# INTERNATIONAL

# JOURNAL OF

# PHYSIOLOGY

IC

Covered by- Index Copernicus

Approved by MCI

# International Journal of Physiology

# International Journal of Physiology

# CONTENTS

**Volume 8, Number 1**                                                          **January-March 2020**

# A Study on the Impact of Exercise on Diabetic and Hypertensive Status of the Patients

**Arpith Mysore Ninge gowda[1] , Revathi Devi Mysore Laxmipathi[2], Dinesh. B[3]**

*[1]Post MD Tutor, [2]Professor and Head Department of Physiology, [3]Associate Professor,  Department of Cardiology, Mysore Medical College and Research Institute, Mysuru, Karnataka, India*

## Abstract

**Introduction and Aim**: Type 2 Diabetes mellitus and essential hypertension are common non communicable disorders that are frequently present together. Hypertension in the type 2 diabetic individual increases the risk and accelerates the course of cardiac disease, peripheral vascular disease, stroke, retinopathy and nephropathy. The hallmark of hypertension in type 1 and type 2 diabetics appears to be increased peripheral vascular resistance. Carbohydrate food intake has a direct effect on postprandial glucose levels in people with diabetes and it is the principal macronutrient of concern in diabetics for glycemic management[1]. In addition, an individual's food choices and energy balance have an effect on body weight, blood pressure and lipid levels directly. To study the importance of influence of exercise on diabetics and hypertensive patients attending medicine Out Patient Department, KR Hospital.

**Materials and Method:** All the patients attending the medicine OPD of KR Hospital, Mysoreor 3 months, 210 patients having both hypertension and diabetes were selected cross sectionally, including both males and females of age group 35-50 years. They were categorized as regularly doing exercise and not doing exercise group. All of patients were screened for diabetic and hypertension by checking fasting blood glucose, post prandial blood glucose and blood pressure, exercise and body mass index were also compared.

**Results**: The study found that, the status of known cases, of type 2 diabetic mellitus and hypertension, who were regularly doing exercise showed statistically significant controlled levelscompared to those who were not doing exercise.

**Conclusion:** The study found that known cases of type 2 diabetic mellitus and hypertension are benefitted by regularly doing exercise when compared with not doing exercise.

***Keywords***: Type 2 Diabetes mellitus; Hypertension; Body mass index (BMI); Exercise.

## Introduction

In India, the younger adults getting type 2 diabetes mellitus (DM) has alarmed the nation and the fear that the productive population getting the disease related complication might decrease the working productivity and in turn development of the nation. India has made an overwhelming economic progress in recent years and is the emerging market but on the other hand it is lagging behind others in healthcare outcomes. Every fifth person in India is diabetic, which made it the world's capital of Diabetics[2]. Hypertension (HTN) is a close associate of diabetes which runs parallel. It is projected that the total number of people with diabetes will rise from 171 million in 2000 to 366 million by 2030. The number of adults with hypertension is predicted to increase by 60% to a total of 1.56 billion people by 2025[3]. Hypertension and Diabetic are common non communicable disorders that share a significant overlap in underlying modifiable

**Corresponding Author:**
**Revathi Devi Mysore Laxmipathi**
Professor and Head Department of Physiology
Department of medicine. Mysore Medical College and Research Institute, Mysuru, Karnataka, India
email: drrevathidevi1@gmail.com

and non-modifiable risk factors (including ethnicity, hereditary, smoking, alcohol, dyslipidemia and lifestyle determinants) and complications[4]. In some cases, both hypertension and diabetic mellitus may be present simultaneously at the time of initial diagnosis.Dietary habits and sedentary lifestyle are the major factors for rapidly rising incidence of non-communicable disorders among developing countries. In type 2 diabetics, recently, elevated HbA1c level has also been considered as one of the leading risk factors for developing microvascular and macrovascular complications. Improvement in the elevated HbA1c level can be achieved through diet management. Thus, the patients could be prevented from developing the diabetes complications. Awareness about diabetes complications and consequent improvement in dietary knowledge, attitude, and practices lead to better control of the disease[5]. Over the past few decades, a lot of effective drugs have been developed for the treatment of hypertension and diabetes mellitus. Hence proper education regarding the disorder, drug dosage and timing, dietary pattern and life style modification play very important role in controlling both hypertension and diabetes mellitus. The objective of this was to study the importance of influence of exercise on diabetic and hypertensive status of population.

## Materials and Method

The study is a randomized cross-sectional study, all the patients attending the medicine Out Patient Department of KR Hospital, Mysuru for 3 months. It is a cross sectional study, 210 patients having both hypertension and diabetes were selected including both males and females of age group 35-50 years. They were then divided into two groups based on exercise status like regularly doing exercise (n=110) and not doing exercise (n=100). All of them were screened for diabetes and hypertension by checking fasting blood glucose, post prandial blood glucose and blood pressure levels.

**Inclusion criteria:**

• Subjects having both Type 2 Diabetes and Hypertension, having at least one of them for 5 years or more and under treatment and compliant with treatment.

• Age group 35-50 years.

• Exercise group consisted of patients who were doing exercise 5 days in a week, 30-45 minutes/day.

• Non-exercise consisted of patients who were not doing any form of exercise.

**Exclusion criteria:**

• Cardiovascular disorders other than hypertension

• H/o of Major surgeries in the past 6 months

• H/o/smoking/alcohol intake

• H/o Reno vascular diseases.

• H/o infectious diseases.

**Statistical analysis**:R software was used for analysis of data. Chi square value and test of significance used.

## Results

Among 210 study population in the KR Hospital, 120 (57%) males and 90 (43%) females are categorized following age groups from 35-50 years (Table 1). In this study of 210 diabetic patients, 110 patients were regularly doing exercises, in that 70 (63%) had a controlled diabetic status and 40 (37%) were not under control; 100 patients were not doing exercises regularly, in that 40 (40%) had a controlled diabetic status and 60 (60%) were not under control. P value <0.001 showed statistically significant (Table 2).

In this study of 210 hypertensive patients, 110 patients were regularly doing exercises, in that 70 (63%) had a controlled hypertensive status and 40 (37%) were not under control; 100 patients were not doing exercises regularly, in that 40 (40%) had a controlled hypertensive status and 60 (60%) were not under control. P value <0.001 showed statistically significant (Table 3).

## Discussion

The present study agrees with the study done by Miller[6]showed regular exercise and dietary education will influence in the non-communicable diseases. Diet, Exercise, Weight loss intervention trial (DEW-IT) showed that exercise incorporated lifestyle interventions can result in significantly better blood pressure and diabetes control among patients taking pharmacotherapy.

The present study reports are also in accordance with the study reports by Khattab[7] dietary counselling must be provided to the patients with type 2 diabetes and hypertension at tertiary care hospital.

The present findings agree with the study done by

Wens J Vermeire[8] who assessed perspectives of type 2 diabetes patients adherence to treatment and concluded that the overall effects of lack of lifestyle measures information would include knowledge and skill deficits and thus, leading to poor glycemic control. This suggests the need for diabetes educational program to improve diet and exercise adherence.

The present result is similar to study done by Egan[9] in which need to identify the specific barriers to exercise in the individual to improve health outcomes.

The study revealed that level of diabetes and hypertension awareness among patients and general population was low. Another study conducted in India by Shah[10] reported that 63% of Type 2 Diabetes Mellitus patients did not had the insight of these diseases and the majority were also unaware about its complications.

**Table 1: Profile of study population**

| Age (years) | Male | Female | Total (%) |
|---|---|---|---|
| 35-40 | 30 | 20 | 50 (24%) |
| 40-45 | 40 | 25 | 65 (31%) |
| 45-50 | 30 | 25 | 55 (26%) |
| >50 | 20 | 20 | 40 (19%) |
| Total | 120 | 90 | 210 (100%) |

**Table 2: Diabetic status and exercise**

| Diabetic status | Regularly doing exercise | Not doing exercise | Total (%) |
|---|---|---|---|
| Under control | 70 | 40 | 110 (52%) |
| Not under control | 40 | 60 | 100 (48%) |
| Total | 110 | 100 | 210 (100%) |

Chi square value 10.804, degree of freedom 1, $p<0.001$.

**Table 3: Hypertension and exercise**

| Hyperten-sion status | Regularly doing exercise | Not regularly doing exercise | Statistical value |
|---|---|---|---|
| Under control | 70 | 40 | 110 (52%) |
| Not under control | 40 | 60 | 100 (48%) |
| Total | 110 | 100 | 210 (100%) |

Chi square value 10.804, degree of freedom 1, $p<0.001$.

## Conclusion

The study found that known cases of type 2 diabetic mellitus and hypertension are benefitted by regularly doing exercise when compared with not doing exercise.

The various studies suggest that T2DM patients and hypertension require reinforcement of DM education including dietary management through health-care providers, health facilities, etc.) toencourage them to understand the disease management better, for more appropriate self-care and better quality of life. The overall purpose of treating T2DM is to help the patients from developing early end-organ complications which can be achieved through proper dietary management and exercise. The success of dietary management and exercise requires that the health professionals should have an orientation about the cultural beliefs, thoughts, family, and communal networks of the patients. As diabetes is a disease which continues for the lifetime, proper therapy methods with special emphasis on diet should be given by the healthcare providers in a way to control the disease, reduce the symptoms, and prevent the appearance of the complications. The patients should also have good knowledge about the disease, diet and exercise, for this purpose, the health-care providers must inform the patients to make changes in their nutritional habits and food preparations. Active and effective life style education may prevent the onset of non-communicable diseases like hypertension and diabetes and its complications.

## Limitations

Possibility of effect of confounders cannot be ruled out by this study design.

The research question can be further explored by clinical trial design or case control design for more validity.

### Recommendations

Health care instructors should be aware of the factors related to the non-adherence of lifestyle modification and should try to intervene them.

**Ethical Clearance-** Taken from institutional committee.

**Source of Funding-** Self.

**Conflict of Interest-** Nil.

## References

1. Holman, R.R., Paul, S.K., Bethel, M.A., Matthews, D.R., Neil, H.A. 10-year follow-up of intensive glucose control in Type 2 diabetes. N Engl J Med 2008; 359:1577-1589.

2. Joshi, S.R., and Parikh, R.M. India - Diabetes Capital of the World: Now Heading Towards Hypertension, JAPI, Editorial. 2007; 55: 323-324.

3. Lago, R.M., Premranjan P Singh and Richard W Nesto, Diabetes and hypertension, Nature clin. practice endo and metabolism, Editorial, Oct 2007 vol 3 no 10,667

4. Amanda, N.Long, DO and Samuel Dagogo-Jack, MD,The Comorbidities of Diabetes and Hypertension: Mechanisms and Approach to Target Organ Protection, J ClinHypertens (Greenwich). 2011; 13(4): 244-251.

5. R. Klein, B.E.K. Klein, K.E. Lee, K.J. Cruickshanks, and S.E. Moss, "The incidence of hypertension in insulin-dependent diabetes,", Archives of Internal Medicine, vol.156,no.6, pp.622-627, 1996.

6. E.R. Miller 3rd , T.P. Erlinger and D.R.Young "Results of the diet, exercise and weight loss intervention trial (DEW-IT)", Hypertension vol. 40, no 5, pp 612-618, 2002.

7. Khatttab, M., Khader, S. Y. Factors Associated with poor glycemic control among patients with Type 2 Diabetes. Journal of Diabetes and its complications 2010, 24, 84-89.

8. Wens, J., Vermeire, E. Perspectives of Type 2 Diabetes patients' Adherence to treatment: A qualitative analysis of barriers and solutions. BMC family practice, 2005: 6: 20-29.

9. Egan, M. A., Mahmood W. A., Barriers to exercise in obese patients with Type 2 Diabetes 2013. 106(7); 635-638.

10. Shah VN, Kamdar PK, Shah N Assessing the knowledge, attitudes and practice of Type 2 diabetes among patients of Saurashtra region, Gujarat. Int J Diabetes Dev Ctries 2009;29: 118-122.

# Assessment of Medical Students' Perception of Educational Environment

**Asmita Phadke[1], Jayshree Kharche[1], Savita Vaidya[2]**

*[1]Associate Professor, Department of Physiology, Bharati Vidyapeeth (Deemed to be University) Medical College, Pune, [2]Former Professor & Head, Department of Physiology, Bharati Vidyapeeth (Deemed to be University) Medical College, Pune*

## Abstract

**Background:** Educationists have established the importance of educational environment in learning. Educational environment is one of the most important factors determining the success of effectiveness of a curriculum. As there was no data available regarding educational environment at our institute; we undertook this study using Dundee Ready Educational Environment Measure (DREEM) to assess students' perception of educational environment at our institute.

**Method:** 60 students of 6th semester were included in the study. Students were administered Dundee Ready Educational Environment Measure (DREEM).

**Results:** We found the mean global DREEM score of our study population to be $121.47 \pm 13.71$ (out of 200). Problem areas identified were – teaching being too teacher centered & factual, teacher being authoritarian, students feeling lonely, bored & stressed out due to academic burden, issue of cheating, no support system for students.

**Conclusion**The present study revealed that students perceived the learning environment at our institute positively. Nevertheless, the study also revealed problematic areas of learning for which we need to put some remedial strategies in place.

***Key words:*** *educational environment, Dundee Ready Educational Environment Measure (DREEM).*

## Introduction

Medical students experience a variety of learning activities in the environs of the medical college. Educational environment is one of the most important factors determining the success of effectiveness of a curriculum[1]. The world over, medical educators are attempting to reform the educational environment so as to make it student friendly without compromising the standards and the quality of learning. Many instruments are available to measure educational environments in undergraduate professional healthcare education, each of which has its own strengths and weaknesses in terms of design, validity and reliability. One of such instruments is the Dundee Ready Educational Environment Measure (DREEM)[2]. DREEM is a 50 item inventory, consisting of 5 subscales. It was originally developed at Dundee and has been validated as a universal diagnostic inventory for assessing the quality of educational environment of different institutions[3,4,5,6,7]. So, in our study, we decided to use Dundee Ready Educational Environment Measure (DREEM) to assess students' perception of educational environment & aimed at identifying weak areas in educational environment & to institute remedial measures to rectify them.

## Material & Method

This was a cross-sectional, questionnaire-based study; data was collected in the year 2015. The medical curriculum in our college is traditional and discipline-based. The course consists of nine semesters over four and a half years followed by one year of internship. Preclinical subjects (anatomy, physiology & biochemistry) are taught in the first two semesters. Paraclinical subjects (pharmacology, pathology, microbiology, forensic medicine & toxicology) are

taught in next three semesters. During this period they also have a limited exposure to medicine, surgery, obstetrics & gynaecology, paediatrics and community medicine. Students are exposed to all clinical subjects in next four semesters.

### Study instrument

We used Dundee Ready Educational Environment Measure (DREEM). The questionnaire had 50 items & assessed five domains:

a. Students' perceptions of learning - 12 items,

b. Students' perceptions of teachers - 11 items,

c. Students' academic self-perception - 8 items,

d. Students' perceptions of atmosphere -12 items and

e. Students' social self-perception - 7 items.

Each item was to be rated on a 5-point Likert scale from 0 - 4 where

0 = strongly disagree, 1 = disagree, 2 = unsure, 3 = agree, and 4 = strongly agree. There were nine negative items (items 4, 8, 9, 17, 25, 35, 39, 48, and 50), for which correction was made by reversing the scores; thus after correction, higher scores indicated disagreement with that item. The maximal global score for the questionnaire was 200 and the global score was interpreted as follows:

0-50 = Academic environment is very poor;

51-100 = Academic environment has many problems;

101-150 = Academic environment is more positive than negative;

151-200 = Academic environment is excellent.

### Study population

Our subjects were 60 students of 6th semester studying at our institute. Students unwilling to take part in the study were excluded. After obtaining approval from institutional ethical committee, students were briefed about the purpose & process of the study. Meanings of some of the terms such as 'course organizers' and 'registrars' were explained. It was also explained that the data would be used for quality assurance as well as for research purpose and their co-operation was requested. The students were instructed not to disclose their identity (by roll number or name) to ensure truthful responses.

After obtaining written consent from the students, Dundee Ready Educational Environment Measure (DREEM)[2] was administered to the students. Each item in the tool was to be rated using a 5 point likert scale ranging from strongly agree (score-4) to strongly disagree (score-0). All questionnaires were distributed and returned the same day which allowed us to achieve a 100% response rate.

**Statistical analysis:** It was performed using Microsoft Excel and Scores of individual domains & global scores were computed. Data was analyzed by computing mean and standard deviation.

Observations & results:

The overall mean global DREEM score of our study population was 121.47±13.71 (Out of 200).

Scores of each domain were as shown in table no.1.

**Table 1: Mean & SD of participants' DREEM scores in each domain**

| Domain | Maximum score | Mean ± SD |
|---|---|---|
| Students' perception of learning | 48 | 29.27 ± 6.24 (61%) |
| Students' perception of teachers | 44 | 27.42 ± 4.47 (62%) |
| Students' academic self-perception | 32 | 20.78 ± 4.46 (65%) |
| Students' perception of Atmosphere | 48 | 27.83 ± 6.15 (58%) |
| Students' social self- perception | 28 | 16.17 ± 3.62 (57%) |
| Total | 200 | 121.47 ±13.71 (60.5 %) |

There was no statistically significant difference between the mean scores for the contributory DREEM domains, which were as follows: perception of learning, 29.27/48 (60%);

perception of teachers, 27.42/44 (62%);

academic self-perception,20.78/32 (64%);

perception of atmosphere, 27.83 /48 (58%),

and social self-perception, 16.17/28 (57%).

In our study, there were 30 items which were scored between 2.50 and 3.00. These items are aspects of the educational environment that could be enhanced.

**Table 2 : Items which were scored between 2.00 and 2.50**

| Item Number | |
|---|---|
| 9 | The teachers are authoritarian |
| 48 | The teaching is too teacher centred. |
| 17 | Cheating is a problem on this course |
| 27 | I am not able to memorize all I need. |
| 28 | I feel lonely |
| 25 | The teaching over emphasizes factual learning |
| 3 | There is no good support system for students who get stressed. |

## Discussion

Over the last four decades there has been growing interest in students' perceptions of their educational environment and the impact this may have on subsequent learning[8,9]. Educational research has demonstrated that the learning environment may influence student behaviour, academic achievement, course satisfaction and aspirations as well as sense of well-being[10,11,12]. Therefore, evaluation of the educational environment is an important consideration for any programmatic or curricular review[13].

In our study, we have found the global score to be 121.47±13.71 (out of 200). According to interpretation of the tool; this is labeled as more positive than negative. It indicates a satisfactory educational environment.

Using the same tool, other researchers have also evaluated educational environment at their institutes & have got comparable results[14,15]. Study of final year medical students in Trinidad reported global mean DREEM score of 109.9/200[1]. A larger scale study, involving students from both final and earlier undergraduate training years, showed a DREEM score of 118/200 in a Nigerian medical school, and 130/200 in a Nepalese medical school[16]. The DREEM global scores for medical schools in Srilanka and UK were reported as 108/200 &139/200 respectively[17,18]. I MBBS Students' perceptions of learning environment in an Indian medical school was found to be 119/200[19].

Based on the responses of students, one can conclude that teaching in our institute is too teacher centered & laying more stress on factual knowledge, students perceive teachers being authoritarian, students feel lonely, bored & stressed out due to academic burden, there is issue of cheating during examinations & support system for students t is non-available. (These items are scored between 2 & 2.5; indicating that these shortcomings are not of severe nature). Steps are being taken to address these shortcomings e.g. we have started with a ward-teacher system in which seven to nine students are assigned to senior teachers & regular meetings are scheduled to deal with students' problems. We are also stressing clinical & practical relevance of various topics in the syllabus. Periodic assessment of DREEM score is also planned so as to know whether the remedial strategies are being helpful to improve educational environment at our institute.

## Conclusion

Our global DREEM score is reassuring but, at the same time, our institute should strive for a score between 150 to 200 which indicates excellent educational environment. To achieve that we need to inculcate remedial strategies; some of which we have started with.

## References

1.  Bassaw B, Roff S, McAleer S, Roopnarinesing S, Lisle JD, Teelucksing S, Gopaul S: Students' perspectives on the educational environment at Faculty of Medical Sciences, Trinidad.Med Teach

2003, 25:522-526.

2.  Sue Roff, Sean McAleer, Ronald M Harden, Mona Al-Qahtani, Ashraf Uddin Ahmed, Horacio Deza, Guido Groenen & Praorn Primparyon (1997) Development and validation of the Dundee Ready Education Environment Measure (DREEM), Medical Teacher, 19:4, 295-299.

3.  De Oliveira Filho GR, Vieira JE, Schonhorst L. Psychometric properties of the Dundee Ready Educational Environment Measure (DREEM) applied to medical residents. Med Teach. 2005;27:343-347.

4.  Jakobsson U, Danielsen N, Edgren G. Psychometric evaluation of the Dundee Ready Educational Environment Measure: Swedish version. Med Teach. 2011;33:267-274.

5.  Till H. Identifying the perceived weaknesses of a new curriculum by means of the Dundee Ready Education Environment Measure (DREEM) Inventory. Med Teach. 2004;26:39-45.

6.  Genn J: AMEE Medical Education Guide No. 23 (Part 1): curriculum, environment, climate, quality and change in medical education: a unifying perspective. Med Teach 2001, 23(4):337-344.

7.  Hammond S, O'Rourke M, Kelly M, Bennett D, O'Flynn S: A psychometric appraisal of the DREEM. BMC Med Educ 2012, 12(1):2.

8.  19Miles S, Swift L, Leinster SJ: The Dundee Ready Education Environment Measure (DREEM): a review of its adoption and use. Med Teach 2012, 34(9):e620-e634.

9.  29 Genn J, Harden R: What is medical education here really like? Suggestions for action research studies of comates of medical education environments. Med Teach 1986, 8(2):111-124.

10. Audin K, Davy J, Barkham M: University Quality of Life and Learning (UNIQoLL): An approach to student well-being, satisfaction and institutional change. J Furth Higher Ed 2003, 27(4):365-382.

11. Plucker JA: The relationship between school climate conditions and aspirations conditions and student aspirations. J Educ Res 1998, 91(4):240-260.

12. Genn J: AMEE Medical Education Guide No. 23 (Part 2) Curriculum, environment, climate quality and change in medical education - a unifying perspective. Med Teach 2001, 23(5):445-454.

13. Till H: Climate studies: can students' perceptions of the ideal educational environment be of use for institutional planning and resource utilization? Med Teach 2005, 27:332-337.

14. Pimparyon P, Roff S, McAleer S, Poonchai B, Pemba S: Educational environment, student approaches to learning and academic achievement in a Thai nursing school. Med Teach 2000, 22(4):359-365.

15. Vieira, J. E., Machado, J. L. M., & Ribeiro, S. M. A. (2015). Assessment of the Environment with DREEM at a Medical School Using Active Methodologies and an Integrated Curriculum. Creative Education, 6, 1920-1935.

16. Roff S, McAleer S, Ifere OS, Bhattacharya S: A global diagnostic tool for measuring educational environment: comparing Nigeria and Nepal. Med Teach 2001, 23:378-382.

17. Jiffry MTM, McAleer , Fernandoo S, Marasinghe RB: Using theDREEM questionnaire to gather baseline information on anevolving medical school in Sri Lanka. Med Teach 2005, 27:348-352.

18. Varma R, Tiyagi E, Gupta JK: Determining the quality of educational climate across multiple undergraduate teaching sites using the DREEM inventory. BMC Med Educ 2005, 5(1):8.].

19. Abraham R, Ramnarayan K, Vinod P, Torke S. Students' perceptions of learning environment in an Indian medical school. BMC Med Educ. 2008;8:20.

# Introduction of Early Clinical Exposure(ECE) in 1st year M.B.B.S Students in the Department of Physiology

**Avjot K Miglani[1], Rajiv Arora[2]**

[1]Professor, [2]Prof & HOD, Department of Physiology. PIMS, Jalandhar

## Abstract

**Background:** Medical council of India regulations on Graduate Medical education 2012 states that "Indian Medical Graduate should possess requisite knowledge, skills, attitudes, values and responsiveness, so that he or she may function appropriately and effectively as a physician of the community while being globally relevant"[1].

Early Clinical exposure (ECE) is a teaching learning methodology, which orients medical students towards actual clinical scenarios and helps them correlate their theoretical knowledge with real life situations in the first year of medical college.

**Methodology:** The project was done in the department of physiology at PIMS Medical College, Jalandhar. The ECE program was used as a supplement to the traditional lectures in Nervous System Physiology. One hundred and fifty (150) first year MBBS students participated in early clinical exposure program conducted in the year 2017–18. A Feedback questionnaire (including both open and closed ended questions), after getting validated by the faculty were filled by the students. Students were also encouraged to give their written open comments anonymously.

Post session feedback from the students was taken by Questionnaire graded on Likert's scale.

**Results:** All the students commented that ECE helped them in their understanding of Nervous system Physiology. Most (63%) appreciated the synchronization of classroom knowledge with clinical exposures, and thought that integrated teaching helped in better understanding of practical applications of physiology (65%).

**Key words:** *Early clinical exposure, first year Medical students, Traditional teaching.*

**Curriculum Innovation Project**

## Background

Medical council of India regulations on Graduate Medical education 2012 states that "Indian Medical Graduate should possess requisite knowledge, skills, attitudes, values and responsiveness, so that he or she may function appropriately and effectively as a physician of the community while being globally relevant" [1]. This mindset needs to be developed. Need to integrate physiology with clinical scenarios is further emphasized by MCI vision 2015 document.

Early Clinical Exposure (ECE) provides a clinical context and relevance to basic sciences learning. It also facilitates early involvement in the healthcare environment that serves as motivation and reference point for students, leading to their professional growth and development.[2]

Early Clinical exposure (ECE) is a teaching learning methodology, which orients medical students towards actual clinical scenarios and helps them correlate their theoretical knowledge with real life situations in the first

**Corresponding author:**
**Dr. Rajiv Arora**
Professor and HOD (Department of Physiology)
PIMS, Jalandhar. Mobile:9815405500.
Email: drrajivarora@yahoo.com

year of medical college.[3]

The early years of medical students hardly provides them with any clinical experience. Not only it is seen that clinical relevance is an important factor for retention of Basic science knowledge, it also forms part of higher domains of learning.

**The need for Early Clinical Exposure:**

For generations, medical students have spent the preclinical years in classrooms and laboratories, memorizing body parts and dissecting specimens, eagerly anticipating the clinical years when they would see and learn from patients. This divide between preclinical and clinical years has been the norm since a century ago providing a theoretical basis for clinical medicine [4]. ECE Act as bridge between preclinical disciplines and clinical disciplines.



ECE also has potential benefits for teachers, healthcare organizations, patients, and populations [4]. Medical students undertaking preclinical studies may become exhausted while coping with the large volume of study contents [5, 6, 7] leading to decreased motivation and inadequate self-directed learning [8]. In addition, a failure to recognize the importance and relevance of the preclinical knowledge to their later clinical studies leads some students to lose their study interest and motivation [9] Motivation comprises intrinsic motivation, which refers to doing something because it is inherently interesting or enjoyable, and extrinsic motivation, which refers to doing something because it leads to a separable outcome [10]. Lack of early clinical experience has shown to demotivate students and make them prone to negative emotions when they finally enter the clinical environment [11] On the other hand early clinical exposure "helps medical students socialize to their chosen profession. It helps them acquire a wide range of subject matter and makes their learning more real and relevant. It has potential benefits for other stakeholders, notably teachers and patients. It can influence career choices [12]. Some of the advantages of early clinical exposure identified

in the literature are that ECE forms a crucial part of initiation into medicine, smoothens the transition from layperson to student physician, provides an opportunity to bring social relevance and contextualize basic science learning, provides teaching and learning of basic clinical skills, enhances student motivation and encourages the students to learn professional behavior[13]. Students perception of advantages of ECE were that it provided important validation of the student's decision to go to medical school, it was a lifeline that helped the student stay focused on their studies and provided opportunity to establish a link between the basic sciences concepts and actual patient cases [12, 13]. Faculty perception of advantages of ECE were that it provided a more integrated approach to teaching basic sciences and clinical medicine, increased excitement for learning by students, provided better comprehension of basic science knowledge [12, 13] . Importance of ECE can be explained in one line by Benjamin Franklin's words of wisdom:" Tell me and I forgot, teach me and I may remember, involve me and I learn" .

## Aims

Introduction of ECE to undergraduate first year MBBS medical students in Physiology.

**Objectives of the study**

The objectives of early clinical exposure of the first-year medical learners are to enable the learner [1]:

(a) Recognize the relevance of basic sciences in diagnosis, patient care and treatment.

(b) Provide a context that will enhance basic science learning.

This will help the students to overcome their pressures and anxieties and motivate them to develop a better insight into the medical profession. [14] It will also lead to a positive influence in the attitude of the student towards medical education which will help them to achieve social as well as professional satisfaction [15]. As students face eternally growing amount of information in the medical sciences, ECE will increase their exposure to clinical problems and thus prepare them to be up-to-date physicians throughout their careers[16].

## Methodology

The project was done in the department of physiology

at PIMS Medical College, Jalandhar.

Conduct of session:

The ECE program was used as a supplement to the traditional lectures in Nervous System Physiology. The program constituted of three case based lectures shown with the help of videos in Basal Ganglia and Cerebellum and Language(Aphasia)pathophysiology. The case based lectures were taken at the lecture hall with the help of an LCD projector.

Study design

Participants

One hundred and fifty (150) first year MBBS students participated in early clinical exposure program conducted in the year 2018-19.

**Data Collection and analysis**

Though attendance for the ECE program was compulsory as per MCI and University guidelines, participating in the program evaluation and giving feedback was strictly on voluntary basis. Informed consent was obtained from the participants. The study was approved by the Institutional Human Ethics committee.

Selection of topics was done with an intention to expose the students to a variety of learning experiences involving all the three domains of learning- cognitive, psychomotor and affective. The learning objectives for each session were chosen carefully in view of student's prior knowledge, the availability of clinical material and relevance. Based on the learning objectives, questionnaires were formulated for all the sessions.

**ECE sessions**

• **Case 1**: case of Cerebellar disease shown using video (downloaded from YouTube) demonstrating all signs and symptoms.

• **Case 2**:Basal ganglia lesions shown using video(downloaded from YouTube) where

a) Parkinsonism patient with all clinical signs and symptoms was discussed.

b) Hemiballismus and Chorea patient with clinical features demonstrated.

• **Case 3**: Patients having various types of Aphasia demonstrated using video(YouTube).

Case history, findings, investigations presented and discussed. Underlying pathophysiology discussed. ECE sessions were done in three lectures both of one-hour duration each. The students were encouraged to ask questions and clarify any doubts.

A Feedback questionnaire (including both open and closed ended questions), after getting validated by the faculty were filled by the students. Students were also encouraged to give their written open comments anonymously.

The questionnaire was meant to assess the impact of the program on the students.

Feedback: Post session feedback from the students was taken by Questionnaire graded on Likert's scale.

Student Feedback Questionnaire

Dear Participants,

**What is ECE?**

ECE is early clinical exposure. ECE means preparing first year MBBS students to meet and learn from patients.

The purpose of this activity is to get feedback on the session of Early Clinical Exposure (ECE). This will help me to evaluate the session and make further improvement. You are requested to give your honest opinion regarding the ECE session and its importance to you. The feedback questionnaire consists of students' perception and feedback on ECE sessions. **You are not required to write Name or Roll Number.**

| Sr. No | Questions | Strongly Disagree | Disagree | Neither agree nor disagree | Agree | Strongly Agree |
|---|---|---|---|---|---|---|
| 1 | ECE is more interesting method of teaching -learning compared to traditional lecture | | | | | |
| 2 | ECE has increased my attention in class | | | | | |

Cont ...

| | | Strongly Disagree | Disagree | Neither agree nor disagree | Agree | Strongly Agree |
|---|---|---|---|---|---|---|
| 3 | ECE motivated me to read more about the topic | | | | | |
| 4 | ECE helped me to understand the topic better | | | | | |
| 5 | ECE has helped me in better retention of the topic | | | | | |
| 6 | ECE helped me in correlating physiology with clinical case | | | | | |
| 7 | ECE made me understand the importance of learning Physiology | | | | | |
| 8 | ECE should be incorporated as a teaching -Learning Method along with regular lectures for other topics in physiology for undergraduates | | | | | |
| 9 | ECE should be incorporated as a teaching -Learning Method along with regular lecture in other Basic Science subjects for undergraduates | | | | | |
| Sr. No | Questions | Strongly Disagree | Disagree | Neither agree nor disagree | Agree | Strongly Agree |
| 10 | ECE should be made a part of curriculum in basic sciences for further batches of MBBS students | | | | | |
| 11 | Enlist three good points about ECE as a method of teaching -learning | | | | | |
| 12. Please give suggestions for improving ECE. | | | | | | |

Data analysis: All Likert's scale responses were categorized into either positive(strongly agree, agree) and negative responses(neutral, disagree and strongly agree).

**Duration of study: 6 months**

## Observations and Results

All the students commented that ECE helped them in their understanding of Nervous System Physiology. Most (63%) appreciated the synchronization of classroom knowledge with clinical exposures, and thought that integrated teaching helped in better understanding of practical applications of physiology (65%).One hundred two (68%) students believed ECE is more interesting method of teaching compared to traditional classroom teaching. In comparison to the other systems where only didactic lectures were involved, 65% of the students felt that ECE in Nervous System Physiology helped them to understand the concepts better.

## Observations and Results



**Fig:1: % students who found ECE more interesting method of teaching-learning compared to traditional lectures (Feedback Questionnaire 1)**
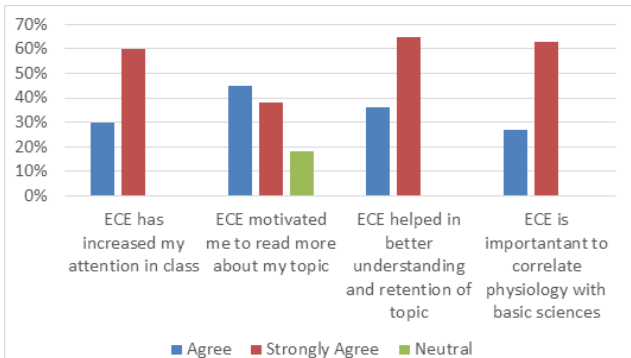


**Fig.2: Feedback Questionnaire (2-7)**
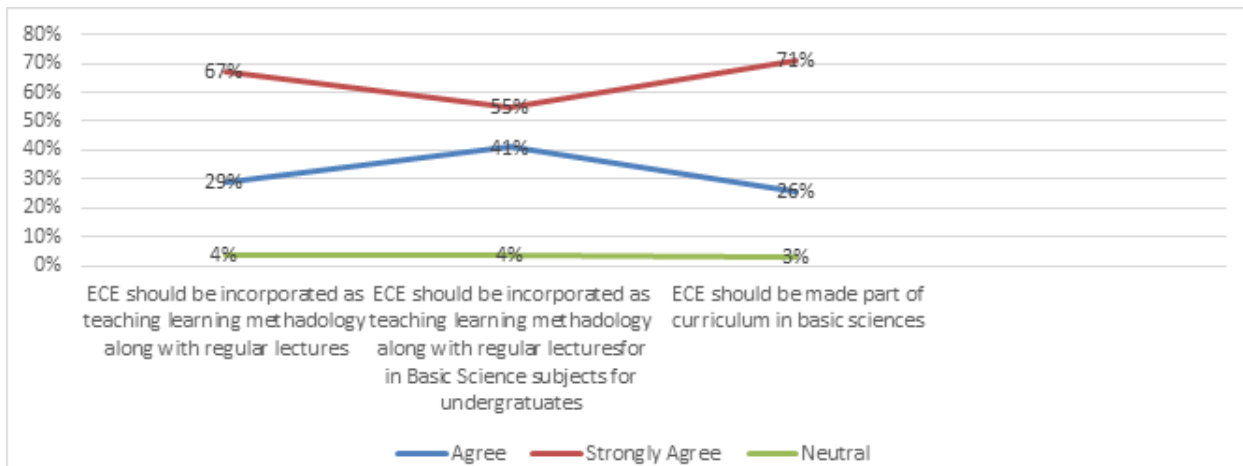
**Fig 3: Feedback    Questionnaire (8,9,10)**

**Typical student comments (Feedback Questionaaire (11)**

**Students' comments about ECE in Nervous system physiology**

| | |
|---|---|
| Understanding of Physiology | "Helped us develop a better understanding of the subject of physiology"<br><br>Helps in correlating clinical with physiology<br>Develops more interest.<br>Makes the topic more interesting.<br>Helps in retention of topic.<br>More visual understanding.<br>Keeps us motivated to work hard. |

**Typical student's suggestions for improving ECE (Feedback Questionnaire (12)**

1.ECE should be incorporated in every theory lecture.

2.ECE should be used in practical classes too for sensitization.

3. ECE should be made part of curriculum of M.B.B.S.

## Discussion

This study aimed to determine the outcomes of the project entitled "**Introduction of the Early clinical exposure(ECE) in 1st year M.B.B.S students in the department of Physiology.**" The model of traditional medical teaching includes more theoretical aspects in the first year of the course followed by exposure to clinics only in the remaining years. Regulatory bodies of the medical education have spelled out clearly that the medical education needs to be geared to train professionals capable of providing holistic care to patients with compassion[1 ]Early clinical exposure, if implemented effectively could very well initiate the changes in the medical education system in the right direction.

Most students appreciated the ECE experience as 'inspiring', 'motivating', 'interesting' and 'good way of learning'. They felt it helped them see the relevance of basic science in clinical practice and evoked self interest.These findings are in accordance with the study conducted by Chari S et al., in which the students were positive about ECE and were full of enthusiasm [17]. Increased motivation of the students with ECE was also seen in the study conducted by Baheti S N et al., [18.]

Tayade et al [19]Chari S, Gupta M, Gade S reported statistically significant difference in the knowledge, skills and attitude of first year M.B.B.S students between ECE and Non-ECE group. Systematic reviews by Dornan

T et al., and Littlewood S et al., concluded that early experience not only helped medical students learn, develop proper attitude towards their studies but also made their learning more relevant and influenced career options [20,21]. Tayade M C et al in their study noted that faculties believed that ECE consumes more manpower, infrastructure, time and requires extra efforts on their part [19].The apparent benefits of ECE include exposure to the health care system, instilling the qualities of a patient centered humanistic physician and increasing motivation for classroom learning [22]. ECE forms a crucial part in the initiation of students into medicine [23]. During a time when students often spend long hours in the classroom, it serves to remind students why they want to be physicians [22]. Most students benefit from active learning strategies over the traditional lecture format [22].

*Outcomes:* What does the study adds

Majority of the students felt that Early clinical exposure has proved for integration of basic with clinical discipline. The perception gathered from students reinforced the affirmative nature of ECE, which provided holistic learning to them.

**Limitations:**

The limitation of the study was about the time constraints in first year MBBS physiology.

## Conclusions

From present study we found early clinical exposure was better learning methodology than traditional teaching for medical students.ECE can serve as a platform for providing the preclinical year students a cosmic variety of experiences involving all the three domains of learning. It can also be adapted as the teaching strategy to introduce the various dimensions of medical profession like scientific, ethical, interpersonal, professional and social. This program if implemented effectively has the potential to be the ideal first step in the making of a holistic doctor. [23]

**Implications**

Introduction of ECE is very important for students. MCI has taken a step further by already implementing in the new curriculum CBME 2019[1].This study proved the importance of ECE as suggested by MCI. The students felt motivated to study as they felt the feeling of being "doctor'' for the first time.

## References

1.  Medical Council of India Vision 2015. Available from http://www.mciindia.org/tools/announcement/MCI_booklet.pdf.

2.  Medical Council of India. Early Clinical Exposure for the Undergraduate Medical Education Training Program, 2019: pp 1-43.

3.  Chinmay Shah. Early clinical exposure- Why and how? Journal of Education Technology in Health sciences DOI: 10.18231/2393-8005.2018.0002.

4.  Dyrbye LN, Harris I, Rohren CH. Early clinical experiences from students' perspectives: A qualitative study of narratives. Academic Medicine, 2007;82(10):979-88.

5.  Dornan T, Littlewood S, Margolis SA, Schrpbier A, Spencer J, Ypinzar V. How can experience in clinical and community settings contribute to early medical education? A BEME systematic review. Med Teach 2006, 28(1):3-183

6.  Bas O, John Yaphe J, Wolfgang Spiegel W, Wilm S, Carelli F, Metsemakers J F M. Early clinical exposure in medical curricula across Europe: An overview. European Journal of General Practice. 2009; 15: 4-1

7.  SHESHGIRI, Chowdapurkar; KOMALA, Nanjundaiah; ASHWINI, ChamanahalliAppaji. Early Clinical Exposure In Anatomy. National Journal of Integrated Research in Medicine, [S.l.], v. 8, n. 5, p. 53-56, feb. 2018. ISSN 0975-9840.

8.  Jouhari Z, Haghani F, Changiz T. Factors affecting self-regulated learning in medical students: a qualitative study. Med Educ Online 20: 28694, 2015. doi:10.3402/meo. v20.28694. 9. Dornan T, Bundy C. What can experience add to early medical education? Consensus survey. BMJ 2006, 329:1-6.

10. McLean M. Sometimes We Do Get it Right! Early ClinicalContact is a Rewarding Experience. Education for Health 2004, 17(1): 42-52. 13. O'Brien-Gonzales A, Blavo C, Barley G, Steinkohl DC, Loeser H. What Did We Learn about Early Clinical Experience? Acad Med 2001, 76 (4): S49

11. Dyrbye LN, Harris I, Rohren CH. Early clinical experiences from students' perspectives: A qualitative study of narratives. Academic Medicine, 2007;82(10):979-88.

12. Rashmi Vyas, Solomon Sathishkumar. Recent Trends In Teaching and Learning In Physiology Education Early Clinical Exposure And Integration, International Journal of Basic and Applied Physiology, 2012;1(1).

13. Michalec B. Clinical experiences during preclinical training: The function of modelled behaviour and The evidence of professionalism principles. Int J Med Edu. 2012;3:37-45

14. Saipanish R. Stress among medical students in a Thai medical school. Med Teach 25: 502–506, 2003. doi:10.1080/0142159031000136716.

15. Sreeramareddy CT, Shankar PR, Binu VS, Mukhopadhyay C, Ray B, Menezes RG. Psychological morbidity, sources of stress and coping strategies among undergraduate medical students of Nepal. BMC Med Educ 7: 26, 2007. doi:10.1186/1472-6920-7-26

16. Yusoff MSB, Abdul Rahim AF, Yaacob MJ. Prevalence and Sources of Stress among university Malaysia Medical Students. Malays J Med Sci 17: 30–37, 2010.

17. Chari S, Gupta M, Gade S. The Early Clinical Exposure Experience Motivates First Year MBBS Students: A Study. Int J Edu Sci. 2015;8(2):403–05.

18. Baheti SN, Maheshgauri D. Early Clinical Micro exposure (Ecmix) (A Path from Early Clinical Micro Exposure to Early Clinical Macro Exposure (Ecmax) Global Journal for Research Analysis. 2015;4(3):1–2.

19. Tayade MC, Bhimani N, Kulkarni NB, et al. The impact of early clinical exposure on first M.B.B.S students. International J of Healthcare and Biomedical Research 2014; 2: 176-181.

20. Dorman T, Littlewood S, Margolis S, Scherpbier A, Spencer J, Ypinazar V. How can experience in clinical and community settings contribute to early medical education? A BEME systematic review. Med Teach. 2006;28(1):3–18.

21. Littlewood S, Ypinazar V, Margolis SA, Scherpbier A, Spencer J, Dornan T. Early practical experience and the social responsiveness of clinical education: systematic review. BMJ. 2005;331(7513):387–91.

22. Dornan T, Littlewood S, Margolis SA, Schrpbier A, Spencer J, Ypinzar V. How can experience in clinical and community settings contribute to early medical education? A BEME systematic review. Med Teach 2006, 28(1):3-18.

23. Impact of a Comprehensive Early Clinical Exposure Program for Preclinical Students Ganesan Vasanth[b] et al Health Professions EducationVolume 4, Issue 2, June 2018, Pages 133-138

# Effect of Peppermint Aroma on Short Term Memory and Cognition in Healthy Volunteers

**B. Deivanayagame[1], A. V. Siva Kumar[2], K. N. Maruthy[3], SK. Kareem[2]**

[1]*Assistant Professor, Dept. of Physiology, Sri Lakshmi Narayana Institute of Medical Sciences, Puducherry,*
[2]*Faculty/Ph.D Research scholar, Department of Physiology, Narayana Medical College, Nellore,*
[3]*Professor& HOD, Department of Physiology, Narayana Medical College, Nellore*

## Abstract

**Introduction:** Aroma therapy is one of the method used to improve alertness, memory and mood enhancements. The previous study conducted on patients suffering from dementia has shown positive effect sleep and improvement in behavior. This pilot study was conducted with Peppermint aroma to see the effect on psychomotor skill and cognitive function.

**Method:** This study was carried out in the department of Physiology,Narayana Medical College, and Nellore.26 young male adults were selected for the study age between 20-30yrs. All the subjects underwent battery of psychomotor and cognitive tests. The tests were conducted in the morning hours of the day between 9 A.M. -11.00A.M.

**Results:** The preliminary analysis of the data showed marked improvement in the Visual and Auditory reaction time which was statistically significant and the p-values are $<0.01$ and $0.005$ respectively. The CFF frequency and the short term memory test did not show any change in the results and also found to be not significant statistically. The counting number test shows remarkable improvement which is significant (P-$<0.001$) which suggests enhancement in concentration with peppermint aroma exposure.

**Conclusion:** we found improvement in Auditory and Visual reaction time and Number countdown test which were statistically significant. This suggests peppermint aroma exposure even for a short time such as 5 min improves math function to a great extent.

***Key Words:*** *Peppermint aroma, Cognition, CFFF, ART, VRT, Attention span*

## Background

Human senses have significant connection to the increased ability to recall information. Sights, tastes and scents are known to bring back one's memory(1). This is known as the recall process.The sense of smell is important to our well being as it stimulates our memory, feeling of creativity and emotions(2). Putative effects of various aromas on aspects of human behavior can betraced back to ancient Greece, where the extracts of aromatic plants were used for cosmetic, religious, and medical purposes(3). Various plant derived essential oils have traditionally been used in the treatment of mental disorders. Acute exposure to a pleasant fragrance has been shown to facilitate the performance of mathematical tasks and vigilance tasks.Aroma therapy is one of the methods used to improve alertness, memory and mood enhancements(4). The previous study conducted on patients suffering from dementia has shown positive effect on sleep andimprovement in behavior(5). Peppermint is one of the mentha species and it's major components are menthol (29%) and menthone (20-30%) (6). Previous research suggests that peppermint aroma can dramatically influence motivation, task activity and alertness(7-11). The objective of this study was to determine the effect of peppermint aroma on short term

**Corresponding Author:**
**Mr. SK. Kareem,**
Faculty & Ph.D Scholar, Department of Physiology, Narayana Medical College, Nellore-524003. Contact: +919885279969, EMAIL: kareem.sb9@gmail.com

memory and cognition in healthy male participants. The measure of influence of peppermint aroma on cognition and short term memory can be evaluated by a battery of tests.

## Materials & Method

The study was carried out in the department of Physiology,Narayana Medical College, Nellore. It was a descriptive cross sectional study in which participants were recruited by non-randomized sampling technique. A total of 26 young adult males who are healthy volunteers age between 20-30yrsselected for the study. All the participants were healthy volunteers. They were inquired about past medical history and illness to rule out the current health status. The entire study protocol was verified and approved by Institutional ethics committee. The informed consent was obtained from all the participants as per the declaration of Helsinki 1975.The investigators were explained the entire protocol in detail to each participant. The participants were instructed to abstain from the caffeinated drinks and centrally acting drugs 24 hours prior to the execution of the tests. They also restricted to physical exercise which may influence the recordings of above mentioned test results. All procedures were performed after light breakfast between 9-11 am.The following tests were done before and after exposure to aroma of peppermint oil.

Computer controlled CFFF test: Critical flicker/ fusion frequency (CFF) is the transition point for an intermittent light of increasing temporal frequency, where the flickering ceases and the light is perceived as continuous.CFF stimulus consists of a red light-emitting diode (LED) with a peak wavelength of 625 nm. Before beginning of test, subject was made familiarize to the test. In this test, the subject was asked to look at the flickering red light (LED) and to appreciate the point of fusion of light. Then the frequency at which fusion of flickering light has occurred was noted downwith the help of NETRA CFF Control Device manufactured by MAVOM Labs pvt.ltd(17).

Determination of Visual Reaction Time (VRT) and Auditory Reaction Time (ART):

Reaction time is usually defined as the time required for an individual to detect the presence of a stimulus. It is a physical skill precisely related to human performance. Determination of Auditory Reaction Time (ART), Visual Reaction Time (VRT) can be done using a computerized recording device (PC 1000) wherein the subjects were asked to respond by pressing a button when he sees the red LED glowing/ when he hears a tone respectively. Then the reaction time was recorded in milliseconds with the help of Audacity.

Mathematical task: This is a part of mini mental test where the subjects were asked to subtract 3 from 999/subtracting 3 from 998 downwards up to 900. It is a verbal test, the subject being asked to subtract 3 from 999, to take 3 away from the answer obtained, and so on. The subjects were placed as much at ease as possible and encouragement was freely given, but no assistance. Each subtraction is considered as a unit with and the interpretations were made on the basis of 33 possible correct answers.

Memory recall test: In this test 15 common objects were displayed to the subject for 1 minute. After one minute the subjects were asked to recollect names of objects in next minute. Depending on number of objects recalled the scores will be given.

First round of data was collected in a separate room (before exposure to peppermint aroma). For the effect of aroma subjects were seated for 10 minutes on a chair in front of the table which was smeared with Peppermint oil (0.5ml) and the tests were performed later.Every day only one subject was recruited to avoid the effect of aroma dose overlap, and the table was cleaned every day. The tests were conducted in the morning hours of the day between 9 A.M. -11.00A.M. in a closed room without any other significant odour to interfere in the results.

Statistical analysis: The data sets were analyzed by graph pad prism & data was represented as mean and SD. Normality of data was tested using Kolmogorov-Smirnov test. A p value of $> 0.05$ indicated normal Gaussian distribution. As the data sets were showed normal distribution, paired "t" test was performed and Pearson correlation was done to find out associations.

# Results

**Table 1: Showing descriptive statistics of basal reaction times and other parameters**

|  | ART(m.sec) | VRT(m.sec) | CFFF(Hz) | Memory test | NCT |
|---|---|---|---|---|---|
| Mean ± SD | 230.7±71.57 | 261.3±64.98 | 39.35±2.24 | 10.15±1.95 | 29.04±10.20 |
| Std.Error | 14.04 | 12.74 | 0.44 | 0.38 | 2 |
| Median | 222 | 255 | 39 | 10 | 29.5 |
| 95% CI | 201.7-259.6 | 235-287.5 | 38.44-40.25 | 9.36-10.94 | 24.92-33.16 |

**Table 2: Showing the comparison of different parameters between pre and post aroma exposure**

| Parameter | Pre aroma exposure | Post aroma exposure | P value |
|---|---|---|---|
| ART | 230.7±71.57 | 196.6±49.29 | <0.01* |
| VRT | 261.3±64.98 | 221.7±70.68 | <0.01* |
| CFFF | 39.35±2.24 | 39.12±2.53 | 0.456 |
| Memory recall test | 10.15±1.95 | 10.54±2.08 | 0.2 |
| Mathematical task | 29.04±10.20 | 39.69±14.12 | <0.001* |

**ART** – Auditory Reaction Time, **VRT** – Visual Reaction Time, **CFFF** - Critical Flicker Fusion Frequency. * Significant

# Discussion

This study was done to determine the effect of exposure to peppermint aroma on in healthy male participants. The study was carried out by comparing the performance of participants before and after the exposure of peppermint aroma. The measures of cognition and attention span were obtained with the help of various cognitive tests and task based tests such as Auditory and Visual reaction time, Critical flicker fusion frequency (CFFF), Problem solving ability and Memory recall test. Similar kind of studies was also done by researchers to identify the effects of peppermint aroma on metal performance, physical ability, alertness and pain threshold. In study conducted by Clive Holmes et.al examined the effect of aroma therapy on memory enhancement in patients with dementia in which they have got significant positive results of peppermint aroma therapy(5). S J Manual et.al done a randomized control trial in 2014 in which they have concluded that peppermint scent enhances the attention of the participants(2). In 2012, Michelle Fox et.al conducted a study to assess the effect of peppermint on memory performance and concluded that peppermint mediate improvements in concentration but consumption of it does not show the effect on memory(12).The current research showed that exposure of participants to the peppermint aroma for limited duration of time enhances their cognition and psychomotor skills. The preliminary analysis of the data showed marked improvement in the Visual and Auditory reaction time which was statistically significant and the p-values are <0.01. The CFF frequency and the short term memory recall test did not show any remarkable change in the results and also found to be not significant statistically. The mathematical test showed notable improvements

which is significant (P value <0.001) which suggests enhancement in concentration with peppermint aroma exposure. Indeed, from the data presented here and evidence from other studies seems conceivable that the exposure to peppermint aroma augments cognition, alertness and concentration. The authors of the current study are planning to execute the influence of peppermint aroma on attention span and cognition by measuring the size and response of the pupil, HRV which relatively indicates the activity of Autonomic nervous system and reticular formation (13, 14, 15). We would also take up the familiarity of peppermint oil for a long time using biofeedback mechanism on psychomotor skills(16).

Limitations: In our study the sample size was limited and it addresses the population of limited geographical area and a separate study needs to describe gender variation in the results. A multicentric study with larger sample size is warranted to understand the effect of peppermint aroma by exploring different domains of cognition and psychomotor skills.

## Conclusion

The results of this study support the effectiveness of peppermint essential oil on individual's alertness, problem solving ability and focus.Our study with limited clinical evidence concluded that peppermint aroma exposure can influence the individual's cognitive ability and attention span to some extent. Therefore, it is clear that more well-designed studies are required to establish the effectiveness of peppermint aroma. And similar kind of studies needs to be done in patients to establish the pharmacological efficacy of exposure to peppermint aroma.

**Conflict of Interest:** On behalf of all authors, the corresponding author states that there is none declared any type of conflict regarding this research work.

**Funding:** There is no financial support for this work to be carried out and publication.

## References

1. Mishkin M, Appenzeller T. The anatomy of memory. Scientific American. 1987 Jun 1;256(6):80-9.

2. Manuel SJ, Syazwan M, Han CW, Fazliyana WN, Awal MB. Peppermint and Lavender Essential Oils : Are They Therapeutic Aromas for Attention and Memory ? 2014;9(1):6–11.

3. Moss M, Hewitt S, Moss L, Wesnes K. MODULATION OF COGNITIVE PERFORMANCE AND MOOD BY AROMAS OF PEPPERMINT AND. 2008;(July 2006):59–77.

4. Gaygen DE, Hedge A. Effect of Acute Exposure to a Complex Fragrance on Lexical Decision Performance. 2009;85–91.

5. Holmes C, Ballard C. Aromatherapy in dementia. 2004;10:296–300.

6. Meamarbashi A, Rajabi A. The effects of peppermint on exercise performance. 2013;1–6.

7. Raudenbush B, Grayhem R, Sears T, Wilson I. Effects of peppermint and cinnamon odor administration on simulated driving alertness, mood and workload. North American Journal of Psychology. 2009 Jul 1;11(2).

8. Herz RS. Aromatherapy facts and fictions: a scientific analysis of olfactory effects on mood, physiology and behavior. International Journal of Neuroscience. 2009 Jan 1;119(2):263-90.

9. McCombs K, Raudenbush B, Bova A, Sappington M. Effects of Peppermint Scent Administration on Cognitive Video Game Performance. North American Journal of Psychology. 2011 Dec 1;13(3).

10. Ilmberger J, Heuberger E, Mahrhofer C, Dessovic H, Kowarik D, Buchbauer G. The influence of essential oils on human attention. I: Alertness. Chemical Senses. 2001 Apr 1;26(3):239-45.

11. Morgan R. The Effects of Peppermint and Orange Aromas on Mood and Task Performance: A Research Study and Process Narration.

12. Fox M, Krueger E, Putterman L, Schroeder R. The Effect of Peppermint on Memory Performance. Journal of Advanced Student Science.. 2012;1:1-7.

13. Kumar AS, Padmavathi R, Maruthy KN, Sowjanya B, KUMAR K. An Innovative Technique to Evaluate Quantitative Pupillary Light Reflex by Dynamic Pupillometry using Infrared Videography. Journal of Clinical & Diagnostic Research. 2019 Apr 1;13(4).

14. Ramadevi P, Maruthy KN, Padmavathi R, Kumar AV, Kareem SK. Evaluation of Static Pupil Size Using Digital Web Camera through Image Analysis. International Journal of Physiology. 2019;7(3):7-11.

15. Pullaganti M, Kumar SA, Maruthy KN, Gurja JP, Chintala KK. Association of stress with heart rate

variability in different phases of the menstrual cycle. National Journal of Physiology, Pharmacy and Pharmacology. 2019;9(3):256-60.

16. Kumar CK, Kumar AS, Madhurima P, Maruthy KN, Preetham GJ. Assessment of psychomotor skills using finger pulse guided biofeedback tool in young medical students. Annals of Medical Physiology. 2018 Dec 31;2(4):36-9.

17. Kumar CK, Maruthy KN, Sasikala P, Gurja JP, Kumar AV, Kareem SK. Impact of chronic alcoholism on temporal cognition and coordination of motor activity. International Journal of Physiology. 2018;6(4):124-7.

# Impact of Obesity on Castelli's Risk Index I and II, in Young Adult Females

**Chandrashekhar D.M[1], Anandkumar[1], Jayalakshmi.M.K[2], Prashanth Babu[3]**

[1]*Assistant Professor,* [2]*Professor,* [3]*Associate Professor, Department of Physiology, Gadag Institute of Medical Sciences, Gadag, Karnataka, India*

## Abstract

**Background:** Obesity is one of today's most blatantly visible, yet most neglected, public health problems. In 2016, 39% of adults worldwide were overweight. Fueled by economic growth, urbanization, an increasingly sedentary lifestyle, and a nutritional transition to processed foods and high calorie diets over the last 30 years, many countries have witnessed the prevalence of obesity in its citizens double, and even quadruple. Obesity especially visceral obesity causes insulin resistance and is associated with dyslipidemia, impaired glucose metabolism, and hypertension all of which exacerbate atherosclerosis, and are risk factors for developing cardiovascular diseases (CVD). The primary dyslipidemia related to obesity is characterized by increased total cholesterol (TC), decreased high density lipoprotein (HDL) levels and abnormal low density lipoprotein (LDL) composition. Lipoprotein ratios are becoming increasingly popular as a way to predict atherosclerosis and CVD.

**Aims and Objectives:** The present study was undertaken to assess the impact of overweight/obesity on lipid profile parameters and lipoprotein ratios- Castelli's Risk Index I and II, in young adult females.

**Materials and Method:** The present study was conducted in KIMS, Hubli, the study and its conduct was cleared by the Ethical committee. Sixty apparently healthy young females were selected for the study. Health status and other personal data were obtained via comprehensive questionnaire. The subjects were divided into two groups based on BMI; Healthy (BMI 18.5-24.99) and Overweight (BMI > 25). Lipid profile was evaluated and lipoprotein ratios calculated. Comparison between the two groups was done using students' t-test.

**Results:** Values for Castelli's Risk Index I & II were found to be significantly higher in the overweight group compared to the control group.

**Conclusion:** Obesity leads to an unfavorable lipid pattern and raises values of both Castelli's Risk Index I & II.

*Keywords: Obesity, Castelli's Risk Index I & II, Lipid profile, Lipoprotein ratios.*

## Introduction

Noncommunicable diseases (NCDs), such as heart disease, stroke, cancer, chronic respiratory diseases and diabetes, are the leading cause of mortality in the world. This invisible epidemic is an under-appreciated cause of poverty and hinders the economic development of many countries. Common, modifiable risk factors underlie the major NCDs. They include tobacco, harmful use of alcohol, unhealthy diet, insufficient physical activity, overweight/obesity, raised blood pressure, raised blood sugar and raised cholesterol[1].

