THE PHYSIOLOGICAL SOCIETY OF SRI LANKA

NEWSLETTER

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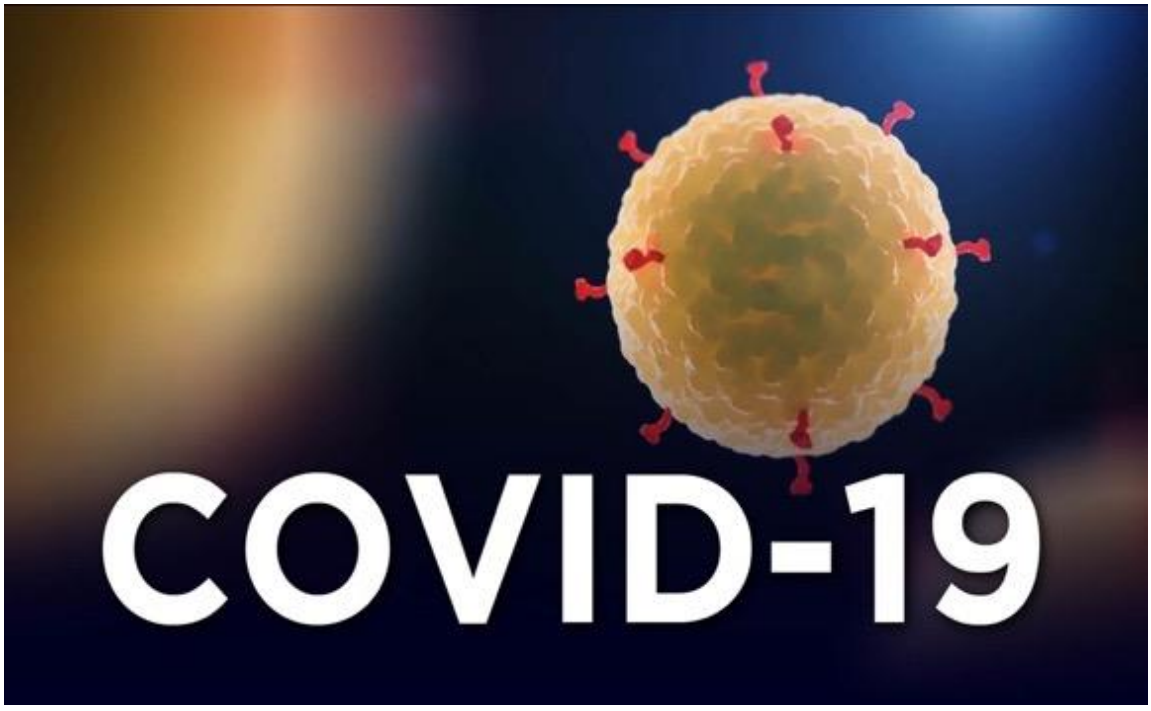
Let's do our part in the fight against



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Editorial

Dear Members,

Welcome to the second issue of the PSSL newsletter for 2019. The release of this issue was delayed due to the emergence of the COVID -19 virus in our country and the sudden unexpected disruption to day to day activities.

The main highlight of this issue is a colourful outline of the 32nd annual sessions, held this time as two components in Colombo and at the Faculty of Medicine, University of Peradeniya in November 2019, a week apart. The scientific sessions commenced with an informative pre-conference workshop on investigation of the upper gastrointestinal tract followed by the K N Seneviratne oration, held in Colombo. The scientific sessions were held in Peradeniya, where Physiologists from all over the country came together for academic and social fellowship and was extremely successful. A most thought-provoking talk on the rise of air pollution, the VB memorial oration and the ACE Koch memorial oration were well received. A well-organized scientific programme unraveled interspersed with free paper sessions, followed by the annual general meeting where the newly elected President and the new committee were elected.

The featured article for this issue is on Cardiovascular Autonomic Functions: Why test it and how to test it? A timely article as stress levels are increasing as the effects of COVID-19 takes toll on us. Stress increases tendency to develop hyperglycemia, predisposing to diabetes mellitus, the commonest known condition to cause cardiovascular autonomic neuropathy (CAN). This article describes investigation of the autonomic nervous system and its role in assessment of CAN with special emphasis on diabetic autonomic neuropathy.

