy
5. Group E - Domiciliary nebulization therapy, public and healthcare workers education and future research

Five groups were constituted originally, but looking at the present global crisis created due to the pandemic of COVID-19 and consequently the apprehensions and concerns raised by spread of infection through nebulization it was thought to include a sixth group in the expert panel to provide guidance to caregivers while nebulizing patients, as follows :

6. Group F - Nebulization Therapy during COVID-19 pandemic and in patients of other contagious viral respiratory infections

Each group discussed the review of scientific evidence by members with intra-group discussions. Evidence and recommendations were presented by individual groups in the final meeting, for deliberations on the recommendations and arrival of consensus. After the meeting concluded, the guidelines were compiled subsequently groupwise and sent to the Convenor for editing. The edited and refined versions of each group draft was circulated to group members for their final comments prior to publication.

The final document of the Indian Guidelines on Nebulization Therapy under the aegis of NCCP(I) consists of six group drafts compiled after systematic review of evidence in order to cover each and every aspect of Nebulization therapy. The guideline document is meticulously compiled and edited with text, level of evidence and grade of recommendation, abbreviations and references.

It gives us immense pleasure to announce to this effect that the compilation of the Indian Guidelines on Nebulization Therapy under the aegis of NCCP(I) is complete and its publication is under progress. We are sure it will be immensely useful as a source of academic knowledge as well as a reference guide for practitioners, teachers, post-graduate medical students, researchers and healthcare workers in the field of Respiratory Medicine, Internal Medicine and other allied sciences which everyone would like to keep ready on their desk.
**ORGANIZERS**

President: Dr. Rajesh Solanki  
Convenor & Chairman: Dr. S. K. Katiyar  
Secretary: Dr. S. N. Gaur  
Coordinator: Dr. Nikhil Sarangdhar

**PARTICIPANTS**

<table>
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<tr>
<th>Group Convenors</th>
<th>Dr. J. C. Suri</th>
<th>Dr. Raj Kumar</th>
<th>Dr. G. C. Khilnani</th>
<th>Dr. Dhruva Chaudhry</th>
<th>Dr. Rupak Singla</th>
<th>Dr. Parvaiz Koul</th>
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<td>Dr. Dhiman Ganguly</td>
<td>Dr. V. K. Vijayan</td>
<td>Dr. Randeep Guleria</td>
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<td>Dr. H. Paramesh</td>
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<th>Group Advisors</th>
<th>Dr. D. Behera</th>
<th>Dr. Rajesh Chawla</th>
<th>Dr. Deepak Talwar</th>
<th>Dr. A. G. Ghoshal</th>
<th>Dr. P. D. Motiani</th>
<th>Dr. V. K. Arora</th>
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<td>Dr. S. N. Gaur</td>
<td>Dr. S. K. Luhadia</td>
<td>Dr. Mohan Kumar T</td>
<td>Dr. K. B. Gupta</td>
<td>Dr. Rajesh Solanki</td>
<td>Dr. A. Mahashur</td>
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<tr>
<th>Group Chairpersons</th>
<th>Dr. D. Behera</th>
<th>Dr. Rajesh Chawla</th>
<th>Dr. N. T. Awad</th>
<th>Dr. Rajendra Prasad</th>
<th>Dr. J. K. Samaria</th>
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<td>Dr. D. J. Christopher</td>
<td>Dr. Mohan Kumar T</td>
<td>Dr. Neetu Jain</td>
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<td>Dr. S. N. Gaur</td>
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<td>Dr. D. Bhattacharya</td>
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<td>Dr. Viswesvaran B</td>
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**GROUP PHOTOGRAPHS OF NCCP(I) – INDIAN GUIDELINES ON NEBULIZATION THERAPY**
NCCP(I) TEXTBOOK OF RESPIRATORY MEDICINE
an educational initiative of
NATIONAL COLLEGE OF CHEST PHYSICIANS (INDIA)

Dr. D. Behera
Editor-in-Chief

Dr. S. N. Gaur
Associate Editor

Dr. S. K. Katiyar
Associate Editor

Dr. S. K. Luhadia
Associate Editor

Dr. K. B. Gupta
Associate Editor

Dr. Bharat Gopal
Associate Editor

National College of Chest Physicians (India) published Textbook of Respiratory Medicine as part of its continuing educational activities. NCCP(I) Textbook of Respiratory Medicine has been edited by Prof. Dr. D. Behera who has been assisted by five associate editors Prof. Dr. S. N. Gaur, Prof. Dr. S. K. Katiyar, Prof. Dr. S. K. Luhadia, Prof. Dr. K. B. Gupta and Dr. Bharat Gopal. This multi-authored textbook contains 41 chapters contributed by senior and experienced authors, both from India and abroad which have been compiled in a single volume so as to provide comprehensive yet concise information on the ever expanding field of respiratory medicine, with special emphasis on the respiratory disorders prevalent in our country. The objective of this book is to address the needs of a diverse audience and become a par-excellent source of information and references for the post-graduate as well as undergraduate medical students as well as serve as a guide to busy practitioners for management of common respiratory illnesses.

NCCP(I) Textbook of Respiratory Medicine begins with an overall review of the respiratory system, including clinical examination, respiratory symptomatology and physiology, followed by a wide array of chapters on diverse topics, taking care to cover all respiratory diseases common to our country. The text is well referenced and lucid in style for better language flow and adequately supplemented by tables, figures and diagrams. Respiratory disorders have been covered according to their prevalence in our country and relevance in clinical practice. Chapters have been well compiled and edited in order to provide updated and relevant information, keeping in mind that the textbook is meant for a diverse readership comprising of post-graduate, undergraduate and post-doctoral medical students of Respiratory and Internal Medicine as well as practicing Chest Physicians. Overall the textbook is well illustrated and informative, a much sought-after valuable addition to the libraries of medical colleges and teaching institutions and has evolved into a highly popular publication as it highlights the current status and updates on various respiratory diseases and their diagnosis and management.

TEXTBOOK CHAPTERS

1. Physical Examination of Respiratory System
2. Common Clinical Symptoms
3. Growth, Development and Morphology of the Respiratory System
4. Normal Respiratory Physiology
5. Defense Mechanisms of the Respiratory System
6. Diagnostic Methods in Respiratory System
7. Interventional Pulmonology & Electromagnetic Navigation
8. Antimicrobials in Respiratory Medicine
9. Pneumonias
10. Anaerobic Pleuropulmonary Infections
11. Parasitic Lung Diseases
12. Tropical Pulmonary Eosinophilia
13. Lung Abscess
14. Bronchiectasis
15. Tuberculosis
16. Non-tubercular Mycobacterial Diseases
17. Bronchial Asthma
18 A. Chronic Obstructive Pulmonary Disease
18 B. Rehabilitation in Chronic Obstructive Pulmonary Disease
19. Aerosol Therapy
20. Respiratory Failure
21. Cor Pulmonale
22. Oxygen Therapy
23. Pulmonary Embolism
24. Acute Respiratory Distress Syndrome
25. Lung Cancer
26. Pulmonary Neoplasms other than Bronchogenic Carcinoma
27. Smoking and Lung Diseases
28. Air Pollution and Respiratory Diseases
29. Essentials of Polysomnography and Recommendations in Adults
30. Sarcoidosis
31. Lungs in Collagen Vascular Diseases and other Systemic Diseases
32. Vasculitis and the Lungs
33. Interstitial Lung Diseases
34. Occupational Lung Diseases
35. Hypersensitivity Pneumonitis
36. Disorders of the Diaphragm and Chest Wall
37. Congenital Anomalies of the Respiratory System
38. HIV and Respiratory Diseases
39. Lung Transplantation
40. Non-invasive Ventilation in Acute Respiratory Failure
41. Pleural Diseases
Dear Colleagues,

You are very well aware that National College of Chest Physicians (India) publishes a National Directory of Chest Physicians in India every five years, with the objective of providing contact details of all Chest Physicians across the country. The last NCCP(I) Directory was published in 2016. We thank all members and fellows of NCCP(I) and request You to inform us in case of any change of residential, official or postal address, mobile number and E-mail ID in order for us to prepare the forthcoming Directory, for which You can fill up the Directory Entry Form in this Newsletter and send by post to the NCCP(I) secretariat address below (or download from our website www.nccpindia.org and send by E-mail to sngaur9@gmail.com).

We also welcome all to submit their plans for events and activities for the forthcoming year. In addition, we would like to ensure You are aware of all your NCCP(I) membership benefits, which include:

- Electronic Voting for Yearly Elections to NCCP(I) Governing Council through E-voting form sent to Your E-mail ID
- Subscription to Indian Journal of Chest Diseases and Allied Sciences (Quarterly issues)
- NCCP(I) National Directory of Chest Physicians (Every 5 years)
- Discounts in Registration for NCCP(I) E-Courses (CPMeC & IPeC)
- Discounts in Registration for participating at National Conferences (including NAPCON), International Conferences (Gulf-Thoracic and others), State Conferences and Workshops and other educational activities under the aegis of NCCP(I)
- Travel Grants for National & International Conferences
- Communications through E-mail and Invitation to attend NCCP(I) Annual General Body Meeting
- Access to NCCP(I) Newsletter – Lung Bulletin (Biennial issues starting from this year)

Should You need any assistance or have any queries regarding Your NCCP(I) Membership or Benefits, please feel free to contact us, our support is always available to help You.

COMMUNICATE WITH US

www.nccpindia.org

Dr. S. N. Gaur, Gaur Clinic, 130 - A, Patparganj Village, Delhi – 110091

sngaur9@gmail.com, ncsarangdhar@rocketmail.com

(+91) 9811271916, (+91) 9029429015
Dear Colleagues,

You will be happy to know that we are going to bring out soon a textbook on ‘Emergencies in Respiratory Medicine’ under the aegis of the National College of Chest Physicians (India) which will be published by Jaypee Brothers. This book is the first of its kind and an excellent step taken by the College in the field of Medical Education. It contains several chapters written by pioneering experts in the field of Respiratory Medicine of our vast country. An attempt has been made to cover each and every aspect of Respiratory Emergencies. Each chapter is meticulously written and edited with abstract, key words, introduction and description of the topic including information on diseases and conditions along with references. It’s our immense pleasure to announce to this effect the work of compilation is under progress. We are sure it will be immensely useful as a source of academic and clinical knowledge for practitioners, teachers, post-graduate medical students and researchers in the field of Respiratory Medicine and other allied sciences which everyone would like to keep ready and have with them.

My observations and experience since last two decades as postgraduate teacher has led to writing a book. Primary aim of the book is to make the beginners in respiratory medicine to understand the basic concepts in a simple way. The book has three sections – interactive case discussions, discussion on chest images and multiple choice questions. One can easily understand the topics as I have tried to present the discussion in interesting way with clinical touch. One can self-assess using the discussions and MCQs.

I express my sincere gratitude to Professor Dr. S. N. Gaur, Honorary Secretary, NCCP(I) for suggesting me to write this book and constantly encouraging me in the process. My sincere gratitude to Professor Dr. P. D. Motiani, President, NCCP(I), for his guidance. I am indebted to NCCP(I) for releasing the book during NAPCON 2020 from January 27-31, 2021. My sincere gratitude to my teacher Prof. Dr. V. K. Arora who has been a guide in my academic career.

I also express my gratitude to all who have contributed and helped me in compiling this book. My sincere thanks to all the Past and present post-graduates in our department who helped me in compiling this book, especially in collecting the images and patient details.
The American Thoracic Society (ATS) and American College of Chest Physicians (ACCP) have agreed to the nomination of 2 delegates as representatives of the National College of Chest Physicians (India) to attend and participate in their annual conferences. These NCCP(I) nominees (Fellows only) will be provided complimentary registration and local accommodation by the Organizers. Travel has to be arranged by the nominees themselves.

In addition, NCCP(I) is providing travel grant worth a fixed amount to its Members and Fellows for participation in national* and international conferences* as follows:
- Rs. 20,000/- for national conferences in India*
- Rs. 80,000/- for international conferences in U.S. & Canada
- Rs. 60,000/- for international conferences in other countries

Those NCCP(I) Fellows or Members interested, can apply to Hon. Secretary, NCCP(I), preferably before March each year (as ATS conference is usually held in May and ACCP annual meeting in October of the calendar year) for consideration providing details on a request letter as follows:

**CHECK-LIST**

1. Name, Dates and Venue of conference
2. Details of Participation in the concerned conference (Delegate/Faculty)
3. Letter of Abstract Acceptance or Invitation at the concerned conference
4. Applicant Particulars (Full name, age in years, gender, Postal address, E-mail ID & Mobile number for communication)
5. Present designation/affiliation
6. NCCP(I) Life Fellowship (LF) or Life Membership (LM) number
7. Number of NAPCONS attended in last 5 years
8. Number of total conferences (national + international) attended in last 5 years
9. Number of publications in last 5 years (attach list)
10. Forwarding letter preferably signed by Head of Department or Institution or a Fellow of NCCP(I)
11. Hard copies of receipts for reimbursement (Registration, Travel, Stay) with breakup of expenses
12. Disclaimer or statement whether availing travel grant/other monetary assistance from any other source for the same

The grant applications should be sent by post addressed to Hon. Secretary, NCCP(I) at the following address:
Dr. S. N. Gaur, Gaur Clinic, 130-A, Patparganj Village, Delhi – 110091. Phone: +91- 9811271916
E-mail: sngaur9@gmail.com

All applicant requests will be scrutinized by a Credential committee at NCCP(I) Governing Council meeting, for those selected, expenses as per norms will be reimbursed by postal cheque in the name of the applicant only.

**NCCP(I) – Prof. S. N. Gaur Young Scientist Award at NAPCON**

- The applicant should not be more than 35 years of age and first author of the abstract submitted for oral presentation at NAPCON mentioning selection for NCCP(I) – Prof. S. N. Gaur Young Scientist award.
- All abstracts forwarded by the NAPCON for NCCP(I) – Prof. S. N. Gaur Young Scientist Award will be scrutinized by an Academic Committee specially constituted by NCCP(I) for this purpose. A maximum of 9 abstracts will be selected for presentation in this award session and the presenters informed accordingly prior to the conference.
- All selected presenters will receive Rs. 5000/- as travel grant by cheque and a certificate of presentation, in addition to certificates and award adjudged for the First, Second and Third prizes.

*For NAPCON, NCCP(I) - Prof. Dr. S. N. Gaur Young Scientist Award is available for Young Scientists. NAPCON Registration is Discounted for All Life Members and Fellows of NCCP(I) and ICS.
For all medical students, continuing medical education (CME) programmes, seminars, updates, workshops and conferences form an integral part of their training apart from the bedside clinical teaching, ward rounds and lectures imparted at medical colleges or teaching institutions. Quiz competition comes as a refreshing change from all these academic activities to enhance and fine-tune their learning and it is something they look forward to with excitement and enthusiasm. To encourage and recognize the budding potential in our Chest Physicians of tomorrow, National College of Chest Physicians (India) undertook the initiative to conduct Post-graduate Quiz Competition in Respiratory diseases with the objective to promote scientific temper in PG students of Pulmonary medicine in India, state-wise as well as nationally.

The NCCP(I) State PG quiz in Respiratory diseases was organised in 15 states at medical colleges/teaching institutions, keeping nationally renowned faculty in Pulmonary medicine as state PG quiz anchors. The first two winners in order of merit in each state were awarded NCCP(I) prize certificate and a cash award of Rs. 5000/- each, with a certificate of participation distributed to all participants. The NCCP(I) State PG quiz programme was a grand success, with a record participation of 290 PG students from different states across the country.

To keep up and carry forward this scientific temper, it was necessary to create a national academic platform to acknowledge and reward this young talent identified among PG students of Pulmonology at state level. Keeping this objective in mind, National College of Chest Physicians (India) organised an All-India PG quiz competition in Respiratory diseases for the first time in our country. Members of the winning team (2 students) from each state were provided a scholarship to participate in the All-India PG quiz with arrangements for accommodation and air travel. All 30 students confirmed their participation and attended the NCCP(I) All- India PG quiz.

The NCCP(I) All-India PG quiz was conducted on Saturday, 21st December 2019 during the 74th National Conference of TB & Chest diseases from 5:00 to 7:15 p.m. at Hotel Leela Palace, Chennai. Dr. Vishnu Sharma, Professor & Head, Department of Respiratory medicine, A J institute of medical sciences, Mangalore was invited to be the National Quiz Master. The PG quiz was inaugurated by Dr. S. N. Gaur (Secretary), Dr. S. K. Katiyar (Chairman, Academic Forum) and Dr. Nikhil Sarangdhar (Coordinator) from NCCP(I) who welcomed all PG students and congratulated them for standing first in the PG quiz in their respective states. After wishing all success, the quiz programme was outlined by Dr. Vishnu Sharma. The preliminary round consisted of 42 multiple choice questions (MCQs) given to all participants to be answered within 20 minutes, at the end of which all answer sheets were collected and each question transparently discussed along with the answer by powerpoint presentation through on-screen display. The individual scores of both students in each team in the preliminary round were combined to compute the final score of each team. The team members from Delhi, Tamil Nadu, Karnataka and Kerala scored the highest in the preliminary round and were selected to participate in the grand round on stage with a buzzer in front. Coordination of each team and functioning of audio-visuals were cross-checked twice and verified with each team before the grand round commenced. 9 rounds of question-answer sessions with first-best answer type pattern were conducted, the answers being discussed at the end of each session. A maximum interval of 5 seconds between pressing the buzzer to answering the question by the respective team was permitted. 10 marks were awarded for correct answers, with negative marking of 5 marks for wrong answers to the respective team. The teams from Delhi, Tamil Nadu, Karnataka and Kerala scored 55, 25, 10 and 35 marks respectively and were congratulated for their performance in the grand round.

For the award ceremony Dr. V. K. Arora, Vice-Chairman, TB association of India and Past-President of NCCP(I) was invited to the dias along with Dr. S. N. Gaur, Dr. S. K. Katiyar, Dr. Nikhil Sarangdhar and Dr. Vishnu Sharma. The first prize carried NCCP(I) prize certificate, cheque of Rs. 25000/- each and plaque of “D.B. Gupta budding talent award” and was awarded to Dr. Tanmay Jain and Dr. Arunachalam from Delhi. The second prize carried NCCP(I) prize certificate and cheque of Rs. 15000/- each and was awarded to Dr. Mahroofa EV and Dr. Archana LP from Kerala. The third prize carried NCCP(I) prize certificate and cheque of Rs. 10000/- each and was awarded to Dr. Amal Johnson and Dr. Vaseema Tabassum from Tamil Nadu. All winners and participants were congratulated.

As a token of appreciation, a certificate of participation from NCCP(I) was personally awarded to all 30 PG students, with congratulations for their efforts and best wishes for their future. A special certificate of appreciation was awarded to Dr. Vishnu Sharma on behalf of NCCP(I) for his efforts towards conducting the NCCP(I) All-India PG quiz in a highly transparent and professional manner.
<table>
<thead>
<tr>
<th>State</th>
<th>PG Quiz Anchor(s)</th>
<th>Venue of State PG Quiz</th>
<th>Winning Team Names</th>
<th>Institute</th>
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<tbody>
<tr>
<td>Delhi</td>
<td>Dr. Vivek Nangia</td>
<td>Fortis Hospital, Vasant Kunj, New Delhi</td>
<td>Dr. Arunachalam Dr. Tanmay Jain</td>
<td>NITRD, Delhi</td>
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<tr>
<td>Gujarat</td>
<td>Dr. Savita Jindal Dr. Sanjay Tripathi</td>
<td>LG Hospital, Ahmedabad</td>
<td>Dr. Palak Bhatt Dr. Trupti Gadhavi</td>
<td>Ahmedabad municipal corporation MET medical college, Ahmedabad</td>
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<tr>
<td>Haryana</td>
<td>Dr. Dhrupa Chaudhry</td>
<td>PGIMS, Rohtak</td>
<td>Dr. Sameer Kotalwar Dr. Ankit Aggarwal</td>
<td>Medanta hospital - the medcity, Gurgaon</td>
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<tr>
<td>Himachal Pradesh</td>
<td>Dr. Malay Sarkar</td>
<td>Indira Gandhi medical college, Shimla</td>
<td>Dr. Swadesh Mohanty Dr. Aseem Sirkeck</td>
<td>Indira Gandhi medical college, Shimla</td>
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<tr>
<td>Karnataka</td>
<td>Dr. Shashi Bhushan</td>
<td>PMSSY Super Speciality block, Victoria hospital, Bengaluru</td>
<td>Dr. Rashmitha MT Dr. Parvathy Pillai</td>
<td>Bangalore medical college &amp; research institute, Bengaluru</td>
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<tr>
<td>Kerala</td>
<td>Dr. Kiran Vishnu Narayan</td>
<td>Indraprastha hotel, Kottayam</td>
<td>Dr. Mahrooфа EV Dr. Archana LP</td>
<td>Institute of Chest diseases, Government medical college, Kozhikode</td>
</tr>
<tr>
<td>Maharashtra</td>
<td>Dr. Sushant Meshram</td>
<td>Government medical college, Nagpur</td>
<td>Dr. Alina Alexander Dr. Abhishek Singh</td>
<td>Government medical college, Nagpur</td>
</tr>
<tr>
<td>Odisha</td>
<td>Dr. Narayan Mishra</td>
<td>Hotel Spectrum, Berhampur</td>
<td>Dr. Biswajit Pati Dr. Saurabh Gupta</td>
<td>VIMSAR med. college, Burla</td>
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<tr>
<td>Puducherry</td>
<td>Dr. S. Yuvarajan</td>
<td>SMV medical college &amp; hospital, Puducherry</td>
<td>Dr. Naren Chandra Dr. Selvaraja</td>
<td>JIPMER, Pondicherry</td>
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<tr>
<td>Punjab</td>
<td>Dr. Vishal Chopra</td>
<td>Government medical college, Patiala</td>
<td>Dr. Leena Chopra Dr. Jain Thomas</td>
<td>Government medical college, Patiala</td>
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<td>Tamil Nadu</td>
<td>Dr. V. Vinod Kumar</td>
<td>Government hospital of Thoracic Medicine, Tambaram, Chennai</td>
<td>Dr. Amal Johnson Dr. Vaseema Thabassum</td>
<td>Apollo hospital, Chennai</td>
</tr>
<tr>
<td>Telangana</td>
<td>Dr. Sailaja K Dr. R. Vijai Kumar</td>
<td>Mediciti institute of medical sciences, Hyderabad</td>
<td>Dr. Lavanya K Dr. Govardhan Reddy</td>
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<td>Uttar Pradesh</td>
<td>Dr. Surya Kant</td>
<td>King George medical university, Lucknow</td>
<td>Dr. Shiv Kumar Verma Dr. Vignesh K</td>
<td>King George medical university, Lucknow</td>
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<tr>
<td>Uttarakhand</td>
<td>Dr. Girish Sindhwani</td>
<td>AIIMS Rishikesh</td>
<td>Dr. Kumar Nishant Dr. Sandeep Kumar</td>
<td>Himalayan institute of medical sciences, Dehradun</td>
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<tr>
<td>West Bengal</td>
<td>Dr. Shelley Shamim</td>
<td>Calcutta National medical college, Kolkata</td>
<td>Dr. Riksoam Chatterjee Dr. D Suresh Kumar</td>
<td>SSKM medical college, Kolkata</td>
</tr>
</tbody>
</table>