Obesity, which broadly refers to excess body fat, has become an important public health problem, and has

**Corresponding author:**
**Chandrashekhar D.M**
Assistant Professor, Department of Physiology
Gadag Institute of Medical Sciences, Gadag,
Karnataka, India, Ph: 9164500454
E mail: chandrashekardm3@gmail.com

reached to epidemic proportions. Large, high-quality longitudinal or prospective studies have confirmed that obesity is a significant risk factor for and contributor to increased morbidity and mortality, primarily from cardiovascular disease (CVD) and diabetes, but also from cancer and other acute and chronic diseases, including osteoarthritis, liver and kidney disease, sleep apnea, and depression. For the majority of these co morbid conditions, weight loss can result in a significant reduction in risk[2].

The primary dyslipidemia related to obesity is characterized by increased triglycerides, decreased HDL levels, and abnormal LDL composition[3]. The pathophysiology of the typical dyslipidemia observed in obesity is multifactorial and includes hepatic overproduction of VLDL, decreased circulating TG lipolysis and impaired peripheral free fatty acid (FFA) trapping, increased FFA fluxes from adipocytes to the liver and other tissues and the formation of small dense LDL[4].

Dyslipidemia is recognized as a prominent risk factor for cardiovascular (CV) disease[5]. Lipid abnormalities, including high levels of low-density lipoprotein cholesterol (LDL-C), elevated triglycerides and low levels of high-density lipoprotein cholesterol (HDL-C), are associated with an increased risk of CVD, thereby serving as contributors to this process[6].

There is now overwhelming experimental and clinical evidence that atherosclerosis is a chronic inflammatory disease. The atherogenic process starts with the accumulation of several plasma lipoproteins in the subendothelial space at sites of flow perturbation and endothelial dysfunction. In the intima, LDL undergoes oxidative modifications by reactive oxygen species, which promote the uptake of ox-LDL (oxidized LDL) into macrophages[7].

Low-density lipoprotein (LDL) cholesterol concentration has been the primary index of cardiovascular disease risk and the main target for therapy. However, several lipoprotein ratios have been defined in an attempt to optimize the predictive capacity of the lipid profile. Castelli's Risk Index I (TC/HDL) and II (LDL/HDL) are two ratios which have shown promise in predicting atherogenicity and CVD[8]. Castelli Risk Index I (CRI-I) has been particularly shown to reflect coronary plaques formation and the thickness of intima-media in the carotid arteries of young adults[9].

The lipoprotein ratios take into consideration both the proatherogenic and antiatherogenic factors, hence they could prove to be a better index for predicting atherogenicity and CVD.

The present study was undertaken to assess the impact of overweight/obesity on lipid profile parameters and the lipoprotein ratios; Castelli's Risk Index I and II (CRI- I and CRI- II), in young adult females.

## Materials and Method

This study was conducted in the department of Physiology, KIMS Hubli, with the assistance of Biochemistry lab, KIMS Hubli. The study and its conduct were cleared by the ethical committee KIMS Hubli.

### Inclusion criteria

1) Healthy young females with the age ranging between 18-35 years

### Exclusion criteria

1) Women on lipid lowering drugs, oral contraceptives, or any medications which may influence lipid profile.

2) Pregnant and lactating women.

### Methods of collection of data

Health status of the volunteers was assessed by comprehensive questionnaire and clinical examination. The collection of the samples, which is an invasive procedure to be performed in the study, was explained to the subjects. The volunteers were asked to do overnight fasting, and the blood sample was collected next day morning, an informed consent for the procedure was taken. They were advised to continue their normal daily diet and working routine.

**Anthropometric data**: Measurements were taken while subjects were relaxed, standing erect and had their arms at their sides and feet together.

- **Body height** was measured by wall mounted Stadiometer.

- **Body weight** was recorded by clinical weighing machine, with subjects dressed in light clothes and no shoes.

- **BMI** was calculated as per formula: Weight

(Kg)/Height (meter)$^2$.

- **Castelli's Risk Index I (CRI-I)** was calculated by the formula: CRI- I= TC/HDL.

- **Castelli's Risk Index II (CRI-II)** was calculated by the formula: CRI-II = LDL/HDL.

The subjects were divided into two groups based on BMI;

- Those with BMI in the range of 18.5-24.99, were placed in the Control group/Controls.

- Those with BMI > 25 were placed in the Study group/Subjects.

Vital parameters like pulse rate, BP were recorded. After selecting the subjects, appointment was scheduled in prior and they were requested to do an overnight fast prior to the day of the test to get fasting blood sample for lipid profile analysis. Between 7am to 10am, 2ml of venous blood was collected, in a plain bulb by venepuncture under aseptic precaution. Serum lipid profile was analyzed in Biochemistry clinical Lab, in KIMS, Hubli, with clinical chemistry Analyzer (Type Model: **XL-300 ERBA**).

## Statistical Analysis

Comparison between the two groups was done by students' t -test. All the analysis was done by using SPSS-20 software.

## Results

The present study had a total of sixty participants, who were divided into two groups based on their BMI. The mean values of serum total cholesterol (STC), serum triglycerides (STG), HDL, LDL, CRI- I and CRI- II is shown in Table 1.

**Table 1: Mean values of lipid profile parameters and Castelli's Risk Index I and II in subjects and controls.**

| Parameter | Subjects (n=30) (Mean ± SD) | Controls (n=30) (Mean ± SD) | 't' value | P value |
|---|---|---|---|---|
| Serum total Cholesterol(mg/dl) | 165.97 ± 26.4 | 141.67 ± 29.16 | 3.383 | 0.001 |
| Serum Triglycerides(mg/dl) | 128.27 ± 48.5 | 126.43 ± 48.3 | 0.147 | 0.884 |
| High density Lipoprotein(mg/dl) | 31.47 ± 7.42 | 33.4 ± 7.85 | -0.981 | 0.331 |
| Low density Lipoprotein(mg/dl) | 108.7 ± 25.86 | 82.95 ± 21.6 | 4.198 | <0.0001 |
| CRI-I (TC/HDL) | 5.43 ± 1.02 | 4.28 ± 0.5 | 5.5 | <0.0001 |
| CRI-II (LDL/HDL) | 3.59 ± 0.95 | 2.54 ± 0.54 | 5.5 | <0.0001 |
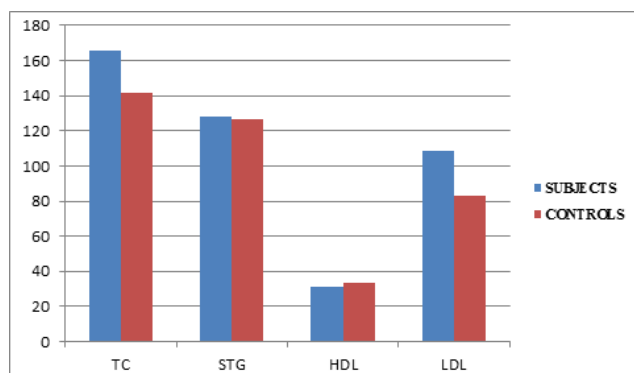
**Graph 1: Bar graph of lipid profile parameters in subjects and controls.**
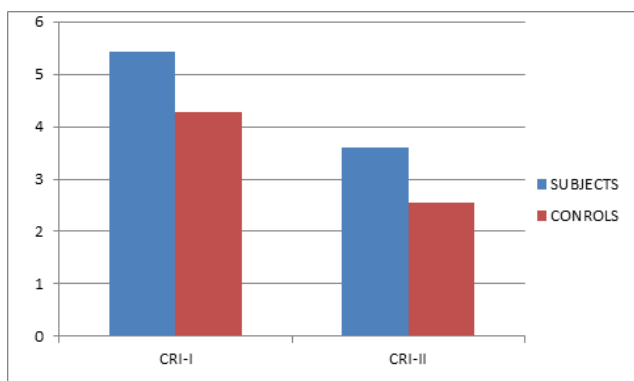


**Graph 2: Bar graph of Castelli's Risk Index 1 and 2 in subjects and controls.**

In the present study we noted that the levels of TC, STG, and LDL were higher in the study group, however the rise in the levels of TC (P value < 0.001) and LDL (P value < 0.0001) was statistically significant. Though the mean value of HDL was slightly lower in the subject group, it was not statistically significant (P value= 0.331).

The mean value for CRI-I in subjects was 5.43 ± 1.02 which was significantly higher compared to the control group value of 4.28 ± 0.5. The difference was highly significant (P value < 0.0001).

The mean value for CRI-II in subjects was 3.59 ± 0.95 which was significantly higher compared to the control group value of 2.54 ± 0.54. The difference was highly significant (P value < 0.0001).

## Discussion

In the present study, the pattern of dyslipidemia associated with the overweight/obese group was an elevation in the level of STC, STG and LDL, and a decrease in the level of HDL. The findings concur with similar studies in the past [10, 11, 12 and 13].

The striking observation in the present study was the significantly higher values of CRI-I and II in overweight/obese group, which was similar to a study done by Myat Su Bo et al., [14].

In the evaluation of dyslipidemia, triglycerides (TGs), LDL, HDL, and total cholesterol (TC) are the lipid profiles that are commonly considered, with emphasis majorly on LDL as "bad lipoprotein"[15]. However using either LDL or HDL alone to predict the risk of atherosclerosis and CVD alone is inadequate, especially in individuals with intermediate risk[16].

Studies have, however, demonstrated that in times when the conventional lipid parameters (TG, HDL-C, LDL-C, and TC) remain apparently normal, lipid ratios such as the Castelli's risk index I and II are the diagnostic alternatives that have been shown in predicting the risk of developing cardiovascular events[17].

Moreover isolated elevation in triglyceride increases the CHD risk but these effects can be balanced by cardio protective lipoprotein of HDL cholesterol[18]. There is evidence suggesting that lipid ratios, such as TC∕HDL, and TG/HDL, which take account of the proportion between the pro-atherogenic and anti-atherogenic fractions, are more effective than single measures of lipids in detecting atherosclerosis, CVD, and Insulin resistance[19].

LDL is considered to be proatherogenic. Oxidation of LDL within the arterial wall may be an important early step in atherogenesis. The uptake of oxidized LDL by macrophages is a likely explanation for the formation of macrophage foam cells in early atherosclerotic lesions. In addition, oxidized LDL has many other potentially proatherogenic properties[20].

High density lipoproteins (HDL) play an important role in reverse cholesterol transport from peripheral tissues to the liver, which is one potential mechanism by which HDL may be anti-atherogenic. In addition, HDL particles have anti-oxidant, anti-inflammatory, anti-thrombotic, and anti-apoptotic properties, which may also contribute to their ability to inhibit atherosclerosis[21].

## Conclusion

Overweight/obesity leads to an unfavorable lipid pattern characterized by high TC, LDL levels, and low HDL levels. Though High LDL levels are traditionally used to predict the risk of CVD, it's the delicate

balance between pro-atherogenic and anti-atherogenic fractions in blood that play a key role in determining atherogenicity, hence we conclude that using lipoprotein ratios like CRI-I and II may be a better way of predicting CVD risk.

In the present study overweight/obesity was associated with high CRI-I and II values, suggesting that such individuals may have a higher risk of developing CVD in the future, unless drastic steps were taken to reduce the weight. These lipoprotein ratios can be used for screening and primary prevention of CVD in overweight/obese individuals.

**Limitations:** Since it was a cross sectional study, no casual relationship can be established. The study is limited as such because it didn't consider the impact of type of diet and level of level of physical activity on lipid profile. Future studies can overcome these limitations by incorporating physical activity, dietary intake pattern, and the influences of social and environmental factors.

**Conflict of Interest:** The authors declare that there are no conflicts of interest regarding the publication of this paper.

**Ethical Clearance**: Taken from the institutional ethical committee.

## References

1) Global Health Observatory (GHO) data. https://www.who.int/gho/ncd/en/. Accessed on 07/09/2019.

2) Pi-Sunyer X. The medical risks of obesity. Postgrad Med 2009; 121(6):21–33.

3) AK Singh, S K Singh, N Singh, N Agrawal, K Gopal. Obesity and dyslipidemia. Int J Biol Med Res 2011; 2(3): 824-828.

4) Boudewijn Klop, Jan Willem F. Elte, and Manuel Castro Cabezas. Dyslipidemia in Obesity: Mechanisms and Potential Targets. Nutrients. 2013; 5(4): 1218–1240.

5) Yusuf S, Hawken S, Ounpuu S, Dans T, Avezum A, Lanas F, McQueen M, Budaj A, Pais P, Varigos J, Lisheng L, INTERHEART Study Investigators. Effect of potentially modifiable risk factors associated with myocardial infarction in 52 countries (the INTERHEART study): case-control study. Lancet 2004; 364(9438):937-52.

6) Miller M. Dyslipidemia and cardiovascular risk: the importance of early prevention. QJM 2009; 102(9):657–667.

7) Wolf D, Ley K. Immunity and Inflammation in Atherosclerosis. Circ Res 2019; 124(2):315-327.

8) Bhardwaj S, Bhattacharjee J, Bhatnagar MK, Tyagi S. Atherogenic index of plasma, Castelli risk index and atherogenic coefficient- new parameters in assessing cardiovascular risk. Int J Pharm Bio Sci 2013; 3:359–64.

9) Nair D, Carrigan TP, Curtin RJ, Popovic ZB, Kuzmiak S, Schoenhagen P, Flamm SD, Desai MY. Association of total cholesterol/ high-density lipoprotein cholesterol ratio with proximal coronary atherosclerosis detected by multislice computed tomography. Prev Cardiol 2009; 12(1):19-26.

10) Maksvytis A, Stakisaitis D. Impact of obesity on lipid profiles in middle-aged women. Medicina (Kaunas). 2004;40(6):553-7.

11) Gilles Plourde. Impact of obesity on glucose and lipid profiles in adolescents at different age groups in relation to adulthood. BMC Family Practice 2002; 3:18.

12) K Suneetha. Study of Lipid Profile in Obese and Non-obese Students in Acharya Nagarjuna University. International Journal of Pharmaceutical and Clinical Research 2018; 10(2):40-42.

13) Bassi R, Sharma S, Kaur M, Sharma A. A study of changes in lipid profile in obese and non-obese females with acne vulgaris. Natl J Physiol Pharm Pharmacol 2014; 4:125-127.

14) Myat Su Bo, Whye Lian Cheah, Soe Lwin, Tin Moe Nwe, Than Than Win, and Myint Aung, "Understanding the Relationship between Atherogenic Index of Plasma and Cardiovascular Disease Risk Factors among Staff of an University in Malaysia," Journal of Nutrition and Metabolism 2018;Article ID 7027624.

15) Third Report of the National Cholesterol Education Program (NCEP) Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults (Adult Treatment Panel III) final report. National Cholesterol Education Program (NCEP) Expert Panel on Detection,

Evaluation, and Treatment of High Blood Cholesterol in Adults (Adult Treatment Panel III). Circulation. 2002; 106(25):3143-421.

16) Superko HR, King S. Lipid management to reduce cardiovascular risk: a new strategy is required. Circulation 2008; 117(4):560-8.

17) Olamoyegun MA, Oluyombo R, Asaolu SO. Evaluation of dyslipidemia, lipid ratios, and atherogenic index as cardiovascular risk factors among semi-urban dwellers in Nigeria. Ann Afr Med 2016; 15(4):194–199.

18) I. Stensvold, A. Tverdal, P. Urdal et al., "Non-fasting serum triglyceride concentration and mortality from coronary heart disease and any cause in middle aged Norwegian women," British Medical Journal 1993;307(6915):1318–1322.

19) Du T, Yuan G, Zhang M, Zhou X, Sun X, Yu X. Clinical usefulness of lipid ratios, visceral adiposity indicators, and the triglycerides and glucose index as risk markers of insulin resistance. Cardiovasc Diabetol 2014; 13:146.

20) Young SG, Parthasarathy S. Why are low-density lipoproteins atherogenic? West J Med 1994; 160(2):153–164.

21) Feingold KR, Grunfeld C. Introduction to Lipids and Lipoproteins. [Updated 2018 Feb 2]. In: Feingold KR, Anawalt B, Boyce A, et al., editors. Endotext [Internet]. South Dartmouth (MA): MDText.com, Inc.; 2000-. Available from: https://www.ncbi.nlm.nih.gov/books/NBK305896/. Accessed on 07/09/2018.

# Physical Factors Influencing FVC in Indian Adult Males

**Chandrashekhar D.M[1], Anandkumar[1], Jayalakshmi.M.K[2], Prashanth Babu[3]**

[1]*Assistant Professor, [2]Professor, [3]AssociateProfessor, Department of Physiology,*
*Gadag Institute of Medical Sciences, Gadag, Karnataka, India*

## Abstract

**Background:** Pulmonary function tests are widely used as a valuable diagnostic tool in detecting and diagnosing various respiratory disorders like COPD, they also play an important role in monitoring therapy effectiveness and course of the disease. Normative values of pulmonary functions of healthy population are affected by different geographic, ethnic, climatic and demographic factors. The most important determining factors of Vital capacity in an individual are anthropometric factors such as age, sex, height, weight, body mass index (BMI) and body surface area (BSA)

**Aims and Objectives:** The aim of the present study was to find the correlation between Forced Vital Capacity (FVC) and anthropometric measurements like height, weight, body mass index (BMI) and body surface area (BSA) in Indian adult males.

**Materials and Method:** Fifty healthy adult males were randomly selected for the study. Ethical clearance was taken from the Institutional Ethical Committee. The physical factors namely height, weight, body mass index (BMI), and body surface area (BSA) were measured. FVC was recorded by using computerized spirometer. The correlation between the various physical factors and FVC was done using Pearson's correlation.

**Results:** Height, weight and body surface area showed significant positive correlation with FVC. However height showed the strongest correlation (r = 0.5807), followed by BSA (r = 0.531) and lastly weight (r = 0.422). However the correlation of BMI with FVC was not statistically significant.

**Conclusion:** The present study showed that body height, body surface area and body weight are important determinant of FVC in Indian adult males, with height being the most important determinant.

*Keywords: FVC, height, weight, BMI, Body Surface Area.*

## Introduction

Pulmonary function test (PFT) is the comprehensive evaluation of the respiratory system in an individual. In a clinical setting, the primary purpose of pulmonary function testing is to identify the severity of pulmonary impairment in various respiratory disorders, mainly chronic obstructive pulmonary disease (COPD).[1]

**Corresponding author.**
**Anandkumar**
Assistant Professor, Department of Physiology
Gadag Institute of Medical Sciences
Gadag, Karnataka, India, Ph: 8971282391
E mail: dranandhugar01@gmail.com

Pulmonary function tests provide objective, quantifiable measures of lung function, which are valuable in evaluating and monitoring diseases that affect both the heart and lung function. They are also useful to monitor the effects of environmental, occupational, and drug exposures, to assess risks of surgery, and to assist in routine health evaluations performed before employment.[2] During last few decades, PFTs have evolved from being used as tool for physiological study to an indispensable clinical investigation in assessing respiratory status of the patients.

The development of pulmonary functions in an individual coexists with the growth of physical parameters i.e. body height and body weight. These

physical parameters are further affected by nutrition and physical activities of growing children.[3] The development of pulmonary function and growth of physical parameters go hand in hand in children.[4]

Obese and overweight people are at a higher risk of respiratory symptoms, such as breathlessness, particularly during exercise, even if they have no obvious respiratory illness. The association between obesity and asthma leads one to question whether the mechanical effects of obesity on the respiratory system could contribute to airway dysfunction that may induce or worsen asthma.[5]

Age has historically been one of the major factors influencing lung function. Pulmonary maturity is reached at about 20–25 years of age,[6] after which lung function progressively begins to decline.[7] Several parameters such as Total Lung Capacity (TLC), FVC and Forced Expiratory volume at one second (FEV1), are affected by height, and they are proportional to body size.[8] This means that in tall individuals, who accordingly have greater lung capacity, lung volume will decrease at a greater rate compared to shorter individuals as they grow older.[9] Obesity can cause airway limitation, causing a parallel reduction in FEV1 and FVC, thus preserving the FEV1/FVC ratio.[10] Abdominal obesity is generally associated with reduced FEV1 and FVC in women and in certain age groups.[11]

Spirometry is the measurement of air moving in and out of the lungs during various respiratory maneuvers. It plays a pivotal role in the diagnosis and monitoring of patients with respiratory disease.[12] Spirometry is easier to perform and even patients with heart and lung diseases can perform it, when instructed properly by well-trained technician and or other health care provider.

The prevalence of childhood pulmonary diseases especially bronchial asthma is increasing worldwide and this necessitates the need for establishing regression equations for predicting pulmonary function in children.[13]

The present study is undertaken to find out which of the physical factors of an individual best correlates with FVC.

## Materials and Method

This is a cross sectional study, which was carried out in the Research laboratory, Department of Physiology, KVG Medical College, Sullia. The study and its conduct were cleared by the ethical committee of the same Institute. Fifty male adult non smokers were randomly selected from population of Sullia taluk. Informed consent was obtained from every subject.

**Inclusion Criteria:**

1) Male subjects in age group of 18 to 30 years.

**Exclusion Criteria:**

1) History of cardiovascular diseases.

2) History of chronic obstructive pulmonary diseases and chronic restrictive lung diseases.

3) Smokers

**Methods of collection of Data:**

• Health status was obtained by comprehensive Questionnaires.

• **Body height** was measured using wall mounted stadiometer.

• **Body Weight** was recorded with portable weighing machine

• **BMI** was calculated as per formula: Weight (Kg)/Height (meter)$^2$.

• **Body surface area** is calculated as per Dubois formula.

$$BSA = 0.007184 \times Weight^{0.425} \times Height^{0.725}$$

Computerized data logging Spiro meter was used for recording the pulmonary function tests (UNI-EM Spiromin 6.24.9 Ink)

Vital parameters like pulse rate, BP were also recorded.

## Statistical Analysis

The correlation between body height, body weight, BMI and BSA with FVC was done using Pearson's correlation coefficient. All the analysis was done by using SPSS-20 software.

## Findings

**Table 1: Mean anthropometric measurements and FVC Of subjects**

| Parameter | Mean ± SD |
|---|---|
| FVC(litres) | 3.95 ± 0.61 |
| Height(centimetres) | 171.4 ± 7.12 |
| Weight(kgs) | 66.12 ± 11.71 |
| BMI(kg/m2) | 22.42 ± 3.15 |
| Body surface area(m2) | 1.77 ± 0.16 |

**Table 2: Correlation of various anthropometric measurements with FVC**

| Parameters | 'r' value | P value |
|---|---|---|
| Height | 0.58 | 0.00001 |
| Weight | 0.422 | 0.002 |
| BMI | 0.19 | 0.186 |
| Body surface area | 0.53 | 0.00007 |
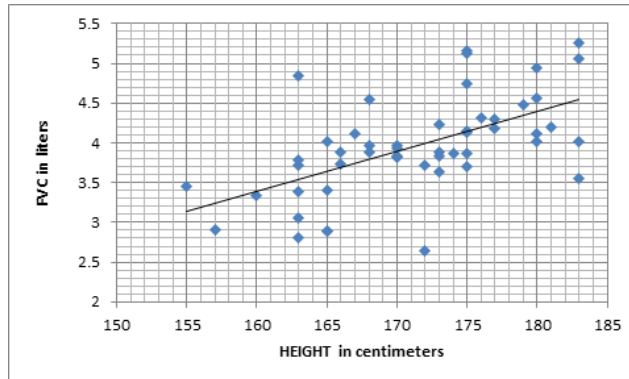


**Graph 1: Correlation of body height with FVC**
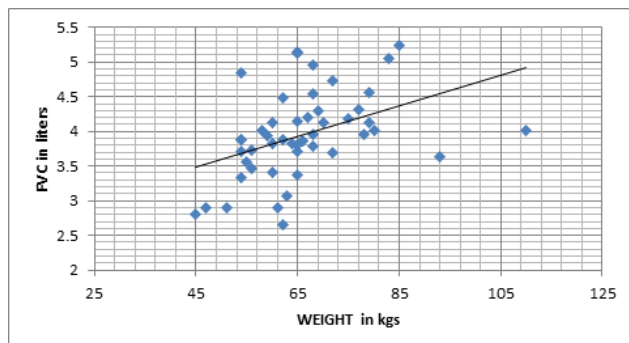


**Graph 2: Correlation of body weight with FVC**
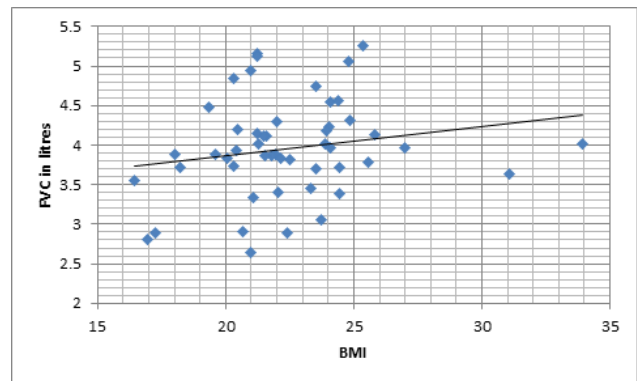


**Graph 3: Correlation of BMI with FVC**
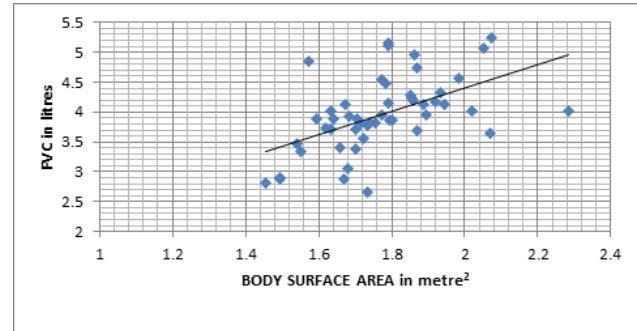


**Graph 4: Correlation of BSA with FVC**

## Discussion

The main objective of this study was to investigate the relationship between the lung function parameter FVC and selected anthropometric variables, which included body height, body weight, BMI and Body surface area.

The present study aimed to explore the influence of anthropometric measurements on FVC. In the present study three physical factors showed significant correlation with FVC, body height showed the strongest correlation (r = 0.5807), followed by BSA (r = 0.531) and lastly body weight (r = 0.422) and BMI showed no statistically significant correlation with FVC.

Results of pulmonary function are influenced by many factors: gender, stature, race, age, technical factors, weight, and others.[14] The relationship between body size and spirometry values also changes with age.[15]

Thoracic dimensions grow more in length than in width.[16] A similar study done by Carel R S et al., in young navy selectees showed that thoracic perimeter and body height are the best predictors for forced vital capacity and $FEV_1$.[17]

In our study it was observed that body height, body weight and body surface area were found to be positively correlated to FVC, the findings were similar to a study

done by Sachin Pawar et al.[18]

A similar study done by Muralidhara and Bhat, in underweight and overweight subjects, found no correlation between BMI, body fat percentage and the pulmonary functions, which concur with the present study.[19]

The present study showed that height was the most important determinant of FVC, which concurs with a similar studies.[20, 21]

Aundhakar et al. established a positive correlation between pulmonary function like FVC, Maximum voluntary ventilation, Peak expiratory flow rate and anthropometric parameters like age, height, weight, BSA, BMI etc.[22]

According to the American College of Sports Medicine, due to increased weight on the chest wall and diaphragm obesity has mechanical effects on respiration. Being obese also causes an increase in energy use as compared to a leaner person at the same workload, so in heavier people the respiratory muscles fatigue at lower intensities. These effects may contribute to the decreases in Functional Residual Capacity, Expiratory Reserve Volume and Total Lung Capacity. But in our study there is no correlation between FVC and BMI in young males.[23]

A study done by Chatterjee et al., reported that FVC, FEV1 and PEFR values increased progressively with age from 9 to 16 years and showed significantly high correlation coefficient with weight and negative correlation of FEV1 % with body surface area.[24]

Height is considered as better index of body size and body size is proportional to lung size. This might be the reason for its best correlation with PFTs .[25]

There are a few studies which however differ from the results of the present study, in some studies BSA showed less correlation than weight and in some studies height was not the primary determinant.[26, 27]

Many of recent studies have observed that pulmonary function shows an inverse relationship with various markers of obesity and fat distribution in children and adolescent.[28]

Markers of both general and visceral obesity like BMI, Waist circumference and Waist hip ratio are reported to influence pulmonary function in children

and adolescent. Some of the recent studies showed that respiratory mechanics can be affected by fat distribution pattern and central obesity, estimated by Waist circumference and Waist hip ratio independent of BMI.[29]

Our study has shown strong positive correlation of FVC with body height followed by BSA and body weight. But BMI has no correlation with FVC, better obesity markers like WC, WHR to be taken to study the effect of obesity on lung functions.

## Conclusion

In conclusion, our study showed body height, body surface area and body weight are the strong determinants of pulmonary function test FVC compared to BMI. BMI showed no statistically significant correlation with FVC in the present study. We conclude that physical factors have a strong influence in determining FVC, and hence these should be taken into account while standardizing the predictive values of PFT.

**Limitation**    The sample size was fifty; future studies can include a larger sample size for accurate predictions. The measure of obesity in the present study was BMI; future studies can include other measures of obesity, especially measures of central obesity like waist circumference and waist hip ratio for better results.

## References

1.  Pulmonary terms and symbols: a report of the ACCP-ATS Joint Committee on Pulmonary Nomenclature, Chest 1975; 67:583

2.  Robert o. Crapo. Pulmonary function testing. N. Engl J Med 1994; 331:25-30

3.  Jiwtode MT, Raikar PR. Comparison of pulmonary function tests in urban and rural children of Nagpur, Maharashtra, India. Int J Res Med Sci. 2017; 5(3):908-11.

4.  Deshpande JN, Dahat HB, Shirole CD, Pandc AH, et al. Pulmonary function and their correlation with anthropometric parameters in rural children. Indian J Pediatrics. 1983; 50:375-8.

5.  Lad UP, Vilas G. Correlation between Body Mass

Index (BMI), Body Fat Percentage and Pulmonary Functions in Underweight, Overweight and Normal Weight Adolescents. J Clinical Diagnosis Res. 2012; 6(3):350-3.

6. Sharma G, Goodwin J. Effect of aging on respiratory system physiology and immunology. Clinical Intervention and Aging. 2006; 1:253–60.

7. Lee B, Park S, Han D. Analysis of the influential factors of maximal-effort expiratory capacity of elderly women. J Phys Ther Sci. 2016;28:2924–8

8. Quanjer PH, CapderouA, Mazicioglu MM,AggarwalAN, Banik SD, Popovic S, et al. All-age relationship between arm span and height in different ethnic groups. Eur Respir J. 2014; 44:905–12.

9. Rufino R, Costa CH, Lopes AJ, Maiworm AI, Maynard K, Silva LM, et al. Spirometry reference values in the Brazilian population. Braz J Med Biol Res. 2017; 50:5700.

10. McCallister JW, Adkins EJ, O'Brien JM. Obesity and acute lung injury. Clin Chest Med. 2009; 30:495–508.

11. Ochs-Balcom HM, Grant BJ, Muti P, Sempos CT, Freudenheim JL, Trevisan M, et al. Pulmonary function and abdominal adiposity in the general population. Chest. 2006; 129:853–62.

12. Golshan M, Nematbakhsh M, Amra B and Crapo RO. Spiro metric reference values in a large Middle Eastern population. European Respir J 2003; 22(3):529-534

13. Mitchell EA. International trend in hospital admission rates for asthma. Arch Dis Child. 1985; 60:376-8.

14. Polgar C, Promadhat V. Standard values. In: Pulmonary function testing in children: techniques and standards. 1st ed. Philadelphia: WB Saunders, 1971; 87-122.

15. Polgar GJ, Weng TR. The functional development of the respiratory system. Am Rev Respir Dis 1979; 120:625-95.

16. Degroodt EG, Van Peit W, Borsboom GJJM, Quanjer PH, van Zomeren BC. Growth of lung and thorax dimensions during the pubertal growth spurt. Eur Respir J 1988; 1:102-8.

17. Carel RS, Greenstein A, Ellender E, Melamed Y, Kerem D. Factors affecting ventilatory lung function in young navy selectees. Am Rev Respir Dis 1983; 128:249-52.

18. Pawar S, Taksande AB. Prediction of pulmonary norms in healthy school boys 5-15 years - A review article. J Mahatma Gandhi Institute Med Sci. 2011; 16(1).

19. Muralidhara DV, Bhat MR. Some aspects of the pulmonary functions in underweight and overweight human subjects. Thai J Physiological Sci. 2007; 20:3-7.

20. Vijayan VK, Reetha AM, Kuppurao KV, Venkatesan P, Thilakavathy S. Pulmonary function in Normal South Indian children aged 7-19 years. Indian J Chest Dis Allied Sci. 2000; 42: 147-156.

21. Tasi MC, Jeng MJ, Chang HL, Taso PC, Yang CF, Peng YY, et al. Spiro metric reference equations for healthy children aged 6 to 11 years in Taiwan. J Chin Med Assoc 2010; 73:218.

22. C.D.Aundhakar. G.J. Kesliwal. V.S. Yajurvedi. M.S. Rewat. S.K.Ganeriwal and Sangam RN.Pulmonary function test in School children. Indian J Physiology pharmacol.1985; Jan-Mar; 29(1):14-20

23. Medarov BI, Strachan P, Cohen R. Effect Of body Mass Index on Pulmonary FunctionTests.Chest. 2005; 128:171-172.

24. Chatterjee S, Mandal A. Pulmonary function studies in healthy school boys of West Bengal. Jpn J Physiol 1991; 41:797-808.

25. Chowgule RV, Shetye VM, Parmar JR.: Lung function tests in normal Indian children. Indian pediatrics 1995-Feb; 32:185-191.

26. Dickman ML, Schmidt CD, Gardner RM.: Spiro metric standards for normal children and adolescents. (ages 5 years through 18 years)Am Rev Respir dis 1971; 104: 680-687.

27. Meenakshi Sharma , Rambabu Sharma , Neelam Singh , Kusum Gaur: FEV1 , FVC, FEV1 /FVC ratio in children of 7-14 years of age from Western Rajasthan. J Bangladesh Soc Physiol. 2013 June; 8(1): 37-41

28. Gundogdu Z, Eryilmaz N. Correlation between peak flow and body mass index in obese and nonobese children in Kocaeli, Turkey. Prim Care Respir J 2011; 20:4036.

29. Soundariya K, Neelambika N. Influence of anthropometric indices on pulmonary function tests in young individuals. World J Med Sci 2013; 9:15761.

# A Study of Nerve Conduction Test & Other Hematological Parameters in Diabetics

**Jasmin J. Parmar[1], Jitendra R. Patel[2]**

[1]*Associate Professor, Department of Physiology, Banas Medical College & Research Institute, Palanpur, Gujarat,*
[2]*Associate Professor, Department of Physiology, Gujarat Adani Institute Of Medical Science, Bhuj, Gujarat*

## Abstract

**Background**: Nerve conduction studies (NCS) are most sensitive, reliable and non-invasive investigation to detect diabetic neuropathy. Sensory nerves are more affected as compared to motor nerves in diabetic neuropathy. Understanding the Nerve conduction studies is useful for early detection polyneuropathy and prevention of its dreaded complications.

**Materials and Method**: The present study was conducted among 30 subjects of type 2 Diabetic and 30 non diabetics subjects. Haematological parameters such as HbA1c, platelet count, prothrombin time, APTT, triglycerides, LDL and HDL levels were estimated. Motor nerve conduction velocity median , ulnar, common peroneal and posterior tibial nerve and sensory nerve conduction velocity median , ulnar and sural nerve were recorded.

**Result**: Mean age of diabetic patients was 51±.4.8 year. Most of the subjects (86.7%) were male. Sensory nerve conduction of sural nerve was signiicantly reduced in diabetic patients as compared to non diabetic patients. SNCV of sural nerve showed deterioration with the duration of diabetes, poor glycemic control and patients with altered lipid profile.

**Conclusion**: Nerve conduction velocity testing of sural nerve is useful for early detection of diabetic neuropathy and reduce the burden of complication and to improve the quality of life in diabetics.

**Keywords**: *Sensory nerve conduction velocity, Sural nerve, Diabetic peripheral neuropathy, HbA1c*

## Introduction

Diabetes mellitus (DM) is a metabolic disorder characterized by chronic hyperglycaemia with abnormalities in carbohydrate metabolism.[1] In India, prevalence of diabetic peripheral neuropathy in Type 2 diabetic patients was 26.1%.[2] Distal symmetric sensorimotor polyneuropathy is the most common clinical type of DPN. Three fourth of neurologically asymptomatic patients may have abnormal nerve conduction.[1] Age, male gender, duration of diabetes, glycated hemoglobin levels and insulin use increase risk of peripheral neuropathy .[3]

**Corresponding Author:**
**Dr. Jitendra R. Patel**
Associate Professor, Department Of Physiology,
Gujarat Adani Institute Of Medical Science, Bhuj,
Gujarat

Nerve conduction studies (NCS) are most sensitive, specific, reliable and non-invasive investigation to detect diabetic neuropathy.[4] Routine NCS include evaluation of motor conduction velocity of the median, ulnar, peroneal and tibial nerves, and sensory conduction velocity of median, ulnar and sural nerves.[1] Sensory nerves are more affected as compared to motor nerves in diabetic neuropathy. [5,6] Relationship between poor glycaemic control and DPN has been reported by various authors. [7,8] The Nerve conduction studies (NCS) is crucial for early detection and prevention of its dreaded complications of DPN.[9,10]

## Aims & Objectives

➢ To study nerve conduction velocity in type 2 diabetes mellitus patients and non diabetic patients.

➢ To study corealtion of haematological parameter

like HbA1c, platelet count, prothrombin time, APTT, triglycerides, LDL and HDL levels with development of polyneuropathy.

## Materials and Method

The present study was conducted among 30 subjects of type 2 Diabetic Subjects in age group (30-65 years) attending out patient clinic of Nalini Chowdhary's neuro center in Hanamkonda, Telangana. The another 30 patients of age group of 30-65 years who seeking for various other medical problems without diabetes were included in control group. Patients with neuropathy due to any other cause were excluded. Detail history regarding complaints of neuropathy was taken. Haematological parameters such as HbA1c, platelet count, prothrombin time, APTT, triglycerides, LDL and HDL levels were estimated in diagnostic center at Hanamkonda.

Nerve conduction studies were performed with Clarity Octopus NCV/EMG machine. Written consent was taken from each patient. Patient's limb was placed in relaxed position. Room temperature was maintained between 21– 23°C as any degree rise or fall in temperature can change the results. Electrode gel was applied under the electrode and affix the electrode with adhesive tape to the skin for reduction of electrode impedance. Motor nerve conduction velocity median, ulnar, common peroneal and posterior tibial nerve and sensory nerve conduction velocity median, ulnar and sural nerve were recorded.

Data was entered in Microsoft Excel 2010 and analyzed using Epi info 7.1 and analyzed in terms of mean and SD. T-test for difference of means was used. "P" values equal to or less than 0.05 was considered as significant.

## Result

Mean age of diabetic patients (51±.4.8) and control group (48.2 ± 9.2) was comparable (p > 0.05). Twenty six male (86.7%) had diabetes. Twenty five patients (83.3%) in control group were male. Mean duration of diabetes was 7.0 ± 3.4 year.

**Table 1: Gender and age wise distibution of case and control group.**

| Characteristicts | Case | Control | P value |
|---|---|---|---|
| Age | 51±.4.8 | 48.2 ± 9.2 | > 0.05 |
| Male | 26 (86.7%) | 25 (83.3%) | > 0.05 |
| Female | 4 (13.3%) | 5 (16.7%) | |

**Table 2: Comparison of various laboratory parameters case and control group.**

| Parameter | Case | Control | p value |
|---|---|---|---|
| HbA1C | 8.1 ± 1.1 | 5.5 ± 0.8 | < 0.05 |
| FBS | 136 ± 13.0 | 81 ± 9.7 | < 0.05 |
| PPBS | 212 ± 35.0 | 130 ± 9.3 | < 0.05 |
| HDL | 25 ± 3.6 | 72 ± 9.5 | < 0.05 |
| TG | 161 ± 23.0 | 36 ± 7.5 | < 0.05 |
| LDL | 130 ± 28.0 | 82 ± 8.9 | < 0.05 |
| Platelet count | 4.4 ± 0.5 | 2.1 ± 0.4 | < 0.05 |
| PT | 13 ± 1.9 | 13 ± 1.9 | > 0.05 |
| APTT | 34.8 ± 2.5 | 33.5 ± 2.5 | > 0.05 |

Table 2 shows comparison of various laboratory parameters in subjects of case and control group. HbA1c, fasting blood sugar (FBS), post prandial blood sugar (PPBS) and platelet count were significantly higher in diabetic patients as compared to control group. There was no any significant difference in PT and APTT among two groups. Lipid profile was also significantly altered in diabetic patients.

**Table 3: Motor nerve conduction velocity (MNCV) among case and control group**.

| MNCV | Case | Control | p value |
|------|------|---------|---------|
| Median nerve | 57.3 ± 3.2 | 57.0 ± 3.3 | >0.05 |
| Ulnar nerve | 57.4 ± 3.1 | 57.3 ± 3.2 | >0.05 |
| Common peroneal nerve | 46.0 ± 2.6 | 46.6 ± 2.5 | >0.05 |
| Posterior tibial nerve | 49.4 ± 3.5 | 46.9 ± 8.1 | >0.05 |

Table 3 shows the MNCV in median, ulnar, common peroneal, and posterior tibial nerves. MNCV of all these nerves were normal in diabetic and non diabetics patients .

**Table 4: Sensory nerve conduction velocity (SNCV) among case and control group.**

| SNCV | Case | Control | p value |
|------|------|---------|---------|
| Median nerve | 48.8 ± 3.9 | 49.2 ± 4.0 | >0.05 |
| Ulnar nerve | 58.3 ± 2.7 | 54.9 ± 9.6 | >0.05 |
| Sural nerve | 46.8 ± 6.8 | 49.7 ± 3.9 | <0.05 |

Results of SNCV of median, ulnar and sural nerve are reported in Table 4. The SNCV of median & ulnar nerves are normal in control & cases group. However, sensory nerve conduction in sural nerve of diabetic patients (46.8 ± 6.8) was significantly lower as compared to control group (49.7 ± 3.9). The normal range of sural sensory nerve conduction velocity is 45.5-56.3m/s. Eleven subjects (36.6%) in cases group were observed with decreased sensory nerve conduction velocity of sural nerve. The mean sensory conduction velocity of sural nerve in these 11 subjects was 38.98m/s. They were having subclinical distal sensory peripheral neuropathy.
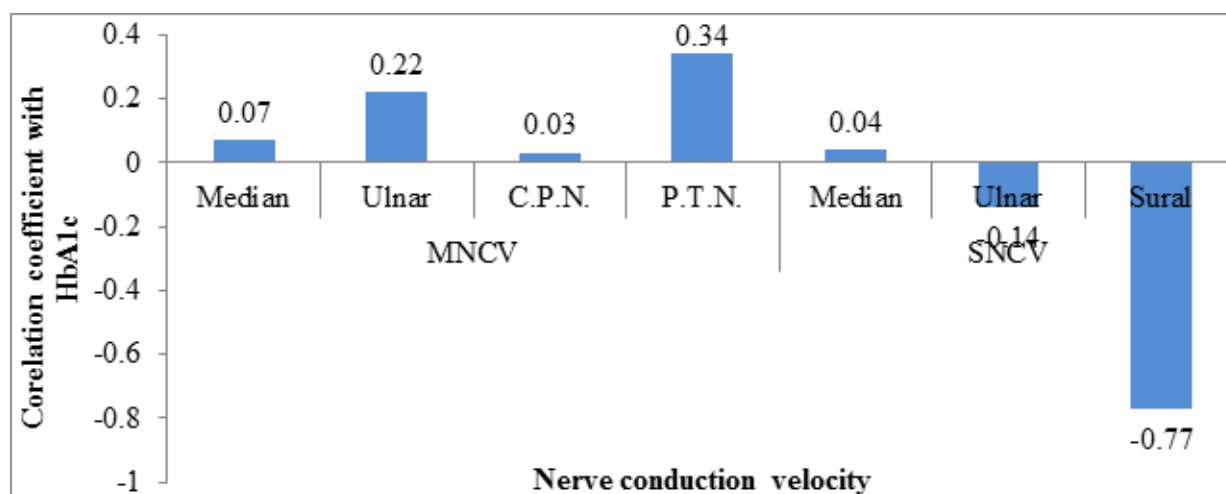


**Figure 1: Correlation of HbA1C with nerve conduction velocity**

Correlation between HbA1c and NCV in different nerves was summarized in figure 1. Only SNCV of sural nerve was negatively correlated with $HBA_1C$ (r=-0.72).
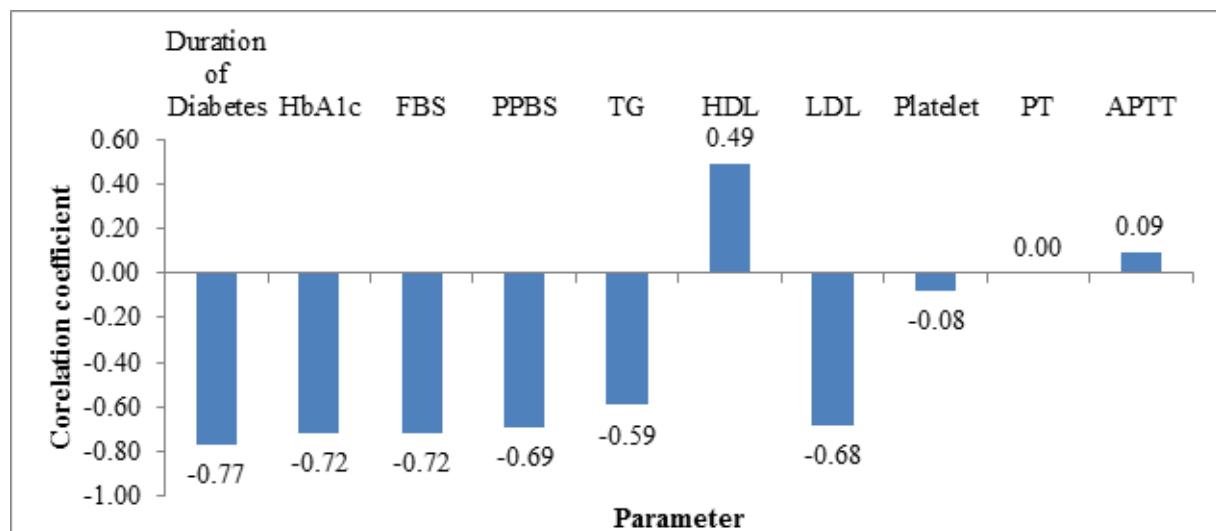
**Figure 2: Correlation of SNCV of sural nerve with laboratory parameter and duration of diabetes.**

SNCV of sural nerve was negatively correlated with duration of diabetes (r=-0.77), HBA₁C (r=-0.72), FBS (r=-0.72), PPBS (r=-0.69), TG (r=-0.59) and LDL (r=-0.68).

## Discussion

In the present study, mean age of diabetic patients was 51±.4.8 year. Therefore, the result is applicable only to patients over 50 year age group. Our study group is comparable to other studies with the same age group of subjects.[11] Most of the subjects (86.7%) were male. Other studies also show a higher prevalence of DM in male as compared to females.[12]

In the present study, sensory nerve conduction of sural nerve was signiicantly reduced in diabetic patients as compared to non diabetic patients. Involvement of sural nerve is suggesting that long nerves are commonly affected. [13] Lower limb is more commonly affected due to length dependent dying back process.We observed that sensory NCS of sural nerve was f inferior with poor glycemic control (higher HbA1c, FBS and PPBS ). Previous study reported that HbA1c > 6.5% increased risk for polyneuropathy in DM patients by more than 5-fold. [14] In the present study, SNCV of sural nerve also showed deterioration with the duration of diabetes. Lee et al.also observed that sural sensory amplitude was significantly associated with polyneuropathy in DM patients. [14] Oguejiofor et al. reported a higher in patients with a duration of DM >15 years and lower prevalence of neuropathy in those with duration of DM <5 years. [15] Various studies suggested risk factors for the neuropathy development such as high cholesterol levels, smoking, hypertension, male sex, older age and poor glycaemic control.[16,17]

## Conclusion

The current study shows that diabetic neuropathy is more common in middle and elderly male patients. It was positively relating with duration of diabetes, poor glycemic control and altered lipid profile. Sural nerve was more commonly involved. Nerve conduction velocity testing of sural nerve is useful for early detection of diabetic neuropathy and reduce the burden of complication and to improve the quality of life in diabetics.

## References

1.  Agarwal S, Lukhmana S, Kahlon N, Malik P, Nandini H. Nerve conduction study in neurologically asymptomatic diabetic patients and correlation with glycosylated hemoglobin and duration of diabetes. National Journal of Physiology, Pharmacy and Pharmacology. 2018;8(11):1533-8.

2.  Pradeepa R, Rema M, Vignesh J, Deepa M, Deepa R, Mohan V. Prevalence and risk factors for diabetic neuropathy in an urban south Indian population: the Chennai Urban Rural Epidemiology Study (CURES-55). Diabetic Medicine. 2008

Apr;25(4):407-12.

3. Franklin GM, Shetterly SM, Cohen JA, Baxter J, Hamman RF. Risk factors for distal symmetric neuropathy in NIDDM: the San Luis Valley Diabetes Study. Diabetes Care. 1994 Oct 1;17(10):1172-7.

4. Claus D, Mustafa C, Vogel W, Herz M, Neundörfer B. Assessment of diabetic neuropathy: Definition of normal and discrimination of abnormal nerve function. Muscle & Nerve: Official Journal of the American Association of Electrodiagnostic Medicine. 1993 Jul;16(7):757-68.

5. Lamontagne A, Buchthal F. Electrophysiological studies in diabetic neuropathy. Journal of Neurology, Neurosurgery & Psychiatry. 1970 Aug 1;33(4):442-52.

6. Hendriksen PH, Oey PL, Wieneke GH, Bravenboer B, Van Huffelen AC. Subclinical diabetic polyneuropathy: early detection of involvement of different nerve fibre types. Journal of Neurology, Neurosurgery & Psychiatry. 1993 May 1;56(5):509-14.

7. Kannan MA, Sarva S, Kandadai RM, Paturi VR, Jabeen SA, Borgohain R. Prevalence of neuropathy in patients with impaired glucose tolerance using various electrophysiological tests. Neurology India. 2014 Nov 1;62(6):656.

8. Herman WH, Aubert RE, Engelgau MM, Thompson TJ, Ali MA, Sous ES, Hegazy M, Badran A, Kenny SJ, Gunter EW, Malarcher AM. Diabetes mellitus in Egypt: glycaemic control and microvascular and neuropathic complications. Diabetic Medicine. 1998 Dec;15(12):1045-51.

9. Chatzikosma G, Pafili K, Demetriou M, Vadikolias K, Maltezos E, Papanas N. Evaluation of sural nerve automated nerve conduction study in the diagnosis of peripheral neuropathy in patients with type 2 diabetes mellitus. Archives of medical science: AMS. 2016 Apr 1;12(2):390.

10. Squintani G, Zoppini G, Donato F, Pineschi E, Donini D, Stoico V, Moretto G, Bonora E, Morini A. Antidromic sensory nerve conduction study of the digital branches of the medial plantar nerve: a novel method to detect early diabetic sensory axonal polyneuropathy. Muscle & nerve. 2014 Aug;50(2):193-9.

11. Gul S, Dar MA, Ali A, Ashraf A, Qadri S. Prevalence of peripheral neuropathy and pattern of involvement on nerve conduction study in patients of type 2 diabetes mellitus with clinical symptoms of peripheral neuropathy in Kashmiri population: a hospital-based study. Imperial J Interdisciplinary Res. 2017;3(2).

12. American Diabetes Association. 2. Classification and Diagnosis of Diabetes: Standards of Medical Care in Diabetes-2018. Diabetes Care. 2018 1;41(Supplement 1):S13-27.

13. Franssen H. Nerve conduction studies in polyneuropathy: practical physiology and patterns of abnormality. Acta neurologica belgica. 2006 Jun;106(2):73-81.

14. Lee WJ, Jang S, Lee SH, Lee HS. Correlation between the severity of diabetic peripheral polyneuropathy and glycosylated hemoglobin levels: a quantitative study. Annals of rehabilitation medicine. 2016 Apr;40(2):263.

15. Oguejiofor OC, Odenigbo CU, Oguejiofor CB. Evaluation of the effect of duration of diabetes mellitus on peripheral neuropathy using the United Kingdom screening test scoring system, bio-thesiometry and aesthesiometry. Nigerian journal of clinical practice. 2010;13(3).

16. Harati Y. Diabetic neuropathies: unanswered questions. Neurologic clinics. 2007 Feb 1;25(1):303-17.

17. Fazan VP, de Vasconcelos CA, Valenca MM, Nessler R, Moore KC. Diabetic Peripheral Neuropathies: A Morphometric Overview. International Journal of Morphology. 2010 Mar 1;28(1).

# Evaluation of CRP in Patients with Asthma

**Karthika M[1], Prakash Chandra Bhardwaj[2], Deisha B Rymbui[3], W Kanan[4], W Asoka Singh[5]**

[1]Senior Resident, Department of Physiology, JIPMER, Puducherry, [2]MD (Physiology), Department of Health Services, Manipur, [3]MD (Physiology), Department of Health Services, Meghalaya, [4]Professor & Head Department of Physiology, Regional Institute of Medical Sciences, Imphal, Manipur, [5]Professor Department of Respiratory Medicine, Regional Institute of Medical Sciences, Imphal, Manipur

## Abstract

Patients with asthma have an ongoing inflammation, which can be assessed by measuring serum C- reactive protein.

**Objective**: To explore whether CRP could be used as a predictor of disease outcome in asthma. **Methods**: A Cross-sectional study was conducted among 50 asthma patients attending Respiratory Medicine outpatient services in Regional Institute of Medical Sciences (RIMS), Imphal from January 2015 to September 2016. Patients aged 18-67 were included in the study after obtaining Ethical approval from the Research Ethics Board, RIMS, Imphal. Computerized Spirometer Helios 401 was the instrument used to measure lung volumes and capacities. BeneSphera™ CRP Latex Slide test kit was used to estimate serum C-reactive protein.

**Results and Observation**: The present study was conducted on fifty asthma patients in which serum CRP level showed association with severity of asthma ( p=0.001) but serum CRP level with spirometric parameters showed significant negative correlation; FVC (r=-0.711, p<0.001), $FEV_1$ (r=-0.665, p<0.001), $FEV_1$/FVC (r=-0.429, p=0.002), $FEF_{25-75\%}$ (r=-0.535, p<0.001), and PEFR (r=-0.515, p<0.001).

**Conclusion**: In our study we found association between serum CRP level and asthma and plasma CRP may be used as a marker of prognosis in patients with asthma.

**Keywords** : C- reactive protein, Asthma

## Introduction

Asthma is one of the most common chronic disease and currently affects nearly 300 million people globally. The prevalence of asthma has risen in affluent countries over the past 30 years with 10-12% of adults and 15% of children affected by the disease.[1]

The NHLBI (National Heart Lung and Blood Institute, US) defined asthma in 2002 as: a chronic inflammatory disorder of the airways in which many cells and cellular elements play a role. [2] The chronic inflammation of the airway causes recurrent episodes of wheezing, chest tightness, breathlessness, and coughing, particularly at night and in the early morning. These episodes are usually associated with wide spread but variable airflow obstruction that is often reversible either spontaneously or with treatment.[2]

Diagnosis is based on the patient`s history, signs, and symptoms, and on the results of spirometry and other pulmonary function tests. Spirometry assesses the obstruction of expiratory airflow, which is the characteristic functional defect. Spirometry is the most effective way of determining the severity of obstructive airway diseases.[3]

C-reactive protein was first isolated in 1930 and was named so because it binds to the C-polysaccharide of the pneumococcus. CRP was first found in the plasma of patients with pneumococcal pneumonia. Modern

**Corresponding author:**
**Dr. Karthika**
M, Senior Resident, Department of Physiology, JIPMER, Puducherry.

molecular studies have determined that CRP is a member of the pentraxin family of proteins. It comprises five protomers, each of 206 amino acids, molecular weight 23 kDa, arranged in cyclic symmetry. With the participation of $Ca^{+2}$ ions, it binds various proteins and phospholipids, particularly phosphocholine. It opsonizes particles and also activates complement via the classical pathway, but its actual biological function is unknown.[4]

C-reactive protein is an abnormal protein that appears in blood in the acute stages of various inflammatory disorders but is undetectable in the blood of healthy persons.[5]

C-reactive protein is an acute phase protein synthesized in the liver during infection, injury and systemic inflammatory conditions. Worldwide studies had shown that it has variable roles in asthma, some showing correlation with spirometric lung function while others with no significant relation. Hence this study was planned to explore its role in the population and also to determine whether it could become an independent predictor of disease outcome in asthma.

## Method

**Study design** A Cross-sectional study was conducted among 50 asthma patients attending Respiratory Medicine outpatient services in Regional Institute of Medical Sciences (RIMS), Imphal from January 2015 to September 2016. Patients aged 18-67 were included in the study after obtaining Ethical approval from the Research Ethics Board, RIMS, Imphal. The participants were recruited by Purposive sampling. Diagnosed asthma patients sent from Respiratory Medicine OPD, RIMS, Imphal were included in this study. Socio-demographic characteristics like age, sex, smoking history were recorded. Thorough physical examination, degree of airflow obstruction and serum C-reactive protein were recorded after obtaining Prior written informed consent form from all the participants.

The patients with chronic disorders like hypertension, diabetes mellitus, cardiovascular diseases, bleeding disorders, inflammatory disorders, infection , malignancy and patients who had recent surgery were excluded from the study.

Computerized Spirometer Helios 401 of the Recorders and Medicare System, Chandigarh,India was the instrument used to measure lung volumes and capacities. The Helios software contains set of prediction equations for computation of predicted parameter values.

The procedure was explained to the patient followed by a demonstration. The patient was asked to "take as deep a breath as possible" and then "blast as fast and hard as you can" and "keep blowing until I ask you to stop" preferably at least 3 seconds followed by a rapid inhalation (inspiration). A tight seal was ensured around the mouthpiece. During the test, soft nose clip was used to prevent air escaping through the nose. Coaching was active and vigorous; instructions were repeated as necessary.Three consecutive maneuvers were performed with a rest of 5 to 10 minutes between two maneuvers.

The best result among the three tests were recorded in the proforma. The results were compared with the predicted values for the same age, sex, height, and weight.

The study variables which include Forced Vital Capacity (FVC), Forced Expiratory Volume in one second ($FEV_1$), $FEV_1$/FVC ratio, Forced Expiratory Flow during 25-75% of expiratory flow ($FEF_{25-75\%}$), and Peak Expiratory Flow Rate (PEFR), were recorded by Helios Computerized Spirometer Model No. 401, in a sitting position. Patients were classified on the basis of GINA Classification of asthma(2006)[6]

BeneSphera™ CRP Latex Slide test kit of Avantor Performance Materials India Ltd, Dehradun, Uttarakhand, was used to estimate serum c-reactive protein in human. The test was expected to be positive with serum CRP levels between 0.6 and 100mg/dl.

## Statistical Analysis

The collected data were entered and analyzed using IBM *SPSS* Statistics *V21*.0 (IBM Corporation, US). Summarizations of data for frequency distribution for variables of interest were carried out by using descriptive statistics such as mean, standard deviation and percentages. Chi-square test was employed to test the association between asthma with variables of interest. A p-value of <0.05 was considered significant.

## Results and Observation

A total of 50 patients with asthma were included in the study. Of which 36% of patients belonged to the age group (38-67years) and 64% of the patients belong to age group (18-37years). About two third of the Obstructive Airway Disease patients were male. Out of 50, 82% patients were non-smoker, 12% were smoker, and 6%

were ex smokers. Socio-demographic characteristics like age, sex, smoking history were recorded. Thorough physical examination, degree of airflow obstruction and serum C-reactive protein were recorded after obtaining Prior written informed consent from from all the participants.

Patients were classified on the basis of GINA Classification of asthma. In our study group shows 46% belonged to intermittent persistent category of asthma ($FEV_1 \geq 0.80$ and PEF variability < 20%), and 20% were in mild persistent category ($FEV_1 \geq 0.80$ and PEF variability 20-30%). 16% of the patients were in moderate persistent category ($FEV_1 = 0.60-0.80$ and PEF variability >30%), while 18% of the patients were in very severe persistent category ($FEV_1 \leq 0.60$ and PEF variability >30%).