COVID-19 rapidly spread around the world due to air travel. Many people who found themselves stranded overseas due to lockdown, had to return to their countries. People with chronic respiratory disease, in whom the virus is found to cause complications, find air travel problematic due to the effects of hypoxia of high altitude. This issue describes respiratory system effects at altitude and the physiological principles of assessing fitness to flying in those with a compromised respiratory system. The article is livened up by some stunning photographs of a visit of a fellow physiologist to the base camp of Mount Everest in Nepal.

As at now, almost the entire world is in a lock down against the deadly COVID-19 virus. The future is uncertain and currently many of us are struggling with many personal battles in our day to day lives. As we are all restricted in our homes, timelines for further activities of the PSSL are not possible. Now is the time for us to be creative and engage in developing online teaching resources for medical students, to be innovative in learning new techniques of teaching and most of all taking time to do recreational activities that we most often delay due to 'lack of time'.
Now that we have been presented with ample time to ponder and create and we will do so until we as a nation will be able to combat COVID-19 and resume our livelihoods as soon as possible. Until then stay safe.

Dr Lakmali Amarasiri - Editor, PSSL

President's message

I am happy to announce that the PSSL completed its 32nd year of existence in year 2019. We were able to hold the regional meeting, the Inter-Medical Faculty Physiology quiz and the annual scientific sessions for the year 2019 with a large number of participants.

The annual scientific sessions were held in the Faculty of Medicine, University of Peradeniya in November and it was dedicated to Professor Carlo Fonseka, a pioneering Physiologist in the country, who passed away a few months before the sessions. In recognition of his contribution to the discipline of physiology as a teacher and a researcher and his contribution to national development, the PSSL at its AGM, decided to hold an oration dedicated to his name from the year 2020.

I wish to thank the PSSL and the executive committee of 2019 for their unstinted support extended to me throughout the year and I wish to extend my best wishes to the new President, Professor Niranga Devanarayana and her executive committee for the year 2020 PSSL activities. I wish all our members good health and hope that all of you would stay safe in these difficult times.

Dr Indu Nanayakkara
President, PSSL

Department of Physiology
Faculty of Medicine
University of Peradeniya

The 32nd Annual Scientific Sessions of the Physiological Society of Sri Lanka was held on the 23nd and 30th November 2019

The events of the annual scientific sessions commenced with a well-attended pre-conference workshop on 'An update on investigating the gastrointestinal tract: high resolution manometry and pH impedance studies' held at Lanka Hospitals on the morning of 23rd November 2020.

The K N Seneviratne oration was held on the same evening, at the New Building Lecture Theatre, Faculty of Medicine, University of Colombo. The chief guest was the Dean of the Faculty of Medicine, Colombo, Professor Jennifer Perera. Highlights of the event included the K N Seneviratne Memorial Oration 2019 on "Lifestyle modifications: a physiological approach to combat metabolic syndrome" delivered by Professor Sudharshani Wasalathanthri of the Department of Physiology, Faculty of Medicine, Colombo and presentation of the K N Seneviratne Memorial Research Award for 2019 and KN Seneviratne Memorial award for Physiology 2019. The event was well attended by the PSSL membership and included the friends and family of the late Professor K N Seneviratne.

The procession

Lighting of the oil lamp by the chief guest, the President of the PSSL and officials of the executive committee, distinguished guests and some senior members of the PSSL committee

Dr Indu Nanayakkara delivering the presidential address

Address by the chief guest Professor Jennifer Perera

Observing a minute of silence in memory of the late Professor Carlo Fonseka, Professor Emeritus

Members of the audience

The dynamic compere Dr Nilanka Wickramasinghe

The K N Seneviratne Memorial Oration delivered by Professor Sudharshani Wasalathanthri

Presentation of the award for the K N Seneviratne Memorial Research Award for 2019 to Dr Amaranath Karunanayake by Professor Jennifer Perera

Presentation of the award for the Professor K N Seneviratne Memorial award for Physiology 2019 to Mr Minura Manchanayake by Mr Nihal Senevirtane brother of the late Professor K N Seneviratne

Professor Dinithi Fernando, Secretary of the PSSL delivering the vote of thanks

Some light moments

Fellowship and dinner

The scientific sessions of the annual sessions were held at the Faculty of Medicine, University of Peradeniya on the 30th of November.

The academic and nonacademic staff of the department of Physiology had worked tirelessly to organize a scientific programme of high standard.