**Report of NCCP(I) State PG Quiz in Respiratory Diseases**

Dr. Vishnu Sharma  
**Quiz Master, NCCP(I) All-India PG Quiz in Respiratory Diseases**  
**Professor & Head, Department of Respiratory medicine, A J institute of Medical Sciences, Mangalore**

Quiz is basically a form of mind sport, in which the players (as individuals or in teams) attempt to answer questions correctly. The word “Quiz” may have originated in student slang and it means to “test knowledge”. Quiz is used in education to test knowledge, abilities or skills of individuals. I have been conducting quiz for respiratory medicine post-graduate students since the last ten years. Post-graduate quiz during a conference with provision of scholarship to meritorious students always generates a lot of excitement, with all students participating enthusiastically. While compiling quiz questions emphasis is laid on must-know facts for the students. A properly conducted quiz with academic focus helps to enhance post-graduate learning. It was a great honour for me to be invited by the National College of Chest Physicians (India) as the Quiz Master to conduct their All-India level quiz for PG students of respiratory medicine. The NCCP(I) All-India PG quiz programme at Chennai was a grand academic success and succeeded in achieving its objective of promoting scientific temper and identifying young talent from the budding post-graduates, who are one day going to be the future of Pulmonary Medicine in our country.
PHOTOGRAPHS OF NCCP(I) STATE PG QUIZ

DELHI

GUJARAT

HARYANA

HIMACHAL PRADESH

KARNATAKA

KERALA

MAHARASHTRA

ODISHA

PUDUCHERRY

PUNJAB

TAMIL NADU

TELANGANA

UTTAR PRADESH

UTTARAKHAND

WEST BENGAL
PHOTOGRAPHS OF NCCP(I) ALL – INDIA PG QUIZ

A. Dr. Vishnu Sharma (Quiz Master) addressing all participants
B & C. Teams qualifying for Grand Round
B. Dr. Arunachalam & Dr. Tanmay Jain (Delhi), Dr. Amal Johnson & Dr. Vaseema Thabassum (Tamil Nadu)
C. Dr. Parvathy Pillai & Dr. Rashmitha MT (Karnataka), Dr. Archana EV & Dr. Mahroofa LP (Kerala)
D. Award of 1st Prize to Dr. Arunachalam & Dr. Tanmay Jain
E. Award of 2nd Prize to Dr. Archana EV & Dr. Mahroofa LP
F. Award of 3rd Prize to Dr. Amal Johnson & Dr. Vaseema Thabassum
G. Group photograph of All participants, Quiz Master – Dr. Vishnu Sharma, Dr. V. K. Arora, and NCCP(I) team – Dr. S. N. Gaur (Secretary), Dr. S. K. Katiyar (Chair, Scientific Committee) and Dr. Nikhil Sarangdhar (Coordinator)
The National College of Chest Physicians (India) organized several conferences since it was formed. The first conference of NCCP(I) (then IACD) was hosted in 1960 at New Delhi jointly with the Association of Physicians of India and other specialist organisations. Subsequent annual conferences were also held jointly with the Association of Physicians of India till 1963, in which year the Association sponsored the 8th International Congress on Chest Diseases in New Delhi. The following year, the Association held its fourth annual conference independently at New Delhi to which the President of the Royal College of Physicians of Edinburgh was a special invitee and guest of honour. In 1974, it held its annual conference jointly with the Tuberculosis Association of India.

Since 1989, NCCP(I) organised its annual conferences, called NACCON (National Chest Conference). These conferences were very successful and popular and were chaired by the then Presidents of NCCP(I). The Indian Chest Society (ICS) was also hosting its annual national conference, called NCRD (National Congress on Respiratory Diseases). In greater interest of the Pulmonary fraternity of our country, the need to have a united conference of both NCCP(I) and ICS, the two largest national bodies on Pulmonary Medicine was felt. After several positive negotiations and meetings spread over almost 8 years, the President, Secretary and Governing Bodies of both the NCCP(I) and the ICS, evolved a consensus to conduct their joint national conference together. From 1999, the NCCP(I) with ICS is having Joint National Conference on Pulmonary Diseases, called NAPCON. The guidelines for organising NAPCON were finalized to assist the organizers and also to have uniformity in organization and maintain a high academic standard of the scientific programme of NAPCON. NCCP(I) and ICS alternatingly select the venue and organisers of NAPCON each year and a similar turn is followed for selection of Chairperson of the Scientific Committee, which consists of equal number of members from both associations. To promote national integration, each year NAPCON is hosted at a different city and has in turn been organised in the north, south, east and western regions of our country, truly reflecting a pan-Indian character. The NAPCON logo, selected jointly by both associations shows two hands representing both NCCP(I) and ICS working together in harmony.

NAPCON as a joint venture of NCCP(I) and ICS has been a grand success right from the beginning, providing opportunity to every person in the specialty of Pulmonary Medicine to come together under one roof to achieve the maximum scientific benefit. NAPCON has been attended by eminent faculty from the American Thoracic Society (ATS), American College of Chest Physicians (ACCP), European Respiratory Society (ERS), Asia Pacific Society of Respirology (APSR) and other Chest Specialists from abroad and from neighbouring Asian countries. The scientific programmes of NAPCONs are also state-of-the-art and widely acclaimed internationally. Not only Chest Physicians but also Physicians, Critical care specialists, Radiologists, Infectious disease specialists, Microbiologists and Pathologists, Cardiologists and Thoracic Surgeons and learned faculties from other allied specialties are invited to deliver guest lectures or participate in debates, panel discussions, practice changing research and symposia to enrich the diversity and academic content of the scientific programme. The scientific programme covers a plethora of topics on different aspects of respiratory diseases and other allied sciences including critical care, pneumonia, tuberculosis, viral and other respiratory infections, diffuse lung diseases, asthma, COPD, interstitial lung diseases, sleep disorders, cardio-thoracic surgery, lung cancer, bronchoscopy, thoracoscopy and other thoracic interventions, pleural diseases, pulmonary vascular disorders, pediatric pulmonology, respiratory allergy and immunology, environmental and occupational problems, pulmonary imaging, sports medicine and rehabilitation apart from several other topics to constitute a unique academic feast.

Apart from the much-awaited scientific programme, delegates are also given the opportunity to participate in several workshops on a wide variety of topics like pulmonary function tests, imaging, research methods and scientific paper writing, critical care, mechanical ventilation, bronchoscopy and interventional pulmonology, allergy, sleep disorders, interstitial lung diseases, tuberculosis and others to refine their technical knowledge and skills. Satellite symposia and free paper oral and poster presentations add to the academic flavour. The Young budding Chest Physicians and Post-graduates eagerly look forward to the opportunity to present their original research work and more than 700 different abstracts are presented at NAPCON year after year. NAPCON is truly a complete scientific and cultural feast, providing opportunity for many pulmonologists and doctors of other specialities of all ages to meet, interact and have discussion with each other to share their knowledge and experiences to evolve strategies for better management of respiratory diseases.

Right since its inception, NAPCON has grown from strength to strength each year to become one of the largest conferences of Pulmonary diseases in Asia and globally with attendance of nearly 3000 delegates annually. NAPCON is a unique success story in itself, a testimony of unity, strength and cooperation between NCCP(I) and ICS and has evolved into a much sought-after ‘Brand name’ and ‘Status symbol’ popular amongst the Chest Physicians and Post-Graduates in India and abroad.
NAPCONs from 1999 till date

<table>
<thead>
<tr>
<th>Sr. No.</th>
<th>YEAR</th>
<th>VENUE</th>
<th>ORGANISING CHAIRMAN</th>
<th>ORGANISING SECRETARY</th>
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<td>1.</td>
<td>1999</td>
<td>Delhi</td>
<td>Dr. J. C. Suri</td>
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<td>2.</td>
<td>2000</td>
<td>Kanpur</td>
<td>Dr. S. K. Katiyar</td>
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<td>2001</td>
<td>Mumbai</td>
<td>Dr. J. C. Kothari</td>
<td>Dr. Rohini Chowgule</td>
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<td>4.</td>
<td>2002</td>
<td>Jaipur</td>
<td>Dr. T. N. Sharma</td>
<td>Dr. N. K. Jain</td>
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<td>5.</td>
<td>2003</td>
<td>Coimbatore</td>
<td>Dr. T. K. Moinudeen</td>
<td>Dr. T. Mohan Kumar</td>
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<td>2004</td>
<td>Ahmedabad</td>
<td>Dr. Gautam Bhagat</td>
<td>Dr. Rajesh Solanki</td>
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<td>2005</td>
<td>Kolkata</td>
<td>Dr. A. K. Ghosh</td>
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<td>2006</td>
<td>Nagpur</td>
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<td>Dr. B. O. Tayade</td>
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<td>2007</td>
<td>Chandigarh</td>
<td>Dr. S. K. Jindal</td>
<td>Dr. Dheeraj Gupta</td>
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<td>2008</td>
<td>Lucknow</td>
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<td>Dr. Rajendra Prasad</td>
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<td>2009</td>
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<td>Dr. C. Ravindran</td>
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<td>Jodhpur</td>
<td>Dr. P. D. Motiani</td>
<td>Dr. K. C. Agarwal</td>
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<td>Dr. V. K. Vijayan</td>
<td>Dr. Raj Kumar</td>
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<td>14.</td>
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<td>Bhubaneshwar</td>
<td>Dr. N. K. Gacchayat</td>
<td>Dr. Narayan Mishra</td>
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<td>15.</td>
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<td>Dr. Vijayalakshmi Thanasekaraan</td>
<td>Dr. B. Rajagopalan</td>
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<td>Dr. A. S. Sachan</td>
<td>Dr. Rakesh Bhargava</td>
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<td>17.</td>
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<td>Jaipur</td>
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<td>Dr. Virendra Singh</td>
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<td>18.</td>
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<td>Mumbai</td>
<td>Dr. K. C. Mohanty</td>
<td>Dr. Agam Vora</td>
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<td>19.</td>
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<td>Dr. A. G. Ghoshal</td>
<td>Dr. Dhrubajyoti Roy</td>
</tr>
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<td>20.</td>
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<td>Ahmedabad</td>
<td>Dr. Rajesh Solanki</td>
<td>Dr. Raj Bhagat</td>
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<td>21.</td>
<td>2019</td>
<td>Kochi</td>
<td>Dr. C. Ravindran</td>
<td>Dr. Rajesh Venkat</td>
</tr>
<tr>
<td>22.</td>
<td>2020</td>
<td>Virtual</td>
<td>Dr. S. N. Gaur</td>
<td>Dr. Nikhil Sarangdhar</td>
</tr>
</tbody>
</table>

All the twenty-one NAPCONs till date were a grand success, appreciated by members and fellows of both NCCP(I) and ICS, faculty, delegates and post-graduate students, as well as the foreign faculty and delegates. Credit for this success goes to team-work from NCCP(I) and ICS, the Organising Committee and the Scientific Committee for working hard in tandem to ensure fabulous conferences of high repute which are appreciated and acclaimed internationally. We are confident the same spirit will continue, year after year, and we look forward to greater participation as well as better conferences in future.
Introduction:  
The respiratory system is essentially concerned with exchange of gases between the inspired air and blood in the alveolar capillaries. It provides a surface for transfer of gases through which blood gets rid of carbon dioxide and absorbs oxygen, in doing so, the pressures of oxygen and carbon dioxide are maintained in the arterial blood at 100 mm Hg and 40 mm Hg respectively while breathing ambient air at sea level.

The process involves ventilation, concerned with movement of gases along the airways to and out of the alveoli, intrapulmonary distribution of air (V) and adequate perfusion of capillaries (Q) matching ventilation and perfusion, thus diffusion of gases over the wide area of the alveolar-capillary membrane. These processes are closely integrated. In addition to the gaseous exchange, lungs play an important role in the maintenance of acid-base balance as they offer a surface for elimination of CO₂ produced in the body during metabolic processes.

History:  
Pulmonary function tests took root with the introduction of spirometer (Latin: spiro-to breathe; meter-to measure) to measure the lung function of a person. The instrument, Spirometer was invented by an English Surgeon, John Hutchinson in 1840s. The original instrument was as tall as an adult person and it consisted a calibrated bucket placed upside down in water. The volume of exhaled air from fully inflated lungs was measured by exhaling into a tube leading to the bucket. This measured the ‘vital capacity’ (term coined by Hutchinson).

Though Hutchinson advocated this ‘capacity for life’ instrument is useful it did not find widespread use and was used in sanatorium to determine the lung volumes of patients with tuberculosis. It was only after a century French Physician Tiffeneau introduced in 1950, the forced measurement of air volumes in a time frame such as forced expiratory volume in 1 second.

A waterless spirometer produced by Jones Medical replaced water type spirometer in 1960. It opened a new era in the diagnostic field in respiratory disorders. Since then spirometry has found a place in the diagnosis and management of obstructive and restrictive lung diseases, pre-operative assessment of patients, and evaluation of cardiac disorders. Spirometry paved the way for various pulmonary function tests involving ventilation and diffusion.

Pulmonary Function Tests  
The clinical significance of pulmonary function tests has been established exemplifying the statement of Sterling ‘the Physiology of today is the Medicine tomorrow.’

Pulmonary function tests are undertaken to find out whether the patient has any lung disease. The results of the pulmonary function tests of a given individual are compared with those obtained from a normal population of comparable height, age and gender. The test is considered abnormal if it falls outside the range based on the standard error of the estimate in which 95% of normal lies.

The methods and measurements obtained in pulmonary function testing are as follows:

1. Airway function: The assessment of airway function includes i) Spirometry (vital capacity VC, forced vital capacity, FVC, forced expiratory volume in 1 second FEV₁, FEV₁/FVC%, tidal volume), ii) flow volume loops, iii) peak expiratory flow rate, PEFR by peak flow meter and iv) airway resistance by body plethysmography.
2. Lung volumes: The following subdivisions of lung volumes are measured utilising spirometry, gas dilution technique and plethysmography (TV, total lung capacity, TLC, functional residual capacity, FRC, and residual volume. RV).
3. Diffusion: Diffusion capacity refers to the magnitude of carbon monoxide gas transfer (DLCO₂) on the volume of the pulmonary capillary bed and the matching between ventilation and perfusion within the lungs. The technique is utilising single breath diffusing capacity.
4. Respiratory muscle function: Breathlessness may be due to muscles to inflate the lungs. Peak inspiratory and peak expiratory maximal mouth pressures are the simple tests to evaluate respiratory muscle strength.
5. Exercise tests: Exercise tests are helpful in demonstration of integrated functions of cardiorespiratory system.
References:

2. Light RW. Clinical Pulmonary function testing, Exercise testing, a Disability evaluation in Clinical Medicine: Essentials of pulmonary and critical care Medicine, George RB, Light RW, Matthay MA, Mathay RA (eds), 3rd edn, Baltimore, Williams & Wilkins, 1995: 151
Lung Volumes and Capacities

Introduction:

Lung volumes (also known as respiratory volumes) refers to the volume of air in the lungs at a given time during the respiratory cycle. Lung capacities are two or more lung volumes added together. The measurement of lung volumes and capacities is an integral part of pulmonary function testing. These volumes tend to vary, depending on the depth of respiration, ethnicity, gender, age, body composition and in certain respiratory diseases. Changes in lung volumes are seen with diseases of lung, pleura, diaphragm and chest wall and hence they play a vital role as a diagnostic tool in Pulmonary Medicine. There are 4 lung volumes and 4 lung capacities, as follows

<table>
<thead>
<tr>
<th>LUNG VOLUMES</th>
<th>LUNG CAPACITIES</th>
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</thead>
<tbody>
<tr>
<td>1) Tidal Volume</td>
<td>1) Inspiratory Capacity</td>
</tr>
<tr>
<td>2) Inspiratory Reserve Volume</td>
<td>2) Vital Capacity</td>
</tr>
<tr>
<td>3) Expiratory Reserve Volume</td>
<td>3) Functional Residual Capacity</td>
</tr>
<tr>
<td>4) Residual Volume</td>
<td>4) Total Lung Capacity</td>
</tr>
</tbody>
</table>

Definitions of Lung Volumes:

- **Tidal volume (TV):** The volume of air inhaled or exhaled with each breath during resting quiet respiration in the respiratory cycle is called the tidal volume. It is around 500 ml in both males and females.
- **Inspiratory reserve volume (IRV):** It is the maximum volume of air that can be inhaled after a normal inspiration. It is around 3100 ml in males and 2100 ml in females.
- **Expiratory reserve volume (ERV):** It is the maximal volume of air which can be expired after a normal expiration. It is around 1200 ml in males and 800 ml in females.
- **Residual volume (RV):** It is the volume of air remaining in the lungs after maximal expiration. It is about 1200 ml.

Definitions of Lung Capacities (Figure 1):

- **Inspiratory capacity (IC):** It is the maximum volume of air that can be inspired from the resting expiratory level. It is calculated from the sum of tidal volume and inspiratory reserve volume. It is around 3600 ml in males and 2600 ml in females. **IC = TV + IRV**
- **Vital capacity (VC):** It is the maximum volume of air that can be exhaled after a maximal inspiration. It is calculated by summing tidal volume, inspiratory reserve volume, and expiratory reserve volume. It is around 4800 ml in males and 3400 ml in females. **VC = IRV + TV + ERV and VC = TLC – RV**
- **Functional residual capacity (FRC):** It is the volume of air which remains in the lung at end of normal expiration. It is calculated by adding together residual volume and expiratory reserve volume. It is around 2400 ml in males and 2000 ml in females. **FRC = ERV + RV**
- **Total lung capacity (TLC):** It is the volume of air contained in the lung at the end of maximal inspiration. It is the maximum volume of air the lungs can accommodate and is calculated by summation of the four primary lung volumes. It is around 6000 ml in males and 4600 ml in females. **TLC = IRV + TV + ERV + RV, also TLC = VC + RV and TLC = FRC + IC**
Factors determining Static Lung Volumes:

- **Age**: lung volumes increase with growth of child
- **Old age**: increase in RV & FRC, decrease in ERV
- **Gender**: males have larger lung volumes
- **Height**: taller individuals have larger lung volumes
- **Race**: different in different ethnicities
- **Reduced in recumbent position than standing**
- **Pregnancy**: FRC decreases by 18-20% due to compression of the diaphragm by the uterus
- **High altitude residents life-long living in relatively hypoxic environments**: have relatively higher lung volumes

Measurement of Lung Volumes:

Various lung volumes and capacities which do not include residual volume (such as vital capacity, inspiratory capacity, inspiratory and expiratory reserve volume and tidal volume) can be measured by simple spirometry. They are compared with predicted values for the given population and are expressed as percent of the predicted value. Value within 80-120% predicted is considered normal. Any value beyond 80-120% predicted is considered abnormal.

All lung volume and capacities are measured directly by spirometer except RV, FRC, TLC. This is because the air in the residual volume of the lung cannot be expired into the spirometer and this volume constitutes part of FRC and TLC.

**RV is measured indirectly in 3 steps**

1) FRC is typically measured using one of the three techniques
   - Helium dilution
   - Nitrogen washout
   - Body plethysmography

2) ERV is measured spirometrically, and

3) RV is calculated as the difference between FRC and ERV

**TLC is calculated by adding the inspiratory capacity, measured from simple spirometry to the FRC. The addition of RV and VC (from spirometry) also provides an estimate of TLC.**

Comparison of plethysmography versus nitrogen and helium dilutional methods

1) Body plethysmography is the gold standard for measurement of lung volumes, particularly in the setting of significant airflow obstruction

2) Advantage of plethysmography is that it is not affected by distribution of ventilation in cases of airways obstruction

3) FRC obtained by dilutional methods is less than that obtained by plethysmography, especially in diseases characterized by air trapping like emphysema and bullous diseases.

4) The difference between FRC by plethysmography and by dilutional methods estimates the volume of gas contained in a non-communicating bulla or cyst

5) Plethysmography cannot be used in patients with marked obesity, skeletal abnormalities or claustrophobia.
Measurement of TLC using imaging techniques:

In subjects with a limited ability to cooperate, radiographic lung volumes using chest x-rays, high resolution computed tomography (HRCT) chest and Magnetic resonance imaging (MRI)may be more feasible than physiological measurements. Measurements of TLC using the chest radiograph or HRCT correlate within 15% of those obtained by body plethysmography\(^1,2\).

Since the TLC is equivalent to the amount of air seen in the lungs on a chest radiograph taken at maximal inspiration, it is important that the subject inhales maximally as the image is created.

Table 1 summarises various methods for measuring lung volumes along with its salient features

<table>
<thead>
<tr>
<th>METHOD</th>
<th>LUNG VOLUME</th>
<th>REMARKS</th>
</tr>
</thead>
<tbody>
<tr>
<td>Multiple-breath Helium (He) Dilution</td>
<td>FRC</td>
<td>Simple, relatively inexpensive; affected by distribution of ventilation in moderate or severe obstruction; multiple-breath; requires IC, ERV to calculate other lung volumes</td>
</tr>
<tr>
<td>Multiple-breath Nitrogen (N(_2)) Washout</td>
<td>FRC</td>
<td>Simple, relatively inexpensive; affected by distribution of ventilation in moderate or severe obstruction; multiple-breath; requires IC, ERV to calculate other lung volumes</td>
</tr>
<tr>
<td>Plethysmography</td>
<td>FRC</td>
<td>Plethysmographic method more complex but very fast; tends to be more accurate in the presence of airway obstruction than gas dilution techniques</td>
</tr>
<tr>
<td>Chest radiograph</td>
<td>TLC</td>
<td>Requires posterior-anterior and lateral chest x-ray films; must breath-hold at TLC; not accurate in the presence of diffuse, space-occupying diseases</td>
</tr>
<tr>
<td>HRCT chest</td>
<td>TLC</td>
<td>Involves radiation exposure and increased cost; must breath-hold at TLC; underestimates lung volumes in the presence of airway obstruction</td>
</tr>
<tr>
<td>MRI</td>
<td>TLC</td>
<td>No radiation exposure; costly; research tool only</td>
</tr>
</tbody>
</table>

Table 1: Methods to measure Lung Volumes

Indications for Lung volume determination:

1) To differentiate obstructive from restrictive disorders
2) To confirm diagnosis of a restrictive disorder
3) To detect combined obstructive and restrictive disorders
4) Assess response to therapeutic interventions
   a. Bronchodilators, steroids
   b. Lung transplantation, lung resection, lung volume reduction
   c. Radiation or chemotherapy
5) Make preoperative assessments of patients with compromised lung function
6) Determine the extent of hyperinflation
7) Assess air trapping by comparison of plethysmographic lung volumes with gas dilution lung volumes
Lung volumes are a pre-requisite in lung volume reduction surgery and surgery for bullous emphysema as the preoperative RV/TLC ratio is directly related to improvement in symptoms of breathlessness.