**Table 1: Mean±SD of FVC, $FEV_1$, $FEV_1$/FVC, FEF25-75%, and PEFR in Asthma patients (N=50)**

| Spirometric parameters | Minimum Value(%pred) | Maximum Value(%pred) | Mean(%pred) | Standard Deviation |
|---|---|---|---|---|
| FVC | 47.00 | 149.00 | 100.02 | 24.64 |
| FEV1 | 28.00 | 138.00 | 90.82 | 30.42 |
| FEV1/FVC | 42.00 | 115.00 | 89.26 | 15.10 |
| FEF25-75% | 7.00 | 138.00 | 58.18 | 30.91 |
| PEFR | 15.00 | 107.00 | 63.40 | 23.52 |

Table 1 shows the lung function test values of Asthma patients as measured by spirometer. All the values were within normal limit.

**Table 2: Association of the serum CRP level with the severity of Asthma**

| Severity of Asthma | CRP | | | Total | p-value* |
|---|---|---|---|---|---|
| | LESS THAN 6mg/L | 6mg/L | 12 mg/L | | |
| Intermittent | 22 | 1 | 0 | 23 | |
| Mild | 10 | 0 | 0 | 10 | |
| Moderate | 5 | 3 | 0 | 8 | <0.001 |
| Severe | 2 | 3 | 4 | 9 | |
| Total | 39 | 7 | 4 | 50 | |

*Chi-Square Test

Table 2 shows that the association between the serum CRP level with the severity of asthma. Patients with intermittent (44% of asthmatic patients) and mild (20% of asthmatic patients) persistent asthma had serum CRP level <6mg/L. Total 7(14%) patients had serum CRP level 6mg/L, in which 1(2%) patient belong to intermittent asthma, 3(6%) patients were in each moderate and severe persistent category of asthma. Out of 9 severe asthmatic patients 4 had serum CRP level 12mg/L (p<0.001).

**Table 3: Association of the serum CRP level with the Visiting day to hospital OPD of Asthmatic patients after asthma attack**

| Visit to Hospital OPD | CRP | | | Total | p-value* |
|---|---|---|---|---|---|
| | LESS THAN 6mg/L | 6mg/L | 12 mg/L | | |
| Same day | 0 | 0 | 4 | 4 | |
| 1 day | 2 | 7 | 0 | 9 | |
| 2 day | 12 | 0 | 0 | 12 | <0.001 |
| More than 2 days | 25 | 0 | 0 | 25 | |
| Total | 39 | 7 | 4 | 50 | |

*Chi-Square Test

Above table shows that association between level of serum CRP in asthma patients with the visiting day to hospital OPD after exaggeration of symptoms. Those asthma patients (8% of asthma patients) who were visited same day of exaggeration of symptoms had serum CRP level 12mg/L, while those asthma patients (74% of asthma patients) who were visited after 2 or more days had serum CRP level <6mg/L (p<0.001).

**Table 4: Correlation of different parameters of spirometry with serum CRP level in Asthma patients (n=50)**

| Parameter | Correlation coefficient(r)* | p-value |
|---|---|---|
| FVC | -0.711 | <0.001 |
| FEV1 | -0.665 | <0.001 |
| FEV1/FVC | -0.429 | 0.002 |
| FEF25-75% | -0.535 | <0.001 |
| PEFR | -0.515 | <0.001 |

*Pearson`s bivariate correlation coefficient

Above table shows that in asthma patients serum C-reactive protein level were significantly correlated with spirometric lung parameters.

## Discussion

Obstructive airway disease (OAD) is a disorder characterized by chronic inflammation of airways which leads to lung function impairment. The term OAD includes bronchial asthma and chronic obstructive pulmonary disease. Inflammation plays an important role in the pathogenesis of asthma. C-reactive protein is an acute phase protein produced by liver during inflammation or injury in the body. Higher level of CRP in blood during exacerbation of obstructive airway disease is not uncommon. However, the ability of serum CRP in predicting asthma control has not been sufficiently investigated yet. We would therefore like to explore its role in our study population and also try to explore whether it could become an independent predictor of disease outcome in asthma.

The present study was conducted on fifty asthma patients in which serum CRP level showed association with severity of asthma ( p=0.001) but serum CRP level with spirometric parameters showed significant negative correlation; FVC (r=-0.711, p<0.001), $FEV_1$ (r=-0.665, p<0.001), $FEV_1$/FVC (r=-0.429, p=0.002), $FEF_{25-75\%}$ (r=-0.535, p<0.00<), and PEFR (r=-0.515, p<0.001).

Increased serum high sensitive C-reactive protein (hs-CRP) in asthma and its association with the disease has been investigated in many studies. In this study significant association was found between serum CRP level and severity of asthma (p<0.001). These results were supported by other authors, such as Zietkowski Z et al[28] reported that serum levels of hs-CRP in all groups of asthmatics were significantly higher than those measured in healthy volunteers. Kilic H et al[39] also noted a significant positive correlation between the levels of hs-CRP and the severity of asthma(p = 0.04, r = 0.38).

Furthermore, a recent population-based study by Kony S et al showed associations of increased levels of serum hs-CRP with a high frequency of airway hyperresponsiveness and low forced expiratory volume in one second ($FEV_1$) among subjects without heart disease[7], suggesting that systemic inflammation may be associated with respiratory impairment. Another recent epidemiological study by *Olafsdottir IS* et al showed that elevated levels of hs-CRP correlate significantly with respiratory symptoms and with prevalence of nonallergic asthma[8].

*Sävykoski T* et al found that the difference in hs-CRP levels remained significant when the comparison was confined to patients with moderate asthma and healthy controls with a normal BMI ($<25 \ kg \cdot m^{-2}$) or nonsmokers[9]. In another study by Shimoda et al., the log serum hs-CRP of 2.3 distinguished asthmatic patients from healthy controls at sensitivity of 69% and specificity of 70%.[10]

In the study by M. Takemura et al, patients with steroid-naive asthma showed increased serum hs-CRP levels compared with healthy controls. Moreover, serum hs-CRP levels correlated negatively with $FEV_1$, $FEV_1/FVC$ and $FEF_{25-75\%}$, positively with numbers of sputum eosinophils and marginally with numbers of macrophages and neutrophils[11] which was similar to our study.

## Conclusion

In this study significant association was found between serum CRP level and severity of asthma(p<0.001). This study has limitations regarding study design which is cross-sectional. The serum CRP has been used for evaluation of treatment in chronic pulmonary airway disease and other inflammatory conditions but several factors other than the presence of asthma might affect serum levels of CRP. CRP may be elevated in obese individuals and also in people who have the habit of smoking. Ageing is also another confounding factor. Another limitation of this study is lack of steroid naive asthma patients. Treatment of patients with corticosteroids may reduce the level of serum CRP. Further longitudinal studies are necessary to evaluate whether CRP could be used an independent predictor of disease outcome in asthma.

## References

1. Asher MI, Montefort S, Björkstén B, Lai CK, Strachan DP, Weiland SK, et al. Worldwide time trends in the prevalence of symptoms of asthma, allergy rhinoconjunctivitis, and eczema in childhood: ISAAC phases one and three repeat multicountry cross-sectional surveys. Lancet 2006;368(9537):733-43.

2. Global strategy for the diagnosis, management, and prevention of chronic obstructive pulmonary disease (2006). Available at: http://www.goldcopd. org. Accessed September 19, 2016.

3. Bellamy D. Spirometry in practice: a practical guide to using spirometry in primary care. 2nd ed. London: BTS COPD Consortium; 2005.

4. Block JL. Evaluation of cardiac injury and function. In: McPherson RA, Pincus MR, editors. Henry`s Clinical Diagnosis and Management by Laboratory Methods. 21st ed. New Delhi: Elsevier; 2009. p. 224-5.

5. Sonnenwirth AC. Serological tests in infectious disease-II. In: Sonnenwirth, Jarett L, editors. Gradwohl`s Clinical Laboratory Methods and Diagonosis. 8th ed. New Delhi: B.I. Publications Ltd; 1990. p. 2328-31.

6. Koshak EA. Classification of asthma according to revised 2006 GINA: Evolution from severity to control. Ann Thorac Med 2007;2(2):45-6.

7. Kony S, Zureik M, Driss F, Neukirch C, Leynaert B, Neukirch F. Association of bronchial hyperresponsiveness and lung function with C-reactive protein (CRP): a population based study. Thorax 2004;59:892–896.

8. Olafsdottir IS, Gislason T, Thjodleifsson B, et al. C reactive protein levels are increased in non-allergic but not allergic asthma: a multicentre epidemiological study. Thorax 2005;60:451–454.

9. Sävykoski T, Harju T, Paldanius M, et al. Chlamydia pneumoniae infection and inflammation in adults with asthma. Respiration 2004;71:120–125.

10. 15. Shimoda T, Obase Y, Kishikawa R, Iwanaga T. Serum high-sensitivity C-reactive protein can be an airway inflammation predictor in bronchial asthma. Allergy Asthma Proc. 2015;36:e23–8.

11. M. Takemura, H. Matsumoto, A. Niimi, T. Ueda, H. Matsuoka, M. Yamaguchi, et al. High sensitivity C-reactive protein in asthma. Eur Respire J 2006; 27: 908-912.

# Relationship between Smoking and Obstructive Airways Disease

**Karthika M[1], Prakash Chandra Bhardwaj[2], Laimayum Amarnath Sharma[3], W Kanan[4], W Asoka Singh[5]**

*[1]SeniorResident, Department of Physiology, JIPMER, Puducherry, [2]MD (Physiology), Department of Health Services, Manipur, [3]Medical officer, DR TB Center, JNIMS Hospital, Imphal, Manipur, [4]Professor & Head Department of Physiology, Regional Institute of Medical Sciences, Imphal, Manipur, [5]Professor Department of Respiratory Medicine, Regional Institute of Medical Sciences, Imphal, Manipur*

## Abstract

Obstructive airways disease is an important cause of mortality and morbidity around the world and smoking has a significant role in the development and progression of the disease. **Objective**: To analyse the relationship between smoking and obstructive airways disease. **Method**: A cross-sectional study was conducted among 100 patients with obstructive airways disease attending Respiratory Medicine outpatient services in Regional Institute of Medical Sciences (RIMS), Imphal from January 2015 to September 2016. Patients aged 18-67 were included in the study after obtaining Ethical approval from the Research Ethics Board, RIMS, Imphal. Computerized Spirometer Helios 401 was the instrument used to measure lung volumes and capacities. **Results and observation**: The present study was conducted on 100 patients with obstructive airways disease. The lung function test values of OAD patients showed that FVC, $FEV_1$, and $FEV_1$/FVC values were within normal limits. But $FEF_{25-75\%}$, and PEFR were lower suggesting that smaller airways were affected in obstructive airway disease. In our study we found that OAD was strongly associated with smoking status($p<0.001$), smoking pack-years($p=0.000$), and smoking duration($p<0.001$). **Conclusion**: From the study results we conclude that that Obstructive airway disease was strongly associated with smoking. Smoking cessation is the best and most effective solution to this problem. Other measures such as screening with spirometric tests in high risk individuals especially the smokers in age group of 40-55 should be considered to reduce the mortality and morbidity due to Obstructive airway disease.

*Keywords - Obstructive airways disease (OAD), smoking, spirometric test*

## Introduction

Obstructive airways disease is a group of condition distinguished by increased resistance and obstruction in the air passages, especially during expiration. The term OAD includes bronchial asthma; chronic obstructive pulmonary disease, consisting of chronic bronchitis and emphysema; bronchiectasis; cystic fibrosis and bronchiolitis.[1]

Asthma is one of the most common chronic disease and currently affects nearly 300 million people globally.

The prevalence of asthma has risen in affluent countries over the past 30 years with 10-12% of adults and 15% of children affected by the disease.[2]

COPD is a characterized by airflow limitation that is not fully reversible. The airflow obstruction is usually progressive and associated with an abnormal inflammatory response of the lungs to noxious particles. There are around 50 million patients with COPD in India and COPD is the second leading cause of death in India. Estimate suggests that COPD will rise from the sixth to the third most common cause of death worldwide by 2020.[3]

The WHO has estimated that there are around one billion smokers around the world of which around two thirds live in the developing countries. Even in the

**Corresponding author:**
**Dr. Karthika. M,**
Senior Resident, Department of Physiology, JIPMER, Puducherry

developed countries one fourth of the adults with asthma are smokers. Active smoking causes increased bronchial responsiveness, increased frequency and exaggeration of acute episodes, also decline in lung function. There is also increased sensitization of occupational agents in patients who smoke compared to non smokers.

Diagnosis is based on the patient`s history, signs, and symptoms, and on the results of spirometry and other pulmonary function tests. Spirometry assesses the obstruction of expiratory airflow, which is the characteristic functional defect. Spirometry is the most effective way of determining the severity of obstructive airway diseases.[4]

The aim of our study is to analyse the relationship between smoking and Obstructive airways disease

## Method

**Study design**: A Cross-sectional study was conducted among 100 patients with Obstructive airways disease attending Respiratory Medicine outpatient services in Regional Institute of Medical Sciences (RIMS), Imphal from January 2015 to September 2016. Patients aged 18-67 were included in the study after obtaining Ethical approval from the Research Ethics Board, RIMS, Imphal. The participants were recruited by Purposive sampling. Diagnosed patients sent from Respiratory Medicine OPD, RIMS, Imphal were included in this study.

Socio-demographic characteristics like age, sex, smoking history were recorded. Thorough physical examination was done and the degree of airflow obstruction was recorded after obtaining Prior written informed consent form from all the participants. The patients with chronic disorders like hypertension, diabetes mellitus, cardiovascular diseases, bleeding disorders, inflammatory disorders, infection, malignancy and patients who had recent surgery were excluded from the study.

**Sample size:**

Sample size was calculated based on the formula

$n = 4pq/l^2$ where, p is prevalence, q=1-p, and l is allowable error

The calculated sample size was 100. There was not much knowledge about the prevalence of OAD in Indian population, so 50% prevalence was taken with 10% allowable error

Computerized Spirometer Helios 401 of the Recorders and Medicare System, Chandigarh, India was the instrument used to measure lung volumes and capacities. The Helios software contains set of prediction equations for computation of predicted parameter values.

The procedure was explained to the patient followed by a demonstration. The patient was asked to "take as deep a breath as possible" and then "blast as fast and hard as you can" and "keep blowing until I ask you to stop" preferably at least 3 seconds followed by a rapid inhalation (inspiration). A tight seal was ensured around the mouthpiece. During the test, soft nose clip was used to prevent air escaping through the nose. Three consecutive maneuvers were performed with a rest of 5 to 10 minutes between two maneuvers.

The best result among the three tests was recorded. The results were compared with the predicted values for the same age, sex, height, and weight.

The study variables which include Forced Vital Capacity (FVC), Forced Expiratory Volume in one second ($FEV_1$), $FEV_1$/FVC ratio, Forced Expiratory Flow during 25-75% of expiratory flow ($FEF_{25-75\%}$), and Peak Expiratory Flow Rate (PEFR), were recorded by Helios Computerized Spirometer Model No. 401, in a sitting position.

### Working Definition

Patients were classified on the basis of following

1) GINA Classification of asthma(2006)[5]

2) GOLD Classification of COPD (2006)[6]

### Statistical Analysis:

The collected data were entered and analyzed using IBM *SPSS* Statistics *V21*.0 (IBM Corporation, US). Summarizations of data for frequency distribution for variables of interest were carried out by using descriptive statistics such as mean, standard deviation and percentages. Chi-square test was employed to test the association between asthma with variables of interest. A p-value of <0.05 was considered to be statistically significant.

## Results and Observation

A total of 100 patients with obstructive airway

disease were included in the study. 50% of the patients were diagnosed with Asthma and 50% of the patients were diagnosed with COPD. In each of the diagnosed group, 17 cases were female and 33 cases were male. Of which Maximum percentage (64%) of patients belong to the age group (38-67 years) while minimum percentage (36%) of the patients belong to age group (18-37 years). Out of 100, 57% patients were non-smoker, 25% were smoker, and 18% were ex smokers.

Patients with COPD were classified on the basis of GOLD Classification of COPD. We found that 22% of COPD patients were in moderate category ($FEV_1$/FVC < 0.70 and $FEV_1$=0.50-0.80), 18% of patients were in severe category ($FEV_1$/FVC < 0.70, and $FEV_1$=0.30-0.50), and 14% of patients were classified as very severe ($FEV_1$/FVC < 0.70, and $FEV_1$ < 0.30). Only 6% of the patients had mild airflow limitation while 40% of COPD patients had no airflow limitation during spirometry.

Patients with Asthma were classified on the basis of GINA Classification of asthma. In our study group shows 46% belonged to intermittent persistent category of asthma ($FEV_1 \geq 0.80$ and PEF variability < 20%), and 20% were in mild persistent category ($FEV_1 \geq 0.80$ and PEF variability 20-30%). 16% of the patients were in moderate persistent category ($FEV_1$=0.60-0.80 and PEF variability >30%), while 18% of the patients were in very severe persistent category ($FEV_1 \leq 0.60$ and PEF variability >30%).

Mean ±SD of different parameters of spirometry in the study participants

**Table 1: Mean±SD of FVC, $FEV_1$, $FEV_1$/FVC, $FEF_{25-75\%}$, and PEFR in OAD patients (n=100)**

| Spirometric parameters | Minimum Value(%pred) | Maximum Value(%pred) | Mean(%pred) | Standard Deviation |
|---|---|---|---|---|
| FVC | 36.00 | 158.00 | 91.99 | 28.99 |
| FEV1 | 9.00 | 155.00 | 75.99 | 34.74 |
| FEV1/FVC | 11.00 | 115.00 | 80.16 | 19.98 |
| FEF25-75% | 6.00 | 138.00 | 46.19 | 31.51 |
| PEFR | 6.00 | 107.00 | 49.05 | 25.96 |

Table 1 shows the lung function test values of OAD patients as measured by spirometer. FVC, $FEV_1$, and $FEV_1$/FVC values were within normal limit. But the mean values of $FEF_{25-75\%}$ and PEFR were lower. The results suggest that smaller airways were more affected in obstructive airway disease.

**Association of OAD with different variables**

**Table 2: Association of Obstructive Airway Disease patients with age (N=100)**

| AGE GROUP ( in years) | OAD | | TOTAL N(%) | p-value* |
|---|---|---|---|---|
| | COPD N(%) | ASTHMA N(%) | | |
| 18-27 | 1(2) | 17(34) | 18(18) | |
| 28-37 | 3(6) | 15(30) | 18(18) | |
| 38-47 | 4(8) | 10(20) | 14(14) | |
| 48-57 | 14(28) | 4(8) | 18(18) | |
| 58-67 | 28(56) | 4(8) | 32(32) | <0.001 |
| TOTAL | 50 | 50 | 100 | |

*Chi-Square test

Table 2 shows that Asthma was associated with younger age group whereas COPD was associated with higher age group (p<0.001).

**Table 3: Association of Obstructive Airway Disease patients with smoking habit (N=100)**

| SMOKING HABIT | OAD | | TOTAL N(%) | p-value* |
| --- | --- | --- | --- | --- |
| | COPD N(%) | ASTHMA N(%) | | |
| SMOKER | 19(38) | 6(12) | 25(25) | |
| NON-SMOKER | 16(32) | 41(82) | 57(57) | <0.001 |
| EX-SMOKER | 15(30) | 3(6) | 18(18) | |
| TOTAL | 50 | 50 | 100 | |

*Chi-Square Test

Above table shows that obstructive airway disease was strongly associated with smoking (p<0.001).

**Table 4: Association of Obstructive Airway Disease patients with smoking pack-years (n=100)**

| SMOKING PACK-YEARS | OAD | | TOTAL N(%) | p-value* |
| --- | --- | --- | --- | --- |
| | COPD N(%) | ASTHMA N(%) | | |
| 0 | 16(32) | 41(82) | 57(57) | |
| Less than 100 | 3(6) | 4(8) | 7(7) | |
| 100-200 | 6(12) | 0(0) | 6(6) | <0.001 |
| 200-300 | 13(26) | 3(6) | 16(16) | |
| More than 300 | 12(24) | 2(4) | 14(14) | |
| TOTAL | 50 | 50 | 100 | |

*Chi-Square Test

Table 3 shows that the obstructive airway disease was strongly associated with smoking pack-years (p<0.001).

**Table 5: Association of Obstructive Airway Disease patients with duration of smoking (N=100)**

| SMOKING DURATION ( in years ) | OAD | | TOTAL | p-value* |
| --- | --- | --- | --- | --- |
| | COPD N(%) | ASTHMA N(%) | | |
| 0 | 16(32) | 41(82) | 57 | |
| Less than 5 | 1(2) | 2(4) | 3 | |
| 5-10 | 1(2) | 3(6) | 4 | |
| 10-15 | 1(2) | 0(0) | 1 | |
| 15-20 | 4(8) | 2(4) | 6 | <0.01 |
| 20-25 | 10(20) | 2(4) | 12 | |
| More than 25 | 17(34) | 0(0) | 17 | |
| TOTAL | 50 | 50 | 100 | |

*Chi-Square Test

Above table shows that the duration of smoking was associated with obstructive airway disease. Obstructive airway disease was more common among the patients who had long history of smoking years (p<0.01).

## Discussion

A total of 100 OAD patients were included of which 50 patients were suffering from asthma and 50 were COPD patients. Among the 100 OAD patients 66% were men and 34% were women. We found that Asthma was associated with younger age group whereas COPD was associated with higher age group (p<0.001).

The lung function test values of OAD patients showed that FVC, $FEV_1$, and $FEV_1/FVC$ values were within normal limits. But $FEF_{25-75\%}$, and PEFR were lower suggesting that smaller airways were affected in obstructive airway disease. In our study we found that OAD was strongly associated with smoking status(p<0.001), smoking pack-years(p=0.000), p<0.001 and smoking duration(p<0.001). The present study found that asthma was more common in men which is in agreement with Razi E et al[7] and Halvani A et al[8] studies. Also in common with their studies, the present study found that the 50 asthmatic patients were in the mean age±SD 34.08±12.83 years.[7,8]

The Latin American Project for the Investigation of Lung Disease (PLATINO), a population-based study of 5,315 subjects including 2,278 non smokers, and 3,036 current or ex-smokers showed that COPD was observed in 3.5% of those who had never smoked and in 7.5% of those who had smoked.[9] The study shows association between smoking and OAD which is in accordance with our study results.

Our findings are in line with the PLATINO study which showed that tobacco smoking was higher in subjects with COPD compared with those without COPD (19.4 vs 9.1 pack-years). The proportions of current smokers and ex-smokers were found to be higher in those with COPD than in those without the disease (current smokers: 36.0% vs 28.8%; ex-smokers: 32.5% vs 26.8%).[10] They also found that females and males with COPD reported higher to-bacco smoking compared with persons without COPD (females: 11.6 pack-years vs 6.0 pack-years; males: 26.5 pack- years vs 14.1 pack-years).[11]

Data from a study conducted by Llordes M et al[12] of subjects at least 45 years of age and with a history of smoking showed that subjects with COPD have a higher smoking index than those without COPD (52.6 pack-yearsvs 32.1 pack-years) which is similar to our study.

The Indian study on Epidemiology of Asthma, Respiratory symptoms and chronic bronchitis (INSEARCH) carried out in a population of 169575 adults over 15 years of age demonstrated odds ratio of 1.82 for cigarette and 2.87 for bidi smoking.[13] Also In a study by Agrawal S et al[14] which used data on self-reported asthma from the India's third National Family Health Survey (NFHS) reported odds ratios of 1.72 and 1.35 for women and men respectively leading to similar conclusion as our study.

## Conclusion

From the study results it is clear that Obstructive airway disease was strongly associated with smoking. Smoking cessation is the best and most effective solution to this problem. But smoking cessation is challenging so optimal approach for smoking cessation should be such as education advice, behavioural intervention along with drug therapy like nicotine replacement therapy with gums, patch or inhaler can be tried for better results. Other measures such as screening with spirometric tests in high risk individuals especially the smokers in age group of 40-55 should be considered to reduce the mortality and morbidity due to Obstructive airway disease.

## Limitations

Although smoking is a widely recognized risk factor for Obstructive airway disease, the interaction of other less traditional risk factors such biomass exposure, occupational pollutants and environmental toxins with smoking have not been studied.

# References

1. Weinberger SE, Rosen IM. Disturbances of respiratory function. In: Loscalzo J, editor. Harrison`s Pulmonary and Critical Care Medicine. New Delhi: McGraw-Hill; 2010. p. 25-35.

2. Asher MI, Montefort S, Björkstén B, Lai CK, Strachan DP, Weiland SK, et al. Worldwide time trends in the prevalence of symptoms of asthma, allergy rhinoconjunctivitis, and eczema in childhood: ISAAC phases one and three repeat multicountry cross-sectional surveys. Lancet 2006;368(9537):733-43.

3. Celli BR, MacNee W, Agusti AA, Anzueto A, Berg B, Buist AS, et al. Standards for the diagnosis and treatment of patients with COPD: a summary of the ATS/ERS position paper. Eur Respir J 2004;23(6):932-46.

4. Bellamy D. Spirometry in practice: a practical guide to using spirometry in primary care. 2nd ed. London: BTS COPD Consortium; 2005.

5. Koshak EA. Classification of asthma according to revised 2006 GINA: Evolution from severity to control. Ann Thorac Med 2007;2(2):45-6.

6. Global strategy for the diagnosis, management, and prevention of chronic obstructive pulmonary disease (2006). Available at: http://www.goldcopd.org. Accessed September 19, 2016.

7. Razi E, Ehteram H, Akbari H, Chavoshi V, Razi A. Evaluation of high-sensitivity C reactive protein in acute asthma. Tanaffos 2012;11(1):32-7.

8. Halvani A, Tahghighi F, Nadooshan HH. Evaluation of correlation between airway and serum inflammatory markers in asthmatic patients. Lung India 2012;29(2):143-6.

9. Perez-Padilla R, Fernandez R, Lopez Varela MV, Montes de Oca M,Muin˜o A, Ta´lamo C, et al. Airflow obstruction in never smokers in five Latin American cities: the PLATINO study. Arch Med Res2012;43(2):159-165

10. Lo´pez Varela MV, Montes de Oca M, Halbert R, Muin˜o A, Ta´lamoC, Pe´rez-Padilla R, et al. Comorbidities and health status in individuals with and without COPD in five Latin American cities: the PLATINO study. Arch Bronconeumol 2013;49(11):468-474.

11. Lopez Varela MV, Montes de Oca M, Halbert RJ, Muin˜o A, Perez-Padilla R, Ta´lamo C, et al. Gender related difference in COPD in five Latin American cities: The PLATINO study. Eur Respir J 2010;36(5):1034-1041.

12. Llorde´s M, Jae´n A, Almagro P, Heredia JL, Morera J, Soriano JB, Miravitlles M. Prevalence, risk factors and diagnostic accuracy of COPD among smokers in primary care. COPD 2015;12(4):404-412.

13. Jindal SK, Aggarwal AN, Gupta D, Agarwal R, Kumar R, Kaur T, Chaudhry K, Shah B. Indian study on epidemiology of asthma, respiratory symptoms and chronic bronchitis in adults (INSEARCH). Int J Tuberc Lung Dis 2012; 16:1270-77.

14. Jindal SK, Aggarwal AN, Gupta D, Agarwal R, Kumar R, Kaur T, Chaudhry K, Shah B. Indian study on epidemiology of asthma, respiratory symptoms and chronic bronchitis in adults (INSEARCH). Int J Tuberc Lung Dis 2012; 16:1270-77.

15. Agrawal S. Effect of indoor air pollution from biomass and solid fuel combustion on prevalence of self-reported asthma among adult men and women in India: findings from a nationwide large-scale cross-sectional survey. J Asthma 2012; 49:355-65

# Sensory Nerve Conduction Velocity in Normal Children Below Five Years of Age

**Md. Zabihullah[1], Dhirendra Kumar Agrawal[2], Tabassum Shahab[3]**

[1]*Assistant Professor, Department of Physiology, GS Medical College and Hospital, Pilkhuwa, Hapur, Uttar Pradesh, India, [2]Ex. Professor and Chairman, Department of Physiology, [3]Ex. Professor, Department of Paediatrics, Jawaharlal Nehru Medial College and Hospital, Aligarh, Uttar Pradesh, India*

## Abstract

**Background:** Peripheral nervous system development begins in prenatal period and continues after birth up to 5 years of age. As a result, Sensory nerve conduction velocity (SNCV) varies during first five years of life of children.

**Aims and Objectives:** The purpose of this study is evaluation of the SNCV of median, ulnar and sural nerve during the first five years of life.

**Material and Method:** SNCV of median, ulnar and sural nerves was measured antidromically in 104 normal under-five children. Statistical analysis of the results was done using SPSS 17.0 software.

**Results:** There was a progressive increase in SNCV of median, ulnar and sural nerves with age. SNCV of all three nerves showed significant positive correlation with age factor.

**Conclusion:** There is significant increase in SNCV of median, ulnar, and sural nerves with age in under-five children. Therefore, age-specific reference values of SNCV for the different nerves in children below 5 years of age are important to evaluate nerve injury or maturational deficit.

***Key Words:*** *Sensory Nerve Conduction Velocity (SNCV), Children, Peripheral nervous system*

## Introduction

Infancy and early childhood are crucial periods in the development of the central nervous system and this critical period in humans extends from the third month of pregnancy to 2 years of age.[1] Myelination of Peripheral nerve also occurs around this critical period, beginning at 15th week of gestation[2] and continuing throughout the first 2 to 5 years of life[3-4]. Axons also undergo progressive maturation during this period, reaching a maximum between ages two and five years.[5]

The ongoing maturation of peripheral nerves in children can be evaluated by measuring conduction velocity as it depends on degree of myelination, diameter of the nerve fibres and internodal distances. The relationship between motor nerve conduction velocity and age has been studied by many authors.[6-7] However, very few studies have been reported on normal sensory nerve conduction velocity (SNCV) during first years of life when nerves undergo maturation. Hence, we planned this study to evaluate the SNCV in normal infants and children below the age of 5 years.

## Materials and Method

The present prospective cross sectional study was conducted in Neurophysiology Laboratory, Department of Physiology, Jawaharlal Nehru Medical College, Aligarh Muslim University, Aligarh. The approval of Institutional Ethical Committee was obtained prior to the commencement of study. An informed consent was

**Corresponding Author:**
**Dr. Md. Zabihullah,**
Flat No. 404, Teaching Staff Quarters,
GS Medical College, Pilkhuwa, Hapur-245304,
Uttar Pradesh, India.
E-mail: drzabihullah81@gmail.com

taken from parents/ guardians of all children before conducting the test. Study sample consisted of 104 healthy children below the age of 5 years born at term, attending the Paediatrics outpatient department for regular check-ups. Subjects having any neuromuscular disorder were excluded from the study.

Sensory nerve conduction velocity test was performed on median, ulnar and sural nerves with Neuroperfect EMG/NCV/EP System. SNCV was measured antidromically by stimulating at a single stimulation site. The distance between the recording electrode and the stimulation site was measured. SNCV was calculated by dividing the distance between the stimulating and recording sites by the latency.[8]

## Statistical Analysis

Statistical analysis of the results was done using SPSS 17.0 software. One-way ANOVA tests and Pearson's correlation were used to analyse the results. A value of p less than 0.05 was considered statistically significant.

## Results

Sensory nerve conduction velocity (SNCV) of median, ulnar, and sural nerve was evaluated in 104 healthy children below 5 years of age. Statistical analysis was done by dividing the children in six age groups.

### Table 1: Age and sex distribution of study subjects

| Groups | Age (Months) | Sex | |
|---|---|---|---|
| | | Male | Female |
| Group 1 | 0 - 6 | 8 | 5 |
| Group 2 | >6 -12 | 9 | 7 |
| Group 3 | >12 - 24 | 9 | 10 |
| Group 4 | >24 -36 | 10 | 9 |
| Group 5 | >36 - 48 | 11 | 8 |
| Group 6 | >48 - 59 | 10 | 8 |

### Table 2: Comparison of mean sensory nerve conduction velocity (SNCV) of median nerve, ulnar nerve and sural nerve between the six age groups using one-way ANOVA

| Parameters | Mean ± SD | | | | | | p-Value |
|---|---|---|---|---|---|---|---|
| | Group 1 | Group 2 | Group 3 | Group 4 | Group 5 | Group 6 | |
| SNCV(m/s) (Median Nerve) | 29.33±2.45 | 37.73±3.16 | 43.52±2.60 | 46.57±2.71 | 48.73±3.15 | 50.95±1.77 | < 0.001* |
| SNCV(m/s) (Ulnar Nerve) | 29.90±2.29 | 38.54±3.08 | 43.74±2.01 | 47.54±1.91 | 49.03±2.12 | 51.28±2.21 | < 0.001* |
| SNCV(m/s) (Sural Nerve) | 29.45±2.97 | 34.92±2.40 | 39.54±2.05 | 45.14±2.43 | 48.59±3.03 | 50.78±1.85 | < 0.001* |

*p<0.05 significance. SD= Standard deviation

**Table 3: Pearson's correlation analysis of effects of age on sensory nerve conduction velocity (SNCV) in the study subjects**

| Nerve | Parameters | r- Value | p- Value |
|---|---|---|---|
| Median nerve | Sensory nerve conduction velocity | 0.863 | < 0.001 |
| Ulnar nerve | Sensory nerve conduction velocity | 0.874 | < 0.001 |
| Sural nerve | Sensory nerve conduction velocity | 0.927 | < 0.001 |

Correlation is significant at the 0.05 level

SNCV for median, ulnar and sural nerve for the six age groups are shown in table 2. SNCV was found to increase progressively from Group1 to Group6 for all the three nerves. One-Way ANOVA test was used for comparing the mean SNCV of median nerve, ulnar nerve and sural nerve between different age groups and was found statistically significant ($p < 0.05$).

Correlation between age and SNCV of median, ulnar, and sural nerve is shown in table 3. Analysis of correlation between age and SNCV was done using Pearson's correlation. A significant positive correlation was found between age and SNCV of median nerve (r-value= 0.863), ulnar nerve (r- value= 0.874), and sural nerve (r- value= 0.927).

## Discussion

Our study assessed the SNCV of median, ulnar, and sural nerve in normal children below the age of five years. The study subjects were divided into 6 age groups. The mean SNCV of all three nerves were found to increase progressively with age, highest being in group 6 comprising children of age group >48-59 months. Significant positive correlation was found between age and the SNCV for all the three nerves. Similar results have been reported by earlier investigators.[9-11]

SNCV depends on degree of myelination, distances between nodes of Ranvier, and diameter of the fibres.[12] Majority of myelin is assembled during the first few years of life. Very rapid myelin synthesis early in development has been demonstrated biochemically by earlier investigaors.[13] Actually Schwann cells begins myelination of nerves in fetal life and it is completed by 5 years of age.[14] Axons also mature simultaneously and reach a peak between 2 to 5 years of age.[5] There exists a direct relationship between the diameter of the nerve fibre and the thickness of the myelin sheath. Remodelling of the nodes of Ranvier also occurs in the first few years of children's life with internodal distances varying linearly with fibre diameter.[15] The significant increase in SNCV of median, ulnar, and sural nerves with age may be explained by varying maturational stages of these nerves. Therefore, age-specific reference values of SNCV for the different nerves in children below 5 years of age are important to evaluate nerve injury or maturational deficit.

## Conclusion

To conclude, SNCV of median, ulnar, and sural nerve increase progressively with age in children below five years of age. It may be due to the ongoing maturation of determinants of conduction velocity of peripheral nerves but future studies should also evaluate the SNCV in children above five years of age.

**Source of Support:** Nil

**Conflict of Interest:** None

## References

[1] Morgane PJ, Austin-LaFrance R, Bronzino J, et al. Prenatal malnutrition and development of the brain. Neurosci Biobehav Rev 1993;17(1):91-128.

[2] Gamble HJ, Breathnach AS. An electron-microscope study of human foetal peripheral nerves. J Anat 1965;99(Pt 3):573-84.

[3] Cottrell L. Histologic variations with age in apparently normal peripheral nerve trunks. Arch Neurol 1940;43(6):1138-50.

[4] Rexed B. Contribution to knowledge of postnatal development of peripheral nervous system in

man: study of bases and scope of systematic investigations into fibre size in peripheral nerves. Acta Psychiatry Neurol Scand Suppl 1944;33:1-206

[5] Thomas JE, Lambert EH. Ulnar nerve conduction velocity and H-reflex in infants and children. J Appl Physiol 1960;15:1-9.

[6] Ja Cho, Kyoung. (1987). Motor Nerve Conduction Velocity in Newborn Infants and Children. Yonsei medical journal. 28. 172-5. 10.3349/ymj.1987.28.3.172

[7] Guchhait B, GuptaS, Sau AK, Sen S. "Evaluation of Motor NCS Parameters between Upper and Lower Limbs and Their Pattern of Maturation in Infants and Children." IOSR Journal of Dental and Medical Sciences (IOSR-JDMS), vol. 18, no. 1, 2019, pp 52-57

[8] Misra UK, Kalita J. Principles of nerve conduction velocity. In: Clinical neurophysiology. 3rd edn. New Delhi: Elsevier India 2014:21-9.

[9] Gamstorp I, Shelburne SA : Peripheral sensory conduction in ulnar and median nerves of normal infants, children and adolescents, Acta Pediat Scand 1965; 54 : 309 – 313.

[10] García, A & Calleja, Jesús & M Antolín, F & Berciano, José. (2000). Peripheral motor and sensory nerve conduction studies in normal infants and children. Clinical neurophysiology: official journal of the International Federation of Clinical Neurophysiology. 111. 513-20. 10.1016/S1388-2457(99)00279-5.

[11] Guchhait B, Sen S, Ganguly G, Sinha M: Peripheral Motor and Sensory nerve conduction studies in normal infants and children in Eastern India, Journal of Dental and Medical sciences,2014(Aug);13(8):26-32

[12] Barrett KE, Barman SM, Boitano S, et al. Excitable tissue: nerve. In: Ganong's review of medical physiology. 25th edn. New Delhi: McGraw-Hill 2016:85-98.

[13] Quarles RH, Macklin WB, Morell P. Myelin formation, structure and biochemistry. Elsevier Publishing 1999: P. 67-8.

[14] Webster HF, Favilla JT. Development of peripheral nerve fibers. In: Dyck PJ, Thomas PK, Lambert EH, Bunge R, editors. Peripheral neuropathy, vol. 1, 2nd ed. Philadephia: WB Saunders 1984; 329-359.

[15] Vizoso, A.D. & Z YOUNG, J. (1948). Internode length and fibre diameter in developing and regenerating nerves. Journal of anatomy 1948; 82: 110-34.

# Evaluation of Differential Levels of Serum Interleukin-6 in Pre-Eclamptic and Normal Pregnancy Women

**Mohd. Nawaz[1], Manish Kumar Verma[2]**

*[1]Assistant Professor. Hind Institute of Medical Sciences, Safedabad, Barabanki, [2]Assistant Professor. Dr.Ram Manohar Lohia Institute of Medical Sciences, Vibhuti Khand, Gomti Nagar, Lucknow*

## Abstract

There has been an increase incidence of Pre eclampsia which is one of the important causes of maternal as well as foetal mortality and morbidity. The changing life style and various stresses in this fast growing world further increase the incidence of Pre eclampsia. The exact cause is still not known beside exploratory study but it seems a state of inflammatory and oxidative stress. Our aim was to evaluate the role of Interleukin 6 (IL6) and to compare its value in normal pregnancy and pre eclampsia. We found in this study that level of IL6 is significantly high in pre-eclamptic compare to normal pregnant women. Our study shows that Pre-eclampsia pregnancy is associated with an enhanced maternal inflammatory condition.

***Key words-*** *Pre eclampsia, Interleukin-6, Inflammatory, Oxidative stress, mortality, Pregnancy*

## Introduction

Pre-eclampsia(PE) is pregnancy-induced hypertension (PIH) of unknown etiology, a life threatening complication of pregnancy that is characterized by new onset of hypertension and proteinuria after 20 weeks of gestation. A clinical diagnosis of PE is confirmed by chart review using the newest guidelines, published in Pregnancy Hypertension [1](*i.e.*, ≥140 mmHg systolic blood pressure and/or ≥90 mmHg diastolic blood pressure with new onset proteinuria or one or more adverse/ severe conditions indicated by the new guidelines).

Preeclampsia is a significant public health problem in both developed and developing countries, causing maternal and perinatal morbidity and mortality globally .The prevalence of preeclampsia varies in different populations and in different ethnic groups [2].

Preeclampsia occurs in 5–8% of pregnancies worldwide, and is the second leading cause of direct

**Corresponding Author:**
**Dr Manish Kumar Verma**
Assistant Professor, Department of Physiology,
Dr Ram Manohar Lohia Institute of Medical Sciences,
Vibhuti Khand, Gomti Nagar, Lucknow -226010
Email id: drmanish24@gmail.com
Contact No. 7704079045

maternal and foetal deaths [3] .Till date there is neither adiagnostic test nor screening tool available for early identification of women at risk of preeclampsia.In the developing world, severe forms of preeclampsia and eclampsia are more common, ranging from a low of 4% of all deliveries to as high as 18% [4]. Around ten million women develop preeclampsia each year around the world and worldwide about 76,000 pregnant women die each year from preeclampsia and related hypertensive disorders and the number of babies who die from this disorders is around 500,000 per annum[5].

In India, a national wise cross sectional study was done by Sutapa Agrawal and Gagandeep k walia for Prevalence and risk factors for Pre-eclampsia in Indian women. They found the highest incidence in Tripura state 87.5% and lowest in Haryana 33%. Over all pregnancy-induced hypertension (PIH) accounts for about 50000 deaths per year [6]. Perinatal mortality is very high (5- fold) in hypertensive disorders during pregnancy and etiology is reduced uteroplacental circulation and remains one of the most common reasons for women to die during pregnancy[7].

The exact cause of pre-eclampsia is still not clearly understood, despite many attemptsto identify possible causes. It is proposed that multiple factors are involved in the initiation and progression of preeclampsia,

including maternal constitutional factors, inflammatory activation, endothelial dysfunction, anti- angiogenic factors [8]. In this study we see the role of Interleukin-6 in pre eclampsia patients and compare the level of same in normal pregnancy and Pre eclampsia.

Interleukin 6 (IL6) is a multifunctional pro and anti inflammatory cytokine [9] that was first reported in 1986[10] and plays important roles in acute and chronic inflammation and autoimmunity[11] and can modulate both pro and anti inflammatory events[9] . IL6 is widely expressed in the gestational tissues and in female reproductive tract and regulates functions in embryo implantation and placental development as well as the immune adaptations required to tolerate pregnancy. It seems that IL-6 is the very important and useful circulating marker of endothelial dysfunction [12]. Among the inflammatory markers that are increased in preeclampsia, Interleukin 6 has been consistently indicated to be present at higher serum concentrations in pre-eclamptic patients than in normal pregnant women [13,14] . Therefore we selected Interleukin 6 as the marker of inflammation in our study.

## Material and Method

### A.   Selection of Cases And Control

The study was conducted on thirty normotensive third trimester pregnant women (control) and thirty newly diagnosed cases of pre-eclampsia in third trimester pregnant women. Both control and cases were selected from Obstetrics and Gynaecology out- patient and antenatal clinic of J.N. Medical College Hospital. A.M.U., Aligarh between December 2014 and October 2016 having no complications like any systemic diseases (diabetes, essential hypertension, chronic renal failure, thyroid disorders, genital tract diseases, urinary tract infection and cervical or vaginal inflammation), since these conditions cause to oxidative stress and inflammation.Informed and written consent was taken from the cases and controls for participation in the study with approval of institutional Ethical Committee, J.N. Medical College Hospital, Aligarh.

### Sample Collection

Antecubital venous blood (5ml) was collected from the subjects in aseptic precautions. The blood was allowed to be kept at 2 to 8 degree Celsius in a refrigerator for one hour. After this serum was obtained by centrifugation at 3000 revolve/minute for 5 minutes. Fresh serum was used for analysis of IL-6.The blood sample was collected in plain vials for serum Interleukin-6 estimation. All the samples were centrifuged in the PG. Lab of physiology Dept. JNMCH AMU and serum was separated for study and stored in deep freezer.

### Serum Il-6 Estimation

Serum IL-6 estimation was done with the help of Human IL-6 ELISA kit as described in the user manual. The ELISA kit for IL6was supplied by Diaclone Research, Cedex, France.

## PRINCIPLE OF IL-6 ESTIMATION AND PRODUCT DESCRIPTION

The Diaclone Human IL-6 ELISA (Enzyme-Linked Immunosorbent Assay) kit is an in vitro enzyme-linked immunosorbent assay for the quantitative measurement of human IL-6 in serum, plasma, cell culture supernatants and urine. This assay employs an antibody specific for human IL6 coated on a 96-well plate. Standards and samples were pipetted into the wells and IL6present in a sample got bound to the wells by the immobilized antibody. The wells were washed and biotinylated antihuman IL6 antibody was added. After washing away unbound biotinylated antibody, HRP-conjugated streptavidin was pipetted to the wells. The wells were again washed, a TMB substrate solution was added to the wells and color developed in proportion to the amount of IL6 bound. The Stop Solution changes the color from blue to yellow, and the intensity of the color was measured at 450 nm by the Elisareader.

### Statistical Analysis of Data

Results were analysed using appropriate statistical tests with the help of Graph Pad Prism software.

- Mean

- Standard Deviation (S.D,)

- Un paired t-test

- P value.

## Observation and Result

The present study was done to know the level of Serum interleukin 6 in pre-eclamptic and normal pregnant women and then these values were compared between these two groups.

Serum IL-6 levels were observed in normal pregnant women and pre-eclampsia patients (Table-1). Mean IL-6 levels in normal pregnant women were found 2.447 ± 0.786pg/ml (N=30) and in pre-eclamptic patients were found 7.456 ± 2.647pg/ml (N=30). This increase in pre-eclampsia patients was found highly significant (P value < 0.01).

**Table-1: Mean levels of serum IL6 in normal pregnant women and pre-eclampsia patients**

| | Normal Pregnant (N = 30) | Pre-eclampsia (N = 30) | P value |
|---|---|---|---|
| Mean level of serum Interleukin-6(ng/ml) | 2.447 ± 0.786 | 7.457 ± 2.647* | <0.05 |

**\*P value < 0.01 which is significant**

## Discussion

The present study demonstrated an elevated mean concentration of inflammatory marker IL6 in the maternal plasma of preeclamptic patients when compared with healthy pregnant Women (Table-1 , Figure-1 ). This increase was highly significant (P value < 0.0001). Our finding indicated that raised level of above marker during pre eclampsia, might be used as markers of inflammation and endothelial dysfunction in the preeclamptic pregnancies. This increase in concentration of IL6 suggested that abnormal cytokine responses in mother might be involved in the pathogenesis of pre eclampsia. Our finding was similar to previous studies done by many investigators .In 2014 by Arjun Jain, et al[15],hypo perfusion induces a preeclampsia like inflammatory response .They found increase in the levels inflammatory cytokines and IL6 in pre eclampsia. In 2011 an study was done by Cristina et al[16] on Inflammatory Disturbances in Preeclampsia: Relationship between Maternal and Umbilical Cord Blood. They found that level of pro-inflammatory cytokines IL-6, significantly higher in Pre- eclampsia pregnant women as compared with normotensive pregnant women. Sharma *et al.[17]*demonstrated that IL-6 and TNF-a were higher in relation to normal pregnancy. These studies supported our study. But conflicting results were found regarding the role of IL-6 in pre eclampsia in different studies. In two different studies, Page et al[18]. and Tosun[19] et al. have shown that maternal serum levels of IL-6 significantly increased in pre-eclamptic patients rising in a way that higher levels are found in patients with severe compared to mild preeclampsia. Studies done by Desai et al. [20] and Maruo et al[21] suggested that IL6 interferes with endothelial cell function and contributes to the systemic endothelial activation and vascular damage suggesting that preeclampsia is associated with elevated plasma IL6 levels. These findings are consistent with a role for pro inflammatory cytokines in the genesis of preeclampsia. It has been seen that IL6 can cause improper angiogenesis [22], inhibit prostacyclin production by down regulating enzymatic activity of Cyclooxygenase and specifically enhances the endothelial cell permeability by altering the ultra structural distribution of tight junctions[22]. But conflicting results were found regarding the role of IL-6 in preeclampsia in different studies [23,24].

## Conclusion

We concluded from our study that probably increased lipid peroxiation of plasma membrane due to oxidative stress is an important factor in the pathogenesis of pre-eclampsia because lipid peroxides damage endothelial cells, produce vasoconstriction and inflammation. Increased IL6 level inhibits vascular remodeling and endothelial damage which may lead to pre-eclampsia. Thus possibly IL6 may be involved in the pathogenesis of pre-eclampsia.

As we have taken both study groups in their third trimester, to know the role of IL6 in early pregnancy and their implementation to control the incidence of pre eclampsia, is required further study.

## References

[1]  Magee LA, Pels A, et al. Canadian Hypertensive Disorders of Pregnancy Diagnosis, evaluation, and management of the hypertensive disorders of pregnancy. 2014.

[2]  Roberts JM, Lain KY et. al: Recent insights into the pathogenesis of pre-eclampsia. Placenta 2002,

[3]  Duley L. The Global Impact of Pre-eclampsia and Eclampsia. SeminPerinatol. 2009;33(3):130

[4]  Villar J, Say L, Gulmezoglu AM, Meraldi M, et. al; Eclampsia and preeclampsia: a health problem for 2000 years.

[5] Kuklina EV, et al. Hypertensive Disorders and Severe Obstetric Morbidity in the United States. ObstetGynecol 2009.

[6] Gupta P., O. P. Ghai. Common complications of pregnancy. In: P. Gupta, O. P. Ghai, eds. Preventive and Social Medicine. 2nd ed. New Delhi, India: CBS Publications; 1998.

[7] Roberts JM, Hubel CA. The two stage model of preeclampsia: variations on the theme. Placenta.2009

[8] Walker JJ: Pre-eclampsia. Lancet 2000; 356:1260–1265. National High Blood Pressure Education Programme Working Group Pediatrics, 2004

[9] Hayashi M, Ueda Y, et al. Interleukin6concentrations in the placentaand blood in normal pregnancies and preeclampsia. HormMetab Res. 2005;37:419 24.

[10] Hirano, T., Yasukawa, K., Harada, H., et al., 1986. Complementary DNA for a novel human interleukin (BSF-2) that induces B lymphocytes to produce immunoglobulin.

[11] Ishihara, K., Hirano, T., 2002. IL-6 in autoimmune disease and chronic inflammatory proliferative disease. Cytokine Growth Factor Rev. 13, 357–368

[12] Erzen B, Sabovic M, et al. Poredos P:pre-eclamptic pregnancies," Gynecological Endocrinology, vol. 24, no. 3, pp. 113–116, 2008.

[13] Singh A, Bhattacharjee J. et al. Role of inflammatory cytokines and eNOS gene polymorphism in pathophysiology of pre-eclampsia.Am J ReprodImmunol 2010.

[14] Kalinderis M, et al. Elevated serum levels of interleukin-6, interleukin-1beta and human chorionic gonadotropin in pre-eclampsia. Am J ReprodImmunol. 2011

[15] Arjunjain et. al on Hypoxic treatment of human paplacental perfusion 2014.

[16] Cristina et al. 2011. Ïnflammatory disturbances in pre-eclampsia.

[17] Sharma A, Satyam A, Sharma JB. Leptin, IL-10, and inflammatory markers (TNF-alpha, IL-6 and IL-8) in preeclamptic, normotensive pregnant and healthy non-pregnant women. Am J Reprod Immunol.2007;58(1):21–30.

[18] Nilsson MB, Langley RR, Fidler IJ. Interleukin6, secreted by human ovarian carcinoma cells, is a potent proangiogenic cytokine. Cancer Res. 2005 PubMed.

[19] Tosun M., H. Celik, B. Avci, E. et al., "Maternal and umbilical serum levels of interleukin-6, interleukin-8, and tumor necrosis factor-α in normal pregnancies and in pregnancies complicated by preeclampsia," 2010.

[20] Desai TR, Leeper NJ, Hynes KL, et. al, Interleukin6 causes endothelial barrier dysfunction via the protein kinase C pathway. J Surg Res. 2002;104:118–23.

[21] Maruo N, Morita I, Ishizaki Y, et al. Inhibitory effects of interleukin 6 on prostaglandin I2 production in cultured bovine vascular Maria IrenoRebelo, Jose Manuel, Nieto Villar. Heart rate variability analysis during normal and hypertensive pregnancy: Dissertation of University of Porto, 2010. . ;PubMed

[22] Desai TR, Leeper NJ, Hynes KL, et. al, Interleukin6 causes endothelial barrier dysfunction via the protein kinase C pathway. J Surg Res. 2002;104:118–23.

[23] S. Al-Othman, A. E. Omu, F. M. E. Diejomaoh, M. Al-Yatama, and F. Al-Qattan, "Differential levels of interleukin 6 in maternal and cord sera and placenta in women with pre-eclampsia," Gynecologic and Obstetric Investigation, vol. 52, no. 1, pp. 60–65, 2001

[24] M. Montagnana, G. Lippi, A. Albiero, G. L. Salvagno, M. Franchi, and G. C. Guidi, "Serum pro-inflammatory cytokines in physiological and pre-eclamptic pregnancies," Gynecological Endocrinology, vol. 24, no. 3, pp. 113–116, 2008

# 5-Alpha-Reductase 2 Deficiency in Newborns: A Review

**Narasimha Prasad Vijayashankar¹, Artem Artemev², Anastasia Pougno², Gopikumar MS³**

*¹Assistant Professor, Department of Pharmacology, Xavier University School of Medicine, Aruba, ²Basic Science Students, Xavier University School of Medicine, Aruba, ³Associate Professor, Department of Physiology, Xavier University School of Medicine, Aruba*

## Abstract

Disorders of sex development (DSD) are mostly associated with gender ambiguity at birth and poses a real challenge in diagnosis. 5 alpha reductase 2 deficiency (5AR2D) is one such DSD associated with deficiency of a key male sex hormone called as Dihydrotestosterone (DHT) due to lack of 5 alpha reductase type 2 enzyme which converts testosterone to Dihydrotestosterone. This hormone is very vital for the development of external genitalia during embryogenesis, and the deficiency leads to partial or abnormal male external genitalia in the affected neonate. 5AR2D is due to mutation in steroid-5- alpha-reductase, alpha polypeptide 2 (SRD5A2) genes on chromosome 2, band p23. Clinical presentation of these children vary from normal male genitalia to a completely female genitalia. 5AR2D is associated with deficiency of DHT and normal or increased testosterone levels. Most of the children with 5AR2D are raised as females and gender conversion to male happens at around puberty due to masculine and voice changes caused by testosterone. Early diagnosis is beneficial in treating this problem. Elevated testosterone: DHT ratio with administration of beta human chorionic Gonadotropin (HCG) is the gold standard test in diagnosis of this defect. Most patients of 5 alpha reductase 2 deficiency are considered infertile, but with recent advancements in assisted reproductive techniques a few 5AR2D have been successful in giving birth to their offsprings.

***Key words:*** *Disorders of sex development (DSD), 5 alpha reductase deficiency, Di-hydro-testosterone (DHT)*

## Introduction

DSD are a group of pediatric disorders associated with inconformity of gonads, chromosomes and hence external genitalia. DSD can be of two types based on the chromosomal status as 46, XX DSD and 46, XY DSD. In 46, XY DSD the primary sex organ is most of the affected individuals is testes, but associated with a deficiency in sex hormones or abnormal response of the individuals to the sex hormones. In majority of 46, XY DSD, if the Y chromosome is normal and the primary sex organ is tests, then the major mutations are seen in either androgen receptor or in SRD5A2 genes. SRD5A2 genes regulate the production of an important enzyme called as DHT which is involved in conversion of

testosterone to DHT. DHT plays a very important role during early embryogenesis by its role in development and differentiation of male external genitalia. Deficiency of DHT during first trimester in the developing male fetus will impact the development of external genitalia leading to ambiguous genitalia in new borns.

46, XY DSD, that too specifically 5AR2D pose a major challenge for the parents and physicians of such individuals, as they must navigate the disorder, correctly diagnose it and manage it to preserve the quality of life. Every effort should be made to accurately diagnose a newborn with ambiguous genitalia. The diagnosis and care for patients with DSD involves a holistic approach and discussion among endocrinologists, surgeons, psychologists, social workers and family members. The degree of patients' masculinity and penis size, response to androgens and treatments, social culture and norms, and quality of life are considered in the care [1, 2].

**Gender Ambiguity in 5AR2D:** The earliest description of the 5AR2D and its surgical repair dates

---

**Corresponding author:**
**Gopikumar MS,**
Associate Professor, department of Physiology, Xavier University School of Medicine, Aruba
No 5, Brebastraat, Ponton, Oranjestad, Aruba
Email: gshivaramaiahmd@xusom.com

to before century, made by the Greek historian Diodorus Siculus. It describes Callo, who is supposedly a married female, who developed a tumor in the genital area. When a physician operated him, he discovered male genital parts with a functioning urethra, which he was able to bring to surface. Thus, the individual became a fully functioning male named Callon [3].

In another study, a 21 year-old-woman, born out of a non-consanguineous marriage presented to physician with undeveloped breasts, ambiguous genitalia, deep voice, and primary amenorrhea. She had normal serum hormone profiles except for elevated total testosterone and an elevated testosterone to DHT ratio. Subsequently, on diagnosis with 5AR2D, she opted to remain as a female and underwent a surgery with hormonal replacement therapy using estrogen [4].

In another study, six Arab subjects were evaluated for male pseudohermaphroditism. Out of six subjects, one subject was pubertal, 2 subjects were prepubertal and the remaining three were post pubertal. All the subjects were raised as females till the point of evaluation. One of the subjects was also married as a female. After their evaluation 2 subjects changed their gender to male, two remained as female and the other two subjects decided to take up a conservative female role and equivocal sex status. [5]

**Genetics of 5ARD2:** The rare 5AR2D was first described in 1974 in patients with pseudovaginal perineo-scrotal hypospadias, micro phallus, and cryptorchid testes [6]. The condition is autosomal recessive and often found in males born in consanguineous parents in areas with high rates of inbreeding [7]. The major gene mutations in 5AR2D occur in the androgen receptor (AR) and steroid-5-alpha-reductase 2, (SRD5A2) genes [1]. Type 1 isoenzyme is encoded by the SRD5A1 gene located on chromosome 5p15, and expressed mainly in the liver and non-genital skin [6]. Type 2 isoenzyme is encoded by the SRD5A2 gene on chromosome 2, band p23, and is expressed at high levels in the prostate, the epididymis, seminal vesicles, genital skin, and its efficiency leads to male pseudohermaphroditism, with incomplete differentiation of male genitalia [2].

SRD5A2 was cloned and shown to contain five exons and four introns, which have over 65 known mutations, including point mutations, deletions, and insertions [1]. The most frequent polymorphism at exon 1, V89L, at the 89th codon, results in valine to leucine substitution, and

Decreases 5-alpha-reductase 2 activity by approximately 30%. V89L polymorphism is also more prevalent in patients with micropenis than in normal males [6, 8]. Results of one major study of 33 subjects confirm the predominance of homozygous (69.1%) *vs.* compound heterozygous mutations (30.9%), whereas deletions and disruptive mutations were relatively rare. Mutations was predominantly seen in exons 1 (35.8%) and 4 (21.7%), whereas exons 3 (11.3%) and 5 (9.4%) seemed to be rare[6, 9].

In another study involving 24 subjects with 5 alpha reductase 2 deficiency from Saudi Arabia born out of a consanguineous marriage. Direct sequencing of their SRD5A2 gene was done from their peripheral blood using PCR technique. Their gene analysis revealed 3 missense mutations (p.R246Q, p.P181L, p.A228T) in 11 subjects, one splice site mutation (IVS1-2A>G) in 11 subjects and 2 nonsense mutations (p.R227X and p.R103X) in 2 subjects [10].

In Spain, 146 index patients with 46, XY DSD were studied between 2002 and 2010. In these patients, gonads were confirmed as testes. SRD5A2 gene mutation was found in 9 index patients. One of the mutations (p.Y188CfsX9) has never been reported before, suggesting that there may be more mutations unrecognized yet [11].