The President of the PSSL, Dr Indu Nanayakkara welcomed everyone to the event and addressed the gathering. The programme commenced with an address by the chief guest Professor Shanthi Mendis, Former Senior Advisor to the World Health Organisation and Consultant Global Health, Geneva Learning Foundation, Geneva.

Thereafter followed the Valentine Basnayake Memorial Oration, delivered by Professor Anoja Fernando, Emeritus Professor, University of Ruhuna. Her topic on Medicine in Literature was delivered with flair and humor making it a memorable experience for the audience.

The plenary lecture on blood pressure; facts, spins and payoffs by Professor Shanthi Mendis was well received and two free paper sessions were presented with enthusiastic discussion.

The afternoon was livened up an interesting and informative symposium on “Toxins, Poisons and Humans” by the dynamic team from the University of Peradeniya, comprising of Professor Indika Gawarammana, Department of Clinical Medicine; Professor Thilini Rajapakse, Department of Psychiatry and Professor Kalana Maduwage, Department of Biochemistry, followed by a lively interactive discussion.

Thereafter, the recipient of the KN Seneviratne Memorial Research Award for 2018 Dr Chanika Alahakoon presented her findings, followed by the awards ceremony for the free papers.

The A C E Koch Memorial Oration 2019 on "Role of brown adipose tissue in energy homeostasis - a fat lot of good?” was delivered by Professor Sudheera Kalupahana, of the Department of Physiology, Faculty of Medicine, Peradeniya offering the audience some food for thought.

The annual general meeting where the new committee was elected concluded the day’s events.

The President of the PSSL, Dr Indu Nanayakkara delivering the welcome address

Lighting of the oil lamp

The chief guest Professor Shanthi Mendis being welcomed

The address by the chief guest

Professor Anoja Fernando Professor Emeritus delivering the Valentine Basnayake Memorial oration

The audience

The free paper session

The symposium on 'Toxins, Poisons and Humans'

Professor Shanthi Mendis delivering the plenary lecture

Recipient of the KN Seneviratne Memorial Research Award for 2018 Dr Chanika Alahakoon presenting her findings

The ACE Koch Memorial Oration being delivered by Professor Sudheera Kalupahana

Some light moments

Featured article

Cardiovascular Autonomic Functions: Why test it and how to test it?

Dr. Indu Nanayakkara (MBBS, MPhil, PhD)
Department of Physiology
Faculty of Medicine, University of Peradeniya

Cardiovascular Autonomic Neuropathy (CAN) results from damage to the autonomic nerve fibers that innervate the heart and blood vessels, resulting in abnormalities in control of heart rate and vascular dynamics (1)(2). The commonest known cause of CAN is diabetes mellitus. It is in fact a frequent and serious complication of diabetes and one of the most disabling in terms of quality of life and life expectancy (3)(4). Objective detection of CAN is essential for confirmation of clinical diagnosis and also to quantify and assess the severity of the disease. The presence of certain symptoms are suggestive of a possible cardiovascular autonomic dysfunction. The presence of features such as dizziness, intermittent visual impairment, exercise intolerance, erectile dysfunction, resting tachycardia and post-prandial hypotension are highly suggestive of the presence of CAN (5). An electrocardiogram seems to suggest autonomic dysfunction when a resting tachycardia is recorded that does not respond to moderate exercise, stress or sleep. Exercise intolerance would also be present with a blunted HR response to exercise. Prolongation of the QT interval, absence of the beat to beat variation in the heart rate (impaired heart rate variability) are two other features which are highly suggestive of CAN. Orthostatic hypotension may be characterized by symptoms like light headedness, palpitations, weakness, dizziness, faintishness, blurred vision, nausea, vomiting and syncope. Reverse dipping and non-dipping patterns in blood pressure where there is increase of blood pressure instead of a reduction is observed in the night, and silent ischeamia are also suggestive of the presence of CAN (2)(5).

Since diabetes is the commonest cause for CAN, there are several reasons why the diagnosis of CAN is necessary for clinical management of the diabetic patient? Firstly, it is necessary for staging of the cardiac autonomic dysfunction – specifically to determine whether the CAN is initial or definitive or severe. Secondly, it is necessary to stratify the degree of cardiovascular risk & the risk of other diabetic complications. There by, it is possible to know the level of risk to the patient. Thirdly, it is necessary to exclude other differential diagnosis of clinical manifestations and to embark on respective treatment. For example, adrenal insufficiency, anaemia, hyperthyroidism may produce symptoms of orthostatic hypotension. Finally, it is also necessary to adapt a goal of HbA1c in patient management on which both the patient and the attending physician can agree upon. For example in advanced CAN, the glycaemic control has to be less stringent in order to avoid asymptomatic hypoglyceamia.