**Clinical Implications and significance of lung volumes and capacities:**

FRC, unlike TLC and RV, is an effort-independent maneuver that is determined by the balance of lung and chest wall recoil at relaxed end-expiration. FRC is reduced in restrictive disorders. Increased FRC is considered pathologic. FRC values greater than 120% of predicted values represent air trapping.

TLC may be increased in patients with obstructive defects such as emphysema and decreased in patients with restrictive abnormalities including interstitial lung diseases, chest wall abnormalities and kyphoscoliosis.

An increased RV indicates that, despite maximal expiratory effort, the lungs contain a larger volume of gas than normal. Increased RV often results in an equivalent decrease in VC. Elevated RV may occur during an acute asthmatic episode but is usually reversible. Increased RV is characteristic of emphysema and bronchial obstruction; both may cause chronic air trapping. RV and FRC usually increase together. Measuring lung volumes is critical to understanding changes in FVC. Decreased FVC can be due to restrictive lung diseases, chest wall defects, neuromuscular weakness, suboptimal effort, or severe obstruction, and therefore knowledge of TLC, FRC, and RV is invaluable in sorting out these possibilities. FRC, RV, and TLC are typically decreased in restrictive diseases. Table 2 lists comparative lung volumes for a healthy adult male and for patients with air trapping (as in emphysema), hyperinflation, restriction (as in interstitial lung disease) and neuromuscular weakness (as in amyotrophic lateral sclerosis).

<table>
<thead>
<tr>
<th>Value</th>
<th>Normal</th>
<th>Air Trapping</th>
<th>Hyperinflation</th>
<th>Restriction</th>
<th>Neuromuscular Weakness</th>
</tr>
</thead>
<tbody>
<tr>
<td>VC (L)</td>
<td>4.80</td>
<td>3.00</td>
<td>4.80</td>
<td>3.00</td>
<td>3.50</td>
</tr>
<tr>
<td>FRC (L)</td>
<td>2.40</td>
<td>3.60</td>
<td>3.60</td>
<td>1.50</td>
<td>2.40</td>
</tr>
<tr>
<td>RV (L)</td>
<td>1.20</td>
<td>3.00</td>
<td>3.00</td>
<td>0.75</td>
<td>1.50</td>
</tr>
<tr>
<td>TLC (L)</td>
<td>6.00</td>
<td>6.00</td>
<td>7.80</td>
<td>3.75</td>
<td>5.00</td>
</tr>
<tr>
<td>RV/TLC (%)</td>
<td>20</td>
<td>50</td>
<td>38</td>
<td>20</td>
<td>30</td>
</tr>
</tbody>
</table>

**Table 2: Comparative Lung Volumes for a Healthy Adult Male and Patients with Air Trapping, Hyperinflation, Restriction, and Neuromuscular Weakness**

**Significance of RV/TLC ratio:**

Restrictive processes due to interstitial lung diseases usually cause lung volumes to be reduced equally. Proportional relationships between lung volume compartments, such as the RV/TLC ratio, may be relatively normal in restrictive diseases. In various forms of extra-pulmonary restriction, such as obesity, kyphoscoliosis, or pleural effusion, RV is often less reduced than TLC, resulting in a relatively elevated RV/TLC ratio despite a low TLC. In obstruction, RV and FRC is usually increased (>120% of predicted) and two different patterns may be observed.

1) This increase in RV and FRC may be at the expense of a reduction in VC (see figure 2) with TLC remaining close to normal in which case the elevated RV/TLC ratio reflects air trapping.
2) In other cases, RV may increase while VC is preserved, so TLC is greater than predicted (>120% of predicted). The term hyperinflation is used to describe this absolute increase in TLC.

TLC may be either normal or increased in obstructive processes such as asthma, chronic bronchitis, bronchiectasis, and emphysema.

The RV/TLC ratio describes the percentage of total lung volume that cannot be emptied during expiration. In healthy adults, the RV/TLC ratio may vary from 20% in young adults to 35% in older patients. Values greater than 35% may result from absolute increases of RV (as in emphysema) or from a decrease in TLC because of a loss of VC. A large RV/TLC in the presence of increased TLC is often indicative of hyperinflation. An increased RV/TLC with a normal TLC indicates that air trapping is present. As an indicator of air trapping, the RV/TLC ratio is a weak but statistically significant indicator of outcome after lung volume reduction surgery (LVRS)

Processes that occupy space in the lungs such as edema, atelectasis, neoplasms, or fibrotic lesions may decrease TLC. Other diseases that commonly result in decreased TLC include pulmonary congestion, pleural effusions, pneumothorax, or thoracic deformities. Pure restrictive defects show proportional decreases in most lung compartments as described for FRC and RV.
When the TLC value is less than 80% of predicted or less than the 95% confidence limit, a restrictive process is present. Reduced VC, along with a normal or increased FEV₁/FVC ratio, is suggestive of restriction, but a measurement of TLC is needed to confirm the diagnosis of a restrictive defect.

Figure 2 depicts a comparison of changes in lung volume compartments and VC (superimposed) in normal, restrictive, obstructive, and neuromuscular weakness patterns. It shows the following:

- **In restrictive patterns**, FRC, RV, and VC are all decreased proportionately, resulting in a decrease in the TLC, which defines restriction. RV/TLC ratio is normal.
- **In obstruction (with air trapping)**, FRC and RV are both increased at the expense of the VC, and therefore TLC remains relatively unchanged. RV/TLC ratio is increased.
- **In obstruction (with hyperinflation)**, FRC and RV are both increased without reduction of VC in which case the TLC increases. RV/TLC ratio is increased.
- **In neuromuscular weakness**, the FRC is normal, but RV and TLC are reduced. RV is often less reduced than TLC, resulting in a relatively elevated RV/TLC ratio despite a low TLC.

A less common pattern is **mixed obstructive-restrictive lung disease**, characterized by a low FEV₁/FVC ratio and a low TLC. This is suggested by simple spirometry when both FEV₁/FVC and FVC are reduced but can only be confirmed by measuring TLC. Measuring the reduction in TLC may allow for more accurate characterization of the concomitant degree of airway obstruction.

**Conclusion:**

Lung volumes and lung capacities form the foundations of understanding the pathophysiology of lung function in health and disease. Specialized equipment is required for its measurement. Their significance is paramount in identifying the functional defect and give clues to identification of the type of disease and the severity of involvement.

- Measurement of Lung volumes help to distinguish between obstructive, restrictive and mixed obstructive and restrictive disorders
- They help to assess volume of closed bulla or cyst
- Lung volumes are mandatory requirement in preoperative evaluation of LVRS
- Besides this their role is also there in assessing response to therapies, prognostication of disease as well as for research.

**References**:

Introduction:

Upper airway (UA) is anatomically the section of the airways that begins from the nose and extends till the carina. Upper airway obstruction (UAO) is a distinct condition that can arise from a group of heterogeneous causes. Clinically it can present with acute or chronic symptoms depending on the etiology and the dynamics of the luminal obstruction. Acute UAO is a medical emergency and the patient may present with stridor, gasping and cyanosis and hypoxia. It is a clinical diagnosis and warrants immediate airway management. A subacute or chronic presentation is usually in form of progressive exertional dyspnea, hoarseness of voice, stridor and cough. These conditions may be frequently misdiagnosed as other common differentials like chronic obstructive pulmonary disease (COPD) or bronchial asthma (BA). In patients with acute UAO, a correct diagnosis is critical, as the definitive therapy often requires surgery. Once diagnosed and treated appropriately the results are gratifying with complete resolution of symptoms. UAO can further be classified as variable if the severity of obstruction varies according to phase of respiration or fixed where the severity of obstruction is unaltered irrespective of the phase of respiration. UAO can arise from either intra or extra thoracic causes. The common intra and extra thoracic causes of UAO are enlisted in Table 1. The mechanisms and pathodynamics of UAO in both these categories are distinctly different. Definitive diagnosis of UAO requires the inspection of UA by maneuvers laryngoscopy or bronchoscopy.

### Table 1: Extrathoracic and Intrathoracic causes of Upper airway Obstruction (UAO)

<table>
<thead>
<tr>
<th>CAUSES OF EXTRATHORACIC UAO</th>
<th>CAUSES OF INTRATHORACIC UAO</th>
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<tbody>
<tr>
<td>Goiter</td>
<td>Tracheal stenosis due to intubation*</td>
</tr>
<tr>
<td>Vocal cord dysfunction syndrome</td>
<td>Foreign body aspiration</td>
</tr>
<tr>
<td>Hypertrophied tonsils</td>
<td>Benign tracheal/bronchial tumors</td>
</tr>
<tr>
<td>Laryngostenosis*</td>
<td>Malignancies</td>
</tr>
<tr>
<td>Postextubation granuloma</td>
<td>Intrathoracic goiter</td>
</tr>
<tr>
<td>Retropharyngeal abscess</td>
<td>Tracheobronchomegaly</td>
</tr>
<tr>
<td>Neoplasms</td>
<td>Acquired tracheomalacia</td>
</tr>
<tr>
<td>Relapsing polychondritis</td>
<td>Right-sided aortic arch</td>
</tr>
<tr>
<td>Bilateral vocal cord paralysis</td>
<td></td>
</tr>
<tr>
<td>Cricoarytenoid arthritis</td>
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</table>

* Causes of fixed upper airway obstruction

Another simple test which gives us a lucid idea about the presence of UAO is spirometry with its flow-volume loop (FVL). Spirometry is the most commonly employed pulmonary function test (PFT) which basically measures the volume and speed of air exhaled and inhaled. This gives an insight into the type and the intensity of the underlying airway disease. It is non invasive, relatively inexpensive test requiring a standard spirometer and can be performed on an outpatient basis. By these virtues it becomes an ideal screening test in cases of suspected UAO. Recording a FVL is a vital and integral component of spirometry. The countenances of the FVL not only help us in determining the presence of UAO but also help us in gauging the site (intrathoracic or extrathoracic) and the nature (fixed or variable) of the obstruction.

### Dynamics of Abnormal Flow Volume Loop in Upper Airway Obstruction:

The underlying mechanisms of obstruction, the pressure variations and the resultant morphology of FVL generated depend on the site of UAO. In variable extrathoracic UAO (VE-UAO), the pressure in the trachea ($P_{tr}$) dips on forced inspiration resulting in a greater transmural pressure at the site of the lesion. This leads to worsening of the obstruction causing flattening of the inspiratory portion of the FVL. On
Expiration the intratracheal pressure increases. This reverses the transmural pressure tending to lessen the degree of obstruction and improve flow (Figure 1). In variable intrathoracic UAO (VI-UAO), with expiration the pleural pressure ($P_{pl}$) exceeds the rise in intratracheal pressure. This results in a reduction in the size of the airway at the site of the lesion causing an expiratory curve flattening$.^{2,3}$ During forced inspiration the pleural pressure becomes markedly negative. This decreases the obstruction by reversing the transmural pressure resulting in improved flow (Figure 2). In a fixed obstruction, irrespective of the site (intrathoracic or extrathoracic) flow is restricted equally in inspiration and expiration. (Figure 3) This flattens both the inspiratory and expiratory curves to form a "boxed" loop. The morphology of FVLs in all these conditions is shown in Figure 4. To avoid any lacunae in diagnosis, reliance on any single ratio or measurement is should be avoided. Hence not only the visual appearance of the FVL but the upper airway indices should be taken into consideration$^5$. The symptoms in UAO appear when the obstruction is relatively severe. However a FVL may show aberrations much before these symptoms manifest. Hence if a high index of suspicion is maintained, FVLs help in the early detection of UAO before symptoms manifest.

**Figure 1**
![Figure 1](image1)

**Figure 2**
![Figure 2](image2)

**Figure 3**
![Figure 3](image3)

**Figure 4**
![Figure 4](image4)

**Figure 1**: Schematic diagram to explain mechanism for Variable extra thoracic upper airway obstruction (VET UAO)
**Figure 2**: Schematic diagram to explain mechanism for Variable intra thoracic upper airway obstruction (VIT UAO)
**Figure 3**: Schematic diagram to explain mechanism for Fixed upper airway obstruction
**Figure 4**: Flow volume loops depicting VET UAO, VIT UAO and Fixed upper airway obstruction

**Recording a Flow volume Loop**: Flow-volume Loops (FVL) were first elucidated by Miller and Hyatt$^5$. In this maneuver, the subject inhales to his total lung capacity (TLC) and then exhales forcefully to residual volume (RV) followed by rapid inhalation to TLC again. A sensitive pressure transducer produces an electric signal, which is computed by a microprocessor, displayed on the screen and recorded. The flow is plotted on the Y-axis and the volume on the X-axis to construct a FVL. A normal FVL shows diminished flow as the lung volume shrinks, seen as slight coving near the RV on the expiratory curve. This occurs due to compressive forces during exhalation and distension by negative pressures during inhalation. The criteria recommended by the American Thoracic Society are used to select the best test of a minimum of three respiratory maneuvers$^6$.

**Visual examination of Flow Volume Loop and criteria for Diagnosing Upper Airway Obstruction**: For UAO, visual inspection of the FVL with respect to flattening of the inspiratory/expiratory portions or both gives a clue to presence, site and nature of UAO. In addition two ratios are calculated
1) Empey’s index$^7$ which is ratio of forced expiratory volume in 1 second (FEV$_1$) and peak expiratory flow (PEF)
2) Mid vital capacity ratio, i.e. the ratio of maximal expiratory flow at 50% of vital capacity (FEF50) and maximal inspiratory flow at 50% of the vital capacity (FIF50).

An Empey's index greater than 8 suggests presence of UAO. Further FEF50/FIF50 greater than 1 indicates variable extrathoracic VE-UAO, whereas FEF50/FIF50 less than 0.3 indicates variable intrathoracic VI-UAO.

**Special FVL caveats:**

Small variations done in the techniques of performing a FVL yield excellent results and help the clinician in clinching the diagnosis. FVLs change their morphology depending on the patient positioning as it affects the dynamics of the UAO. FVLs should be plotted in those positions in which symptoms are maximal to detect postural UAO. In some cases of goiter when sitting FVL is normal, supine FVL helps in detecting UAO. This also helps in determining the subsets of the patients who will benefit the most from surgery. FVLs are particularly important in obstructive sleep apnoea (OSA), as it is a simple and inexpensive objective method to predict the presence and the severity of UAO. The saw-tooth sign on FVL in OSA was first described by Sanders et al. in patients with OSA. It is hallmark by presence of three or more consecutive peaks and troughs occurring at regular intervals (Figure 5). The underlying mechanism has been attributed to turbulent flow due to sporadic narrowing of the upper airway due to the tissue redundancy. In addition to OSA this could also be seen in intrathoracic central airway stenosis, tracheomalacia and laryngeal dyskinesia.

Patients with OSA needs to triaged as per the clinical severity and the pretest probability scores so that those that need a polysomnography (PSG) on priority get an opportunity to be tested at the earliest. This clinical determination of the pretest probability is further supported by an evidence of an UAO on a FVL. Posture related worsening of UAO also occurs in subjects with obstructive sleep apnoea OSA), as nasopharyngeal resistance increases in the supine position. Hence in OSA when conventional oral and sitting FVLs are normal, supine position and nasal FVLs are indicated. Distinct patterns of the FVL like the biphasic spirogram also called as two can effect gives a clue to the possibility of main stem bronchial narrowing as seen in malignancy with an endobronchial mass causing complete obstruction of a mainstem bronchus. This can be confirmed subsequently by other techniques like FOB. In unilateral mainstem bronchial narrowing the emptying and filling of the lungs occurs as two distinct compartments the normal lung fast and the one with the narrowed bronchus much slower resulting in this biphasic pattern of the FVL. Spirometry in patients with tracheal stomas or tracheostomy tubes is difficult due to failure to achieve a good seal between the tube and the mouthpiece of the spirometer. This can be overcome using adapters. Thus FVL is an inexpensive screening test and can be performed in specific positions and via alternative routes to evaluate UAO.

![Flow-volume loops depicting “Saw tooth” appearance](image)

**Case Report:**

A 60-year-old male, non-smoker, was referred to us for assessment of breathlessness. He complained of progressive exertional dyspnea since 2 to 3 years which worsened in the recumbent position. Now, he also complained of difficulty in his day-to-day activities since 2 months. He also gave a history of wasting of hand muscles since 6 months. Patient was hypertensive since 7 years. On neurological examination, the patient had a stooped posture with masseter muscle wasting, thenar and hypothenar muscle wasting, miniasporemyoclonus of both hands and brisk reflexes. The respiratory system examination was within normal limits. The baseline saturation of the patient measured with a pulse oximeter (SpO2) was normal; however, there was a desaturation to 80% with paradoxical thoraco-abdominal movements on lying supine. Chest radiograph was suggestive of bilateral elevated diaphragm. High-resolution computed tomography of chest was normal. With a suspicion of a neurological disease, the patient was evaluated with an electromyography and nerve conduction velocity study which showed acute and chronic denervation of C5 to C8 and T1 nerves, with site of lesion being anterior horn cells/nerve roots. Spirometry was suggestive of forced expiratory volume in the first second (FEV1) of 1.25 L (44%), forced vital capacity (FVC) of 1.85 L (53%) with FEV1/FVC ratio of 68% in sitting position. While in supine position, they were reduced to FEV1 of 0.42 L (15%) and FVC of 0.89 L (25%). The flow volume loops (FVL) revealed upper airway UAO.
The Empey’s index was 8. The FV loop characteristically demonstrated a biphasic (“two-can” effect) expiratory loop along with fall in lung function in supine position (Figure 6). To rule out SDB, PSG was done which showed apnea–hypopnea index (AHI) of 29 with predominant CSA. Titration with bi-level positive airway pressure (PAP) showed reduction in CSA AHI. The patient was diagnosed as a case of SDB, CSA with UAO due to MND. Spirometry with FVL plays an important role in conditions like neuromuscular diseases particularly motor neuron disease. (MND). It is not only an important tool in assessing respiratory functions but it also can demonstrate peculiar characteristics of in the FV loop which may help in clinching the diagnosis. The “two-can” effect is defined as the biphasic nature of expiratory FVL. This phenomenon occurs due to sudden laryngeal muscle contractions in MND. Patients with MNDs also have associated sleep related breathing disorders like OSA or CSA. This leads to flaccidity of the UAs and leads to peculiar FVL changes. Our patient also demonstrated the characteristic two can effect on his FBL which occurred due to his MND with CSA as diagnosed on his holistic workup.

![Figure 6: Schematic diagram to explain mechanism for Biphasic loop (Two-can effect) in case of central sleep apnea.](image)

**Conclusion:**
UAO continues to be an under diagnosed and underreported entity. Spirometry with FVL is an excellent screening tool for UAO by virtue of its simplicity, ease of availability and non-invasiveness. A timely index of suspicion and a low threshold for performance of a spirometry with FVL in the appropriate clinical context can aid in the early diagnosis of UAO and hence shift the patient prognosis favorably.

**References:**

Introduction:

Spirometry is the basic lung function test which is most commonly done in pulmonary practice. Although spirometry is not a test to arrive at a clinical diagnosis, it is helpful for the clinician in several different ways as follows:

1. To categorize the disease into obstructive, restrictive or mixed pattern
2. Determination of best lung function of the patient and disease severity
3. Subclinical disease detection in a risk-category
4. Evaluation of treatment follow-up
5. Prediction of outcomes
6. Intensive monitoring of treatment and disease progression

Spirometry is also an important tool for screening in epidemiological research.

Other pulmonary function tests (PFT) needed for differential diagnosis are done as and when necessary depending upon a particular clinical condition. They include dynamic Studies such as Flow-Volume Loops and static lung volumes such as the FRC measurements. Measurement of gas transfer is done with the assessment of Diffusing Capacity and partial pressures of arterial blood gases.

Spirometry provides important information on the following different lung parameters important to interpret for both diagnosis and treatment purposes:

1. Lung volumes and capacities such as tidal volume (TV), vital capacity (VC) and their derivatives
2. Dynamic lung functions- expiratory flows such as FEV\textsubscript{1}, FEF\textsubscript{200-1200} and FEF\textsubscript{25-75%}
3. Flow – Volume loops

Bronchodilator reversibility (BDR) test consists of spirometry performed before and after bronchodilator inhalation following a specifically designed protocol. BDR is done in cases of obstructive pattern particularly to differentiate between asthma and chronic obstructive pulmonary disease (COPD).

Pitfalls of Spirometry:

Spirometry is fraught with many pitfalls and inaccuracies. Errors may arise during the testing procedure as well as with reference to the acceptability of the graph and interpretation of the test. Some of these important pitfalls are discussed as under.

1. Related to the testing procedure: Spirometry requires active involvement of the patient and the technician throughout the procedure. A standardized procedure is therefore essential for meaningful interpretation.
The standardized test requires that the patient should be comfortable while sitting or standing. Nose clips and tight sealing of lips over mouthpiece are recommended to prevent any air-leak during the procedure. The test begins with normal tidal volume breaths. After a few breaths when the TV stabilizes, perform a maximal inspiration at end-expiration to total lung capacity followed by exhalation as hard, as fast, and as completely as possible.

2. **Related to the Test Quality**: A good quality test should reflect the procedure with essential acceptable criteria such as the following:
   i. Stable tidal volume without any leak
   ii. Complete inhalation during to the tidal volume before exhalation
   iii. Satisfactory and uninterrupted exhalation
   iv. Satisfactory duration of the procedure
   v. Without the presence of artifacts
   vi. The reproducibility criteria should also be satisfied. It is normally desirable that 3-8 manoeuvres should be done for this purpose. It is normally aimed to obtain two largest values within 0.2 litres for both vital capacity and forced expiratory volume in 1st second (FEV₁). Interpret 3 best tests if the earlier stated criteria are not met even after 8 trials.

3. **Related to the Graph**: The graph obtained with spirometry depicts volumes and flows at ambient temperature and pressure (ATP). For the purpose of standardization, these are converted to body temperature, pressure, water vapor saturated (BTPS) conditions.

4. **Interpretation**: For interpretation of spirometry, one needs to know the normal values. Unlike many other physiological parameters, there are no fixed spirometric values. They vary depending upon gender, age, height and body-mass index (BMI). For proper interpretation, one needs to know the values predicted for an individual. The test-determined value is thereafter changed as percent of the predicted values. Predicted values are obtained from different regression equations or nomograms relevant for the population being tested. Sometimes, the age-specific means are used for prediction if the regression equations are not available. The other options to express abnormality, the values are expressed as either ‘lower limits of normal (LLN), fixed percent, lower fifth percentile or lower 95th confidence interval (C.I).