**Epidemiology:** The largest 5AR2D affected kindred known are New Guinean, Dominican and Turkish. The New Guinean kindred's mutation was the first group described, with deletion of the 5α-reductase 2 gene of more than 20 kb resulting in a full loss of enzymatic activity. The Dominican kindred have a missense mutation in exon 5, substituting thymidine for cytosine and resulting in a substitution of tryptophan for arginine, resulting in reduction in binding of 5α-reductase-2 to its critical cofactor NADPH and a great decrease in enzymatic activity. The Turkish kindred have a single base deletion in exon 5, causing a frame shift mutation with complete loss of enzymatic activity [7].

5AR2D was also identified in populations not considered at risk of inbreeding, such as Europeans or North Americans, particularly in Quebec [12].

**Clinical Presentation:** 5-alpha- reductase 2 deficiency is characterized by external female phenotype at birth, with a shallow vaginal pouch, hypospadias, which is an opening of the urethra on the underside of the penis, and a clitoral-like phallus. Presence of bilateral

testes and normally developed internal male genitalia [7], but an underdeveloped prostate and a bifid scrotum. This is due to the inability to convert testosterone to dihydrotestosterone (DHT). DHT is responsible for the differentiation of genital tubercle and urogenital sinus into the prostate, urethra and external genitalia. Thus, male differentiation fails to occur despite high circulating testosterone levels [13, 14].

**Gender Choice and Fertility:** Most of the patients with 5 alpha reductase 2 deficiency are assigned female gender at birth and are raised as female child. At puberty, the surge in testosterone production prompts virilization, and enlargement of the genitalia with appearance of secondary sexual characteristics like development like muscle growth deepening of voice, pubic and axillary hair [1, 7]. Lack of breast development and amenorrhea prompts the suspicion of diagnosis at puberty for most of the subjects with 5 alpha reductase 2 deficiency. [6, 16].

Fertility is a challenge due to low sperm production, defective transformation of spermatogonia into spermatocytes, and the inability to liquefy semen due to a lack of prostate specific antigen and seminal fluid [7]. Two known extremes of the phenotype are an arginine substitution for glycine at position 35, resulting in a female phenotype with minor virilization at puberty, and a serine substitution for a glycine at position 196 resulting in a predominantly male phenotype [15]. Other mutations lead to subtle abnormalities in the enzyme may underlie some forms of commonly encountered urogenital birth defect in males as well as androgen-dependent disorders such as male pattern baldness, acne, hirsutism, and benign or cancerous growth of the prostate [14,15].

**Diagnosis:** Traditionally, the diagnosis relies on DHT measurement, but the results being equivocal can sometimes lead to misdiagnosing the condition. An alternative approach for diagnosis of 5AR2D is urinary steroid profiling (USP), a readily available testing option. In one study, of the 15 patients undergoing USP, all showed low ratios in at least 2 of the 4 pairs of 5-alpha- and 5-beta-reduced steroid metabolites. USP is considered as an ideal test for biochemical phenotyping in 5AR2D 3 months after the birth of the child. [12, 13]. Mutational analysis of *SRD5A2* by PCR and direct DNA sequencing of all 46, XY DSD patients is the key to the diagnosis of 5AR2D, as this is the ultimate tool for diagnosis. Making use of this wonderful technology for identifying the chromosomal abnormalities of DSD will

probably help to get a confirmed diagnosis [12, 16].

The biological diagnosis of 5AR2D is usually supported by an increase in the T/DHT ratio after human chorionic gonadotropin (HcG) stimulation testing, which is first line of diagnosis in infants and pre-pubertal children [1,6]. In some cases, however, the diagnosis cannot be ruled out by if there is no evidence of elevated T/DHT ratio after HCG stimulation. [6, 13] T/DHT ratio greater than 8.5 is suggestive of the deficiency and these babies and infants should be investigated further with molecular analysis of the 5-alpha-reductase-2 gene [12].

In another major study, over 72% of patients presented a ratio above 10, confirming that this ratio is a good indicator and could easily be used for screening patients [6]. Adult subjects deficient in 5α-reductase-2 activity also have elevated T/DHT ratios with normal-to-elevated testosterone levels compared with normal subjects. What distinguishes them from androgen-insensitive subjects who may also have abnormal T/DHT ratios is abnormal ratios of 5β- to 5α-reduced glucocorticoid and other steroid metabolites, indicating that this condition is a generalized defect in hepatic steroid 5α- metabolism [1, 17].

The definitive diagnosis of mutation causing 5AR2D can now be made by using special techniques like complementary DNA analysis by using the biopsy material of the tissues, peripheral blood and cultures obtained from fibroblasts [7, 18]

**Treatment options in 5AR2D**: Early diagnosis of 5AR2D is a key factor in its treatment. Early diagnosis allows the children to be raised as males at an early age and children can avoid embarrassment of gender conversion at a later age. In an interesting case study, two siblings, 3 years apart, both undergoing DHT treatment were successful in increasing phallic size. The siblings were born to Pakistani parents who were first cousins and practising Muslims. When the first child was born, early diagnosis and management of 5AR2D encouraged them to raise this baby as a male in accordance with the chromosomal sex. The younger sibling was born just when the older one was being diagnosed. [1].

DHT gel treatment for pediatric micropenis was studied with 76 pediatric patients with 46, XY DSD, who were treated with DHT gel (0.1-0.3 mg/kg/day) for three to six months. 22 of these patients had SRD5A2 mutations. The penis length of the patients significantly improved with DHT treatment. The length of the penis

increased significantly after long term treatment with DHT resulting in greater improvement. Therefore, the study shows that local application of DHT gel can promote penis growth effectively without systemic adverse reactions [1].

Often these individuals require long-term psychological support to aid in making an informed decision. If the parents chose to raise a female, gonadectomy and surgical correction of the external genitalia are indicated to avoid masculinization, with vaginoplasty for a healthy sexual life. An estrogen-only hormone replacement therapy must be administered these patients throughout life until the 50th year of age [3]. In New Guinea, where tribal culture is gender-segregated with very traditional male and female roles, rituals and rules, the upbringing and transition from female to male is very difficult for 5AR2D individuals [7].

## Conclusion

The multiples studies on 5AR2D underline the wide spectrum of phenotypes and biological profiles in patients with the condition. So far, no genotype- phenotype relationship could be determined [6]. Diagnosis is made by a combination of criteria such as DSD, virilization at puberty, and a marked increase in the T/DHT ratio after HCG testing. Due to often false negatives, DNA sequencing of the entire SRD5A2 gene is necessary. Physicians must be informed and aware of DSD and 5AR2D in particular, and make informed clinical decisions with the family of the affected individuals, due to the often-sensitive matter in a variety of communities and societies. 5AR2D should be considered in all XY newborns with ambiguous genitalia and normal or high plasma testosterone secretion [9]. The decisions made should consider the fact that at puberty, the individuals often become masculine and identify as males. Also, management decisions should aim at preserving functionality, quality of sexual and day-to-day life.

## References

1.    Fu X, Zhang W, Qu X. Correlation of androgen receptor and SRD5A2 gene mutations with pediatric hypospadias in 46, XY DSD children. Genetics and Molecular Research. 2016; 15(1).

2.    Odame I, Donaldson M, Wallace A, Cochran W, Smith P. Early diagnosis and management of 5 alpha-reductase deficiency. Archives of Disease in Childhood. 1992;67(6):720-723.

3.    Markantes G, Deligeoroglou E, Armeni A, Vasileiou V, Damoulari C, Mandrapilia A et al. Callo: The first known case of ambiguous genitalia to be surgically repaired in the history of Medicine, described by Diodorus Siculus. HORMONES. 2015;

4.    Maleki N, Kalantar Hormozi M, Iranparvar Alamdari M, Tavosi Z. 5-Alpha-Reductase 2 Deficiency in a Woman with Primary Amenorrhea. Case Reports in Endocrinology. 2013;2013:1-4.

5.    Al-Attia H. Male pseudohermaphroditism due to 5 alpha-reductase-2 deficiency in an Arab kindred. Postgraduate Medical Journal. 1997;73(866):802-807.

6.    Maimoun L, Philibert P, Cammas B, Audran F, Bouchard P, Fenichel P et al. Phenotypical, Biological, and Molecular Heterogeneity of 5α-Reductase Deficiency: An Extensive International Experience of 55 Patients. The Journal of Clinical Endocrinology & Metabolism. 2011;96(2):296-307.

7.    Kang H, Imperato-McGinley J, Zhu Y, Rosenwaks Z. The effect of 5α-reductase-2 deficiency on human fertility. Fertility and Sterility. 2014;101(2):310-316.

8.    Sasaki G, Ogata T, Ishii T, Kosaki K, Sato S, Homma K et al. Micropenis and the 5α-Reductase-2 (SRD5A2) Gene: Mutation and V89L Polymorphism Analysis in 81 Japanese Patients. The Journal of Clinical Endocrinology & Metabolism. 2003;88(7):3431-3436.

9.    Maimoun L, Philibert P, Cammas B, Audran F, Pienkowski C, Kurtz F et al. Undervirilization in XY newborns may hide a 5α-reductase deficiency: report of three new SRD5A2 gene mutations. International Journal of Andrology. 2010;33(6):841-847.

10.    Alswailem M, Alzahrani O, Alghofaili L, Qasem E, Almohanaa M, Alsagheir A et al. Molecular genetics and phenotype/genotype correlation of 5-α reductase deficiency in a highly consanguineous

population. Endocrine. 2018;63(2):361-368.

11. Fernández-Cancio M, Audí L, Andaluz P, Torán N, Piró C, Albisu M et al. SRD5A2 gene mutations and polymorphisms in Spanish 46,XY patients with a disorder of sex differentiation. International Journal of Andrology. 2011;34(6pt2):e526-e535.

12. Chan A, But B, Lee C, Lam Y, Ng K, Tung J et al. Diagnosis of 5 -Reductase 2 Deficiency: Is Measurement of Dihydrotestosterone Essential?. Clinical Chemistry. 2013;59(5):798-806.

13. Al-Jurayyan N. Ambiguous Genitalia: Two Decades of Experience. Annals of Saudi Medicine. 2011;31(3):284-288.

14. Akcay T, Fernandez-Cancio M, Turan S, Güran T, Audi L, Bereket A. ARandSRD5A2gene mutations in a series of 51 Turkish 46,XY DSD children with a clinical diagnosis of androgen insensitivity. Andrology. 2014;2(4):572-578.

15. Thigpen A, Davis D, Milatovich A, Mendonca B, Imperato-McGinley J, Griffin J et al. Molecular genetics of steroid 5 alpha-reductase 2 deficiency. Journal of Clinical Investigation. 1992;90(3):799-809.

16. Deeb A, Suwaidi H, Ibukunoluwa F, Attia S. Phenotype, Sex of Rearing, Gender Re-Assignment, and Response to Medical Treatment in Extended Family Members with a Novel Mutation in the SRD5A2 Gene. Journal of Clinical Research in Pediatric Endocrinology. 2016;8(2):236-240.

17. Fernández-Cancio M, Nistal M, Gracia R, Molina M, Tovar J, Esteban C et al. Compound Heterozygous Mutations in theSRD5A2Gene Exon 4 in a Male Pseudohermaphrodite Patient of Chinese Origin. Journal of Andrology. 2004;25(3):412-416.

18. Ng W, Taylor N, Hughes I, Taylor J, Ransley P, Grant D. 5 alpha-reductase deficiency without hypospadias. Archives of Disease in Childhood. 1990;65(10):1166-1167.

# Somatosensory Evoked High Frequency Oscillations in a Homogeneous Population of Drug Naive Migraineurs

**Ravi Prakash[1], Sowmya. P[2]**

*[1]Director, Veditha Mind Care Clinic, Bangalore, Karnataka, [2]Assistant Professor, Department of Physiology, Bowring and Lady Curzon Medical College and Research Institute, Bangalore, Karnataka*

## Abstract

Introduction: High band pass filtering of evoked potentials have produced the results of decreased early High frequency oscillations (HFOs) in previous studies of migraine patients. However, in most of these studies, the study population has been heterogeneous. The only study till date which used homogenous drug naive group of patients has actually produced contradictory results to the previous studies showing. Aim: We aimed to clarify the status of HFO amplitudes and latencies in drug-naive newly diagnosed patients. Methods: In the present study, we evaluated the 20 newly diagnosed and drug naive migraine patients using electrophysiological assessments using Somatosensory evoked potentials (SSEPs) and compared their SSEP parameters with those of the healthy age and gender matched normal controls. Median nerve SSEP was obtained by standard protocols. High frequency oscillations were extracted from the broad-band evoked potentials by digitally filtering using high pass filtering at 450Hz-1KHz. Early HFOs were identified when occurred before the N20 peak. Results: No differences were observed in the broad-band SSEPs i.e., N20 amplitudes and latencies. The maximal peak to peak amplitudes in the drug naïve migraineurs in between the attacks for the early HFOs (occurring before the N20 peak) were significantly smaller than the normal controls (p=0.046). The number of negative peaks were also fewer in the migraineurs in a statistically non-significant way. Discussion: This is the first HFO study on a homogenous population of migraineurs which shows decreased early HFOs, thus implicating weaker thalamocortical activity and contradicting the results by Lai et al[1].

*Keywords: SSEP, HFO, drug naïve migraineurs, thalamocortical activity*

## Introduction

Migraine is one of the most common neurological disorders with a prevalences of nearly 8% and 12-15% in males and females respectively[2]. It is a disabling condition characterized by excruciating headaches which vary widely in duration and intensity and almost universally the condition causes severe interference with daily life activities of the patient. The headaches are characterised by throbbing in nature and unilateral in location along with the additional features of nausea, sensitivity to light, sound, and exacerbation with body and head movements. The International Classification of Headache Disorders (ICHD) has provided the operational clinical criteria for migraine diagnosis which includes the migraine with and without aura. Inspite of its high prevalence and being studied scientifically for several decades now, the etiopathogenesis of migraine remains poorly understood. Recently the theoretical standpoint for the etiopathogenesis of headaches has shifted from the vascular hypersensitivity theory to the cortical hyperactivity theory proposing that it is the abnormal cortical excitability leading to enhanced pain perception.

Electrophysiological methods have proved to be vital for assessments in such conditions because they have allowed in vivo measurements of the migraineur's cortical responses to various sensory stimuli. Especially, the SSEPs have been studied for exploring the neurophysiological abnormalities in migraine. Some very important electrophysiological findings have been noted in migraineurs in-between the attacks. The most well established electrophysiological abnormality

**Corresponding author:**
**Sowmya. P**
Email id: sowmya.panju@gmail.com

in migraine has been the lack of adequate habituation response of somatosensory cortex to stimuli meaning that the migraine patients fail to show a decremental response to repeated stimuli[3]. This finding led the researchers in this field to search and speculate for the possible neurophysiological mechanisms underlying the failure of this habituation response. It has been hypothesized that pre-activation excitability of cortex could be the etiology of this lack of habituation[4]. However, the mechanisms underlying this pre-activation excitability remain a matter of speculation. Among the hypotheses, altered activity of thalamo-cortical connections leading to cortical inhibition has been proposed [3,5] which subsequently result in the lack of habituation response. This abnormal information processing has been proposed to be responsible for the hypersensitivity of cortex leading to enhanced perception of pain.

In order to develop a clearer comprehension and clarity of the mechanisms underlying the deficit seen in habituation in migraine, more sophisticated analysis of evoked potentials (especially the somatosensory evoked potentials) have been applied. Analysing the HFOs in evoked potentials is one such sophisticated method. HFOs are the low amplitude (around 100 nVs) high frequency (>600 Hz) waves embedded within the broad-band fluctuations obtained in the usual fluctuations of the SSEPs. These can be elicited by using digital high-pass filtering (>400 Hz) during the recording or offline analysis of various evoked potentials. HFOs in SSEPs are classified into two types according to their theoretical sources of origin. Whereas the early HFOs (those occurring before the peak of N20) are thought to reflect spike activity in the activation from thalamo-cortical cholinergic fibres, the late HFOs (following the peak of N 20) originate theoretically due to the cortical inhibitory GABAergic interneuron[6]. A study of SSEP HFOs in migraine therefore theoretically explores the thalamo-cortical activation as well as the intrinsic inhibition of the parietal cortex simultaneously. In recent years, few studies have been conducted for evaluating the early and late HFOs in migraine. Mostly, HFOs in migraineurs have been studied on the SSEPs. Earliest study with this methodology was conducted by [7] who found that HFOs in SSEPs were decreased generally in migraine patients in between the attacks. Subsequently this finding was reproduced by few other studies [3,9]. However, the conclusion from these studies is far from universally accepted. Whereas a majority of such studies show a decreased HFOs (maximal amplitude and area

under curve) in migraineurs as compared to healthy controls [3,9] some studies actually show the opposite findings with increased HFO amplitudes between migraine as compared to controls[1]. The study [1] raises some important issues because it was the only study that we could find which was conducted on untreated migraine patients. Therefore it raises the speculation that untreated migraine patients could be having a hyperexcitable rather than a hypoexcitable cortex between the attacks. Therefore, the treatment naive condition of such patients could be the reason that the HFO findings were different from other patient samples who are undergoing treatment or whose clinical pictures are improving [3,9]. This raises the question whether the earlier findings of low HFO amplitudes between the attacks could be because of inhomogeneity in the sample population. Especially, the effects of chornicity of migraine or treatment related improvements could have influenced other findings. It has been observed that the changes in the clinical status in the course of migraine is associated with changes in HFOs with the amplitudes decreasing with worsening of symptoms and increasing with improvement in the symptoms[8]. In another recent study the maximal peak to peak amplitudes in Chronic migraine patients were similar to the normal subjects whereas the maximal amplitudes in interictal periods of migraine were significantly smaller than normal subjects[9]. Therefore, the variations in findings in these previous studies could have been due to inhomogenous population of samples with regards to chronicity and clinical status. We aimed to control these variables by selecting a sample of young and newly diagnosed patients suffering from migraine who were treatment naive. We conducted broad band SSEPs as well as high band filtering to obtain early (presynaptic) HFOs in a homogenous population.

## Methodology

**Sample:** In present study, we evaluated 20 patients newly diagnosed as migraine and compared them with healthy age and gender matched normal controls. The patients were recruited from the out patients department of the neurology department. All the patients were newly diagnosed with migraine. The diagnosis was made as per the guidelines provided by the International classification of headache disorders 3rd edition[12]. Subjects were excluded if they fulfilled the criteria of chronic migraine according to the ICHD criteria or any other neurological condition. The electrophysiological examination was conducted on the next day of diagnosis before beginning

with the medications. Therefore during the examination, the patients were drug-naive. It was also assured that the patients did not receive any specific anti migraine medications in past. Even when they had been taking non-specific analgesics, it was not on a regular basis to avoid the inclusion of medication overuse headache. All the subjects had given written informed consent and the study was approved by the institutional ethical committee.

### Somatosensory evoked potentials:

Stimulus delivery: Electrical stimuli in the form of constant current duration of 0.2 ms width square wave pulses were applied to the right wrist for stimulation of the right median nerve. The stimulus were delivered at a regular interval with a stimulation frequency of 4.2 Hz. The anode was placed in strategic way at the wrist such that it overlapped the median nerve. The location was just at the proximal palmar crease. The cathode was placed at a location of 3 cm proximal to the anode. The stimulus intensity was calculated using the motor threshold and was set slightly above the same. The placement of recording electrodes was done at C3' and Fz as per the guidelines of the International 10-20 System. The reference electrode was placed on the forehead. Impedances of the electrodes were kept below the levels of 10 kΩ. A trial of 500 stimuli was applied and the consequent responses were used for averaging SEP. Subjects were asked to sit in a relaxed posture on a comfortable chair. The room was adequately and the subject was asked to keep their eyes opened with their attention fixed on the wrist movement. Fifty milliseconds duration of the post-stimulus period were sampled at a frequency of 5000 Hz.

*Broad band and High frequency oscillation analysis:* The usual broad-band SEP responses were obtained by using a band-pass filter of the range from 0.5-2000 Hz. The HFOs were separated and isolated from the underlying N20 primary cortical responses, by offline analysis. HFOs were extracted from the broad band recording by altering the band frequency settings at 450 Hz and 1 KHz which made evident the small amplitude waves existing between this frequency range (Fig.1). The HFOs were marked only for the oscillations which were at least double the amplitude of the background noise[1]. Two separate bursts of HFOs could be identified in most of the SSEP response traces: the first burst, also known as the early burst was the one occuring before the onset of N20 peak which usually began with the initiation of

the ascending limb of N20 or just before it. In literature, this has been referred to as the early or pre-synaptic HFOs (Coppola et al 2005). In general, the frequency of oscillations was observed to be higher in the first HFO burst as compared to the second burst. For marking these HFOs, we included the wavelets occurring before the peak of N20 as the early HFOs and those occurring after the peak of N20 as the late HFOs. We chose to a 5ms gap before and after the onset of this 5 ms because sometimes the onset and offsets of HFOs are controversial as they may originate before the ascending limb/onset of N20. Therefore, to maintain uniformity, we chose to have a fixed time interval.

In HFOs, we measured the latency of the negative oscillatory maximum, the number of negative peaks and the maximum peak-to peak amplitudes. The latency was automatically marked by the software on the X-axis and the negative peaks were manually counted. All measurements were performed separately on the two HFO bursts-early and late. Standardised measures of latencies and peak to peak amplitudes were used to calculate the parameters of Latencies of the negative oscillatory maxima and maximum peak-to-peak amplitudes for the early HFO-components defined as those peaking before the peak of N20 waveform. Latencies and amplitudes of the late HFO subcomponents were computed in the same way as for the early HFO burst peaking after the N20 maximum[6]

Statistical analysis: For statistical analysis, SPSS 16th version was used. The data was expressed as means and standard deviations. Independent samples t test was applied to find the difference between broad-band as well as the HFOs variables between the cases and the controls. For categorical variables, chi-square test was applied.

### Results

The results have been expressed in Tables 1,2 and 3. Table No.1 shows the comparison of the sociodemographic variables between the cases and controls. All the subjects of cases and control groups were right handed. There were no statistical differences between the two groups on the parameters of age and gender and height. Controlling these four variables (Handedness, age, gender and height) was important as these are known to influence the SSEP waveform parameters[10].The comparison of N20 wave form amplitudes and latencies obtained from broad

band filtering of SSEP have been presented in Table No.2. The results show that there were no statistical differences between the amplitudes and the latencies between the groups. Table No.3 show the comparison of HFOs parameters between the groups. We found statistically significant difference only in the maximum peak to peak amplitudes of early HFOs such that the amplitude was lesser in the migraine patients (0.07± 0.049 μV) as compared to the normal controls (0.12 ± 0.10 μV). The number of negative peaks were also lesser in the migraine patients (2.73 ± 0.71) as compared to the normal controls (3.07 ± 0.76).

### Table No.1: Sociodemographic Variables of Cases and Controls

|  | Cases | | Controls | | |
|---|---|---|---|---|---|
|  | Mean/N | SD/% | Mean /N | SD/% | p-value |
| Age (Years) | 24.25 | 5.92 | 25.05 | 5.57 | 0.66 |
| Gender (Males) | 7 | 17.9 | 9 | 23.1 | 0.32 |
| (Females) | 13 | 33.3 | 10 | 25.6 | |
| Height (cms) | 164.89 | 4.15 | 166.78 | 4.97 | 0.34 |
| Handedness (Right) | 20 | 100% | 20 | 100% | 1 |
| (Left) | 0 | | 0 | | |
| Education status | | | | | |
| Undergraduate | 0 | 0 | 0 | 0 | 0.11 |
| Graduate | 14 | 35.9 | 8 | 20.5 | |
| Postgraduate | 6 | 15.4 | 11 | 28.2 | |
| Employment status | | | | | |
| Student | 14 | 35.9 | 14 | 71.8 | 0.27 |
| Unemployed | 4 | 10.3 | 1 | 12.8 | |
| Employed | 2 | 5.1 | 4 | 15.4 | |

No significant differences were observed among the cases and controls over the variables of age, gender, educational status and employment status

### Table No.2: Broad Band Pass Filtering of SSEP Showing N20 Amplitudes and Latencies

|  | CASES | | CONTROLS | | |
|---|---|---|---|---|---|
|  | MEAN | SD | MEAN | SD | p-value |
| N20 AMPLITUDE (μV) | -1.65 | 1.39 | -2.27 | 1.89 | 0.49 |
| N20 LATENCY (ms) | 18.64 | 1.30 | 18.51 | 1.14 | 0.81 |

No significant differences were observed in the broad band variables of N20 in both the amplitude and latency

**Table No.3: Electrophysiological parameters of high frequency oscillations of SSEP**

| | CASES | | CONTROLS | | |
|---|---|---|---|---|---|
| | MEAN | SD | MEAN | SD | p-value |
| LATENCY OF MAXIMUM NEGATIVE PEAK (ms) | 16.20 | 1.54 | 16.08 | 1.92 | 0.82 |
| NUMBER OF NEGATIVE PEAKS | 2.73 | .71 | 3.07 | .76 | 0.14 |
| MAXIMUM PEAK TO PEAK AMPLITUDE (μV) | .07 | .049 | .12 | .10 | 0.046 |

No significant differences were observed in the latency of maximum negative peak. A trend difference was observed in the number of negative peaks with lesser number of peaks in migraine patients as compared to healthy controls. The maximum peak to peak amplitude was significantly reduced in cases as compared to controls (p=0.046)

## Discussion

In this small study, we aimed to evaluate the HFOs in newly diagnosed drug-naïve migraine patients. But more importantly, this study was aimed to investigate if the HFO related findings in earlier studies were affected by the inhomogenity of their sample populations. In order to make our study sample homogeneous, we eliminated the variations in chronicity, age and drug treatment status across the patient group. Ours is the first study conducted with this level of controlling of patient parameters. The mean age of our patients group was 25 years showing that they were in the early stages of migraine. This was important in our study to eliminate the element of chronicity for which we also excluded the subjects fulfilling the ICHD criteria for chronic migraine. Such an age-specific assessment was necessary because it has been observed that the clinical features of migraine change with age[11]. Other sociodemographic conditions like gender were also controlled so that there were no significant differences between the two groups (Table No.1). Our study findings are important to clarify the exact status of HFOs in newly diagnosed drug naive patients. Whereas most of the studies have reported decreased HFO amplitudes in the interictal period of migraine [3,9], the patients included in these studies have been heterogenous in terms of chronicity and clinical condition. On the other hand the only study which recruited homogenous drug naive patients with lack of chronicity showed contradictory results by eliciting high HFO amplitudes in between the attacks[1]. This raises the speculation that if the drug naivety and lack of chronicity the patient's group were the reasons for this contradictory finding[1]. Therefore we aimed to clarify these findings. In our study we controlled these factors by only recruiting newly diagnosed patients before being started on any anti-migraine medications. The importance of recruiting young patients has been discussed above.

However, inspite of our sample being similar to that of Lai et al (2011), our results are in sharp contrast to theirs and are in line with previous study results by Coppolla et al (2005;2013). We found that HFO amplitudes in migraineurs were smaller than the healthy controls. Additionally we also found lesser number of negative peaks in early HFOs in our migraine patients (Although not statistically significant, p=0.15) again strengthening the idea that the early HFOs are weakened in migraine patients in between the attacks. Our results therefore echo the idea that in between the attacks, activity in the thalamocortical excitatory cholinergic afferents are decreased, thus indicating subcortical hypoexcitability [3,9]. On the basis of our results we therefore conclude that in drug naive and non-chronic migraine patients, the thalamocortical projection activity is less leading to a state of hypoexcitability of cortex. However, we recommend that before any

electrophysiological assessment, it is important to keep the migraine patient group as homogeneous and as well defined as possible because the demographic related or treatment related variables can very well affect the clinical condition of migraine thereby causing errors in the assessment findings.

## Conclusion

Our study clarifies the prevailing confusion regarding status of SSEP HFO amplitudes in newly diagnosed drug naive patients by showing that even in homogenous drug naïve patients, the early HFO amplitudes are smaller. Thus we conclude that in drug naïve young patients suffering from migraine, the HFO negative peaks are smaller and lesser in number which is indicative of hypexcitability of cortex. In future studies, homogeneity of the population should be ensured to avoid any interference on the results arising from the disease related or treatment related confounding variables.

**Conflict of Interest:** 'The author(s) declare(s) that there is no conflict of interest'

**Source of Funding:** Self

**Ethical Clearance:** Obtained from the Institutional Ethical committee.

## References

1. Lai KL, Liao KK, Fuh JL, Wang SJ. Subcortical hyperexcitability in migraineurs: a high-frequency oscillation study. Can J Neurol Sci. 2011 Mar;38(2):309-16.

2. Diener H C, Katsarava Z, Limmroth V. Current diagnosis and treatment of migraine. Schmerz 2008, 22 (Suppl. 1), 51–58.

3. Coppola G, Pierelli F, Schoenen J. Habituation and migraine. Neurobiology of Learning and Memory 92 (2009) 249–259

4. Schoenen J. Deficient habituation of evoked cortical potentials in migraine: a link between brain biology, behavior and trigeminovascular activation?Biomed Pharmacother 1996; 50: 71–8.

5. Tommaso M, Ambrosini A, Brighina F, Coppola G, Perrotta A, et al. Altered processing of sensory stimuli in patients with migraine. Nature Reviews Neurology 10, 144–155 (2014)

6. Gobbelé R, Waberski TD, Thyerlei D, Thissen M, Darvas F, Klostermann F, Curio G, Buchner H. Functional dissociation of a subcortical and cortical component of high-frequency oscillations in human somatosensory evoked potentials by motor interference. Neurosci Lett. 2003 Oct 23;350(2):97-100.

7. Sakuma K, Takeshima T, Ishizaki K, Nakashima K. Somatosensory evoked high-frequency oscillations in migraine patients. Clin Neurophysiol. 2004;115(8):1857-62.

8. Restuccia D, Vollono C, Del Piero I, Martucci L, Zanini S. Somatosensory High Frequency Oscillations reflect clinical fluctuations in migraine. Clin Neurophysiol. 2012 Oct;123(10):2050-6.

9. Coppola G, Iacovelli E, Bracaglia M, Serrao M, Lorenzo C D and Pierelli F. Electrophysiological correlates of episodic migraine chronification: evidence for thalamic involvement. Coppola et al. The Journal of Headache and Pain 2013, 14:76

10. Poornima S, Ali SS, Balaji PA, Shankar V, Kutty K. Median nerve somatosensory evoked potentials in medical students: Normative data. Adv Biomed Res 2013, 2:56: 1-6

11. Martins K, Bigal M E, Bordini CA, Speciali JG: Migraine in the elderly. Headache 2006, 46:312–316.

12. Headache Classification Subcommittee of the International Headache Society. The international classification of headache disorders. Cephalalgia 2004, 24 (Suppl. 1), 9–160.

# A Comparitive Study: Autonomic Functions in Cerebral Palsy Individuals and Their Siblings

**Sowmya. P[1], Govindaraj.J [2]**

[1]*Assistant Professor, Department of Physiology, Bowring and lady Curzon medical college and research Institute, Bangalore, Karnataka, [2]Assistant Professor, Department of Physiology, Vijayanagar Institute of Medical Sciences, Ballari, Karnataka*

## Abstract

**Introduction**: It is defined as an "umbrella term covering a group of non-progressive, but often changing, motor impairment syndromes secondary to lesions or anomalies of the brain arising in the early stages of its development"[1] primarily leading to a disorder of movement and posture. **Objective:** To assess autonomic functions in cerebral palsy individuals and their siblings. **Method:** Twenty cerebral palsy and sibling volunteers with no neurological damage were recruited for the study. Heart rate variability was used to assess the autonomic function. HRV was recorded in supine position for 5 minutes under quiet, calm conditions. Time domain parameters and frequency domain parameters were analyzed. **Results:** There was no statistically significant difference in any of the HRV parameters. There was no significant difference in HRV parameters in children with CP and healthy children indicating a normal sympatho-vagal balance. **Conclusion:** The presence of normal sympatho vagal balance in CP predicts that patients with CP have the same predilection as the general population to abnormalities associated with sympatho-vagal balance.

***Key Words***: *Cerebral palsy, HRV, sympathetic activity, parasympathetic activity, sympatho vagal balance.*

## Introduction

Cerebral palsy (CP) is defined as an "umbrella term covering a group of non- progressive, but often changing, motor impairment syndromes secondary to lesions or anomalies of the brain arising in the early stages of its development".[1] The brain is the most complex organ in the human body and any injury may bring disturbances of function, both along the line of motor control and sensation. Cerebral palsy (CP) is the major physical disability affecting the functional development of children (Boyle et al 1994, Thorogood 2001), characterized by inability to control motor functions, this problem with movement and posture makes certain activities difficult, and has the potential to have a negative effect on the overall development of a child by affecting the child's ability to explore, speak, learn,

and become independent.[2] By convention, brain injuries occurring at any stage antenatally and postnatally till the age of 2 years are included in the definition of CP.[3,4] 75% - 80% of the cases are due to prenatal injury. Less than 10% is due to significant birth trauma or asphyxia.[5]

Autonomic Nervous System(ANS) has two main branches: the sympathetic and parasympathetic nervous system. The normal variability in HR is due to autonomic neural regulation of the heart and the circulatory system. Balance between sympathetic nervous system (SNS) and parasympathetic nervous system (PNS) controls the HR. Increased SNS or diminished activity of PNS results in cardio-acceleration. Conversely, a low SNS activity or a high PNS activity causes cardio-deceleration. Heart rate variability (HRV) is used as a non - invasive tool to assess autonomic functions. It can be measured non-invasively by using changes in an heart's response as a proxy for changes in ANS activity. HRV refers to beat-to-beat alterations in heart rate.[6] The parasympathetic influence on heart rate is mediated by the vagus nerve.[7]

Variations in heart period are largely dependent on vagal modulation.[8] The vagal and sympathetic activity

**Corresponding author:**
**Dr. Govindaraj.J**
Assistant Professor, Department of Physiology,
Vijayanagar Institute of Medical Sciences
Email id: sowmya.panju@gmail.com

constantly interact.[6] HRV is used to demonstrate the development of tonic vagal influence on the heart.[9]

The peak of QRS complex is R wave, the duration between two consecutive R wave peaks is termed the RR interval. It is also called NN intervals when the heart is beating at sinus rhythm. This difference is assessed by calculating both the time domain and frequency domain of HRV using Power Spectral Density (PSD). Time intervals between each successive normal QRS complex are first determined.All abnormal beats not generated by sinus node depolarizations are eliminated from the HRV analysis.

## Objectives of the Study

To compare any disturbances in autonomic functions in children with cerebral palsy and their sibling volunteers.

## Material and Method

HRV was used to determine autonomic function in twenty five known cerebral palsy cases (mild to moderate CP)of age group 6-16 years (study). They were recruited from schools for individuals with special needs. Fifteen sibling volunteers within the same age group and similar nutritional status were analysed using HRV (control). Testing procedure and protocol was explained to study group, control group and their parents/guardian. An informed written consent was obtained from parents/guardian of both study and control group to participate in the study. A detailed medical and family history was taken. Any history of medications affecting the ANS was noted. A complete systemic examination of cardiovascular and respiratory system were conducted. Consumption of stimulants and beverages like coffee or tea was avoided on subjects at least a day before the scheduled HRV recording. All recordings were conducted from 10am to 12pm to eliminate the effect of circadian influence. Repeated recordings were done in CP patients in order to avoid artefacts. Subjects were rested comfortably in supine position for 5 minutes and then ECG of the subjects were recorded in Lead II for 5 minutes using three electrodes placed in right infraclavicular region, left infraclavicular region and left iliac region. HRV parameters - Time and Frequency domain were measured according to the Task Force of the European Society of Cardiology and North American Society of Pacing and Electrophysiology. The obtained artefact free recording was analysed using RMS Vagus HRV software (RMS, India)

The following HRV parameters were analyzed:

**TIME DOMAIN:**

- **SDNN**- Standard deviation of Normal to Normal RR interval, it reflects all the cyclical components responsible for variability in the period of recording.

- **RMSSD**- It is square root of the mean squared difference of successive Normal to Normal RR interval.

- **NN50** - Number of interval differences of successive Normal to Normal RR intervals greater than 50ms.

- **pNN50** - The proportion of successive Normal to Normal RR interval greater than 50 milliseconds.

**FREQUENCY DOMAIN:** Power spectral analysis was used to determine the frequency domain parameters.

- **Low Frequency (LF)**- is influenced by both parasympathetic and sympathetic activity.

- **High Frequency (HF)** - influenced by parasympathetic activity.

- **LF/HF ratio** - indicates sympatho-vagal balance.

- **VLF** - is influenced by non-neuronal components affecting the heart like Renin- Angiotensin system, local factors, and thermoregulation.

## Statistical Analysis

Mann-Whitney U test was used to compare the difference in HRV values in in cerebral palsy individuals and their siblings. The level of significance was fixed at $p < 0.05$ Data analysis was carried out using Statistical Package for Social Science (SPSS Software, Version 20).

## Result

The data from twenty five cerebral palsy cases and fifteen sibling volunteer were analyzed. The time domain and frequency domain parameters were measured. Mann-Whitney U test was employed to compare the values. The values are expressed as median and range. However a trend towards reduction is seen in all the time domain parameters (Table no 1) and absolute LF, absolute HF, normalized HF in controls (Table no 2). Whereas a trend toward reduction in cases was seen in

normalized LF and LF/HR ratio (Table no 2).

**Table 1: Comparison of Time domain parameters between CP and sibling volunteers.**

| Group | | Median (Range) | p value |
|---|---|---|---|
| SDNN (ms) | Case | 56.11 (16-89) | 0.423 |
| | Control | 50.41 (20-75) | |
| RMSSD | Case | 33.24 (10.7-59.15) | 0.563 |
| | Control | 29.67 (15.31-63.4) | |
| NN50 (ms) | Case | 36(0-90) | 0.901 |
| | Control | 28 (1.0-115). | |
| pNN50 | Case | 17.85(0-48.3) | 0.432 |
| | Control | 14.23 (0.35-45.10) | |

Mann Whitney U test was employed with the level of significance fixed at $p < 0.05$

**Table 2: Comparison of Frequency domain parameters between CP and sibling volunteers.**

| Group | | Median (Range) | p value |
|---|---|---|---|
| LF (ms$^2$) | Case | 138 (31.00-455) | 0.850 |
| | Control | 135.5 (55-425) | |
| LF (nu) | Case | 65 (53.50-69.10) | 0.646 |
| | Control | 65.1(53.50-77.60) | |
| HF (ms$^2$) | Case | 84.5 (17-305) | 0.921 |
| | Control | 84 (38-225) | |
| HF (nu) | Case | 32.1(20.80-44.60) | 0.645 |
| | Control | 30.95(22.90-42.50) | |
| LF/HF | Case | 2.18 (1.15-3.69) | 0.659 |
| | Control | 2.32 (1.35-3.17) | |

Mann Whitney U test was employed with the level of significance fixed at $p < 0.0$

## Discussion

This study provides a unique and value added addition to the autonomic functional status of Cerebral palsy individuals and their comparison with the sibling volunteers. HRV was recorded only in the resting supine position without subjecting them to any special test (Orthostatic tilt test). No significant difference in supine position for the low frequency component, high frequency component or the low frequency/high frequency ratio between the study and control groups. Time domain parameters between the cases and controls also showed no significant difference. The effect of posture has not been evident since HRV was recorded only in resting supine position. There is very few literature on Autonomic disturbances between CP and their siblings among the Indian population. The

findings from our study contributes as an important addition to the HRV in CP patients and their siblings, suggesting that HRV can be similar in them. Our study as well as the other studies have presented data in terms of median rather than mean as there was high range of standard deviation obtained. For such cases measures are presented in median and found to be statistically more accurate. Most of the studies so far have produced contradictory findings regarding the HRV related data in CP patients. Park et al., Yang et al., etc. The CP group in supine position presented greater sympathetic autonomic predominance in heart rate than the normal group by Zamenur et al (2011).

## Conclusion

This study concludes that there is no statistically significant difference between cases and controls in resting supine position across all the parameters of HRV i.e. both time and frequency domain parameters studied. The calm, relaxed conditions during our recording, normal nutritional status, resting supine position and inclusion of mild to moderate CP children could have contributed to the significantly similar Heart rate variability parameters among the Cerebral palsy and their siblings. The presence of normal sympatho vagal balance in CP predicts that patients with CP have the same predilection as the general population to abnormalities associated with sympatho-vagal balance.

**Conflict of Interest:** 'The author(s) declare(s) that there is no conflict of interest'

**Source of Funding:** Self

**Ethical Clearance:** Obtained from the Institutional Ethical committee(MSRMC, Bangalore)

## References

1. Mutch L, Alberman E, Hagberg B, Kodama K, Perat MV. Cerebral palsy epidemiology: where are we now and where are we going. Dev Med Child Neurol. 1992; 34:547-551.

2. 23 Martha Wilson Jones, Elaine Morgan, Jean E. Shelton, Christine Thorogood. Cerebral Palsy: Introduction and Diagnosis (Part 1). MD J Pediatr Health Care. 2007; 21(3):146-152.

3. Bass N. Cerebral palsy and neurodegenerative disease. Curr Opin Pediatric 1999; 11: 504-507.

4. United Cerebral Palsy (2005) Press room: cerebral palsy - facts & figures. Available from: URL http://www.ucerebral palsy.org/ucerebral palsy_ generaldoc.cfm/1/9/37/37-37/447.

5. MacLennan A. A template for defining a causal relation between acute intrapartum events and cerebral palsy: international consensus statement. BMJ 1999; 319:1054-1059.

6. Heart rate variability. Standards of measurement, physiological interpretation, and clinical use. Task for of the European Society of Cardiology and the North American Society of Pacing and Electrophysiology. Eur Heart J 1996; 17:354–81.

7. Jalife J, Michaels DC. Neural control of sinoatrial pacemaker activity. In: Levy MN, Schwartz PJ, eds. Vagal Control of the Heart: Experimental Basis and Clinical Implications. Armonk, NY: Futura; 1994:173-205.

8. Chess GF, Tam RMK, Calaresu FR. Influence of cardiac neural inputs on rhythmic variations of heart period in the cat. Am J Physiol.1975; 228:775-780.

9. Larson SK, Porges SW. The ontogeny of heart period patterning in the rat. Dev fsychobiol 1982; 15:519.

# Spectral Analysis of Heart Rate Variability During Mobile Phone Usage in First Year Medical Students

**Rekha[1], Rashmi Ramanathan[2], Jensy Sekar[3], Ram Mohan[4], Kalpaka[4], S.Jeevithan[5]**

*[1]Assistant Professor, [2]Associate Professor, Department of Physiology, KMCH Institute of Health Science and Research, Coimbatore, [3]Final MBBS Part I, Karpagam Faculty of Medical Sciences and Research, Coimbatore, [4]Tutor in Physiology, KMCH Institute of Health Science and Research, Coimbatore, [5]Associate Professor, Department of Community Medicine, KMCH Institute of Health Science and Research, Coimbatore*

## Abstract

**Background:** The electromagnetic radiations emitted by cellular telephones may influence the autonomic tone which modifies the functioning of the circulatory system.

**Aim and Objectives:** To estimate the effect of using mobile phones on heart rate variability (HRV) in first year medical students.

**Method:** ECG and HRV parameters are recorded in three periods, - when the subject is at rest without mobile phone (Period-I), with mobile phone kept on the left chest (Period-II) and when responding to a call by keeping the mobile over his right ear (Period-III) on 100 healthy subjects aged between 18 and 21 years of either sex.

**Results:** When the mobile was kept on chest or while attending a call,a decrease in high frequency (HF) in normalized units and increase in low frequency (LF) in normalized units and increase in the ratio of LF to HF (LF/HF) was observed in the frequency domain measures. Decrease in RR intervals, RMSSD and NN50 was observed in the time domain measures

**Conclusion:** The participants showed a higher sympathetic tone and a lower parasympathetic tone while attending a call. The study proved the intense effects of handset radiofrequency radiations on autonomic balance in healthy subjects.

***Key words** :Heart rate Variability, electromagnetic radiations, mobile phone usage.*

## Introduction

Recent years have witnessed unprecedented increase and ubiquitous use of mobile phone and enormous attention about its ill effects on human health. Presently, mobile phones are being used everywhere because these devices do not use the physical cable/wire that is demanded for the communications purposes [1] .The intense use of mobile phones has led to cardiac disorders, malignancy, infertility,sleep disturbances, headaches, memory problems, and nervous disorders like changes in the electroencephalographic activity and brain tumours on long term usage. Electromagnetic fields emitted by cellular phones interfere with the work of cardiac pacemakers and other implantable medical devices [2,3]. So caution should be exercised when using smartphones around sensitive electro medical equipments in hospital intensive care units and on airplanes.

Electromagnetic waves are non-ionizing radiations which causes tissue heating or induce electrical currents in tissues which is the principal mechanism of interaction

**Corresponding author:**
**Dr. Rashmi Ramanathan** M.D.,
Associate Professor, Department of Physiology,
KMCH Institute of health science and research,
Coimbatore
Mail : rashmikumar82@gmail.com
Phone: 9486127040.

between radiofrequency energy and the human body Tissue heating is the best-established mechanism for RF radiation induced effects in biological systems[4]. Since heart is an electrically excitable tissue, more research is required to assess its susceptibility to radio-frequency electromagnetic radiation (RF-EMF).

**Exposure levels :** Mobile phones are low-powered radiofrequency transmitters, operating at frequencies between 450 and 2700 MHz with peak powers in the range of 0.1 to 2 watts [5]. The present standards indicate that the maximum powers that the mobile phones is permitted to transmit are 2 and 1 W for 900 and 1800 MHz respectively [6,7]. High level EMF may change the complexity of cardiac system behaviour [8].

**Heart rate variability** Heart rate variability (HRV) analysis helps to evaluate the equilibrium between the sympathetic and parasympathetic effects on heart rhythm by measuring the beat-to-beat variations of R-R interval [9].

In response to public and governmental concern, WHO established the International Electromagnetic Fields (EMF) Project in 1996 to assess the scientific evidence of possible adverse health effects from electromagnetic fields [10].

It is now known that among the prominent frequency bands in the HRV spectra, the high frequency (HF) component is attributed to the para-sympathetic influences on the heart and that the low frequency (LF) component is due to both the parasympathetic nervous system (PNS) and the sympathetic nervous system (SNS) activities [11,12].

Heart rate variability has gained wide-spread acceptance as a clinical tool for the evaluation of cardiac autonomic changes. It was shown that the call with a mobile phone may change the autonomic balance in healthy subjects due to its electromagnetic field. But the exact contributions of the parasympathetic and the sympathetic divisions of the autonomic nervous system to this variability remain controversial. Therefore, the present study was undertaken to test the intense effects of handset radiofrequency radiations on cardiac autonomic modulation.

## Aim and Objectives

1. To study the effect of radio-frequency electromagnetic radiation (RF-EMF) emitted from mobile phones on cardiac autonomic nervous system in first year MBBS students.

2. To compare the HRV parameters in individuals with and without smart phone radiations.

## Methodology

After getting Institutional Ethical Committee clearance and informed consent from voluntary second year MBBS students, 100 first year MBBS students (male: 50, female: 50 ) in the age group between 18 and 21 years of either sex were included in the study. Students taking medications affecting cardio-respiratory response (anti psychotics, antidepressants, anti arrhythmic) or with any serious cardiovascular disease including arterial hypertension, ischemi/rheumatic heart diseases, diabetes mellitus, metabolic and neurological disorders which could influence the HRV analysis were excluded from the study. Detailed history taking, demographic data & vitals like BP, HR of all the participants were obtained.

### Pre-test preparations

Subjects were instructed not to eat, drink, smoke, exercise & play for at least an hour prior to the test. HRV recording was done between 9:00 and 11:00 am in a semi-darkened, temperature controlled research laboratory. The subject was maintained at rest in supine position for a period of 10 min. The standard procedures for short-term HRV analysis were followed. The changes in the heart rate were measured on the polygraph by Recorders and Medicare System (RMS) polyrite D data acquisition system.

### Method of HRV analysis

Then ECG and HRV parameters are recorded under following conditions.

1. When the subject is at rest without mobile phone for 3min (Period-I)

2. When the mobile is kept over the front pocket or left chest for 3 min (Period-II)

3. When the subject is responding to a call by placing the mobile over his right ear for 3 min (Period-III)

These 3 conditions were studied at an interval of 5 min**.**We used Xiomi Redmi mobile phone for our research project. The highest SAR value for use of this device at the ear is 0.75 W/kg. The recorded HRV parameters are

time domain and frequency domain measures.

The **time domain parameters** obtained during HRV measurements are 1) Mean RR i.e., the average of all RR intervals. 2) SDNN ie.The standard deviation of all NN intervals. 3) NN50 – It is the number of pairs of adjacent RR intervals differing more than 50 ms. 4) RMSSD ie., the square root of the mean of the sum of the squares of differences between adjacent RR intervals. 5) Mean HR.

The **frequency domain parameters** obtained by Lomb periodogram are high frequency (HF, 0.15-0.4 Hz)), low frequency (LF 0.04-0.15 Hz), very low frequency (VLF), and LF: HF ratio. Artifacts were eliminated by computer-based artifact detection and beats were rejected if they varied by more than 40% from the preceding beat.

It is known that the efferent vagal activity is a major contributor to the HF component [17,18] Likewise, it has been reported that RMSSD andPNN50 reflect short-term HRV and are predominantly influenced by the parasympathetic tone, whereas SDNN is influenced by both the sympathetic and parasympathetic tone and express long-term HRV.A predominance of sympathetic tone in cardiac activity contributes to LF component and it induces tachycardia and reduces beat-to-beat variations LF/HF ratio is computed to analyze the predominant activity of the heart.

**Statistical analysis** was done by using the SPSS version 23. All the data were expressed in the form of mean+SD. One way ANOVA was used to test the significant difference among the 3 periods. *P* <0.05 was taken as statistically significant.

**Table 1: Demographic characteristics of the study volunteers**

| Parameters | Male (n=50) | Female(n=50) | P value |
|---|---|---|---|
| Age (years) | 20 ± 0.81 | 19.96 ± 0.90 | 0.816 |
| Height (cm) | 164.34 ± 5.965 | 156.22 ± 5.46 | 0.000** |
| Weight (Kg) | 66.20± 7.071 | 48.10± 7.33 | 0.000** |
| BMI (Kg/m²) | 24.49± 2.05 | 19.70 ± 2.79 | 0.000** |

**Table 2: Comparison of Frequency domain measures of Heart rate variability in three different conditions**

| Parameter | | Period I (mean ± SD) | Period II (mean ± SD) | Period III (mean ± SD) | p value |
|---|---|---|---|---|---|
| LF nu | Males | 45.25 ± 17.43 | 65.55 ± 24.28 | 65.33 ± 19.70 | 0.000** |
| | Females | 42.33 ± 19.07 | 58.04 ± 23.56 | 83.44 ± 32.79 | 0.000** |
| HF nu | Males | 51.01 ± 16.95 | 34.20 ± 15.84 | 33.86 ± 12.49 | 0.000** |
| | Females | 55.83 ± 20.03 | 50.96 ± 18.61 | 48.01 ± 19.51 | 0.129 |
| LF/HF Ratio | Males | 1.14 ± 0.89 | 2.41 ± 1.88 | 2.60 ± 2.74 | 0.000** |
| | Females | 1.01 ± 0.78 | 1.34 ± 0.77 | 2.02 ± 1.18 | 0.000** |
| HF Power | Males | 40.11 ± 20.24 | 23.46 ± 14.25 | 27.38 ± 12.25 | 0.000** |
| | Females | 40.98 ± 21.81 | 40.20 ± 22.74 | 41.19 ± 27.72 | 0.977 |
| VLF Power | Males | 29.08 ± 23.01 | 37.56 ± 26.99 | 26.63 ± 21.40 | 0.060 |
| | Females | 22.37 ± 20.44 | 42.29 ± 55.30 | 40.20 ± 26.78 | 0.015 |

**Table 3: Comparison of time domain measures of Heart rate variability in three different conditions**

| Parameter | | Period I (mean ± SD) | Period II (mean ± SD) | Period III (mean ± SD) | p value |
|---|---|---|---|---|---|
| Mean RR (s) | Males | 0.73 ± 0.11 | 0.68 ± 0.13 | 0.63 ± 0.17 | 0.375 |
| | Females | 0.71 ± 0.10 | 0.69 ± 0.13 | 0.72 ± 0.13 | 0.493 |
| Mean HR | Males | 89.24 ± 27.21 | 84.98 ± 16.83 | 92.42 ± 26.24 | 0.298 |
| | Females | 87.96 ± 13.30 | 90.34 ± 19.14 | 88.78 ± 15.82 | 0.759 |
| SDNN (ms) | Males | 73.87 ± 45.27 | 78.67 ± 49.60 | 84.13 ± 42.06 | 0.535 |
| | Females | 88.98 ± 56.66 | 79.41 ± 47.41 | 97.28 ± 75.51 | 0.344 |
| RMSSD | Males | 76.01 ± 63.67 | 64.47 ± 34.84 | 78.66 ± 40.21 | 0.293 |
| | Females | 97.32 ± 77.20 | 66.03 ± 41.65 | 112.09 ± 98.71 | 0.010* |
| NN50 (count) | Males | 33.97 ± 23.67 | 33.14 ± 21.67 | 45.44 ± 25.55 | 0.017* |
| | Females | 38.83 ± 23.89 | 38.03 ± 24.76 | 29.52 ± 24.94 | 0.113 |
| pNN50 (%) | Males | 27.63 ± 18.46 | 34.45 ± 34.86 | 34.61 ± 16.88 | 0.277 |
| | Females | 33.28 ± 20.04 | 31.86 ± 18.16 | 39.36 ± 34.03 | 0.287 |

**Table 4: Comparison of bloodpressure among the subjects in three different conditions**

| Parameter | | Period I (mean ± SD) | Period II (mean ± SD) | Period III (mean ± SD) | p value |
|---|---|---|---|---|---|
| SBP | Males | 119.4 ± 5.19 | 127.12 ± 4.48 | 127.84 ± 5.64 | 0.000** |
| | Females | 110.4 ± 8.94 | 121.96 ± 8.14 | 117.22 ± 8.91 | 0.000** |
| DBP | Males | 77.96 ± 5.17 | 83.8 ± 3.34 | 83.26 ± 4.45 | 0.000** |
| | Females | 72.4 ± 7.24 | 78.04 ± 6.37 | 78.14 ± 8.76 | 0.000** |

## Discussion

Given the enormous number of smart phone users, it is essential to investigate and monitor any potential health impact of it and mainly on the cardiovascular system.[13] Heart rate variability is a physiological phenomenon that reflects the autonomic functions ie.,parasympathetic and sympathetic influence on sinus node activity. In healthy individuals, parasympathetic tone predominates over sympathetic tone..

Among the **frequency domain measures**(table 2), LF power and LF/HF ratio in period II(2.41±1.88) and III(2.6±2.74) is increased in males than period

I(1.14±0.78) .This strongly emphasise that sympathetic tone has been increased when the mobile phone is kept on chest and while attending call than at rest and. LF power and LF/HF ratio in period III is increased in females than period I and II. This shows that sympathetic tone has been increased more while attending call than other conditions and it is consistent with the research work reported by Ryszard[14] and Berkay Ekici [15]. HF power and HF nu was less in period I and II, ie. less parasympathetic activity which is a considerable cardiac morbidity. Difference in HF power, VLF power, and HF nu among the 3 periods are all statistically insignificant in females and VLF power among the 3 periods is statistically insignificant in males.

Among the time domain measures, (table 3) decrease in mean RR interval and increase in SDNN in period II and III in males strongly depicts that sympathetic tone is increased when the mobile phone is kept on chest and while attending a call. RMSSD was less in period II in both sex ie.,less parasympathetic activity This shows vagal modulation of the autonomic nervous system is decreased while the smart phone is kept on chest.and the sympathetic tone is increased [16] . There is statistically significant difference in RMSSD among the 3 periods in females and NN50 in males which signifies the decrease in vagal tone.

Similar results are derived in the study done by Kodavanji B [17] *et al*emphasised that the time and frequency domain measures of the HRV have provided prognostic information and mobile users had a higher sympathetic tone and a lower parasympathetic tone as compared with the mobile nonusers.

**Hemodynamic** parameters like SBP,DBP (table-4) are increased in period II and III among both ssexes when compared to period I and it is consistent with the study reported by Vangelova [18] et al that electromagnetic radiation exposure increases blood pressure. Heart rate (table-3) showed a steady increase in period II, III in both males and females. Thus it is evident that the phone usage may reflect a change in sympathovagal balancein favour of an increased sympathetic tone which may cause cardiac morbidity when used for prolonged duration.

The novelty our work was to study the effects of long-term exposure to Mobile phones on the cardiac electrical activity. Further randomized control trials should be carried out to show the unexplored effects of autonomic dysfunction on cardiovascular system so that we can prevent majority of cardiac illness by creating awareness among people especially the youngsters.

## Conclusion

In this study, we have shown that the intense effects of handset radiofrequency radiations on autonomic balance in healthy subjects.Consequences of over usageof mobile phones for more than 15 years is lacking and this might warrant further research in this area.WHO also promotes research priorities for radiofrequency fields to fill gaps in knowledge through its research agendasWe conclude by saying that reserve the cell phones for shorter conversations and it is always better to prefer landline phones when available. Liberal use of headphones or keeping the phone little awaywhile attending a call and placing it at least three feet away from bed while sleeping might reduce the exposure to radio frequency energy.

**Limitations of the study**: First, the study population was relatively small and, therefore, the results could not be directly applied to the general population. Secondly, the study recorded HRV in short term. Inclusion of 24 h HRV would be more reliable and conclusive to validate the results.

## References

1.  Suhag AK, Larik RSA MangiGz, Khan M.Impact of Excessive Mobile Phone. Usage on Human Journal of Computer Science & Systems Biology 2016;9(6):173-177.

2.  Altamura G, Toscano S, Gentilucci G, Ammirati F. Influence of digital and analogue cellular telephones on implanted pacemakers. European Heart journal.1997;18:1632–41.

3.  Barbaro V, Bartolini P, Donato A, Militello C.1999. Electromagnetic interference of analog cellular telephones with pacemakers: in vitro and in vivo studies.1999;**22**:626–634.

4. Sofia Kottou, DimitriosNikolopoulos, EfstratiosVogiannis, DionysiosKoulougliotis, ErmioniPetraki,Panayiotis. How Safe is the Environmental Electromagnetic Radiation?Journal of Physical Chemistry and Biophysics. 2014;4(3)

5. Electromagnetic fields and public health: mobile phones 2014 ;[cited oct 8 2018]. Dpwnloaded from https://www.who.int/

6. Guidelines for limiting exposure to time-varying electric, magnetic, and electromagnetic fields (up to 300 GHz). International Commission on Non-Ionizing Radiation Protection. Health Phys 1998;74:494-522.

7. Media centre electromagnetic fields and public health:mobile phones.WHO 8th October 2014 [cited 2ndapril 2019]. Downloaded from https://www.who.int/news-room/fact-sheets/detail/electromagnetic-fields-and-public-health-mobile-phones

8. Analysis of the Mobile phone effect on the Heart rate variability by using the largest lyapunovexponent.Journal of medical system, springer link.2010;34(6):1097–1103.

9. Karthik S and Pal GK. Sympathovagal imbalance in thyroid dysfunctions in females: correlation with thyroid profile, heart rate and blood pressure. Indian J PhysiolPharmacol. 2009;53(3):243–52.

10. What is the International EMF Project? World health organisation.downloaded from www.who.int/peh-emf/project-2016; Aug 4

11. Ori Z, Monir G, Weiss S, Sayhouni X, Singer DH. Heart rate variability: frequency domain analysis. CardiolClin 1992; 10: 499Â–537.

12. Akselrod S, Gordon D, Obel FA, Shannen DC, Barges AC, Cohen RJ. Power spectral analysis of the heart rate fluctuation: a quantitative process of the beat-to-beat cardiovascular control. Science 1981; 213: 220Â–22

13. Barutcu I, Esen AM, Kaya D, Turkmen M, Karakaya O, Sağlam M, etal. Do mobile phones pose a potential risk to autonomic modulation of the heart? Pacing ClinElectrophysiol 2011;34: 1511-4.