Diagnostic tests for CAN include the standards cardiovascular autonomic reflex tests (CARTs) (5) and the heart rate variability (HRV) indices (6). Resting heart rate and the QT interval could also be used in the absence of the CARTs and HRV (5).

In fact, the gold standard tests for the diagnosis of CARTs are sensitive and simple to perform. The CARTs which could be performed easily are the beat-to-beat heart rate variation during deep breathing, heart rate response to standing and the heart rate response to Valsalva maneuver, systolic blood pressure response to standing and diastolic blood pressure response to isometric exercise. The normal, borderline and abnormal values for each of these tests are established (7).

The Deep breathing test (DBT), records the variation in RR interval and hear rate with respiration. Accuracy of this test depends on slow, smooth, deep breathing and therefore, should be performed at a rate of 6 breathing cycles per minute. Over 120 beats are analyzed in order to calculate the E:I ratio (ratio between longest RR interval and shortest R-R interval). Postural Index (PI) is calculated using RR interval after standing up from recumbent position (Lying to standing test) and the PI is equal to the ratio between longest RR between 20-40 beats and the shortest RR between 5-25 beats. Valsalva ratio is calculated by performing the Valsalva maneuver with an intra-thoracic pressure of 40 mmHg held for 15 seconds and then determining the ratio between the longest RR in Phase IV and shortest RR in phase II of the maneuver. Handgrip test determines the diastolic blood pressure response to isometric exercise while the orthostatic test determines the systolic blood pressure response to standing from a supine posture. A rise of diastolic blood pressure of more than 16 mmHg is considered normal for the Handgrip test. A definite orthostatic hypotension is considered to be present if the drop in systolic blood pressure is more than 30 mmHg on standing up. The normal response for standing from a supine posture is a transient fall of systolic blood pressure below 10 mmHg and a fall between 11 to 29 mmHg is considered borderline. Some of these tests such as the deep breathing test has cut-off limits which are age related since the variation in heart rate reduces with advancing age. Interpretation of CARTs results should be done with caution. No single test would suffice. Ideally all 4 tests have to be used in arriving at a diagnosis. The degree of cardiovascular autonomic dysfunction could be staged according to test results. If one test is abnormal or two tests are borderline, the autonomic dysfunction is considered as being borderline while abnormal results of two tests would indicate definite autonomic dysfunction. The presence of orthostatic hypotension indicates severe autonomic dysfunction. Heart rate tests have a high sensitivity and specificity. On the contrary, the orthostatic test has a low sensitivity but high specificity.

Heart rate variability is defined as the oscillation of RR intervals between each heart beat because of the autonomic nervous activity on the SA node. It has been consistently confirmed that reduction in the variability reflects, suppression of the vagal modulation & sympathetic dominance. This in turn will cause higher mortality & arrhythmias. Heart rate variability measurements are quantified by analysis of either the spontaneous variations of RR intervals (commonly known as HRV)(8) or experimentally induced variations of RR (CARTs). This has become relatively easy because of the modern digital data aquisition systems which are able to analyze the ECG recordings with long durations. Spontaneous variation in HRV could be analyzed in the time domain and frequency domain. In time domain analysis, the fluctuation of RR between each heart beat around the average value over a period of time is obtained by statistical analysis of RR intervals. In frequency domain analysis the magnitude of fluctuation of RR in a predetermined range of frequency is obtained by spectral analysis using mathematical algorithms. In fact, HRV testing is very useful in early detection of cardiac autonomic neuropathy (9).

Screening for CAN should be performed in all patients with Type II diabetes at diagnosis and patients with Type I diabetes after 5 years of disease - especially those with poor glycaemic control (HbA1C>7g/dl), those with one major cardiovascular disease risk factor and those with other chronic complications of diabetes. Screening should be performed in asymptomatic patients for pre-operative risk assessment before major surgery and also in patients who are unaware of hypoglycaemia having features associated with CAN. The testing is also done to exclude other drug effects & co-morbidities that mimic CAN. It is also recommended to be repeated every year in diabetics to assess progression especially when the patients have poor glycemic control, microangiopathic complications and high cardiovascular risk (5).