   - It is futile and often confusing to use more spiographic variables than the necessary since computerized analysis of the graph may provide a large number of values. One generally needs only VC, FEV₁ and FEV₁/VC% from a good quality satisfactory graph. Mid-flow rates may sometimes help.
   - Interpret values well above or well below lower limits of normal with confidence. Borderline values should be interpreted with great caution. Such values should be interpreted along with the clinical information. Other reports which may result in fallacious interpretations include false positive & false negative results. Mixed defects also need a careful interpretation in the light of clinical details. One may erroneously categorize a defect as restrictive or obstructive.

**Common errors during testing**:

1. Unsatisfactory start– a slow start or hesitation when starting exhalation: Spirometry is patient-dependent test involving cooperation and effort on his part. Even a slight hesitation or a delayed start produces erroneous results. Both hesitation in exhalation and slow manoeuvre will cause a delay in the peak flow and falsely increase the FEV1.
2. Coughing during the test
3. Hesitation or swallowing during exhalation
4. Obstruction due to patient’s tongue or leak at the mouthpiece

A good spirogram should comprise of a full inspiration, rapid achievement of highest flow followed by smooth and continuous decrease in expiratory flow gradually terminating to the resting value. Expiration should ideally last for three seconds or more.
In spite of a good graph, spirometry interpretation also requires other essential criteria as below:

1. **Demographic information** such as the gender, age, height, weight and ethnicity. Normal values used for prediction depend upon an individual’s age, height and gender. Incorrectly entered values may not be even noticeable to the person reporting the graph.
2. **Instrument calibration**: This should be ensured on regular basis to avoid errors.
3. **Selection of the graph** as per standard recommendations without the presence of artefacts due to a hesitant start, slow expiration, glottis closure, breath holding or overlap of breaths. Poor selection frequently leads to false-negative or false-positive results.

**Reference equations** : It is generally recommended including the ATS/ERS guidelines that each pulmonary function laboratory should have its own reference equations based on the local population. This may not be possible in all scenarios when such nomograms are not available. It is important to mention that reference equations themselves are meaningful only when they have been derived from an adequate sample size which included different ethnicities, ages and heights using standard statistical analyses.

In summary, spirometry is a test which is highly dependent upon cooperation and effort of the patient as well as the expertise and endurance of the technician. It is fraught with several pitfalls responsible for relatively high rates of false positive and false negative results. It looks like a simple test but with potential to provide several erroneous results. Interpretation of spirometry requires expertise and background clinical information. Computerized interpretation of disease state based on spirometric values must always be avoided.

**Points of interest**:

1. **Although not a diagnostic test**, spirometry is highly useful for disease- categorization and severity, determination of best lung function, evaluation of treatment follow-up and prediction of outcomes.
2. It requires cooperation and efforts on the part of both the patient and the technician.
3. It is important to select a good spirometric graph for a meaningful interpretation.
4. Patient demographic information is crucial for spirometry interpretation.
5. Spirometry interpretation should always be done in the light of clinical available data rather than a computerized categorization as an obstructive, restrictive or mixed disease.

**References**:

Case 1.

A 35 year old woman presents with history of dyspnoea on exertion and orthopnea since 2-3 years. She is a never smoker and there is no history of asthma or any other chronic pulmonary or cardiac diseases. She underwent a surgery for mitral valve repair a year back. Her spirometry is as follows :

<table>
<thead>
<tr>
<th></th>
<th>Pre - bronchodilator</th>
<th>Post - bronchodilator</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Actual</td>
<td>Predicted</td>
</tr>
<tr>
<td>FVC (l)</td>
<td>1.13</td>
<td>2.65</td>
</tr>
<tr>
<td>FEV₁ (l)</td>
<td>0.75</td>
<td>2.15</td>
</tr>
<tr>
<td>FEV₁/FVC</td>
<td>66.3%</td>
<td>81.1%</td>
</tr>
</tbody>
</table>

* FVC: forced vital capacity; FEV₁: forced expiratory volume in 1 second

Her spirometry was repeated in supine position and was as follows :

<table>
<thead>
<tr>
<th></th>
<th>Supine</th>
<th>% Change from upright position</th>
</tr>
</thead>
<tbody>
<tr>
<td>FVC</td>
<td>0.85</td>
<td>- 24.7%</td>
</tr>
<tr>
<td>FEV₁</td>
<td>0.66</td>
<td>- 12%</td>
</tr>
<tr>
<td>FEV₁/FVC</td>
<td>77.6%</td>
<td></td>
</tr>
</tbody>
</table>

Interpretation:

The patient has reduced FEV₁ and FVC with pre and post bronchodilator FEV₁/FVC ratio less than 70%. Bronchodilator reversibility is not significant (less than 200 ml and 12% change in FEV₁ ). There is a mixed pattern with either air trapping or true restrictive process which
can be confirmed by measurement of static lung volumes. In view of her surgical history patient was also asked for a repeat test in supine position. There is more than 10% decrease in FVC which suggests respiratory muscle weakness (diaphragmatic paralysis could be explained due to phrenic nerve injury during the operation). Unilateral diaphragmatic paralysis is usually associated with a decrease in VC of 15 to 25 percent; bilateral diaphragmatic paralysis can be associated with a decrease in supine VC approaching 50 percent. A decrease of more than 10% in supine from sitting/upright position is considered significant for diagnosis of respiratory muscle weakness (1).

Case 2.

A 63 year old man, current smoker, presented with history of left sided chest pain and exertional breathlessness since 5 months. On examination of the chest a monophonic wheeze was heard in the left upper areas of chest. Spirometry was done and below are the forced expiratory spirogram and flow volume loop of the patient

![Volume Graph](image)

**Interpretation:**

The flow volume loop shows a **biphasic pattern** that is characteristically seen in mainstem obstruction (left side in the present case)(2). In this pattern the initial half of curve is normal due to rapid emptying of unaffected side while the second half has a straight line appearance due to constant expiratory flow because of the fixed airway resistance on the diseased side. Similarly, during forced inspiration the curve shows a pronounced slowing of maximum inspiratory flow towards the end of inspiration. The inspiratory curve flattening is more specific than that of the expiratory part for recognising mainstem bronchial narrowing, as the end inspiratory tail (straightening) is not a feature of generalised airways obstruction. The volume time spirogram seen above illustrates the contribution of slowly emptying lung compartments due to obstruction.

Case 3.

A 54 year old man, ex-smoker (history of smoking 30 pack year), presented with chronic cough and exertional dyspnoea. He was diagnosed as a case of COPD and was given inhaled bronchodilators with only partial relief. There was history of wheezing and breathlessness which increased on lying down. Patient underwent spirometry that revealed the following result:

<table>
<thead>
<tr>
<th></th>
<th>Pre-bronchodilator</th>
<th>Post - bronchodilator</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Actual</td>
<td>Predicted</td>
</tr>
<tr>
<td><strong>FVC (l)</strong></td>
<td>2.72</td>
<td>3.56</td>
</tr>
<tr>
<td><strong>FEV₁</strong></td>
<td>1.90</td>
<td>2.95</td>
</tr>
<tr>
<td><strong>FEV₁ /FVC</strong></td>
<td>69.8%</td>
<td>82.8%</td>
</tr>
</tbody>
</table>
**Flow-Volume Loop**

**Interpretation:**

Both FVC and FEV$_1$ are reduced and reduced pre bronchodilator ratio reveals obstructive pattern with no significant bronchodilator reversibility. Although post bronchodilator ratio is more than 70% hence a diagnosis of COPD is not suggestive with these values. Also seen in flow volume loop are oscillation in the late expiratory part of the curve. There is sharp peak in expiratory curve with a rapid decline with tailing of end part suggestive of obstructive pattern. These oscillations can be caused by tracheobronchomalacia, redundant pharyngeal tissue, neuromuscular disease, or structural or functional disorders of the larynx.(3), (4). Bronchoscopy was done and it revealed dynamic airway collapse with more than 50% collapse during expiration suggestive of tracheobronchomalacia. The sharp peak with decline is also associated with collapse of airways due to negative transmural pressure in this condition.

**Case 4.**

A 16 year old girl presented with complaints of breathlessness and cough with expectoration for 5 years which are usually seen with change of season and during episodes there is diurnal variation with more symptoms at night. Family history (mother) of allergic rhinitis is present. On examination (done during the episode) rhonchi are heard bilaterally. Chest X ray is normal. Her spirometry result is shown below.

<table>
<thead>
<tr>
<th></th>
<th>Pre bronchodilator</th>
<th></th>
<th>Post bronchodilator</th>
<th></th>
<th>Change</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Actual</td>
<td>Predicted</td>
<td>% Predicted</td>
<td>Predicted</td>
<td></td>
</tr>
<tr>
<td>FVC (l)</td>
<td>2.35</td>
<td>3.40</td>
<td>69.1%</td>
<td>2.40</td>
<td>70.5%</td>
</tr>
<tr>
<td>FEV$_1$ (l)</td>
<td>2.18</td>
<td>2.80</td>
<td>77.8%</td>
<td>2.21</td>
<td>78.9%</td>
</tr>
<tr>
<td>FEV$_1$/FVC</td>
<td></td>
<td>82.4%</td>
<td>92.1%</td>
<td></td>
<td></td>
</tr>
<tr>
<td>FET</td>
<td>6.2 sec</td>
<td></td>
<td>5.8 sec</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

*FET : forced expiratory time*
Flow volume loop

**Interpretation:**

The spirometry values are suggestive of restrictive pattern. The flow volume curve is smaller but parallel to the predicted curve and ‘Knee’ pattern (convex inflection) can be appreciated on the expiratory limb. The knee pattern is a normal variant usually seen below 35 years of age due to a proximal flow limiting choke point in the airways that moves distally with age due to loss of elastic recoil of parenchyma. The flow volume curve is more towards normal and comparable to the predicted curve though the values suggest restrictive disease.

Static lung volumes were done for the patient and TLC (total lung capacity) was normal (88% of predicted) and RV/TLC was slightly increased (124% predicted). This is pseudo restriction and is known as ‘non-specific ventilatory abnormality’. This is usually seen in asthma (mild or quiescent asthma), obese individuals or children and young females. In the current case asthma explains the above findings which can be confirmed by doing bronchoprovocation test.

**Case 5**

A 22 year old boy complained of intermittent wheezing, chest tightness and breathlessness. The symptoms were exaggerated few minutes after exercise and used to subside in an hour. There was no history of smoking or environmental exposures. Family history was insignificant. Chest X ray revealed no abnormality. Spirometry was done and values and curve are given below:

<table>
<thead>
<tr>
<th></th>
<th>Pre - bronchodilator</th>
<th>Post - bronchodilator</th>
<th>Change</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Actual</td>
<td>Predicted</td>
<td>%</td>
</tr>
<tr>
<td>FVC (l)</td>
<td>3.25</td>
<td>3.60</td>
<td>90.2%</td>
</tr>
<tr>
<td>FEV₁ (l)</td>
<td>2.55</td>
<td>3.10</td>
<td>82.3%</td>
</tr>
<tr>
<td>FEV₁ /FVC</td>
<td>78.4%</td>
<td>86.1%</td>
<td></td>
</tr>
<tr>
<td>FEV₆</td>
<td>3.20</td>
<td></td>
<td></td>
</tr>
<tr>
<td>FEV₁ / FEV₆</td>
<td>79.6%</td>
<td></td>
<td></td>
</tr>
<tr>
<td>FEF₂₅-⁷₅ (l/sec)</td>
<td>2.20</td>
<td>3.90</td>
<td>56.4%</td>
</tr>
</tbody>
</table>
Interpretation:

FVC, FEV₁ and the ratio of two are all within normal limits for the patient. Although, maximal mid expiratory flow (FEF₂₅₋₇₅) are drastically reduced. Though reduction of this parameter is non-specific for small airway diseases but they are among the earliest signs of airflow obstruction in such diseases and hence can help in narrowing to the diagnosis and directing further investigations. Another parameter seen in the above spirometry is FEV₆ (forced expiratory volume at 6 seconds). It has been seen in various studies that FEV₁/FEV₆ <73% and FEV₆ <82% predicted can be used as a valid alternative for the FEV₁/FVC <70% and FVC <80% predicted cut-off points for the detection of obstruction and restriction, respectively(6,7). It is considered as an effective screening tool in the primary care centres for early detection of obstructive airway diseases especially COPD among high risk individuals (smokers, >40 years of age). It has the following advantages:

1. Easier to perform especially for older patients
2. More reproducible than FVC
3. Reduce risk of syncope as it involves shorter manoeuvre

In view of his history and MMEF values, differential diagnosis of airway disorder was kept and patient was further taken up for bronchial provocation test (post exercise), as below:

<table>
<thead>
<tr>
<th>Post challenge</th>
<th>5 minutes</th>
<th>10 minutes</th>
<th>20 minutes</th>
<th>30 minutes</th>
</tr>
</thead>
<tbody>
<tr>
<td>FEV₁ (l)</td>
<td>2.45</td>
<td>2.44</td>
<td>2.31</td>
<td>2.24</td>
</tr>
<tr>
<td>Change (%)</td>
<td>-3.9%</td>
<td>-4%</td>
<td>-9.4%</td>
<td>12.1%</td>
</tr>
</tbody>
</table>

Interpretation: Exercise induced bronchial challenge is a physical challenge indirect test that has low sensitivity and high specificity. A negative challenge does not rule out asthma but a positive test is highly specific for exercise induced asthma. The test is considered positive when there is fall of >10% in adults and >13% in paediatrics from baseline FEV₁. The above results shows a positive response to exercise challenge and suggest exercise induced asthma in the patient.

Case 6.

A 36 year old woman presented to emergency department complaining of difficulty to clear secretions and shortness of breath. 3 weeks prior to this even patient was hospitalised for pneumonia and underwent percutaneous tracheostomy. On examination tracheostomy scar was noticed which was healed and she was tachypnoeic. On auscultation of chest prolonged inspiratory phase was appreciated with harsh breath sounds. A bedside spirometry was done which revealed the following values:
### Flow Volume Loop

**Interpretation:**

FEV₁ is slightly reduced and FVC is within normal range. Looking at the flow volume loop one can clearly appreciate the flattening of both inspiratory and expiratory part which is indicative of fixed upper/central airway obstruction. Also upper airway obstruction diagnosis is supported by calculating Empey index which is the ratio of FEV₁ (ml)/PEFR (l/min). In a healthy individual this ratio is less than 10 while in a person with upper airways obstruction ratio is usually greater than 10 and the higher the index the more severe the obstruction[8]. This is due to a much more reduction of PEFR than FEV₁ in upper airway obstruction. The initial part of expiratory limb (flow at higher lung volumes) of flow volume loop is effort dependent and hence the flow is reduced to a greater extent due to increased resistance because of upper airway obstruction whereas at lower lung volumes flow is effort independent and is primarily determined by collapse of bronchioles.

Hence the ratio of PEFR and FEV₁ is above 10 in cases of upper airways obstruction. In the present case the ratio is 12.7 and hence is suggestive of upper airway obstruction and the graph points towards a fixed obstruction. Considering her history and spirometry, bronchoscopy was performed which revealed subglottic stenosis.

During inspiration the forced maneuver is effort dependent whereas the major part of expiration is effort independent (except the initial portion wherein peak flow is achieved). Due to this, the flow during the middle of inspiration measured at 50% of the FVC (FIF50%) is usually greater than the maximal expiratory flow at 50% of FVC (FEF50%). Ratio of FIF50%/FEF50% is, therefore, usually less than 1[9]. In variable extrathoracic lesions, the ratio is increased (usually greater than 1), while in variable intrathoracic lesions, the ratio is diminished (0.2 or less). In fixed obstructions (intrathoracic or extrathoracic), the ratio is expected to be close to 1.
Key Points:

Interpretation of spirometry is done in following steps:

1. Demographics (age, sex, height, race) for reference values and quality of test (such as technical comments for acceptability, reproducibility and end of test criteria) are first analysed.

2. Analysis of size and shape of flow volume loop:
   a. Scalloped curve / concave expiratory limb: obstructive disorders
   b. Small size with steep slope and low MMEF (maximal mid expiratory flow) with normal or reduced PEFR giving a witch’s hat appearance: parenchymal restrictive disorders
   c. Low PEFR, low MMEF with parallel slope to predicted curve: chest wall restrictive disorders
   d. Non sharp peak producing a convex curve: poor effort or neuro muscular disorders
   e. Flattening of any limb:
      i. Only expiration is flat (variable intra-thoracic obstruction)
      ii. Only inspiration is flat (variable extra-thoracic obstruction)
      iii. Both inspiratory and expiratory limbs are flat (fixed upper airway obstruction)

3. Analysis of spirometry values:
   
4. Grading of severity: ATS grading of severity of any spirometric abnormality based on FEV1. After determining the pattern to be obstructive, restrictive or mixed, FEV1 is used to grade severity:
   - Mild: FEV1 > 70 (% pred.)
   - Moderate: 60–69
   - Moderately severe: 50–59
   - Severe: 35–49
   - Very severe:<35

5. Correlation of the data with clinical history and other investigations: As spirometry is not specific for any disease and is a supportive test it is always to be interpreted with correlation to patients history and other investigations.
References:


Peak Expiratory Flow : Estimation and Clinical Applications

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Nishtha Singh²

Introduction:

Peak expiratory flow rate (PEFR) is the maximal flow obtained on maximal short exhalation after a complete inspiratory effort. It occurs in the first 200 milliseconds of maximal expiratory effort from the total lung capacity. PEFR correlates well with the value of forced expiratory volume in one second (FEV₁) in patients of asthma and thus is useful in monitoring of asthma control at places where spirometry is not available.¹²

Peak flow depends on expiratory muscle strength, voluntary effort, the last complete inspiratory flow and calibre of large airways. It varies with the effort of the patient. It is mainly an index of larger airways therefore may underestimate severity of asthma predominantly involving smaller airways.

PEFR is measured by both spirometers and peak flow meters. The peak flow meters are small mechanical devices although electronic devices are also present. There is no single calibration metre for all the devices and the specifications of the instruments should matched with the international guidelines.³

Method of performing PEFR:

These steps are to be followed while performing PEFR. These include:

1. The person should sit or stand straight
2. A full deep breath is taken in
3. The mouthpiece is put inside mouth between teeth
4. When the mouth seal is formed, a short breath with maximal effort is blowed out
5. The exhalation should be short for 2 seconds
6. The result on the peak flow meter should be recorded and this process is repeated 2 more times (total 3 times).
7. The highest of all the readings should be recorded.

The patient should be taught the technique of using the peak flow meter at the time of prescription. Thereafter, the technique should be checked at every follow up visit. In addition, patients should be taught to observe the variability in PEFR apart from recording of absolute values.

Normal values- The values of PEFR are similar to the values obtained of forced expiratory volume (FEV₁) and forced vital capacity (FVC) in spirometry. These values also depend on the age, sex and height of the patient. PEFR values are denoted in L/minute in peak flow meter, However, in spirometry, these values are denoted in L/sec. Conversion can be performed from L/sec to L/minute by multiplying with 60 sec/min. The PEFR values obtained from spirometer are lower than peak flow meter readings. This is due to the fact that spirometer requires a prolonged expiratory effort rather than a rapid expiratory blow with a peak flow meter. The predicted PEF values are obtained by taking the reference values from the peak flow meter and not from the spirometer.

Generally, the values of PEF are slightly lower in the morning than in the afternoon or evening.⁴ the highest values are generally seen between 2pm to 4pm. The mean diurnal variation in healthy school children was studied to be 6.2%.⁵ This variation is usually less than 20% in well controlled asthmatic patients.

Correlation with lung functions:

PEF values correlates well with severity of asthmatic symptoms assessed by asthma control test (ACT).⁶⁻⁸ It varies slightly in overweight individuals.⁹ It also correlate well with spirometric values like FEV₁.
Limitations:

The validity of PEF results depends a lot on the correct technique of the patient. Suboptimal effort can give false low values even in healthy subjects. Therefore, asking a patient to perform the peak flow technique in front of a doctor or trained health care personnel is important in assessing the correct technique.

Restrictive diseases affecting chest wall can cause inadequate expiratory effort leading to false low peak flow value. Therefore, in cases where the peak flow values are less than 80 percent, a check spirometry should be done.

Peak flow meter has high sensitivity in assessing severity of COPD but specificity is low.

In severe asthma, PEF can underestimate the severity of disease.

Personal Best PEF

When a patient uses peak flow values for self-management of asthma, a personal best peak flow reading should be recorded. It is usually recorded when the patient is completely well after taking maximal inhaler therapy. A value less than 80 percent of the personal best is considered abnormal taking into account the normal diurnal variation in the airflow. Revaluation of the personal best reading should be done every year to account for the lung growth in children and in patients with changing disease severity. The personal best value reaches a peak at the age of 18-20 years, remains at this level till 30 years in males and 40 years in females. Thereafter, the value declines.

Peak flow diaries

For recording the personal best PEF, the peak flow charting should be done two to four times a day for two weeks when the asthma is well controlled. This chart should be examined at the next visit in order to calculate the patients personal best PEF.

Self-Management of Asthma

The role of peak flow meter in the monitoring of asthma is still not determined. It provides an objective parameter for asthma control. Conclusive evidence of its role in improving asthma outcomes could not be determined by randomised control trials. But guidelines support the use of peak flow meter charting in supplementing other assessments of asthma control in moderate to severe asthma. The frequency of peak flow meter reading can be changed according to the needs of patients. A patient who is a poor perceiver of symptoms may benefit from diurnal record of peak flow charting. Symptom diaries may not be very useful in such patients. On the other end, in case of stable disease, the patient needs to measure the PEF once daily only. In cases when asthmatic symptoms are expected to increase such as during pollen season or viral infection, the PEFR recording frequency is increased.

Asthma action plan:

This is a written diary where medications are suggested according to severity of asthma. Patient is provided guidance to assess and identify decline in asthma control, and to change treatment plan according to the symptomatology and PEF. When the patients combine their symptomatology with PEF monitoring, they get both subjective and objective ways to correlate trigger exposure with severity of asthma. The patient is also guided in deciding the treatment and catching early warning signs of possible deterioration.

Green, Red and Yellow zones categorisation:

These are the zones denoting a colour scheme according to which the patients can self treat at home. These are as follows:

Green zone - This signifies that the PEF is between 80-100 percent of the personal best value and symptoms are not present. The patient in this zone should continue his medicines as earlier.

Yellow zone - The PEF values are between 50-80 percent of the personal best. This is a warning zone or a zone of caution. The patient should take his/her prescribed medicines along with additional medicines suggested by clinician in action plan.

Red zone - Here the PEF lies below 50 percent of the personal best. The patient should immediately contact his clinician and get therapy started.

Adherence:

Long term adherence to PEF monitoring is difficult, therefore attaching PEF monitoring with self-treatment activities leads to improved adherence.

Efficacy:
The effectiveness of PEF in changing the asthma outcomes is questionable. Studies could not find the advantage of using PEF in modifying asthma outcomes such as morbidity and quality of life. PEF monitoring is most advantageous in patients with severe asthma who have poor perception of symptoms.