14. Ryszard Andrzejak, RafalPoreba, MalgorzataPoreba.The Influence of the Call with a Mobile Phone on Heart Rate Variability Parameters in Healthy Volunteers. Industrial Health ,Field Report 2008;46:409–417.

15. BerkayEkici and AslıTanındı .The effects of the duration of mobile phone use on heart rate variability parameters in healthy subjects Anatol J Cardiol 2016; 16: 833-8.

16. Malliani A, Pagani M, Lombardi F, CeruttiS. Cardiovascular neural regulation explored in the frequencydomain, 1991;84:1482–92.

17. Kodavanji B, Mantur VS, Kumar NA, Pai SR. A pilot study on long term effects of mobile phone usage on heart rate variability in healthy young adult males. J Clinical diagnosis Res.2012;6:346-9.

18. Vangelova K, Deyanov C, Israel M. Cardiovascular risk in operators under radiofrequency electromagnetic radiation. Int J Hyg Environ Health 2006;209:133-8.

# Electrodiagnostic (EDX) Predictors of Sensory Neuropathy in Primary Hypothyroidism and its Association with Age and Body Mass Index (BMI): A Cross Sectional Study

**Sangeeta Jain[1], Balaji Ghugare[1], Mukesh Dinkar[2], Komal Makwana[3], Ravindra Ninama[4], Himanshu Chauhan[4]**

*[1]Associate Professor, GMERS Medical College and Hospital, Gotri Vadodara, Gujarat, [2]Professor & Head, GMERS Medical College and Hospital, Vadnagar, Gujarat, [3]Assistant Professor, [5]Tutor, GMERS Medical College and Hospital, Gotri Vadodara, Gujarat*

## Abstract

**Background:** Sensory neuropathy occurs in hypothyroidism often early as compared to motor. Involvement of median and sural nerve is reported earliest in literature. There is controversial electrophysiological data with respect to predictors of neuropathy and its association with age and body mass index.

**Objectives:** Present study was aimed to find out EDX predictors of sensory neuropathy and its association with age and BMI in primary hypothyroidism patients.

**Materials and Method:** Total 28 out of 39 hypothyroid cases referred to clinical neurophysiology laboratory were selected for analysis. These cases satisfied the biochemical and EDX criteria. Percentages of abnormalities in different variables of nerve conduction study were calculated. Also coefficient of correlation between age, BMI and different variables evaluated for association between them if any.

**Results:** Study population was having age range from 23-75 years, 15 female and 13 male. Female preponderance was observed. Correlation between Age, BMI and different variables of NCV were statistically not significant. Sensory conduction velocity was most common abnormality observed (49%), followed by SNAP amplitude (42%) and onset latency (25%).

**Conclusion:** We concluded that age and BMI have no effect on occurrence of neuropathy in primary hypothyroidism. Sensory Conduction velocity was most common predictor of sensory neuropathy in these cases.

***Key Words:*** *Hypothyroidism, Neuropathy, Nerve conduction study, Onset latency.*

## Introduction

Hypothyroidism is caused by the low level of circulating thyroid hormones and raised TSH. It is estimated to affect 3.8–4.6 % of general population, with four times common in women.[1] Hypothyroidism is an endocrine disorder which results in neurological dysfunction. It also affects brain, peripheral nerves and muscular system. The neurologic manifestations, which may be noted incidently, occur in conjunction with the systemic features of the disease. The symptoms and signs of neurologic dysfunction may be the presenting feature in some patients and can contribute significantly to disability.[2]

In adults, the neurological manifestations of clinical hypothyroidism include decreased mental status, bradycardia, hypothermia, poor concentration and short-term memory, peripheral neuropathy, entrapment

**Corresponding Author:**
**Dr Balaji W Ghugare**
Associate Professor Physiology, GMERS Medical College Gotri Vadodara 390021,
email ID: mulbala@gmail.com, mob No: 9427119413.

neuropathy and myxoedema coma. In some patients with clinical hypothyroidism, the main and presenting manifestation may be the peripheral nerve dysfunction. In overt hypothyroidism, the frequency and severity of neuromuscular disease depends mostly upon the severity and duration of thyroid hormone deficiency.[3] In hypothyroidism, metabolic alteration occurs due to hormonal imbalance which affects the Schwann cell and induces a segmental demyelination. It has been shown electrophysiologically and pathologically that there is primary axonal degeneration.[4] The function and the ability of electrical conduction of the motor and sensory nerves can be evaluated by nerve conduction study. With the steady improvement and the standardization of these methods, nerve conduction studies have become reliable tests in clinical settings. Nerve conduction studies often can define whether the underlying pathophysiology is demyelination or axonal loss and they can differentiate between a primary demyelination and a primary axonal neuropathy.[5] Most of the studies on hypothyroidism and neuropathy evaluated prevalence of neuropathy in primary and overt hypothyroid cases and effect of age, gender, obesity, duration of disease on occurrence of neuropathy. With above background this study was done to find out the effects of age and BMI on sensory nerve conduction study variables in primary hypothyroid patients. We also evaluated the frequent EDX predictors of sensory neuropathy in these patients.

## Material and Method

A cross sectional study was done by analysing the data retrieved from 28 hypothyroid patients that were referred to clinical neurophysiology laboratory at GMERS Medical College Gotri Vadodara. Total 39 cases were referred for electrodiagnostic evaluation during period January 2015 to December 2018. All the Cases were referred from department of Medicine. Out of these 11 patients were excluded as it did not meet our clinical, EDX and biochemical criteria for inclusion. Data in these cases was insufficient. Patients suffering from diabetes mellitus, liver and kidney disease and patients with hypothyroidism secondary to pituitary disease were excluded from the study. 28 cases fulfilled above mentioned inclusion criteria and hence their data selected for analysis.

RMS EMG Portable Aleron machine was used for nerve conduction study at clinical neurophysiology laboratory, Department of Physiology. All the patients underwent Sensory nerve conduction study that included the determination of onset latency, sensory nerve action potential (SNAP) amplitude and conduction velocity of median, ulnar nerves bilaterally.

## Procedure in brief

Sensory nerve conduction study (antidromic) involved stimulation of sensory nerves proximally and recording SNAPs with electrodes placed distally over the dermatomic distribution. Sensory nerve conduction velocity was calculated by dividing the distance between active electrode and cathode of stimulator by onset latency. Sensory nerve action potential amplitude was taken from peak to base. Ground electrode was placed between stimulating and recording electrodes.

Antidromic study was done using ring electrode. Ring electrode was placed on index finger for Median nerve on little finger for ulnar nerve. In all cases cathode and anode were 3 cm apart. For upper and lower limbs, duration was 100 μs, sweep speed 2 ms/D and filter was between 20Hz to 3 KHz.

## Data storage and Statistical analysis

Data was stored in Microsoft excel sheet. Graph Pad prism software was used for data analysis. Correlation coefficient was obtained to see the effect of age and BMI on different nerve conduction study variables in hypothyroid cases. Demographic profile was obtained for the study population. Frequency of abnormalities in sensory nerve conduction study variables was obtained. Further, correlation coefficient to evaluate the effect of age and BMI on different variables was measured.

## Results

Total 28 cases data was selected for analysis. Out of that 15 were female and 13 were male. Table number 1 shows demographic and anthropometric profile of participants.

**Table No. 1: Demographic profile of participants in study (n=28, 15 female, 13 male)**

| Parameters | Mean ±SD | Range (Minimum-Maximum) |
|---|---|---|
| Age (Years) | 50.12±13.5 | 23-75 |
| Weight (Kg) | 60.41±11.35 | 45-88 |
| Height (Cm) | 158.2±5.62 | 150-168 |
| BMI (Kg/M2) | 24.23±4.89 | 17.6-34.2 |

(Note: SD- standard Deviation, Kg- Kilograms, Cm- centimetres, BMI- Body mass index, M²- Square meter)

Table number 2 shows frequency of abnormalities observed in sensory nerve conduction study parameters. Total 112 nerves (56 median and ulnar each) were assessed for observation of abnormalities. Nerve conduction velocity abnormalities were most common and onset latency variable was least affected. Absent SNAP, Present but reduced amplitude and CV and Prolonged OL were noted as abnormalities in variables.

**Table No.2: Distribution of abnormalities in different sensory nerve conduction study variables in study population (n=112)**

| NCV Variables | Right side | | Left side | | Total (%) |
|---|---|---|---|---|---|
| | Median | Ulnar | Median | Ulnar | |
| OL | 6 | 05 | 09 | 08 | 28 (25%) |
| SNAP Amplitude | 14 | 07 | 15 | 12 | 48 (42.85%) |
| CV | 17 | 08 | 17 | 13 | 55 (49.10%) |

(Note: NCV- Nerve conduction velocity, OL- Onset latency, SNAP- sensory nerve action potential)

Table number 3 shows whether BMI and age had any association with Neuropathy in hypothyroid patients or not? Table shows that there was no statistically significant association observed between age, BMI and different NCV variables (r value was <0.5 for all variables). Although a negative trend with respect to age, BMI and SNAP amplitude was observed, it was not statistically significant.

**Table No. 3: Association of age and BMI with sensory nerve conduction study variables in study population.**

| NCV variables Age | | Onset latency | | Amplitude | | Conduction velocity | |
|---|---|---|---|---|---|---|---|
| | | BMI | Age | BMI | Age | BMI | |
| Right side | Median | -0.14 | -0.16 | 0.021 | -0.31 | 0.30 | 0.16 |
| | Ulnar | 0.008 | -0.08 | -0.13 | 0.05 | 0.07 | 0.20 |
| Left side | Median | 0.137 | -0.15 | -0.36 | 0.05 | 0.04 | 0.19 |
| | Ulnar | 0.4 | 0.04 | -0.23 | 0.15 | -0.03 | 0.27 |

(Note- NCV- Nerve conduction study, BMI- Body mass index, all numerical values represent 'r' values)

## Discussion

Present study enlightens our existing knowledge about frequently abnormal sensory NCV variables in hypothyroid patients and also about trends in abnormality of these variables with respect to age and BMI.

We analysed 112 sensory median and ulnar nerves bilaterally to find out most common predictor of neuropathy in hypothyroids. Conduction velocity was most frequently occurring abnormality (49.10%) followed by SNAP amplitude abnormality (42.85%). Onset latency abnormalities remained least common predictors of neuropathy in hypothyroid cases (only

25%).

Our observations are coinciding with the previous studies that concluded median and sural nerve demyelinating sensory polyneuropathy are common in hypothyroidism. Reduction in amplitude in 60% cases for median and sural sensory nerves and slowing of conduction velocity in 71% for these nerves was noted. In most of these studies early sensory involvement affecting prolonged latencies, reduced amplitude and CV have been observed. [6, 7, 8]

Neurological dysfunction associated with disorders of thyroid gland could be the result of hormonal imbalance or immune mechanism accompanying thyroid disease. Metabolic alteration in hypothyroidism affects Schwann cells leading to segmental demyelination which is reflected as a decrease in conduction velocity. [9]

Present study also highlighted about effect of age and BMI on different NCV variables in hypothyroid cases. Although there was no statistically significant correlation observed between age, and BMI with different variables; a negative association of advancing age and neuropathy in hypothyroidism was attributed to persistent reduction in SNAP amplitude. S Karne et al reported that there was independent correlation of advanced age and female gender with development of neuropathy among primary hypothyroid cases. They also observed that obesity was also one of the risk factor for neuropathy although we could not find any such association between BMI and EDX variables of Neuropathy. [10]

## Conclusion

We concluded that age and BMI have no effect on occurrence of neuropathy in primary hypothyroidism. Sensory Conduction velocity was most common predictor of sensory neuropathy in these cases. Onset latency of SNAP was least abnormal among different EDX variables. Smaller sample size remains major drawback of study to extrapolate findings in general population.

**Ethical Clearance:** Institutional Ethics committee was informed and permission to publish neurophysiology laboratory data from institute/department head was duly obtained.

**Conflict of Interest**: None declared

**Sources of Support:** Nil.

## References

1. G. Velayutham, B. Kabali. Pattern Reversal Visual Evoked Potentials in Hypothyroid Females. National Journal of Basic Medical sciences. 2012; 2:365-68.

2. Adikesavan Balraman,Gowdhanam Natarajan, B. Vishwanatha Rao and Balasubramanian Kabali. A Study of Nerve Conduction Velocity in Newly Diagnosed Hypothyroid Females. World Journal of Medical Sciences. 2013; 9:198-201.

3. Jalilzadeh SH, Bahrami A, Eftekharosadat B, Mobasseri M, Pezeshki Z. Peripheral Nerve Function in Subclinical Hypothyroidism: A Case-Control Study. Int J Endocrinol Metab. 2006; 4:78-83.

4. Raffaello Nemni, Edo Bottachi, Raffaella Fazio, Angelo Mamoli, Massimo Corbo, Massimo Camerlingo et al. Polyneuropathy in hypothyroidism. Clinical, electrophysiological and morphological findings in four cases. J Neurol Neurosurg Psychiatry. 1987; 50: 1454-1460.

5. Preston DC, Barbara E.Shapiro. Electromyography And Neuromusc 2nd edition. USA: Elsevier; 2005: pg5.

6. Asia A, Warkar A. Nerve conduction studies in recently diagnosed untreated hypothyroid patients. Indian Journal of Basic and Applied Medical Research; 2015;4(4):330-334.

7. Yeasmin S, Begum N, Begum S, Rahman SMH. Sensory neuropathy in hypothyroidism: Electrophysiological and clinical findings. Journal of Bangladesh Society of Physiologist. 2007;2: 1-6.

8. Khedr EM, El Toony LF, Tarkhan MN, Abdella G. Peripheral and central nervous system alterations in hypothyroidism: electrophysiological findings. Neuropsychobiology. 2000;41(2):88-94.

9. S Waghmare, S Pawar, V shende, S Pajai. Sensory neuropathy in hypothyroidism: A case control study. International journal of contemporary medical research. 2016; 3(11):3262-3265.

10. SS Karne, NS Bhalerao. Nerve conduction studies in primary hypothyroidism. Thyroid research and practice.2016; 13(3):131-135.

# Study of Parasympathetic Activity in Newly Diagnosed Patients of Moderate Depression Using High Frequency of HRV

**Sat Pal[1], Neena Mishra[2], Priti Singh[3], Praveen Prashant[4]**

*[1]Associate Professor, [2]Ex-Professor, Dept. of Physiology, [3]Professor, Dept. of Psychiatry,
Pt. B D Sharma PGIMS, Rohtak, [4]Biochemistry Dept. BPSGMCW Khanpur Kalan*

## Abstract

**Introduction: -** Heart Rate Variability (HRV) is a noninvasive technique to measure parasympathetic activities of heart. In this study, we measured the Parasympathetic activity in newly diagnosed patients of moderate depression by High Frequency (HF) parameter of HRV. Depression is the leading cause of mental disability and by the year 2020, depression is projected to reach 2nd place of the ranking of disease *disability-adjusted life year* (DALY).

**Objective:** Parasympathetic activity in newly diagnosed patients of moderate depression.

**Method:-** this study was planned on 40 newly diagnosed male patients of moderate depression according to ICD-10 (the international classification of diseases-10) taken from department of Psychiatry PGIMS Rohtak and 40 healthy males subjects of same age group. High Frequency (HF) parameter of HRV was recorded by using RMS polyrite machine.

**Result: -** The HF ($ms^2$) component in frequency domain of HRV in patients of moderate depression in group II (166.55±41.48) compared to normal subject in group I (179.48±41.68) which reduced but not significantly. The HF expressed in normalized unit (HFnu) and group II (35.30±12.58) were compared to Group I (38.74±1276) and which reduced but not significantly. And HF expressed in power percent (HFpp) and values of group II (18.83±5.04) were compared to the Group I (20.58±5.02), the reduction was not significant.

**Conclusion:-**We found that Parasympathetic activity was not reduced significantly in patients of moderate depression. So the parasympathetic activity is not a problem to cause cardiac disease in patients of moderate depression.

***Keywords:** patients of moderate depression, Heart rate variability, parasympathetic activity.*

## Introduction

The psychological disorder is multifaceted and it affects an individual's mental and physical health.[2] Depression is the leading cause of mental disability and the 4th leading contributor to the global burden of disease *disability-adjusted life year* (DALY) in 2000.[3]. At this rate of increase, by the year 2020, depression is projected to reach 2nd place of the ranking of DALYs calculated for all ages, both sexes, after heart disease.[4] According to the international classification of diseases-10 (ICD-10), depression is generally ranked in terms of severity as mild, moderate and severe.[1] The main typical symptoms are depressed mood, loss of interest, reduced energy leading to increased fatiguability and diminished activities. Other symptoms are reduced concentration, reduced self-esteem, idea of guilt, pessimistic views of the future, idea of self harm or suicide, disturbed sleep and diminished appetite. In moderate depression, at least two typical symptoms and three of the other symptoms must be present.[14] It has been reported that major factors associated with depressive disorders were female sex,

**Corresponding Author:**
**Praveen Prashant**
Biochemistry Dept. BPSGMCW Khanpur Kalan

middle age, low level of education, financial constraints and relationship problems. It was also found that there was an increasing trend in the prevalence of depression with increasing age in both genders. [5,10,11] There is a linear rise with age of self-reported symptoms of depression.[6] The older individuals are more vulnerable to depression.[7] In 1992, in USA, the depression reaches its lowest level in the middle age at about age 45, with a rise in later life (80 years). In India, instead of decline in the middle age, there was a steady increasing trend seen with age. [8] Women have higher depression rates than men.[9, 10] In Pakistan, the prevalence of depression is high. At its worst, depression can lead to suicide. There is loss of about 850000 lives every year.[12]

The human heart beat in a healthy individual is not absolutely regular. It varies as a result of interplay of many factors including respiration (Respiratory sinus arrhythmia), stress (both physical and mental), exercise, blood pressure, Renin-angiotensin system, circadian rhythm and other unknown complex mechanisms. Hence heart rate normally fluctuates around a mean heart rate. This fluctuation of heart rate is called as Heart rate variability (HRV). In a normal person the increase in heart rate is due to dominant effect of efferent sympathetic activity and decrease is due to dominant parasympathetic activity. From the above reason and by pharmacological experiments now it is proved that the heart rate variability indicates the balance between parasympathetic and sympathetic function of autonomic nervous system.[14]

HRV analysis provides a quantitative marker of the autonomic nervous system (ANS) because the regulation mechanisms of HRV originate from the sympathetic and parasympathetic nervous systems.[15] Furthermore; patients with mood disorders such as depression have an imbalance in cardiac autonomic tone with excessive sympathetic excitation, which results in decreased HRV.

## Review of Literature

The HF component is correlated with the parasympathetic dominance, resting vagal tone and cardiac vagal control (CVC).[17]

Julian et al (1998) observed that non depressed male subjects had greater HF than depressed male subjects; however, non-depressed female subjects had less HF than depressed female subjects. He quoted Yeragani et al (1991) who did not find a difference in HF between depressed patients and healthy subjects, Ryan et al

(1994) observed higher HF in female than male subjects and Carney et al (1995) suggested decreased in HF in psychiatric populations having depression and decrease HF is a risk factor for cardiac events.[18]

Jonathan Rottenberg has done a critical analysis of role of cardiac vagal control in relation to HF and cited that physical activity level is positively associated with CVC (Mølgaard et al.1994), depression is associated with reduced physical activity and impaired exercise performance (Hollenberg et al 2003), Agelink et al. (2001) studied patients of major depression and found no relation between change in depression score and HF power.[19]

## Material and Method

The present study was conducted in Department of Physiology in collaboration with Department of Psychiatry at Pt. B. D. Sharma, PGIMS, Rohtak. Forty male patients of moderate depression according to ICD-10 (the international classification of diseases-10) in the age group of 18-40 years were drawn from the psychiatry department of this institute. Forty normal age matched male subjects were drawn from our staff members, medical students and healthy attendants accompanying the patients to the Institute. They were arranged into two groups GROUP I- 40 normal healthy male subjects. And GROUP II- 40 male patients of moderate depression.[13]

### Inclusion Criteria

Patients of moderate depression according to the international classification of diseases-10 (ICD-10) in which moderate depression has at least two typical symptoms and three of the other symptoms were subjected for the study. The main typical symptoms are depressed mood, loss of interest, reduced energy leading to increased fatiguability and diminished activities. Other symptoms are reduced concentration, reduced self-esteem, idea of guilt, pessimistic views of the future, idea of self harm or suicide, disturbed sleep and diminished appetite. Subject having two typical symptoms and three other symptoms were included. [13]

### Exclusion Criteria:

The following patients were excluded from the study if having:-

- History of any other major illness in the previous one year.

- History of drug intake for any other ailments in last one month.

- Co-morbid psychiatric disorder.

- Patients with severe depression or with suicidal tendencies

- Unwilling to undergo the procedure.

- Patients having endocrine disorders.

Informed and written consent was taken from every patient and subject for undergoing the whole procedure. All experiments were conducted between 10am to 1pm to avoid diurnal variations. The whole procedure was explained in detail to each subject in their own language in order to alley any fear or apprehension. The machine used was RMS digitalized polygraph, "Polyrite-D" system (RMS India Pvt. Ltd, Chandigarh). By this machine wide variety of physical phenomena can be recorded simultaneously and individual customization can be done. Along with automatic analysis, the data can be stored in hard disk and auto-regeneration of report is possible at the time of requirement.

The subject was asked to lie down on a couch in front of the POLYRITE D system. Then 3 disposable adhesive electrodes were attached to left arm, right arm, and left leg respectively and 3 electrodes for ECG were attached to them for measuring HRV. Then the basal recording of E.C.G (lead II) was done for 5 minutes.

The artifacts produced due to the movement may cause a discrepancy in the result which is a major limitation in the interpretation of the same.[16] For which in our study we have taken utmost care to keep the subject absolute immobile to eliminate the movement factor.

## Result

**Table 1: Comparison of High Frequency (power percent) in normal subjects (Group I) and patients of moderate depression (Group II)**

| Parameter | Group I Mean ± SD | Group II Mean ± SD | p Value |
|---|---|---|---|
| HF p% | 20.58±5.02 | 18.83±5.04 | NS |

HF expressed in power percent (HFpp) and value of the Group I (20.58±5.02) and group II (18.83±5.04) were compared. HF decrease in Group II but not significantly (Table 1)

**Table 2: Comparison of High Frequency (normalized unit) in normal subjects (Group I) and patients of moderate depression (Group II)**

| Parameter | Group I Mean ± SD | Group II Mean ± SD | p Value |
|---|---|---|---|
| HF(nu) | 38.74±1276 | 35.30±12.58 | NS |

HF expressed in normalized unit (HFnu) in the Group I (38.74±1276) and group II (35.30±12.58) were compared. HF decrease in Group II but not significantly (Table 2)

**Table 3: Comparison of High Frequency (millisecond square) in normal subjects (Group I) and patients of moderate depression (Group II)**

| Parameter | Group I Mean ± SD | Group II Mean ± SD | p Value |
|---|---|---|---|
| HFms2 | 179.48±41.68 | 166.55±41.48 | NS |

HF expressed in millisecond square in group I (179.48±41.68) and group II (166.55±41.48) were compared. HF decrease in Group II but not significantly (table 3)

## Discussion

The HF ($ms^2$) component in frequency domain of HRV in patients of moderate depression in group II (166.55±41.48) and normal subject in group I (179.48±41.68) were compared (table 3), the HF expressed in normalized unit (HFnu) and the Group I (38.74±1276) and group II (35.30±12.58) were compared (Table 2) and HF expressed in power percent (HFpp) and value of the Group I (20.58±5.02) and group II (18.83±5.04) were compared (Table 1). In all these different units of expression of HF of both groups, no statistically significant change was seen.

In our study, the decrease in HF was not statistically significant as we have used male patients only ( avoided gender difference) and patient of moderate depression

(not major depression) thus the decrease in HF have shown that there is insignificant alternation of vagal tone in patient of moderate depression.

## Conclusion

As per the above discussion we saw that the major depression is associated with various co-morbid conditions of cardiac origin. In case of moderate depression the autonomic imbalance is not a problem to cause cardiac disease.

## References

1. Wittchen HU, Pittrow D. Prevalence, recognition and management of depression in primary care in Germany: the depression 2000 study. Hum Psychopharmacol 2002;17:1–11.

2. Michaud CM, Murray CJ, Bloom BR. Burden of disease: implications for research. JAMA 2001;285:535-9.

3. Pothen M, Kuruvilla A, Philip K, Joseph A, Jacob KS. Common mental disorders among primary care attenders in Vellore, south India: nature, prevalence and risk factors. Int J Soc Psych 2003;49:119-25.

4. Nambi SK, Prasad J, Singh D, Abraham V, Kuruvilla A. Explanatory models and common mental disorders among patients with unexplained somatic symptoms attending a primary care facility in Tamil Nadu. Natl Med J India 2002;15:331-5.

5. Mirza I, Jenkins R. Risk factors, prevalence, and treatment of anxiety and depressive disorders in Pakistan: a systematic review. BMJ 2004;328:794.

6. Stordal E, Mykletun A, Dahl AA. The association between age and depression in the general population: a multivariate examination. Acta Psychiatrica Scandinavica 2003;107:132-41.

7. Lerer B, Gillan D, Lietenberg P, Gorfine M, Gelfin Y. Inter relationship of age, depression and central serotonergic function: evidence from fenfluramine challenge studies. Int Psychogeriatry 1996;8:83-102.

8. Mirowsky J, Ross CE. Age and depression. J Health Soc Behav 1992;33:187-205.

9. Blazer DG, Kessler RC, McGonagle KA, Swartz MS. The prevalence and distribution of major depression in a national community sample: the national co-morbidity survey. Am J Psychiatry 1994;151:979-86.

10. Bebbington PE, Dunn G, Jenkins R. The influence of age and sex on the prevalence of depressive conditions: report from the national survey of psychiatric morbidity. Psychol Med 1998;28:9-19.

11. Salokangas RK, Poutanen O. Risk factors for depression in primary care: findings of the TADEP project. J Affect Disord 1998;48:171-80.

12. Poongothai S, Pradeepa R, Ganesan A, Mohan V. Prevalence of Depression in a large urban south indian population: the Chennai urban rural epidemiology study. Plos One 2009;4(9):e7185.

13. ICD-10 Classification of mental and behavioural disorder. Clinical descriptions and diagnostic guidelines. Indian ed. Delhi: A.I.T.B.S. Publishers; 2007. p. 110-11.

14. Katira R, Narain VS, Puri VK. Heart rate variability. JAPI 1997;45:49-51.

15. Juha PN P, Tarvainen PO, Ranta PA, Karjalainen. Software for advanced HRV analysis. Comptr Methds Progrms Biomed 2004;76:73-81.

16. Brenner KM, Thomas S, Shepherd RJ. Autonomic regulation of the circulation during exercise and heat exposure: inferences from heart rate variability. Sports Med 1998;26:85-99.

17. Malik M, Bigger JT, Camm AJ, Kleiger RE, Mallini A, Moss AJ et al. Task Force of the European Society of Cardiology and the North American Society of Pacing and Electrophysiology. Heart rate variability standards of measurement, physiological interpretation, and clinical use. Euro Heart J 1996; 17:354-81.

18. Julian FT, Smith M, Rossy LA, John JS, Friedman BH. Heart period variability and depressive symptoms: gender differences. Biol Psych 1998;44:304-6.

19. Rottenberg J. Cardiac vagal control in depression: a critical analysis. Biological Psychol 2007;74:200-11.

# Prevalence of Hypertensive Disorder of Pregnancy in North India

**Seema Kumari**

*Asstt Professor (Physiology) P.M.C.H. Patna*

## Abstract

**Aims and Objective:** Hypertensive disorders of pregnancy (HDP) are among the commonest medical disorders during pregnancy constituting one of the greatest causes of maternal and perinatal morbidity and mortality worldwide . This study aimed to analyze the prevalence of hypertensive disorder of pregnancy in North India

**Material and Method:** The study group consisted of a total of 150 pregnant women attending Maharaja Agrasen hospital OPD/IPD during their second trimester (14-20 weeks) of pregnancy from Dec 2016 to Nov 2017.

**Results:** Out of 146 cases studied , 129 cases remained normotensive and 17 cases developed HDP. Out of 17 cases ,8 cases had mild HDPs and 9 cases had severe HDPs. The prevalence of HDP was 11.7% in present study.

**Conclusion:** The study concluded that the incidence of hypertensive disorders in pregnancy was higher than worldwide average and hence early diagnosis and intervention through regular antenatal checkup is pivotal to prevent hypertensive disorders of pregnancy and its complications.

***Key words:*** *HDP, Hypertension , Pregnancy, North India*

## Introduction

Hypertensive disorders of pregnancy (HDP) are among the commonest medical disorders during pregnancy constituting one of the greatest causes of maternal and perinatal morbidity and mortality worldwide. [1-4] HDP complicate up to 5-10% of all pregnancies.[5] It accounts for 10-15% of maternal deaths specially in the developing world.[6] The spectrum of disease ranges from mildly elevated blood pressures with minimal clinical significance to severe hypertension and multi-organ dysfunction .Besides perinatal death ,HDP lead to preterm delivery, fetal intrauterine growth restriction, low birth weight. HDP can also trigger severe

forms of maternal complications, such as cardiovascular and cerebrovascular diseases, liver and kidney failure, placental abruption, disseminated intravascular coagulation (DIC) and HELLP syndrome.

Hypertension in pregnancy is defined as a systolic BP of 140 mmHg and higher, and a diastolic BP of 90 mmHg and higher. Severe pregnancy hypertension is defined as SBP ≥160 mmHg or a DBP ≥110 mmHg. The systolic value was reduced from 170 mmHg by most international societies after recognition that a SBP≥160 mmHg is associated with an increased risk of stroke in pregnancy.

Hypertensive disorders during pregnancy are classified into four categories, as recommended by the National High Blood Pressure Education Program (2000) Working Group on High Blood Pressure in Pregnancy[7] :

- Gestational hypertension (transient hypertension of pregnancy)

- Preeclampsia-eclampsia

**Corresponding Author:**
**Dr Seema Kumari**

Dr Seema Kumari , Asstt Professor (Physiology) , P.M.C.H. , Patna, North Patel Nagar, Patna 800024
E-mail: drseemakumari2@gmail.com

- Preeclampsia superimposed on chronic hypertension

- Chronic hypertension

**Aims & Objective:** To find the prevalence of Hypertensive disorder of pregnancy in North India.

## Material and Method

Study site: Maharaja Agrasen Hospital, Punjabi Bagh, New Delhi.

Study population : Woman with singleton pregnancy visiting Obs & Gynae OPD/IPD of Maharaja Agrasen hospital, Punjabi Bagh during their second trimester (14-20 weeks) of pregnancy

Study Design : Prospective observational study

Sample size: 150

**Inclusion Criteria**

▶ 1.All pregnant women in their second trimester (14 - 20 weeks) above 18 years and below 40 years of age with informed consent.

▶ 2. Pregnancy confirmed by ultrasonography

▶ 3. Singleton pregnancy

▶ 4. Previously normo tensive and non proteinuric

**Exclusion Criteria**

The established **exclusion criteria** are as follows:

▶ Age less than 18 and more than 40 years

▶ Multiple pregnancy

▶ Chronic Hypertension

▶ Gestational trophoblastic diseases in present or previous pregnancy

▶ Down syndrome/any congenital anomalies

▶ Pregnancy associated with Germ cell tumors

▶ Diabetes mellitus

## Methodology

- A total of 150 pregnant women attending Maharaja Agrasen hospital OPD/IPD during their second trimester (14-20 weeks) of pregnancy were enrolled in the study.

Diagnostic criteria for **Gestational Hypertension** was new onset of hypertension ($\geq$140 mmHg systolic and/or $\geq$90 mmHg diastolic) after 20 weeks gestation without Proteinuria and signs of end organ dysfunction.

Diagnostic criteria for **Pre-eclampsia (revised ISSHP, 2014)** was Hypertension after 20 weeks gestation and the coexistence of one or more of the following new-onset conditions:

1. Proteinuria (spot urine protein/creatinine >30 mg/ mmol [0.3 g/mg] or >300 mg/day or at least 1 g/L['2 + '] on dipstick testing).

2. Other maternal organ dysfunction: renal insufficiency (creatinine >90 umol/L; 1.02 mg/dL), liver involvement (elevated transaminases – at least twice upper limit of normal $\pm$ right upper quadrant or epigastric abdominal pain), neurological complications ( eclampsia, altered mental status, blindness, stroke,or more commonly hyperreflexia when accompanied by clonus, severe headaches when accompanied by hyperreflexia, persistent visual scotomata), haematological complications (thrombocytopenia, , DIC, haemolysis).

3. Uteroplacental dysfunction (foetal growth restriction)

Diagnostic criteria for **Eclampsia** was Pre-eclampsia associated with convulsion.

## Observation & Results

**Table 1: Mean Age of the three groups (Normotensive, Mild HDP and Severe HDP)**

| GROUPS | MEAN +/_ SD | RANGE | MEDIAN | P VALUE |
|---|---|---|---|---|
| NORMOTENSIVE (B) | 27.86+/-3.71 | 19-37 | 28 | |
| MILD HDP( A1) | 29.87+/-4.63 | 21-38 | 30 | 0.10 |
| SEVERE HDP (A2) | 29.11+/-4.40 | 24-39 | 29 | |
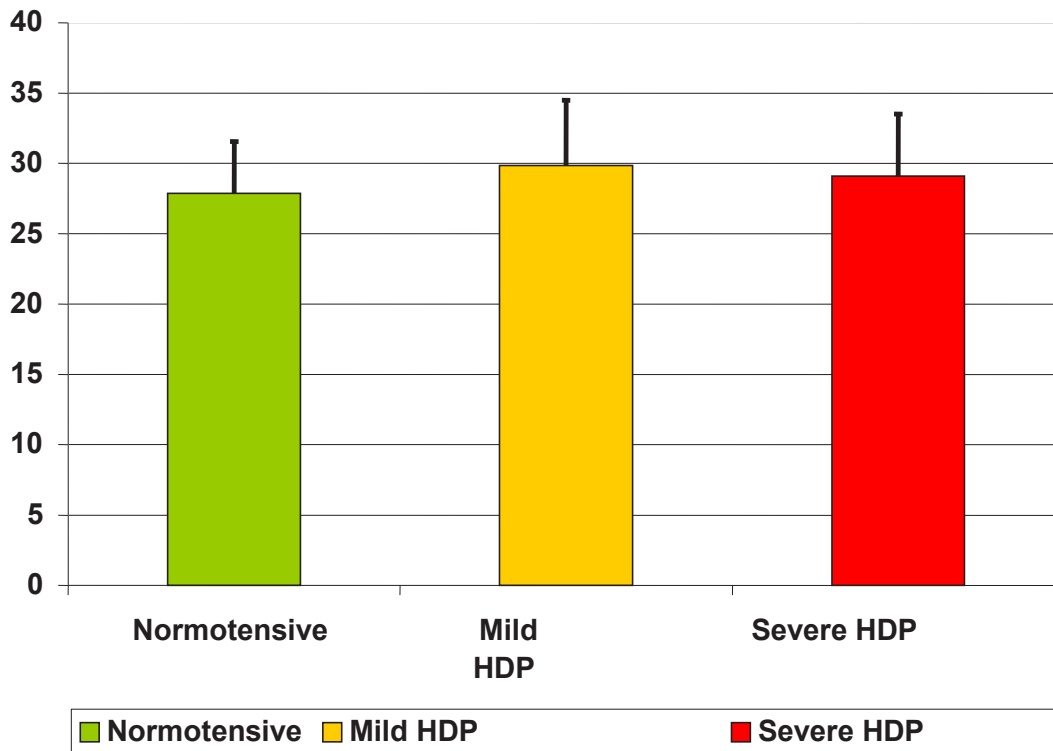
The mean age of normotensive group was 27.86+/-3.71, whereas in Mild HDP and Severe HDP group was 29.87+/-4.63 and 29.11+/-4.40. There was statistically no significant difference between age of three groups.
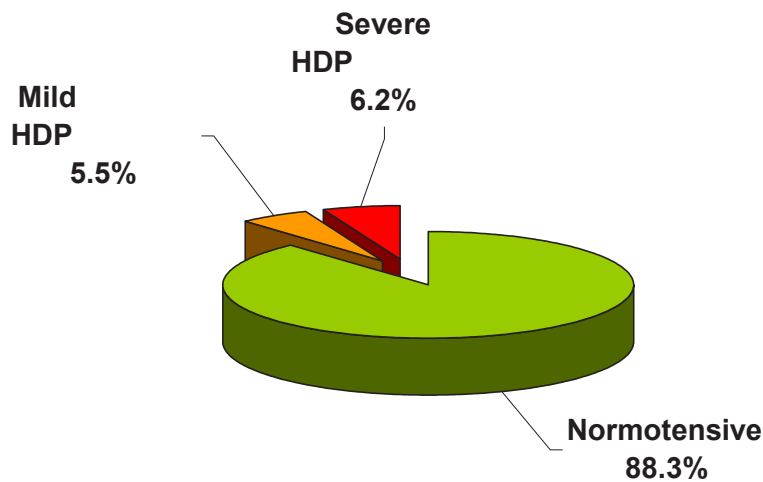


Corrected Chi-square () test ( =13.28; p=0.10 NS-Not Significant) showed that there was no significant association between age and cases of the three groups (p=0.10). Thus the cases of the three groups were more or less equally distributed over age.

One ANOVA showed that there was no significant difference between the mean age of the cases of the three groups ($F_{2,143}$=2.11;p=0.12). Thus the cases of the two groups were age matched.

**Table-2: Prevalence of HDP**

| Group | Number | % |
|---|---|---|
| Normotensive | 129 | 88.4% |
| Mild HDP | 8 | 5.5% |
| Severe HDP | 9 | 6.2% |
| Total | 146 | 100.0% |

Out of the 146 cases under study 129(88.4%) cases were normotensive, 8(5.5%) were Mild HDP and rest 9(6.2%) were severe HDP. In overall 17(11.7%) were HDP. Thus the prevalence of HDP was 11.7%.

## Discussion

Hypertensive disorders of pregnancy (HDP) is a major challenge in overcoming pregnancy complications that are responsible for poor maternal and prenatal outcome in developed as well as underdeveloped countries of the world. These disorders comprise of chronic hypertension, gestational hypertension, preeclampsia and eclampsia.

The spectrum of HDP ranges from mildly elevated blood pressures with minimal clinical significance to severe hypertension and multi-organ dysfunction.

In my study, 150 cases were initially enrolled. However, only 146 cases (97.3%) could be evaluated for the final results. The 4 cases were lost to follow up.

These cases were divided into two groups:

• **Group A** - who developed HDP.

• **Group B** - who remained normotensive

Group A was divided further into two group i.e. Group A1 and Group A2 based on features of severity. Cases in Group A1 were mild hypertensive and A2 were severe hypertensive.

The mean age of the cases for my study was 27.86 (Group B normotensive) 29.87 (Group A1, mild hypertensive) and 29.11 (Group A2 ,severe hypertensive).

There was no statistically significant correlation found between the age and the occurrence of HDP which was in concordant with the results of study conducted by Vishal Sharma et al (2016) [7], who observed that there was no statistically significant difference between age of subjects and HDP. 76.34 % of the patients in my study were between 21 and 30 years of age thus rendering a very young population morbid and at risk of mortality.

The prevalence of HDPs observed in my study is 11.7 % . Incidence of hypertensive disorders in India is found to be 10.08 % as observed through the data collected by the National Eclampsia Registry (NER) (11,266 out of 1,11,725 deliveries) over the 3 consecutive years[8,9] . The prevalence matches out with NER data considering the fact that my study place is tertiary care centre where high risk patients having more visits.

In a study conducted by Vidyabati R K et al (2010)[10] prevalence rate of HDPs was 17.68%. In the study by Charu sharma et al (2017)[11], the incidence of HDP came out to be 6.92%. The prevalence of hypertension during pregnancy was found to be 6.9% in the study conducted by Bharti Mehta et al(2015)[12]. Hypertensive disorders of pregnancy were reported to be 15.5, 5.38, and 8.96%, respectively, in other various hospital-based studies in India. [13,14,15]

## Summary & Concusion

In my study, 150 cases were initially enrolled. However, only 146 cases (97.3%) could be evaluated for the final results. The 4 cases were lost to follow up . Out of 146 cases studied , 129 cases remained normotensive and 17 cases developed HDP. Out of 17 cases ,8 cases had mild HDPs and 9 cases had severe HDPs. The prevalence of HDP was 11.7% in my study.

The mean age of the cases for my study was 27.86(Group B normotensive ) 29.87 (Group A1, mild hypertensive ) and 29.11 (Group A2 ,severe hypertensive ). There was no statistically significant correlation found between the age and the occurrence of HDP.

**Conflict of Interest** - Nil

# References

1.  Alessia Mammaro, Sabina Carrara. Hypertensive Disorders in Pregnancy Journal of Prenatal Medicine 2009; 3 (1): 1-5

2.  Bombrys AE, Barton JR, Habli M et al. Expectant management of severe preeclampsia at 27(0/7) to 33(6/7) weeks' gestation: maternal and perinatal outcomes according to gestational age by weeks at onset of expectant management. Am J Perinatol 2009;26:441-46.

3.  Berg CJ, Chang J, Callaghan WM. Pregnancy-related mortality in the United States 1991-1997. Obstet Gynaecol. 2003;101(2):289-96.

4.  Berg CJ, Harper MA, Arkinson SM. Preventability of pregnancy related deaths. Obstet Gynaecol. 2005;106(6):1228-34.

5 .  Hypertensive disorders , Williams obstetrics ,24 edition , page 728-779

6.  Vigil-De Gracia P, Montufar-Rueda C, Ruiz J.Expectatnt management of severe preeclampsia and preeclampsia superimposed on chronichypertension between 24 and 34 weeks gestation. Eur J Obstet Gynecol Reprod Biol 2003;107(1): 24-7.

7.  Vishal Sharma, Preeti Sharma,Nighat Firdous BETA HCG in Mid Trimester as a Predictor of Pregnancy Induced Hypertension International Journal of Science and Research (IJSR) Volume 5 Issue 9, September 2016.

8.  http://www.ner-fogsi.in/

9.  Gupte Sanjay , Wagh Girija . Preeclampsia–Eclampsia ; The Journal of Obstetrics and Gynecology of India (January–February 2014) 64(1):4–13

10.  Vidyabati RK, Davina H, Singh NK, et al. Serum βhCG levels and lipid profile in early second trimester as predictors of pregnancy induced hypertension. J Obstet Gynecol India. 2010;60(1):44–50. doi: 10.1007/s13224-010-0008-1.

11.  Charu Sharma, Smriti Gupta, Mamta Tyagi ; Maternal & Perinatal outcome in Hypertensive Disorders of Pregnancy in a Tertiary Care Hospital in Northern India; OGIJ 06 00229 Volume 6 issue 6 2017

12.  Mehta B, Kumar V, Chawla S, Sachdeva S, Mahopatra D. Hypertension in pregnancy: A community-based study. Indian J Community Med [serial online] 2015

13.  Mohan BS. Pregnancy induced hypertension and prior trophoblastic exposure. J Obstet Gynecol Ind 2004;54:568-70.

14.  Prakash J, Pandey LK, Singh AK, Kar B. Hypertension in pregnancy: Hospital based study. J Assoc Physicians India 2006;54:273-8.

15.  Bangal VB, Giri PA, Mahajan AS. Maternal and foetal outcome in pregnancy induced hypertension: A study from rural tertiary care teaching hospital in India. Int J Biomed Res 2011;2:595-9.

# Mind Mapping Applied as a Pedagogical Tool for MBBS Students

**Shyma P[1], Jai Prakash[2], Nandkishor[3], Rajajeyakumar M[4]**

*[1]Associate Professor, Department of Physiology, [2]Professor & Head, Pharmacology, [3]Statistician, Community Medicine. The Oxford Medical College, Hospital and Research Centre, Bangalore, [4]Assistant Professor, Department of Physiology, Trichy SRM Medical College Hospital & Research Centre (Affiliated by The Tamilnadu Dr. MGR Medical University, Chennai)*

## Abstract

**Background:** To assess efficacy of mind mapping technique in lecture classes versus traditional didactic lectures on immediate and long-term memory.

**Materials & Method:** 150 First MBBS students, randomized into two groups (75 each), by simple random technique (odd roll number-group 1, even roll number-group 2). Study group (Mind mapping group) Control Group (didactic lecture /non mind mapping group).Batches reversed for another topic to address ethical issues.MCQ test was taken after class for short term memory, after a month for long term memory.

**Results:** Descriptive and inferential statistics (paired t test and independent t test) was used to compare the data. P value of Mind Mapping group when compared to Non Mind Mapping group in terms of both short term and long term memory was highly significant (p< 0.0001) for both topic 1 and 2. P value was not statistically significant when short term and long term memory of mind mapping group was compared. But highly significant p value (p< 0.0001) was observed when short term and long term memory of Non Mind Mapping group was compared.

**Conclusion:** The performance of Mind Map Group is better than the Non Mind Map Group both in terms of short term and long term memory.

Mind mapping used as a pedagogical tool can improve the academic scores of students.

***Keywords:** Didactic lectures, Mind Map, MBBS course, Pedagogical tool, Physiology examinations Reproducibility of concepts,*

## Introduction

Only a moderate number of first-year medical students (60%) could pass the university Physiology examination in the first attempt at our college during the past 3 years. Feedback from students revealed their difficulty in listening to a theory class for an hour, reproducing the concepts delivered by a teacher and the voluminous syllabus to be covered, studied and reproduced for MBBS university exams.

Mind map is a diagram used that offers an overview of a topic and its complex information, allowing students to comprehend, create new ideas and build connections. A mind map has a basic theme placed at the centre and sub branches related to the subject are added to the basic theme.

When the mind map is read, the central word/ sentence forms the starting point and the branch to the top right-hand of the central image is the first branch inspected. [1] Throughout the whole process imagery,

**Corresponding Author:**
**Shyma P,**
Associate Professor, Department of Physiology,
The Oxford Medical College, Hospital and Research
Centre, Bangalore. Mobile No: 8971433432
Email Id: shymarajeev@gmail.com

color and the visual-spatial arrangement of the material are emphasized. Mind maps can be drawn by hand, either as "rough notes" during a lecture or meeting, or as higher quality images when more time is available [2].

For instructional purposes, concept mapping fulfills many important roles; by granting pupils a way to reflect their knowledge about a certain subject, by acting as a tool to ease the burden of studying with the means of providing comprehensive understanding on a given subject, by supporting the creation of new ideas and the way these are organized and by facilitating the learning of new concepts and their intricate relationships. [3]

Visual representation also allows the development of holistic understanding that words alone cannot convey, because the graphical form allows representations of parts and whole in a way that is not available in sequential structure of text. [4]

While no one can actually claim to have invented the mind mapping technique (since people have been using it to learn for ages) Tony Buzan is widely regarded as the author who "patented" it.

Tony Buzan, in his work, claims that the brain's natural preference in receiving and adopting new information is a nonlinear graphical form compared to the traditional and established forms like reading where the information is acquired from left to right (or right to left) and top to bottom[5].

The world is becoming more and more competitive. The quality of performance has become the key factor for personal progress. . In our society academic achievement is considered as a key criterion to judge one's total potential.

Medical college curriculum and syllabus are a difficult and rigorous endeavour, but what exactly makes it so difficult? Teachers are continuously developing and refining their teaching skills. Can a change of teaching methodology make a difference to the students.

In view of this, a study was conducted to evaluate first year MBBS students' performance in Medical Physiology on the basis of constructive teaching method of mind mapping to answer the following questions:

## Aim

To determine whether "Mind Mapping" as a teaching tool, compared to the didactic lectures, improve the performance of first year MBBS students in Physiology

To determine whether the attention span of students and recollection of contents be improved through the technique of teaching through mind mapping.

## Objectives

To assess efficacy of mind mapping technique in lecture classes versus traditional didactic lectures on immediate and long-term memory.

## Materials and Method

All students are admitted to I MBBS course through the National Eligibility Entrance Exam (NEET) year.

150 First MBBS students, randomized into two groups (75 each), by simple random technique (odd number-group 1, even number-group 2)

↓

Study group (Mind mapping group)

Control (didactic lecture /non mind mapping group)

↓

Batches reversed for another topic to address ethical issues.

↓

MCQ test after class for short term memory, after a month for long term memory

## Results

**Table 1 Results of pre- and post test scores of MMT and NMMT groups for Topic A**

| | | Topic A | | |
|---|---|---|---|---|
| | **Short Term** | **Long Term** | | |
| **Academic Performance** | **Pre Test (n=75) (Mean±SD)** | **Post Test (n=75) (Mean±SD)** | **Mean Difference (Outcome)** | **P value** |

***Cont...* Table 1 Results of pre- and post test scores of MMT and NMMT groups for Topic A**

| | | | | |
|---|---|---|---|---|
| Mind Mapping Technique Group1(MMT) | 7.89±1.5 | 7.81±1.24 | 0.08 | 0.7071 |
| Non Mind Mapping Technique Group1 (NMMT) | 4.54±1.74 | 2.74±1.96 | 1.8 | 0.0001 |
| Mean Difference (outcome) | 3.35 | 5.07 | | |
| **P Value** | **0.0001\*** | **0.0001$** | | |

\* Independent "t" test between mean of MMT and NMMT for short term memory

$ Independent "t" test between mean of MMT and NMMT for longt term memory

**Table 2 Results of pre- and post test scores of MMT and NMMT groups for Topic B**

| | Topic B | | | |
|---|---|---|---|---|
| | **Short Term** | **Long Term** | | |
| **Academic Performance** | **Pre Test (n=75) (Mean±SD)** | **Post Test (n=75) (Mean±SD)** | **Mean Difference (Outcome)** | **P value** |
| Mind Mapping Technique (MMT) | 7.51±1.49 | 7.55±1.51 | 0.04 | 0.8525 |
| Non Mind Mapping Technique (NMMT) | 3.7±1.78 | 2.34±1.76 | 1.36 | 0.0001 |
| Mean Difference (outcome) | 3.81 | 5.21 | | |
| P Value | 0.0001\* | 0.0001$ | | |

\* Independent "t" test between mean of MMT and NMMT for short term memory

$ Independent "t" test between mean of MMT and NMMT for longt term memory

Descriptive and inferential statistics (paired t test and independent t test) was used to compare the data. P value of Mind Mapping group when compared to Non Mind Mapping group in terms of both short term and long term memory was highly significant (p value 0.0001) for both topics 1 and 2. P value was not statistically significant when short term and long term memory of mind mapping group was compared for both topics 1 and 2. But highly significant p value was observed when short term and long term memory of Non Mind Mapping group was compared for both topics A and B.

**Graph 1: Bar chart showing comparison of SD pre and post test score (MMT and NMMT) of Topic-A**
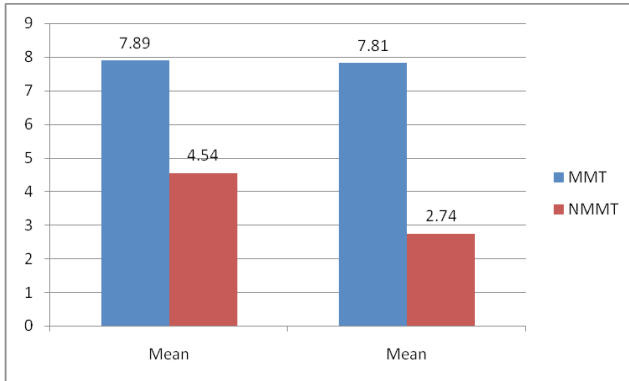


**Graph 2 - Bar chart Showing Comparision of the Mean of Pre & Post Test Score Of Topic-A**
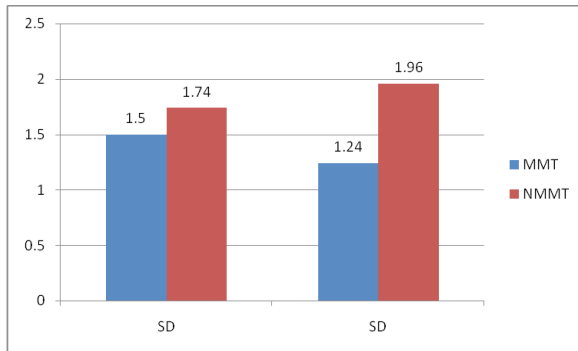


**Graph 3 -Bar chart Showing Comparision of the SD of Pre & Post Test Score Of Topic-B**



**Graph 4- Bar chart Showing Comparision of the Mean of Pre & Post Test Score Of Topic-B**
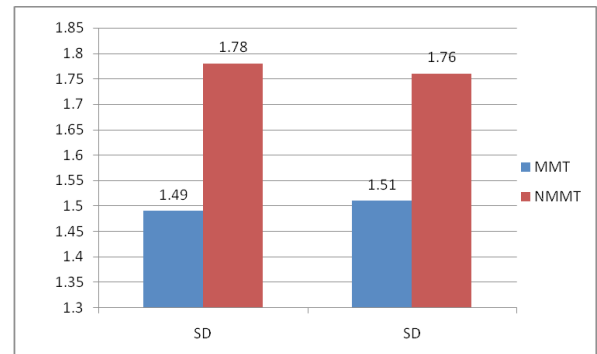


**Group-A**

- Independent "t" test between mean of MMT and NMMT before intervention show statistical significant difference, p value<0.001

- Independent "t" test between mean of MMT and NMMT after intervention show statistical significant difference, p value<0.001

- The difference between pre and post test score of MMT short and MMT long were analyzed by paired t test and was found to be statistically non significant ,MMT p value=0.70
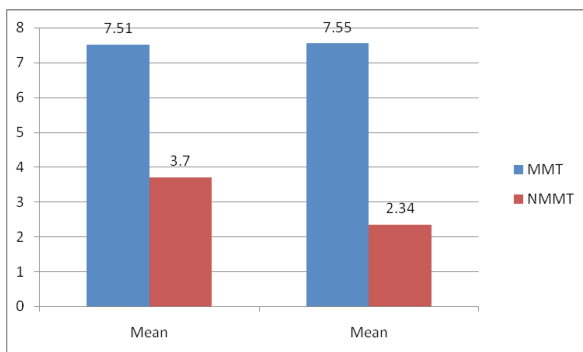
- The difference between pre and post test score of NMMT short and NMMT long were analyzed by paired t test and was found to be statistically significant. NMMT p value <0.001.

**Group-B**

- Independent "t" test between mean of MMT and NMMT before intervention show statistical significant difference, p value<0.001

- Independent "t" test between mean of MMT and NMMT after intervention show statistical significant difference, p value<0.001

'• The difference between pre and post test score of MMT short and MMT long were analyzed by paired t test and was found to be statistically non significant ,MMT p value=0.85

- The difference between pre and post test score of NMMT short and NMMT long were analyzed by paired t test and was found to be statistically significant. NMMT p value <0.001

## Discussion

Analyzing the data shows that, as a strategy to improve memory for written information, the Mind Mapping technique has the potential for an important improvement in efficacy as a pedagogical tool.

In the case of both Group A and B mind map technique resulted in 90% recall of lecture contents, in case of short term and long term memory and it was statistically significantly (p <0.001) .

The recall advantage with the mind map technique was very impressive for the test given immediately after class and also the test given after a month compared to the performance in both the test for the non mind map group.

That differences seen in between the groups in performance suggests that improvements that arise with mind maps are likely to be dependent upon improvements in cognitive processing.

Our results are in concurrence with the results of the study by Jain S who evaluated Mind Mapping in urban school children.[2]

Dr. Roger Sperry, a Nobel Prize winner, is the man who put mind mapping into a scientific frame.

He proved what was commonly known or at least suspected - visual forms of note making and learning were superior to the traditional ones. He showed that the part of the brain which was the latest to evolve "the thinking cap" is (as well as the brain itself) divided into two hemispheres that perform complex tasks which are called cortical skills. These skills include logic, daydreaming, imagination, color recognition and several others. [5].

It is proven that with the use of a Mind Map more of these functions will cooperate and will be in synchronization, creating a lasting information and impression in the brain.

There is definitely more than enough evidence to get a person to start using mind maps as a pedagogical and learning tool.

There are plenty of reasons for implementing Mind Mapping as a go-to technique when teaching or learning something new. Visual stimulants of different colors and symbols combined with connections made will engage one's brain much more effectively than traditional methods.

It is likely that mind map as a pedagogical tool encourages a deeper level of processing of information by the student compared to conventional didactic lectures .

This paper has shown the efficacy of using mind maps as a pedagogical tool in medical colleges.

For the effective implementation of competency based medical education, the use of mind maps as a pedagogical tool should be encouraged while handling large groups and adequate educational materials provided to the faculty for the use of mind maps.

## Conclusions

The performance of Mind Map Group was better than the Non Mind Map Group both in terms of short term and long term memory. Due to the emphasis on Problem Based Learning (PBL) to create a competent medical graduate, it is very important that the teachers formulate a teaching method which will help students improve their memory for written material whilst complementing the deeper level of learning obtained with PBL Such a teaching technique is particularly important given that a current weakness of PBL is that students perform worse on examinations which require recall of factual contents. Mind Mapping stimulates prefrontal cortex (short term memory) and reinforces information in the medial temporal lobe (long term memory). Mind maps are active learning approaches that integrate information on a Meta cognitive level.

The limitations of this study are that it was carried out with two topics in a small number of students.

We would recommend that the technique of Mind Mapping be used in a larger number of students, for longer periods of time to validate its superiority over didactic lectures, especially in the MBBS course.

## References

1.    Farrand P, Hussain F, Hennessy E. The efficacy of the mind map' study technique. Medical education. 2002 May;36(5):426-31.

2.    Jain S. The comprehensive study of how mind mapping technique helps to understand concepts

and ideas in science teaching. International Journal of Scientific and Research Publications. 2015 Dec;5(12):284-6.

3.  Jones BD, Ruff C, Snyder JD, Petrich B, Koonce C. The effects of mind mapping activities on students' motivation. International Journal for the Scholarship of Teaching and Learning. 2012;6(1).

4.  Goodnough K, Woods R. Student and Teacher Perceptions of Mind Mapping: A Middle School Case Study. Paper presented at the Annual Meeting of the American Educational Research Association (New Orleans, LA, April 1-5,2002).

5.  I mind Super User Account 3 Psychology Research Studies Regarding Mind Mapping (internet)( Accessed on Wednesday, December 20, 2017). Available from https://www.imindq.com/blog/3-psychology-research-studies-regarding-mind-mapping#

# Study of Cognitive Evoked Potentials in Type 2 Diabetes Mellitus

**Sindhuja.A[1], R.Ramya[2]**

*[1]Assistant Professor, Department of Physiology, The Oxford Medical College Hospital and Research centre, Yadavanahalli, Attibele, Bangalore. Karnataka, [2]Assistant Professor, Department of Physiology, Apollo Institute of Medical Sciences and Research, Murakambattu, Chittoor, Andhra Pradesh*

## Abstract

**Background:** The completesyndrome of diabetes mellitus,related metabolic aberrations anddiabetic complications is posing a major threat in the 21st century. Cognitive dysfunction is a well known complication of diabetes which continues to be investigated.

**Aims and Objectives:** To evaluate the cognitive functions using electrophysiological ($P_{300}$ latencies) tests in diabetics and non diabetics in the age group of 40 - 59 years with same gender proportion, to analyze whether cognition is affected in diabetics when compared to non diabetics and to know the usefulness of electrophysiological ($P_{300}$latencies) tests in detecting subtle cognitive changes.

**Materials and Method:** The study was conducted on 50 diabetics and 50 non diabetics aged between 40 and 59 years. Cognition was assessed using $P_{300}$ potential.The evoked potential data analysis was done using Student unpaired T test to compare the mean of two groups.

**Results:** The absolute peak latencies of $P_3$ component of endogenous cognitive evoked potentials was significantly prolonged among diabetics (334.8 $\pm$ 20.8) as compared to controls (285.7 $\pm$ 14.9). There was no statistically significant difference between groups when analyzed for $N_2$ in $C_z$and $F_z$.

**Conclusion:** This study identifies prevalence of cognitive dysfunction in diabetic patients when assessed using electrophysiological tests.Good cognitive function is critical to safely manage diabetesand draws attention to various challenges in their management. Clinicians should consider screening for cognitive function in diabetics using $P_{300}$, as it is effective in detecting subtle changes much before their clinical manifestation.