All cardiovascular autonomic reflex tests are safe and have a high value to risk ratio. Valsalva test may theoretically cause intra-ocular hemorrhage, but no such events have been documented in diabetics. However, Valsalva maneuver may be avoided in conditions such as severe uncontrolled hypertension and proliferative retinopathy. CARTs should be avoided in morbidly obese patients (can interfere with DBT), in hyperglycaemia or hypoglycaemia, at least 2 hours until after insulin is given. Caution should be exercised in interpreting results in chronic obstructive pulmonary disease & respiratory failure, heart disease particularly failure and in chronic kidney disease.

Cardiovascular autonomic neuropathy is a common but often under-diagnosed complication of diabetes which is strongly associated with an increased rate of cardiovascular morbidity and mortality. As the development and progression of cardiovascular denervation can be slowed down and is partly reversible in the early disease stages, it is recommended to perform screening for that complication among diabetic patients.

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Am I fit to fly?

The respiratory effects of high altitude exposure and the basis of assessment of fitness to fly in patients with chronic respiratory disease

Air travel is common, and as a result many people in the older category and those with disease are seen to use air travel increasingly nowadays. Air travel exposes an individual to a low oxygen and high pressure environment, with the most significant effect being a reduction in the partial pressure of oxygen (PO_2). Normal subjects and the majority of respiratory patients can tolerate this reduction in PO_2 without experiencing any respiratory distress. However, patients with chronic respiratory disease, with or without coexistent cardiac disease are at risk of developing hypoxia or worsening of pre-existing hypoxia. Hence, patients with preexisting pulmonary disease are now being subject to preflight medical screenings.

Preflight screening evaluation of patients with chronic respiratory disease include; a thorough medical history and physical examination to detect underlying comorbidities, a spirometry assessment in the absence of any contraindication, pulse oximetry / arterial blood gas at rest and a hypoxic inhalation challenge test. Certain patients with pulmonary disease should be instructed not to fly. These include patients who pose risk to others such as those with active infectious diseases such as tuberculosis or influenza, those with haemoptysis, unresolved pneumothorax, and those who require supplemental oxygen excess of 4 L/minute at sea level. In other patients with chronic respiratory disease, the likelihood to desaturate must be assessed to determine whether they are fit to fly or not. These tests expose patients to the conditions they are likely to encounter during air travel or use sea-level arterial blood gas analysis.

The first part of this article highlights the physiological adaptations of the respiratory system to high altitude and then describes the basis of assessment of likelihood to desaturate in patients with chronic respiratory disease, when cruising at high altitude.

The barometric pressure falls as altitude or vertical height above sea level increases. The percentage of oxygen in the atmosphere remains constant (20.9%), and so, as the barometric pressure decreases, the partial pressure of oxygen in inspired air (PiO_2) decreases proportionately. This condition is referred to as hypobaric hypoxia.

Acute ascent rapidly to extreme heights results in acute mountain sickness, with pulmonary and cerebral oedema in unacclimatized individuals, and can be fatal. However, if the body is gradually exposed to increasing altitude, it can adapt and survive. This process called acclimatization involves beneficial adaptive physiological changes to restore oxygen delivery towards sea-level values. However, even in some susceptible acclimatized individuals, long term exposure to hypoxaemia and tissue

hypoxia may lead to detrimental effects such as excessive production of red blood cells, hypoxic pulmonary vasoconstriction leading to pulmonary hypertension and congestive heart failure.

Reduced atmospheric PO_2 and humidification of air as it travels through the air passages leads to a further decrease in the PO_2 by the time the air reaches the alveoli. The alveolar partial pressure of oxygen (PAO_2) is predicted by the alveolar gas equation $PAO_2 = PiO_2 / (PACO_2 / R)$ and accordingly, due to presence of CO_2 in alveoli, the partial pressure of oxygen in alveoli is further reduced. This leads to subsequent reduction in arterial PO_2 and an initial reduction in oxygen delivery to the tissues, resulting in increase in alveolar ventilation. At sea-level, hypoxia where the arterial PO_2 is above 60 mmHg, does not cause an appreciable increase in alveolar ventilation, because central chemoreceptor inhibition by reduced cerebral extracellular PCO_2 cancels out the hypoxic stimulation of peripheral chemoreceptors. However, during acclimatization there is an increase in alveolar ventilation for any given partial pressure of oxygen in arterial blood. This is thought to be due to a decrease in cerebrospinal fluid bicarbonate levels and increase in hydrogen ion stimulation of central chemoreceptors. As change in arterial PCO_2 is inversely proportional to alveolar ventilation, the increase in alveolar ventilation results in increased removal of carbon dioxide and produces a respiratory alkalosis, which is metabolically compensated for by renal loss of bicarbonate ions.