**Other uses of PEFR:**

PEF may also be useful in the monitoring of occupational asthma, assessment of severity in acute asthmatic exacerbation and home monitoring of the disease. Monitoring of PEF at work and away from work gives a good index for initial evaluation of occupational asthma. For diagnosis of asthma, spirometry is the preferred method, but, in cases where spirometry is not available, peak flow meter charting is valuable.

Peak flow variability is also a valuable index. It is expressed in percentage of minimum PEF and calculated by the difference between the maximum peak flow value to the day’s minimum value. If the within day or between days peak flow variability is greater than 20 percent, it is characteristic of asthma.

The role of PEF has become more important in the present COVID-19 pandemic scenario where usage of spirometry has declined drastically and value of home monitoring of asthma is increased.

**References:**

10. Reddel HK, Marks GB, Jenkins CR. When can personal best peak flow be determined for asthma action plans? Thorax 2004; 59:922.
Measurement of Diffusion Lung Capacity

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Introduction:
The fundamental action of lungs is gaseous exchange that involve three components named ventilation, perfusion & diffusion. In lungs, oxygen moves from alveolar gas into capillary blood by diffusion & into neighbouring cells by the process of diffusion. Carbon dioxide also moves by diffusion but in direction opposite to that of oxygen.

Diffusion capacity is the volume of the given gas that diffuses across the respiratory membrane (alveolar-capillary membrane) per unit time($V_G$) in response to the difference in mean pressure of the gas within the alveolus ($P_{AG}$) and pulmonary capillary($P_{CG}$).

The diffusion capacity of the lung to any gas = $DL_G$

$$DL_G = \frac{V_G}{P_{AG} - P_{CG}}$$

In other words, the diffusion capacity is volume of gas that diffuses through respiratory membrane each minute for a pressure difference of 1 mm Hg and is expressed as ml/min/mmHg.

The gases that can be used to measure Diffusion capacity of lungs (DL) are Oxygen($O_2$), Carbon monoxide (CO) and nitric oxide (NO) due to their unique ability to combine with Haemoglobin (Hb). Measurement of $DLO_2$ is difficult because oxygen transfer may be limited by ventilation-perfusion mismatch and shunting apart from diffusion. Moreover, a changing capillary $PO_2$ during capillary transit cannot be accurately determined. Because of difficulty in estimating the mean capillary oxygen pressure ($PCO_2$) which is required for calculation of pressure gradient across the alveolar-capillary membrane, CO is widely used for measuring the DL. In case of CO, mean capillary CO pressure ($PCO$) can be assumed to zero due to extremely high affinity of Hb to CO (240 times than $O_2$) so its partial pressure in the blood remains almost zero in spite of significant HbCO concentration.

$$DL_CO = \frac{V_{CO}}{P_1 - P_2} = \frac{V_{CO}}{P_1} = \frac{V_{CO}}{P_{CO}}$$

There are two components of DL i.e. membrane component and the intravascular component. These can be expressed as the inverse of their effective diffusing capacity as shown in following equation by Roughton and Forster: -

$$\frac{1}{DL} = \frac{1}{D_M} + \frac{1}{\Theta \cdot V_C}$$

Where the DL is diffusion capacity; $D_M$ is membrane component of resistance to diffusion; $\Theta$ is rate of reaction of CO with Hb (ml CO/min/Pka/ml of blood) and $V_C$ is volume of blood in the pulmonary capillary bed. $1/ \Theta \cdot V_C$represent intravascular resistance to diffusion.

Diffusion capacity for NO($DL_NO$) is 4 to 5 times greater than $DL_CO$ in the same subject. Since binding of NO with haemoglobin is much more fast, the time required for binding of NO with intracellular haemoglobin (Intravascular component)is negligible and contribute little to the measured value of $DL_NO$. Therefore, $DL_NO$ is assumed to be equal to $D_MNO$. Again measurement of $DL_NO$ is not routine except research settings in view of high reactivity with oxygen, need for special equipment for analysis and potential cardiovascular effects etc.

There are three methods to measure Diffusion capacity:
Single-Breath Method: this was first described by Marie Krogh in 1914 and is almost exclusively utilized in clinical settings. The patient exhales to residual volume and then takes a maximal inhalation (up to vital capacity) of the test gas containing 0.3% CO and a diluent inert gas, 10% helium in air, holds the breath for around 10-seconds and then exhales maximally. The rate of disappearance of CO from the alveolar gas during the 10-sec breath hold is calculated. At the end of breath-holding period, a sample of alveolar gas is obtained after discarding the dead space. The exhaled sample is then analysed for CO using an infrared analyser. The inert tracer gas is used to measure the alveolar volume by dilution. Figure 1 shows the single breath method of DLCO measurement.

\[
\text{DL}_{\text{CO}}\text{sb} = K \times \text{VA} \times \ln \left( \frac{\text{PB} - 47}{\text{t}} \right)
\]

Where \( \text{VA} \) is the alveolar volume in liters, ‘t’ is breath holding time in sec, and ‘K’ is constant. The fractional concentrations of CO and helium in inspired and sample gas (\( F_A\text{CO} \) and \( F_A\text{He} \), respectively) are indicated by appropriate terms. The patients result are interpreted by comparing with the predicted values of the lower limit of normal person and severe respiratory impairment is defined as DL below 45% of the predicted values. This method is easy, safe, non-invasive, rapid and widely used test to measure the diffusion capacity.

In many patients with pulmonary diseases, the single breath diffusion capacity is reduced. This decrease is usually caused by uneven ventilation-perfusion distribution and diffusion-perfusion properties in diseased lungs rather than actual change in diffusion across the respiratory membrane. Such diseased lungs tend to empty unevenly, and the post dead space sample of exhaled gas that is analysed for CO does not represent that of whole lungs and for this reason, in Europe, the diffusion capacity is termed as ‘transfer factor’ to emphasize that it is more a measure of the lungs overall ability to transfer a gas into the blood rather than a specific test of diffusion. Nevertheless, the test provides considerable information about gas exchange in normal lungs. Even in patients with advanced lung disease, the results provide useful information to assess the severity and the type of pulmonary abnormality.

Steady-state Method: In this method, the subject breathes a low concentration of CO (about 0.1%) for about 30 secs, until a steady state of gas exchange has been reached. The constant rate of disappearance of CO from alveolar gas is than measured for a further short period, along with the alveolar concentration of the gas. This technique is better suited for measurements during exercise, when breath hold becomes a problem. This method is technically difficult and gives lower values than single breath method. This method is primarily employed in research settings.

Intrabreath Method: More recently, with the development of rapidly responding infrared analyser, the diffusing capacity can be measured using a single breath-slow exhalation, or ‘intrabreath technique’. The gas concentrations are monitored continuously during slow inhalation and exhalation. Multiple estimates of DL can be made during a single exhalation, giving DL as a function of lung volume. Alternatively, a single estimate of DL can be obtained by applying a linear regression to exhaled CO concentration continuous measured during slow exhalation.

The measurement of DLCO is variable compared to spirometric observations and criteria for acceptable measurement of DLCO have been based upon relative or absolute difference between repeated measurements. The ATS/ERS consensus statement recommends reporting the average of two measurements, both of which agree within 3.0 ml/min/mm Hg or within 10 percent of the higher measured value. The other technical considerations also accept the time of breath-holding maneuver in range of 8 to 12 seconds. It is also important to note that patient should refrain from smoking for at least 12 hours to avoid elevation of carboxyhaemoglobin levels. Inhaled bronchodilators are avoided on the day of the test and there should be no supplement oxygen for at least 15 minutes prior to or during the test.

Conditions causing decrease in DLCO are:

1. Factors/conditions affecting \( \text{VA} \) or pulmonary capillary bed either directly or indirectly i.e. pulmonary vascular disorders, pulmonary emboli, pulmonary vasculitis etc.
(2) Conditions causing changes in \( V_c \) in patients with infiltrative disorders of inter alveolar septum that obliterate/destroys capillaries i.e. sarcoidosis, diffuse interstitial fibrosis, berylliosis, collagen vascular disorders etc.

(3) Conditions causing changes in \( D_m \) by intra alveolar filling process or increasing blood diffusing pathway or true alveolar capillary block i.e. pneumonia, pulmonary oedema, pulmonary alveolar proteinosis etc.

(4) Conditions causing decrease in both \( V_c \) & \( D_m \) i.e. removal of lung tissue by surgery (pneumonectomy), destruction of lung tissue by disease process (emphysema).

(5) Obstructive lung diseases with non-uniform \( V_a/Q \) distribution

(6) Conditions causing decrease in \( \Theta \) (Hb concentration) i.e. anaemia.

(7) During oxygen inhalation

Conditions causing increase in DLCO are:

(1) Increase DL\(_{CO}\) signifies increase in \( V_c \) secondary to haemodynamic changes in pulmonary circulation i.e. increase in pulmonary arterial or left atrial pressure or increase in pulmonary blood flow i.e. early stages of LVF, left to right intra cardiac shunt etc.

(2) Increase in \( \Theta \) i.e. polycythaemia, high altitude (decrease capillary PO\(_2\)).

(3) During attack of bronchial asthma.

(4) Pulmonary haemorrhage, Mueller maneuver, supine position etc.

Measurement of diffusion lung capacity is important in defining abnormalities and responses to treatment in interstitial lung diseases. In sarcoidosis change in DL\(_{CO}\) are more sensitive indicator of response to treatment. It is useful to measure fresh pulmonary haemorrhage in good pasture syndrome. This is utilized to rule out COPD from asthma and also to rule out extra-parenchymal causes of restrictive lung diseases.

**Suggested Reading:**


(2) Klocke RA. Diffusion, chemical reactions and diffusion capacity. Fishman’s Pulmonary Diseases and Disorders. 4\(^{th}\) edn, Vol 1, McGaw Hill Medical, New York, 2008.


Introduction:
Postoperative pulmonary complications are an important source of perioperative morbidity and mortality. The rate of postoperative pulmonary complications across all types of surgery was 6.8 percent in a systematic review. Routine preoperative testing is unnecessary. It should be triggered by findings on examination, patient history, and review of systems and should be appropriate for the scheduled surgery.

Perioperative Pulmonary Physiology:
Reduced lung volume after surgery is a major factor contributing to the development of postoperative pulmonary complications. Thoracic and upper-abdominal surgeries are associated with a reduction in lung volumes in a restrictive pattern. Reduction of the FRC below closing volumes contributes to the risk of atelectasis, pneumonia, and ventilation/perfusion (V/Q) mismatching with consequent postoperative hypoxemia. Lower abdominal surgery is associated with similar changes but to a lesser degree. Reductions in lung volumes are generally not seen with surgery on the extremities.

Preoperative Risk Assessment:
Preoperative evaluation include focused history taking, evaluation of pertinent medical records, ordering and review of indicated testing, a patient interview, and a focused physical examination followed by assessment of lung function.

Clinical Evaluation:
A complete history and physical examination are the most important elements of preoperative risk assessment. Any history suggesting unrecognized chronic lung disease or heart failure, such as exercise intolerance, unexplained dyspnea, or cough, requires further consideration. All patients prior to major surgery should be screened for obstructive sleep apnea, in particular, through the use of the STOP-BANG questionnaire. Physical examination should be directed toward evidence for obstructive lung disease, especially noting decreased breath sounds, wheezes, rhonchi, or prolonged expiratory phase.

Pulmonary Function Testing:
PFTs are not needed in the majority of patients undergoing extra-thoracic surgery. However, all candidates for lung resection should have preoperative pulmonary function tests performed. PFTs may also be useful in patients with known or suspected respiratory disease (e.g., reduced exercise tolerance, unexplained dyspnea, cigarette smoking >20 years, chronic obstructive pulmonary disease [COPD], interstitial lung disease). Spirometry is widely available, and measures of the forced expiratory volume in one second (FEV₁) and forced vital capacity (FVC) are commonly used.

Assessment of Oxygenation and Hypoxia:
Assessment of SpO₂ can help stratify risk, particularly before high-risk surgeries. Arterial blood gas (ABG) analysis is rarely needed. ABG might be useful in patients with a resting SpO₂ <93 percent, an abnormal serum bicarbonate, and severe abnormalities on PFTs (e.g., FEV₁ <1 liter). Current data do not support the use of preoperative arterial blood gas analyses to stratify risk for postoperative pulmonary complications.

Chest Radiographs:
Routine chest radiography is not recommended by the American College of Radiology but may be indicated in the presence of symptoms, findings on examination, or prior abnormal radiograph. It is reasonable to obtain a preoperative chest radiograph in patients with known cardiopulmonary disease and in those over age 50 years undergoing high risk surgical procedures, including upper abdominal, aortic, esophageal, and thoracic surgery.

Exercise Testing:
Cardiopulmonary exercise testing (CPET), which includes calculation of maximum oxygen uptake and ventilatory anaerobic threshold, is used to assess patients with abnormal PFTs to determine the safety of planned lung resection surgery. CPET may also have a role in the evaluation of patients with unexplained dyspnea who are undergoing non-cardiopulmonary surgery. A simplified form of exercise testing that can be accomplished in an office setting is the six-minute walk test.
**Estimating Post-operative Pulmonary Risk of Respiratory Failure:**

Risk prediction tools are useful to stratify risk when advising patients before surgery and, in some cases, to identify patients most likely to benefit from risk-reduction interventions. The ARISCAT index\(^\text{10}\) use readily available clinical information and provides an estimate of the risk of any postoperative pulmonary complications. The two Gupta risk calculators are used to establish the risk of a single complication, either pneumonia or respiratory failure\(^\text{11}\). The Arozullah index\(^\text{12}\) will be of use primarily in research settings. These tools are a useful starting point when estimating pulmonary risk before major non-cardiac surgery.

**Pre-operative pulmonary physiologic evaluation for Lung Resection:**

Lung resection is frequently considered in patients with lung cancer, and less commonly, in patients with some benign disorders (eg, localized bronchiectasis). In many cases surgical resection needs to be considered in patients with impaired pulmonary function who have risk factors for complications. The evaluation involves assessing the effect of resection on the postoperative level of lung function as well as on the development of cardiopulmonary complications.

**General Assessment including Cardiovascular Risk:**

For patients who are being evaluated for pulmonary resective surgery (i.e., lobectomy, pneumonectomy, wedge resection), a general history and examination should be performed. The clinician should also specifically look for a past history of resections (eg, surgery for old tuberculosis or bronchiectasis), and presence of other chronic lung diseases like COPD, ILD which can affect tolerability of lung resection and post resective lung function.

**Chest Computed Tomography:**

With the specific resective surgery in mind (ie, pneumonectomy versus lobectomy), imaging should be evaluated for the anatomy of the region of the lung to be resected and can also be used to count segments for postoperative lung function prediction.

**Pre-operative Pulmonary Function:**

The forced expiratory volume in one second (FEV\(_1\); ie, spirometry) and the diffusing capacity for carbon monoxide (DLCO) should be measured in all patients in whom resectional surgery is being considered. If testing has not been performed within the previous 6 to 12 months, new testing should be requested. American College of Chest Physicians (ACCP)\(^\text{13}\) and the European Respiratory Society/European Society of thoracic surgeons (ERS/ESTS)\(^\text{14}\) guidelines are available for physiological evaluation of patients being planned for resective surgery especially for lung cancer.

Patients with a preoperative FEV\(_1\) and DLCO that are both ≥80 percent predicted do not need to undergo further testing for assessing postoperative lung function or risk. These patients are considered low risk and can generally tolerate lobectomy or pneumonectomy.

Patients with a preoperative FEV\(_1\) or DLCO <80 percent predicted need to undergo further evaluation to allow calculation of predicted postoperative (PPO) lung function. While the ACCP supports PPO lung function assessment in this group, the ERS/ESTS suggest performing a cardiopulmonary exercise test (CPET).

PPO values for FEV\(_1\) and DLCO take into account the preoperative values, the amount of lung tissue to be resected and its contribution to overall lung function. The contribution of the region of lung that is to be resected to overall lung function can be determined by quantitative lung scintigraphy or by lung segment counting; the latter is typically performed on chest computed tomography. Perfusion scintigraphy is the most widely used method in patients undergoing pneumonectomy while lung segment counting is recommended for patients undergoing lobectomy.

**Quantitative lung scintigraphy: Perfusion method (pneumonectomy):**

\[
PPO\ FEV_1 = \text{preoperative FEV}_1 \times (1 – \text{fraction of total perfusion in the resected lung measured on radionuclide perfusion}).
\]

The absolute value obtained is then compared with the predicted value for FEV\(_1\) for that individual’s height, age, and gender to obtain the percent predicted postoperative FEV\(_1\). The same formula can be used to predict PPO DLCO by substituting values for diffusing capacity.

**Segment counting: Anatomic method (lobectomy):**

\[
PPO\ FEV_1 = \text{preoperative FEV}_1 \times (1 – a/b)\]

where “a” is the number of segments to be resected and “b” is the total number of unobstructed segments.

Patients with both PPO FEV\(_1\) and PPO DLCO ≥60 percent predicted are considered low risk and should tolerate surgical lobectomy or pneumonectomy.

For patients with either PPO FEV\(_1\) or PPO DLCO <60 percent predicted, and where both values are ≥30 percent predicted, a low technology exercise test (either stair climb or a shuttle walk test) should be performed. An incremental shuttle walk test (ISWT) distance greater than 400 meters (ie, 40 x 10 meter “shuttles”) has been associated with a maximum oxygen uptake (VO\(_{2}\)max) ≥15 mL/kg/minute [1]; these patients can undergo major thoracic surgery and do not need a CPET. Patients whose exercise ability is equal to or above 22 meters on the stair climbing test are considered low risk and the patient is deemed to have sufficient pulmonary function to undergo resectional surgery.
If either PPO FEV₁ or PPO DLCO is < 30 percent, a formal CPET with measurement of VO₂max should be performed. Where those who achieve a VO₂max > 20 mL/kg/minute (or over 75 percent predicted) are considered low risk, and < 10 mL/kg/minute (or < 35 percent predicted) are high risk. Patients who achieve a VO₂max between 10 and 20 mL/kg/minute have a wide range of risk and are considered as “moderate risk” per the ACCP guidelines, which also suggests individualizing the approach in this population (Fig 1).

In clinical practice, a rule of thumb which can be applied if methods to calculate PPO or access to CPET are not there is to follow the recommendations of the BTS¹⁵ which states that no further respiratory function tests are required for a lobectomy if the post-bronchodilator FEV₁ is >1.5 litres and for a pneumonectomy if the post-bronchodilator FEV₁ is >2.0 litres, provided that there is no evidence of interstitial lung dis-ease or unexpected disability due to shortness of breath.

Another useful practice point in patients posted for thoracic surgeries and major upper abdominal surgeries, is to start them on breathing exercises especially incentive spirometry in the preoperative period itself. This has been shown to greatly reduce the incidence of post operative pulmonary atelectasis.

Figure 1: Algorithm for pulmonary preoperative assessment of patients requiring lung resection

Conclusion:
Patients with pulmonary pathologies require thorough evaluation and planning for optimal outcomes following surgical procedures. The preoperative assessment is thus a valuable opportunity to mitigate risk and optimize and educate patients.

References:


Assessment of Fitness to Fly

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In-flight Physiology:
Atmospheric pressure is maximum at the sea level and decreases logarithmically with ascent due to decrease in gravitational forces. The atmospheric pressure at the sea level is 760 mm Hg. By Dalton’s law, the total pressure of the mixture of the gases is equal to the sum of the pressures exerted by each gas in the mixture. Oxygen constitutes approximately 21% of air, so the partial pressure of the oxygen at the sea level is 160 mm Hg. Commercial flights fly through the troposphere at an altitude of 38000 feet, but the cabin is pressurized to altitude of 8000 ft. The gas composition of troposphere is constant. At an altitude of 8000 ft, the atmospheric pressure is 564 mm of Hg, so the partial pressure of oxygen is 118 mm of Hg, which is equivalent to breathing 15% oxygen at sea level leading to hypobaric hypoxia. Normal individuals can tolerate this change and do not develop symptoms. Their SpO2 drops to 85-91%. However, those with pre-existing lung or cardiovascular diseases may not tolerate these changes. By Boyle’s law, the volume of a gas is inversely proportional to the pressure to which it is subjected, at a constant temperature. As the atmospheric pressure decreases, the volume of the gas in the closed cavities increase, human body temperature being constant at 37 degree Celsius. Thus, there is a risk of rupture of bullae or increase in size of closed pneumothorax if present.

Pre-flight assessment:
The following equations were recommended by BTS for estimation of in-flight PaO2:

\[ \text{(1) PaO2 (Alt) (mmHg)} = 0.410 \text{ PaO2 (ground) (mmHg)} + 1.7652 \]
\[ \text{(2) PaO2 (Alt) (mmHg)} = 0.519 \text{ PaO2 (ground) (mmHg)} + 11.855 \text{ FEV1} - 1.760 \]
\[ \text{(3) PaO2 (Alt) (mmHg)} = 0.453 \text{ PaO2 (ground) (mmHg)} + 0.386 (\text{FEV1%}) + 2.44.9 \]
\[ \text{(4) PaO2 (Alt) (mmHg)} = 22.8 - (2.74 \times \text{altitude in thousands of feet}) + 0.68 \times \text{PaO2 (ground) (mmHg)} \]

Martin et al in their study, assessed the performance of these predictive equations against hypoxic challenge test. Predictive equations led to an over-estimation of the requirement of in-flight oxygen.

Edvardsen et al in their landmark study conducted a prospective trial of patients using hypoxic challenge test as gold standard. They established the value of 6 minute walk test and oxygen saturation at sea level as a reliable means to diagnose the need for in-flight oxygen as well as preventing the need to conduct a resource intensive test like hypoxic challenge in a large proportion of patients.

If the baseline SpO2 < 92%, patients will require supplemental oxygen during flight. If the baseline SpO2 > 95% and SpO2 post 6MWT is > 84%, they may not require oxygen during flight. If SpO2 > 95% and SpO2 post 6MWT is < 84%, they will require HCT. If the baseline SpO2 is 92-95% and SpO2 post 6MWT is < 84% they will require supplemental oxygen during the flight. If SpO2 is 92-95% and SpO2 post 6MWT is > 84% they would require HCT.

With HCT if the PaO2 < 50 mmHg or Spo2 < 85 % while breathing a mixture with 15% oxygen concentration, then in-flight oxygen may be required.

Although the study was restricted to patients with COPD, general contours of the problem and the likely solutions could be drawn from it. A subsequent study did not show the effect of 6MWT as a good discriminating feature in obese individuals, highlighting the potential that remains to be addressed in this area.