***Key words****: Diabetes Mellitus; Cognition; Cognitive evoked potential; Event related potentials; NIDDM; P_{300}.*

## Introduction

Diabetes mellitus (DM) is taking its place as a main threat to human health in the 21st century. Type 2 DMis a complex metabolic disorder that results from an interaction between genetic predisposition and environmental factors. It accounts for about 90% of all cases of diabetes.[1]The prevalence of DM is rapidly increasing as a result of longevity, urbanization, traditional family structure, mechanized work and associated lifestyle changes.[2]

The number of people with DM worldwide has increased more than two fold over the past three decades. In 2011, approximately 366 million people worldwide had DM, 90% of whom had type 2 DM. The number of people globally with DM is estimated to be 552 million (87 million in India) by 2030, which will represent

**Corresponding Author:**
**Dr. R.Ramya,**
Assistant Professor, Department of Physiology,
Apollo Institute of Medical Sciences and Research,
Murakambattu, Chittoor, Andhra Pradesh. PIN- 517 127. Email: ramyamayudhar@gmail.com,
Ph. No. : 9562452446

7.8% of the total adult population of the world in the age group of 20–79 years.The major burden of DM is in developing countries. 80% cases of DM live in less developed countries.[3]DM is known to have devastating complications on multiple organs in the body. The chronic course of DM is associated with renal, retinal, cardiovascular, nervous system complications like brain hemorrhage, ischemia, peripheral and autonomic neuropathy.[4] Higher brain activities like message comprehension and mnemonic capacities have not been monitored in diabetics due to non availability of biological markers.[5]

A well recognized nervous system complication of elderly diabetics which is less addressed is cognitive dysfunction.[6]

Cognitive impairment in diabetes in terms of speed of processing, memory and attention was observed in patients with DM especially during hyperglycemia.[7]

Cognitive $P_{300}$ potential has been used as a non-invasive, objective and quantitative method to assess higher cognitive functions of human brain. These potentials express the aptitude of human brain to discriminate, classify, decide and memorize the significance of an exogenous stimulus.It serves as a tool to check the sequelae caused by hypoglycemia in hippocampus region, which is evident even before there is clinical manifestation of nervous system damage. [8,9]

Concomitant cognitive dysfunction in diabetic patients generally goes unnoticed. Cognitive dysfunction is an important co morbidity that needs to be addressed in diabetic population.This aroused the need for screening subtle cognitive dysfunction in diabetics which are often unrecognizable.

Recognizing these asymptomatic cerebral changes and modifiable risk factors that influence cognitive changes in diabetes can put forward preventive treatment of the condition and thereby improve the quality of life so as to - "Achieve tightest possible glycemic control with lowest possible chances of hypoglycemia".

## Materials and Method

The present study was conducted in the auspices of research laboratory of Department of Physiology, S.S.Institute of Medical Sciences & Research Centre, Davangere. Study design was of case control type.

**Selection of participants:**

- The study comprised of 100 randomly selected subjects in the age group of 40 – 59 years.

- The case group comprised of 28 diabetic males and 22 diabetic females.

- The control group included 30 non diabetic males and 20 non diabetics females in the same age group.

**Inclusion criteria:**

❖ **Cases:**

- Known diabetic between 40 and 59 years of age.

- Newly diagnosed cases between 40 and 59 years of age.

❖ **Controls:**

- Non diabetics between 40 and 59 years of age in same gender proportion.

- Same socioeconomic and educational background as diabetics.

**Exclusion criteria**

- Hearing loss.

- Old age (above 60years)-Dementia, Alzheimer's disease.

- Stroke.

- Recurrent hypoglycemic episodes.

- Parkinson's disease.

- HIV dementia complex.

- Psychiatric disorders like Schizophrenia and depression.

- Nutritional deficiency - Vitamin $B_{12}$.

- Hepatic encephalopathy.

- Alcoholism.

After obtaining Institutional ethics committee approval and written informed consent, the patient's blood glucose levels were estimated to check their glycemic status on the day of cognitive assessment

Electrophysiological test was recorded using RMS EMG EP MARK II supplied by recorders and medicare system (P) limited, Chandigarh.

## Data Analysis

Data analysis was done using SPSS software. Student unpaired T test to compare the mean of evoked potentials between the two groups.Differences were considered significant at $P<0.001$.

## Results

The study was conducted among diabetics and non diabetics to find out relationship between diabetes and cognition, in the age group of 40-59 years.The data was analyzed by using SPSS 18 and excel was used to generate graphs, tables etc.

Analysis was done using appropriate statistical tests to compare mean values of different parameters between 2 groups.P value was significant at $\leq 0.001$. Mean and standard deviation were calculated for baseline characteristics and electrophysiological test. Cases and controls were compared using Student's unpaired T test. Table 1 shows the comparison between the baseline characteristics of subjects.Table 2 shows the Comparison of cognitive evoked potential in study group and control group. It shows that the absolute peak latencies of $P_{300}$ component of endogenous cognitive evoked potentials was significantly prolonged among diabetics as compared to controls. Graph 1 illustrates that Amplitude of $P_{300} - N_2$ is higher in cases than that of controls.

**Table 1: Baseline physical characteristics.**

| Parameter | Case | Control | P* Value |
|---|---|---|---|
| Age (Years) | 52.7+7.1 | 50.06+6.3 | 0.052 |
| Pulse (bpm) | 73.8+7.1 | 74.72+6.1 | 0.46 |
| SBP (mmHg) | 127.6+13.0 | 126+7.6 | 0.45 |
| DBP (mmHg) | 77.8+6.2 | 79.2+4.0 | 0.17 |
| BMI (Kg/m2) | 25.3+3.5 | 23.9+3.1 | 0.03 |
| * Student's unpaired t test, | | | |

I.    **Comparison of cognitive evoked potential in study group and control group.**

**Table 2. Cognitive evoked potential.**

| Parameter | Cases | Controls | P* Value |
|---|---|---|---|
| N100 latency (ms) | 76.0 + 20.9 | 77.4 + 19.5 | 0.73 |
| P200 latency(ms) | 167.3 + 26.3 | 165.4 + 29.3 | 0.73 |
| N200 latency(ms) | 185.0 + 31.2 | 179.7 + 29 | 0.30 |
| P300 latency(ms) | 334.8 + 20.8 | 285.7 + 14.9 | <0.001* |
| P300-N2(μV) | 6.0 + 3.8 | 5.8 + 4.7 | 0.79 |
| * Student's unpaired t test, P < 0.001 | | | |
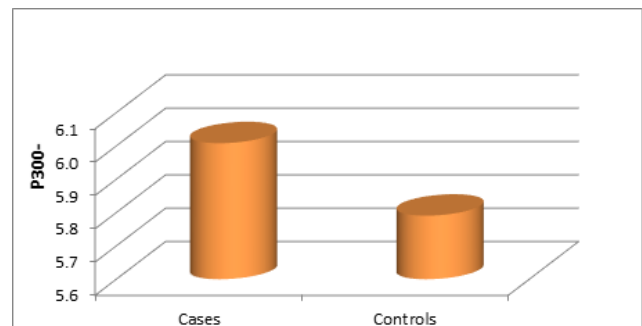


**Graph 1: Amplitude of $P_{300} - N_2$ among cases and controls.**
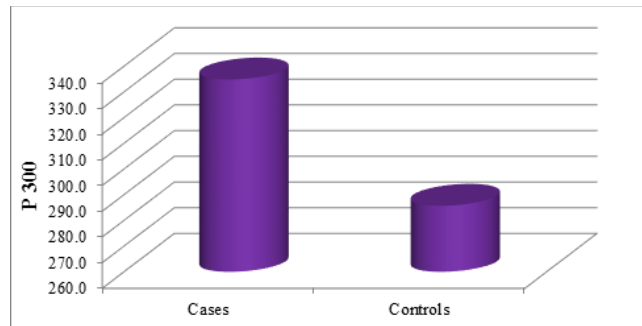


**Graph 2: Latency of $P_{300}$ among cases and controls.**

Mean $P_{300}$ latency for case group was 334.8+20.8ms and that of controls was 285.7+14.9ms. The difference was highly significant with a P value of <0.001. There was no significant difference in amplitude of $P_{300} - N_2$, latencies of $N_2$ and $P_2$.

## Discussion

The diabetic patients complain of loss of memory and poor ability to concentrate. Self management of diabetes including avoidance of hypoglycemia is complex. The cerebral mechanism underlying the cognitive deficits and the responsible brain structures remains to be delineated. They are the topics of intense research, but brain atrophy and vascular changes have

both been assumed.[10]

In previous studies, $P_{300}$ latencies in diabetics were either prolonged or tend to be prolonged in $C_z$ and $F_z$ compared to controls. [11,12] In a study $P_{300}$ latency was not elevated significantly at all sites but longer latency was observed at $F_z$ and $C_z$.[13]

Concerning the amplitude of $P_{300}$ the data are sparse. Most authors did not report measurement of amplitude[14, 12] In others, there was no statistically significant difference between patients and control. [15]

In our study absolute peak latencies of $P_{300}$ component of endogenous cognitive evoked potentials was significantly prolonged among diabetics as compared to controls. The amplitude did not differ significantly in the two groups.

The $N_{100}$ and $P_{200}$ components are believed to reflect the activity in neural areas that are activated by sensory modality and are independent of the attention of the subject.[16] The $N_{200}$ component is related to the unexpectedness of the stimulus. It is regarded as a measure of the time of early stimulus processing, engaging orientation and attention. $P_{300}$ latency is regarded as a measure of stimulus classification, speed, reflection of memory and storage that are initiated in the hippocampus which is considered to be $P_{300}$ generator. The $P_{300}$ amplitude represents online updating of working memory and / or attention process involved in working memory. [17,18]

Since the latencies of $N_{100}$, $N_{200}$ and $P_{200}$ did not differ between cases and controls, prolongation of $P_{300}$ latencies cannot be attributed to delay in perceptual encoding. It is thought to be produced by interaction between frontal lobe, hippocampus, temporal and parietal process. Hippocampus is involved in learning and memory. The delayed $P_3$ in NIDDM therefore reflects inhibition or possible damage of this area.[14] Diabetic milieu causes delay in cognitive processes by interacting with $N_{200}$ and $P_3$ generators in cerebral cortex.[17]

The observed electrophysiological abnormality reflects impairment in attention, memory and speed of information processing which is indicative of early cognitive impairment in diabetes.

Medical care alone in the absence of adequate self-care is rarely effective for chronic illnesses like Diabetes. Self care in diabetes has important clinical and public health implications[19] Since the incidence of Alzheimer's disease is increasing in diabetics, assessing cognitive function is very essential in preventing this co morbidity in the elderly diabetics.[6] Older women with diabetes have poorer cognitive functioning and a more rapid cognitive decline than women with normal blood glucose level.[20] Studies show that improving the metabolic control in IDDM patients with vigorous and continuous insulin, further deteriorates their cognition.[21]

## Limitations of the Study

• The present study is a case control study where subjects were randomly selected from population. A large population based study is required to extrapolate these findings to general population.

• The study does not correlate glycemic status and duration of diabetes mellitus with that of cognitive function, which would establish a better association of cognitive function and glycemic status.

## Conclusion

The aim of this study was to assess cognitive function in type 2 DM and to find out the usefulness of electrophysiological tests in detecting cognitive changes.

In general we found that there was statistically significant difference in $P_{300}$ latency between diabetics and non diabetics, with higher latency in diabetic group when compared to non diabetic group. The amplitude did not differ among the two groups. These results prove that diabetes affects cognition. The cognitive impairment was evident with electrophysiological tests. This highlights the fact that electrophysiological tests are highly sensitive in detecting early cognitive impairment in diabetic patients.

## References

1. Fauci AS, Braunwald E, Kasper DL, Hauser SL, Longo DL, Jameson JL, et al. Harrison's principles of internal medicine. 17th ed. McGraw-Hill; 2008. 2275 -2304.

2. Zimmet P. Alberti K G & Shaw J. Global and societal implications of the diabetes epidemic.

Nature 2001; 414: 782 – 789.

3.  IDF Diabetes atlas, 5th edition. International Diabetes Federation 2012.

4.  The Diabetes Control and ComplicationsTrial Research Group: The effect of intensivetreatment of diabetes on the developmentand progression of long-term complications in insulin-dependent diabetes mellitus. N Engl J Med 1993; 329: 977–986.

5.  Worral G, Moulton N, Briffett E. Effect of type II diabetes mellitus on cognitive function. J Fam Pract1993; 36: 639-643.

6.  Arvanitakis Z, Wilson RS, Bienias JL,Evans DA, Bennet DA. Diabetes mellitus and risk of Alzheimer disease and decline in cognitive function. Arch Neurol 2004; 61: 661 – 666.

7.  Sommerfield AJ, Deary IJ, Frier BM. Acute hyperglycemia alters mood state and impairs cognitive performance in people with type 2 diabetes. Diabetes care. 2004; 27 (10): 2335-2340.

8.  Donchin E, Ritter W, Mccallum WC. Cognitive psychophysiology: The endogenous components of the ERP. InEvent-Related Brain Potentials of Event related Brain Potentials in Man. Academic Press, New York 1978; 349-411.

9.  Di Leo MA, Di Nardo W, Cercones S, Ciervo A, Lo Monaco M, Greco AV, et al. Cochlear dysfunction in IDDM patients with subclinical peripheral neuropathy. Diabetes Care 1997; 20 (11): 1800-1803.

10. Biessels GJ, Koffeman A, Scheltens P. Diabetes and cognitive impairment, Clinical diagnosis and brain imaging in patients attending a memory clinic. J Neurol 2006; 253: 477-482.

11. Hissa MN, Artur CJ, D'Almeida, Cremasco F, Bruin VMS. Event related potential in NIDDM patients without cognitive impairment and its relationship with previous hypoglycemic episodes. Neuroendocrinollet 2002; 23: 226-230.

12. Pozzessere G, Valle E, Crignis S D, Cordischi VM, Fattapposta F, Rizzo PA et al. Abnormalities of cognitive function in insulin dependent diabetes mellitus revealed by P300 event related potential analysis compared with short latency evoked potentials and psychometric tests. Diabetes1991; 40(8): 952-958.

13. Mooradian AD, Perryman K, Fitten J, Kavonian GD and Morley JE . Cortical function in elderly noninsulin dependent diabetes patients. Arch Intern Med 1988; 1489(11): 2369-2372.

14. Tandon OMP, Verma A and Ram BK. Cognitive dysfunction in NIDDM: p3 event related evoked potential study. Indian J PhysiolPharmacol 1999; 43 (3): 383-388.

15. Andreadou E, Mitrakou A, Constantindes VC, Triantafyllou N. Auditory P300 event related potentials in patients with type 2 diabetes mellitus. J Diab Res Clin Met 2012; 1-7.

16. Picton, TW and Hillyard SA. Human auditory evoked potentials. II. Effects of attention. Electroencephalogr Clin Neurophysiol 1974; 36: 191-199.

17. Ritter W, Ford JM, Gaillard AWK, Harter RM, Renault DMK, Rohrbaugh J Cognition and event-related potentials. I. The relation of negative potentials and cognitive processes. Ann N Y Acad Sci 1984; 425:24-38

18. Polich, J. Attention, probability, and task demands as determinants of P300 latency from auditory stimuli. Electroencephalogr Clin Neurophysiol 1986; 63: 251-259.

19. Korff VM, Gruman J, Schaefer J, Curry SJ, Wagner EH: Collaborative management of chronic illness. Ann Intern Med 1997; 127: 1097 –1102.

20. Gregg EW, Yaffe K, Cauley JA, Rolka DB, Blackwell TL, Narayan KMV et al. Is diabetes associated with cognitive impairment and cognitive decline among older women? Arch Intern Med 2000; 160: 174 – 180.

21. Connis RT, Taylor TR, Gordon MJ, Mecklenburg RS, Li ljenguist JE, Stephens JW, Baker MS. Changes in cognitive and social functioning ofdiabetic patients following initiation of insulin infusion therapy. Exp Aging Res 1989; 15: 51-60.

# Effect of Computer Usage on Visual Reaction Time in Information Technology Professionals of Bangalore City

**Sini Sreenivasan[1], M.S. Kusumadevi[2]**

*[1]Post graduate, [2]Professor, Department of Physiology, Bangalore Medical College and Research Institute, Fort, K R Road, Bengaluru, Karnataka, India*

## Abstract

**Background:** Reaction time is the elapsed time between presentation of a sensory stimulus and the subsequent behavioural response. Time taken to respond to visual stimulus gives visual reaction time. In the modern world, the usage of computers for occupational purpose is increasing with increased duration of usage, especially in IT profession. Few studies have been done on reaction time in mobile phone users and video game players, but no study is done in occupational computer users.

**Objective:** To compare visual reaction time between occupational computer users (use computer for work >40hrs/week) and the controls (recreational computer users : <15hrs/week)

**Materials and Method:** Study was done on 30 occupational computer users (use computer for work >40hrs/week) and 30 recreational computer users (<15hrs/week) in the age group 20-35 years, after institutional ethical committee clearance and consent from all subjects. Visual Reaction Time was measured using Human Benchmark Software. Student's t test was used for statistical analysis and p value <0.05 taken statistically significant.

**Results:** Results showed a statistically significant decrease in visual reaction time in occupational computer users with a p value of 0.01

**Conclusion:** Usage of computer for longer hours especially for occupational purpose decreases the visual reaction time in the individuals when compared with those who use it for lesser hours like recreational purpose.

***Keyword:*** *Visual reaction time, IT profession, Occupational computer users, Recreational computer users.*

## Introduction

Reaction time is the elapsed time between presentation of a sensory stimulus and the subsequent behavioural response[1]. Reaction time determines the alertness of a person[2] and also represents the rate of processing of sensory stimulus by CNS and its execution in the form of motor response[3]. There are three types of RT (1) Simple RT: Here there is one stimulus and one response. (2) Recognition RT: Here there is some stimulus that should be responded to and other that should not get a response. (3) Choice RT: Here there are multiple stimulus and multiple responses. The concept of Reaction Time was first described by Abu Rayhan al-Biruni. Franciscus Cornelis Donders (1865), a Dutch Physiologist was the first to systematically measure human RT. He used a telegraph like device invented in 1840 by Charles Wheastone for his study[4]. Reaction time can be to visual stimulus or auditory stimulus. The time required to respond to a visual stimulus is known as Visual Reaction Time (VRT) and to an auditory stimulus is known as Auditory Reaction Time (ART) [5].

In the modern world Computers have become an integral part of daily life[6]. Usage of computer can be for occupational purpose or recreational purpose.

**Corresponding author**
**M.S. Kusumadevi**
Professor, Department of Physiology
Bangalore Medical College and Research Institute
Fort, K R Road, Bengaluru- 560002, Karnataka, India

India has been in the forefront in cyber world with IT industry developing into a major service provider. It was estimated in the 1990's that 40-80 million Visual Display Terminals (VDTs) were there in the workplace. There are approximately six-computers/1000 population with an installation of 18 million Personal Computers (PCs) and their number increasing all the time[7].

Computer use is an interactive activity involving psychomotor ability in using the computer mouse, to sensory and cognitive abilities such as speed of searching for icons, to issues with learning, memory and executive functions[6]. There are no studies done on visual reaction time in computer users. Many studies explored several cognitive domains in computer users. Some previous studies have shown that greater usage of computers can help in maintaining cognitive abilities.

## Objective

To compare visual reaction time between occupational computer users (use computer for work >40hrs/week) and the controls (recreational computer users: use computer for <15hrs/week).

## Materials and Method

This is a case-control study, done on 30 occupational computer users (use computer for work >40hrs/week)[8] and 30 recreational computer users (use computer for <15hrs/week)[8]of either sex in the age group 20-35 years. The study was started after obtaining the ethical clearance from the institutional ethical committee. Occupational computer users were selected from different IT companies across the Bangalore city after considering the inclusion and exclusion criteria's and also age matched controls were selected.

A detailed history taking and relevant clinical examination was done for all subjects. Following which, subjects who had any medical/surgical illness, those having any visual disturbances, alcoholics and smokers were excluded. Also those who are involved in any sports or activities (like video games) which can improve VRT were excluded. Written informed consent was obtained from all subjects and each subject was explained about the whole procedure and objective of the study. Visual Reaction Time (VRT) was assessed using Human Benchmark Software[9]. This software consist of a red coloured screen on the laptop monitor. The subject is instructed to focus on the screen and when the red colour changes to green colour, he/she should press the enter key on the keyboard. Five trials were given and average time was taken as the visual reaction time. Lower the reaction time better is the subject's response to visual stimuli and also attention and fine motor skills are better in the subjects.

Data is presented as mean $\pm$ SD. Students t test is used for comparing the visual reaction time between two groups with p value of < 0.05 as statistically significant.

## Results

**Table 1 shows demographic details of study group and control group**

| Parameters | Study group | Control group |
|---|---|---|
| Age (yrs.) | 25.36 + 0.73 | 26.03 + 0.90 |
| Male | 17 | 14 |
| Female | 13 | 16 |

*Table 2* shows VRT in study group and control group. The VRT in study group (which includes occupational computer users) is seen to be better than the VRT in control group (which includes recreational computer users) with a statistically significant p value of 0.01.

| Test | VRT | p value |
|---|---|---|
| Study group | 385.93 + 13.82 | 0.01* |
| Control group | 409.70 + 12.05 | |
| *p value <0.05 statistically significant | | |

## Discussion

In the present study done on 30 occupational and 30 recreational computer users, the visual reaction time is seen to be better in those who use computer for occupational purpose ie., for longer duration. Lesser reaction time indicates faster response of the individuals to visual stimuli. Thus these individuals have a better sensory motor performance. The rate of processing of sensory stimulus by CNS and its response in the form of motor performances are faster in occupational computer users[1].

There are no previous studies done on VRT in computer users. But few studies are done on cognition in computer users. These studies have proved an improvement in cognitive performances in computer users. The domains explored in those studies include attention, executive functions, learning & memory etc. Patricia A Tun et al in 2010, found that frequent computer activity is associated with good cognitive function particularly executive control[6]. Slegers K et al in 2012 also reported similar findings. They used Visual Verbal Learning Test to measure verbal memory, Letter–Digit Substitution Test to measure processing speed and Stroop Colour Word Test for selective attention and susceptibility to interference. They also found protective effects of the computer use for measures of selective attention and memory in both older (>50yrs) and younger (24-49yrs) participants[10]. Another study done by Juno et al in 2017, found that the occupational computer users have better mental speed, sustained attention, response inhibition and visual memory compared to recreational computer users[8]. Johnson GM et.al, did a study on cognitive processing differences between frequent and infrequent internet users. Four modified cognitive assessment system subtests, each assessing one dimension of the PASS model of cognitive processing (ie., planning, attention, simultaneous and successive processing) were tested. The study showed that frequent internet users excelled in cognitive performances when compared with infrequent internet users[11]. There are few other studies done on mobile phone users and reaction time, which gives contrary results. In a study done by Chinmay Shah et al, VRT and ART were found to be prolonged with concomitant mobile use when compared to controls[12].

Computer use requires continuous inputs from the users and therefore provides mental stimulation. Computer use is an interactive activity which requires visuomotor control like, visual-cognitive input (i.e., viewing a screen) and manual output (i.e., manipulation of peripheral devices such as a keyboard and mouse)[8]. The continuous usage of computer thus increases their sensory motor performances and improvement in the visual reaction time.

## Conclusion

Usage of computer for longer hours especially for occupational purpose decreases the visual reaction time in those individuals.

## References

1.  Shelton J, Kumar GP. Comparison between Auditory and Visual Simple reaction time. Neuroscience & Medicine. 2010;1:30-32.

2.  Dube SP, Mungal SU, Kulkarni MB. Simple visual reaction time in Badminton players: A comparative study.National Journal of Physiology, Pharmacy & Pharmacology.2015;5(1):18-20.

3.  Solanki J, Joshi N, Shah C, Mehta HB, Gokhle PA. A study of correlation between auditory and visual reaction time in healthy adults. Int.J.Med.Public health. 2012;2(2):36-38.

4.  Jain A, Bansal R, Kumar A, Singh KD. A comparative study of visual and auditory reaction times on the basis of gender and physical activity levels of medical first year students. International Journal of Applied and Basic Medical Research. 2015;5(2):124-27.

5.  Bhabhor MK, Vidja K, Bhanderi P, Dodhia S, Kathrotia R, Joshi V. A comparative study of visual reaction time in table tennis players and healthy controls. Indian J.Physiol Pharmacol. 2013;57(4):439-442.

6.  Tun PA, Lachman ME. The association between computer use and cognition across adulthood: use it so you won't lose it? Psychol Aging. 2010;25(3): 560–568.

7.  Sharma AK, Khera S, Khandekar J. Computer related health problems among information technology professionals in Delhi. Indian J of Community Med.2006;31(1):36-38.

8.  Cyril JM, Rajaram S. A comparative study of cognitive functions in Occupational & recreational computer users. International Journal of Physiology. 2017;5(2):186-90.

9.  Murray, M.M., Foxe, J.J., Higgins, B.A., Javitt, D.C., Shroeder, C.E. (2001). Visuo-spatial neural response interactions in early cortical processing during a simple reaction time task: a high-density

electrical mapping study. Neuropsychologia, 39, 828-844.

10. Slegers K, van Boxtel MPJ, Jolles J. Computer use in older adults: Determinants and the relationship with cognitive change over a 6 year episode. Comput Human Behav. 2012;28(1):1–10.

11. Johnson GM. Cognitive processing differences between frequent and infrequent Internet users. Comput Human Behav. 2008;24(5):2094–2106.

12. Shah C, Gokhle PA, Mehta HB. Effect of Mobile Use on Reaction Time. Al Ameen J Med Sci (2010) 3(2):160-164.

# Effect of Duration of Disease and Glycemic Control on Attention, Executive Function and Visual Reaction Time in Type 2 Diabetes Mellitus Patients of Bangalore

**Sini Sreenivasan[1], M.S. Kusumadevi[2]**

[1]*Post graduate, [2]Professor, Department of Physiology, Bangalore Medical College and Research Institute, Fort, K R road, Bengaluru, Karnataka, India*

## Abstract

**Background**: Type 2 diabetes mellitus a common endocrine disorder is on constant rise in the world. Diabetes mellitus is associated with premature mortality and several complications such as neuropathy, nephropathy and cardiovascular diseases. It can lead to decrease in psychomotor speed, executive functions, verbal memory, processing speed, complex motor functioning, working memory, visual retention and attention. These consequences have been attributed to glycemic control and duration of disease.

**Objective**: **1)** To correlate duration of disease with attention, executive function and visual reaction time in type 2 diabetes patients.

**2)** To correlate HbA1c with attention, executive function and visual reaction time in type 2 diabetes patients.

**Materials and Method**: 60 uncomplicated Type 2 diabetes mellitus patients of either sex, aged 40-60 years were recruited from Diabetic Clinic Victoria Hospital, Bangalore. Ethical clearance & informed consent taken. After detailed examination & history taking, 2ml of fasting blood sample were taken for estimation of HbA1c. The cognitive tests include Digit vigilance test for attention, Stroop test for executive functions and visual reaction time using Human Benchmark software. Correlation was found using Pearson correlation, p value<0.05 taken as statistically significant.

**Results:** The Attention and executive function scores and visual reaction time are positively correlated with both duration of disease and HbA1c. Significant correlation is found with attention and Visual Reaction time with p value<0.05.

**Conclusion:** With progressive increase in the duration of disease and HbA1c levels there is decline in Attention and executive function & increase in Visual reaction time.

*Keywords: Type 2 Diabetes mellitus, Attention, Executive functions, Reaction time, HbA1c.*

## Introduction

Type 2 diabetes mellitus is a common endocrine disorder[1] and is on constant rise in the world.[2] Diabetes mellitus is associated with premature mortality[3] and

**Corresponding author**
**M.S. Kusumadevi**
Professor, Department of physiology
Bangalore Medical College and Research Institute
Fort, K R road, Bengaluru- 560002, Karnataka, India

several complications such as neuropathy, nephropathy and cardiovascular disease.[1] It can also affect brain leading to accelerated cognitive decline and an increased risk of dementia.[4] In Cardiovascular Health Study, the prevalence of mild cognitive impairment was 19% in individuals of age > 65years and 29% in those aged >85years.[3] Cognitive decline can be seen in both type 1 and type 2 diabetes mellitus patients, but the affected domains remain distinct in these two types with executive functions, memory, learning, attention and psychomotor efficiency being more affected in type 2 diabetes mellitus

patients. In diabetes patients, the executive functions are particularly important as they involve behaviours, such as insight into a particular problem, problem-solving, judgment, stopping or changing old behaviours and starting new habits, thus affecting the individual's self-care[5]. In long standing diabetes mellitus there can be signs of autonomic dysfunction, slowing of reflexes and increase in reaction time to a number of external stimuli of different modalities[2]. The reason for all these consequences of diabetes has been linked to the degree of hyperglycaemia as measured by HbA1c which is a marker of chronic hyperglycemia and also the duration of disease. There are some, but not all studies showing a negative impact on cognitive functioning in type 2 diabetes individuals with longer duration and elevated HbA1c levels[6]. The ACCORD-MIND Study evaluated the relationship between type 2 diabetes mellitus and glycemic control and proved that poor glycemic control can lower the cognitive function[5]. The present study is designed with the objective to correlate duration of the disease and HbA1c with attention, executive function and visual reaction time.

## Objective

1) To correlate duration of disease with attention, executive function and visual reaction time in type 2 diabetes patients.

2) To correlate HbA1c with attention, executive function and visual reaction time in type 2 diabetes patients.

## Materials & Method

This is a cross-sectional study done on 60 type 2 diabetes mellitus subjects of either sex under the age group of 40-60 years from Diabetic Clinic, Victoria hospital, Bangalore. The study was started after getting ethical clearance from the Institutional Ethical Committee. The subjects who are able to understand English were enrolled for the study. Written informed consent was taken and each subject was explained about the whole procedure and objective of the study.

A detailed history taking and relevant clinical examination was done for all subjects. Following which, subjects who had hypertension, dyslipidaemia, and any diabetic complications were excluded. Also, diabetic subjects on insulin, those with visual disturbances were excluded. Fasting venous blood samples (2ml) were taken for estimation of HbA1c. HbA1c is measured

in BIORAD D-10 machine using latex agglutination inhibition assay. Cognitive tests that measured performances in specific domains of interest were chosen. This includes Digit Vigilance Test (DVT) for attention and Stroop Test for executive functions. Visual Reaction Time, a measure of attention and fine motor skills were also considered.

**Digit Vigilance Test:** DVT was administered according to the instructions provided in the NIMHANS Neuropsychology Battery[7]. This test consist of numbers 1-9 arranged randomly and placed in rows in a sheet. Digits are closely packed on the sheet in 50 rows and 30 digits per row. The subject is instructed to cancel the digits 6 and 9 as fast as possible without missing targets or cancelling wrong numbers. Time taken for completion of the test forms the score and was noted using a stopwatch. Lower score indicate better sustained attention.

**Stroop test:** This test was administered according to the instructions provided in the NIMHANS Neuropsychology Battery[7]. The test consist of a paper in which the colour names BLUE, GREEN, RED, YELLOW are printed. Colour of the print occasionally corresponds with the colour designed by the word. There are 16 rows and 11 columns. The subject is instructed to read the words column wise as fast as possible. Time taken to read all the 11 columns were noted down using stopwatch. Next the subject was asked to name the colour in which the word was printed. The time taken to name all the colours in the column wise was also noted. Reading time and naming time were converted into seconds. Reading time was subtracted from naming time to get the Stoop effect score. Lower score indicates better executive functions.

**Visual Reaction Time:** VRT was assessed using Human Benchmark Software[8]. This software consist of a red coloured screen on the laptop monitor. The subject is instructed to focus on the screen and when the red colour changes to green colour, he/she should press the enter key on the keyboard. Five trials were given and average time was taken as the visual reaction time. Lower the reaction time better is the attention and fine motor skills.

**Statistics:** Data presented as Mean $\pm$ Standard deviation. Pearson correlation was used to correlate duration of disease and HbA1c on attention, executive function and visual reaction time. P value <0.05 was considered statistically significant. Statistical analysis

was done using Microsoft Excel 2013.

## Results

**Table 1 Shows demographic details of the study group**

| Parameters | Study group |
|---|---|
| Age (yrs.) | 54.01+ 4.41 |
| BMI (kg/m2) | 26.52+1.95 |
| Male | 32 |
| Female | 28 |

*Table 2* shows the correlation between duration of disease and different tests done on 60 type 2 diabetes mellitus subjects. Here duration of disease was positively correlated(r=0.31) with score of Digit Vigilance Test and p value statistically significant (0.014). A positive correlation (r=0.20) was also seen with Visual Reaction time and p value statistically significant (0.045). Stroop test score also shows a positive correlation (r=0.14) but without any statistical significance.

| Test | r | p value |
|---|---|---|
| DVT | 0.31 | 0.014* |
| VRT | 0.20 | 0.045* |
| STROOP TEST | 0.14 | 0.255 |
| *P value <0.05 statistically significant | | |

*Table 3* shows the correlation between HbA1c and different test done. Here HbA1c was positively correlated(r=0.55) with the score of Digit Vigilance Test and p value statistically significant (0.01). A positive correlation (r=0.31) was also seen with Visual Reaction time and p value statistically significant (0.01). Stroop test score also shows a positive correlation (r=0.13) but without any statistical significance.

| Test | r | p value |
|---|---|---|
| DVT | 0.55 | 0.01* |
| VRT | 0.31 | 0.01* |
| STROOP TEST | 0.13 | 0.30 |
| *P value <0.05 statistically significant | | |

## Discussion

Type 2 diabetes mellitus individuals are at increased risk of developing cognitive dysfunction. There is 1.5 times more likelihood of developing cognitive decline and frank dementia when compared with those individuals without diabetes[3]. The present study reveals that both duration of the disease and HbA1c has got a negative impact on certain cognitive domains. With increase in duration of the disease and poor glycemic control, there is a decrease in sustained attention and executive function and an increase in reaction time to visual stimuli.

Sustained attention was tested by DVT. It refers to the capacity to attend a task for a required period of time. Right fronto-parietal network mediates sustained attention. Executive functions tested by Stroop test indicate Response inhibition by the brain. Response Inhibition measures the ease with which a perceptual set can be shifted both to conjoin changing demands and by suppressing a habitual response in favour of an unusual one. Pre-frontal areas are essential for response inhibition.[7] Visual reaction time was tested for attention and fine motor skills. It indicates the time taken for processing of sensory stimulus by central nervous system and its execution in the form of motor response.[2]

The cause for cognitive dysfunction in diabetes patients is said to be due to a combination of factors like hyperglycemia, vascular diseases, hypoglycemia and insulin resistance.[5] Glucose serves as a fuel for the brain and is necessary for cognitive performances. Hyperglycemia alters the cognitive functions through a variety of mechanisms like, polyol pathway activation, increased formation of Advanced Glycated End products (AGEs), diacylglycerol activation of protein kinase C and increased glucose shunting in the hexosamine pathway. Hyperglycemia decreases the glucose availability to the brain by impairing the transfer of glucose across the Blood-Brain-Barrier and between the intra and extracellular fluids in the brain, thus impacting cognitive performance. Even a reduction in availability of acetylcholine caused by decrease transport of glucose across the BBB is attributed to cognitive decline.[9] Diabetes Mellitus affects the peripheral nerves in the somatosensory and auditory system, slows psychomotor responses and has cognitive effects all of which may affect reaction times. Some studies states that axonal degeneration of both myelinated and unmyelinated fibres, axon shrinkage, axon fragmentation, thickening

of basement membrane and micro thrombi delays motor nerve conduction velocity, thus delaying the reaction time.[2]

Finding in the present study are in consistent with the study done by Lamport and Colleagues in 2009. The study concluded that worsening of glucose tolerance is associated with cognitive impairment, the most prevalent of which are deficits in verbal memory but also include attentional control, executive function and processing speed deficits.[10] In a cross-sectional analysis done by Shorr et al. on 378 high functioning diabetes subjects, higher HbA1c but no fasting plasma glucose levels were consistently associated with lower scores on two cognitive tests.[11] There are few limitations for the study. It would have been more effective if a large study group was used which can represent the general population.

## Conclusion

Type 2 diabetes mellitus is a risk factor for cognitive impairment. In the present study, HbA1c which is a marker of chronic hyperglycemia and duration of disease are correlated with few cognitive domains like attention, executive function and reaction time. With increase in duration of the disease and poor glycemic control, sustained attention and executive functions are declining. Also there is an increase in visual reaction time. Diabetes is a disease which require proper self-care and monitoring. The decline in cognitive functions can affect their activities like glucose monitoring, medications or insulin injection patterns, diet and exercise timing. Hence identification of cognitive impairments and their relation with Diabetes is an important step for prevention of cognitive decline.

## References

1. Biessels GJ, Van Der Berg E, Craen AJM, Gussekloo J, Westendrop RGJ. The impact of diabetes mellitus on cognitive decline in the oldest of the old: a prospective population- based study. Diabetologia (2006); 49:2015-23.

2. Sidhu J, Mittu S, Sidhu H. Visual reaction time changes in the type 2 diabetics and non-diabetics. Arch.Appl.Sci.Res.2015; 7(7):59-61.

3. Yaffe TC, Gerstein HC, Williamson JD, Lazar RM, Lovato L, Miller ME et al. Relationship between baseline glycemic control and cognitive function in individuals with type 2 diabetes and other Cardiovascular risk factor. Diabetes Care feb 2009; 32(2):221-25.

4. Ruis C, Beissels GJ, Kappelle LJ, Donk M, Gorter KJ, Rutten G. Cognition in the early stages of type 2 diabetes. Diabetes care 2009; 32:1261-65.

5. Munshi MN. Cognitive dysfunction in older adults with diabetes: What a clinician needs to know. Diabetes Care 2017; 40:461-67.

6. Gluck ME, Ziker C, Schwegler M, Thearle M, Votruba SB, Krakoff J. Impaired glucose regulation is association with poorer performance on the Stroop task. Physiol Behav 2013 October 2; 122.

7. Rao SL, Subbakrishna DK, Gopukumar K. NIMHANS Neuropsychology Battery 2004.

8. Murray, M.M., Foxe, J.J., Higgins, B.A., Javitt, D.C., Shroeder, C.E. (2001). Visuo-spatial neural response interactions in early cortical processing during a simple reaction time task: a high-density electrical mapping study. Neuropsychologia, 39, 828-844.

9. Kodl CT, Seaquist ER. Cognitive Dysfunction and Diabetes Mellitus. Endocr Rev. 2008 Jun; 29(4):494-511.

10. Lamport DJ, Lawton CL, Mansfield MW, Dye L. Impairments in glucose tolerance can have a negative impact on cognitive function: a systematic research review. Neurosci Biobehav Rev. 2009; 33:394–413.

11. Shorr R, de Rekeneire N, Resnick NK, Yaffe K, Somes GW,Kanaya M. Glycemia and cognitive function in older adults using glucose-lowering drugs. J Nutr Health Aging 2006.Jul-Aug; 10(4):297-301.

# A Study to Quantify & Compare Stress Levels & Lipid Profile in Working & Non- Working Women of Bangalore

**Sneha G Deshapande[1], Girija B[2]**

[1]Post-Graduate Student, [2]Professor & HOD, Department of Physiology, Bangalore
Medical College & Research Institute, K R Road, Bengaluru

## Abstract

**Background**- Dyslipidemia is prevalent worldwide, mental stress & sedentary life style being major risk factors. Among working women lack of sleep, long working hours, commuting, meeting deadlines amount to physical stress that is part and parcel of job commitment. In non-working women there is availability of house help at hand & online facilities which reduce their physical activity. Many family issues & odd working hours may add to stress in them. Present study intended to quantify stress levels & compare it with lipid profile in working & non-working women.

**Objective**-

1) To quantify stress levels & lipid profile in working & non- working women.

2) Compare stress & lipid profile in working & non-working women.

**Materials & Method**- The study is done on 60 working & non- working women of Bangalore in age group of 20-40 years. Subjects with history of DM, HTN, CVD, thyroid disease were excluded. Informed consent was taken from all participants. After general examination & history taking -Stress score was assessed with perceived stress scale questionnaire. Lipid profile was assessed with 2ml venous sample after 8hrs of fasting. Students 't' test is used for statistical analysis.

**Results**- Stress levels, LDL & Total cholesterol levels are significantly higher in working women. HDL levels were lower in same with P value of <0.05.

**Conclusion**- Dyslipidaemia found in working population may be due to increased stress score in them.

*Key Words*: dyslipidaemia, PSS score, working women, stress.

## Introduction

Dyslipidaemia is highly prevalent among women[1]. The management of dyslipidaemia is a cornerstone in the prevention of both primary and secondary cardiovascular events, such as myocardial infarction, ischemic stroke, and coronary death[1]. It is estimated that one in every two women die of a heart-related disorder, which represents more deaths than due to cancer, chronic lung conditions, and accidents combined. There is a gap of approximately 10 years in mortality rates between women and men[1]. Dyslipidaemia contributes to a major portion of Cardio-vascular disease. Mental stress & sedentary life style being the major risk factors[2].

Women are the magnificent creation of God, a multi-faceted personality with the power of benevolence, adjustability, integrity and tolerance[3].

Women in India have come a long way after independence[4]. In Bengaluru, women constitute about 39% of the workforce[5]. They have started to reach the highest places and to occupy the most exciting positions of power within the society which is leading women to face more stress at work place[6].

**Corresponding Author:**
**Girija B2**
Professor & HOD, Department of Physiology,
Bangalore Medical College & Research Institute,
K R Road, Bengaluru

In modern times, higher education facilitated women's entry into gainful employment. [1]

Working women experience high amount of stress in their life. Women in modern global world have to play a dual role as housewife and career builder[3]. Families today are seeing rapid changes due to the increased pace of growth and modernization. Indian women belonging to all classes have entered into various professions which causes stress in their personal and professional life. Among working women lack of sleep, long working hours, commuting, meeting deadlines amount to physical stress that is part and parcel of job commitment[7]. In these women there is also a lack of physical activity due to lack of time in managing both professional & personal life.

In non-working women there is lack of physical activity leading sedentary type of life style. It may be due to modernisation and availability of all facilities at hand, like online grocery shopping, online banking, and house help is also handy further reducing the physical activity in them. They are also stressed in order to meet varied demands of the family members, like odd working hours of partners, schedule of children etc,. This causes stress and lack of physical activity paving way for development of dyslipidaemia in them[2].

Our study aims at assessing the stress levels in both the groups and compares the lipid profile in them.

## Objective-

1) To quantify stress levels & lipid profile in working & non- working women.

2) Compare stress & lipid profile in working & non-working women.

## Materials and Method

The study was conducted on 60 working and non-working women of Bangalore aged between 20-40 years who were following sedentary life style during December 2017 to May 2018. Informed consent was taken from all participants and ethical clearance was obtained from institutional ethical committee. The participants were divided into 2 groups Group 1 comprising of non-working women and group 2 comprising of working women with 30 participants in each group. Subjects were matched for both age and BMI, women who were in their menstruating phase were excluded from the study.

Subjects with BMI > 30kg/m$^2$, history of Smoking, Alcohol Intake, Substance Abuse, Dyslipidemia, Diabetes Mellitus, Hypertension, Psychiatric Illness were excluded from the study.

Subjects from both groups were given the Perceived stress score questionnaire[8] and were asked to answer the questionnaire. Has 10 questions, Scores range from 0-40, The answers are graded on a 5-point Likert Scale ranging from never=0, almost never = 1, sometimes = 2, fairly often = 3, to very often = 4, Positively framed questions 4, 5, 7, and 8 are reverse scored, that is never = 4 to very often = 0, and the scores are summed, with Higher scores indicating more perceived stress.

2 ml of Blood samples were obtained from all the participants at 7 am after 8 hours of fasting, for analysis of lipid profile in the biochemistry sample collection room of victoria hospital, Bangalore.

Data was analyzed using Microsoft excel 2010, expressed as Mean $\pm$ SD. Students 't' test is used for statistical analysis. P value of <0.05 was taken as statistically significant.

## Results

Working women showed significant increase in PSS score than Non-working women.

**Table 1: Demographic data of the participants**

|  | Group 1 | Group 2 | P value |
|---|---|---|---|
| AGE | 30.9+5.9 Yrs | 30.5+4.5 Yrs | 0.811 |
| BMI | 23.42+2.28kg/m2 | 24.69+3.28 kg/m2 | 0.087 |
| PSS Score | 20.2+2.99 | 25.3+3.90 | <0.001* |

**Table 2: Lipid profile of all participants.**

| Group 1 |  | Group 2 | P value |
|---|---|---|---|
| 125.1+14.41 | Total Cholesterol | 146+18.41 | <0.001* |
| 44.3+14.06 | HDL | 38.2+5.71 | 0.0318 |
| 90.54+9.81 | LDL | 117.8+30.38 | <0.001* |
| 116.1+45.92 | Triglycerides | 137.8+19.54 | 0.020 |
| 83.5+11.68 | Non-HDL cholesterol | 107.9+19.40 | <0.001* |
| 2.82+0.76 | TC/HDL ratio | 3.9+0.73 | <0.001* |

**\* indicates statistically significant change** Working women have higher levels of Total cholesterol, LDL, triglycerides, Non- HDL cholesterol & total cholesterol HDL ratio, the increase was statistically significant with p value <0.001.

## Discussion

Working women have more stress than the non-working women. Women are Expected to be perfect home maker & mother. Ones who go out of their way often end up with a stressful life[7]. Stress in general and job stress in particular-part & parcel of their life[4]. Job stress- alters the homeostasis by Altered sleep wake cycle. Abnormal eating patterns. Stress is inevitable due to Increased competition, lack of co-operation from both sectors of life[9].

Chronic Stress leads to altered lipid metabolism by several mechanisms which interact with each other leading to Dyslipidemia[11] . The following figure is a schematic representation of the cascade of events which leads to Dyslipidaemia.
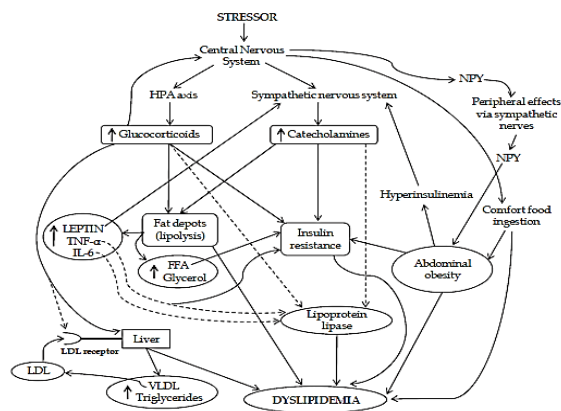


**Figure 1. Hypothalamic-pituitary-adrenal axis (HPA), low-density lipoprotein (LDL), very low- density lipoprotein (VLDL), free fatty acids (FFA), neuropeptide Y (NPY), tumor necrosis factor (TNF-α), interleukin 6 (IL-6). Solid arrows show stimulatory effects; dashed arrows indicate inhibitory effects.**

Chronic stress causes the activation of HYPOTHALAMO-PTUITARY ADRENAL axis, leading to increased secretion of glucocorticoids and catecholamines, which in turn causes hyperinsulinemia, insulin resistanc, increased fat deposits and increased levels of **leptin**[11] . They also inhibit the activity of Lipoprotein lipase which inturn causes accumulation of fats leading to DYSLIPIDEMIA. Another mechanism through which stress acts is the release of Neuropeptide Y which causes abdominal obesity which in turn triggers the cascade of insulin resistance and dyslipidemia[11] . Another proposed mechanism is that chronic stress induces binge eating phenomenon in which person eats comforting foods which are loaded with fats and carbohydrates which in turn leads to Dyslipidemia[11] .

In a study done on working women in Hyderabad, there was a higher level of occupational stress among married working women as explained in terms of traditional trends, demands of society and responsibilities assigned to them as a mother, wife, and homemaker[12]. In another study done on software employees, there was no significant difference in the level of job stress among married and unmarried working women[13].

## Conclusion

Working women had more perceived stress score than non- working women. Dyslipidaemia found in working women may be attributed to chronic stress in them.

### Limitations

It is done only in one pattern of job, can be done in many patterns of job to compare the effects. Done only in women of Bangalore- need to do in all cities to generalise the findings.

### Implications

Stress relieving activities can be promoted in the job sectors in order to reduce the Stress in working population. Following activities can be tried

- Meditation

- Art promoting activities- painting/singing/dancing.

- Yoga exercise

- Routine get-togethers.

**Conflict of Intrest:** Nil

## References

1. Phan BAP, Toth PP. Dyslipidemia in women: Etiology and management. Int J Womens Health. 2014;6(1):185–94.

2. Kasper DL, Fauci AS, Hauser SL, Longo DL, Jameson JL LJ. Harrion's Principles of Internal Medicine. 19th ed. USA: McGraw-Hi11 Education; 2015. 1930-1934 p.

3. Adhikari H. Anxiety and Depression : Comparative Study Working and Non-Working Mothers etween Anxiety and DepressionComparative Study Between Working and Non-Working Mothers Anxiety and Depression : Comparative Study between Working and Non-Working Mothers. Glob J Hum Soc Sci. 2012;12(1):1–9.

4. Bhuvaneshwari M. A Case Study on Psychological and Physical Stress Undergone By Married Working Women. IOSR J Bus Manag. 2013;14(6):38–44.

5. Susan.R. Women at work. In: F.H.Tyrer, K.Lee, editors. A synopsis of Occupational Medicine. 2nd ed. Bristol: John wright & sons; 1985. P 81.

6. Rukmini.S. Bangalore on top: Women form 39% of city's workforce. The Times of India Bangalore [Internet]. 2013 May 13;

7. Dr. Latha Krishnan. Factors Causing Stress among Working Women and Strategies to Cope Up\n. IOSR J Bus Manag [Internet]. 2014;16(5):12–7. Available from: http://www.iosrjournals.org/iosr-jbm/papers/Vol16-issue5/Version-5/C016551217.pdf

8. Cohen S. Perceived stress scale - 10-item version. Psychology. 1994;1–3.

9. Islahi F. Women , Work and Stress : Analysing the Liaison. Int J Indian Psychol. 2017;5(1):145–57.

10. Costa G. Shift work and health: Current problems and preventive actions. Saf Health Work. 2010;1(2):112–23.

11. Klein F, das Neves VJ, Costa R, Sanches A, Sousa T, Sampaio Moura MJC, et al. Dyslipidemia Induced by Stress. Dyslipidemia - From Prev to Treat. 2012;(May 2014).

12. Parveen N. Investigating Occupational Stress Among Married and Unmarried Working Women. Dep Psychol Univ Sindh, Jamshoro. 2009;5(January):21–37.

13. J sudha; Dr P Karthikeyan. Work life balance of women employees: a literature review. Int J Serv Oper Manag. 2018;29(1):100.

# Assessment for Hypertension Risk Score in Pune Urban Population

**Sudhir Gavali[1], Deepak Tambe [2], Shishir Joshi[3], A Pranita[2], J.S. Kharche[1], Gayatri Godbole[2], Anuradha Joshi[4]**

*[1]Associate Professor, [2]Assistant Professor, Department of Physiology, Bharati Vidyapeeth (Deemed to be University) Medical College, Pune, [3]Consulting Physician, Sanjivani Health Care Center, Pune, [4]Professor & Head, Department of Physiology, Bharati Vidyapeeth (Deemed to be University) Medical College, Pune*

## Abstract

**Introduction:** The World health statistics 2012 report highlight on the growing problem of the non-communicable diseases burden due to urbanisation and change in the life styles. According to the WHO report, one in three adults worldwide, has hypertension a condition that causes around half of all deaths from stroke and heart disease.In this study, we are using the common modifiable risk factors like obesity and exercise status to evaluate risk of hypertension in later life. So the present study is planned to calculate the risk score for the development of hypertension. **Material & Method**: It was a cross sectional study. Study was conducted on about 521 individuals between the age group of 18-50 yrs in Pune urban population. Risk factors like obesity,physical activity .family history and depression were used to assess score **Results**: In our study total 521 participants were included out of which 27%,58% and 15 % have mild, moderate and high risk of developing hypertension respectively in future. **Conclusion**: The study concludes that majority of urban Pune population have moderate risk of hypertension. This highlights need of preventable measures like counselling sessions, regular follow up, life style modification in form of increased physical activity and healthy diet.

*Kaywords* : Hypertension , Risk score, Urban population

## Introduction

The World health statistics 2012 report highlight on the growing problem of the non-communicable diseases burden due to urbanization and change in the life styles.

According to the WHO report, one in three adults worldwide, has hypertension a condition that causes around half of all deaths from stroke and heart disease.[1]

Most of these people remain undiagnosed, although many of these cases could be treated with low-cost medications, which would significantly reduce the risk of death and disability from heart disease and stroke.

Also left untreated can lead to cardiovascular disease, blindness and kidney failure.[2]

Globally cardiovascular disease accounts for approximately 17 million deaths a year, nearly one third of the total .Of these, complications of hypertension account for 9.4 million deaths worldwide every year. Hypertension is responsible for at least 45% of deaths due to heart disease and 51% of deaths due to stroke [3]

In an analysis of worldwide data for the global burden of HTN, 20.6% of Indian men and 20.9% of Indian women were suffering from HTN in 2005. The rates for HTN in percentage are projected to go up to 22.9 % and 23.6 % for Indian men and women, respectively by 2025. Recent studies from India have shown the prevalence of HTN to be 25% in urban and 10% in rural people in India. According to the WHO 2008 estimates, the prevalence of raised BP in Indians was 32.5% (33.2% in men and 31.7% in women). However, only about 25.6% of treated patients had their

**Corresponding Author:**
**Dr. Deepak Tambe,**
Address: 805, Neelam Apartments, Shree Mahalaxmi Nagar, Chaitraban, Bibwewadi, Pune – 411037.
Phone no.:8796974215.
Email: sudhirgavali0069@gmail.com

BP under control, in a multicenter study from India on awareness, treatment and adequacy of control of HTN. [5]

Previously a disease of the middle-aged and elderly hypertension has recently escalated in all age groups and is now being identified in younger and younger age groups, including adolescents and children, especially in high-risk populations adds to the total cardiovascular disease burden and threatens to slow progress toward the goals for heart disease and stroke mortality through the remainder of the decade. [5,6] This underscores the need for mass awareness and screening programmes to detect HTN at an early stage.

Physical inactivity and overweight or obesity with genetic component and stress are the important risk factors for the development of hypertension.

In this study, we are using the common modifiable risk factors like obesity and exercise status to evaluate risk of hypertension in later life. So the present study is planned to calculate the risk score for the development of hypertension.

## Material & Method

It was a cross sectional study. Study was conducted on about 521 individuals between the age group of 18-50 yrs in Pune urban population.

Nature of the study was explained to all the subjects & written consent was taken. All subjects was assessed for risk score as per the Proforma using standard techniques. The waist circumference was taken at the midpoint between the iliac crest and the lower border of the ribs after a normal expiration. Systolic and diastolic blood pressure (BP) was be recorded in the right arm in supine position by using a mercury sphygmomanometer after 5 min rest. Two readings of SBP and DBP were recorded and the mean of each was used.

Assessment of physical activity was done according to Global Recommendations on Physical Activity for Health, WHO 2010 [6] An individual is defined as physically active if he/she is performing at least 150 minutes of moderate-intensity aerobic physical activity throughout the week such as walking 30 min /day for 5 days or doing at least 75 minutes of vigorous-intensity aerobic physical activity such as football and netball, as well as activities such as dancing , running and swimming laps throughout the week or an equivalent combination of moderate- and vigorous-intensity activity.

An individual is defined as physically less active if he/she is performing less than 150 minutes of moderate-intensity aerobic physical activity throughout the week such as walking less than 30 min /day for & not for maximum 5 days / week or doing at less than 75 minutes of vigorous-intensity aerobic physical activity such as football and netball, as well as activities such as dancing , running and swimming laps throughout the week or an equivalent combination of moderate- and vigorous-intensity activity.

An individual is defined as physically inactive if he/she is performing only activities of daily living.

Depression is common among persons with hypertension. Unfortunately the diagnosis of depression is often missed by health care professionals. Using a short questionnaire as the WHO-5 can help to monitor emotional well-being. For assessment of stress or psychological well being a simple screening tool i.e WHO -5 well being index was used.

The WHO-5 Well-being Index [7] is a short, self-administered questionnaire covering 5 positively worded items, related to positive mood (good spirits, relaxation), vitality (being active and waking up fresh and rested), and general interests (being interested in things). It has shown to be a reliable measure of emotional functioning and a good screener for depression. Administering the WHO-5 Well-being Index takes 2-3 minutes and can be integrated in clinical routine, both in primary and secondary care.

Each of the five items is rated on a 6-point Likert scale from 0 (= not present) to 5 (= constantly present). A score below 13 indicates poor wellbeing .If the raw score is below 13 or if the patient has answered 0 to 1 to any of the five items it is an indication for further assessment to confirm depression.

The levels of blood pressure were taken as per the Indian guideline of blood pressure for hypertension.[8]. We score each of the factors in multiples of 10 for easy counting. Statistical analysis was done by nonparametric test using computerized 'SPSS' software version 10.

**Table 1: Risk score for Hypertension**

| Risk factors | HTN Score |
|---|---|
| Age | |
| < 35 | 0 |
| 35 – 49 | 20 |
| > 50 | 30 |
| Waist Circumference (cm) | |
| Female / Male | |
| <80 / <90 | 0 |
| 80 – 89 / 90 – 99 | 10 |
| > 90 / > 100 | 20 |
| Physical activity | |
| Physically active | 0 |
| Physically less active | 10 |
| Physically inactive | 20 |
| WHO - 5 Well being score | |
| > 13 | 0 |
| < 13 / 0 or 1 | 10 |
| Family history | |
| No family history | 0 |
| Either parent (Mother /Father ) | 10 |
| Both parent | 20 |
| Systolic BP | |
| <130 | 0 |
| 130-139 | 10 |
| Diastolic BP | |
| <85 | 0 |
| 85-89 | 10 |
| Total Score | |
| Max score | 120 |
| Mild risk | <50 |
| Moderate risk | 50-70 |
| High risk | >80 |

## Result

**Table 2: Hypertension Score**

| Hypertension score | | | % |
|---|---|---|---|
| Mild risk | <50 | 141 | 27 |
| Moderate risk | 50-70 | 303 | 58 |
| High risk | >80 | 77 | 15 |
| Total | | 521 | 100 |

In our study total 521 participants were included out of which 27%,58% and 15 % have mild, moderate and high risk of developing hypertension respectively in future.

## Discussion

It was a cross sectional study .Study was conducted on about 521 individuals between the age group of 18-50 yrs in Pune urban population. In our study total 521 participants were included out of which 27%, 58% and 15 % have mild, moderate and high risk of developing hypertension respectively in future.

As per the risk score maximum number of participants i.e 58 % (303) were in the moderate risk and 77 i.e. (15%) were at high risk may need to take early interventions with drug treatment after detailed investigations. The 141 i.e. 27% were in the mild risk may need to take precautions regarding weight loss or physical activity stress management so that they should not progress for high risk.

In various studies [9,10] presence of cardiometabolic risk factors (such as central obesity and high BMI) may have been the contributing factors for high prevalence of HTN in urban and rural parts of India. Increase in HTN with advancing age was shown by various studies. It was observed that sedentary lifestyle and central obesity also increases risk for hypertension.

The differences in HTN prevalence between urban and rural areas noted in our study could be explained by the differences in socioeconomic conditions, risk factors, and quality of healthcare services provided. Lifestyle changes (harmful dietary practices and sedentary habits) occurring because of rapid urbanization and economic progress in urban areas have also contributed to the growing epidemic of HTN in urban areas of India.

Prevalence of Physical Inactivity of 50.2%. Overall prevalence of Hypertension was 65.1% . There was a linear positive correlation between Age with Diastolic Blood Pressure and Systolic Blood Pressure. [11]

In one of the study they found positive correlation between depression and hypertension. The underlying causes of depression need to be addressed and community programs need to be initiated to raise awareness regarding long-term complications of untreated depression . [12]

Thus, if hypertension and it's related complications are detected at an early stage it will help to reduce morbidity and mortality related to hypertension.

## Conclusion

The study concludes that majority of urban Pune population have moderate risk of hypertension. This highlights need of preventable measures like counselling sessions, regular follow up, life style modification in form of increased physical activity and healthy diet.