The physiological response to acute hypobaric hypoxia also includes increase in cardiac output, increasing oxygen delivery to tissues and increase in haemoglobin concentration, increasing the oxygen content in blood. The increase in haemoglobin occurs as a result of initial reduction in plasma volume and over time with increase in secretion of erythropoietin.

The initial hyperventilation and respiratory alkalosis shifts the oxygen haemoglobin dissociation curve to the left, increasing affinity of haemoglobin for oxygen. Over a period of days to a week, an increase in 2,3-diphosphoglycerate shifts the curve to the right, favoring unloading of oxygen in the tissues and with time restores the curve to the sea-level position in fully acclimatized individuals. Individuals at altitude are frequently on the steep segment of the oxygen haemoglobin dissociation curve, where a small increase in arterial PO_2 leads to a significant increase in oxygen saturation.

Cruising altitudes of commercial aircraft typically range from approximately 30,000 to 40,000 feet, and most aircraft cabins are usually pressurized to increase the partial pressure of oxygen of inspired air (PiO_2) to approximately 15.1% of that at sea level, equivalent to that if an altitude of around 5,000-8,000 ft. This results in a partial pressure of inspired oxygen of 100–105 mm Hg and PaO_2 of approximately 60–70 mm Hg in healthy individuals, assuming a normal $PaCO_2$ and a respiratory quotient of 0.8

It is not possible to increase the PiO_2 to that of at sea level, as increasing the pressure beyond this limit would have adverse effects. As the aircraft ascends, the decreasing cabin air pressure results in in a temporary increase in the volume of any trapped gas within body cavities as the pressure slowly equalises with the cabin pressure. This is the reason for the “popping” sensation experienced in the ears during ascent. This may be of significance in patients with either cystic or bullous disease, where the increase in the volume of the trapped gas could compress adjacent healthy tissue affecting lung mechanics. Hence, unresolved pneumothorax, is an absolute contraindication to air travel because air trapped in the pleural space will expand at altitude.

A hypobaric environment is also associated with a decrease in the FEV_1 and FVC and increase in residual volume, functional residual capacity, and the total lung capacity in both normal individuals and those with COPD. Detection of clinically significant hyperinflation before air travel may therefore be relevant in some patients with obstructive lung disease.

The sigmoid shape of the oxygen dissociation curve (figure 1) allows healthy individuals to ascend to normal altitude of cruising in commercial airplanes without any appreciable hypoxaemia.

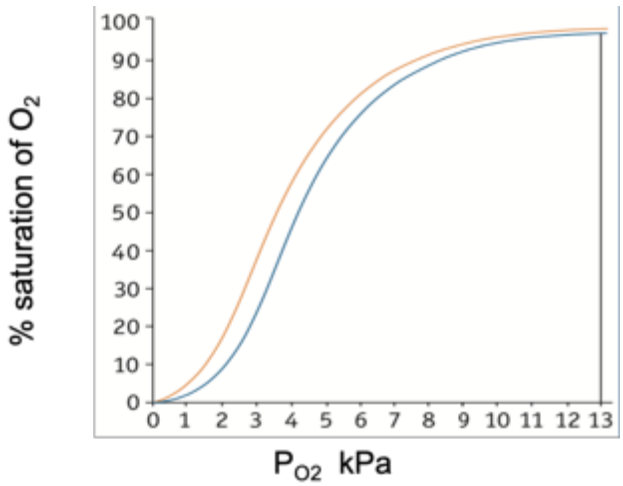

Figure 1
This diagram shows the oxygen haemoglobin dissociation curve in a healthy individual (red curve) and a patient with chronic respiratory disease (blue). *Reproduced from A.G. Robson J.A. Innes. Breathe, 2006, Volume 3, No 2, 141-146*

At sea level, in a healthy individual the PaO_2 is approximately 13 kPa or 97.5 mmHg. Around 2400m, the PaO_2 still remains around 9.7 kPa (or 72.7 mmHg), during which the oxygen saturation is maintained around 92%. In patients with respiratory disease there is a rightward shift in the oxygen dissociation curve due to chronic respiratory acidosis. This would lead to decreased affinity of haemoglobin for oxygen, increasing the possibility for the development of desaturation. At approximately 2400m the oxygen saturation is less than 90% and if the aircraft goes beyond this altitude, as the curve is steep, significant hypoxia would quickly develop.