Hypoxia altitude simulation test:
Also known as Hypoxic Challenge Test (HCT) is considered as gold standard in assessing the need for in-flight supplemental oxygenation. This involves drawing a sample of ABG at the beginning of the test. 15.1 percent oxygen mixture is administered via a tight-fitting mask or mouthpiece. Repeat ABG sample is drawn anytime Spo2 drops below 85% or at the end of 20 minutes. If PaO2 in less than 50 mmHg at the end of the study, patients may require in-flight supplemental oxygenation.

Hypobaric chambers:
These chambers simulate the reduced pressure conditions as well as reduced oxygen concentration as would be present in a flight. These devices have been used in a few studies; however, they have limited availability for routine use.
### Diseases | Remarks
--- | ---
**Asthma** | Risk of bronchospasm is present due to mucosal water loss due to low cabin humidity. HCT should be performed for patients of severe asthma irrespective of their baseline oxygen saturation. In case of a recent history of exacerbation, patients should be allowed to fly only when they are stable and the need for the use of rescue medications have reached their usual baseline. All patients should keep their rescue inhaler with a spacer device in their hand baggage. Patients of severe asthma should also keep oral corticosteroids with them⁷.

**COPD** | Risk of bronchospasm is present due to mucosal water loss due to low cabin humidity. In case of a recent history of exacerbation, patients should be allowed to fly only when they are stable usually 6 weeks after an exacerbation. All patients should keep their inhalers with a spacer device in their hand baggage. Patients should be evaluated for the presence of pneumothorax and bullous lung disease. Those with untreated closed pneumothorax should not be allowed to fly. In those with a prior history of pneumothorax, the risk of recurrence should be explained. Risk of recurrence is low if pleurodesis has been performed. They are at a high risk of VTE. For the prevention of VTE, they should be advised to drink plenty of water, remain mobile, may consider the use of graduated compression stockings, avoid alcohol, LMWH or NOAC’s should be used when the flight duration is more than 6 hours. LMWH 40 mg s.c on the morning of the flight and on the next day is recommended⁷.

**Interstitial lung diseases** | HCT should be considered if DLCO<50% of predicted, PaO2 < 70 mm of Hg or SpO2 < 95% after exercise⁷.

**Infectious diseases** | Patients of acute otitis media should travel only after two weeks of the episode. Those with highly contagious infections like measles, mumps, chicken pox, SARS, MERS, COVID-19 should be allowed to fly only when they become non-infectious. Patients with drug sensitive pulmonary tuberculosis should be allowed to fly only when two sputum smears are AFB negative. Those with drug resistant pulmonary TB should be allowed to fly when two consecutive AFB cultures are negative⁷.

**Obstructive sleep apnea** | Should avoid overnight travel. If already on CPAP, and overnight travel is unavoidable, should carry the CPAP device. Prior approval from the concerned airlines is necessary for carriage of the device and its battery. Alcohol should be avoided as it increases upper airway collapsibility⁷.

**Pulmonary embolism** | In-flight hypoxia

- Pulmonary vasoconstriction

- Worsening of V/Q Mismatch

- Increased pulmonary pressure’s

- Right heart strain

It is prudent to delay air travel for at least 2 weeks following the episode. Patient should be on anticoagulation to prevent recurrent embolism⁷.

**Pulmonary hypertension** | Those in NYHA WHO class I and II are fit to fly. Those in NYHA WHO class III and IV should receive in-flight oxygen by nasal prongs @ 2L/min. If already on LTOT, then flow rate should be doubled during travel⁷.

**Lung cancer** | They can travel if stable and if the oxygen saturation is normal at the sea level. However, they are at an increased risk of venous thromboembolism, pneumothorax, and infections particularly if receiving chemotherapy.

For the prevention of VTE, they should be advised to drink plenty of water, remain mobile, may consider the use of graduated compression stockings, avoid alcohol, LMWH or NOAC’s should be used when the flight duration is more than 6 hours. LMWH 40 mg s.c on the morning of the flight and on the next day is recommended⁷.

**Chest wall and neuromuscular disorders** | If FVC is less than 1 litre, HCT should be performed. For patients who are on NIV, they will require NIV during flight. Prior airline approval should be taken for the use of the device in-flight. Battery arrangements must be done beforehand and permission for carrying the same should also be taken. NIV settings may need adjustment at high altitudes. They should preferably travel during daytime and avoid sleeping during daytime in the flight⁷.

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**Table 1 : Disease specific recommendations for Fitness to Fly**
References:


Body Plethysmography

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Introduction:

Body Plethysmography is a technique which provides a detailed assessment of lung function by measurement of static lung volumes and specific airway resistance. The word Plethysmography is derived from the Greek ‘plethysmos’ which means increase or enlargement and ‘graphe’ means writing, hence plethysmograph refers to a device which records changes in the volume of an organ or limb. It was nearly 65 years ago in 1956 that Arthur B. DuBois, Julius H. Comroe Jr and their colleagues published two papers on the use of body plethysmography. The foundation of the technique was laid by Eduard Pflüger who described a “pneumonometer” as far back as 1882 followed by Jere Mead, who pointed out that there are three important factors altering the pressure when a person is breathing inside a closed chamber. These are the increase in temperature as a result of body heat, the change in the number of gas molecules as oxygen is taken up and carbon dioxide is eliminated, and any change in volume of some part of the gas. DuBois’s simple but brilliant contribution was to make the person breathe shallowly and rapidly, thus making it practical to record lung gas volume and airway resistance without confounding factors.

Rationale for Body Plethysmography:

- Body Plethysmography can record static lung volumes and capacities like Residual Volume (RV), Functional Residual Capacity (FRC) and Total Lung Capacity (TLC); as well as airway resistance and airway conductance; parameters that cannot be measured by Simple Spirometry which involves the measurement of dynamic lung volumes and flow rates. It also contributes in the procedure of measurement of static compliance of the lungs.
- Body Plethysmography can record all of these during breathing at rest and forced manoeuvres are not required, hence can be done even by relatively dyspnoeic patients.
- Body Plethysmography can even measure spirometric data using the same flow meter as used for resistance measurements.
- Thus it is a complete lung function testing system that measures lung mechanics parameters during normal and forced breathing in a single sequence of linked measurements.
- Moreover, Body Plethysmography measures lung volumes of both well ventilated and poorly ventilated areas of the lung compared to the Dilutional methods of measurement. Thus volume of non-communicating bullae can be estimated by the difference in volume measured by these two methods.

Indications for Whole-Body Plethysmography:

1) Measurement of lung volumes to distinguish between restrictive and obstructive processes as these may not be obvious from simple spirometry
2) Diagnosis of hyperinflation and evaluation of obstructive lung diseases, such as bullous emphysema and cystic fibrosis, that may produce artificially low results if measured using other methods
3) Estimation of volume of non-communicating bullae
4) The finding of a significant difference between FVC and SVC is an indication to determine static lung volumes
5) Quantification of restrictive lung disease
6) Further assessment of respiratory symptoms (breathlessness, cough, sputum)
7) Measurement of lung volumes when the subject is unable to perform multibreath tests
8) Follow-up assessment of the course of disease and response to treatment
9) Airway obstruction reversibility and provocation tests
10) Evaluation of resistance to airflow

11) Lung volumes are a pre-requirement in lung volume reduction surgery (LVRS) and surgery for bullous emphysema as the preoperative RV/TLC ratio is directly related to improvement in symptoms of breathlessness

Contraindications for Whole-Body Plethysmography:

1) The general contraindications for Pulmonary Function testing apply to Body Plethysmography also, thus it should not be performed in patients of:
   - Recent abdominal, thoracic, or eye surgery
   - Hemodynamic instability
   - Unstable angina/ recent MI within 1 month
   - Symptoms of acute severe illness like Chest pain, nausea, vomiting, high fever, dyspnea
   - Recent hemoptysis
   - Pneumothorax
   - Recent history of abdominal, thoracic, or cerebral aneurysm

2) Specifically, body plethysmography is impractical in patients with marked obesity, skeletal abnormalities or claustrophobia

Principle of Body Plethysmography:

The principle of body plethysmography is based on Boyle's Law which states that in an enclosed gaseous system under isothermal conditions, the changes in pressure and volume of a gas are inversely related. This means that the product of pressure and volume of the gas remains constant. Thus \( PV = (P+\Delta P) (V+\Delta V) \), where \( P \) and \( V \) are the pressure and volume of a mass of gas respectively, \( \Delta P \) is the change in pressure and \( \Delta V \) is the associated change in volume of the same mass of gas in the defined space and at constant temperature.

Technique of Body Plethysmography:

Apparatus: Body plethysmograph, colloquially known as Body Box, is essentially a rigid closed box where the patient sitting inside it makes respiratory manoeuvres resulting in small changes of pressure, which are recorded. There are three different types of body plethysmographs available: i) the pressure plethysmograph, in which pressure during breathing varies while volume remains constant ii) the volume plethysmograph, in which the volume varies during breathing while the pressure remains constant iii) the pressure corrected, flow plethysmograph which combines the characteristics of pressure and volume plethysmographs. The conceptual basis of all three devices is the same, but the most commonly used is the pressure plethysmograph. The pressure body plethysmograph or body box is a chamber resembling a transparent telephone box in which a person can be comfortably seated and which is closed with an airtight seal during measurement (Figure 1).

Figure 1: Schematic Diagram of Pressure Body Plethysmograph
It has a pneumotachograph and transducer for measuring flow and volume and two pressure transducers, one for measuring pressure inside the box relative to ambient pressure (Pbx) and the other for pressure at the mouth (Pm). A solenoid operated shutter mechanism is situated between the mouth piece and the pneumotachograph. The three transducers are connected to an amplifying system so that Pbx and Pm are displayed simultaneously on the X and Y axes respectively, of an oscilloscope.

**Method for determination of FRC by Body Plethysmography**:

The patient is seated comfortably within the box with nose clip in place, hands on his cheek to minimize pressure changes due to the oral cavity and asked to breathe quietly (tidal breathing) into the mouthpiece. After few breaths, at end of quiet expiration as seen on spirogram, the shutter is closed and patient is asked to pant (rapid shallow breaths) gently into the mouthpiece. In this closed system with no air flow as shutter is closed, the respiratory movements cause changes in both mouth pressure and box pressure. The box pressure is calibrated to volume changes initially itself by introducing a small known volume of air into the sealed box and recording the pressure changes. The mouth pressure reflects the alveolar pressure when the shutter is closed as there is no airflow. The changes in mouth pressure and box pressure or lung volume caused by panting against a closed shutter in the enclosed body box, appear on the oscilloscope as a closed loop (Fig 2). The change of volume by which the lung generates positive or negative alveolar pressure is also known as shift volume. Measurement of the slope of this loop is used to determine the volume of the gas in the lungs. When shutter closure is at end tidal expiration, the lung volume would be the FRCpleth, also known as thoracic gas volume or TGV.

**Figure 2 : Pressure Volume tracing of Body Plethysmograph**

Pm= pressure at the mouth which reflects alveolar pressure when shutter closed,  
Pbx= pressure in the box which reflects lung volume as change in Pbx is calibrated initially to record the change in volume,  
ΔP= change in pulmonary pressure produced by respiratory efforts,  
ΔV=change in gas volume in the lungs

Measuring airflow through the mouthpiece during tidal breathing, maximal inspiration to TLC and maximal expiration to RV followed by the measurement of TGV is done as a combined procedure. This should be followed by a prolonged forced expiration to determine the forced expiratory volume in 1 s (FEV1) and the forced vital capacity (FVC). The spirometric data can be conveniently recorded by the same flow meter as used for resistance measurements. In this way information on lung mechanics during normal and forced breathing can be obtained in a single sequence of linked measurements enabling both static and dynamic lung volumes to be calculated by the body plethysmograph. The procedure is repeated until reproducible results are obtained.

**Mathematical Application of Boyles Law to FRC determination by Body Plethysmograph**:

Boyles law states that:

\[ PV = (P+\Delta P) (V+\Delta V) \]

On inspiration, pressure at the mouth falls relative to atmospheric pressure and due to chest wall expansion, the box pressure rises. Thus above equation is written as:

\[ PV = (P-\Delta P) (V+\Delta V) \]

Thus, \( PV=PV+P\Delta V-V\Delta P-\Delta V\Delta P \)
As $\Delta V \Delta P$ value is negligible, it can be ignored and then the equation is written as:

$$P \Delta V = V \Delta P \text{ or } V = \frac{P \Delta V}{\Delta P}$$

Where $V = \text{FRC}$ since shutter is closed at end tidal expiration, $P$ is alveolar pressure or $P_m$ which is known and $\frac{\Delta V}{\Delta P}$ is the inverse of the slope of the loop on the oscilloscope. Thus FRC can be calculated by Body Plethysmography.

**Method for determination of Airway Resistance by Body Plethysmography:**

Airway resistance and specific airway conductance are measured at the same time as lung volumes in body plethysmograph. Airway resistance ($R_w$) is the pressure required (alveolar to mouth) to generate a unit of airflow through the airways. Thus it is the ratio of the driving pressure for flow to the actual rate of air flow along the airways including mouth, nasopharynx, larynx, and central and peripheral airways. However, since the alveolar pressure that is needed for the determination of the proper airway resistance is not available during free breathing, the measurement is done by relating flow rate to box pressure, which are directly measurable by body plethysmography. The ratio of box pressure to flow rate, expressed in suitable units, is called specific airway resistance or $s_{Raw}$. It is actually not a resistance as it is the product of airway resistance and the lung volume at which the $R_w$ was measured and depends on both lung volume and airway resistance. If airflow is plotted on the vertical axis versus box pressure on the horizontal axis, closed curves are obtained. The reciprocal slope of these breathing loops represents specific airway resistance. In healthy subjects the curves are approximately straight lines, while in patients with respiratory diseases various patterns can be recognized (Figure 3).

![Figure 3: Schematic representation of $s_{Raw}$ loops in 1) normal subject, 2) patient with increase large airways resistance, 3) patient with chronic airflow obstruction, 4) patient with obesity or diaphragmatic paralysis, and, 5) patient with upper airway obstruction](image)

In case of asymmetries in the loops, for correct interpretation, it is necessary to determine an average $s_{Raw}$, which is also known as effective specific airway resistance or $s_{Raw}^{eff}$ and this is done by modern software by computing a weighted average over the breathing cycle.

Airway conductance ($G_w$) is the reciprocal of $R_w$, thus it is the airflow generated per unit of pressure. Specific airway conductance ($SGaw$) is usually reported because the conductance of the airways increases with lung volume. $SGaw$ is calculated by dividing airway conductance by lung volume. Thus $SGaw = G_w / V$ where $V$ is the lung volume at which $G_w$ was measured.

**Mathematical determination of Airway Resistance by Body Plethysmograph:**

The patient seated in the Body Box is asked to pant at a rate of 2 breaths/second against the mouthpiece, while airflow is measured using a pneumotachograph. The reciprocal of the slope of the curve obtained by plotting of box pressure on x axis and airflow on y axis, gives the $s_{Raw}$. While the panting continues, the shutter at the airway opening is closed so that airflow is transiently interrupted and the changes in pressure in the plethysmograph ($P_{bx}$) which correspond to changes in lung volume, and pressure at the mouth ($P_m$) are recorded on the x and y axes respectively, of the oscilloscope. Since there is no airflow as shutter is closed, $P_m$ equals the alveolar pressure ($P_A$). This is the same technique which is used to measure $FRC_{pleth}$ and in fact $R_w$ is normally estimated at FRC to standardize the measurement.

The first procedure with open shutter provides the relationship between airflow and $P_{bx}$. The second procedure with closed shutter determines the relationship between $P_A$ and $P_{bx}$. Airway resistance is calculated by dividing the slope of the loop obtained by plotting $P_A$ versus $P_{bx}$ while the shutter is closed by the slope obtained by plotting flow versus $P_{bx}$ while the shutter is open.

$$R_w = \frac{P_A}{P_{bx}} \frac{V}{P_{bx}}$$
Thus $\text{Raw} = \frac{P_a}{V}$

Where $\text{Raw} = \text{airway resistance (cmH}_2\text{O/L/s)}$, $P_a = \text{alveolar pressure (cmH}_2\text{O)}$, $V = \text{airflow (L/s)}$

**Interpretation of Body Plethysmography report**:

A Pulmonary function test report cannot be viewed in isolation and must be interpreted along with the clinical diagnosis as well as supporting lab data and imaging. A body plethysmography report is to be read with the accompanying simple spirometry report. It specifically displays the FRC, RV, TLC, RV/TLC ratio, ERV and effective $\text{SRaw (sReff)}$ as seen in Figure 4.

**Figure 4 : Body Plethysmograph Report**

1) Body Plethysmograph report has to be read in conjunction with simple spirometry and it is determined whether spirometry is normal or shows restriction or obstruction.

2) A **restrictive disorder** can be suspected from spirometry when vital capacity (VC) is reduced and the ratio of forced expiratory volume in 1 s (FEV$_1$) to FVC (FEV$_1$/FVC) is normal or elevated.
   a) However, it is definitely proven only by a decrease in TLC. In fact, restrictive disorders are defined as TLC being below the 5th percentile of normal values.
   b) There is also a reduction in ALL lung volumes i.e TLC, FRC, RV in a restrictive disorder.
   c) If the restriction is due to an intrinsic cause like interstitial lung disease, the RV/TLC ratio remains normal.
   d) If the restriction is due to extrinsic causes like kyphoscoliosis or neuromuscular disease, then the TLC is reduced because of mechanical limitation to chest wall expansion or due to respiratory muscle weakness while RV is normal because lung tissue and elastic recoil are normal; hence RV/TLC ratio will be high.

3) An **obstructive disorder** can be suspected from spirometry when the ratio of forced expiratory volume in 1 s (FEV$_1$) to FVC (FEV$_1$/FVC) is reduced.
   a) The dynamic compression of airways in obstructive lung diseases and decrease in lung recoil due to destruction of elastic tissue especially in emphysema; results in increase of some lung volumes i.e TLC, FRC and RV. The Vital capacity however may be normal or may be reduced due to air trapping.
   b) The increase in RV is disproportionate to the increase in TLC leading to an increased RV/TLC ratio in obstructive lung disease. It is called Air trapping if the TLC is normal with an increase in RV/TLC; while an increase in both TLC and RV/TLC is called hyperinflation.
c) Values of RV above the 95th percentile but below 140% predicted are indicative of mild, values between 140 and 170% predicted of moderate, and values above 170% predicted of severe hyperinflation.

d) The disproportionate increase in RV leads to reduction in FVC as TLC=RV+VC, this is called pseudo restriction.

4) In some patients there may be a **mixed pattern** of restriction and obstruction. This may be indicated by an obstructive pattern on spirometry combined with reduced lung volumes.

5) Lung volumes are also useful when there are **equivocal findings** on spirometry. A finding of raised TLC or RV supports a diagnosis of obstructive airway disease even when FEV1 and FVC are in normal range, albeit at lower limits.

Table 1 summarizes the interpretation of lung volumes measured by Body Box.

<table>
<thead>
<tr>
<th>VOLUME</th>
<th>RESTRICTIVE INTRINSIC</th>
<th>RESTRICTIVE EXTRINSIC</th>
<th>AIR TRAPPING</th>
<th>HYPERINFLATION</th>
</tr>
</thead>
<tbody>
<tr>
<td>TLC</td>
<td>DECREASED</td>
<td>DECREASED</td>
<td>NORMAL</td>
<td>INCREASED</td>
</tr>
<tr>
<td>VC</td>
<td>DECREASED</td>
<td>DECREASED</td>
<td>DECREASED</td>
<td>N or slight increase</td>
</tr>
<tr>
<td>FRC</td>
<td>DECREASED</td>
<td>DECREASED</td>
<td>INCREASED</td>
<td>INCREASED</td>
</tr>
<tr>
<td>RV</td>
<td>DECREASED</td>
<td>NORMAL</td>
<td>INCREASED</td>
<td>INCREASED</td>
</tr>
<tr>
<td>RV/TLC %</td>
<td>NORMAL</td>
<td>INCREASED</td>
<td>INCREASED</td>
<td>INCREASED</td>
</tr>
</tbody>
</table>

**Table 1 : Summary of Interpretation of Body Plethysmography**

6) **Interpretation of Airway Resistance**:  
Defining the range of normal for Raw is difficult because of inter and intra individual variations of Raw with lung volumes. One classification proposes defining abnormal Raw in adults in whom FRC exceeds 2L as follows: Mild increase is defined as Raw 2.8 - 4.5 cmH2O/L/s, moderate as between 4.5-8.0, severe as > 8.0 cmH2O/L/s. It is prudent to evaluate both Raw and sRaw because resistance to airflow varies at different lung volumes as airways are wider at high lung volumes than at low lung volumes. Patients with COPD and severe hyperinflation may show only a moderate elevation of Raw as they have high FRC, while sRaw will be severely affected.

**Conclusion**:

Body Plethysmography is a non-invasive lung function test providing important additional information on lung volumes and airway resistance, over and above that provided by spirometry. It is less effort dependent than spirometry, and a relatively quick procedure. The measurements are performed during quiet breathing; hence it is more physiological. Though the equipment required is quite expensive, yet Body Plethysmography is valuable in assessing respiratory disorders and is considered the gold standard for lung volume measurements.

**References**:

Introduction:
Cardio-pulmonary exercise testing (CPET) is a form of exercise testing which involves the analysis of gas exchange during exercise at incrementally increasing intensity. A computerised protocol provides breath by breath measurement of volume of oxygen consumption (VO2), volume of carbon dioxide (VCO2) production, air expired (VE) and cardiac parameters like blood pressure, pulse rate etc. It provides information beyond standard exercise testing and is also a reproducible quantitation of cardiorespiratory fitness. It also provides diagnostic and prognostic information for cardiovascular and pulmonary diseases.

Indications (American Heart Association):

Class I - (Indicated)
1. Evaluate exercise capacity and response to therapy in heart failure patients being considered for transplantation.
2. Differentiate cardiac versus pulmonary limitation for dyspnea on exertion

Class IIa - (good supportive evidence)
1. Evaluate exercise capacity when indicated for medical reasons when subjective estimates (exercise test time or work rate) are unreliable.

Class IIb - (weak supportive evidence)
1. Evaluate response to intervention in which exercise tolerance is an important end point.
2. Determine exercise training intensity for cardiac rehabilitation.

Class III - (Not indicated)
Routine use to evaluate exercise capacity.

Procedure:
The patient exercises on a Treadmill or on a bicycle ergometer. The latter is preferred because work rate can be directly measured. The patient is connected to a mouthpiece attached to a spirometer and a metabolic cart. Hence real-time measurements on ventilatory and gas exchange parameters are obtained. Other parameters apart from electrocardiography and non invasive blood pressure, which are continuously monitored are pulse rate, oxygen saturation with pulse oximetry, flow, volume, exhaled oxygen and carbon dioxide concentrations. Incremental or constant work load protocol may be used. The test needs to be terminated if the patient (a) develops significant hypoxemia (b) gets fatigued or exhausted or (c) develops myocardial ischemia, arrhythmia, significant blood pressure elevation etc, suggesting cardiovascular instability.