**Ethical Clearance** was taken from institutional ethical committee.

**Source of Funding** Bharati Vidyapeeth Deemed to be University

**Conflict of Interest** – Nil

## References

1. Global health risks: mortality and burden of disease attributable to selected major risks. Geneva, World Health Organization, 2009 http:// www. who. int/ healthinfo/ global_ burden_ disease/ Global Health Risks_report_full.pdf).

2. A global brief on hypertension Silent killer, global public health crisis April 2013 .WHO reference number: WHO/DCO/WHD/2013.2

3. Global Recommendations on Physical Activity for Health, WHO 2010 . http://www.who.int/ dietphysicalactivity/factsheet_recommendations/ en/index.html

4. WHO(Five) Well being index. 1998 Version Psychiatric Research Unit WHO Collaborat ing Centre in Mental Health

5. Indian Guidelines Management of Hypertension 2001. Hypertension India 2001; 15 (2): 1-34.

6. Gupta R, Pandey RM, Misra A, Agrawal A, Misra P, Dey S, et al. High prevalence and low awareness, treatment and control of hypertension in Asian Indian women. *J Hum Hypertens* 2012; 26:585–593

7. Meshram II, Arlappa N, Balkrishna N, Rao KM, Laxmaiah A, Brahmam GN. Prevalence of hypertension, its correlates and awareness among adult tribal population of Kerala state, India. *J Postgrad Med* 2012; 58:255–261 [PubMed] [Google Scholar]

8. Joshi SR, Saboo B, Vadivale M, Dani SI, Mithal A, Kaul U, et al. SITE Investigators Prevalence of diagnosed and undiagnosed diabetes and hypertension in India: results from the Screening India's Twin Epidemic (SITE) study. *Diabetes Technol Ther* 2012; 14:8–15 [PubMed] [Google Scholar]

9. Hypertension and its risk factors in tea garden workers of Assam. Hazarika NC, Biswas D, Narain K, Kalita HC, Mahanta J Natl Med J India. 2002 Mar-Apr; 15(2):63-8

10. Kinra S, Bowen LJ, Lyngdoh T, Prabhakaran D, Reddy KS, Ramakrishnan L, Gupta R, Bharathi AV, Vaz M, Kurpad AV, Smith GD, Ben-Shlomo Y, Ebrahim Socio demographic patterning of non-communicable disease risk factors in rural India: a cross sectional study S BMJ. 2010 Sep 27; 341():c4974.

11. Physical Inactivity and Its Association with Hypertension in Adult Female Population of Srinagar, India: a Community Based Cross-Sectional Study Abdul Rouf1, Mahbooba Rasool2, Mohammad Salim Khan3, Mohd Saleem Sheikh2 National Journal of Community Medicine. Volume 9( 9) , 2018 : 693-697

12. Samar Mahmood , Syeda Z Hassan , Muqadus Tabraze, Mohammad O Khan, Iqra Javed, Ameer Ahmed , et al . Prevalence and Predictors of Depression Amongst hypertensive Individuals in Karachi, Pakistan. Cureus. 2017 Jun; 9(6): e1397.

# Serum Uric Acid Level in Primigravidae with Pre-Eclampsia: A Case Control Study from Karnataka

**Veena H C[1], Manjunatha S[2]**

[1]*Assisstant Professor, Kodagu Institute of Medical Sciences, Madikeri, Karnataka,*
[2]*Professor and HOD, Dept of OBG, Kodagu Institute of Medical Sciences, Madikeri, Karnataka*

## Abstract

**Introduction:** Pregnancy inducedhypertension is an exclusive condition affecting 10% ofpregnant women. The study of uric acid in serum is an interesting problemespecially in normal pregnancy and pregnancy inducedhypertension (PIH). **Objective:** To compare the changes of serum uric acid level in healthy non pregnant, PIH and normotensive pregnant women. **Methodology:** Cross sectional observational study conducted in the Obstetrics and Gynecology department, hospital, Navodaya medical college , Raichur, Karnataka involving 30 each pregnant, PIH and non-pregnant women. Serum uric acid levels were measured and analysed the data using SPSS 23 version software. **Results:** Comparison ofmean serum uric acid level in pregnant normotensive (4.14±1.05) and in PIH women (6.03±1.61) was found significant. Comparison of mean serum uric acid level in pregnant normotensive women (4.14±1.05) and in non pregnant women ( 3.39±0.5) found significant. Mean serum uric acid level in PIH women was 6.03±1.61 whereas in non pregnant women was 3.39±0.5 ($p<0.001$). **Conclusion:**Serum uric acid level is at higher side in PIH women as compared to pregnant normotensives and non pregnant women in our study.

*Key words: Serum uric acid, PIH, preeclampsia, preganancy*

## Introduction

Quality of life of mother and newborn has become an important concern in todays era. Hypertensive disorders are amongst the most common medical disorders during pregnancy. It is also responsible for maternal and perinatal morbidity and mortality.[1]In developing countries 7-10% of all pregnancies complicated by some form of hypertensive disorder and lead to various maternal and fetal complications.[2] Pre-eclampsia and eclampsia is still reported as "a disease of theories" since its etiology is still obscured. The crux in the pathophysiology of pre-eclampsia appears to be endothelial cell dysfunction.[3] Pre-eclampsia is a multisystem disorder characterized by hypertension to the extent of 140/90 mm Hg or more, proteinuria (≥300mg/day) and edema induced by pregnancy after 20th week.[4]We know that serial changes

occur in serum uric acid level in normal pregnancy and pregnancy induced hypertension.[5, 6]The raised levels of uric acid in the pregnancy induced hypertension were considered to be due to its diminished destruction in liver. Stander and Cadden[7] did not find impairment of uric acid excretion, but, Seitchik[8] showed that there was excessive reabsorption of urate by renal tubules in toxaemic conditions.Reduced uric acid clearance secondary to reduced glomerular filtration rate, increased reabsorption, and decreased secretion may be at the origin of elevated serum levels in women with preeclampsia.[9,10]

So, in view of the greater emphasis being placed on maternal and child health in present era, we want to revisit serum uric acid as a useful biomarker, which is extremely cheap & widely available and early screening will definitely help in prevention of medical emergency.

### Objectives:

- To compare the changes of serum uric acid level in healthy non pregnant women and healthy pregnant women

**Corresponding Author**:
**Dr Anitha K,**
Associate Professor, Dept of Physiology, Navodaya Medical College, Raichur, Karnataka.
Email ID dranithaks@gmail.com

- To compare the changes of serum uric acid level in healthy pregnant women and pre-eclampsia patients

- To compare the changes of serum uric acid level in healthy non pregnant women and pre-eclampsia patients

## Methodology

**Study population:** The study was conducted in the Obstetrics and Gynecology department, Navodaya medical college , Raichur, Karnataka. Total 90 women volunteers were selected out of which 30 healthy non-pregnant women, 30 pregnant women and 30 pregnant women with preeclampsia were considered as subjects. 30 healthy non-pregnant women were considered as control. 30 normal pregnant and 30 pre-eclamptic women were taken from the outpatients and inpatients wards of Obstetrics and Gynecology department. Ethical Clearancewas taken fromInstitutionalEthical Committee.

**Study duration:** one year

**Study design:** Case control study

**Inclusion criteria**:

- Age >18 years, singleton pregnancy beyond the 20th week pregnancy was taken for the study

- 30 healthy pregnant women were considered for the study.

- 30 pregnant women with pre-eclampsia who fulfill above criteria was considered.

**Exclusion Criteria**: Patients with history of hyperuricemia, preexisting diabetes, hypertension, renal disease, thyrotoxicosis, liver disease, cardiovascular illness, and symptomatic infectious diseases were excluded.

The diagnosis of preeclampsia was based on the definition of American College of Obstetrics and Gynaecologists. [14]The criteria is as stated below:

1. Systolic blood pressure greater than 140 mm Hg or a rise of at least 30 mm Hg, and/ or

2. Diastolic blood pressure greater than 90 mm Hg or a rise of at least 15 mm Hg (manifested on two occasions at least 6 hours apart), and/ or

3. Proteinuria of 300 mg or greater in 24 hours urine collection or protein concentration of 1gm/ L (on two occasions at least 6 hours apart).

30 normal, healthy non-pregnant female volunteers selected for control study. They were of comparable age, and physical activity. They were non-smoker, not taking tobacco and free from any other metabolic or organic disorders. We collected necessary information about subject in Performa.

**Method of collection of data:**Each subject taking part in the study was explained about the purpose of the study and procedure to be adopted in the study. Informed consent was taken from all the subjects. A detailed history followed by clinical examination was carried out for each one of the subject.

**Procedure:** The blood pressure of the participants were measured with a standard mercury sphygmomanometer. Three readings were taken at the interval of 10 minutes. Proteinuria analysis were performed using standard procedures.Blood samples of participants were taken from right or left cubital vein would be collected in plain tubes and serum levels of uric acid was measured.

Determination of uric acid was carried out by quantitative estimation on colorimetric method by enzymatic uricase method, which has many advantages like single reagent system, one step procedure, prevents protein precipitation. Highly sensitive and specific and the reagent was stable. It is a very speedy method and one can determine uric acid within 5minutes and very small amount of serum was required.

**Statistical analysis:** Data was entered in MS excel sheet and analysed by using SPSS 23.0 version. The quantitative data was expressed as mean and standard deviation. Comparison of mean between two groups was done by suing unpaired t test. A p value less than 0.05 was considered as significant whereas p<0.001 was considered as highly significant.

## Results

**Table 1 depicts distribution of study population according to age group.** In our study we involved 30 women in each group. Commonly observed age group in our study was between 21 to 30 years in all three groups. 93.3% pregnant women, 63.3% PIH women and 60% non pregnant women belongs to this age group.

**Table 2 shows distribution according to mean age group of study population.** Mean age of pregnant normotensive women was 25.53± 3.16 years. Mean age of PIH women was 24.33± 4.70 years. Mean age of non-pregnant women was 21.53± 2.46 years.

**Table 3 explains about comparison of mean serum uric acid level in pregnant normotensive and PIH women.** Mean serum uric acid level in pregnant normotensive women was 4.14±1.05 whereas in PIH women was 6.03±1.61. When we compared the uric acid levels in both groups, it was found to be statistically significant (p<0.001). It means serum uric acid level in PIH group was considerably higher as compared to pregnant normotensive women in our study.

**Table 4 explains comparison of mean serum uric acid level in pregnant normotensive and non pregnant women.** Mean serum uric acid level in pregnant normotensive women was 4.14±1.05 whereas in non pregnant women was 3.39±0.5. When we compared the uric acid levels in both groups, it was found to be statistically significant (p<0.001). It means serum uric acid level in pregnant normotensive group was at higher side compared to non pregnant women in our study.

**Table 5 shows comparison of mean serum uric acid level in PIH and non pregnant women.** Mean serum uric acid level in PIH women was 6.03±1.61 whereas in non pregnant women was 3.39±0.5. When we compared the uric acid levels in both groups, it was found to be statistically significant (p<0.001). It means serum uric acid level in PIH group was at higher side compared to non pregnant women in our study.

**Table 1: Distribution according to age group of study population**

| Frequency | | Pregnant | | PIH | | Non pregnant | |
|---|---|---|---|---|---|---|---|
| | | Percent | Frequency | Percent | Frequency | Percent | |
| Age group in years | ≤ 20 | 1 | 3.3 | 8 | 26.7 | 12 | 40.0 |
| | 21-30 | 28 | 93.3 | 19 | 63.3 | 18 | 60.0 |
| | > 30 | 1 | 3.3 | 3 | 10.0 | 0 | 0 |
| | Total | 30 | 100.0 | 30 | 100.0 | 30 | 100.0 |

**Table 2: Distribution according to mean age group of study population**

| | Mean | Std. Deviation |
|---|---|---|
| Pregnant | 25.53 | 3.16 |
| PIH | 24.33 | 4.70 |
| Non pregnant | 21.53 | 2.46 |

**Table 3: Comparison of mean serum uric acid level in pregnant normotensive and PIH women**

| | Group | N | Mean | Std. Deviation | t | p | Inference |
|---|---|---|---|---|---|---|---|
| Uric acid | Pregnant | 30 | 4.14 | 1.05 | -5.403 | 0.0001 | Highly significant |
| | PIH | 30 | 6.03 | 1.61 | | (<0.001) | |

**Table 4: Comparison of mean serum uric acid level in pregnant normotensive and non pregnant women**

| | Group | N | Mean | Std. Deviation | t | p | Inference |
|---|---|---|---|---|---|---|---|
| Uric acid | Pregnant | 30 | 4.14 | 1.05 | 3.500 | 0.001 | Highly significant |
| | Non pregnant | 30 | 3.39 | .50 | | (≤0.001) | |

**Table 5: Comparison of mean serum uric acid level in PIH and non pregnant women**

| | Group | N | Mean | Std. Deviation | t | p | Inference |
|---|---|---|---|---|---|---|---|
| Uric acid | PIH | 30 | 6.03 | 1.61 | 8.571 | 0.0001 | Highly significant |
| | Non pregnant | 30 | 3.39 | .50 | | (<0.001) | |

## Discussion

In our study we involved 30 women in each group. Commonly observed age group in our study was between 21 to 30 years in all three groups. 93.3% pregnant women, 63.3% PIH women and 60% non-pregnant women belong to this age group. (table 1)

Mean age of pregnant normotensive women was 25.53± 3.16 years. Mean age of PIH women was 24.33± 4.70 years. Mean age of non-pregnant women was 21.53± 2.46 years (table 2).

Ajay kumar Singh et al[11] conducted the study in Uttar Pradesh and reported the mean age 27.40 ± 3.55 years in Normotensives and 27.03 ± 3.91 years in PIH women in his study.

Mean serum uric acid level in pregnant normotensive women was 4.14±1.05 whereas in PIH women was 6.03±1.61. When we compared the uric acid levels in both groups, it was found to be statistically significant (p<0.001). It means serum uric acid level in PIH group was considerably higher as compared to pregnant normotensive women in our study. (table 3)

Ajay kumar Singh et al[11] found that mean serum uric acid level in normotensives was 4.94 ± 0.83 whereas in PIH it was 7.63 ± 0.59. The difference was significant.

Jasmin Diwan et al[12] from Jamnagar, Gujrat reported that mean serum uric acid level was 4.6 ± 0.42 in normal pregnancy and it was 7.6 ± 0.76 in PIH women. They also found the difference between two groups serum uric acid significant.

Patel Tejal et al[13] determined the mean serum uric acid level in the lasttrimester of pregnancy for the normal women to be3.5 ± 0.6 mg% and in preeclampsia group it was 6.4±1.7mg%.

Thereare couple of previous studies that highlights on the uric acid level in preeclampsia and its significant rise.[14-16]

Serum uric acid is one of the parameters used in earlydiagnosis of pregnancy induced hypertension. It was reported that there is positive correlation between hyperuricaemia and hypertension which distinguishes between pregnancyinduced hypertension and chronic hypertension.

Mean serum uric acid level in pregnant normotensive women was 4.14±1.05 whereas in non- pregnant women was 3.39±0.5. When we compared the uric acid levels in both groups, it was found to be statistically significant (p<0.001). It means serum uric acid level in pregnant normotensive group was at higher side compared to non-pregnant women in our study. (table 4). Mean serum uric acid level in PIH women was 6.03±1.61 whereas in non pregnant women was 3.39±0.5. When we compared the uric acid levels in both groups, it was found to be statistically significant (p<0.001). It means serum uric acid level in PIH group was at higher side compared to non pregnant women in our study. (table 5)

Shah C J et.al[17]stated that serum uric acid levels are significantly raised in pre eclampsia than normal pregnancy and there is a high positive correlation with the disease severity in relation to hypertension and proteinuria. Lim KH et al[18]also stressed on the clinical utility of serum uric acid measurements in hypertensive diseases of pregnancy.

In non-pregnant women, hyperuricemia is anindependent predictor of cardiovascular and renaldisease in general population and in chronic hypertension.Elevated uric acid level in maternal blood, presumablydue to reduced renal urate excretion is frequently notedin preeclampsia.

## Conclusion

Serum uric acid level is at higher side in PIH women as compared to pregnant normotensives and non pregnant women in our study. Present study show that uric acid isone of the important laboratory tests for screening of pre-eclampsia.The importance of continuousantenatal surveillance and thereof uric acid by laboratorytest in early identification of suspected and establishedcases of hypertension in pregnancy is thus evident. Thedisease can be identified early and its deterioration preventedby proper management.

**Conflict of Interest :** Nil

**Source of Funding :** Self

**Ethical Clearance** : Obtained from ethical clearance obtained from Human Ethical committee of Navodaya Medical College and Research center, Raichur.

## References

1. Shah M R. Hypertensive disorders in pregnancy. 1st edn published by Jaypee. 2007:1-10.

2. Barrilleaux PS, Martin JN; Hypertensive therapy during pregnancy. ClinObstet Gynecol., 2002;45(1):22-34.

3. Cotter AM, Molloy AM, Scott JM, Daly SF. Elevated plasmahomocysteine in early pregnancy: a risk factor for the development of severe preeclampsia. American Journal of Obstetrics and Gynaecology.2001;185(4):781- 85.

4. Dutta D.C (2001), Hypertensive disorders in pregnancy, in :Textbook.

5. Lind T, Godfrey KA, Otun H. Changes in serum uricacid concentration during normal pregnancy.
Br Jr of obst and Gyne 1984; 91:128-132.

6. Mustaphi R, Gopalan S, Dhaliwal L, Sarkar AK. Hyperuricaemia and pregnancy induced hypertension-Reappraisal. Ind. J med Sci 1996(3);50:68-71

7. Stander HJ, Cadden JF. Blood chemistry in preeclampsia and eclampsia. Am. J of Obst. Gynecol 1934; 28:856-9.

8. Sietchik J. The metabolism of urate in preclampsia. Am. J of obst Gynecol 1956; 72:40-7.

9. Powers RW, Bodnar LM, Ness RB, Cooper KM, Gallaher MJ, Frank MP, Daftari AR, Roberts JM. Uric acid concentrations in early pregnancy among preeclamptic women with gestational hyperuricemia at delivery. Am J Obstet Gynecol. 2006;194:160:e1– e8.

10. Jeyabalan A, Conrad KP. Renal function during normal pregnancy andpreeclampsia. Front Biosci. 2007;12:2425–2437.

11. Singh AK, Kumar R, Singh VK, Srivastava S, Sharma A. Serum uric acid levels in pregnancy induced hypertension preeclampsia. Int J Clin Biochem Res. 2018;5(3):365-368

12. Jasmin Diwan, Chinmay Shah, Dixit, Anand AK. A Comparative Study of Serum Uric Acid Level in Normal Pregnancy, and Pregnancy Induced Hypertension. Int. J. Med. Public health 2011;1(1): 1 39-41

13. Patel Tejal, Dudhat Astha. Relationship of Serum Uric Acid Level to Maternal and Perinatal Outcome in Patients with Hypertensive Disorders of Pregnancy. Gujarat medical journal 2014 ; 69(2):45-47

14. Hickman PE, Michael CA & Potter JM, Serum uric acidas a marker of pregnancy-induced hypertension. *Aust N ZJ Obstet Gynaecol.* 1982;22:198-202.

15. Osakwe CR, Ikpeze OC, Ezebialu IU, Osakwe JO& Mbadugha NN, The predictive value of serum uricacid for the occurrence, severity and outcomes of preeclampsiaamong parturients at nnewi Nigeria. *Niger J Med.* 2015;24(3):192-200.

16. Mazzali M, Hughes J, Kim YG, Jefferson JA, Kang DH, Gordon KL, Lan HY, Kivlighn S & Johnson RJ, Elevated uric acid increases blood pressure in the rat by a novel crystal-independent mechanism. *Hypertension.* 2001;38(5)1101-1106.

17. Shah CJ.serum uric acid levels in PIH. Int. J.Med.

Public health,2011;1(1):39-42

18. Lim KH, Friedman SA, Ecker JL, Kao L and. Kilpatrick SJ. The clinical utility of serum uric acid measurements in hypertensivediseases of pregnancy. Am J Obstet Gynecol. 1998 May;178(5):1067-71.

# Sleep Habits in First Year Medical Students at AIIMS Patna and its Impact on their Academic Performance

**Yogesh Kumar[1], Pramita Dubey[2], Kamlesh Jha[1], Ramji Singh[3]**

*[1]Associate Professor, [2]Senior Resident, [3]Professor, Department of Physiology AIIMS Patna*

## Abstract

**Introduction: -** Sleep is an important biological necessity. Sleep timing and duration affects many functions of our body like, endocrine, metabolic, and neurological functions which are critical to the maintenance of individual health. College students often have erratic sleep schedules, poor sleep hygiene and poor sleep quality, which might affect their performance and cognitive functions. **Objective:** To characterize sleep habits and day and night habits in medical students using sleep quality assessment (PQSI scale) and Epworth sleepiness scale; to estimate how subjective sleep quality is associated with sleep problems in students; to estimate how academic progress is associated with subjective sleep quality. **Materials and Method:** A cross-sectional design- A self-administered paper questionnaire was administered of first-year through final-year MBBS students. Students data on sleep quality was collected routinely as part of orientation program to assess students' need in department of Physiology. Data on academic performance (first professional marks) was accessed from examination controller. Pittsburgh sleep quality index and Epworth sleepiness scale scoring was done. **Results:** A total of 90 medical students of AIIMS Patna, aged 16 to 25 years completed the study. Sleep habits of students shows an extreme range of 2 to 4 hrs minimum sleep to 7 to 9 hrs of maximum sleep. Maximum students take 6 to 7 hrs sleep. No statistically significant difference in academic performance between the different Epworth sleepiness score is observed. Negative correlation was obtained between the PSQI and grade average.

*Keywords: Sleep deprivation, academic performance, ESS score, Global PQSI*

## Introduction

Today, prolonged wakefulness is a widespread phenomenon[1]. Chronic sleep restriction is endemic in modern society[2]. Sleep timing and duration affects many functions of our body like, endocrine, metabolic, and neurological functions which are critical to the maintenance of individual health. If left untreated, sleep disorders and chronic short sleep can lead to heart disease, high blood pressure, Obesity, Diabetes and all-cause mortality chronic disabilities and disorders such as arthritis, kidney disease, pain, human immunodeficiency virus (HIV), epilepsy, Parkinson's disease, and depression are also common due to Sleep deprivation. Among older adults, health-related quality of life decreases due to untreated sleep disorders which affect cognitive and medical functioning of the body, leading to functional limitations and loss of independence, and are associated with increased chances of death[3].

Adequate sleep optimally impacts mental functioning also. It impacts students' performance on examinations and ultimately grades received. The pattern of sleep one experiences in a 24-hour period directly correlates with physical health, mood, and mental functioning. Suboptimal sleep is a national problem, with many not obtaining the recommended 7 hours of sleep each night. Increasing awareness of the positive effects of adequate sleep and increasing the proportion of adults who obtain sufficient amounts of sleep to improve health, wellness, productivity, quality of life, and public safety is a goal for our healthcare system[4] Studies assessing the impact of sleep on academic

**Corresponding Author:**
**Dr Pramita Dubey**
Postal address: Physiology Department,
2nd Floor, Dr. RMLIMS, Gomtinagar, Lucknow,
226010, Email: pramita3107@gmail.com

performance focus primarily on teens, adolescents, and students[4]. Few studies have explored sleep habits in populations of undergraduate medical students. Those studies that have, found sleep complaints were common in medical students and poor sleep habits were correlated with changes in academic performance[5,6]. However, studies that provide knowledge about sleep habits among students have yet to be conducted in India. In a study, the sleep-wake pattern and effect of academic schedules and individual characteristics on the sleep-wake cycle and academic performance were examined. The relation between attention and working memory, and sleep deprivation is well established[7]. In previous studies, long-term memory with sleep deprivation has been measured with a variety of tasks, and the results are variable[1].

Sleep deprivation also affects other parameters. It impairs visuomotor performance, which is measured with tasks of digit symbol substitution, letter cancellation, trail-making or maze tracing. A study suggests that Sleep deprivation impedes engagement of spatial attention, which can be observed as impairments in saccadic eye movements[7]. Reasoning ability during Sleep Deprivation has for the most part been measured with Baddeley's logical reasoning task or its modified versions. Again the results are inconsistent (deteriorated performance was reported by few no effects were noted by others[8,9]. In addition to the cognitive domains described above, total Sleep deprivation affects several other cognitive processes as well. It increases difficulties in utilizing new information in complex tasks requiring innovative decision-making. Deterioration in decision-making also appears as more variable performance and applied strategies, as well as riskier behaviour[9,10].

According to the well-controlled studies, the less sleep obtained due to sleep restriction, the more cognitive performance is impaired[11]. Several tasks have been used in the sleep deprivation studies. For example, motor function, rhythm, receptive and expressive speech, and memory measured with the Luria-Nebraska Neuropsychological Battery deteriorated after one night of SD, whereas tactile function, reading, writing, arithmetic and intellectual processes remain intact[12].

More recent meta-analysis shows that Sleep deprivation causes a significant decrease in both the clinical and overall performance of both residents and non-physicians[13]. Certain studies suggest factors like social and academic demands, part-time jobs and irregular school schedules, affect the sleep-wake cycle of college students[5].

**Aim and objectives:**

1. To characterize sleep habits in medical students using sleep quality assessment (PQSI scale) and Epworth sleepiness scale;

2. to estimate how subjective sleep quality is associated with sleep problems in students;

3. to estimate how academic progress is associated with subjective sleep quality;

4. to estimate the prevalence of self-reported sleep problems in first year medical students at AIIMS Patna.

**Material and method:**

The study was conducted in Department of Physiology, AIIMS Patna after taking ethical clearance. Ninety first year medical students of AIIMS Patna, aged 16 to 25 years were included for the study.

After getting informed consent baseline demographic data was collected. All subjects were made to fill a questionnaire through which sleep quality was assessed. Pittsburgh sleep quality index and Epworth sleepiness scale scoring was calculated based on the responses obtained. Academic performance was assessed based on aggregate of total marks obtained in Anatomy, Physiology and Biochemistry by students in 1st Professional MBBS Examination.

Data obtained is presented using appropriate chart type. Kruskar wallis test is used to find if statistically significant difference occurs in academic performance between the different Epworth sleepiness score
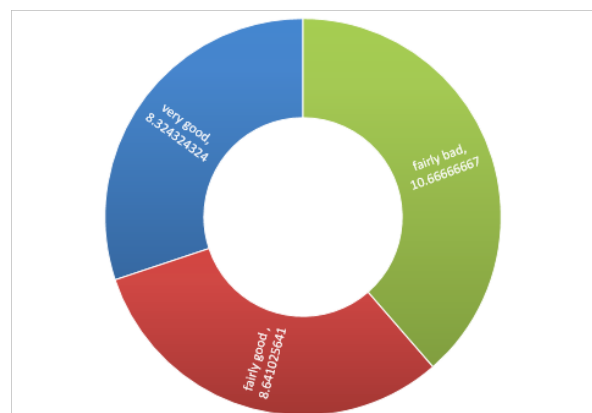
**Results:**



**Fig 1 Relation between ESS score and self-rating of sleep**

As expected those students who rate their sleep as fairly bad were having maximum Epworth Sleepiness Scale indicating chances of day time sleepiness.

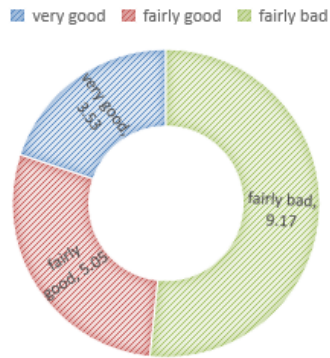## GLOBAL PQSI AND SELF RATING OF SLEEP GROUP



**Fig 2:**

Pittsburgh Sleep Quality Index (PSQI) is a self-report questionnaire that assesses sleep quality over a 1-month time interval. When global PQSI scores were calculated than it was found that students who had reported their sleep quality as fairly bad were having maximum scores (9.17) while those who rate their sleep quality as very good were having least global PQSI score (3.53).
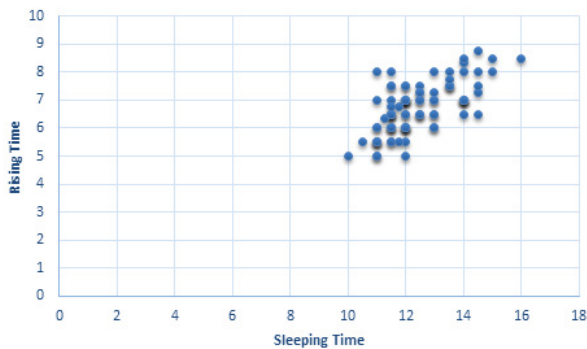


**Fig 3 Relation between rising time and sleeping time**

Time has been converted into continuous scale. On X axis 5 represents 5 pm, 10 represents 10 pm, 15 represents 3 am and 20 represents 8 am. On Y axis 0 represents 12 am, 1 represents 1 am and likewise 10 represents 10 am

Table 1: Kruskar wallis test to find if statistically significant difference occurs in academic performance between the different Epworth sleepiness score

**Kruskar Wallis Test**

| Groups | N | Mean rank |
|--------|-----|-----------|
| 1 | 39 | 45.86 |
| 2 | 16 | 38.94 |
| 3 | 35 | 48.10 |
| Total | 90 | |

Independent / grouping variable= ESS score (Epworth sleepiness score)

Dependent variable= academic performance

Students were divided into three groups on the basis of Epworth sleepiness score; first thirty-nine students with lowest Epworth sleepiness score were taken into first group, next 16 in second group and next 15 with maximum Epworth sleepiness score in third group

Groups: is the classification of students in three groups (1, 2 and 3) on the basis of Epworth sleepiness score

N: is the no. Of students in different groups based on Epworth sleepiness score

Mean rank: Mean rank for each group can be used to compare the effect of different Epworth sleepiness score.

Test statistics

| | Epworth sleepiness score |
|--------|-----|
| Chi square | 1.366 |
| Df | 2 |
| Asymp. sig. | 0.505 |

Kruskar wallis H test shows that there was no statistically significant difference in academic performance between the different Epworth sleepiness score, $\chi^2(2)= 1.366$, p=0.505, with a mean rank percentage marks score of 45.86 for Epworth sleepiness score 1-7, 38.94 for Epworth sleepiness score 8-9 and 48.10 for Epworth sleepiness score 10-15.

**Table 2: Chi square test to find an association between academic performance and PSQI score**

**Chi-Square Tests**

| | Value | df | Asymp. Sig. (2-sided) |
|---|---|---|---|
| Pearson Chi-Square | 44.828[a] | 37 | .176 |
| Likelihood Ratio | 57.269 | 37 | .018 |
| Linear-by-Linear Association | .230 | 1 | .631 |
| N of Valid Cases | 90 | | |

a. 76 cells (100.0%) have expected count less than 5. The minimum expected count is .34.

The value of the test statistic is 44.828

The corresponding p value of the test statistics is 0.176

Since p value is more than significance level ($\alpha = 0.05$), we do not reject the null hypothesis. We conclude that there is no enough evidence to suggest an association between academic performance and PSQI score.

**Table 3: Chi square test to find association between academic performance and early and late risers.**

**Chi-Square Tests**

| | Value | df | Asymp. Sig. (2-sided) |
|---|---|---|---|
| Pearson Chi-Square | 33.375[a] | 36 | .594 |
| Likelihood Ratio | 40.348 | 36 | .284 |
| Linear-by-Linear Association | .017 | 1 | .896 |
| N of Valid Cases | 86 | | |

a. 74 cells (100.0%) have expected count less than 5. The minimum expected count is .30.

The value of the test statistic is 33.375

The corresponding p value of the test statistics is 0.594

Since p value is greater than significance level ($\alpha = 0.05$), we do not reject the null hypothesis. We conclude that there is not enough evidence to suggest an association between academic performance and early and late risers.

**Discussion**

Characterizing sleep habits is necessary in medical students so as to obtain their prevalent sleep pattern and advise changes to improve their sleep hygiene which may affect their academic performance. Sleep habits of students of first year MBBS were studied which showed an extreme range of 2 to 4 hrs minimum sleep to 7 to 9 hrs of maximum sleep. Maximum students take 6 to 7 hrs sleep. It was found that maximum students sleep between 11 pm and 1 am. Sleep quality based on intake of sleep medication was studied. Although maximum students do not take sleep medication , few students (around 15 to 20%) take medication less than once a week and around 5% take medicine more than once a week. This proportion is higher than normal population[14]. It may be because of increased awareness or increased stress among medical students. Maximum students rate their sleep quality as very good or fairly good. It can be said that maximum students have 5 to 9 hrs of sleep which can be said to be optimal period of sleep and be rated as good.

ESS score is widely used in the field of sleep medicine as a subjective measure of a patient's sleepiness during day time. The higher the score, higher is the propensity of person's average sleep in daily life or their day time sleepiness. Figure 1 finds no relation between ESS score and self rating of sleep when ANOVA was applied. However, a higher ESS score is found among students rating fairly bad quality of sleep. While, figure 2 depicts a correlation between PSQI scale and self rating of sleep. PSQI is a self report questionnaire that assesses sleep quality over a 1-month time interval.

Figure 3 shows that those who go to bed early rise early while those who go to bed late rise late in the morning. No relation is found between ESS score and hrs of sleep. A person's need for hrs of sleep may vary from individual to individual. No statistically significant trend was found between hrs of sleep and self rating of sleep, again implying that individual's need varies from person to person. However a trend is obtained.

Further analysis is done to find if academic performance is affected by sleep hygiene. Table 1 shows Kruskar wallis test which shows that there was no statistically significant difference in academic performance between the different Epworth sleepiness score. Table 2 is chi square test to find association between academic performance and PSQI. It shows that there is no enough evidence to suggest an association between academic performance and PSQI score. Table 3 is a chi squre test to find association between academic performance and early and late risers. It also shows that there is not enough evidence to suggest an association between academic performance and early and late risers.

A study[5] determined if sleep deprivation and in a sample of non depressed university students was associated with lower academic performance. A statistically significant negative correlation was obtained between the PSQI and grade average proving that poor sleep quality is associated with lower academic performance. However the findings are not similar in our studies, which need explanation.

A study by Rodrigues RN et al[15] observed that the sleepier students did not achieve as well as the others on their final examinations.

It was shown by Jamaan M. Al-Zahrani et al[16] that there is a high prevalence of excessive day time sleepiness among medical students in Alkharj, Saudi Arabia. The disturbed sleep pattern is not associated with academic performance. Study says that longitudinal investigation are warranted to determine whether prolonged sleep disturbances eventually influence the academic performance of this cohort.

Those with lower PSQI (near 1) have good academic performance. It is also found that those with higher PSQI like 10 an 15 also had good academic performance. Poorest performance is of a student with PSQI score 4 . This implies that academic performance depends upon multiple factors apart from sleep pattern. There is no statistically significant difference in academic performance based on hrs of sleep. However the differences are academically significant. Those who sleep for more than 8 hrs have poor academic performance (acad perf score around 49%) while those with 6 to 8 hrs of sleep have a good academic performance score of around 59%. This implies that good sleep hygiene is necessary for a good academic performance.

## Conclusion

A total of 90 medical students of AIIMS Patna, aged 16 to 25 years completed the study. Sleep habits of students shows an extreme range of 2 to 4 hrs minimum sleep to 7 to 9 hrs of maximum sleep. Maximum students take 6 to 7 hrs sleep. No statistically significant difference in academic performance between the different Epworth sleepiness score is observed. Negative correlation was obtained between the PSQI and grade average.

## References

1.   Paula A and Päivi P-K; Sleep deprivation: Impact on cognitive performance; Neuropsychiatr Dis Treat. 2007 Oct; 3(5): 553–567.

2.   Gregory B N J, Wesensten D R, Thorne M L, Thomas Helen C, Sing D P, Redmond et al; Patterns of performance degradation and restoration during sleep restriction and subsequent recovery: a sleep dose-response study; Journal of sleep research; March 2003;12(1):1-12.

3.   Healthy People. 2020, Office of Disease Health and

Promotion, U.S. Department of Health and Human Services. Sleep health. http://www.healthypeople.gov/2020/topicsobjectives2020/overview.aspx?topicid=38. April 21, 2014.

4. Megan L Z , Matthew J. S, Matthew Song, Leanna M. K, Jingjing Qian, Paul W et al; Sleep Duration and Academic Performance Among Student Pharmacists; Am J Pharm Educ. 2015 Jun 25; 79(5): 63.

5. Ana L D. M, Denise B.F. M, Patrícia F. L &John F. A; The Relationships between Sleep-Wake Cycle and Academic Performance in Medical Students; Journal of Biological Rhythm Research; 2001; 32(2)

6. Veldi M, Aluoja A, Vasar V; Sleep quality and more common sleep-related problems in medical students; Sleep Med. 2005 May;6(3):269-75.

7. Bocca ML, Denise P. Total sleep deprivation effect on disengagement of spatial attention as assessed by saccadic eye movements. Clin Neurophysiol.2006; 117:894–9.

8. Blagrove M, Alexander C, Horne JA. The effects of chronic sleep reduction on the performance of cognitive tasks sensitive to sleep deprivation. App Cogn Psych.1995; 9:21–40.

9. Linde L, Bergstrom M. The effect of one night without sleep on problem-solving and immediate recall. Psychol Res. 1992; 54:127–36.

10. Harrison Y, Horne JA; One night of sleep loss impairs innovative thinking and flexible decision making.; Organ Behav Hum Decis Process. 1999 May; 78(2):128-45.

11. Dinges DF, Pack F, Williams K, Gillen KA, Powell JW, Ott GE, Aptowicz C, Pack AI; Cumulative sleepiness, mood disturbance, and psychomotor vigilance performance decrements during a week of sleep restricted to 4-5 hours per night.; Sleep. 1997 Apr; 20(4):267-77.

12. Kim DJ, Lee HP, Kim MS, et al. The effect of total sleep deprivation on cognitive functions in normal adult male subjects. Int J Neurosci. 2001; 109:127–37.

13. Philibert I; Sleep loss and performance in residents and non-physicians: a meta-analytic examination; Sleep. 2005 Nov; 28(11):1392-402.

14. Maurice M. Ohayon, Epidemiological Overview of sleep Disorders in the General Population; Sleep Medicine Research (SMR) 2011; 2(1): 1-9

15. Rodrigues RN, Viegas CA, Abreu E Silva AA, Tavares P ; Daytime sleepiness and academic performance in medical students. Arq Neuropsiquiatr. 2002 Mar;60(1):6-11.

16. Jamaan M. Al-Zahrani , Khaled K. Aldossari, Imad Abdulmajeed , Sameer H. Al-Ghamdi , Abdullah M. Al-Shamrani and Nawaf S. Al-Qahtani ; Daytime Sleepiness and Academic Performance among Medical Students; Health science journal; 2016, 10(3):13.

# Impact of Fuel Exposure on Ventilatory Function among Petrol Pump Attendants in Thies, Senegal

**Arame Mbengue[1], Mame Saloum Coly[2], Salimata Houndjo Faye[4], Abdou Khadir Sow[3]**
**Mor Diaw[3], Fatou Bintou Sarr[1]**

[1]*Professor,* [2]*Assistant Professor, Laboratory of Physiology, UFR of Health Sciences of Thies, Service Functional Explorations of the Regional Hospital of Thies, Senegal,* [3]*Senior Assistant Professor,* [4]*Assistant Professor, Laboratory Physiology and Functional Explorations, FMPO/UCAD, Dakar, Senegal*

## Abstract

**Introduction:** Petrol pump attendants are exposed to toxic substances in fuels. The release of petrol fumes during fuel refuelling is a repeated and often harmful source of exposure for petrol pump attendants. The purpose of our study was to evaluate the ventilatory disorders related to fuel exposure among petrol pump attendants.

**Materials and Method:** Forty petrol pump attendants have been recruited. The study included a medical survey with a questionnaire that examined anthropometric characteristics, seniority, smoking habits and working conditions. The study included a medical survey with a questionnaire that examined anthropometric characteristics, seniority, smoking habits and working conditions. All our subjects received a basic and post-bronchodilator spirometry. The targeted respiratory functional disorders were bronchial obstruction, restrictive syndrome and mixed syndrome. Obstructive syndrome has been reported on the basis of: FEV1/ FVC and/or FEF25-75% less than 80%. Functional restriction was defined on the basis of a decrease in FVC <80% and the normal FEV1/FVC ratio. The mixed syndrome was retained firstly before the combination of the two obstructive and restrictive spirometric anomalies.

**Results:** We noted 20% of active smokers and 25% of ex-smokers. The average length of service was 9.4 ± 7.05 years. All of the petrol pump attendants used incomplete means of protection. No employee was receiving medical follow-up. Most subjects (52.5%) were unaware of the risks associated with fuel exposure. Respiratory functional disorders were dominated by isolated obstructive ventilatory disorder (OVD) in 45% of subjects and were mainly located in the distal bronchi (65%). Besides, 12.5% of subjects had an isolated restrictive functional disorder (RFD) and a mixed syndrome in 27.5% of cases. Obstructive and mixed ventilatory disorders were more important according to smoking status. The different ventilatory disorders (VD) appeared from the beginning of exposure with a predominance of obstructive ventilatory disorders. Mixed ventilatory disorders increased significantly after 10 years of exposure.

**Conclusion:** Exposure to petrol / diesel fumes has harmful effects on the ventilatory function of petrol pump attendants.

***Key words:*** *petrol pump attendant, fuel, ventilatory disorders, spirometry, Senegal.*

**Corresponding Author:**
**Arame Mbengue,**
Department of Biology, UFR of Health Sciences,
University of THIES, SENEGAL,
email: aramembengue08@live.fr

## Introduction

Personnel working in petrol stations are exposed not only to air pollutants, but also to organic and inorganic components in fuels (diesel oil and petrol). During the distribution chain of these fuels, hydrocarbon fumes are released, leading to overexposure of employees working

in petrol stations, particularly petrol pump attendants. The fuels contain a variety of chemicals including small amounts of benzene and sometimes lead that are absorbed by the human body through the respiratory tract or through skin contact. These can cause respiratory symptoms and impaired ventilatory functions[1]. Animals exposed to diesel exhaust gas have been shown to develop impaired lung function [2,3]. In Senegal, a developing country, the fuel trade is growing considerably with the advent of new local fuel distribution companies. Every day, about fifty employees working in the 26 petrol stations in the city of Thiès are directly exposed to the fuels and the additives they contain. The purpose of this study was to determine the ventilatory disorders related to fuel exposure among petrol pump attendants working in the city of Thiès (Senegal).

## Material and Method

### Participants

The study protocol was approved by the ethics committee of the University of Thiès. We conducted a descriptive and analytical cross-sectional study over a three-month period (29 June to 02 October, 2018). Our study population consisted of fuel-exposed petrol pump attendants, all male. All these workers had at least one year of seniority. The study included a medical survey (questionnaire, clinical examination and spirometry). The questionnaire covered physical characteristics (gender, height, weight, age), seniority in the activity, working conditions, smoking habits. For cigarette smoking intoxication, we have identified active smokers, ex-smokers and non-smokers. Subjects are categorized as smokers: those who smoked during the 30 days preceding the survey, former smokers: those who smoked for more than 30 days but did not smoke during the 30 days preceding the survey; non-smokers are those who had never smoked or who had smoked for less than 30 days. The exclusion criteria were the presence of a thoracic deformity or a progressive cardiopulmonary pathology known at the time of the study.

Spirometric parameters: Basic and post-bronchodilator spirometry were performed in all our subjects. Spirometry was performed using a regularly calibrated Jaeger PNEUMO Care Fusion device coupled with a computer in which data analysis software is installed. Explanations and supporting illustrations were provided in advance regarding the conduct of the examination for better cooperation of the subjects. After the various tests were performed, lung volumes and bronchial flows were examined and the rates of variation of the different parameters were taken into account with respect to the reference standards (ERS/ATS 2005)[4]. The best test was selected taking into account the subject's degree of cooperation and the aspect of the flow-volume curve. Volumes, lung capacities and bronchial flows were examined (basic and post-bronchodilator spirometric parameters). The rates of variation of the various parameters in relation to the reference standards have been taken into account.

The parameters measured were the Maximum Expiratory Volume at the first second (FEV1), the Forced Vital Capacity (FVC), the Tiffeneau ratio (FEV1/FVC) and the maximum expiratory flows (FEF) including FEF75%, FEF50%, FEF25% and FEF25-75%. Obstructive syndrome was defined on the basis of a lower FEV1/FVC ratio (less than 80%) and FEV1 < 80% of the predicted value. The diagnosis of obstructive ventilatory disorders was made according to the level of impairment in the airways:

- total obstruction: FEV1/FVC < 80% of the predicted value and $FEF_{25-75\%}$< 80% of the predicted value,

- proximal obstruction only: FEV1/FVC < 80% predicted value and $FEF_{25-75\%}$ normal,

- distal obstruction only: $FEF_{25-75\%}$< 80% of the predicted value and FEV1/FVC normal.

The functional restriction was retained ahead of a decrease in CVF < 80% of the predicted value and a normal CVF1/CVF ratio (>80%).

## Statistical Analysis

Data collection was based on pre-established survey forms and spirometric data forms using Excel 2013 software. The statistical analysis was done using Excel and Prism software. Qualitative variables were expressed in proportions and quantitative variables in terms of averages and standard deviations. The Student test was used to search for a link between the parameters. The limit of significance was set at $p < 0.05$.

## Results

A total of forty (40) petrol pump attendants were recruited. The average age was $37.77 \pm 3.99$ years with a more represented age group of 40 to 50 years (35%).

The average BMI was $21.77 \pm 3.05$ kg/m² (Table I). The seniority in the job was estimated at $9.4 \pm 7.05$ years. In addition, our subjects worked an average of $76 \pm 12$ hours per week with extremes ranging from 60 to 96 hours. Our study population was mainly composed of non-smokers (55%) with a lower proportion of active smokers (20%) having an average smoking consumption of 5.6 packs per year with extremes ranging from 2 to 13.5 packs per year.

All of our subjects wore safety shoes and uniforms based on the colour of their petrol station. However, none of them used gloves and only one gas station attendant reported that he wore a face mask during his shift. Four (4) employees had received a pre-employment medical examination, representing 10% of our study population. On the other hand, no employee received regular medical follow-up. Nevertheless, 52.5% of the petrol pump attendants recruited were unaware of the occupational risks related to fuel exposure when questioned.

The prevalence of respiratory function abnormalities was 85% in the study. Obstructive, restrictive or mixed ventilatory disorders were noted in our subjects. The most common ventilatory disorders (VD) were isolated obstructive types (45%) which were mainly located in the distal bronchi (65%). Besides, 27.5% of the subjects had mixed ventilatory disorders and a restrictive syndrome in 12.5% of the cases (Figure 1).

Depending on seniority in activity (duration of exposure), we noted at the beginning of exposure (group 1 to 10 years) a significantly higher prevalence of obstructive ventilatory disorders compared to restrictive syndrome (p=0.0001) and mixed syndrome (p=0.02). In addition, mixed ventilatory disorders increased significantly after 10 years of exposure (26.9% of workers with 1 to 10 years of exposure compared to 28.6% of the group with more than 10 years of seniority (p=0.0001) (Figure 2).

Obstructive syndrome was significantly higher among ex-smokers than among smokers (p=0.005). Furthermore, this syndrome is also significantly higher among active smokers compared to non-smokers (p=0.0001). Mixed ventilatory disorders were significantly higher among smokers compared to non-smokers (p=0.0001) (Figure 3).

The analysis of post-bronchodilation spirometry data made it possible to objectify 10% of work-related asthma cases in our study population (Figure 4). We noticed a negative correlation between the base FEV1 and seniority (p= 0.0193 and r=-0.36) (Figure 5).

## Discussion

This study has identified the main work-related risks of the petrol pump attendant job in the informal economy in our country. Paradoxically, the decisive role of the petrol pump attendant in the operation of the petrol station exposes him to the health risks arising from the handling, especially of fuel distribution pistols, with the failure to comply with the safety measures often observed among these exposed groups. Workers at these petrol stations are in continuous contact with fuels and constantly inhale the particles emitted by the vehicles they refuel[5]. Moreover, the need for petrol pump attendants sometimes to sniff the tank cap to check the fuel type in order to avoid errors, results in overexposure to fuel fumes[6]. In our cross-sectional study, the results confirm a ventilatory function impairment of petrol pump attendants exposed to petrol / diesel fumes. Similar results have been observed in previous studies[7,8]. In our study, abnormalities in ventilation function are significant in 85% of the exposed subjects, which could be explained by the lack of use of personal protective measures (PPM) during work, poor air quality in petrol pumps, multiple tasks (refuelling, storage and workplace cleaning), and lack of knowledge about the harmful effects of fuels. Obstructive syndrome predominated in our exposed subjects. Pellegrino et al[9] had found similar results with changes in FEV1 and FEV1 / FVC ratio more common than changes in FVC. The reduction in the FEV1 / FVC ratio may indicate a slight obstruction of the respiratory tract. Our results are consistent with what is found in the observations of Kesavachandrani et al.[10] and Ayres et al.[11] which showed that workers exposed to diesel and automatic exhaust systems had increased respiratory tract resistance. Studies involving particle distribution in the human lung have shown a major impact site in the terminal bronchioles[12] where most chronic obstructive pulmonary disease begins[13]. It is clearly established that the loss of bronchiolar attachment due to the destruction of the extracellular matrix secondary to the deposition of toxic particles is the cause of the reduction in FEF25-75%[14,15].

The restrictive syndrome and the mixed deficit observed in our study seem predictable with regard to the substances handled and the working conditions. Indeed, repeated chemical aggression initially leads to obstruction, secondary alveolitis will lead to pulmonary

fibrosis whose ventilatory expression is the restrictive syndrome [16]. Our study found an increase in respiratory failure among smokers. Many previous studies have shown the potentiating effect of tobacco on respiratory risk at work-related exposures[17,18]. Weisel et al.[19] found that work-related inhalation exposure to benzene could be increased among employees who smoke.

The prevalence of asthma in our subjects is 10%. Studies in healthy and asthmatic adult populations have shown a clear association between exposure to benzene and total volatile organic compounds (VOCs) on the one hand and, the occurrence of asthmatic respiratory manifestations (diagnosed asthma, clinical or functional symptoms suggestive of asthma)[20,21] on the other hand.

The increasing tendency of lung parenchyma involvement through an increase in mixed syndrome relative to seniority in exposure suggests a probable influence of exposure duration on respiratory function. Our results corroborate those of Solanki et al[22] who observed that, as the duration of exposure increases, a progressive decrease in FEF25-75 and FEV1 / FVC, pointing to obstructive pathology and leading to a mixed type of respiratory deficit. In our work, negative correlations were found between job tenure and FEV1. These results could suggest an increase in bronchial muscle tone leading to a certain degree of bronchoconstriction in relation to an irritant effect related to VOC exposure[23].

**Table I: Anthropometric data**

| Anthropometric data | Average | Min | Max |
|---|---|---|---|
| Age (years) | 37.77 ± 3.99 | 21 | 59 |
| Weight (kg) | 71.15 ± 10.54 | 54 | 96 |
| Height (cm) | 180.65 ± 5.74 | 168 | 190 |
| BMI (kg/m²) | 21.77 ± 3.05 | 17 | 27 |

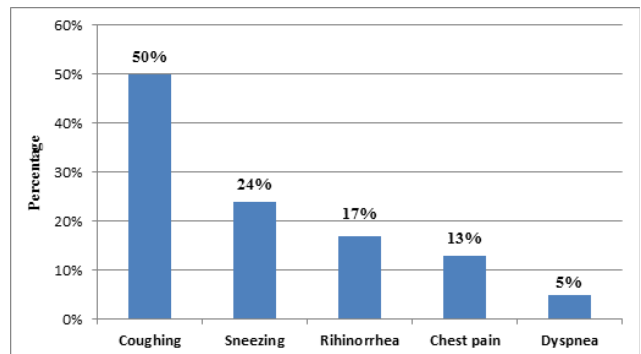**BMI :** Body Mass Index



**Figure 1 : Distribution of the population according to the different ventilatory disorders.**
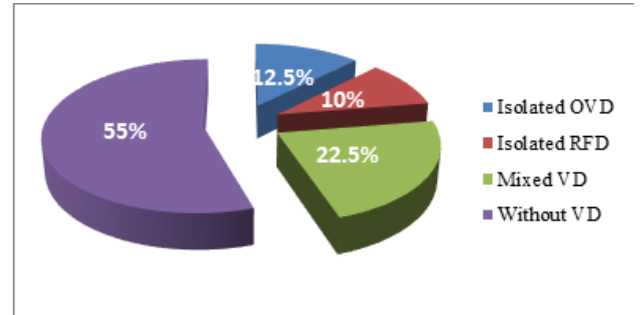


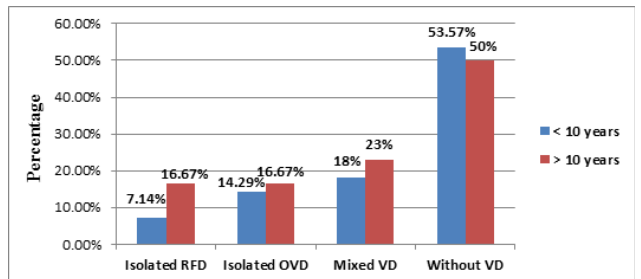**Figure 2: Distribution of ventilatory disorders by seniority (years).**



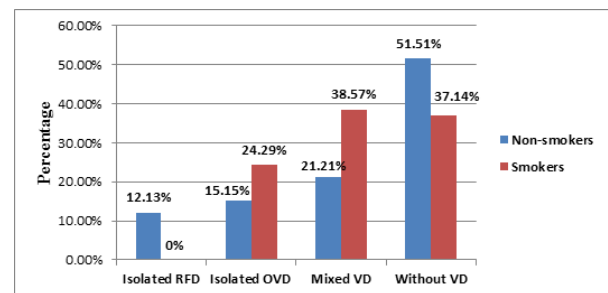**Figure 3: Distribution of ventilatory disorders by smoking status**



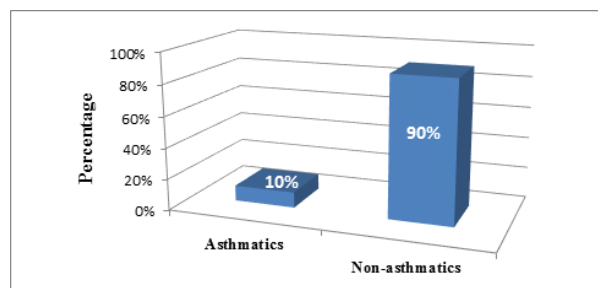**Figure 4: Prevalence of asthma in our population.**



**Figure 5: Correlation between FEV1 and seniority.**

## Conclusion

The prevalence of ventilatory disorders is high in the petrol pump worker environment. Small airways are probably the most critical site of lung damage. Smoking and seniority in the job increase the risks associated with fuel exposure. Frequent health check-ups and regular follow-up of ventilation function would be beneficial. The proper use of safety measures such as protective masks, gloves could be adopted to reduce the health risks associated with chronic exposure to fuels. Enhanced individual and collective protection measures must be implemented to reduce the health risks associated with fuel exposure.

## References

1.    Kaung S, Liang W. Clinical analysis of 43 cases of chronic benzene poisoning, *Chemico-Biological Interactions* 2005 : 153-154

2.    Vinegar A, Carson A, Pepelko WE. Pulmonary function changes in Chinese hamsters exposed six months to diesel exhaust. Env Internal 1981; 5: 369–71.

3.    Mauderly JL. Life-span of rodents inhaling diesel exhaust: results through 30 months. Inhal Toxicol Res Inst Ann Rep 1983 : 305–16.

4.    Brusasco V, Crapo R, Viegi G. Recommandations communes de l'ATS et de l'ERS sur les explorations fonctionnelles respiratoires. Revue des Maladies Respiratoires 2007 **;** 24(3) : 11-14.

5.    Wiwanitkit V, Suwansaksri J, Nasuan P. Research note: Urine trans, trans-muconic acid as a biomarker for benzene exposure in gas station attendants in Bangkok, Thailand. Ann. Clin. Lab. Sci 2001 ; 31 : 399–401.

6.    Ferreira MC, Freire ON. Carga de Trabalho e Rotatividade naFunção de Frentista. RAC 2001; 5 :175–200.

7.    Singhal M, Khaliq F, Singhal S, Tandon OP. Pulmonary function in petrol pump workers: A preliminary study. Indian J Physiol Pharmacol 2007;51:244-5.

8.    Hulke SM, Patil PM, Thakare AE, Vaidya YP. Lung function test in Petrol Pump workers. National J PhysiolPharmaPharmacol2012;2:71-5.

9.    Pelligrino R, Viegi G, Brusasco V, Crapo RO, Burgos F, Casaburi R et al. Interpretative strategies for lung function test. Eur. Respir. J 2005 ; 26 : 948-968.

10.    Kesavachandrani C, Rastogi SK, Anand M, *et al.* Lung function abnormalities among petrol-pump workers of Lucknow, North India. *Current Science* 2006; 90: 1177-1178.

11.    Ayres SM, Evans R, Licht D, Griesbach J. Health effects of exposure to high concentrations of automotive emissions. *Arch Environ Health* 1973; 27: 168-78.

12.    Pinkerton KE, Green FH, Saiki C, Vallyathan V, Plopper CG, Gopal V, *et al*. Distribution of particulate matter and tissue remodeling in the human lung. Environ Health Perspect 2000; 108: 1063-9.

13.    Roux M, Duracher C. Physiologie bronchique. Physiologie humaine appliquée 2$^{ème}$ édition. Editions Arnette 2017: 900 p**.**

14.    Udwalia F.E, Shetye V.M. The Maximum expiratory flow volume curve in normal subjects in India. Chest Journal 1986; 89(6): 852-56.

15.    Moorman WJ, Clark JC, Pepelko WE, Mattox J. Pulmonary function responses in cats following long-term exposure to diesel exhaust. J Appl Toxicol 1985; 5(5): 301–5.

16.    Foray JP**.** Rappel de la situation réglementaire en matière de pollution : quelles implications de la nouvelle directive COV-Solvants sur le secteur de la peinture industrielle ? In : Peintures industrielle et nouvelles technologies. La Documentation Professionnelle, Paris Euroforum 1998: 20p.

17.    Laraqui HC. H, Laraqui HO, Rahhali AE et al**.** Risques respiratoires chez les ouvriers des menuiseries ébénistes artisanales. Rev Mal Respir 2001; 18:615-622.

18.    Wieslander G, Norback D, Edling C. Occupational exposure to water-based paint and symptoms from the skin and eyes. Occup Environ Med 1994; 51:181-6.

19.    Weisel CP. Benzene exposure: An overview of monitoring methods and their findings. Chem. Biol. Interact 2010; 184: 58–66.

20. Rumchev K, Spickett J, Bulsara M et al. Association of domestic exposure to volatil organic. Compounds with asthme in young children. Thorax 2004; 59: 746-751

21. Mitha N, Lévy J, Annesi-Maesano I, Lafortune J, Magnier AM, Ibanez G. Pollution de l'air intérieur et asthme chez l'adulte. Revue des Maladies Respiratoires 2013; 30: 374 - 413

22. Solanki RB, Bhise AR, Dangi BM. A study on spirometry in petrol pump workers of Ahmedabad, India. Lung India 2015; 32 (4) : 347-352.

23. Noertjojo HK, Dimich-Ward H, Peelen S et al. Western red cedar dust exposure and lung function: a dose-response relationship. Am J Respir Crist Care Med 1996; 154 : 968-73

# Lung Function of Traditional Bakers and Pastry Makers Expsosed to Flour Dust in the City of Thies, Senegal

**Arame Mbengue[1], Mame Saloum Coly[2], Mor Diaw[3], Abdou Khadir Sow[3], Salimata Houndjo Faye[4], Fatou Bintou SAR[1]**

*[1]Professor, [2]Assistant Professor, Laboratory of Physiology, UFR of Health Sciences of Thies, Service Functional Explorations of the Regional Hospital of Thies, Senegal, [3]Senior Assistant Professor, [4]Assistant Professor, Laboratory Physiology and Functional Explorations, FMPO/UCAD, Dakar, Senegal*

## Abstract

**Introduction:** Cases of restrictive and obstructive diseases have been reported in workers exposed to flour dust. In Senegal, studies on the lung function of bakery-pastry employees are rare. The purpose of our study was to evaluate the clinical respiratory manifestations and ventilatory disorders (VD) related to flour dust exposure among traditional bakers and pastry makers in the city of Thies, Senegal.