Usually as a compensation for the reduced PO_2, there is an increase in cardiac output and minute volume. However, in patients with cardiac or respiratory limitations, compensatory mechanisms may be poor and thus result in alveolar and tissue hypoxia. In patients with chronic respiratory disease, other mechanisms of desaturation include a blunted ventilatory response to hypoxia, either due to chemoreceptor insensitivity; airway obstruction limiting an increase in ventilation or increased shunting in the lungs.

Hypoxic challenge testing, is performed under either normobaric or hypobaric conditions to assess whether a patient has the likelihood of desaturating at cabin cruising altitudes, using a decreased fraction of inspired oxygen (FiO_2) to simulate the hypoxic conditions at altitude. Hypobaric hypoxic challenge testing using hypobaric chambers or modified body plethysmograph chambers reproduces an environment most similar to that encountered during actual air travel; however, it is not widely available. Hence, assessment for hypoxia is most commonly performed using a normobaric hypoxic challenge test. The basis of the test is where the patients breathes in a hypoxic gas mixture (FiO2 to 15.1%) from a cylinder or a Douglas bag through a non-rebreathing valve or by using a 40% venturi-type oxygen mask driven by 100% N_2, for 20-30 minutes, assuming that this is sufficient time for any physiological changes to take place. Studies for longer durations have found that there was an initial

fall in PaO_2 once the flight had reached cruising altitude, and that this was maintained throughout the flight.

Normobaric hypoxic challenge test

Hypobaric hypoxic challenge test

The oxygen saturation (SpO_2) and pulse rate are measured continuously throughout the procedure and observed for desaturation. Some laboratories perform arterial blood gases before and after the procedure. After the administration of a 15% fractional concentration of inspired oxygen for 20 minutes, a PaO_2 greater than 50 mm Hg or an SpO_2 of at least 90% could suggest that in-flight oxygen is not required. However, if desaturation beyond these values is observed, oxygen is administered and the test repeated to determine the oxygen requirement of the patient during flying.
Sitting values of PaO_2 may differ from those during light exercise (such as walking within the aircraft), hence this test cannot predict whether a patient will desaturate at high altitude during light exercise.

Whether patients require supplemental oxygen and guidelines on disease specific management are available. The BTS guidelines suggest that those with a resting oxygen saturation >95% at sea level will be able to fly without the risk of developing significant hypoxia, those with an oxygen saturation <92% should not fly without supplemental oxygen, and patients already on long term oxygen therapy should have their usual flow rate increased by 2 litres per minute for the duration of the flight to compensate for the reduction in PO_2.

Dr Lakmali Amarasiri
Senior Lecturer
Department of Physiology, Faculty of Medicine, University of Colombo

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The inspiration for the above article came from the following stunning photographs shared by Professor Tharaka Dassanayake, taken during a visit to the Annapurna base camp of Mount Everest with an altitude of approximately 4000m.

Photo memories of a visit to a base camp of Mount Everest

The summit of Mount Everest is the highest point above sea level on the earth's surface, at approximately 8,850 metres altitude, and has a partial pressure of oxygen about one-third of the sea-level value.

Figure 1: Life in thin air: Nestled against a hillside in the Everest Region, Namche Bazaar is the capital of the Sherpa people. Notably, it is the home of Tenzing Norgay, the first man who summitted Mount Everest with Edmund Hillary. Many of his descendants continue the world's most dangerous job (November 2016).

Figure 2: Perceptual constancy: Distances are deceptive in the mountains. Although looking like a moderately sized mountain that lies few kilometres away, the Everest peak (top left) towers 5200m above 28km North East from where my wife and travel partner, Deva stands (November 2016).

Figure 3: Annapurna Sanctuary: In this winter trek, huffing and puffing for 8 days brings us to the Annapurna Base Camp at an altitude of 4130m. Even an ardent non-believer cannot help that primeval feeling that he is closer to the heaven – or is it altitude sickness? (December 2018)

Newsletter compiled and edited by Dr Lakmali Amarasiri

Editor
Physiological Society of Sri Lanka

Cover image from WHO framework convention on tobacco control
https://images.app.goo.gl/vrxyZLDpBt2zAqCV7