Physiology of Coupling of External to Cellular Respiration:
The journey of oxygen extraction by the lungs from the air and its transport via the pulmonary and systemic circulation till its utilisation at the cellular level is depicted in Figure 1.
Figure 1. Gas transport mechanisms coupling cellular (internal) respiration to pulmonary (external) respiration.

Circ = circulation; CO$_2$ = carbon dioxide; Consum = consumption; Creat = creatine; Lac = lactate; HR = heart rate; Mito = mitochondria; PO$_4$ = phosphate; O$_2$ = oxygen; Periph = peripheral; Prod = production; Pulm = pulmonary; Pyr = pyruvate; QCO$_2$ = carbon dioxide production; QO$_2$ = oxygen utilization; SV = stroke. Takes into account delivery, extraction and utilisation of oxygen.

(From: Principles of Exercise Testing and Interpretation, 3rd ed, with permission from Lippincott Williams & Wilkins.)

**Fick Equation – Clinical Interpretation:**

\[ VO_2 = (\text{CARDIAC OUTPUT}) (A - VO_2) \]

\[ VO_2 = HR \times SV \times (CaO_2 - CvO_2) \]

HR \( \rightarrow \) variation indicates Sinus node dysfunction or maybe due to the effect of drugs

SV \( \rightarrow \) abnormalities point towards a possibility of Cardiomyopathies, contractility issues, EDV – ESV

CaO$_2$ \( \rightarrow \) is dependent on the PaO$_2$, hemoglobin and the pulmonary capacity

CvO$_2$ \( \rightarrow \) Skeletal muscle, Blood flow

**CPET – Important Terms:**

VO$_2$ = oxygen consumption (measure of CV-R fitness)

- Absolute (L/min) vs relative (ml/kg/min)

RER = respiratory exchange ratio (measure of effort)

- VCO$_2$/VO$_2$ - amount of CO$_2$ per O$_2$ consumed

- RER > 1.0 extra Co$_2$ produced-corresponds to lactate production

- RER > 1.10 considered maximal effort

VE/VCO$_2$ slope + vent efficiency/dead space

- Prognostic in HF: > 34 \( \rightarrow \) worse prognosis

Dyspnea index = peak exercise ventilation/MVV

- > 50 per cent = onset of dyspnea

- > 80 per cent = exercise ceases usually within 1 minute
Breathing reserve = (1- Dyspnea index )

$O_2$ pulse = $VO_2$/HR – SV x AVO2 difference. Surrogate for stroke volume

Several parameters are being measured and a lot of data is generated during CPET. However, all available information needs to be interpreted and clinically correlated judiciously.

$VO_2$- equals metabolic oxygen consumption. It increases linearly with the level of exercise/work intensity until it reaches a plateau (= $VO_2$ max- which is the best index of aerobic capacity) due to cardiac limitations and/or tissue extraction.

$VCO_2$- is the measured carbon dioxide output and is the same as its metabolic production. It increases at the same rate as $VO_2$ at lower work levels, but at higher work levels the $VCO_2$ rises more steeply. This is consequent to bicarbonate buffering of increased lactate accumulation due to onset of anaerobic metabolism.

**Aerobic threshold**- is the point at which the $VCO_2$ increases disproportionately to $VO_2$ due to onset of increased lactate production and bicarbonate buffering. It is a marker for the onset of metabolic acidosis during incremental work levels and usually occurs at 50-75 per cent of $VO_2$ max.

**Oxygen pulse**- is the ratio of $VO_2$ and heart rate and indicates the oxygen consumed per cardiac cycle.

The etiologies of exercise limitation are categorized into three types:

1. Cardiovascular diseases
2. Respiratory diseases
3. Physical deconditioning

CPET can help to identify the cause of exercise limitation. Cardiovascular causes can be assessed by analysing the oxygen pulse, anaerobic threshold and relationship to intensity of work. The pulmonary causes can be assessed by analysing the $paCO_2$, maximal respiratory rate and the respiratory reserve (peak VE/MVV). Gas exchange abnormalities can be derived from physiological dead space assessment, pulse oximetry, and calculating the alveolar-arterial oxygen gradient ($PA--aO_2$). The quantum of decrease in $VO_2$ max or maximal exercise capacity on CPET is indicative of the severity of the exercise limitation. The normal cardio-pulmonary response to exercise is shown in Figure 2.

### Normal Cardiopulmonary Response to Exercise

<table>
<thead>
<tr>
<th></th>
<th>Rest</th>
<th>Exercise</th>
<th>Increase</th>
</tr>
</thead>
<tbody>
<tr>
<td>$VO_2$, L/min</td>
<td>0.250</td>
<td>3.0-4.5</td>
<td>12-18 x</td>
</tr>
<tr>
<td>HR, bpm</td>
<td>70</td>
<td>180</td>
<td>2.5-3 x</td>
</tr>
<tr>
<td>SV, ml</td>
<td>70</td>
<td>105-140</td>
<td>1.5-2 x</td>
</tr>
<tr>
<td>CO, L/min</td>
<td>5</td>
<td>20-25</td>
<td>4-5 x</td>
</tr>
<tr>
<td>$V_E$, L/min</td>
<td>8</td>
<td>180</td>
<td>20-25 x</td>
</tr>
</tbody>
</table>

### Figure 2 . Normal Cardio-Pulmonary Response to Exercise

**V-Slope Method**:

When the net increase in lactate accumulation produces an acidosis, $VCO_2$ accelerates faster relative to $VO_2$. When you plot $VCO_2$ versus $VO_2$, the relationship is two separate, but linear, components. The intercept of these 2 slopes is the Ventilatory or Lactate or Anaerobic Threshold. This is shown in Figure 3 and Figure 4 respectively.
A sample CPET report of a 76 years old male is depicted in Figures 5 & 6.

The typical findings seen on CPET in cardiovascular disease and pulmonary disease are compared in Table 1.

**Table 1. Comparison of CPET findings in Cardiovascular and Pulmonary Diseases**

<table>
<thead>
<tr>
<th></th>
<th>Cardiovascular</th>
<th>Pulmonary</th>
</tr>
</thead>
<tbody>
<tr>
<td>Peak VO&lt;sub&gt;2&lt;/sub&gt;</td>
<td>Reduced</td>
<td>Reduced</td>
</tr>
<tr>
<td>Ventilatory threshold</td>
<td>Reduced</td>
<td>Normal or reduced</td>
</tr>
<tr>
<td>∆VO&lt;sub&gt;2&lt;/sub&gt;/∆WR</td>
<td>Often reduced</td>
<td>Normal</td>
</tr>
<tr>
<td>Peak HR</td>
<td>May be reduced</td>
<td>May be reduced</td>
</tr>
<tr>
<td>Peak VO&lt;sub&gt;2&lt;/sub&gt;/HR</td>
<td>Often reduced</td>
<td>May be reduced</td>
</tr>
<tr>
<td>Breathing reserve, 1</td>
<td>&gt;20%</td>
<td>&lt;15%</td>
</tr>
<tr>
<td>Postexercise FEV&lt;sub&gt;1&lt;/sub&gt;</td>
<td>Unchanged from rest</td>
<td>May decrease compared with rest</td>
</tr>
<tr>
<td>Pa&lt;sub&gt;O2&lt;/sub&gt; or Sa&lt;sub&gt;O2&lt;/sub&gt;</td>
<td>Normal</td>
<td>Often reduced</td>
</tr>
<tr>
<td>VD/Vt (tidal volume or Ve/Vco&lt;sub&gt;2&lt;/sub&gt;)</td>
<td>May be elevated</td>
<td>Often elevated</td>
</tr>
</tbody>
</table>

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Conclusion:

CPET can be performed in order to determine the cause of dyspnea viz. pulmonary versus cardiac (or both) versus deconditioning versus obesity. It is a very sensitive test and can identify even subclinical disease. It can therefore be performed to objectively measure functional capacity as for preoperative assessment or disability evaluation and as a run up to heart and/or lung transplantation. CPET can also be performed to determine prognosis and measure response to therapy in heart failure patients, optimise settings for rate-adaptive pacemakers and research.

References:

Assessment of Respiratory Muscle Strength

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Introduction:
Assessment of respiratory muscle strength forms a critical part of pulmonary function testing. It is highly under-utilised in clinical settings and needs to be understood better by clinicians to make the best use of testing. Respiratory muscle strength testing can be done by multiple methods such as the maximal inspiratory pressure (MIP), the maximal expiratory pressure (MEP), and sniff nasal inspiratory pressure (SNIP) etc. The MIP identifies the strength of the diaphragm and other inspiratory muscles, and the MEP tells us about the strength of the expiratory muscles, including abdominal muscles. SNIP measurement is one additional test of inspiratory muscle strength which is relatively easier to perform.

Indications: The indications for respiratory muscle strength testing include:
1. Suspicion of respiratory muscle weakness such as known neuromuscular disease or unexplained dyspnea
2. Reduced vital capacity of unknown aetiology
3. Follow-up of patients with respiratory muscle weakness to assess progression/response to therapy

Various methods:
Respiratory muscle strength can be assessed using voluntary movement test as well as non-voluntary movement tests. Commonly used tests include MIP, MEP, SNIP and transdiaphragmatic pressure measurement. Some measures which can be used as an indirect measure of respiratory muscle weakness include fall in FVC in the supine position and isolated fall in maximum voluntary ventilation. Here we will discuss the principles and performance of the commonly used tests for respiratory muscle testing.

Performance of the procedure:
Maximal inspiratory pressure (MIP) and maximal expiratory pressure (MEP) can be easily measured using the lung function testing machines with some additional hand-held attachments and software downloaded on a computer and provides a real-time graph of the procedure.

Maximal inspiratory pressure (MIP):
The procedure aims to assess the generated maximum negative pressure during inspiration. For patient's best effort, it is essential to explain and demonstrate the manoeuvre to the patient. The rubber mouthpiece with flanges is placed, and the patient is asked to seal his lips around the mouthpiece. He is, then, asked to exhale slowly to empty the lungs. Following this, the patient is instructed to breathe-in forcefully and ask to maintain the inspiratory pressure for at least 1.5 seconds. The largest negative pressure sustained for at least one second should be taken as MIP rather than the transient peak. This manoeuvre is repeated five times, and the variability between these manoeuvres should be less than 10 cm H2O. If the patient has facial muscle weakness, he may be asked to use hands to press their lips around the mouthpiece to avoid leakage. The report should include the maximum value as well as the predicted value.

Maximal expiratory pressure (MEP):
It is the most commonly used measure of expiratory muscle strength. It is rapid and simple to perform as it uses low-cost equipment and has well-established reference values with a lower limit of normal being 150 cm H2O for males and 120 cm H2O for females. However, it requires patient co-operation and co-ordination between the patient and examiner. It can high false-positive results due to submaximal efforts or leaks around the mouth, which is common in patients with facial weakness. It is measured using a pressure manometer, usually in a sitting position with a nose clip in place. The patient is asked to perform maximum forceful exhalation, and it should be sustained for 1-2 seconds. This manoeuvre should be repeated 3-8 times, and the highest value recorded is used for interpretation. This test can be performed from total lung capacity to exhalation or from function residual capacity to exhalation. The values obtained via TLC to exhalation are usually higher than those obtained from FRC. This test helps in the assessment of cough strength, especially in patients with neuromuscular weakness.
Sniff nasal inspiratory pressure (SNIP):

Sniff nasal inspiratory pressure is a non-invasive way of measurement of inspiratory muscle strength, including the diaphragm. It usually accurately reflects the oesophageal pressure (Pes), having the advantage of avoiding the oesophageal catheter. However, the correlation between SNIP and oesophageal pressure is reduced in patients with airway obstruction. It should be used as an additional test of inspiratory muscle strength and MIP as a single test may overestimate muscle weakness and use of more than one test reduces the chances of false-positive results. It has the advantage of being inexpensive, easily doable and reproducible. The lower limit of normal for males is usually 70 cm H2O while for females it is 60 cm H2O. However, the test requires patient co-operation and cannot be used for patients on mechanical ventilation. It also underestimates the muscle power in patients with airway obstruction. The test is usually performed in the sitting position. The patient is asked to completely close one nostril by nose plug to prevent leaking whereas the other nostril is patent. The patient is asked to take a deep inspiration with mouth closed. This inspiration should be very short and explosive, causing the collapse of the unplugged nostril. At least ten tests should be performed to avoid any false-positive results.

Transdiaphragmatic pressure:

Transdiaphragmatic pressure (Pdi) is the difference between gastric pressure (Pga) and Pes (Pdi = Pga – Pes). It depicts the force generated by the diaphragm during inspiration. As the diaphragm is a chief inspiratory muscle, responsible for 60-70% of inspiratory force, Pdi is one important parameter of inspiratory muscle strength. This test has been there for a long time, and the lower limit of normal value for males is 80 cm H2O while for females it is 70 cm H2O. However, it is an invasive test as it requires catheter placement into oesophagus and stomach, which can be discomforting for the patient. The availability of catheters may also be a difficult and cost is also high. The correct placement of the catheter requires an experienced operator.

Two air-filled latex balloon catheters are placed, one in the stomach and other in the oesophagus. A single catheter with two balloons can also be used. These catheters are connected to the manometer, which displays the oesophageal and gastric pressure curves on the screen. To ensure correct positioning, it is necessary to observe the Pes and Pga curves. During inspiration, Pes is negative, while Pga is positive. Pdi can be measured during quiet breathing as well as during sniff manoeuvres. Additionally, magnetic stimulation of the phrenic nerve can also be done to measure Pdi. This method is complex and costly and does require a high level of competence for correct recording and interpretation.

Electrical and magnetic phrenic nerve stimulation:

These are non-volitional tests for respiratory muscle strength assessment and are extremely useful for patients who have difficulty in understanding commands or are on mechanical ventilation. To obtain maximal involuntary inspiratory contraction, electrical stimulation or magnetic stimulation of the phrenic nerve can help. These tests are based on the principle of stimulating the cervical phrenic nerve and incite a diaphragm contraction. Electrical nerve stimulation requires electrode placement and is painful, while magnetic phrenic nerve stimulation causes less discomfort and is tolerated by most patients. It works on the principle of creation of a magnetic field in the neck by using the coils. The PDI values measured by electrical and magnetic stimulation are usually similar. Due to its more comforting nature, magnetic stimulation has replaced electrical stimulation in current practice. The lower limit of normal for Pdi by magnetic stimulation is 20 cmH2O. The disadvantage is that it can sometimes be non-specific and may stimulate neck muscles, causing jerky contraction. The instrument used is costly and not readily available in all centres.

The device consists of a generator with coils attached to it. Most often, a 90-mm circular coil is used, and it gives unilateral stimulation. However, a figure of eight coil can also be used, which provides bilateral stimulation. The major disadvantage of Pdi measurement is the need for placement if gastric and oesophageal balloon catheters. It is used most often in research settings and mechanically ventilated patients.

Diaphragm Ultrasound:

Ultrasonography of the diaphragm can also be used to assess the strength and thickness. It can be done by the vascular probe in the zone of apposition where diaphragm appears as a thick band between the pleural and peritoneal line. The thickness of the diaphragm and thickness fraction can be measured to identify and follow-up the diaphragm function. Use of a curved probe allows for measuring diaphragm excursion during quiet breathing as well as during sniff manoeuvre.

Conclusion:

Use of various tests discussed above helps in measurement of respiratory muscle weakness. The principles, advantages and disadvantages of each test should be understood well to use them appropriately. MIP and MEP remain most easily available and used tests for this purpose. However, in current times the use of ultrasound for this purpose is increasing.
Impulse Oscillometry

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Introduction:

Pulmonary function tests are the cornerstone in the diagnosis of obstructive airway disease. In 1956, DuBois et al. described the forced oscillation technique (FOT) as a tool to measure lung function using sinusoidal sound waves of single frequencies generated by a loud speaker and passed into the lungs during tidal breathing. In 1975, Michaelson et al. improvised the technique to use multiple frequency sound waves which was named impulse oscillometry (IOS). In 1998 Jaeger made computerised IOS commercially available.

Principle of Impulse Oscillometry:

Superimposition of sound waves on normal tidal breathing, which leads to disturbances in flow and pressure across the airways, leading to an ultimate output of respiratory resistance, reactance and impedance. This principle of IOS is derived from the Ohm’s law which states that resistance is a product of division of pressure and flow.

<table>
<thead>
<tr>
<th>Terminology</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>Impedance (Xrs)</td>
<td>A calculation of the total force needed to propagate a pressure wave through the pulmonary system, comprising resistance and reactance</td>
</tr>
<tr>
<td>Resistance (R)</td>
<td>Energy required to propagate a pressure wave through the airways; to pass through the bronchi and bronchioles, and to distend the lung parenchyma. Resistance is determined when a pressure wave is unopposed by airway recoil and is in phase with airflow</td>
</tr>
<tr>
<td>Reactance (X)</td>
<td>Energy generated by the recoil of the lungs after distention by a pressure wave out of phase with airflow</td>
</tr>
<tr>
<td>Area of reactance (AX)</td>
<td>Area under the curve between the reactance values for 5Hz and the resonance frequency. (Figure 3)</td>
</tr>
<tr>
<td>Resonance frequency (Fres)</td>
<td>The frequency at which the lung tissue moves from passive distention to active stretch in response to the force of the pressure wave signal; graphically when reactance is zero. (Figure 3)</td>
</tr>
</tbody>
</table>

Table 1. Terminologies used in Impulse Oscillometry

Sound waves with higher frequencies (20 Hz) travel shorter distances, generally till the large airways. Hence, the resistance at 20 Hz (R20) represents the resistance of the large airways. Sound waves with lower frequencies (5 Hz) travel larger distances and generally till the small airways. Hence, the resistance at 5 Hz (R5) represents the total airway resistance. Subtracting R20 from R5 (R5 – R20) reflects resistance in the small airways. (Figure 1,2)
How to perform IOS:

The IOS machine has a pneumotach and pressure transducer connected in series, with a speaker at one end and a mouthpiece at the other. (Figure 4) The IOS instrument should be calibrated every day for volume using a 3-L syringe and for resistance using a reference resistance of 0.2 kPa·L⁻¹·s⁻¹ to ensure that the sensors are working accurately. The patient should be explained the procedure. Sitting position is preferred with legs kept uncrossed in order to reduce extra-thoracic pressure and a nose clip should be worn. The mouthpiece of the IOS should be at a comfortable height so that the neck is slightly extended. Ensure that there is a tight seal between the mouthpiece and lips to prevent air leak. The cheeks should be held firmly either by the patient with his/her hands or by an assistant who presses the cheeks firmly from behind. (Figure 5) The patient should be instructed to perform normal tidal breathing in a relaxed state. The recording should be performed for at least 30–45 seconds. During this period, around 120–150 sound impulses are pushed into the lungs from which the mean reactance and resistance values are determined at frequencies from 5 to 20 Hz. A minimum of three such tests should be performed. Care should be taken to ensure reproducible results without any artefacts. For bronchodilator reversibility assessment, a short-acting bronchodilator is administered and after 20 minutes an equal number of measurements are performed in the same manner as above.
Advantages of IOS:

The patient needs to perform simple tidal breathing maneuvers that require less effort and co-operation than spirometry, hence children less than 5 years, elderly and those with physical and cognitive limitations who cannot perform spirometry easily and can therefore perform this test easily. Moreover, it can be performed in patients on ventilators and also during sleep. IOS has much greater sensitivity to detect peripheral airways obstruction than spirometry.

<table>
<thead>
<tr>
<th>IOS Resistance (R)</th>
<th>IOS Reactance (X)</th>
<th>Spirometry</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal</td>
<td></td>
<td>R5, R20 , X5 and Fres normal</td>
</tr>
<tr>
<td></td>
<td></td>
<td>R5 &gt; 150% predicted , R20 is normal, increased X5 and Fres.</td>
</tr>
<tr>
<td>Distal Obstruction</td>
<td></td>
<td>R5,R20 &gt; 150% predicted ,Normal X5 and Fres.</td>
</tr>
<tr>
<td>Proximal Obstruction</td>
<td></td>
<td>R5, R20 normal , X5 and Fres is increased</td>
</tr>
<tr>
<td>Restrictive Lung Disease</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

References:

FENO : Measurement and Clinical Applications

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Introduction :

Fractional exhaled nitric oxide (FENO) is increasingly being used in clinical practice to guide the management of airway diseases. This investigation has emerged in a big way in the last decade due to more research on this topic worldwide. The initial machines used for FENO testing were bulkier and not available as point-of-care tests. Since the availability of portable machines, the test is increasingly being used to make treatment decisions.

Nitric oxide (NO) plays a key role in modulating type 2 inflammation and in the regulation of type 2 immune responses. It is derived endogenously from the L-arginine amino acid, and its synthesis is catalyzed by one of the three forms of the enzyme NO synthase (NOS). Two constitutive NO synthases (cNOS) are generally expressed in platelets, neuronal cells, and epithelial cells and regulate the airway function. The inducible form of the enzyme (iNOS) is expressed in macrophages, neutrophils, mesangial, endothelial and vascular smooth muscle cells. It is produced in response to airway inflammation and in host defence against infection. This expression can be induced by inflammatory cytokines, such as tumour necrosis factor α, interferon γ and IL-1β. It has also been found to be upregulated by IL-13, leading to increased levels of FENO. Nitric oxide is a messenger molecule, and its activity depends on the level of oxidative stress and its uptake by antioxidant molecules. It regulates multiple biological functions including platelet reactivity, blood flow, neurotransmission and neurological memory. At high concentrations, it works in cytotoxic defence mechanisms against tumours and pathogens. It is also a key inflammatory mediator in the respiratory tract. There is emerging evidence that highlights several roles for NO in regulating pulmonary function and pulmonary disease, as an endogenous modulator of airway function and as a pro-inflammatory and immunomodulatory mediator. In patients with bronchial asthma, airway inflammation is associated with increased symptoms and airway obstruction. Higher levels of FENO in asthma are associated with eosinophilic airway inflammation and increased expression of corticosteroid-sensitive inducible NOS. Higher levels of FENO may also be associated with exacerbations and disease severity.

Measurement of FENO :

FENO measurement is easy to perform; it is reproducible and has a high degree of acceptance by patients and can be a useful marker for asthma patients. The current NICE guidelines recommend FENO to be used the management of patients who remain symptomatic on inhaled corticosteroids. Several commercially available analyzers can perform FENO measurement. These analyzers differ in some aspects such as methods of measurements, complexity, or setup required. Stationary analyzers usually use the chemiluminescence method, while handheld devices use electrochemical principles to measure FENO levels. Regardless of the used technology, the analyzers should follow the standardized measurement procedures recommended by ATS (American Thoracic Society) and ERS (European Respiratory Society) in order to assure reliable FENO measurements. There is limited data available on whether the results of the different analyzers are comparable. It is important to know whether the instruments used to measure FENO can be used interchangeably in clinical practice. The various analyzers available to measure FENO levels include the handheld FENO analyzer Vivatmo pro (BV; Bosch Healthcare Solutions, Waiblingen, Germany), another handheld FENO analyzer NIOX VERO (CN; Circassia Pharmaceuticals plc, Oxford, United Kingdom) and the stationary Ecomedics analyzer CLD 88 (EC; Eco Medics AG, Duerten, Switzerland). A recent study has shown that for the range between 0 and 70 ppb, FENO levels measured with all three devices are statistically equivalent within predefined acceptance criteria and do not differ in a clinically relevant way.