**Materials and Method:** A descriptive cross-sectional study was conducted. Forty subjects were recruited. Subjects received a basic and post-bronchodilator spirometry. The targeted respiratory functional disorders were bronchial obstruction, functional restriction and mixed syndrome.

**Results:** The average length of service at the workplace was 7±6,7 years. Smokers represented 17,5%. A respiratory symptoms were found in 65% of our subjects. Coughing, sneezing and rhinorrhea were more frequent with 50%, 24% and 17% respectively. The most common ventilatory disorders among exposed bakers were of the mixed type (22,5%). On the other hand, 12,5% had an isolated obstructive ventilatory disorder (OVD) and a restrictive functional disorder (RFD) was found in 10%. The obstructive ventilatory disorders were dominated by obstruction of the small airways (80%). The severity of ventilatory disorders was of a moderate deficit type (87% and 62% respectively). Respiratory function abnormalities were more common in the smokers with a prevalence of ventilatory disorders that increased with age of employment. However, the effect of exposure appears to be clear because among non-smokers, 12,13 % of the exposed patients had respiratory functional abnormalities with isolated restrictive functional disorder type.

**Conclusion:** This study showed that bakery and pastry workers have various respiratory symptoms and ventilatory disorders. Smoking and job tenure remain factors that increase these flour dust-related risks.

***Keywords:*** *bakery, flour dust, respiratory symptoms, spirometry, ventilatory disorders*.

## Introduction

Traditional bakers and pastry makers are exposed to flour dust every day, various irritant chemicals and the heat generated during baking. In 1713, Ramazzini reported for the first time otolaryngological and bronchial manifestations in bakers[1]. Since then, a lot of studies have shown that exposure to flour dust causes respiratory symptoms and is associated with impaired lung function[2,3]. The respiratory symptoms are thus common among workers exposed to organic dust. These are often complex and can contain different types of materials from animals and plants. Furthermore, they can cause lung diseases such as work-related asthma, chronic bronchitis and allergic alveolitis[4]. Obstructive and restrictive respiratory diseases have been reported in grain handlers and bakery workers in other parts of the world[5] In Senegal, respiratory risks are probably

**Corresponding Author:**
**Arame Mbengue,**
Department of Biology, UFR of Health Sciences,
University of THIES, SENEGAL,
email: aramembengue08@live.fr

higher because working conditions are very hard and individual and collective preventive measures are absent in this professional sector. The purpose of our work was to evaluate the clinical respiratory symptoms and ventilatory disorders related to flour dust exposure in traditional bakers and confectioners in the city of Thies.

## Materials and Method

A cross-sectional descriptive study was conducted from november 2018 to february 2019 after approval of the study protocol by the ethics committee of the University of Thies.

### Study population

The study population whose free and informed consent was obtained, consisted of 40 bakers, all male, with at least one year's seniority in the profession. Subjects who had a refusal to participate or a known progressive cardiopulmonary pathology were not included in our study.

### Method

The study included a medical survey with a questionnaire that examined physical characteristics, seniority, smoking habits, respiratory symptoms and atopic history. The subjects are categorized as smokers: those who smoked during the 30 days preceding the survey, former smokers: those who smoked for more than 30 days but did not smoke during the 30 days preceding the survey; non-smokers are those who had never smoked or who had smoked for less than 30 days. All our subjects received a complete physical test and then a basic and post-bronchodilator spirometry using a *Jaeger PNEUMO CareFusion* brand spirometer. Lung volumes and bronchial flows were examined and the rates of variation of the different parameters with respect to the reference standards were taken into account (ERS/ATS 2005). The parameters measured were the Forced Expiratory Volume at the first Second (FEV1), the Forced Vital Capacity (FVC), the Tiffeneau ratio (FEV1/FVC) and the maximum expiratory flow rate 25-75% ($FEF_{25-75\%}$). We have defined a restrictive function disorder on the basis of a decrease in vital capacity (VC) of less than 80% and a normal FEV1/FVC ratio (>80%). The obstructive ventilatory disorder is defined by a measured FEV1/FVC ratio of less than 80% of the theoretical value. Small airway obstruction has been defined by a FEV1/FVC ratio of more than 80% and a $FEF_{25-75\%}$ of less than 80% of the theoretical value. FEV1 and FVC

quantify the importance of obstructive or restrictive syndrome according to the following classification: mild ventilatory disorder (70≤FEV1 or VC<80%), moderate ventilatory disorder (50%≤FEV1 or VC<70%), severe ventilatory disorder (30%≤FEV1 or VC <50%) and very severe ventilatory disorder (FEV1 or VC<30%)[6]. In case of obstructive syndrome, a reversibility test was performed 15 minutes after inhalation of 400 mg of salbutamol. The mixed syndrome was retained in front of the association of the two obstructive and restrictive spirometric anomalies[7].

## Statistical Analysis

The data were entered and analyzed using Excel software version 2013. The percentage averages and standard deviations were determined for the quantitative parameters. The Student test made it possible to compare the average. Correlations were also searched using the Pears on Test. The significance rate was a p-value<0.05.

## Results

### Working conditions and work-related exposure

The bread making rooms were mostly poorly ventilated with a very hot environment due to the heat released by the oven during the baking and the lack of a ventilation system. Moreover, these locations served as dormitories for some employees during their off-duty hours. Our subjects worked an average of 48±22 hours per week extremes ranging from 18 to 105 hours/week. The majority of our bakers (58%) worked between 30 and 60 hours a week. However, 27% of the subjects had a weekly working time of more than 60 hours and only 15% worked less than 30 hours per week. Thus, all these working conditions contributed to exposure to air contaminants in rooms that are often crowded, badly maintained and poorly ventilated.

### General characteristics of the population

Our results showed that our subjects were 26±10 years old with an average BMI of 21,22±3,57 kg/m². The average length of service in the workplace was 7±6,7 years (Table1). Smokers represented 17,5% of our study population.

### Prevalence of respiratory symptoms

Respiratory symptomatology was found in 65% of our subjects, 25% of whom had presented a notion of atopy. Coughing, sneezing and rhinorrhea were

more common with 50%, 24% and 17% respectively (Figure1).

### Prevalence of ventilatory abnormalities

The most common ventilatory disorders in exposed subjects were of the mixed type, (22,5%). On the other hand, 12,5% of the subjects had pure obstructive ventilatory disorders and a restrictive functional disorder was found in 10% of the cases (Figure 2). The obstructive ventilatory disorders were dominated by a small airway syndrome (80%). The severity of obstructive or restrictive ventilatory disorders was of a moderate deficit type (87% and 62% respectively). The prevalence of ventilatory abnormalities were higher in the smokers (mixed ventilatory disorder 38,57% vs 21,21%) and increased significantly along with seniority in the post (p=0.0001 for the mixed ventilatory disorder) (Figure3). The effect of exposure to flour dust appears to be clear because 12,13% of the non-smokers had respiratory functional abnormalities with an isolated restrictive functional disorder. However, 10% of work-related asthma cases were found in our study population.

### Table 1: General characteristics of the study population.

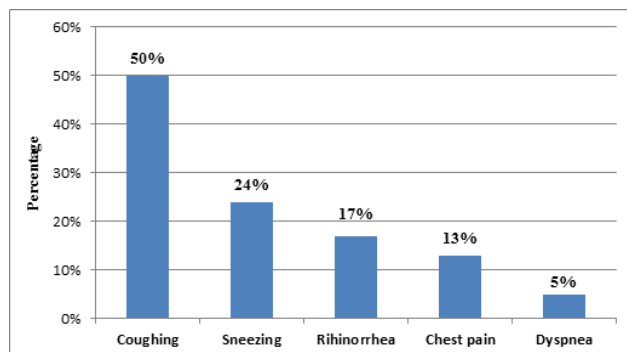| Anthropometric data | Average | Min | Max |
|---|---|---|---|
| Age (years) | 26.15 ± 10.18 | 15 | 52 |
| Weight (kg) | 68.30 ± 10.97 | 51 | 95 |
| Height (cm) | 179.42 ± 8.08 | 150 | 195 |
| BMI (kg/m²) | 21.22 ± 3.57 | 16 | 30 |
| Waist size (cm) | 74.95 ± 9.08 | 61 | 95 |
| Seniority (years) | 7 ± 6.7 | 1 | 25 |

BMI : Body mass index



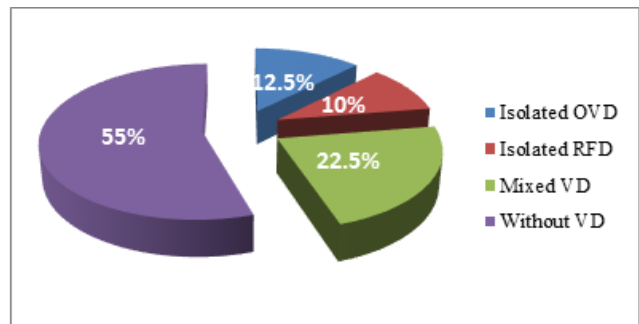Figure 1: prevalence of respiratory symptoms.



Figure 2: prevalence of ventilatory abnormalities.
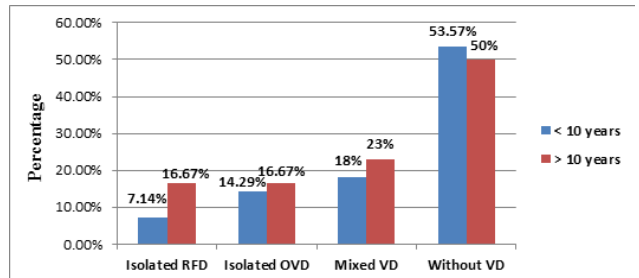


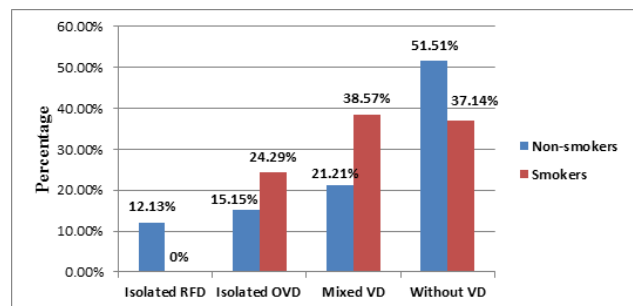Figure 3: distribution of VD according to seniority (years).



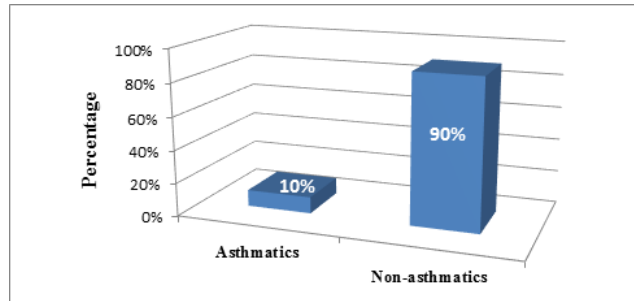Figure 4: distribution of VD by smoking status.



Figure 5: Prevalence of asthma in our study population.

### Discussion

This study made it possible to identify the main work-related risks of the bakery and pastry profession that are part of the informal economy in our country. Our recruited subjects did not receive medical follow-up and did not use any personal protective equipment against flour dust during the exercise of their profession. However, bakers' lack of protective measures could be linked, on the one hand, to a failure by employers and, on the other hand, to ignorance of the health risks incurred due to the low levels of education combined with a lack

of adequate training. This is consistent with observations made by Hentschel et al.[8] in other job categories where workers are exposed to dust.

### *General characteristics of the population*

Our study population was relatively young (average age 26±10 years). The age distribution does not differ from the traditional demographic characteristics of African countries. The proportion of workers with less than 5 years' professional experience predominated, i.e. 45% of the subjects. This observation was also made by Aba[9] who found 59.9% of workers with less than 5 years' seniority respectively among carpenters. Seniority is a determining work-related risk factor in the occurrence of certain pathologies related to exposure to organic dust. In fact, young workers lack experience and do not sufficiently control the risks associated with the different activities they carry out. In Senegal, there are currently no specific recommendations concerning the exposure limit value for flour dust in relation to the exposure threshold of 10 mg/m$^3$ for harmful dust [10].

### *Clinical respiratory manifestations*

Our study revealed the presence of a large number of complaints among bakers and pastry makers with a clear predominance of cough (50%), sneezing (24%) and rhinorrhea (17%). Backlate et al[11] similarly observed that coughing, mucus production, wheezing and dyspnea were acute symptoms of exposure to flour dust. Sneezing and rhinorrhea are the most common symptoms in the studies of Ige et al.[12] and Backlake et al.[11]. Flour has been implicated in triggering symptoms in 65% of our bakers. Our results corroborate those of Laraqui et al[13] who observed that workers exposed to cereal dust handling large quantities of grain daily and without any means of personal protection were more symptomatic (64.3%) than unexposed subjects. Tabka et al [14], in a cross-sectional study on respiratory pathology related to cereal dust in the Sousse region (Tunisia) made the same observations, suggesting that these clinical manifestations depend on the level of dusting.

Several surveys among bakers in Europe have reported lower prevalences than those found in our study: 14 to 17% for Vanhanen et al.[15], 19% for Prichard et al.[16]. These studies concerned more modern bakeries where hygiene conditions and methods of individual and collective technical prevention were better. The relatively small number of exposed subjects in our study (40 bakers) may contribute to underestimate the importance of the problem.

In our study, the prevalence of 25% of atopic subjects would be due to the lack of orientation of young atopic people towards respiratory risk-free jobs, and to the absence of work-related medical services. Its prevalence in the literature is so differently assessed: 16% for Vanhanen et al[15], 38% for Houba et al[17]. This discrepancy may be related to the definition of atopy in the methodologies of surveys based on the questionnaire only.

### *Spirometric parameters*

In our study, the abnormalities of ventilatory function are very important, accounting for 45% of our subjects. The prevalence of ventilatory abnormalities in other Moroccan studies was 31,6% among mill workers[18]. These changes in respiratory function would probably be caused by airway obstruction due to bronchoconstriction in response to flour dust. The healthy worker effect would explain that ventilatory disorders were more of an obstructive type characterized by small airway damage with a moderate degree of obstruction. Indeed, workers with severe disorders were not present at the time of the survey because they had left the profession on their own or were on sick leave.

The combination of tobacco and exposure to organic dusts impairs respiratory function with effects that potentiate, add up or multiply. In our study, we found that work-related exposure associated with smoking leads to an increase in respiratory functional disorders (Figure 4). Cotton et al[19] in a study conducted in cereal workers showed lower spirometric values in exposed and smokers. This finding suggests an interaction between tobacco and work-related exposure in the development of ventilatory abnormalities. In another study, comparing a group of cereal workers to a control group, the same authors found an increase in the prevalence of symptoms and a decrease in respiratory function values in the exposed and smoking group, but in this study, these effects appeared to be more than synergistic[20]. Moreover, our study found a relationship between the observed ventilatory disorders and seniority in this activity. Variations in ventilatory parameters in the bronchodilation test showed 10% of asthmatic subjects. Many cross-sectional studies have reported asthma prevalence in bakers ranging from 5 to 21%[21]. These differences in results can be explained by the type of bakeries (artisanal or industrial), the level of

dust (presence or absence of effective prevention or ventilation measures) and the criteria for defining asthma that vary from one study to another.

## Conclusion

This study showed that bakers and pastry makers exhibit various respiratory symptoms and ventilatory disorders related to the constant inhalation of flour dust during the course of their work. Poor working conditions and the premises housing these bakeries and pastry factories thus favour the occurrence of these work-related risks. Smoking and seniority in the profession increase these functional anomalies related to dust. The strengthening and implementation of individual and collective preventive measures remain a priority for better health for these workers.

## References

1. Ramazzini B. De morbis artificum diatriba. 1700. D'après la traduction de A. de Fourcroy : Alexitère éd. 1990.

2. Emls J, Beckett P, Griffin P, Evans P, Sams C, Roff M et al. Job categories and their effects no exposure to fungal alpha-amylase and inhalable dust in UK Baking industry. AIHAJ 2003;64 (4): 467-71.

3. Burstyn I. Teschke K, Kennedy SM. Exposure levels and determination of inhalable dust in bakeries. *Ann Occup Hyg* 1997; 41: 609-24.

4. Lucas D, Loddé B, Bronstein JA, Dewitte JD, Jegaden D. Asthme au soja, la problématique professionnelle chez les dockers à partir de deux cas. Medicina Maritima 2008; 8:20.

5. Occupational health and safety. International Labour Office, Geneva 1990 WHLD Pub, Vol11/LZ: 98-100.

6. Soriano JB, Miravitlles M, Garcia-Rio F, Munoz L, Sanchez G, Sobradillo V et al. Spirometrically-defined restrictive ventilator defect: population variability and individual determinant .Prim Car Respir J. 2012; 21(2):187-193.

7. Weitzenblum E. Indication des explorations fonctionnelles respiratoires. Paris: Éditions margaux orange; 2004:169–78

8. Hentschel T, Hruschka F, Priester M. Global report on artisanal and small scale mining. Mining minerals and sustainable development. World Business Council for sustainable development/ IIED; 2002.

9. Aba LN. Amélioration des conditions de travail et de la productivité dans les garages automobiles à Abidjan : Proposition d'application de la méthodologie du WISE. Thèse Med, Abidjan ; 2007. n°4562: 96..

10. Valeurs limites d'exposition professionnelle aux substances dangereuses en France. INRS, Cahiers de Notes Documentaires, 1988; 133: 691-706.

11. Blecklake M, Broder I, Chan-Yeung M, Dosman JA, Ernst P, Herbert FA et al. Recommendations for reducing the effects of grain dust on the lungs. Canadian Thoracic Society Standard Committee. CMAJ 1996; 155 (10)1399-1403.

12. Ige OM, Awoyemi OB. Respiratory symptoms and ventilatory function of the Bakery workers in Ibadan, Nigeria. WAJM 2002; 21; (4):316-18.

13. Laraqui CH, Caubet A, Laraqui O, Benghalem A, Harourate K, Bichara M et al. Prevalence des symptômes respiratoires et évaluation du degré de sensibilisation chez les ouvriers du souk de céréales de Casablanca. *Rev Mal Respir* 2000; 17: 947-55.

14. Tabka F, Mrizek N, Tabka Z, Choudat D, Conso F. Pathologie respiratoire liée à la poussière de céréales. Etude dans la région de Sousse (Tunisie) ; *Arch mal prof*, 1999;60(2):101-6.

15. Vanhanen M, Tuomi T, Hokkanen H, Tupasela O, Tuomainen A, Holmberg PC et al. Enzyme exposure and enzyme sensitization in the baking industry. Occup Environ Med. 1996; 53: 670–676.

16. Prichard MG, Ryan G, Musk AW. Wheat flour sensitisation and airways disease in urban bakers. Br J Ind Med 1984; 41(4): 450-4.

17. Houba R, Heederik JJ, Doekes G, Van Run PEM. Exposure sensitization relationship for α-amylase allergens in the baking industry. Am J Respir Crit Care Med 1996;154(1): 130-6.

18. Laraqui CH, Alaoui Yazidi A, Rahhali A, Verger C, Caubet A, Belemallem I, et al. Prévalence des symptômes cliniques respiratoires et la sensibilisation dans un échantillon de minotiers au Maroc. Int J Tuberc Lung Dis 2003;7(4):382-9.

19. Cotton D, Graham B. Effects of smoking and occupational exposure on peripheral airways function in young cereal grain workers. Am. Rev. Resp. Dis. I 982; 26. 660-665.

20. Cotton D, Graham B. Effects of grain dust exposure and smoking on respiratory symptoms and lung function. J. Occup. Med.1983; 25:131-141.

21. Aka I. Amélioration des conditions et de la productivité dans les menuiseries et ébénisteries à Abidjan : Proposition d'application de la méthodologie du WISE. Thèse Med, Abidjan; 2008. n° 4716:106.

# Effects of Habitual Physical Activity Level (PAL) on Simple Visual and Auditory Reaction Time in Healthy Indian Adults

**Suparna Ghosh[1], Roopashree K[2], Nandini C[1], An Jubin John[2]**

[1]*Associate Professor,* [2]*Assistant Professor, East Point College of Medical sciences and Research Centre, Bangalore, Karnataka*

## Abstract

**Aim**  To find the correlation between physical activity level(PAL) and simple visual and auditory reaction time(SVRT and SART), which are tests for  cognition .

**Material and Method:** One hundred(100) healthy volunteers of both the genders(males 52 and females 48), aged between 18 and 50 years were recruited. Reaction time (RT), is defined the elapsed time between the presentation of a stimulus and the subsequent behavioral response to occur. Subjects were presented with red light for VRT and pure tone sound stimuli for ART.  Reaction time is a measure of the coordination between the sensory and motor system occurs. The physical activity level (PAL) was determined by administering a physical activity level questionnaire developed by the Division of Nutrition, St. John's Medical College, Bangalore. The PAL of an individual classified as follows: sedentary<1.4 moderately active 1.55 to 1.75, and  heavily active .1.75.

**Result:**    Statistical analysis of  data was done by one-way ANOVA with  Post-hoc by Turkey HSD test. The results were found to be significant.  There is a negative correlation between the SVRT and PAL with $R^2$ value of 0.006;  SART and PAL with $R^2$ value of 0.001  .

**Conclusion:**   In this study we observed that when there is increase in the PAL, there is significantly faster ART and VRT.  Therefore,  more physically active individuals have better reaction times and are more coordinated at motor tasks when compared to less physically active.

***Key words***: *BMI( Body mass index), Auditory reaction time(ART), Visual reaction time(VRT), PAL(Physical activity level)*

## Introduction

Chronic diseases are already the primary disease cluster in India.  Physical inactivity is one of the important risk factor in common for several chronic diseases including coronary artery disease, hypertension, diabetes, certain types of cancers (breast and colon), respiratory disorders, obesity and osteoporosis being responsible  up to 60% of all deaths[1].The problem is of particular concern in countries like India that has transitional economies and changing lifestyles. These changing lifestyles may compound increased risks for ethnic groups.  There are some data, for instance they suggest that Indians have a genetically determined risk for coronary artery disease.  Physical inactivity would enhance that risks including cognitive disturbances[2].

Physical activity comprises all types of muscular activity that increase energy expenditure substantially. Exercise is a regular and structured subset of physical activity, performed deliberately as preparation for athletic competition or the improvement of some aspect of health[3].

Reaction time(RT) is defined as the time elapsed between administration of a stimulus and the appearance of appropriate voluntary  response in a person.  Luce and Welford descried three types of RT  1.  Simple RT: here

**Corresponding author:**
**Dr. Roopashree K.**
Flat no. 207, T. M Enclave, 38th cross, 19th Main, 5th block, HBR layout, Bangalore - 560043   E-mail: roopamysore5@yahoo.co.in  Ph no:  7349564985

there is one stimulus and one response. Recognition RT: here there is some stimulus that should be responded to and other that should not get a response. 3. Choice RT: here there are multiple stimuli and multiple responses[4]. Simple reaction time is usually defined as the time required for an observer to detect the presence of a stimulus. It is a physical skill closely related to human performance. It represents the level of neuromuscular coordination in which the body through different physical, chemical and mechanical processes decodes visual or auditory stimuli which travel via afferent pathways and reach the brain as sensory stimuli. Simple reaction time can be determined when an individual is asked to press a button as soon as a light appears or sound heard. Research done by Pain & Hibbs shows that simple auditory reaction time has the fastest reaction time for any given stimulus[5].

Exercise can improve reaction time. In 1971Levitt and Gutin showed that subjects had the fastest reaction times when they were exercising sufficiently to produce a heart rate of 115 beats per minute and in 1980 Welford found that physically fit subjects has faster reaction times[6,7]. In 2005, Kashihara and Nakahara found that vigorous exercise did improve the choice reaction time , but only for the first 8 minutes after exercise.

Some benefits occur immediately after a session of moderate-to-vigorous physical activity, commonly referred to as the "last bout effect." Reduced feelings of anxiety, improved sleep, and improved cognitive function are examples of benefits that can occur after a single episode of moderate-to vigorous physical activity. If participation in physical activity becomes regular, reductions in routine (baseline) feelings of anxiety occur, the last bout effect on deep sleep becomes more pronounced, and components of executive function continue to improve. Executive function includes the processes of the brain that help organize daily activities and plan for the future. Tasks such as the ability to plan and organize; monitor, inhibit, or facilitate behaviors; initiate tasks; and control emotions all are part of executive function[8,9,10]

Many studies have been conducted to assess reaction time in soccer players, badminton players, basketball players, yoga practitioners but no studies have yet been done in the general population with measured regular general cores and exercise is not there. We undertook this study to assess the correlation between measured habitual physical activity and simple reaction times[11,12,13]

## Material and Method

One hundred(100) volunteers, aged between 18-50 years ,male(n=52) and female(n=48) were recruited for the present study[2]. The subjects had no history of diabetes, hypertension, visual and auditory disturbances, alcohol intake, smoking, and no history of recent illness from any diseases. Ethical clearance for the study was obtained from the Institutional Ethical Committee. Participation in the study was voluntary and a written informed consent was obtained from every participant[13,14].

PC 1000 Hertz's Reaction Timer*:* We used an in house build add on device called PC 1000, to measure visual reaction time. PC 1000 is a1000 hertz square wave oscillator which has a soft key for start and stop function. PC 1000 Reaction timer instrument has two components (A &B) connected to each other.

First component (A) has a start button which is handled by the examiner only.

Second component (B) has a stop button which will be handled by the subject alone and also it has a small red LED which receives the visual stimulus. Red light is selected for the experiment as it persists for a long time in retina. Component A and component B are in turn connected to a personal computer which has audacity software installed in it. Audacity software records the reaction time with 0.001sec accuracy in wave format[10]. Minimum five trials were given for SVRT and SART measurements. Minimum time recorded was considered as the final SVRT and SART[15,16].
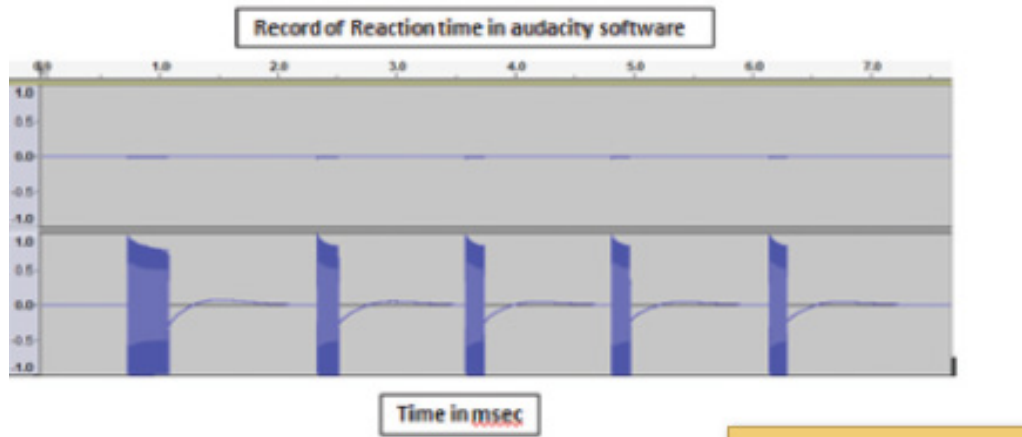
**Physical activity level (PAL):** This is used as composite index of Physical activity patterns and is calculated as:24 hr energy expenditure/basal metabolic rate. 24 hr energy expenditure is calculated as the sum of energy expenditures of all reported activities computed for a single day. Basal metabolic rate is calculated from age and gender specific regression equations recommended by WHO, that include height and weight as predictor variables.

Cutoffs for PAL's that describe grades of physical activity are <1.4 sedentary, 1.55-1.6=moderately active and >1.75= heavily active. Thus, lower PAL's indicate more sedentary physical activity profiles[2].

**Calculation of 24 hour energy expenditure:** The activity reported for one month are recomputed for 24 hours as the sum of energy expenditure related to sleep, occupational energy expenditure, discretionary leisure time energy expenditure and "residual energy expenditure". In order to calculate energy expenditure for each of these components BMR/min is first computed. For every reported activity MET (metabolic equivalent) which is essentially a multiple of BMR is applied. Higher MET 's indicate higher levels of physical activity.

"Residual energy expenditure" relates to those periods in a day which are unaccounted for by recall, and for which intensities of activities have to be assumed. Individuals tend to underreport sedentary, therefore we employ a uniform MET of 1.4 for all "residual time"[11,12,17,18].

## Results

Statistical Analysis: Data was entered into Microsoft Excel (Windows 7; Version 2007) and analyses were done using the Statistical Package for Social Sciences (SPSS) for Windows software (version 16.0; SPSS Inc, Chicago). Descriptive statistics such as mean and standard deviation (SD)for continuous variables, frequencies and percentages were calculated for categorical Variables were determined. Association between Variables was analyzed by using Chi-Square test for categorical Variables. Comparison of mean of quantitative variables were analyzed using unpaired t test and ANOVA (Analysis of Variance) respectively for categories having 2 and more than 2 categories. Levels of significance was set at $P<0.05$. In table 1 the subject characteristic of male and female is depicted

**Table 1:Descriptive Statistics of Male and Female**

| Subject Characteristics | Male | Female |
|---|---|---|
| AGE(yrs) | 24±7.05 | 25.27±8.75 |
| BMI (kg/m2) | 23.24±3.67 | 23.72±4.32 |
| PAL | 1.66±0.34 | 1.64±0.42 |
| VRT( msec) | 0.198±0.044 | 0.233±0.059 |
| ART (msec) | 0.176±0.046 | 0.193±0.065 |
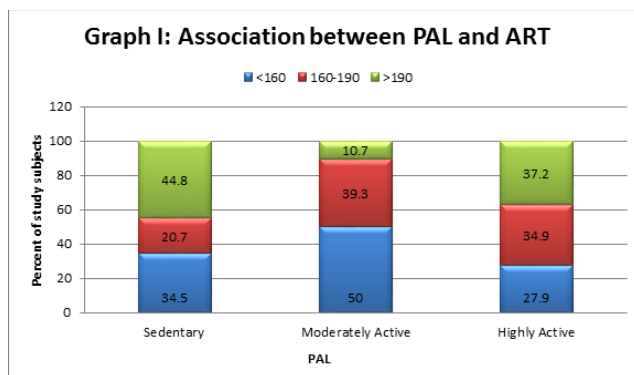
Graph 1 tells the association between PAL and ART
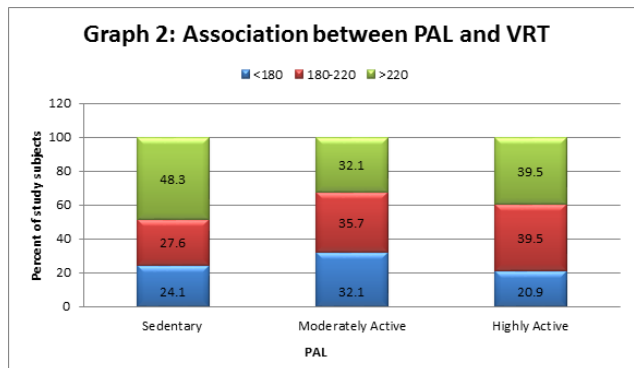


Graph 2 depicts the association between PAL and VRT

## Discussion

This cross-sectional study was conducted on hundred healthy students and staff of age group of 18 to 50 years of both the genders. The questionnaire assessed physical activity of the past month across multiple domains including discretionary leisure time, household chores, work, sleep, sedentary activities and other common daily activities. The frequency and average duration for each activity were documented. Frequencies were ascertained using fixed categories of 'daily, once a week', '2–4 times a week', '5–6 times a week', 'once a month' and '2–3 times a month'. When all reported activities did not cumulatively account for 24 h, a standard MET (metabolic equivalent) of 1.4 was applied to the 'residual time', as in previous studies(12). For manual occupational activity, the integrated energy index (IEI) of the activity was applied instead of the MET value. Unlike MET, IEI accounts for 'rest' periods that participants are likely to take when engaged in manual activities[14]. PAL cut-offs have been described to classify physical activity patterns into sedentary/light, moderately and vigorously active lifestyles[17].

Since the introduction of the 1993 Compendium, many studies have used the coding scheme and standard MET values to assign intensity levels to PA questionnaires globally. The results from these studies have supported conclusion that regular physical activity is health enhancing and that physical inactivity is a major risk factor for chronic diseases and premature mortality. The 2011 Compendium provide 821 codes that reflect 21 major headings, numerous specific act ivies and their detailed descriptions, and associated MET values that can be used to identify the energy cost of PA[17,18]. Compendium provides a timely update with adoption of evidenced-based MET values

RMR represents as 3.5ml/kg/min. **Resting metabolic rate** (also called RMR) is the rate at which our body burns energy when it is at complete rest. We can **calculate your resting metabolic rate** to see how many calories our body needs to perform basic functions like breathing and circulation. Our RMR or resting metabolic rate is part of our **total daily energy expenditure (TDEE)** or the total number of calories you burn each day.

Moderate evidence indicates that moderate-to-vigorous physical activity can have beneficial effects on cognition though the intensity of activity required to preserve cognitive function remains unclear. Relative to studies of children and older adults, there is dearth of several reviews (SR) and meta-analyses on the relationship of PA and cognition in young and middle-aged adults(18-50 yr). The review of the 2018 physical activity guidelines concluded the grade as not assignable[25.]

The cardio-metabolic profile also shows improvements soon after an episode of moderate-to-vigorous physical activity. Blood pressure is reduced, and insulin sensitivity is increased. These cardio-metabolic benefits persist for hours to days after the last bout. They also may be sufficient to lower the blood pressure of people with pre-hypertension and hypertension into normal ranges for a major portion of the day. Other benefits, such as reduced risk of cardiovascular disease (CVD), diabetes, falls, and fall-related injuries among older adults, and improved physical function accrue as the physiologic adaptations to greater physical activity transpire. Improved cardio-respiratory and muscular fitness and biomarkers of disease risk start to accrue within days, and for a given amount of physical activity, maximize after a few months. Additional benefits accrue if physical activity volume is further increased. The reductions in risk apply every day and at all ages, including young adults, even though their risk for chronic disease is lower than for middle-aged and older

adults[24,3].

Prospective study of older women, higher levels of long-term regular physical activity were strongly associated with higher levels of cognitive function and less cognitive decline. Specifically, for getting the apparent cognitive benefits of greater physical activity were needed. Walking the equivalent of at least 1.5 hours per week at a 21-30 min/mile pace was also associated with better cognitive performance[18,19,20,,21,22,24].

This cross-sectional study shows that the ART is faster than VRT which supports the other studies conducted in India and elsewhere[13,14,19,22]. The mean and standard deviation of ART in our study is 176±0.046 ms and VRT is 198±0.044 ms in males and 193±0.065 msec, and 233±0.059 msec in females(Table 1). Researches by Kemp et al., show that an auditory stimulus takes only 8-10 milliseconds to reach the brain, but on the other hand, a visual stimulus takes 20-40 milliseconds[6]. This implies that the faster the stimulus reaches the motor cortex, faster will be the reaction time to the stimulus. Therefore since the auditory stimulus reaches the cortex faster than the visual stimulus, the auditory reaction time is faster than the visual reaction time. There is an increase of reaction time both ART and VRT in females than males irrespective their age which also supports other data[19,21].

Our study shows a significant decrease in reaction time in both the auditory and visual forms with the increase of measured habitual activity level of (>1.4 to <1.75). The ART is decreased from 190.35 msec to 162.07milisec and VRT from 220.58 msec to 199.78 msec in males. The difference is not so evident in sedentary(<1.4) and moderately active(PAL 1.4-1.75) people but quite evident in moderately and heavily active people(PAL>1.75). From this study, we understood that to acquire better cognitive benefit one person has to do more vigorous physical activity than habitual (PAL>2).

## Conclusion

The more active people definitely has the faster reaction time than the lesser active and cognitive function is better. The dose of the activity can be calculated and introduced to other form of activities like sports and exercises in future studies as they are all measurable.

## References

1. Mondal S, Van Belle S India's NCD strategy in the SDG era: are there early signs of a paradigm shift? Global Health. 2018 Apr 25;14(1):39. doi: 10.1186/s12992-018-0357-6.

2. Bharti AV, Sandhya N, V az M The development and characteristics of physical activity level questionnaire for epidemiological studies in urban middle class Indians. Indian J Med Res 2000; 111: 95-102

3. Bouchard C, Shephard RJ, Stephens T. Physical activity, fitness and health. Champaign, IL: Human Kinetics, 1994:77-88.

4. Shephard RJ. How much physical activity is needed for good health. Int J Sports Med 1999;20:23–7.

5. Teichner, Warren H. Recent studies of simple reaction time. Psychological Bulletin 51.2 (1954):128.

6. T.G.Matthew Pain and A. Hibbs, "Sprint Starts and the Minimum Auditory Reaction Time," Journal of Sports Sciences, Vol. 25, No. 1, 2007, pp. 79-86.

7. Duke-Elder S. Franciscus Cornelis Donders. Br J Ophthalmol. 1959;43:65–8

8. Welford AT. Choice reaction time: Basic concepts. In: Welford AT, editor. Reaction Times. New York: Academic Press; 1980. pp. 73–128.

9. Luce RD. London: Academic Press; 1968. [Last accessed on 2011 Aug 08]. Information Theory of Choice. Reaction Times. Available from http://www.biology.clemson.edu/bpc/bp/Lab/110 reaction.htm.

10. Levitt S and Gutin B Multiple choice reaction time and movement time during physical exertion. Research quarterly 1971;42: 405-410

11. Kashihara K and Nakahara Y. Short term effect of physical exercise at lactate threshold on choice reaction time. Perceptual and Motor Skills 2005 100;2: 275-291.

12. Ainsworth BE, Haskell WL, Herrmann SD, Meckes N, Greer JL, Vezina J, Basett DR. Jr, Tudor - Locke

C, Whitt- Glover MC, Jacobs DR. Jr., Leon AS. 2011 Compendium of Physical Activities: a second update of codes and MET values Med Sci Sports Exerc. 2011 Aug;43(8):1575-8, doi:10 1249/MSS. 0b03e 31821 ecs

13. Thomas JI, Venkatesh D. A comparative study of the effects of superbrain yoga and aerobic exercise on cognitive functions. National Journal of Physiology, Pharmacy and Pharmacology. 2017;7(9):895.

14. 14.Jain A, Bansal R, Kumar A, Singh KD A comparative study of visual and auditory reaction times on the basis of gender and physical activity levels of medical first year students. Appl BasicMed Res 2015 May-Aug ; 5(2): 124-127

15. Karia RM, Ghuntla TP, Mehta HB, Gokhle PA, Shah CJ. Effect of gender difference on visual reaction time: A study on medical students of Bhavnagar region. IOSR-PHR. 2012;2:452-4

16. R. Niruba and Maruthy KN. Assessment of auditory and visual reaction time in Type 2 Diabetes- A case control study. Al Ameen J MedSci (2011)4(3):274-279

17. Physical Activity Guideline Advisory Committee. Physical Activity Guideline Advisory Committee Report. Washington(DC): US Department of Health and Human Services; 2008. p 23.

18. Skandan KP, Mehta SK, Mehta YB, Gaur HK.Visuo motor co-ordination time in normal children. Ind Ped 1980; 17: 275-278.

19. Gavkare AM, Nanaware NL, Surdi AD. Auditory reaction time, visual reaction time and whole body reaction time in athletes. Indian Medical Gazette2013; June: 214-219.

20. Jain AK. Manual of Practical Physiology. New Delhi: Avichal Publishing Company, 2000;p291

21. Shenvi D and Balasubramanian P. A comparative study of visual and auditory reaction time in males and females. Indian J Physiol Pharmacol, 1994; 38: 229-231.

22. Nikam LH and Gadkari JV. Effect of age, gender and body mass index on visual and auditory reaction times in healthy Indian population. Indian J Physiol Pharmacol 2012; 56(1): 94- 99.

23. Gunstad J, Lhotsky A, Wendell CR, Zonderman AB. Longitudinal Examination of Obesity and Cognitive Function: Results from the Baltimore Longitudinal Study of Aging Neuroepidemiology.2010 May; 34(4): 222–229.

24. Weuve J, Kang JH, Manson JE, Breteler MMB, Ware JH, Grodstein F. Physical Activity, Including Walking, and Cognitive Function in Older Women*JAMA*. 2004;292(12):1454-1461. doi:10.1001/jama.292.12.1454

25. Erickson KI, Hillman C, Stillman CM, Ballard RM, Bloodgood B, Conroy DE et al. Physical Activity, Cognition, and Brain Outcomes: A Review of the 2018 Physical Activity Guidelines. Med Sci Sports Exerc. 2018 Jun ;51(6): 1242-1251.

# Physical Fitness among Doctors Working in a Tertiary Care Teaching Hospital

**Shivappa GC[1], Revathi Devi ML[2], Manjunatha SN[3]**

*[1]Assistant Professor, Department of Physiology [2]Professor & Head, Department of Physiology,*
*[3]Associate professor, Department of Community Medicine, MMCRI, Mysuru*

## Abstract

**Background:** Physical fitness is the capacity to perform a sedentary task efficiently and also a sense of physical wellbeing and the capacity to deal with emergencies demanding unaccustomed physical effort. The fit man carries on a given grade of moderate work with less displacement of his physiological equilibrium.

**Objectives**

1.  To measure the Physical Fitness Index of doctors at tertiary care hospital using modified Harvard Step Test.

2.  To correlate physical fitness scoring with Body Mass Index, gender and physical activity.

**Method**: A cross sectional study was conducted on 156 doctors working in tertiary care teaching hospital attached to Mysore Medical College and Research Institute during June 2018 to May 2019. Modified Harvard step test was used to measure physical fitness. SPSS 17 version was used for statistical analysis.

**Results:** Males constituted 42.3% and females constituted 57.7% of the study population with a mean age of 33.5 years. BMI of females was little higher (24.01±3.77) when compared to males (24.03±3.01). Forty percent of females had excellent physical fitness whereas only 6.06% of males had excellent physical fitness. Only 14.40% of the female doctors had poor physical fitness whereas 36.36% of males had poor physical fitness. It was found that BMI and pulse rate had a statistically significant negative correlation with physical fitness where as physical activity, Systolic and diastolic blood pressure had positive correlation.

**Conclusion:** Physical activity is an important determinant and predictor of physical fitness. Females are having better physical fitness as most of them are pursuing some physical activity.

***Key Words:*** *Modified Harvard Step Test; Physical Fitness Index; Pulse Rate Variability*

## Introduction

Physical fitness is the capacity to perform a sedentary task efficiently and also a sense of physical wellbeing. It is also a capacity to deal with emergencies demanding unaccustomed physical effort.

The fit man carries on a given grade of moderate work with less displacement of his physiological equilibrium.[10] He can establish steady states of higher grades of work. Regular muscular fitness training program improves endurance and flexibility of trunk and lower limb musculature[14].

In today's life there is more stress and less physical activity. Fitness becomes more important both physiologically and psychologically. Fitness is defined as state of mental and physical harmony which enables one to perform his daily activity to the best.

**Corresponding author-**
**Dr Shivappa GC,**
Assistant professor, department of physiology,
MMCRI, Mysuru. Email – shivappagc@gmail.com

Doctors require absolute physical and mental fitness during there working. The present study is to assess physical fitness among doctors working in a tertiary care teaching hospital.

## Objectives

1. To measure the Physical Fitness Index of doctors at tertiary care hospital using modified Harvard Step Test.

2. To correlate physical fitness scoring with Body Mass Index, gender and physical exercise.

## Materials and Method

The study sample consisted of 156 doctors of MMC&RI, 66 male doctors and 90 female doctors. The study was conducted for the period of one year (June 2018 to May 2019) in the Department of Physiology, Mysore Medical College and Research Institute, Mysore.

### Inclusion Criteria

a. Healthy male and female doctors aged between 25 and 60 years.

### Exclusion Criteria

a. Doctors with musculoskeletal disability.

b. History of cardiovascular disorders.

### Method of Collection of Data

All the exercise data were collected during morning hours between 7 am to 9 am to avoid any possible diurnal variation effect. Subjects were told to report an hour before exercise. The details of the procedure of exercise test were explained to the subjects and actually demonstrated before in order to allay apprehension. Written informed consent was obtained from them. They were asked to refrain from eating or drinking at least for an hour and allowed to take rest for half an hour before exercise. Before the exercise test, the physical anthropometric parameters like height, weight and BMI and physiological parameters like pulse rate, respiratory rate and blood pressure of the subjects were recorded.

### Recording of Physical Fitness Index by Using modified Harvard Step Test

Physical fitness index of each subject was recorded by using modified Harvard step test with step height 40 cms (instead of 50 cms of original HST) for males and 33 cms for females, which is a wooden step, heavily constructed such that it remains steady always during the test (validated)[11]. The observer calls the rhythm, at the signal "start" stopwatch is started, metronome is turned on. The subject places one foot on the platform and later the other, and immediately steps down, bringing down first the same foot which he placed up first. At exactly 3 seconds intervals the signal 'up' is given and rhythm is maintained by giving the following count up, -2-, -3, -4 up, -2, -3, -4. The subject should "lead-off" with the same foot each time and not try to alternate the foot. The subject is exercised at the rate of 20 times (instead of 30 times of original HST) a minute for 5 minutes continuously unless he stops from exhaustion.

All subjects were stopped at 5 minutes if they could go that long. Exhaustion is defined as when the subjects could not maintain the stepping rate for 15 per minute. The subjects who did not complete the duration of test (5 minutes) are not included in the result because it is difficult to assess the fitness.

When the subject successfully completes the test recovery time starts counting. He is made to lie quietly on the cot. Three readings (radial pulse) were taken during this recovery phase. The first reading was from 1 minute to 1 minute 30 seconds after the exercise, the second reading was from 2 minutes to 2 minutes 30 seconds after the exercise and the third reading from 3 minutes to 3 minutes 30 seconds after the exercise.

The Physical Fitness Index is calculated by using following formula.

$$PFI = \frac{\text{Duration of exercise in seconds} \times 100}{2 \times \text{Sum of the three half minute post exercise pulse counts}}$$

Blood pressure (SBP and DBP) at 3 minute 30 seconds and 4 minutes 30 seconds after the exercise were also recorded in supine position.

According to modified Harvard Step test the following scores are used for classifying the physical fitness of study subjects.[13]

### PFI rating and PFI score

| PFI Rating | PFI Score | |
| --- | --- | --- |
| | Male | Female |
| Excellent | >115 | >91 |
| Good | 103-115 | 84-91 |
| Fair | 91-102 | 77-83 |
| Poor | <91 | <77 |

**Statistics:** Statistical package for Social Science 17 version was used for analyses of the data. Descriptive statistics like mean and SD were used. Chi-square test, student t-test, correlation coefficients were used for the analysis of the data.

# Results

**Table 1: Profile of study subjects**

| Gender | Number (%) | Age (years) (Mean ± SD) | BMI (kg/m2) (Mean ± SD) |
|---|---|---|---|
| Male | 66 (42.3) | 34.88 ± 9.78 | 24.03± 3.01 |
| Female | 90 (57.7) | 32.93 ± 8.22 | 24.01 ± 3.77 |

It was interesting to find a great disparity in the physical fitness levels of males and females. This gender difference was statistically significant.

**Table 2: Physical Fitness Index among study subjects**

| PFI rating | Male (%) | Female (%) | Total (%) |
|---|---|---|---|
| Excellent | 4(6.06) | 36 (40) | 40(25.6) |
| Good | 11 (16.67) | 18 (20) | 29(18.6) |
| Fair | 27 40.91) | 23 (25.60) | 50 (32.1) |
| Poor | 24 (36.36) | 13 (14.40) | 37 (23.7) |
| Total | 66 (42.3) | 90 (57.7) | 156 (100) |

Chi square: 110, df: 6, p<0.0001

The average BMI of females was slightly higher than males. Doctors having poor physical fitness had higher BMI when compared to doctors with better physical fitness. This was true for both males and females. Body mass index was inversely related to physical fitness in the present study

**Table 3: Physical fitness according to Body Mass Index**

| PFI rating | BMI (kg/m2) | |
|---|---|---|
| | Male (Mean ± SD) | Female (Mean ± SD) |
| Excellent | 23.5±3.7 | 24.03± 3.85 |
| Good | 23.36±1.75 | 24± 2.77 |
| Fair | 24.22± 2.81 | 22.83± 3.76 |
| Poor | 24.21±3.65 | 26.42± 4.77 |

Recovery pulse rate after the exercise is used to assess the physical fitness of an individual in most of the methods as it is influenced by physical exercise. . Modified Harvard step method also relies on this parameter for deducing the physical fitness of the individual. The basal pulse rate (before exercise) and pulse rate after the exercise was measured among the study subjects.

**Table 4: Pulse rate of study subjects (males & females) before and after the exercise according to physical fitness**

| PFI rating | Pulse Rate (beats per minute) in males | | t-value | Sig* |
| | Before Exercise (Mean ± SD) | After Exercise (Mean ± SD) | | |
| --- | --- | --- | --- | --- |
| Excellent | 75.5±10.63 | 79.5±3.11 | 2.345 | NS |
| Good | 75.5±7.31 | 94.27±4.61 | 3.201 | NS |
| Fair | 80±8.08 | 105.22±3.82 | 7.865 | S |
| Poor | 80.38±10.69 | 119±6.69 | 9.543 | S |

* S = Significant; NS = Not significant

| PFI rating | Pulse Rate (beats per minute) in females | | t-value | Sig* |
| | Before Exercise (Mean ± SD) | After Exercise (Mean ± SD) | | |
| --- | --- | --- | --- | --- |
| Excellent | 78.15±7.96 | 100.64±7.05 | 2.214 | NS |
| Good | 85.2±7.03 | 114.44±2.43 | 6.987 | S |
| Fair | 85.44±9.07 | 124.7±3.47 | 7.543 | S |
| Poor | 77.91±7.36 | 131.15±5.9 | 11.152 | S |

* S = Significant; NS = Not significant

The mean change in the pulse rate (before exercise and after exercise) among doctors with excellent physical fitness was only 13 units. The mean difference among subjects with poor physical fitness was 46 units. This clearly shows that heart rate variability is least among people with excellent physical fitness. This difference of pulse rate among various groups was found to be statistically significant.

The physical activity, BMI, pulse rate & BP are correlated with physical fitness. Physical activity of study subjects was measured in three grades. Grade '0' meant sedentary life style without any physical exercise other than routine work. Grade '1' meant physical exercise at least 30 minutes a day, 5 days a week e.g. walking, yoga, etc. Grade '2' meant participation in sports, athletics, gym, etc.

**Table 5: Correlation of physical fitness with BMI and physical activity and cardiovascular variables**

| | PFI score | BMI | Physical activity scores | Pulse rate | SBP | DBP |
| --- | --- | --- | --- | --- | --- | --- |
| PFI score | 1 | | | | | |
| BMI | 0.76 | 1 | | | | |
| Physical activity scores | 0.21 | 0.89* | 1 | | | |
| SBP | 0.73 | 0.43 | 0.91* | 0.94* | 1 | |
| DBP | 0.79 | 0.29 | 0.51 | 0.34 | 0.86* | 1 |

* Significant at 5%

## Discussion

Modified Harvard Step Test is proven to be a suitable method for assessing physical fitness of Indians. Many authors have demonstrated the utility of this method in Indian subjects.

This method was adopted by Sunil KR Das, Mahapatra S, Bhattacharya G, Mukherjee D[13], in the year 1993 at Laboratory of Human Performance Assessment, Department of Physiology, University College of Science and Technology, Culcutta.

Ganeriwal SK, Sen SL, Khandare SS[8] in the year 1968 performed the HST in Indian    51 female medical college students in the age group of 17 to 25 years using a stepping height of 18 inches. Banerjee PK and Chatterjee S[3] in the year 1983 studied the effectiveness of the Harvard step test in assessing the physical fitness in 54 Indian adolescent boys.

The present study showed that 40% female subjects had excellent physical fitness and 14.40 had poor physical fitness

Das SK, Nath N, Ray A[6], in the year 1990 studied the PFI using Astride Jump Test as an alternative of Harvard Step Test in 218 school girls having age 12 to 16 years in suburban area of Calcutta. In which 19% girls had excellent and 16% had poor physical fitness.    Shivappa GC, Revathidevi ML, Manjunatha SN[12] in the year 2012 studied the physical fitness in the 365 medical students having age 17 to 22 years in a government medical of Karnataka by modified Harvard Step Test. In which 46 % girls had excellent and 6% had poor physical fitness.

In our study 6.06% of the male subjects had excellent physical fitness and whereas 36.36% had poor physical fitness.

Das SK, Adhikari A, Goshthakur[5], in the year1988 studied the PFI using Harvard Step Test in 134 athletic boys having age ranging from 12 to 15 years belonged to various training camps in and around Calcutta. In which only 3% of male subjects had excellent and 23% had poor physical fitness.

Shivappa GC, Revathidevi ML, Manjunatha SN[12] in the year 2012 studied the physical fitness in the 365 medical students having age 17 to 22 years in a government medical of Karnataka by modified Harvard Step Test. In which 5.7% male subjects had excellent and 44% had poor physical fitness.

It was found that 50 percent of male & 45 percent of females studied had grade '1' & '2' physical activity. This study has clearly established that physical activity is important determinant and predictor of physical fitness.

There are no sufficient studies to compare PFI rating among males and females.

The average BMI of males was found to be24.03+3.01 kg/sq m and the average BMI of females was found to be 24.01+3.77 kg/sq m. It was found that BMI was inversely proportion to physical fitness both among males and females.

The present study is consistent with the study by Ganeriwal SK, Sen SC and Khandare(1968)[8], in which there was negative correlation between fitness index and body weight, pulse rate and positive correlation between fitness index and body height.

The present study is consistent with the study by Shivappa GC, Revathidevi ML, Manjunatha SN[12,] , in which there was negative correlation between fitness index and body weight, pulse rate and positive correlation between fitness index and body height.

Pulse rate variability (pre and post exercise) was minimum among subjects who had excellent physical fitness and it was maximum among subjects who had poor physical fitness index.

The present study is consistent with the study by Hammond(1987)[9] , which says that endurance athletes have lower resting and exercise heart rates which may be in part relate to down regulation of cardiac beta-adrenergic receptors secondary to repeated and prolonged episodes of sympathetic stimulation during exercise.

Systolic and diastolic blood pressure variabilities (pre and post exercise) were minimum in all the groups of physical fitness.

The present study is consistent with the study by Dalia A Biswas and Jayanth R Kher(1996)[4] , reported that heart rate, systolic blood pressure, double product and respiratory rate rose linearly with increasing grades of exercise while diastolic blood pressure recorded a fall.

Physical fitness was correlated with BMI, physical activity, gender and cardiovascular variables. Physical fitness had a statistically significant negative correlation with BMI.

The present study agrees with the study by Anabel NR, Anselmo JP, et al. (2007)[2] which reported that the faster heart rate was observed in the group with lower physical fitness (male and female). Additionally it is known that overweight and obese individuals exhibit lower levels of physical fitness.

In the present study subjects who had regular physical activity had better physical fitness and two variables were positively correlated.

The present study agrees with the study by Dipayan C, Soma C, et al. (2002)[7] which showed that non-residential school children who did not have regular physical activity showed a less physical fitness index scores as compared to residential school children were engaged in regular physical activity.

In the present study females had better physical fitness as majority of them are pursuing physically active lifestyle though the study had established a strong positive correlation between physical fitness and female gender. It may be due to the confounding by physical activity.

Pulse rate had a statistically significant negative correlation with the physical fitness and also physical activity in our study.

The present study agrees with the study by Albert W (1971)[1,] who reported that a lower arterial blood pressure and bradycardia are two prominent characteristics brought about by training.

## Conclusion

1. Physical fitness of doctors working in a tertiary care teaching hospital attached to Mysore Medical College and Research Institute, Mysore is not satisfactory. This may be due to the sedentary life style and lack of sporting activities and also over emphasis on hospital work.

2. Females are having better physical fitness as most of them are pursuing some physical activity.

3. Regular physical activity is an important determinant of physical fitness.

4. Overweight and obesity decreases the physical fitness of individuals.

5. Heart rate variability is less among physically fit individuals during physical exercise.

## References

1. Albert W. A physiological analysis of the effects of two training programmes. J Sports Med 1971;11:252 6.

2. Anabel N R, Anselmo JP, Luciana C, Nazare S B.The association between cardio- respiratory fitness and cardiovascular risk in adolescents. J Paediatria (Rioj) 2007;83(5):1-12

3. Banerjee PK and Chatterjee S. Harvard Step Test as a measure of physical fitness in adolescent boys. Indian J Med Res 1983;79:413-7.

4. Dalia AB and Jayant RK. Cardio-respiratory changes associated with graded exercise and determination of aerobic power in male medical students. Indian J Physiol and Pharmacol 1996;40(1):79-82.

5. Das SK, Adhikari A and Gosthakur D. Gradation of Harvard Step Test scores in athletic boys. Indian J Physiol and Allied Science 1988;42(1):8-10.

6. Das SK, Nath N, Ray A. Studies of physical fitness index (PFI) in school girls. Indian J Physiol and Allied Science 1990;44(1):4-6.

7. Dipayan C, Soma C, Vasantha AK. Physical fitness and non-residential schools. Indian J Physiol and Pharmacology 2002;46(3):328-32

8. Ganeriwal SK, Sen SC and Khandare SS. Test of physical fitness (Harvard Step Test) in Indian females. Indian J Med Res 1968;56:845-9.

9. Hammond. Association of decreased myocardial beta-receptors and chronotrophic response to isoproterenol and exercise in pig following chronic dynamic exercise. Circulation Research 1987;60:720-6.

10. Johnson RE. Applied physiology. Am Rev Physiol 1946;81:585.

11. Rhyming I. A modified Harvard Step Test for evaluation of physical fitness. Arbeistphysiologie, Bd 1954;15 S:235-250.

12. Shivappa GC, Revathidevi ML, Manjunatha SN. A study of physical fitness among the students in a government medical college of Karnataka by modified Harvard Step Test. Indian J physiol and

Allied Science 2014;68(4):111-19.

13. Sunil KR Das, Mahapatra S, Bhattacharya G and Mukherjee D. Determination of Physical Fitness Index (PFI) with modified Harvard Step Test (HST) in young men and women. Indian J Physiol and Allied Science 1993;47(2):73-6.

14. Swetha Satish DP, Sumaiya AK, Rauf I and Rmakrishna KS. Assessment of endurance, power and flexibility of navi Mumbai traffic police: Int J Med Pharm Sci, july 2014;4(11):20-25.

# Call for Papers/ Article Submission

## Article submission fee

- Please note that we charge manuscript handling charges for all publications. Charges can be enquired by sending mail.
- In cases of urgent publication required by author, he /she should write to editor for discretion.
- Fast tracking charges are applicable in urgent publication
- Please note that we charge only after article has been accepted for publication, not at the time of submission.
- Authors have right to withdraw article if they do not wish to pay the charges.

## Article Submission Guidelines

Please submit paper in following format as far as applicable

1. Title
2. Names of authors
3. Your Affiliation (designations with college address)
4. Corresponding author- name, designations, address, E-mail id
5. Abstract with key words
6.  Introduction or back ground
7. Material and Method
8. Findings
9. Discussion / Conclusion
10. Acknowledgement
11. Conflict of Interest
12. Source of Support
13. References in Vancouver style.
14. Word limit 2500-3000 words, MSWORD Format, single file
15. Please quote references in text by superscripting.

# International Journal of Physiology

## CALL FOR SUBSCRIPTIONS

ABOUT THE JOURNAL **International Journal of Physiology** is a double blind peer reviewed international journal which has commencedits publication from January 2013. The journal is quarterly in frequency. The journal covers all aspects ofphysiology. The journal has been assigned ISSN 2320-6039 (Print Version) and ISSN 2320-608X (Online Version).The journal is covered by Index Copernicus, Poland and many other international data bases.

| Journal Title | Print Only |
|---|---|
| **International Journal of Physiology** | **INR 9000** |

## NOTE FOR SUBSCRIBERS

- Advance payment required by cheque/demand draft in the name of **"Institute of Medico-Legal Publications"** payable at Noida, Uttar Pradesh.
- Cancellation not allowed except for duplicate payment.
- Claim must be made within six months from issue date.
- A free copy can be forwarded on request.