Clinical applications :

FENO values are currently used to predict and document the response to inhaled corticosteroids, monitor adherence to inhaled corticosteroids, and as a diagnostic tool in treatment-naïve patients. The introduction of FENO testing in primary care settings can be achieved with a very low effort with respect to measurement procedures and data interpretation, while simultaneously improving the quality of care.

Various biomarkers used to identify type 2 airway inflammation include serum IgE levels, blood as well as sputum eosinophils, FENO and serum periostin levels. Increase in the sputum and bronchial epithelial eosinophils is considered the “gold standard” for identifying type 2 airway inflammation. However, the bronchial biopsy is an invasive procedure, and sputum eosinophils levels testing is not available in a large proportion of centres as it requires a lab setup. This is where the initial role of FENO testing comes. It adds an additional dimension to traditional clinical testing, with advantages including the non-invasive nature of the test, the ease of repeat measurements and its relatively simple use in patients with severe airflow obstruction, where other tests may be difficult to perform. It has been to predict...
sputum eosinophilia in patients adults with asthma, irrespective of severity, atopy and smoking status. They also correlate well with the level of inflammation and show a decrease following ICS treatment. FENO levels may not always correlate with peripheral eosinophilia as they result from different inflammatory processes. Cytokines IL-4 and IL-13 regulate IgE synthesis and increase FENO levels, while IL-5 drives the development, recruitment and activation of eosinophils. So, FENO should not be considered a surrogate marker for sputum eosinophils; however, it is a parallel marker of type 2 airway inflammation. At present, severe asthma management protocol involves the use of FENO levels and blood eosinophil counts for phenotyping and guide treatment decisions. A simultaneously increased FENO, as well as blood eosinophils, are associated with a higher chance of uncontrolled asthma.

**FENO and exacerbations:**

A higher FENO level is a predictive factor for asthma exacerbations. Multiple systematic reviews of clinical trials in asthma management have demonstrated that altering asthma therapy based on FENO levels helps in reduction of future exacerbations. A meta-analysis comparing the use of FENO to guide treatment and management based on clinical assessment demonstrated that the number of individuals with one or more asthma exacerbations was significantly lower in the FENO-guided treatment group than in the control group (odds ratio 0.60). A similar analysis in children has demonstrated that the number of children having one or more asthma exacerbations was significantly lower in the FENO-guided group than in the control group (OR 0.58). FENO is recommended for diagnosis of suspected asthma cases by the National Institute for Health and Clinical Excellence (NICE) guidelines in the UK with values >40 ppb in adults and >35 ppb in children. However, this decision depends on the clinical probability of asthma and is supplemented by additional bronchial provocation testing to determine airway hyper-responsiveness. GINA suggests ≥20 ppb FENO complemented by other features such as blood eosinophils ≥150 cells/μL and/or sputum eosinophils ≥2%, could indicate type 2 immune response. ATS also recommends FENO testing at initial diagnosis of asthma and for monitoring of airway inflammation. According to the ATS guidelines, high FENO levels are defined as more than 50 ppb. There may be country-specific cut-offs; however, ATS recommends against these due to non-standardized data.

**FENO in asthma diagnosis:**

FENO is recommended for diagnosis of suspected asthma cases by the National Institute for Health and Clinical Excellence (NICE) guidelines in the UK with values >40 ppb in adults and >35 ppb in children. However, this decision depends on the clinical probability of asthma and is supplemented by additional bronchial provocation testing to determine airway hyper-responsiveness. GINA suggests ≥20 ppb FENO complemented by other features such as blood eosinophils ≥150 cells/μL and/or sputum eosinophils ≥2%, could indicate type 2 immune response. ATS also recommends FENO testing at initial diagnosis of asthma and for monitoring of airway inflammation. According to the ATS guidelines, high FENO levels are defined as more than 50 ppb. There may be country-specific cut-offs; however, ATS recommends against these due to non-standardized data.

**FENO as a response predictor:**

A high FENO value (>50 ppb) is highly suggestive of response to inhaled corticosteroids therapy in adults. A significant interaction was found between the baseline FENO levels and treatment response in a randomized controlled trial. For every ten ppb higher FENO, the change in the Asthma Control Questionnaire (ACQ) score increased by 0.071 (p=0.044), more in the patients on inhaled steroids than placebo. Based on multiple studies, there is evidence to suggest that FENO can be a useful tool for ICS dose titration and guiding asthma management.

**FENO to assess treatment adherence:**

It can also be used to monitor adherence to inhaled steroids because persistently high levels suggest non-adherence to therapy. Among patients with difficult asthma, seven days of directly observed ICS therapy led to a significantly greater reduction in FENO levels in non-adherent patients as compared to adherent patients (52.4% versus 20.4%; p<0.003). This reduction in FENO after observed ICS therapy identified the patients who were thought to have a refractory disease but were actually not adherent to the prescribed therapy.

**Cost constraints:**

The high cost is one of the most commonly cited reasons for not adopting FENO in general clinical practice. In a cost-effectiveness study from the UK, FENO’s use for the diagnosis of asthma led to 43 GBP reduction in cost and using FENO to guide treatment led annual cost reduction of 341 GBP per patient compared to using lung function and other parameters. In the Indian context, the cost-effective analysis is not yet available. However, it appears appropriate to use FENO for diagnosis in high pre-test probability patients as it will prevent additional investigations and may do phenotyping at diagnosis as well. Follow-up FENO can be cost-effective in non-responsive patients as it will help guide treatment and may prevent additional investigations.

**Limitations to FENO testing:**

Higher FENO levels may be seen in few other conditions such as eosinophilic bronchitis, allergic rhinitis, and atopy. In upper respiratory tract infections as well as in upper airway allergy, the levels may be very high. Some patients with COPD also have high FENO levels which may be the subgroup which has asthma–COPD overlap. In a study among COPD patients, FENO levels were found to be lower in those receiving ICS therapy. Currently, GINA does not recommend the routine use of FENO in all patients with asthma for guiding management.

**Conclusions:**

Recent advances in standardization and better technology have simplified the measurement of FENO levels. It is being used as a marker of airway inflammation and is a biomarker for type 2 inflammation. It can help in the diagnosis of asthma, monitoring adherence and therapy for patients with difficult asthma.
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4. Determination of Normative Values of Fractional Nitric Oxide in Exhaled Breath (FeNO) in Healthy Indian Population - CHEST [Internet]. [cited 2021 Jan 18]. Available from: https://journal.chestnet.org/article/S0012-3692(16)56211-4/fulltext  
Arterial Blood Gas Interpretation

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Introduction:
As pulmonologists, ensuring optimum oxygen (O₂) delivery is our basic duty. That is more relevant in the present COVID era. So, analysis of Arterial Blood Gas (ABG) should start from partial pressure of arterial oxygen (PaO₂). ABG gives us a good additional information about pH, bicarbonate (HCO₃⁻), metabolic status of the body like acidosis and alkalosis.

Before analysing ABG, the validity of report should be assessed first by a formula “80 – x = 24 x PCO₂(partial pressure of carbon-di-oxide)/HCO₃⁻”, where x is the first two digits after decimal in pH value. Arterial and venous blood gas can be easily differentiated by seeing oxygen saturation of capillary blood (SpO₂). ABG analysis should be done step-wisely. (Fig. 1)

Step-wise analysis of ABG:

A. First step (Analysis of PaO₂):
Hypoxaemia is defined as PaO₂ less than 80mm of Hg. Hypoxaemia is classified into i) Mild, when PaO₂ is between 79 - 60mm of Hg, ii) Moderate, when PaO₂ is between 59 - 40mm of Hg and iii) Severe, when PaO₂ is less than 40mm of Hg. Due to peculiarity of oxygen dissociation curve, a sharp fall of oxygen saturation below PaO₂- 60mm of Hg, Respiratory failure is defined as PaO₂ less than 60mm of Hg in room air at sea level at normal atmospheric pressure.

PaO₂ depends on O₂ therapy and calculated or measured fraction of inspiratory oxygen (FiO₂). In emergency situation, it is not ethical, moral or practical to hold oxygen delivery till ABG sample is taken. In that situation, expected PaO₂ can be calculated as “PaO₂= 500 x FiO₂”.

In presence of hypoxemia, three parameters should be looked for:

1. $P_{aCO_2}$: $P_{aCO_2}$ is inversely proportional to alveolar ventilation. Raised $P_{aCO_2}$ indicates alveolar hypoventilation. Causes of hypoventilation may be in the respiratory centres, nerves, muscles, chest wall and in COPD (due to increased dead space ventilation). Whereas, decreased $P_{aCO_2}$ indicates hyperventilation and that usually occurs in type I respiratory failure. All type I respiratory failure will convert into type II respiratory failure at terminal stages.

2. Ratio between PaO₂ and FiO₂: Ratio less than 300 may indicate acute respiratory distress syndrome (ARDS) subject to fulfilment of other criteria. The ratio is also used to assess the severity of ARDS.

3. Alveolo-arterial oxygen gradient $D(PA_O₂ - PaO₂)$: $PaO₂$ is calculated with alveolar gas equation “$PAO₂ = FiO₂{PB – PH₂O (water vapor pressure)} – PaCO₂ / R (respiratory quotient)” . Normal level of $D(PAO₂ – PaO₂)$ is age divided by 3. High gradient indicates lung parenchymal involvement.

B. Second step (Analysis of pH):
Normal value of pH is 7.4±0.05. High pH indicates alkalosis and low pH indicates acidosis. Both acidosis and alkalosis may be caused by respiratory and metabolic disorders. Respiratory disorders are characterized by primary change of $P_{aCO_2}$ (increased in acidosis and decreased in alkalosis). Whereas, metabolic disorders are characterized by primary change in $HCO_3^-$ level (increased in alkalosis and decreased in acidosis). Sometimes, pH may be near normal level due to compensatory mechanism, as compensation makes pH returns towards normal but not to the normal value. In that case, 7.4 may be considered as reference value.

C. Third step (Analysis of PaCO₂):
The normal value of PaCO₂ is 40±5mm of Hg. A raised PaCO₂ indicates respiratory acidosis or compensation to metabolic alkalosis. On the other hand, low PaCO₂ indicates respiratory alkalosis or compensation to metabolic acidosis. pH level can differentiate among them. Basic understanding is that PaCO₂ changes in opposite direction to pH i.e. PaCO₂ increases with a fall in pH (respiratory acidosis) and decrease with a rise in pH (respiratory alkalosis). If PaCO₂ changes in the same direction to that of pH, respiratory pathology is not the primary responsible event. The clinical setting, PaO₂ and HCO₃⁻ level are also helpful. As for example, a low PaO₂ indicates respiratory disorder.
D. Fourth step (Analysis of $\text{HCO}_3^-$):

$\text{HCO}_3^-$ rise may be due to metabolic alkalosis or compensation to respiratory acidosis. Whereas, $\text{HCO}_3^-$ fall is due to metabolic acidosis or compensation to respiratory alkalosis.

Base excess (BE) or base deficit indicates an excess/deficit of base in the blood, respectively. They indicate the amount of a fixed acid or base that must be added to a blood sample to achieve a pH of 7.4, under standard condition. Normal range of base excess is between $-2$ to $+2$ mEq/L. More than $+2$ mEq/L indicates metabolic alkalosis and less than $-2$ mEq/L indicates metabolic acidosis. BE is calculated as $0.93 \times ([\text{HCO}_3^-] - 24.4 + 14.8 \times (\text{pH} - 7.4))$ or $0.93 \times [\text{HCO}_3^-] + 13.77 \times \text{pH} - 124.58$.

E. Fifth step (assessment of compensation):

Following points are very important for analysis of compensation.

1. Respiratory compensation is fast, as lungs are quick to respond, but the compensation is usually incomplete. Whereas, metabolic compensation is slow, as kidneys are lazy organs, but the compensation is usually complete.
2. Respiratory disordered are classified into acute with minimum compensation and chronic events with almost total compensation.
3. Compensation (secondary changes) occurs in same direction to the primary changes, but overcompensation never occurs and it indicates mixed disorder. $\text{PaCO}_2$ and $\text{HCO}_3^-$ going in different direction also indicate a mixed disorder and, in that case, predominant pathology has to be interpreted clinically.
4. For metabolic disorder the respiratory compensation can be simply calculated as: i) $\text{PaCO}_2 = (1.5 \times [\text{HCO}_3^-]) + 8 \pm 2$ in case of metabolic acidosis; and ii) $\text{HCO}_3^- + 15$ (when $\text{HCO}_3^-$ level is within 10 to 40 mmol/L) in case of metabolic alkalosis.
5. For respiratory disorders the compensation can be calculated by "1-2-3-4-rule. $\text{HCO}_3^-$ increase will be 1 and 4 mmol/L for 10 mm of Hg increase of $\text{PaCO}_2$ in acute respiratory acidosis and chronic respiratory acidosis, respectively. Similarly, $\text{HCO}_3^-$ decrease will be 2 and 3 mmol/L for 10 mm of Hg decrease of $\text{PaCO}_2$ in acute respiratory alkalosis and chronic respiratory alkalosis, respectively.
6. Less or more secondary changes than the calculated levels of compensation indicate mixed disorder.

F. Sixth step (Calculation of Anion Gap (AG)):

Anion gap (AG) is calculated as Na$^+$ — [Cl$^- + \text{HCO}_3^-$] and it indicates unmeasured anions like albumin, phosphates, sulfates and organic anions. Its normal value is 8 to 10 mmol/L. AG calculation is particularly important in metabolic acidosis. Causes of high AG acidosis include diabetic ketoacidosis, lactic acidosis, renal failure, acidosis due to toxins, etc. Whereas, causes of normal anion gap acidosis include diarrhea (bicarbonate lose), renal tubular acidosis, K$^+$ sparing diuretics, ACE inhibitor. etc.

Sometimes, AG may be the only abnormality in ABG report. As for example, a person with ketoacidosis with severe vomiting may have apparently normal ABG [PH - 7.40, $\text{PaO}_2$ - 90 mm of Hg, $\text{PaCO}_2$ - 40 mm of Hg, $\text{HCO}_3^-$ - 25 mmol/L, Na$^+$ - 135 mEq/L, K$^+$ - 3 mmol/L, Cl$^-$ - 80 mEq/L. Here high AG suggests abnormality in ABG.

It should be kept in mind that meticulous attention for BE, AG and lactate level can be helpful in identify hidden metabolic acidosis.

G. Seventh (Clinical correlation and application): Interesting cases

1. Following vivax malaria, a 30 years old lady developed acute onset respiratory distress. Her ABG showed PH - 7.49, $\text{PaO}_2$ - 73mm of Hg, $\text{PaCO}_2$ - 34mm of Hg and $\text{HCO}_3^-$ - 25.6mmol/L. ABG could be interpreted as simple respiratory alkalosis. The lady was receiving oxygen @ 5L/min with calculated $\text{FiO}_2$ - 0.4 and $\text{PaO}_2/\text{FiO}_2 = 182.5$. Her chest X-ray showed bilateral lung infiltrates, her echocardiography did not show any abnormality and she was diagnosed as ARDS.

2. A man presented with respiratory distress and ABG showing PH - 7.34, $\text{PaO}_2$ - 54mm of Hg, $\text{PaCO}_2$ - 50mm of Hg and $\text{HCO}_3^-$ - 29mmol/L. The report was naturally interpreted as respiratory acidosis (based on low PH and high $\text{PaCO}_2$), commonly found in exacerbation of COPD and that could have been managed conventionally with biphasic positive airway pressure (BiPAP). On the other hand, in asthmatics ABG usually shows respiratory alkalosis with low $\text{PaCO}_2$. A rise of $\text{PaCO}_2$, even normal level, is considered abnormal in asthmatics. Same ABG in asthmatics should be taken seriously and they may require invasive ventilation.

3. A man admitted in ICU with ABG showing respiratory acidosis [$\text{PaCO}_2$ - 70mm of Hg and $\text{HCO}_3^-$ - 32mmol/L]. The compensatory increase of $\text{HCO}_3^-$ is expected to be 3 or 12 mmol/L for acute or chronic respiratory acidosis, respectively. ABG report can be interpreted as ‘respiratory acidosis with metabolic alkalosis’ in acute clinical setting or ‘respiratory acidosis with metabolic acidosis’ in chronic clinical setting.

4. A man with acute exacerbation of COPD showing PH - 7.164, $\text{PaO}_2$ - 66mm of Hg, $\text{PaCO}_2$ - 89mm of Hg and $\text{HCO}_3^-$ - 36.2mmol/L was put on invasive ventilation. Next day his ABG showed PH - 7.61, $\text{PaO}_2$ - 86mm of Hg, $\text{PaCO}_2$ - 46mm of Hg and $\text{HCO}_3^-$ - 39.2mmol/L. In isolation the second report may be interpreted as metabolic alkalosis. But, comparing with first report the diagnosis was respiratory alkalosis (rapid rise of pH can only be explained by substantial fall of $\text{PaCO}_2$, not by small rise of $\text{HCO}_3^-$) that needed adjustment of ventilatory set up.
Conclusion:

There is always a gap between knowledge and its application. Never read ABG report in isolation. ABG reports must be interpreted on the background of clinical setting. Do not interpret ABG in isolation but on the background of previous ABG. All ABG reports must include FiO₂ or level of O₂ therapy. “Treat the patient not the ABG report”.

**Figure 1. Flow chart for analysis of ABG**

- **Look for**
  - $\text{PaCO}_2$
  - $\text{PaO}_2/\text{FiO}_2$ Ratio
  - $D(\text{PAO}_2 - \text{PaO}_2)$

- **Analysis of $\text{PaO}_2$**
  - To assess respiratory failure

- **Analysis of pH**
  - For acidosis or alkalosis

- **Analysis of $\text{PaCO}_2$**
  - For assessment of respiratory disorders

- **Analysis of $\text{HCO}_3^-$**
  - For assessment of metabolic disorders

- **Assessment of compensation**

- **Clinical correlation and application**
  - Calculation Base excess or base deficit
  - Calculation of Anion Gap

- **Confirm arterial sample**
- **Assess the validity of report**
Introduction:

Pulmonary function tests form an important objective diagnostic investigation in the diagnosis and management of respiratory disorders. During the current year, World has witnessed an unprecedented pandemic of COVID-19 due to Severe acute respiratory syndrome coronavirus-2 (SARS-CoV-2). The causative agent is highly contagious that can spread by persons infected with COVID-19 who may be symptomatic or asymptomatic. Undertaking spirometry in such a background is highly risky as the test is associated with cough and production of droplets and aerosols. The infective agent spreads through them. There is need to take all precautions to minimise the risk of infections to patients, healthcare workers and staff undertaking the test.

In this background, it is necessary to determine the need for testing. Many factors come in the picture on the decision to undertake the test or defer it. It has to be established whether the risk to the patient and the staff outweigh the risk to the patient of deferring the test. If it is concluded that the test in not that essential, then it can be delayed until the prevalence of COVID-19 has subsided.

Screening:

It is necessary to screen the patients to know whether they are infected. They should be questioned about having acute respiratory symptoms such as fever, cough, rhinorrhoea, sore throat, body ache, and loss of smell and taste. In presence of such symptoms, pulmonary function tests should not be undertaken as they are capable of transmitting disease. It should be noted that even patients who do not exhibit any symptoms, may still be infected but asymptomatic or pre-symptomatic with a capability to spread the disease to others. All these patients/persons should undergo nucleotide amplification assay tests such as RT-PCR. If the result is positive, spirometry should not be undertaken. If the test is negative, spirometry may be undertaken with all safety measures if the test is absolutely necessary.

Precautions and Performance:

The testing room should have proper ventilation and negative pressure (air flow from clean to potentially contaminated area). The exhaust should be from the room to outside, of through filters capable of capturing droplets and aerosols, such as high-efficiency particulate air (HEPA) filters. It is advisable to undertake the test at the end of the day so as to allow sufficient time for ventilation, cleaning and disinfection of the room. Such a precaution is necessary when the test is performed in clinic rooms used for multi-purpose.

While performing spirometry social distancing must be maintained. There should be 2-meter distance between the technician and the patient. The physical positioning should be made in such a way so that the technician is out of the direct plume of exhaled air or cough. Staff performing the test should wear personal protection equipment (PPE) consisting of face mask, face shield, N95 respirator, gown and gloves that limits aerosolized droplet acquisition for staff. Hands should be thoroughly washed before and after each test. Only the patient to be tested and the technician should be present in the room.

The spirometer used should have a high-efficiency in-line bacterial and viral filter (BVF). They prevent cross contamination with corona virus. It must be noted that spirometers routinely used may not be adequate to prevent cross contamination. These filters are single use and are discarded following their use by each patient. It must be remembered the filter placed on the mouthpiece may affect the accuracy of the measurement. Since there is a chance of inhaling virus particles in the room by the patient, nose clips have to be applied and inhale maximally through the filter. It is necessary they should forcefully exhale maximally through the filter. This will prevent virus in their lung gaining entry into the testing area.

It is necessary to give detailed instructions about the testing procedure. Since there is a chance of inhaling virus particles in the room by the patient, nose clips have to be applied and inhale maximally through the filter. It is necessary they should forcefully exhale maximally through the filter. This will prevent virus in their lung gaining entry into the testing area.
Lung function tests using aerosol-generating protocols should be deferred. It is necessary to restrict the pulmonary function tests and to undertake only those tests essential for immediate treatment decisions. All protective measures have to be taken for the staff and the persons being tested. The surfaces of the testing area should be wiped with cleansing agents. The risk of transmission depends on the prevalence of the virus in the community, age of the patient, severity of lung disease and presence of immunosuppression.

Impaired pulmonary function and exercise capacity remain for a long time even after recovery from COVID-19 pneumonia. A study of 110 patients discharged after suffering from COVID-19 found significant fall in diffusing capacity. Higher impairment in lung volumes was noted in severe cases. There was restrictive ventilatory defect.3

References:

DEAR COLLEAGUES,

AS MEMBERS / FELLOWS OF NATIONAL COLLEGE OF CHEST PHYSICIANS (INDIA),

If You, Your Spouse or Children have been Awarded / Felicitated / Recognized / Appreciated this year for any Outstanding Achievement(s),

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► How can We all make NCCP(I) Lung Bulletin better together?
► Is there anything You would like us to consider for our next issue?

Your personal feedback is always appreciated!

Please do write to The Editor, NCCP(I) Lung Bulletin at ncsarangdhar@rocketmail.com
SALUTES

The front runners in this Covid scenario